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Oral Health and its Implications in Late Pleistocene Western Eurasian Humans

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Dedicated to Lola

ABSTRACT OF THE DISSERTATION

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Systematic paleopathology research on the Late Pleistocene can provide a new perspective on the health, demographics and lifestyle of Paleolithic peoples; however oral pathologies, which can reveal both health and diet, have rarely been discussed beyond individual diagnoses. This project sampled Late Pleistocene humans from across Western Eurasia and collected data on dental and alveolar health, focusing on caries, periapical lesions, periodontal disease, and antemortem tooth loss. This research presents a number of new findings as well as reaffirming temporal patterns identified through other research lines (e.g., developmental stress and trauma), suggesting Early Upper Paleolithic peoples were healthier than the preceding Neandertals, but health declined around the Last Glacial Maximum in response to environmental degradation.

Caries prevalences are higher than any previous publication had estimated and reached an individual prevalence of over a quarter of the sample by the Late Upper Paleolithic; however, severe carious lesions and multiple affected teeth in one individual remain rare. Caries also pattern latitudinally with more caries along the Mediterranean, though this cline eases over time. This suggests that subsistence patterns varied regionally, but also shifted over time with the introduction of increased dietary carbohydrates well in advance of agriculture. Periapical lesions increased with age, but did not pattern over time or geography.

Periodontal disease was extensive in the Late Pleistocene. Early Upper Paleolithic modern humans have a slight decrease in disease severity relative to Neandertals, but the overall pattern of the Late Pleistocene is one of high morbidity. Periodontal disease also increases through the aging process, with all elderly individuals exhibiting at least mild alveolar resorption.

Neandertals have more tooth loss than Early Upper Paleolithic humans, suggesting comparisons between Neandertals and recent humans for this trait have produced dichotomies unrepresentative of the Upper Paleolithic transition. Tooth loss then increased again in the Late Upper Paleolithic, though this may represent a relaxing of tooth-loss related mortality.

All the pathologies except caries are correlated with one another suggesting age as approximated by dental wear and periodontal disease produce more tooth loss than caries. Subsistence shifts that occurred in response to cultural and environmental change produced differential health for Late Pleistocene groups, and oral disease was more common than previously thought.

Chapter 1: Introduction

The transition from the Middle to the Upper Paleolithic in Western Eurasia has been a major focus of recent paleobiological research for a number of reasons (See Table 1.1 for definitions of archaeological/ geological temporal terminology). First there is a relative abundance of human fossils and archaeological sites in Western Eurasia, so material is readily available, whereas preservation and access may limit research in other regions. Archaeological research also has a long antiquity in this part of the world, so there is a large canon of literature from which to draw. Finally the domination of modern humans over various other hominin groups has an anthropocentric appeal to anthropologists and the general public.

Previous researchers have principally approached the establishment of modern humans in the Upper Paleolithic from the perspectives of differential lithic technology (e.g., Bar-Yosef and Kuhn, 1999; Ambrose, 2001), subsistence (e.g., Grayson and Delpech, 2003; Richards and Trinkaus, 2009), symbolism (e.g., Lindly, 1990; McBrearty and Brooks, 2000; Henshilwood and Marean, 2003), or functional anatomy (e.g., Niewohner, 2001; Ruff et al., 2006; Maki, 2013) to explain the evolutionary success of one population—Early Modern Humans—over all other archaic hominin groups (White et al., 1982; Bar-Yosef, 2002), especially Neandertals in Europe because of the reasons detailed above. However, the key to modern humans' demographic ascendancy is fundamentally one of both differential mortality *and morbidity*, and this is often attributed to their "superior" behaviors in a variety of fields and their implications. Different subsistence patterns, technology, and social structure can result in improved health outcomes for the members of these groups, so inquiries into the differential health/morbidity of Neandertals and early modern humans could add to this Upper Paleolithic transition debate. Although topics

such as differential mortality (and its inherent paleontological biases), assessments of developmental non-specific stress indicators, and traumatic lesions and survival have been addressed in the literature, the richest potential source of data on Late Pleistocene morbidity—oral pathology—has never previously been systematically studied. Teeth preserve well taphonomically and interact directly with the environment, so their health can reflect the overall health of an individual, providing a unique opportunity to explore differential health in the past.

	Date Range	Comments
Middle Paleolithic	300,000-27,000 bp	Prepared core stone tool technology, e.g., Mousterian tool industries in Europe and Southwest Asia, used by multiple hominin taxa
Upper Paleolithic	50,000-10,000 bp	Fully modern tool technologies associated with modern humans (with some exceptions, i.e., Initial Upper Paleolithic industries in Europe associated with Neandertals)
Early Upper Paleolithic	50,000-22,000 bp	Upper Paleolithic tool industries, often dominated by stone blades, before the Last Glacial Maximum (e.g., Aurignacian, Gravettian) associated with the Earliest modern humans in Europe
Late Upper Paleolithic	22,000-10,000 bp	Tool industries after Last Glacial Maximum (e.g., Solutrean, Magdalenian); especially known for polychromatic parietal cave art, microliths
Epi-Paleolithic	18,000-10,500 bp	Regional term, usually applied to Southwest Asia and North Africa. Transitional period in Latest Upper Paleolithic before agriculture. Natufians are Late Epi-Paleolithic
Late Pleistocene	120,000-10,000 bp	Geological period
Holocene	10,000 bp-today	Geological period, also known as Anthropocene

Table 1.1: Definition of Temporal Terms (Hovers et al., 1998; Gorin-Morris, 2002; Mai et al., 2005)

Historically, pathology was the domain of physicians who considered little of the osteological effects of disease (Angel, 1981). Studies of skeletal pathology became more common post-World War II, and the methods refined on recent historical skeletal samples

became broadly available to bioarchaeologists and paleoanthropologists beginning in the 1970's (Buikstra and Cook, 1980; Angel, 1981; Ortner and Putschar, 1985). In large cemetery samples, questions of morbidity and pathology prevalence could be asked with sufficient sample size and largely complete individuals. Life history patterns were thought to be easily discernable and were initially considered analogous to public health studies on living populations.

However these methods are not always considered appropriate for use in fossil samples due to issues related to the Osteological Paradox (Wood et al., 1992). Unless one is looking at a catastrophic culling of a population (e.g., a volcano, flood, slaughter), the individuals who are dead are not necessarily an accurate portrayal of the age distribution and stress levels of a population in any point in time. Are individuals that die with evidence of stress (e.g., Harris lines, dental enamel hypoplasias) less healthy than their contemporaries because *they are dead,* or are they potentially healthier because they survived environmental insults that others died from—or rather they lived long enough to leave the signature of the survival of that assault on their bones? Unfortunately these questions pervade all studies of the dead and become exaggerated when one attempts to define samples that represent "real" populations in the Pleistocene (Wood et al., 1992). With the widespread availability of radiographs, CT scanning, isotope analysis, and DNA testing today, paleopathology research is increasing and broadening its scope within and outside bioarchaeology.

Much paleopathology research has focused on the Agricultural Revolution (e.g., Cohen and Armelagos, 1984; Oxenham et al., 2006; Starling and Stock, 2007; Tayles et al., 2009; Eshed et al., 2010). There was a primary dietary shift from wild gathered products to domesticated cereals and vegetables and secondary health and bodily stress shifts that occurred with this new subsistence pattern. Caries increased (Larsen, 1995) as did musculoskeletal indicators of stress

(Eshed et al., 2004), but the timing of this so-called revolution was different across the Old and New World. Other dental paleopathology research has tried to identify differences in oral health by sex (Peterson, 2002; Lukacs, 2008; 2011; Watson et al., 2010) or socioeconomic status (Pechenkina et al., 2002; Cucina and Tiesler, 2003; DeWitte and Bekvalac, 2010). However these studies of oral pathology have not been taken much further back than the Pleistocene/Holocene boundary. Despite some of the problems identified above, these methods can and should be applied to the deeper human fossil record.

Certain pathologies have been well studied in Neandertals and early modern humans. Dental enamel hypoplasias reflect bouts of severe stress during development (e.g., starvation, fever), and therefore have been popular for reconstructions of seasonal food stress in Pleistocene populations as well as by extension "overall health" in young individuals (Ogilvie et al., 1989; Brennan, 1991; Skinner, 1996; Hillson and Bond, 1997; Guatelli-Steinberg, 2004; Guatelli-Steinberg et al., 2004). Idiosyncratic examples of developmental pathology and trauma are published as individual descriptions or included in monographs (Wu et al., 2011; 2013 and references within). Only recently have broader, comparative analyses of pathology begun to be conducted (Berger and Trinkaus, 1995; Guatelli-Steinberg, 2004; Holt and Formicola, 2008). However oral pathologies are often only side notes in paleontological descriptions—if mentioned at all—lacking standardized categorization (with the partial exception of dental enamel hypoplasias) and rarely systemically comparing discrete groups. Considering the myriad of detailed oral pathology surveys in historic and pre-historic Holocene samples, this is surprising. Also, the little attention paid has been principally on the Neandertal specimens and their relative lack of oral pathology, and even less attention is given to the more abundant Upper Paleolithic modern humans who make a more logical contrast to Neandertals than Holocene samples. The

few studies that have looked at multiple individuals tend to use either narrow geographical (Brennan, 1991) or age sampling methods (Skinner, 1996; 1997) and/or examine a single pathology (Ogilvie et al., 1989; Guatelli-Steinberg et al., 2004).

In the context of previous Late Pleistocene pathology research, I have designed a comprehensive survey of oral pathologies from the available fossil material (i.e., mandibles, maxillae, and both *in situ* and loose teeth) of Late Pleistocene Eurasian humans across Europe and Southwest Asia using standardized radiographic and visual measurements and scoring methods from the dentistry and bioarchaeology literature. This work was designed to assess relative levels of oral health, and by extension, morbidity. The focus pathologies of this study are caries, periapical lesions, periodontal disease, and antemortem tooth loss, though rarer anomalies and other pathologies were also recorded. Distributions in age-adjusted oral lesion prevalence across these samples are used to test hypotheses on disease level changes between the Middle Paleolithic (Neandertals and early modern humans), Early Upper Paleolithic, Late Upper Paleolithic, and Epi-Paleolithic/Holocene temporally and taxonomically defined samples. The comparative Holocene samples were chosen because there is the possibility that "taxonomy" (i.e., Neandertals versus modern humans) has no role in differentiating pathology prevalence for hominin groups, and the difference is ultimately one of the Pleistocene versus the Holocene.

The Holocene, or last ~10,000 years (11,700 Cal BP (Walker et al., 2009)), was initiated with fluctuating global temperatures and saw increasing human population densities, and the establishment of agriculture and sedentary living (Gilead, 1988; Larsen, 2006). It has already been firmly demonstrated that agriculture precipitated a major shift in health for humans (Cohen and Armelagos, 1984; Larsen, 2006; Lukacs, 2008; Fields et al., 2009; Pinhasi and Stock, 2011). Detailing how oral health has changed over the last 120 ky provides an additional perspective on

Late Pleistocene modern human emergence and the health of those populations, especially when considered with the latest paleosubsistence and other paleobiology research. Culture does not equal biology, but studying oral pathology gives a biological context to the body's response to behavioral strain (e.g., diet, use of teeth-as-tools), and ultimately inferences can be made about the effects of these interactions on overall population health.

The oral health and overall health connection

Studies in modern populations show oral health is a good proxy for overall health status (Dolan et al., 1991; Gift and Atchison, 1995; Hujoel, 2009). Oral health shows a positive association with socio-economic status and general health (Samuelson et al., 1971; Dye and Thornton-Evans, 2010; Mashoto et al., 2010; Jamieson et al., 2011) and psychological well-being (Kiyak and Mulligan, 1987) and negative associations with mental stress (Marcenes and Sheiham, 1992) and cardiac disease (Slavkin and Baum, 2000; Meurman et al., 2004). Dental health research has an advantage over other skeletal indicators when dealing with fossils, because the preservation bias of teeth allows a greater possible sample size, and there is a medical literature supporting an oral pathology and overall morbidity association at the individual and population level (Dolan et al., 1991; Meurman and Hämäläinen, 2006; Hujoel, 2009).

Teeth are also the only mineralized portion of the body that interacts directly with the environment. This contact means that the oral cavity is often the first signal of poor diet and resultant poor overall health. In a meta-analysis of previous publications on oral health and systemic health, the oral cavity was identified as a "warning bell" for systemic diseases that hit later in life, namely cardiac disease, diabetes and cancer (Hujoel, 2009). The directionality of the association seems to indicate that a high carbohydrate diet causes oral pathology—specifically

caries and periodontal disease—in the short term (even in a matter of weeks) and systemic disease in the long term (Hujoel, 2009). This has broader implications for the interpretation of the results of this study when considered with Pleistocene dietary reconstructions. Various oral pathologies can confidently be used collectively to infer Late Pleistocene health as has been done with other bodily pathologies (Brennan, 1991; Steckel, 2003; Holt and Formicola, 2008) and may even be a superior data source in that the connection between the oral cavity and overall health is so well documented. The larger subfield of dental paleopathology is contextualized in the background chapter.

Research Questions and Hypotheses

Four hypotheses were devised to look at changes over time and region in oral pathology prevalence, nested within three broader research questions, namely: 1) Did oral pathology prevalence change significantly between the Holocene and Pleistocene when I remove agriculture as a variable (Hypothesis 1); 2) How did oral pathology prevalence change over time in the Late Pleistocene (Hypotheses 2 & 3); and 3) Did oral pathology prevalence vary geographically in the Late Pleistocene (Hypothesis 4)? The assumptions based on previous research specific to each hypothesis are discussed below.

$H1_0$. There are no significant differences between Late Pleistocene groups and Holocene pre-agricultural comparative samples- The documented changes between Pleistocene and Holocene groups, i.e., increased population density, decreased mobility from sedentism, more intense resource exploitation, etc., suggest that health and morbidity could have been evolving in response as well before the introduction of agriculture (Eshed et al., 2010). I expect to see greater prevalences of oral pathology in the Holocene groups because of changing subsistence patterns and the results of other paleopathology research (e.g., Frayer, 1989; Holt, 2003). Alternatively

there could be no significant differences between the Holocene and Pleistocene samples due to changing sources of morbidity, but not changing prevalence, and ultimately agriculture would be the major driver of morbidity changes for past humans in the early and mid-Holocene. In this scenario the null hypothesis would not be rejected.

$H2_0$. There are no significant differences between the Middle Paleolithic (Neandertals and modern humans) and Early Upper Paleolithic - Previous research suggests health improved in Early Upper Paleolithic modern humans relative to Middle Paleolithic Neandertals (Brennan, 1991; see review in Holt and Formicola, 2008), and this is what is predicted here. But if oral pathology prevalence increases in the Early Upper Paleolithic relative to the Middle Paleolithic Neandertals, it would suggest that morbidity levels increased in the Early Upper Paleolithic yet did not affect the success of Early Upper Paleolithic modern humans in Western Eurasia. Their colonization of the Old World would be in spite of their poor health, and other stress indicator research (stature: Formicola and Giannecchini, 1999; Formicola and Holt, 2007; Meiklejohn and Babb, 2011; dental enamel hypoplasias: Brennan, 1991; Skinner, 1996; iron deficiency: Brennan, 1991) does not support this hypothesis. If there are no differences, it suggests morbidity differences cannot explain the replacement of Neandertals by Early Upper Paleolithic modern humans. Middle Paleolithic modern humans are also sampled and compared against Early Upper Paleolithic modern humans to look at changes over time within one taxonomic group.

$H3_0$. There will are significant differences between the Early Upper Paleolithic and the Late Upper Paleolithic - Past studies indicate that health declined slightly in the Late Upper Paleolithic relative to the Early Upper Paleolithic (Frayer, 1989; Brennan, 1991; Formicola and Holt, 2007; 2008). I would therefore expect to see increased pathology prevalence over all age groups, i.e., pathologies affect individuals at a younger age or more intensely over the aging

process. The cultural/technological changes associated with the transition from the Early to Late Upper Paleolithic coincide with increased population density and environmental degradation around the time of the Last Glacial Maximum (Drucker et al., 2003; Bocquet-Appel et al., 2005). As there is already a well-documented increase in non-dental stress indicators in the Late Upper Paleolithic, if there is no increase in oral pathology prevalence, this would indicate that either oral health indicators do not reflect the appropriate type of population stress, or the increase in stress around the Last Glacial Maximum was mild enough to be non-significant statistically in increasing oral pathologies.

$H4_0$. There are no significant differences between the three identified regions of western Eurasia- Subsistence research that took regionality into account has found regional heterogeneity in the dietary resources utilized by Late Pleistocene humans (Aranguren et al., 2007; Henry and Piperno, 2008; Hardy, 2010; Revedin et al., 2010; Hardy and Moncel, 2011), which should predict differences in dental pathologies. Other research found decreased cultural heterogeneity across Europe in the Early Upper Paleolithic relative to the Middle Paleolithic that then increased again in the Late Upper Paleolithic (cf., Holt and Formicola, 2008). The higher Late Upper Paleolithic regional cultural heterogeneity is attributed to contracting preferred environments and a need to culturally differentiate one's group as population densities rose and group territory contracted in degraded environments. I would therefore expect morbidity differences across regions to also increase in response to varying subsistence and behavior.

Structure of the Thesis

I begin in chapter 2 with a background on research in the intersecting fields relevant for this study: dental anthropology, dentistry, bioarchaeology/paleopathology, and paleoanthropology. In chapter 3, the field, laboratory, and statistical methods of the research will

be described. Caries and periapical lesions (Chapter 4), periodontal disease (Chapter 5), and antemortem tooth loss and agenesis (Chapter 6) each have their own results and pathology specific discussion chapters. Each pathology has separate etiology and environmental and bodily correlates, so they each warrant their own results and discussion. Chapter 7 is a summary results and discussion chapter for all the pathologies, where covariance amongst the explored pathologies is tested and discussed, and the research results are used to test the four project hypotheses. The overall implications for overall health, subsistence and quality of life will also be discussed. Finally in chapter 8, the major findings are summarized and the project concluded with suggestions for broader implications and future directions.

Chapter 2: Background

Introduction:

This research project lays at the intersection of many different academic fields. This chapter begins with a history of the study of teeth within an anthropological framework. It is followed by descriptions of the focus pathologies, the history and methods of their study within dentistry and anthropology, and discuss their relationship to overall health. Much of this methods work for skeletal remains has been done within the subfields of osteo- and bioarchaeology. The scope can then be narrowed onto paleopathology research of Late Pleistocene specimens, which provides the specific precedent for the formulation of the four research hypotheses. Most of this literature resides within paleoanthropology. To provide context for some of the broader implications, other paleoanthropology research on Late Pleistocene diet, isotope analysis, oral bacteria, and non-pathological stress indicators will be reviewed. Once all these disparate fields are explored within the context of this project, I can focus on the specific holes present in the academic literature that this project investigates. By sourcing methods from and building on the results of dentistry, medical pathology, bioarchaeology, demography, global health, evolutionary medicine, and biological anthropology research, this paleoanthropology project can develop broad and interesting implications for its results and conclusions.

The Study of Teeth and their Pathology:

The human dentition and oral bone have always been a popular subject of study in paleoanthropology: they preserve well, they are relatively abundant, and they are the only hard tissue that interacts directly with the environment. Because of the plethora of anthropology research on teeth, dental anthropology is recognized as a distinct subfield, with its own society,

jargon and the other accouterments that comes along with having a named subfield. Teeth have intricate and diverse morphologies that are not particularly susceptible to environmental influences during development and tend to pattern across human geography (Scott and Turner, 1997). This allows a number of types of questions to be asked of teeth: namely questions of the relatedness of groups (from the presence of discrete traits and metric characters), diet (from dental wear intraspecies or cusp morphology interspecies), and health (from oral pathology). The latter two have principally been of interest to bioarchaeologists and the former of physical anthropologists. This may explain why paleoanthropologists, within physical anthropology, have spent little effort to document oral pathology in Pleistocene humans.

Historically the study of pathology was the domain of physicians, who viewed teeth-pulling barber surgeons as beneath them, and therefore thought little of the pathology of teeth until the last 200 years. Luckily certain physicians eventually took an amateur interest in archaeology and the field of paleopathology was born (see Hillson and Rose (2012)). Archaeological skeletal assemblages are dominanted by teeth and make a logical study subject. Now within the fields of bioarchaeology and paleopathology, dental pathology research is quite common. Methodology similar to the ones used in this study have been utilized in recent, large archaeological samples (e.g., Corruccini et al., 1987; Kerr, 1991; Marin et al., 2005; Caglar et al., 2007; Cucina et al., 2011). Radiographs are still integrated sporadically, but standardized scoring is typical with a number of the techniques now commonly used in dry skeletal samples being developed from these analyses (e.g., Costa 1982; Maat and van der Velde, 1987; Kerr, 1990; Lavigne and Molto, 1995; Lanfranco and Eggers, 2010). Multiple pathologies are scored when considered inter-related, such as caries and antemortem tooth loss (e.g., Costa, 1980a; 1980b; Lukacs, 1995; Molnar, 2008; Oxenham and Matsumura, 2008). Paleoanthropologists

unfortunately have not adopted many of these scoring methods, often developed by archaeologists in conjunction with members of dentistry departments. This hinders specimen comparisons across the literature.

Dentistry:

Pathologies:

Dental wear: Teeth are normally subject to wear from a variety of sources including food and foreign objects (abrasion), occlusal and interproximal tooth-on-tooth contact (attrition), and chemical etching and dissolution (erosion) (Begg, 1954). Because enamel and primary dentin are not remodeled, wear is progressive and irreversible. Additional dentin can fill in the pulp chamber as a mechanism to protect the tooth from pulpal exposure when wear rates are high, and this secondary dentin is visible on the occlusal surface and in radiographs (Hillson, 2000). Interproximal wear narrows the teeth and promotes mesial drift, shortening the dental arcade over time (Begg, 1954). If a tooth is lost, the now unopposed matching occlusal tooth may supererupt because occlusal forces alleviate (Hillson, 2000). All three kinds of wear and continuous eruption—i.e., slow, occlusal migration of a tooth over its lifetime—are normal changes in the life of a tooth, even though severe wear and continuous eruption may implicate or encourage other oral pathologies (e.g., root caries from root exposure, antemortem tooth loss from excessive eruption) (Kerr, 1990; Hillson, 2008).

Occlusal wear can be used to estimate age-at-death by comparing each tooth's degree of wear to a population appropriate attrition model calibrated by eruption sequence (see Chapter 3: Methods) (Miles, 1963; Brothwell, 1972; Smith, 1984). Error in all aging techniques increases with age, and correction is needed since more distal molars wear more slowly in addition to erupting later in development (Walker et al., 1991). Dental wear is correlated with a number of

the focus pathologies of the project—negatively with caries (Maat and van der Velde, 1987; but see Meiklejohn et al., 1992), causing (Brothwell, 1963) and accelerated by (Lovejoy, 1985) tooth loss—in addition to contributing to the calculation of the wear categories; therefore it is an important variable to record and consider when producing a pathology diagnosis.

Patterns of dental wear have been well documented in bioarchaeological studies as well as in fossil hominids (e.g., Molnar, 1971; Whittaker et al., 1985; Skinner, 1997; El Zaatari et al., 2011; Dawson and Brown, 2013). Pleistocene individuals show elevated levels of wear with age (across all age categories), similar to many non-industrial recent populations (Smith, 1984; Skinner, 1997; Rose and Ungar, 1998; Fiorenza et al., 2011). The "Attritional Occlusion model" assumes human teeth have evolved for high levels of wear, and mesial drift of posterior teeth, lingual tipping of anterior teeth, and continuous eruption of all the teeth are considered compensatory mechanisms for maintaining good oral health over an individual's lifespan in a high dental wear environment (Begg, 1954; Kaifu et al., 2003). This may explain higher rates of malocclusion in contemporary populations with very low levels of dental wear and partially explain our higher rates of dental disease—but this has yet to be tested (Kaifu et al., 2003). Reduction in dental wear in the same Holocene populations is assumed to correspond to reductions in overall tooth size, but other selective forces (e.g., oral infection avoidance) have also been presented as driving dental reduction (Calcagno and Gibson, 1991). In skeletal samples, numerous wear categorization schemes are available (e.g., Molnar, 1971; Scott, 1979; Smith, 1984; Bardsley, 2008) and wear is a generic change that all teeth, especially Pleistocene ones, are assumedly subjected.

Hypoplasia: Systemic disturbances, as well as oral trauma, that occur during odontogenesis can interrupt the activity of ameloblasts, cells that secrete enamel matrix and are

extremely sensitive to changes in metabolism (Commission on Oral Health, 1982; Hillson, 1996; Hillson and Bond, 1997). When their activity is depressed, ameloblasts excrete thin, poorly calcified enamel, or they may quit production permanently from prolonged stress (Hillson, 1996; Hillson and Rose, 2012). This produces visible defects on the enamel surface known as dental enamel hypoplasias. Perikymata are the small grooves in the external enamel produced by a normal pause in enamel production every seven to ten days of life (Commission on Oral Health, 1982). Analysis of the external perikymata—or internal Striae of Retzius (also known as Wilson bands) which are brown-colored planes within the structure of the crown—relative to a hypoplasia may be used to "date" within an approximately six month window of accuracy when a disturbance occurred in an individual's development, depending on the position of the arrest on the tooth and the tooth's formation timing (Rose et al., 1978; Hillson and Bond, 1997; Hillson and Rose, 2012). Dental enamel hypoplasias can range from random dots or dotted lines (pit enamel hypoplasia), to solid lines (linear or furrow enamel hypoplasias) or whole enamel cusps missing (planar or cuspal hypoplasias) depending on the timing, severity, and length at which the individual's health was compromised (Hillson, 1996; Ogden, 2008). Hypoplasias are generally recorded at a macroscopic level by type and distance of the disturbance from the cemento-enamal junction (CEJ) (Goodman and Armelagos, 1988; Brennan, 1991). At higher levels of magnification, the individual perikymata can be counted.

Dental enamel hypoplasias represent *stress* during development, and therefore not necessarily health (Hillson and Rose, 2012). There has been a recent push to clearly define and differentiate the terms "stress" and "health" in the anthropology literature (i.e., the issue had its own session at the 2013 American Association of Physical Anthropologists meetings (McIlvaine and Reitsema, 2013)), and the confounding of the two terms relates back to problems identified

within the Osteological Paradox (Wood et al., 1992). Are the individuals with dental enamel hypoplasias the healthiest individuals because they survived the insults on their bodies? The literature suggests otherwise. An increasing number of hypoplasias can have an increasingly negative effect on lifespan, i.e. increased mortality (Goodman and Armelagos, 1988). Also individuals often exhibit more than one hypoplasia, suggesting cyclically recurring stress such as seasonal food shortages (73.9% of affected southwestern French Pleistocene individuals had multiple hypoplasias (Brennan, 1991)) as well as multiple hypoplasias have a multiplying effect on increased mortality (Palubeckaitė et al., 2002).

As a result, dental enamel hypoplasias have been a popular research variable for those asking morbidity and "health" questions of fossil samples. These data have been collected globally on recent populations—skeletally (e.g., Duray, 1992; Keenleyside, 1998; Palubeckaitė et al., 2002; Cucina et al., 2006; Lieverse and Link, 2007; Starling and Stock, 2007) and through dental practices (e.g., Pascoe and Seow, 1994; Lai et al., 1997) —and back though the Pliocene and Pleistocene (e.g., Sognnaes, 1956; Johanson et al., 1982; Molnar and Molnar, 1985; White, 1988; Ogilvie et al., 1989; Brennan, 1991; Skinner, 1996; Hillson and Bond, 1997; Brunet et al., 2002; Guatelli-Steinberg, 2004; Guatelli-Steinberg et al., 2004, 2011; Lacruz et al., 2005). Dental enamel hypoplasias have been shown to be associated with low social status, poor childhood health (high infant morbidity), and poor nutrition.

Hypoplasias also predispose teeth to other further dental pathology. In a sample of Australian Aborigine children, where 99% of them had at least one tooth with hypoplasias due to high infant morbidity, dental caries were present predominately on teeth, which also had dental enamel hypoplasias (Pascoe and Seow, 1994). These affected teeth have additional furrows and pits for bacteria to thrive in, as well as thinner, compromised enamel that can be destroyed at a

more rapid rate when pH levels drop in the mouth. Hypoplasias represent physiological stress during dental development (*in utero* through approximately the age of sixteen when the third molar crown finishes forming (AlQahtani et al., 2010)) whereas the following pathologies can occur at any point during life, providing a broader perspective on individual morbidity.

Caries: Caries is a serious, pervasive issue in modern dentistry; however, before the advent of agriculture, caries is considered a relatively rare condition (though not unknown). Carious lesions are areas of demineralization caused by the secretions of acidogenic microorganisms (Scott and Turner, 1988; Hillson, 2008). Demineralized areas progress in alternating periods of rapid development and quiescence until they invade the dentine and pulp cavity and result in pulpal necrosis (Pine and Ten Bosch, 1996; Hillson, 2001). The formation of carious lesions is a complex process determined by the species of cariogenic bacteria, plus the interplay of host resistance (Wang et al., 2010) and dietary factors including the mineral content of local water supplies (Adatia, 1975; Scott and Turner, 1988; Hildebolt, 1987). Recent research on dental calculus suggests that the biodiversity of oral flora has actually decreased over time, resulting in a predominance of the caries-causing species in modern post-Industrial Revolution populations (Adler et al., 2013). Caries is often associated with agricultural practices (Larsen et al., 1991), but it is documented in nonhuman primates (Schultz, 1956), Middle Paleolithic humans (Sognnaes, 1956; Lalueza et al., 1993; Tillier et al., 1995; Lebel et al., 2001; Trinkaus and Pinilla, 2009; Walker et al., 2011; Lacy et al., 2012) and earlier hominin taxa (Carter, 1928; Brodrick, 1948; Robinson, 1952; Clement, 1956; Grine et al., 1990; Lacy, n.d.), though at significantly lower rates than modern populations.

Carious lesions are associated with increased risk for systemic disease (e.g., diabetes, heart disease) as well as general medical morbidity (Pascoe and Seow, 1994; Hujoel, 2009);

however, it appears that both caries and systemic diseases can be symptoms of excessive carbohydrate consumption over short (a matter of weeks and months) and long (decades) time scales, respectively (Hujoel, 2009). Without a lifestyle intervention, caries may be an early warning sign of systemic diseases to come later in life (Hujoel, 2009). Caries is also the most common chronic childhood disease in the United States with poor children suffering from twice as much dental disease as affluent ones (Bagramian et al., 2009). Poor children are less likely to seek or have access to treatment. Therefore in modern populations, caries is strongly negatively associated with overall health, but the causality is indirect at best and is likely a symptom of other related causes (e.g., diet, socioeconomic status).

Much caries research has been done on cemetery samples, but less on older archaeologically-derived samples (e.g., Nelson et al., 1999; Cucina and Tiesler, 2003; Delgado-Darias et al., 2006; Lieverse and Link, 2007; Liebe-Harkort, 2012; Halcrow et al., 2013). Because caries prevalence is closely related to diet (an observation first made by Mummery in 1870), caries prevalence has been used as data to test questions related to sex, socio-economic, and age differences in dietary patterns. Individuals who consume food at more frequent intervals (e.g., women and children), consume less high quality or diverse foods (e.g., low socio-economic individuals) or consume pre-processed foods (e.g., the very young and old) show higher rates of caries (Lukacs, 2008; 2011; Mashoto et al., 2010; Halcrow et al., 2013). Broken Hill, a Middle Pleistocene hominid from Kabwe, Zambia, has the most numerous and severe carious lesions known from the Pleistocene (Koritzer and St. Hoyme, 1979; Puech et al., 1980; Bartsiokas and Day, 1993; Lacy, n.d.). The specimen also has other pathology including multiple temporal lesions, and his poor oral health can easily be assumed to represent severe morbidity if not the ultimate cause of mortality in the individual (Montgomery and Williams, 1994).

There are only six currently recognized incidents of caries in Neandertals, <0.5% of known teeth (Walker et al., 2011). The reason for this has been debated: is it a diet low in carbohydrates with high wear; or the absense of severely cariogenic oral flora (Soltysiak, 2012; Tomczyk, 2012)? Research on oral flora preserved in the calculus of two Neandertals, from SW Asia and Central Europe, indicate climatically differing cariogenic bacteria species (Vandermeersch et al., 1994; Pap et al., 1995; Arensburg, 1996). This matches work on phytoliths in fossil calculus showing regional differences in plant foods consumed (Henry, 2011). Calculus, a mineralized deposit, has an inverse relationship with caries (demineralization) (Hillson, 2001). Calculus is common on fossil specimens (personal observation), and this, plus high levels of wear, may partially explain low caries incidence in these samples (Maat and van der Velde, 1987; Hillson et al., 2010).

Periapical lesions: Periapical lesions are cavities formed in the alveolar bone around the tooth root (usually the apex or periapical region), usually caused by a topical bacterial infection or pulpal death from infection, attrition, caries, or trauma (Scott and Turner, 1988; Dias and Tayles, 1997; Hillson, 2000). An inflammatory response in the periapical region may cause bone resorption around the tooth root, or pus can burst through the buccal or lingual alveolar plate through a fistulous tract (Dias and Tayles, 1997). This disease process can also cut off blood and nerve supply to the pulp cavity causing necrosis, if it was not the causal condition (Scott and Turner, 1988). As most periapical lesions identified in fossils by radiograph were not likely painful or causing systemic infection, the blanket term "periapical lesion" is more appropriate than "abscess" if a more specific diagnosis is unavailable (e.g., chronic or acute granulomata, cysts, osteomylitis, etc.) (Dias and Tayles, 1997).

Skeletal studies of periapical lesions are usually done in conjunction with caries research as they are often associated, at least in populations that have high caries rates (Keenleyside, 1998; Liebe-Harkort, 2012) or with wear (e.g., Kieser et al., 2001). Oral infections allow direct access for oral bacteria to enter the blood stream. Specific oral species have been found in arterial plaques associated with coronary artery disease, and bactermia and septicemia have direct mortality consequences (Williams et al., 2008). The oral cavity can, therefore, become both a source of infection from lesions and periodontal disease, and also a source of pro-inflammatory cytokines from the body's response to these pathologies (Gendron et al., 2000). Circulating inflammation products/mediators (e.g., cytokines) from both localized and general inflammation are associated with mortality and systemic disease (e.g., kidney disease) (Ioannidou et al., 2011). The increased rate of these associated systemic diseases and septicemia from vividans group *Streptococci* in Western societies has been suggested to be a result of increased dentalism, i.e., the retention of more teeth into old age (Rautemaa et al., 2007). Dental extraction is one of the oldest medical intervention utilized in cases of localized oral infection (Zias and Numeroff, 1986; Forshaw, 2009), as infections tend to clear up when the tooth is removed (O'Reilly and Claffey, 2000), but this is becoming less common with the availability of antibiotics in recent clinical settings. Excessive occlusal wear is also correlated with periapical lesions because of the potential for pulpal exposure and general trauma to the alveolus (Clarke and Hirsch, 1991; Kieser et al., 2001; Molnar, 2008). Individuals with evidence of infection in archaeological contexts unsurprisingly show decreased life expectancy (Goodman and Armelagos, 1989).

Some population studies have been done of periapical lesions in modern humans (e.g., Abbott, 2004), but there is little systematic research in fossils, just individual diagnoses

(Dastugue, 1967; Heim, 1976; Trinkaus, 1985; Trinkaus et al., 2006; 2014; Mann et al., 2007; Shang and Trinkaus, 2010; Liu et al., 2011; Condemi et al., 2012; Lacy et al., 2012). This is likely because caries rates are low in fossil Pleistocene populations, and periapical lesions are often assumed to be the product of extreme carious lesions—i.e. periapical lesions were assumed to be not present if not visible in the absence of caries. Today, with the widespread availability of radiography and computed tomography imaging, it would be easy to test this assumption. Data on general "abscesses" in Late Pleistocene fossils has been collected, but since no significant patterns was found, only the per-individual prevalences were published (Frayer, 1989)

Antemortem tooth loss: Tooth loss can be caused by severe attrition, trauma, or any other oral pathology that either kills the dental pulp or destroys the bone or periodontal ligaments holding the tooth in the alveolus (Scott and Turner, 1988; Hillson, 2000; Bahrami et al., 2008). Some researchers question the assumed relationship between periodontal disease and antemortem tooth loss because supporting data are sparse especially among skeletal studies (Costa, 1980a; Clarke et al., 1986; Kerr, 1991). However, in a recent human dentistry study, periodontal disease was responsible for tooth loss in fewer patients than other causes, but it was responsible for the loss of more individual teeth overall than any other cause (Al-Shammari et al., 2005). In other words, if periodontal disease is severe enough to cause tooth loss, it will cause a higher number of teeth to be lost in that individual than other causes (e.g., caries, fractures) (Al-Shammari et al., 2005). There is also an inverse relationship between the number of teeth present and chronic heart disease suggesting that tooth loss, or rather its causes, have a negative impact on overall health/morbidity. Periodontal disease and less than ten teeth present was associated with a 25-30% increased risk of chronic heart disease (Cullinan et al., 2009).

Antemortem tooth loss has been recorded in skeletal studies, though often in tandem with other pathologies, namely caries (e.g., Costa, 1980a; Keenleyside, 1998; Nelson et al., 1999; Cucina and Tiesler, 2003; Lieverse and Link, 2007; Liebe-Harkort, 2012). Since it is assumed, at least in agricultural populations, that much tooth loss is caries related, a caries correction factor can be derived to estimate how many teeth lost were due to caries (Lukacs, 1995; Márquez-Grant, 2009). The relationship between other pathologies and tooth loss in skeletal samples has not been thoroughly explored though (e.g., periodontal disease, trauma (cf. Lukacs, 2007)).

It must be noted that not every individual has 32 teeth to begin with when scoring antemortem tooth loss (Hillson, 2001), including some Late Pleistocene specimens (Heim and Granat, 1995; Hillson, 2006). However these are pathological developmental or genetic examples of agenesis, and therefore are not related to antemortem tooth loss, as a tooth was never present (Agenesis is reported here with antemortem tooth loss in chapter six). Antemortem tooth loss is known from the Late Pleistocene (Sergi, 1974; Trinkaus, 1983; 1985; Tappen, 1985; Shang and Trinkaus, 2010), but rarely scored systematically or compared between groups or individuals (cf. Gilmore, 2011; n.d.).

Periodontal disease: Periodontal disease is a complex process that often occurs with other pathologies and is the most prevalent chronic infection in modern humans (Rautemaa et al., 2007). It has two levels: gingivitis, which affects only the gum margins and 95% of recent people; and periodontitis, which results in destruction of the alveolar bony crests and periodontal ligaments and is present in between 10%-56% of recent human samples (Clarke et al., 1986; Jenkins and Kinane, 1989; Kerr, 1991; Oliver et al., 1998; Hugoson et al, 2008; Eke et al., 2012). Only periodontitis leaves a signature skeletally through degeneration of the alveolar bone (Clarke et al., 1986; Jenkins and Kinane, 1989; Kerr, 1991; Oliver et al., 1998; Hugoson et al, 2008; Eke

et al., 2012). Plaque deposits predispose one to periodontal disease by inflaming the gingiva and giving a home to bacterial biofilm (Hillson, 2001).

The modern dentistry definition of periodontal disease is a distance of >2 mm between the cervico-enamel junction and the alveolar crest (CEJ-AC) (Clarke et al., 1986; Kerr, 1991; Hillson, 2000; Ogden, 2008). This definition is problematic in fossils because 1) alveolar crests preserve poorly, and 2) it assumes little dental wear or continuous eruption. Therefore levels of wear, crest morphology, and porosity should also be scored in dry bone to avoid confusion (Costa, 1982; Kerr, 1988; Clarke et al., 1986). However, it has been argued that generalized horizontal bone loss from periodontal disease is rare in archaeological assemblages (i.e., it is often localized) and therefore unlikely to be confused with arch-wide continuous eruption from general dental wear (Clarke et al., 1986; Clarke, 1990; Kaifu et al., 2003). Localized supraeruption is possible though, so antemortem tooth loss and unusual wear patterns should also be taken into account.

Periodontal disease is correlated with many other diseases in recent modern humans, such as cardiac disease (Slavkin and Baum, 2000; Meurman et al., 2004; Williams et al., 2008), diabetes (Garcia et al., 2001; Hujoel, 2009), low birth weight of offspring, osteoporosis, arthritis (Ogden, 2008 and citations within), preeclampsia (Shetty et al. 2010), and others. The directionality of these relationships is complicated though, and periodontal and other systemic diseases may both be symptoms of another larger cause (carbohydrate consumption: Hujoel, 2009, genetics: see Cullinan et al., 2009 for full discussion). There are a number of potential mechanisms for these inter-disease correlations including: common genetic predispostions, periodontopathic bacteria entering the blood stream, cross-reactivity of bacterial and human heat-shock proteins (antibodies), inflammation and its mediators, and obesity and its associations

(Cullinan et al., 2009). Eating a high amount of carbohydrates can directly cause periodontal disease by feeding periodontogenic oral bacteria, but it can also cause obesity, which leads to systemic inflammation, diabetes, and coronary heart disease, all of which are also associated with periodontal disease (Cullinan et al., 2009; Hujoel, 2009). Because of the complexity of these relationships, the causalities have not been fully identified, and the call for more research continues to be made under the new cross-disciplinary term "periodontal medicine" (Garcia et al., 2001). The exact connections between periodontal disease and other systemic health issues are only beginning to be more fully explored.

Ultimately periodontal disease and poor oral health directly affect mortality. Periodontitis is associated with a 46% increase in mortality risk for 25-74 year olds (DeStefano et al., 1993), and mortality increases with poor oral health even when deaths from cardiovascular disease are removed (Jansson et al., 2002). Life history profiles are also affected by fertility, and periodontal disease has a number of effects on reproductive women including intensified periodontal disease during pregnancy, preeclampsia, decreased birth weight of infants born to those women, as well as spontaneous abortion and pre-term birth (Cohen et al., 1969; Garcia et al., 2001; Lieff et al., 2004; Shetty et al., 2010). Dental plaque, as well as horizontal tooth mobility, increases throughout pregnancy with tooth mobility decreasing post-birth, but not to pre-pregnancy levels (Cohen et al., 1969). This is likely where the old adage "a tooth per child" comes from, referring to tooth loss of periodontal disease origin during and immediately after pregnancy (Lanfranco and Eggers, 2012). Whether relaxin or other pregnancy-related hormones are directly involved is untested. Lower fetal birth weight is also associated with developing hypertension, diabetes and high cholesterol in these infants as adults (Trevathan, 2007; Baker et al., 2008), suggesting multigenerational effects of periodontal disease.

These pregnancy-related periodontal changes also tend to be most severe in black women, women who smoked during pregnancy, and those on public assistance, all three variables associated with lower socio-economic status in the United States (Lieff et al., 2004). This suggests that the subset of women who are most susceptible to pregnancy-aggrevated periodontal disease are also those with the least access to professional oral care. The implications of this for the deeper fossil record are unclear though. It can at least be said that populations suffering from high levels of periodontal disease are also likely to be experiencing higher mortality and possibly some negative effect on fertility compared with those populations with lower rates of periodontal disease.

Assessments of periodontal disease have been done in many historic and recent pre-historic samples (Costa, 1982; Ronderos et al., 2001; Delgado-Darias et al., 2006; Wasterlain et al., 2011; Marin et al., 2012). Periodontal disease is associated with both mortality and low social status (Keenleyside, 1998; DeWitte and Bekvalac, 2010; 2011). However with a skeletal sample, the cumulative effect of periodontal disease over one's life is assessed and not current disease activity at the time of death, making comparisons with modern living human studies problematic (Garcia et al., 2001). Methods for diagnosing periodontal disease in skeletal material are well accepted in bioarchaeology now, but have yet to be widely applied to the fossil record.

As an exception, Brennan (1991) noted periodontal disease without scores in Southwestern French Late Pleistocene humans and found that the prevalence decreased in the Late Upper Paleolithic from the Early Upper Paleolithic. The Krapina Neandertal sample shows more labial/buccal side alveolar resorption than historic and modern populations that show more interdental resorption (Topić et al., 2012). A few monographs or articles have also mentioned the periodontal status of their subjects, but without populational context (Neandertals: Banyoles

(Lalueza et al., 1993); El Sidron 2 (Prieto, 2005); Guattari 2 and 3 (Mallegni, 1995); Zafarraya (Torrent, 1997); Modern humans: Caldeirao 1 (Trinkaus et al., 2001); Cova Foradá (Lozano et al., 2013); Dolní Věstonice 16 (Trinkaus et al., 2006); Mladeč 2 and 8 (Teschler-Nicola et al., 2006); Skhul 5 (Smith, 1977)). Some authors have acknowledged that periodontal disease rates may be high in the Pleistocene based on anecdotal evidence, but have had no studies or data to reference (Calcagno and Gibson, 1991; Lanfranco and Eggers, 2010). On a population level, high rates of periodontal disease may indicate high general morbidity. Modern studies of the ways that periodontal disease differentially affects individuals across populations along various variables can be used to seek more specific implications for individual fossil diagnoses and sample wide patterns.

Oral Health and Overall Health:

The above pathologies should not be viewed independently from one another. All can be co-morbidities and the cause or product of a number of other disease processes. For example dental wear may inhibit caries, but result in pulpal pathology (Maat and van der Velde, 1987; Molnar, 2008). When providing a diagnosis for an individual, all of the available information on their oral health can and should be weighted together. Surveying multiple orodental disease processes and their frequencies at a population level will elucidate more distinguishable and informative patterns than individual diagnoses. Some of this has been done with modern samples through dentistry and public health research using an oral health score. Jansson et al. (2002) made an overall health score for their study on the relationship between oral health and mortality in a modern Swedish sample with detailed dental records and death certificates. Their score included four variables divided by the maximum value: total lost number of teeth, marginal bone loss, number of teeth with caries and number of teeth with periapical lesions/abscesses. The

scores therefore ranged from 0-4 with the oldest age category who had died during the sample period having an average score of 1.1. With a skeletal sample that contains many incomplete individuals, this score could still be utilized as it is a proportion. Brennan (1991) made a score to indicate change in health indicators over time by dividing the N of total body stress indicators showing the same trend (increase or decrease) divided by the total number of variables examined minus one. This was used with Late Pleistocene human samples, and was adapted for this study.

There is an assumption that oral health—as a whole through the aggregate of multiple pathology indicators—represents the overall health of an individual to a certain degree. The assumption is well founded in the public health literature, and a number of these studies are referenced above per pathology (e.g., Garcia et al., 2001; Migliorati and Madrid, 2007; Rautemaa et al., 2007; Williams et al., 2008; Cullinan et al., 2009; Hujoel, 2009). Because most recent human tooth loss is the result of caries and periodontal disease and their resultant infectious lesions, these four pathologies are closely related and have warranted general oral health assessments. Poor pooled oral health is associated with increased mortality (Jansson et al., 2002), heart disease (Meurman et al., 2004), and decreased quality of life measures (Gift and Atchison, 1995). If one views the oral cavity as a constant potential source of infection and inflammation because of its flora and direct environmental interaction, any oral pathology can potentially have systemic affects on the individual resulting in morbidity and even mortality (Gendron et al., 2000). At a population level, high oral pathology rates have already been shown to produce negative affects on life span and quality of life in recent modern human (e.g., Gift and Atchison, 1995; Jansson et al., 2002) and skeletal studies (e.g., Palubeckaitė et al., 2002; DeWitte and Bekvalac, 2010). For fossil samples, one can not have access to life-long medical histories, and therefore it is the cumulative affects of stress on the individual that is being

observed. However one can infer health at the time of death for the individual from oral pathology, and at a population level, these values will inform morbidity prevalence.

Background of Samples:

Comparative Samples:

The Natufians lived during the Final Late Pleistocene (13,100 - 9650 calibrated BC) in Southwest Asia (Eshed et al., 2010), and were the culture from which agriculture in the region would arise. They were mostly sedentary unlike other Late Pleistocene peoples, but still practicing hunter-gatherer subsistence, gathering wild cereals and hunting wild ungulates like gazelle (McCorriston and Hole, 1991; Nadel and Hershkovitz, 1991; Eshed et al., 2010). Though they were consuming a diet similar to later agricultural peoples, i.e., large amounts of processed grains, they were still generally living a Pleistocene lifestyle, but with decreased mobility (Eshed et al., 2004). Because the shifts in diet and health are so marked with the origins of agriculture (Cohen and Armelagos, 1984), the Natufians have been a popular study sample in that they can be used to test hypothesis about whether diet or lifestyle were driving changes seen in the Neolithic (Smith and Peretz, 1986; Nadel and Hershkovitz, 1991; Eshed et al., 2004; 2010).

The North American samples from Indian Knoll and Point Hope (Ipiutak) have also been the subject of multiple studies, and they vary considerably in diet and lifestyle. The Indian Knoll peoples were Archaic period (4,500-6,100 BP (Winters, 1974)), pre-agriculturalists in the American Midwest woodlands (Leigh, 1925; Webb, 1974). Though they are considered pre-agricultural, there is evidence for some small-scale garden agriculture and intense processing of gathered food items (Leigh, 1925; Cassidy, 1972; Webb, 1974). The lifespan of the people from Indian Knoll was similar to that of other hunter-gatherers (Johnston and Snow, 1961), and this population was physically stressed (Cassidy, 1972; Perzigian, 1977). The Point Hope peoples

include the Ipiutak and Tigara, though only the Ipiutak were included in this study. The Point Hope Ipiutak hunted caribou and utilized marine resources in what is now Alaska from 100 BC to 500 AD, but were not reliant on whale hunting like the later Tigara (Rainey, 1941; 1971; Larsen and Rainey, 1948). Their use of large terrestrial mammals and high protein consumption suggest a diet roughly similar to Late Pleistocene Europeans, justifying their selection as a comparative sample (e.g., Guatelli-Steinberg et al., 2004; Krueger and Ungar, 2012).

Considering their large sample sizes and accessibility, the three chosen comparative samples have all been used previously for paleopathology research within bioarchaeology and physical anthropology. The majority of the dental research on the Indian Knoll material has focused on dental wear and crown morphology (Rabkin, 1943; Perzigian, 1976). The Point Hope material was previously analyzed for wear, antemortem tooth loss, caries and periodontal disease, but since this research was conducted, newer scoring methods have been developed (Costa, 1977; 1980a; 1980b; 1982). Also radiographs were not taken, and therefore bone remodeling from periapical lesions and hypercementosis could not be fully assessed. A recent study of dental and overall health indicators (including caries, antemortem tooth loss, and "alveolar defects") in Hokkaido Jomon and Okhotsk used Costa's published data as their comparative group (Oxenham and Matsumura, 2008).

The Natufian dentition has been extensively surveyed in the past (Smith 1970; 1972). A more recent study focused on wear and pathology in Natufian and Neolithic peoples from the Levant, but did not use radiographs (Eshed et al., 2006). Scoring of pathologies was less detailed than was used here. Eshed and colleagues (2006) cite that their lack of radiography limits their assessment of "abscesses", and therefore they could not use the preferred methods of Dias and Tayles (1997). In this study periodontal disease was not scored beyond present/absent, and wear

was only recorded for the first molar. The Holocene human data desired for this project were therefore not available in the published literature and needed to be collected; however, where methods are similar, some publications can be used to provide more comparisons per pathology (see specific results chapters). These collections were chosen because they are all Holocene or Epi-Paleolithic collections of individuals not practicing true agriculture, but diverse hunter-gatherer subsistence patterns. They are also collections with a large number of relatively complete individuals available for study.

Late Pleistocene Sample:

The Neandertals were a taxonomic group of humans in Europe and Southwest and Central Asia from 250-27 kya known for possessing a distinctive suite of morphological characteristics that differentiate them from other contemporaneous human groups (Stringer and Gamble, 1993; Trinkaus and Shipman, 1993; Tattersall, 1995; Mellars, 1996). They were physically well adapted for cold environments (Ruff, 1994; Holliday, 1997; Churchill, 1998; Steegmann et al., 2002; Weaver, 2003) and utilized the large terrestrial mammal resources available (e.g., Bar-Yosef, 2004; Bocherens, 2009; Rivals et al., 2009). Because Neandertals were the earliest identified fossil human that was not "modern", there is a long history of their study (King, 1864; Trinkaus and Shipman, 1993). Much of this research has focused on differentiating Neandertals from modern humans, especially from the perspective that they are less "evolved" than *Homo sapiens sapiens* (cf. Trinkaus, 2013).

Modern humans arose ~200 kya in East Africa (White et al., 2003; McDougall et al., 2005), spread into Southwest Asia (Bar-Yosef, 1994) and South China (Liu et al., 2010; Shen et al., 2013) after 100 kya, and by 30 kya were the only hominin group on Earth (Trinkaus, 2005; Stringer, 2012), barring the late survival of *Homo floresiensis* in Indonesia (Brown et al., 2004).

Though these fossil modern humans are within the range of variation of recent humans, there were still changes over time in body size and shape as they adapted to their newly colonized regions (cf. Holt and Formicola, 2008). And to do this massive expansion across the Old World, other hominin groups had to be displaced or absorbed (Stringer, 2002; Templeton, 2002; Conard, 2006; O'Connell, 2006). This shift from modern human to Neandertal anatomy and associated cultural traditions in Western Eurasia is described as the Upper Paleolithic transition, even though there is evidence that late Neandertals were using Upper Paleolithic cultural industries (i.e., culture change does not equal biological change) (Hublin et al., 1996; Gravina et al., 2005; Peresani, 2008). This project aims to contribute to the research around the causal factors in this shift. The previous health and subsistence research conducted on these Late Pleistocene humans, including that designed specifically around the Upper Paleolithic transition, are discussed below.

Current Late Pleistocene Research:

Paleopathology Studies:

Past studies which sought to answer questions of Late Pleistocene stress and morbidity have generally focused on developmental stress indicators, traumatic lesions, and developmental disorders. The few that included any dental components are addressed in the next section. The latter two types of research studies (lesions and developmental disorders) have been concerned principally with culturally mediated differential risk, survival and mortuary treatment in additional to differential diagnoses of lesions (Trinkaus 2005b; Formicola, 2007; Trinkaus and Buzhilova, 2012; Wu et al., 2011). The first (e.g., Harris/Transverse lines and dental enamel hypoplasias) represent stress during development and have been studied, sometimes in tandem, to identify general populational stress levels, though they actually only reveal stress that occurred while the individual was young (Ogilvie et al., 1989; Brennan, 1991; Skinner, 1996; Guatelli-

Steinberg et al., 2004). Other more general indicators have also been studied including stature (Formicola and Giannecchini, 1999; Formicola and Holt, 2007; Meiklejohn and Babb, 2011), robusticity (Frayer, 1981; Ruff et al., 1993; 1994; Trinkaus et al., 1994; Churchill, 1998; Shackelford, 2005) and body proportions (Holliday, 1997; 1999). These do not represent any specific periods of hardship, but general trends of health, activity, and nutrition over time (see Holt and Formicola (2008) and Trinkaus (2013) for summaries). Only a few of the more systematic Late Pleistocene paleopathology studies have incorporated oral pathology.

Dental Fossil Pathology Studies:

Any Pleistocene dental paleopathology research has generally focused on individuals. Monographs on specific fossils have addressed the issue for their subject sample and typically at a macroscopic level without scoring or detailed descriptions of the lesion (e.g., Carter, 1928; Borgognini et al., 1980; Molnar and Molnar 1985; Tillier et al., 1989; Tillier et al., 1995; Buzhilova 2000; Lebel et al., 2001; Trinkaus et al., 2006; 2014; Liu et al., 2010; Shang and Trinkaus, 2010; Walker et al. 2011; Condemi et al., 2012). If they summarize other known cases of the pathology, it is not done systematically. Interproximal caries and periapical lesions are likely underestimated in more complete specimens as radiographs are rarely taken. A few wider fossil surveys have been conducted (e.g., Frayer, 1989; Ogilvie et al., 1989; Brennan, 1991; Skinner, 1996, 1997; Guatelli-Steinberg et al., 2004; Gilmore, 2011; n.d.), but all focused on a single pathology or did not use current methodologies and recently discovered specimens—as well as some older specimens. Their work, however, lays the groundwork for my research aims.

The broader studies have focused primarily on dental enamel hypoplasia. Skinner's (1996; 1997) work on wear and hypoplasias in immature Late Pleistocene individuals from Europe was thorough, but his sample was biased towards Late Upper Paleolithic adolescent

specimens. Only hypoplasias were scored, and only from juveniles, i.e., those who did not survive into adulthood, which may distort the interpretation of the overall stress levels of the population (Wood et al., 1992). Others have looked at dental enamel hypoplasias from Neandertal samples (Ogilvie et al., 1989; Guatelli-Steinberg et al., 2004), but not a broad sample of early modern humans.

Brennan (1991) surveyed Middle and Upper Paleolithic individuals in southwestern France for dental enamel hypoplasias, caries, periodontal disease, and a variety of non-dental stress indicators. Caries increased over time, but radiographs were taken only if a lesion was unclear. Periodontal disease increased in the Late Upper Paleolithic. Stress indicators were pooled to compute a Health Stress Index (positive change from Middle Paleolithic to Early Upper Paleolithic, negative from Early Upper Paleolithic to Late Upper Paleolithic). Brennan's samples were constricted in geography, and therefore, her interpretations may not be applicable to other regions.

Frayer (1989) examined caries, alveolar disease, and antemortem tooth loss in Early Upper Paleolithic, Late Upper Paleolithic, and Mesolithic modern humans in Europe. Caries increased continuously over his chronological sample with none being found in the Early Upper Paleolithic. More recent research that used more detailed temporal variables suggests caries may have decreased at the end of the Paleolithic and inflected upwards again in the Mesolithic (Caselitz, 1998). Frayer (1989) also showed that caries prevalence correlated negatively with latitude in the Mesolithic and emphasized a possible transition from a high protein to a high carbohydrate diet beginning in the Late Upper Paleolithic (also Cachel, 1997). This suggests a null hypothesis to be tested with the data from this study. Alveolar disease increased (no scores used), and Frayer felt this could not be attributed to carbohydrates, but did not speculate further

into the implications of this pattern. Antemortem tooth loss did not correlate with time or any other variables. Radiographs and severity scores were not utilized, and some casts and photographs were used instead of original specimens.

Gilmore (2011) has noted less antemortem tooth loss in Neandertals than recent humans, which she attributed to less "modern" behavior in her sample of 26 Neandertals compared with Holocene recent humans (The sample is reported as containing 27 Neandertals, but Guattari 1 and 2 are most likely the same individual (Mallegni, 1991)). Specimen age was not accounted for, and radiographs were not used. The continuation of this research has included non-human primates (Gilmore, 2013), and a more detailed study is forthcoming (Gilmore, n.d.).

There are serious gaps in research on dental pathology and oral health in Late Pleistocene Western Eurasia, even though non-dental paleopathology research results supply predictions for this project. First, previous samples have all had spatial, geochronological, and/or age constraints. Second, the focus has generally been on dental enamel hypoplasias; this is a developmental defect and may only predispose individuals to degenerative processes later in life. Third, there is a dearth of radiographic imaging and standardized scoring methods in fossil dental pathology research. The research in the following chapters is designed to remedy these omissions and expand our understanding of human differential stress through the Late Pleistocene.

Demography Issues:

Mortality patterns have been assessed for Pleistocene *Homo,* and especially for Neandertals and modern humans, to explain the Upper Paleolithic transition (Caspari and Lee, 2004; Trinkaus, 2011). No Late Pleistocene differences in mortality profiles were identified (Trinkaus, 2011), but multiple factors affect such profiles (Trinkaus, 1995). Morbidity is likely to be a better signal of past populational stress, because it represents long-term stressors. Mean-age-

at-death is also not a reliable statistic of life expectancy because differential fertility rates drives mortality profiles as much as differential mortality (Sattenspiel and Harpending, 1983), and populations were likely unstable throughout the Middle and Upper Paleolithic (Trinkaus, 1995; Hovers and Belfer-Cohen, 2006; Powell et al., 2009; Wu et al., 2013). Otherwise healthy individuals can die from accidents and swiftly progressing disease.

The effects of mortality and life history profiles on pathology prevalence need to be taken into account with the interpretation of this study's results as all these dental pathologies increase with age, i.e., overall prevalences are directly a result of the age distribution of the sample. It has been argued that Neandertals and modern humans have different developmental timing (Dean et al., 1986; Rozzi and deCastro, 2004; Smith et al., 2007; 2010), but this is difficult to account for (Dental wear is used to age individuals in this study). Interestingly enough periodontal disease is reported to have multiple negative reproductive consequences (e.g., pre-term birth, spontaneous abortions, low birth weight, etc.), and its effect on fertility is something that can be explored further with the discussion of the results of this research. Since differential mortality has been unable to distinguish Middle Paleolithic and Upper Paleolithic humans, differential morbidity may be able to do so.

Subsistence Issues:

Recent work has focused on the degree of change in Late Pleistocene diets using faunal collections (e.g., Marean and Kim, 1998; Grayson and Delpech 2003; Bar-Yosef, 2004; Adler et al., 2006) and stable isotopes to assess dietary protein sources (e.g., Bocherens et al., 2001; Richards et al., 2008; Richards and Trinkaus, 2009); and organic residues on lithics plus phytoliths and starch grains in dental calculus and grindstones to identify possible food plants (Hardy et al., 2001; Lev et al., 2005; Revedin et al., 2010; Hardy and Moncel, 2011; Henry,

2011; Henry et al., 2011; Adler et al., 2013). The elucidation of Paleolithic diets provides a framework for assessments of oral pathology in that periodontal disease has correlations with protein deficiency (Kerr, 1962) and both caries and periodontal disease are associated carbohydrate consumption (Larsen et al., 1991; Hujoel, 2009), and species of oral flora (Loesche, 1996; Adler et al., 2013; Warinner, 2013). Yet, as procurement technology evolved through the Late Pleistocene, there appears to have been little change in the dietary resources exploited, suggesting that ecozonal variation in available foods best explains dietary shifts that may relate to aspects of oral health (Stiner, 1994; Hardy, 2010; Fiorenza et al., 2011; Henry, 2011; Trinkaus, 2013). Therefore I would not expect subsistence differences to explain Neandertal and early modern humans' differential oral health and hence morbidity, although oral health patterns may be variable across regions reflecting regional subsistence differences (Fiorenza et al., 2011; Henry, 2011) (see project hypothesis four). Reduced environmental productivity, changing foraging costs, decreased mobility, resource use intensification, and increasing cultural heterogeneity around 20 kya may also have exaggerated these regional issues (cf., Holt and Formicola, 2008).

Evolutionary Medicine:

Modern human populational health is a partially a result of the specific population's adaptations—though recent human mirgration, colonial history, etc. are confounding factors today (see debate in Farmer et al., 2013). But how does our current environment reflect the environment within which the majority of the human evolution has taken place? Evolutionary medicine is a paradigm through which modern biological responses to pathology and infection can be interpreted by asking questions about how adaptive are these responses if one assumes a "Stone Age" environment (Williams and Nesse, 1991; Nesse and Williams, 2008; Trevathan,

2007; Gluckman et al., 2009). As far as oral health, there has been a "host-parasite arms race" between cariogenic and periodontopathic oral flora and the human host (Williams and Nesse, 1991; Warinner et al., 2014). What about the presentation of periodontal disease and caries are actually an adaptive response on the part of the host to dealing with these bacteria? The swelling, inflammation and fever associated with periapical lesions, periodontitis, and other oral infections have been explored as far their adaptive role. Besides being side effects of the immune systems response to infection, fever may make the host less aminable to the bacteria as well as encouraging the liver to store iron and therefore robbing the bacteria of a necessary element (Bullen, 1981; Kluger, 1991).

Only a few previous studies have used this paradigm to ask questions about Pleistocene dentition. The decrease in dental wear in (relatively) recent times could be driving an increase in oral disease as our dentition evolved in a high wear environment (Kaifu et al., 2003). Scissor occlusion of the anterior teeth (with overjet and underbite) and the interlocking on the cusps of the posterior teeth—which in modern dentistry is considered a normal condition—may actually be a retention of the juvenile condition due to minimal amounts of wear even in adults (Kaifu et al., 2003). This is encouraging malocclusion, impacted teeth, and larger interdental spaces for plaque to accumulate and promote periodontal disease, caries, and periapical lesions, as well as temporomandibular joint dysfunction and arthritis.

Dental reduction may also be an adaptation in minimizing oral pathology. The rate of tooth reduction in the Holocene is twice that of the Upper Paleolithic (Brace et al., 1987), and there is a 45% reduction in occlusal area of the posterior teeth in early *Homo* through the Neolithic (Calcagno and Gibson, 1991). Some have argued this is merely the result of decreased selection for large teeth through reduced wear causing either genetic drift or a metabolic savings

known as the "probable mutation effect" (Brace, 1964; McKee, 1984). But even a minor reduction of oral pathology through smaller teeth could be enough to drive that selection. Calcagno and Gibson (1991) suggest that smaller, less complex teeth confer resistance to caries and periodontal disease in a low wear environment (i.e., soft, less abrasive diet) by minimizing crowding/malocclusion and interdental spaces as well as producing teeth with less crenulous surfaces for cariogenic bacteria to thrive (this latter assertion is not convincing as there is no reference for smaller teeth being less morphologically complex). They acknowledge that they do not have the data to test this hypothesis in the Pleistocene, though they assume high periodontal disease in the Upper Paleolithic based on the available radiographs (Skinner and Sperber, 1982).

Genes that predispose individuals to caries have already been identified (Nariyama et al., 2004; Wang et al., 2010). However the morbidity effect of rampant caries may minimally affect fitness if it hits later in life. Any gene that has fatal or impairing consequences towards the end of the expected life span may still be selected for if it confers even minor benefits earlier in life; this is known as "deferred costs" (Williams and Nesse, 1991; Worthman and Kohrt, 2005; Trevathan, 2007). To understand the genetic predispositions to oral pathology, such as lower oral pH that encourages caries, it should be ask whether or not these states confer any advantages, especially early in life. Evolutionary medicine has been used as a paradigm through which to ask questions of the fossil record mostly; however, it could inform larger questions about differential health over human evolution, and this is explored in the interpretation of the results of this study.

What the Field of Pleistocene Paleopathology is Missing:

No one has previously conducted a thorough survey of oral pathologies: namely, caries, periodontal disease, periapical lesions, and antemortem tooth loss. Dental enamel hypoplasias have been well studied in the Pleistocene, but as it has already been pointed out, they only

represent systemic stress on an individual during growth and development. Much selection occurs on individuals during this period, but it is not the sum of stress on an individual during their lifetime. Adult morbidity has a major contribution to the overall health of a population and also indirectly affects the health and survival of those individuals' offspring and the next generation. The connection between oral health and systemic health is unquestioned at this point, making a study of Pleistocene oral health a logical pursuit if one wants to answer questions about differential morbidity and health in these fossil samples. Multiple publications have concluded their studies with a call for a more detailed study of oral health in the Pleistocene, and yet the calls went unheeded for over twenty years (Brennan, 1991; Calcagno and Gibson, 1991; Kaifu et al., 2003; Holt and Formicola, 2008)

The few smaller assessments of oral health from Pleistocene individuals often suffer from one of two problems: narrow sample selection making conclusions potentially unapplicable to a larger population; or the failure to utilize better methods developed outside paleoanthropology. To ask questions of Neandertals more broadly and their extinction more specifically, why use contemporary modern humans as a comparison when Upper Paleolithic modern humans specimens are available? Specifically with oral pathology, there is such an extensive bioarchaeology literature of the study of caries, periodontal disease and their resultant pathologies in skeletal collections, one cannot claim there is no tested method to reference.

It is within this current dearth of research that this study orients itself. The methods necessary are widely available, the questions have not been answered, and the implications of the results are expansive. How did oral pathology—and therefore morbidity and health more broadly—vary across populations of Late Pleistocene Western Eurasia? Did health differ over time, and if there are differences, can they be attributed to changing environmental conditions or

evolving technology and culture? Did health differ between Neandertals and early modern humans to an extent that one can make inferences about the Upper Paleolithic transition and the extinction of Neandertals? The larger global health, dentistry, and evolutionary medicine implications of this research could provide a counterpoint for those fields. Many studies have been based on the premise that oral health was good in the Pleistocene, and that the advent of agriculture was the inflection point for the decline of oral health and the rampant oral disease we see today. However that premise has never been fully tested, and where there is have evidence to the contrary, there are some interesting implications for the current narrative of the history of human health.

Chapter 3: Materials & Methods

Materials:

Fossils Materials:

Materials were available for research in twelve countries: Spain, France, Italy, Romania, Croatia, Austria, Czech Republic, Germany, Belgium, England, Israel, and the United States. Universities and museums were visited from March 2012 through January 2013; pilot work was completed in June and July 2010. Fossil materials were selected for inclusion through reviews of regional fossil catalogs and publications. Preservation level was not taken into account as this could underestimate pathology, which weakens alveolar bone, if only nearly complete specimens were selected (Marin et al., 2005). The list was narrowed based upon which specimens were actually available for study. I visited in total 35 museums, universities, and laboratories in 32 cities (Table 3.1).

Country:	City:	Institute:
Spain:	Malaga	Delegado Provincial de la Consejerría de Cultura de la Junta de Andalucía
	Nerja	Museo Historia de Nerja
	Murcia	Zoología y Antropología Física, Universidad de Murcia
	San Sebastian	Centro de Depósito de Materiales Arqueológicos y Paleontológicos de Guipúzcoa
	Madrid	Consejo Superior de Investigaciones Científicas
France:	Paris	Muséum national d'Histoire Naturelle
	Paris	Institut de Paléontologie Humaine
	Les-Eyzies-de-Tayac	Musée National de Prehistoire
	Bordeaux	Laboratoire d'Anthropologie, Université Bordeaux 1
	Perigueux	Musée d'art et d'archéologie du Périgord
	Lussac-les-Châteaux	Musée de Préhistoire de Lussac-les-Châteaux
	Saint Marcel	Musée de Argentomagus

Country:	City:	Institute:
Italy:	Torino	Dipartimento di Anatomia, Farmacologia e Medicina Legale, Università di Torino
	Ventimiglia	Museo Nazionale Preistorico dei Balzi Rossi
	Roma	Museo Nazionale, Preistorico Etnografico, Luigi Pigorini
	Roma	Dipartimento di Biologia Ambientale, Sapienza-Università di Roma
	Anagni	Istituto di Paleontologia Umana (formerly in Roma)
	Pisa	Dipartimento di Biologia, Universita di Pisa
	Ferrara	Dipartimento di Biologia ed Evoluzione, Sezione di Paleobiologia, Preistoria e Antropologia, Università di Ferrara
Romania:	Craiova	Muzeul Olteniei
	Bucureşti	Institutul de Speologie "Emil Racoviţă"
Croatia:	Zagreb	Institute for Quaternary paleontology and geology
Austria:	Vienna	Naturhistorisches Museum Wien
Czech Republic:	Dolní Věstonice	Centrum pro Paleolit a Paleoetnologii Dolní Věstonice
	Brno	Moravian Museum
Germany:	Bonn	Rheinisches Landesmuseum
	Mettman	Neanderthal Museum
	Tübingen	Senckenberg Center for Human Evolution and Paleoecology, Paläoanthropologie, Eberhard-Karls-Universität
Belgium:	Brussels	Laboratory of Anthropology and Prehistory, Royal Belgian Institute of Natural Sciences
	Liège	Geology Department, Université de Liège
England:	London	Natural History Museum
Israel:	Tel Aviv	Department of Anatomy and Anthropology, University of Tel Aviv
United States:	Lexington	Webb Museum, University of Kentucky
	New York City	American Museum of Natural History
	Cambridge	Peabody Museum, Harvard University

Table 3.1: Institutes visited for data collection

Details of specimens included in the study and their sites of origin are provided in Appendix 1 and are organized first by temporal/ taxonomic group, then by country, then alphabetically by site. The minimum number of Late Pleistocene individuals is 253, some of which are represented by only one tooth. The comparative samples are represented by

comparatively more complete burials and are represented by at least one arcade (maxilla or mandible). Their samples sizes (minimum number of individuals, MNI) are as follows: 23 Natufians from Tel Aviv University, 29 Natufians from the Harvard Peabody Museum (52 Natufians total), 23 from Point Hope, and 75 from Indian Knoll.

Research materials:

Measurements were taken with a pair of Mitutoyo metal calipers. Photographs were taken with a Nikon D90 digital camera and a Tamron macro lens and Sigma 18-200mm lens. Light sources varied. All digital radiographs were taken with a Nomad eXaminer x-ray generator gun and a Digirex digital dental radiography system with size #1 digital sensor and software (Dentamerica Inc, Industry, CA), except for the Indian Knoll material where a larger Bowie veterinary x-ray generator was used.

Methods:

Laboratory Methods:

Each specimen was scored for presence and severity of caries, periapical lesions, periodontal disease, hypercementosis, and antemortem tooth loss (details below). Considering the strong interconnectivity among these disease processes and other changes in the oral cavity, dental wear, hypoplasia, and crown morphology were recorded to test for covariance between morphology and oral disease in future analyses. Dental and bony size and shape metrics were also recorded (e.g., buccolingual and mesiodistal dental measurements, mandible width and length). All available Neandertal oral specimens with reasonable dental preservation (MNI=121), Middle and Upper Paleolithic and selected Epi-Paleolithic adult SW Asian and European modern human oral specimens (Middle and Upper Paleolithic moderns MNI=132, Epi-

Paleolithic/Holocene N=52), and selected North American archaic and historic era specimens (N=98) were subjected to examination both macroscopically and radiographically following a standardized data collection protocol detailed below.

To be included in the sample, the mandible, maxilla, and/or tooth had to date to the appropriate time period. The pathologies of periodontal disease, periapical lesions, and ante-mortem tooth loss require alveolar bone for diagnosis; caries only requires a tooth. Therefore sample size varies per pathology measurement and those values are provided in the introduction of each results chapter. Oral pathologies, such as alveolar infections, can weaken alveolar bone, and therefore, choosing only well-preserved specimens would greatly underestimate pathology incidence (Marin et al., 2005). To avoid at least some sampling bias, preservation level was not taken into account with the selection of fossils for inclusion in the study.

Each specimen was placed within one of four age categories based on dental development (AlQahtani et al., 2010) and/or occlusal scores (the ratio and shape of dentin to enamel on the occlusal surface) (Smith, 1984): juvenile/subadult (under 18 years of age); young adult (approximately third decade of life); mid-aged adult (approximately fourth decade of life); and elderly (over ~40 years of age) (Frayer, 1989; Watt et al., 1997). Wear is population specific, so Smith's (1984) scale for hunter-gatherers is the best scale available (Walker et al., 1991). Dental wear is considerably less in extant groups practicing a Westernized diet. It is critical to assess age, because average life span affects oral pathology incidence and severity in a population, as well as allowing for the identification of age-specific trends (Caspari and Lee, 2004; Caglar et al., 2007). These wear categories are theoretically broad enough to avoid inaccuracy (Nagar and Hershkovitz, 2004) and issues such as the possible aging effects of

differences in enamel thickness between Neandertals and early modern humans (Molnar et al., 1993; Olejniczak et al., 2008).

Faunal lists from each site and layer plus published climatic reconstructions are used to infer climatic assignment (Temperate vs. Cold) to test correlation with caries incidence (see Appendix 1 and Table 4.7). Every specimen is also assigned to one of three geographic categories (i.e., Mediterranean, Atlantic, and Continental), but because these samples span a broad time range, these regions can represent different environments over time. Therefore where possible, an additional variable of "climate" (temperate versus cold) is introduced because it may explain pathology patterning better than "region". Climate dictates the available food resources in an environment, and dietary sugars can produce both caries and periodontal disease in recent humans samples (Hujoel, 2009).

Macroscopic Methods:

Photographs and caliper measurements were used to augment visual assessment of specimens. Pathologies acquired during life were differentiated from post-mortem changes (such as fossilization discoloration, pseudocaries, and tunneling mycelium damage) and were recorded (Poole and Tratman, 1978; Kerr, 1990; Whittaker et al, 1990; Hillson, 2001). A Nikon D90 camera with macro lens was used to take detailed photos of each instance of pathology and an 18-200mm lens was used for whole specimen shots. The photos were used for record keeping and re-examination during data analysis when questions arose.

Radiographic Methods:

For radiographic images, a Nomad eXaminer portable x-ray generator was used along with a Digirex digital dental radiography complete system with size #1 digital sensor connected to a PC laptop computer for immediate imaging results using the associated Digirex software

(Dentamerica Inc, Industry, CA). Exposure of 15 mA and 40 kV were used with adjusted exposure time depending on the specimen's preservation and mineral content starting with 0.1 seconds up to 0.4 seconds. The x-ray sensor was placed against the specimen one foot from the source. A longer distance of one meter is preferred with traditional portable x-ray machines, but with a Nomad eXaminer, this is not necessary. All specimens were radiographed by SL with the exception of the samples from Bordeaux, where exisiting radiographs were available (Bois-Ragot, Les Battut, and Baousso de Torre), and the Sima de las Palomas material, where 2-D x-ray images from micro-CTs were available.

Per pathology methods:

Caries: Caries were scored visually with the aid of 10x hand magnification following an ordinal 8-level protocol for location, surface, and severity (Hillson, 2001). The scoring takes the size of the lesion and which dental tissues are affected into account, i.e. how deep is the lesion, and does it affect the pulp, etc? Score 1 is staining, and score 2 is staining with an etched texture. Because of the confounding effects of taphonomic preservation and post-mortem damage, score 1 and 2 caries are difficult to diagnosis definitively in fossils and were therefore not included here. Score 3 is a confirmed lesion that only affects the enamel, and score 5 is a lesion that affects the enamel and dentin, or cementum and dentin in root caries (score 4 is dentin exposure that is not definitely carious and therefore also not included in this study). Therefore the vast majority of caries in this sample are scores 3 and 5. Score 6 is caries that affects one surface and the pulp chamber; score 7 is a lesion that affects multiple surfaces, but not the pulp chamber; and score 8 involves multiple surfaces and the pulp chamber (Hillson, 2001). There is an additional 9-15 scoring regime for grossly severe caries with associated alveolar lesions; its use was rarely necessary. Most lesions were either a level 3 (clear cavitation, but enamel only) or level 5 (dentin

exposed in the cavity). To avoid misdiagnosis confused by diagenetic change, discolorations were recorded separately (Poole and Tratman, 1978; Hillson, 2001). Prevalence per tooth type, tooth surface, location (occlusal, approximal, root, etc.), and severity are tabulated in the caries chapter. Some carious lesions are better diagnosed with radiographs for *in situ* teeth. The same 8-level scoring technique was used for caries radiographic diagnoses and checked against what could be seen visually since radiographs were available instantaneously in the field (Hillson, 2001).

Periapical Lesions: Periapical lesions were diagnosed visually when the cortical bone was affected and further classified by defect size with digital calipers (height and width). Associated teeth, side of alveolar process affected (lingual, labial/buccal), and descriptive traits were also recorded (Dias and Tayles, 1997). Radiographs were used for periapical lesions that could not be well assessed visually or to diagnose ones that were not visible externally, i.e., were confined to the trabecular bone. A number of previously unpublished lesions were identified through radiographs. Values are presented as the number of alveoli present in a sample affected by lesions.

Periodontal Disease: Periodontal disease manifests as deterioration of the alveolar bone, interdental septa, and lamina dura and the formation of bony pockets (Costa, 1982). To assess periodontal disease here, I measured the distance from the cemento-enamel junction (CEJ) to both the occlusal surface (crown height) and the alveolar crest (visible root height, or CEJ-AC distance) at buccal and lingual midpoints using digital calipers (Armitage, 2004; Wiebe and Putnins, 2000). CEJ-AC measurements are used to diagnosis periodontal disease in modern clinical settings, as well as to record attachment loss over time. In living humans, a distance of more than 1-2 mm is considered slight disease though in dried bone or fossils, the alveolar bone

may also recede for taphonomic reasons. Up to 3mm may be healthy and needs to be considered with the condition of the bone (Goldman et al., 1976; Lavigne and Molto, 1995). Recording six CEJ-AC measurements per tooth is advocated for (Lavigne and Molt, 1995; Eke et al., 2012), but that was not feasible here because of the delicate nature of fossil alveolar bone, especially interproximally. More than the standard two measurements (mid-point buccal and lingual) were taken when CEJ-AC distances varied greatly around the tooth, a condition more common in multi-rooted teeth. CEJ-AC distances were averaged per tooth, tooth type, and individual with locations of severe angular defects noted (Hildebolt and Molnar, 1991).

Costa (1982), however, considered CEJ-AC distances to be somewhat untrustworthy for periodontal disease diagnosis because of the other processes that enlarge CEJ-AC distances besides periodontal disease (e.g., continuous eruption, lesions of pulpal origin (Clarke, 1990)), and presented his own method. Following Costa's (1982) method of two scores for each present interdental crest (porosity and shape), an ordinal/binary score for alveolar condition was also assigned (Table 3.2). The first ordinal category refers to the shape of the alveolar septa between teeth (convex, flat, or concave) and the second binary score refers to the presence or absence of porosity. Other authors have considered Costa's method to be too subjective (Lavigne and Molto, 1995); therefore, both kinds of data were collected. Also the extent of continuous eruption can be a confounding factor in periodontal disease diagnosis (Costa, 1982; Whittaker et al, 1990; Clarke and Hirsch, 1991; Danenberg et al., 1991; Newman, 1998; Dewitte and Bekvalac, 2010). Therefore periodontal disease diagnosis is presented using two different diagnostic protocols. Porosity can manifest before increasing CEJ-AC distance, so septa condition can hypothetically catch early disease (Costa, 1982). Alveolar topography and/or

deformities were also recorded (Karn et al., 1984). These can be related to periodontal disease, infections/lesions, and trauma.

Using a dental probe to test the depth of infrabony periodontal pockets was not done to protect the delicate fossil materials, though it is advocated in periodontal studies of other skeletal materials (Costa, 1982; Lavigne and Molto, 1995). In addition to delicate preservation, many of the specimens examined here did not preserve full dentition. The CEJ-AC distances of a small subsection of an individual's teeth predict the overall average CEJ-AC distance for an individual with a small amount of error though (Shrout et al., 1990); suggesting that where a subsection of alveoli in an individual are preserved, I can reasonably assume they reflect the CEJ-AC distance average of the whole individual. CEJ-AC distances were then surveyed as averages per tooth type and individual.

Ordinal Score	0	1	2	3
CEJ-AC measurement[1]	0-1mm	2-3mm	4-5mm	>6mm
Septa Condition Scores[2]	No Porosity; Convex or Flat	Porosity; Convex shape	Porosity; Flat shape	Porosity; Concave shape
Diagnosis	No disease	Mild disease	Moderate disease	Advanced disease

Table 3.2: Diagnosis for periodontal disease in specimens using both CEJ-AC distances and interdental septa scores: [1]Modified from Lavigne and Molto (1995) who modified it from Ramfjord (1967). Clarke et al. (1986) used a similar method with three levels (0-2mm, 2-4mm, >4mm); [2]Costa, 1982

Because two types of periodontal diagnosis were used, differences between the protocols was assessed to test whether high CEJ-AC distances can occur without degraded inter-dental septa condition (i.e., continuous eruption). These results are included in Appendix 3 and found generally that the two methods are highly correlated with one another (except for the upper left

central incisor). High levels of wear are common in Pleistocene individuals, especially in the oldest age category (Krueger, 2011). Teeth can super-erupt without matching occlusal forces and mesially drift in response to interproximal wear while still maintaining good alveolar bone health (Begg, 1954; Hillson, 2001; Kaifu et al., 2003). The use of two methods of periodontal disease diagnosis should minimize false positives in these populations given their high levels of dental wear, though the strong relationship between the two diagnostic methods suggests this is not a problem (Appendix 3).

Antemortem Tooth Loss (AMTL): Teeth present and alveoli (tooth sockets) present were recorded. Any visual evidence of alveolar resorption or missing teeth were noted, and antemortem tooth loss was recorded as "number of sockets missing a tooth with evidence of resorption per all identifiable sockets" (Buikstra and Ubelaker, 1994). Due to high preservation variance in these samples, number of lost antemortem teeth per individual is inappropriate, so percentages per individual are used. Some alveoli were difficult to definitively state whether a tooth was lost ante- or postmortem, so there are separate categories for definitive loss and definitive plus probable loss (Gilmore, 2013). Unerupted teeth seen in radiographs or in partly open crypts were also recorded, as were deciduous teeth. Considering eruption timings, some present deciduous teeth appeared to be pathologically persistent. Agenesis of teeth, especially third molars, can be difficult to differentiate from tooth loss in older individuals, therefore agenesis results were also reported in the antemortem tooth loss results chapter. Considering that dental agenesis has not been analyzed previously for the Pleistocene, the reporting of the results is warranted.

Occlusal Wear: Dental wear was scored using an ordinal 8-level scoring method per tooth type with some sub-categories for the posterior teeth (Smith, 1984). Wear is used to assign an

age category along with dental development (AlQahtani et al., 2010). When the pulp chamber is exposed, any indications of demineralization or carious activity, as well as secondary dentin were noted to differentiate caries-induced versus wear-induced pulp chamber exposure (Hillson, 2001). Secondary dentin can also be seen radiographically, though it is difficult to quantify. Exposed pulp chambers were cross-referenced with evidence of infection or periapical lesions.

Statistical Methods:

From this raw data, oral pathology prevalences were computed. Rate (occurrence of new cases of disease with a specified time period) and incidence (risk of developing disease within a specified time period) for pathology can be calculated for living humans, but considering the biased structure presented in an cementary or archaeological sample, only prevalence can accurately be assessed (total number of cases in sample) (Hillson, 2008)—though the term "rate" is commonly but inaccurately used in anthropological research (e.g., Frayer, 1989). The majority of the data derived from this study are ordinal, but a few forms of data are nominal or continuous. I did not anticipate normal distributions for any continuous data—with the exception of size metrics—as most of these pathologies intensify over the aging process, and many individuals will show no pathology. Because of this, Kruskal-Wallis tests (non-parametric, multiple sample groups) were usually used for determining significant difference between samples and Chi-square goodness-of-fit to test whether distribution patterns differed from predicted. The alpha value for significance is set at 0.05.

Percentage of carious teeth over all teeth present per group, tooth type, severity, etc. are compared as well as proportion of individuals with various levels of periodontal disease severity within variously defined groups. Antemortem tooth loss is a more simple mathematical diagnosis: presence/absence per alveolus. The number of alveoli missing their tooth and showing

some form of alveolar resorption over all the alveoli present was calculated per specimen. These ratios were compared between individuals and groups. Periapical lesions were also calculated per alveoli present in a sample group. By using "number of alveoli affected" for lesions, it is somewhat more confounding than using the number of lesions as large lesions can affect more than one tooth. Using number of alveoli affected may overestimate the number of contained lesions (periapical lesion prevalence), but it gives an idea of the severity of affliction for an individual. Also it is alveoli affected, not teeth affected, as no loose tooth was diagnosed as having a periapical lesion (though Eshed et al., (2006) reports their lesion percentages as per tooth). An alveolus with a tooth missing postmortem could still be diagnosed as "lesioned"—and lesions can cause antemortem loss. A lesion that caused antemortem loss well in advance of death may go undiagnosed though, if healing is extensive.

All of these pathologies are assumed to increase in frequency with age, and therefore age needs to be accounted for in the data distribution. The age distributions per group were tested for statistical difference (Kruskal-Wallis), which as a technique has a precedent, but is not perfect (e.g., Bridges, 1991). Two other techniques were experimented with: comparisons per age categories (e.g., Keenleyside, 1998; Dewitte and Bekvalac, 2011) and regression holding age constant. The first two methods are done in each pathology results chapter and regression is attempted in the overall health discussion chapter. This has to be done per pathology since the sample numbers vary considering what was preserved in each individual. Certain pathologies are also assumed to differently affect men and women. Unfortunately with fossils, sex is rarely a known variable and therefore corrections for sex could not be made.

Drawing from Brennan's (1991) "Overall health statistic" used to identify direction and magnitude of changing health indicators between consecutive time periods—a positive value

indicates increasing health, a negative value decreasing health—a total dental health statistic is used to help describe the rejected null hypotheses about changing oral health in the discussion chapter (see Equation 3.1). There are 4 indicators (caries, periodontal disease, antemortem tooth loss, periapical lesions) meaning that the highest possible value is +/- 5.33. This is a crude measure, but it is comparative within the same study and gives us a holistic indication of changes over time. The overall null hypotheses are tested per pathology and where the pathologies show conflictory signals, the total dental health statistic could help to explain which signal is stronger.

$$\frac{\pm \#\ of\ indicators\ showing\ same\ directional\ trend}{Total\ \#\ of\ indicators - 1}\ x\ Total\ \#\ of\ indicators$$

$$= Brennan's\ Overall\ Health\ Statistic$$

Equation 3.1: Overall morbidity score between each temporal group (from Brennan, 1991)

The null hypotheses were drafted to reflect no change, and therefore would be rejected under the current understanding in the literature of health and stress in the Late Pleistocene. Tests of statistical differences between groups for distribution of pathologies are used individually and in aggregate to test the hypotheses (see Chapter 7: Oral Health & Systemic Health). Where null hypotheses fail to be rejected, this might be explained by one of three issues: population morbidity does not differentiate samples; oral health does not reflect population morbidity; or sample size is inadequate. Since oral health correlates well with mortality (Goodman and Armelagos, 1989; Lovell, 1991) and overall health (Dolan et al., 1991; Gift and Atchison, 1995), the former is the most likely explanation (i.e., the null hypothesis truly fails to be rejected). Sample size issues could exaggerate type II error in the study; however, testing was done by tooth type in addition to specimen/individual to increase sample sizes (but introduces

interrelatedness amongst data points). Sample size is always a problem in paleoanthropological research. This is addressed per pathology in the results chapters as each pathology analysis has a different sample size.

Chapter 4: Caries & Periapical Lesions

Introduction:

Caries are not well known—or rather have rarely been identified—before the advent of agriculture across most studied regions of the world. In this survey, new instances of caries were identified and others were confirmed (47 carious teeth seen in total out of 1869 teeth studied, or 2.5%). Prevalences remain low overall, but are higher than previously reported (e.g., Frayer, 1989; Brennan, 1991; Walker et al., 2011 (see Table 4.1 and 4.7)). Though sample sizes for various caries-positive sub-groups are small, patterns by time period/taxonomy and region are discernable. Caries are often analyzed with periapical lesions in recent samples, as severe carious lesions may eventually result in pulpal inflammation, necrosis, alveolar inflammation, or periapical lesion formation. Here there are 67 lesioned alveoli in 2131 alveoli examined (3.1%). Few periapical lesions are directly associated with carious teeth in these samples, and few carious lesions are advanced enough to effect the pulp chamber. The significance of this lack of association is discussed, and lesions are also compared in chapter seven to periodontal disease prevalence—another potential source for the introduction of bacteria into the alveolus.

Results:

Caries can be analyzed as presence/absence and severity either by individual or by tooth. Because fossil individuals rarely preserve all of their teeth—and in the Pleistocene a single tooth may represent a named specimen—analysis by tooth is the only reasonable approach for a sufficient sample size. This makes cross-comparison with some other Holocene studies inappropriate, as per-individual prevalences are commonly reported and are much higher than per-tooth prevalences (see Table 4.1). It only takes one lesion to diagnose all 32 teeth in an

individual as having the disease "caries" using this method. If the minimum number of individuals is used (MNI) (not all catalog numbers correspond to the fossil remains of only one individual), the Late Pleistocene caries analysis sample contains at least 253 individuals and 36 individuals diagnosed with caries, making the pooled caries individual prevalence 14.2%. These carious "individuals" range from a single tooth to a full dental arcade.

	This Study	Frayer, 1989	Other studies
Neandertal	3.50% [6.7%]	-	-
MPMH	40% [43.8%]	-	-
EUP	15.90%	0%	2.9% (Holt & Formicola, 2008)
LUP	26.20% [27.4%]	11.30%	7.3% (Holt & Formicola, 2008)
Mesolithic	-	19.10%	14.6%[t] (Wittwer-Backofen and Tomo, 2008), 14.9% (Meiklejohn et al., 1988)
Natufians	15.40%	-	* (Eshed et al., 2006)
Point Hope (Ipiutak)	8.70%	-	* (Costa, 1980b)
Indian Knoll	66.70%	-	30% (Leigh, 1925), 21.3% (Rabkin, 1943)

Table 4.1: Comparison of this study's results (caries percentage, individuals per group) with previous publications; Values in parenthesis include published examples. [t]Calculated from values available in the paper; *None of the previous relevant publications report this value, nor the necessary variables to compute it

In this study, the per-individual values are: Neandertal: 3.5% (4 of 116); Middle Paleolithic Modern Humans (MPMH): 40% (6 of 15); Early Upper Paleolithic: 15.9% (10 of 63); and Late Upper Paleolithic: 26.2% (16 of 61) (Table 4.1). Pre-Columbian Native American samples' per-individual caries prevalences range from 1.9% to 89.6% (Wells, 1975) and the comparative samples analyzed in this study range from 8.7% (4 of 23 at Point Hope) to 66.7%

(50 of 75 at Indian Knoll) indicating that this value varies wildly for human samples and disguises severity and multiple carious lesions in an individual. All further results are presented as per-tooth. Considering antemortem and postmortem loss, the per-tooth values likely underestimate the caries prevalence to a small degree.

Known from Literature	Newly Identified Examples
Neandertal	**Neandertal**
Sima de Palomas 25, 59	Amud 1
Aubesier 5, 12	Tabun 2
Kebara 1*, 27	**Middle Paleolithic EMH**
Banyoles 1*	Qafzeh 7, H4, 4, 9, 11
Middle Paleolithic EMH	**Early Upper Paleolithic**
Qafzeh 3	Les Rois 28
Skhul 2	Grotte des Abeilles
Early Upper Paleolithic	Dolní Věstonice 15
Cro Magnon 4	Předmostí A17088 (whole mand.)
Les Rois 50-4 (23), 51-15(40)	Barma Grande 2
Pavlov 1	**Late Upper Paleolithic**
Dolní Věstonice 13[t]	Bois Ragot
Paglicci*	Le Morin
Late Upper Paleolithic	Gough's Cave 1
Grotte des Enfants 4*	Bruniquel (Abri Lafaye)
Arene Candide 1*	Ohalo 1, 2
Lalinde	Laugerie-Basse 2
Romito 1	Vindija 22.2 & 22.7
Ortucchio 1*	Romanelli 29 & loose tooth #7
Roc del Migdia 1*	Continenza 4
Urtiaga B1*	Saint Germain 1970-7
Balauziere*	
Aurensan*	
Aveline's Hole 174*	
Kent's Cavern EM501*	

Table 4.2: All the specimens with at least one carious lesion; * indicates it is in the literature, but is not a part of the sample because the descriptions were inadequate; [t]published as "maybe" having caries (Hillson, 2008), but considered as definitive caries here

Table 4.2 lists the fossil catalog names for all the identified individuals with caries, as well as other Pleistocene specimens published as having caries, but I did not study. Specimens

were not chosen for initial inclusion in this study based on any previous knowledge of their pathology status, so many specimens known to have caries were not included for visual inspection for a variety of reasons (See Chapter 3: Methods for a description of sample selection). Some of these specimens were initially described as explicitly not having caries or maybe having caries (e.g., Dolní Věstonice 13 & 15, respectively) (Trinkaus et al., 2006; Hillson, 2008), although that now appears to be inaccurate, i.e., they both have caries. For example, the Banyoles specimen was originally described as explicitly not possessing carious teeth (Herńandez-Pacheco and Obermaier, 1915; Tillier et al., 1995), but subsequent publications corrected that initial assertion (Lalueza et al., 1993). The same conflictory diagnostic situation describes Arene Candide 1 (no caries: Formicola, 1988; caries: Frayer, 1989) and Grotte des Enfants 4 (no caries: Formicola and Repetto, 1989; caries: Frayer, 1989). Published examples of caries that included sufficient descriptions were included in the sample as well, as a parenthesized value following the original sample value. This included: Neandertals: Aubesier 5 (Trinkaus et al., 2000), Aubesier 12 (Lebel and Trinkaus, 2002b), Kebara 27 (Tillier et al., 1995), and Banyoles 1 (Lalueza et al., 1993; Trinkaus et al., 2000); Middle Paleolithic modern human: Skhul 2 (Sognnaes, 1956; Tillier et al., 1995); and Late Upper Paleolithic modern human: Romito 1 (Fabbri and Mallegni, 1988). No additional Early Upper Paleolithic specimens were identified from the literature for inclusion. All of these additional examples come from the Mediterranean region and raise the overall percentage (2.8% (53 of 1875)) and the per-individual values (Neandertal: 6.7% (8 of 120); Middle Paleolithic modern humans: 43.8% (7 of 16); Late Upper Paleolithic: 27.4% (17 of 62)) (Table 4.1).

This list suggests that the relative lack of caries in the literature does not mean that carious lesions do not exist (Figure 4.2). Many of the caries examples in the “published” lists

were pulled from lists in summary publications (Sognnaes, 1956; Frayer, 1989; Brennan, 1991); there are few publications explicitly describing these individual examples of pathology (I included all of the sufficiently described ones above). Many anthropologists who were unfamiliar with dental pathologies may not have identified the carious lesions in these specimens. They were not looking for these pathologies, and therefore they remained undescribed.

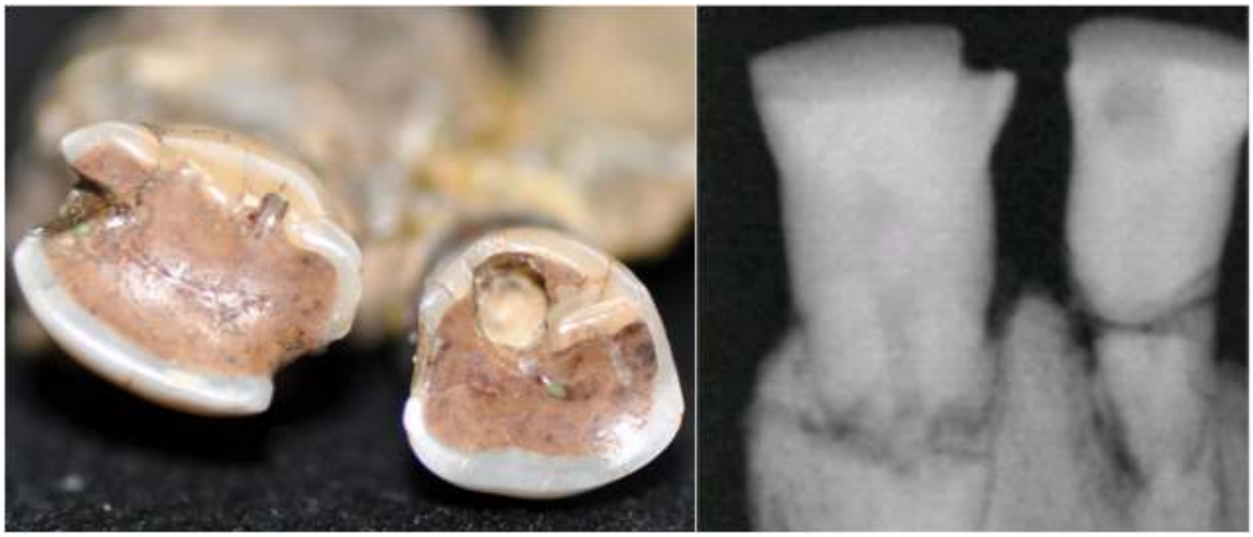


Figure 4.1: Qafzeh 3 lateral incisor caries photograph (left) and radiograph (radiograph)

Table 4.3 gives a summary of the distribution of these carious teeth based on time period against region. The relevance of the distribution across time and geography are discussed in sub-sections below. There is no Middle Paleolithic Modern Human in Western Eurasia outside of Southwest Asia (Mediterranean region), hence the dashes, as a value of zero would indicate that no caries were present in a sample.

	Atlantic	Continental	Mediterranean	Sum
NEAN	0	0	4 [8]	**4 [8]**
MPMH	-	-	16 [17]	**16 [17]**
EUP	5	5	1	**11**
LUP	8	0	8 [9]	**16 [17]**
Sum	**13**	**5**	**29 [35]**	**47 [53]**

Table 4.3: Number of carious teeth organized by region and time period (NEAN: Neandertals; MPMH: Middle Paleolithic modern humans; EUP: Early Upper Paleolithic; LUP: Late Upper Paleolithic); Values in parentheses include published examples

Age distribution:

The age distributions of each time period and region were tested for statistically significant differences. Because the tooth sample size for the caries analysis is different from the alveoli sample size for the periodontal disease analysis, this testing is done again in the next chapter. Caries organized by age category is analyzed separately below. Age distribution by MNI and number of teeth present was plotted by region and time period (Figs. 4.2, 4.3, 4.4, 4.5). The Sima de las Palomas sample with its large number of single loose teeth with their own catalog numbers—and therefore counted as separate individuals—warps the Neandertal distribution for individuals (MNI). Using Kruskal-Wallis for all tests, age distribution by time period for individuals was significantly different when Palomas was included (p-value: 0.048), but not significant if Palomas is removed (p-value: 0.116), or if the Middle Paleolithic modern humans, who are without elderly individuals, are removed (p-value: 0.354) (Figure 4.2). There are non-significant differences between regions using MNI (without Palomas: p-value: 0.105; with Palomas: p-value: 0.120) (Fig. 4.3). When testing for age distribution differences by number of teeth, by region (p-value: 0.276) (Fig. 4.4) and time period (p-value: 0.255) (Fig. 4.5), there is also no statistical difference between groups. All the temporal/ taxonomic and regional samples

pattern similarly with respect to age distribution, with more young and mid-aged adults than adolescents and elderly. Therefore because age differences are not statistically significant, comparisons do not require control for age.

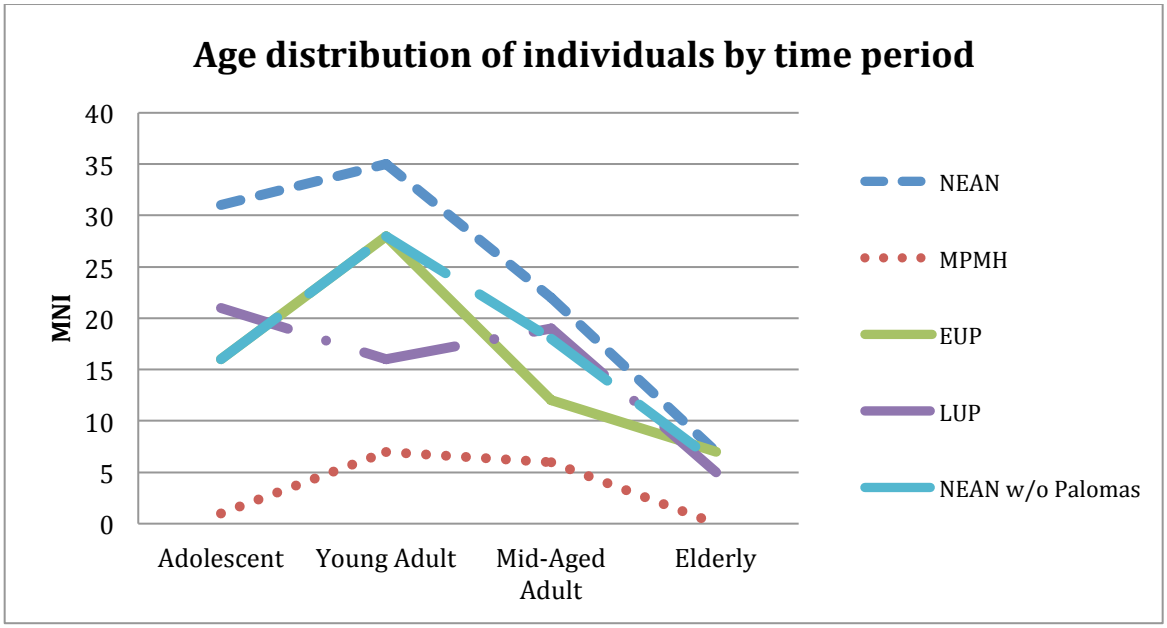


Figure 4.2: Age distribution of individuals by time period: The Neandertals are plotted both with and without the Sima de las Palomas individuals (With Palomas: p-value: 0.048; without Palomas: p-value: 0.116)

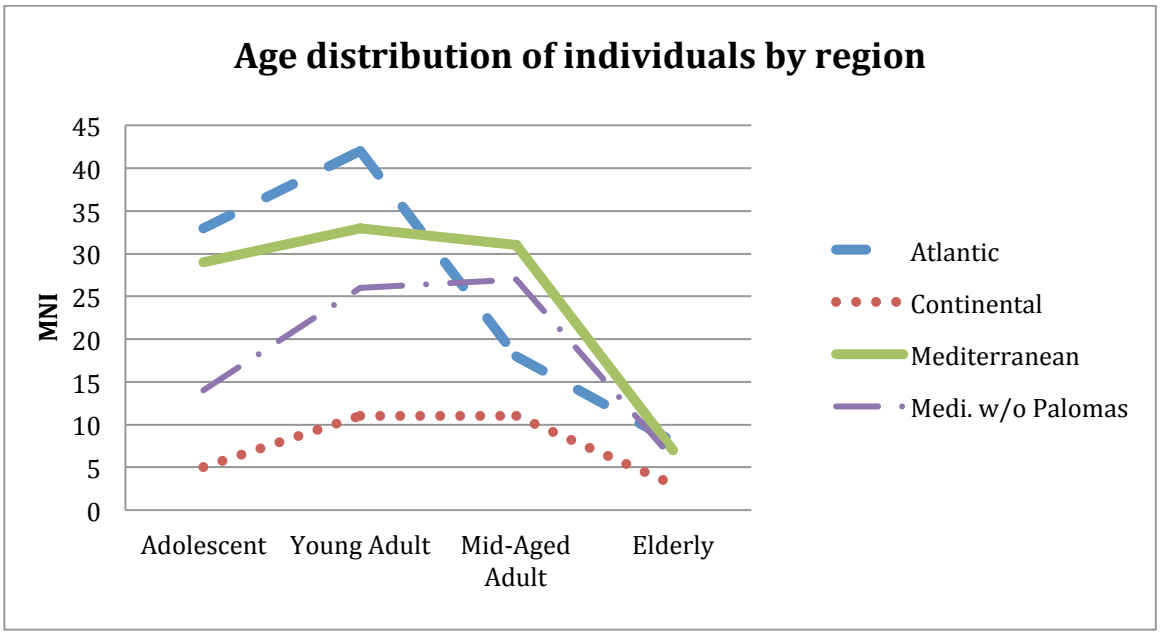


Figure 4.3: Age distribution of individuals by region: The Mediterranean region is plotted both with and without the Sima de las Palomas individuals (without Palomas: p-value: 0.105; with Palomas: p-value: 0.120)

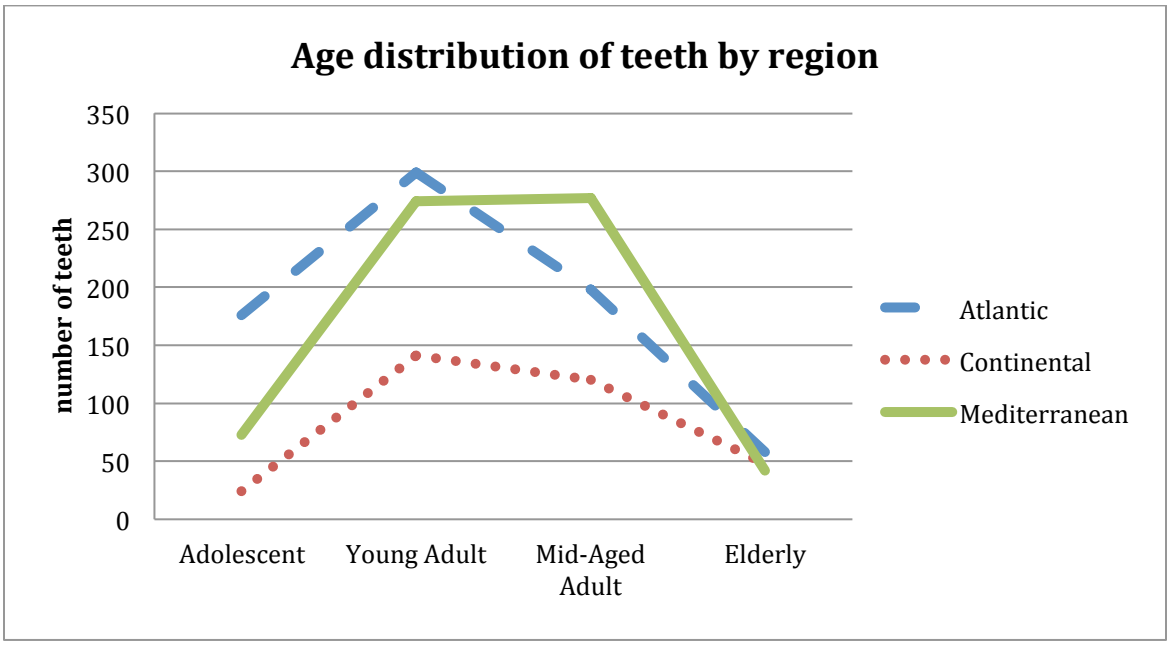


Figure 4.4: Age distribution of caries sample by number of teeth per region: The Mediterranean region is plotted both with and without the Sima de las Palomas individuals (p-value: 0.276)

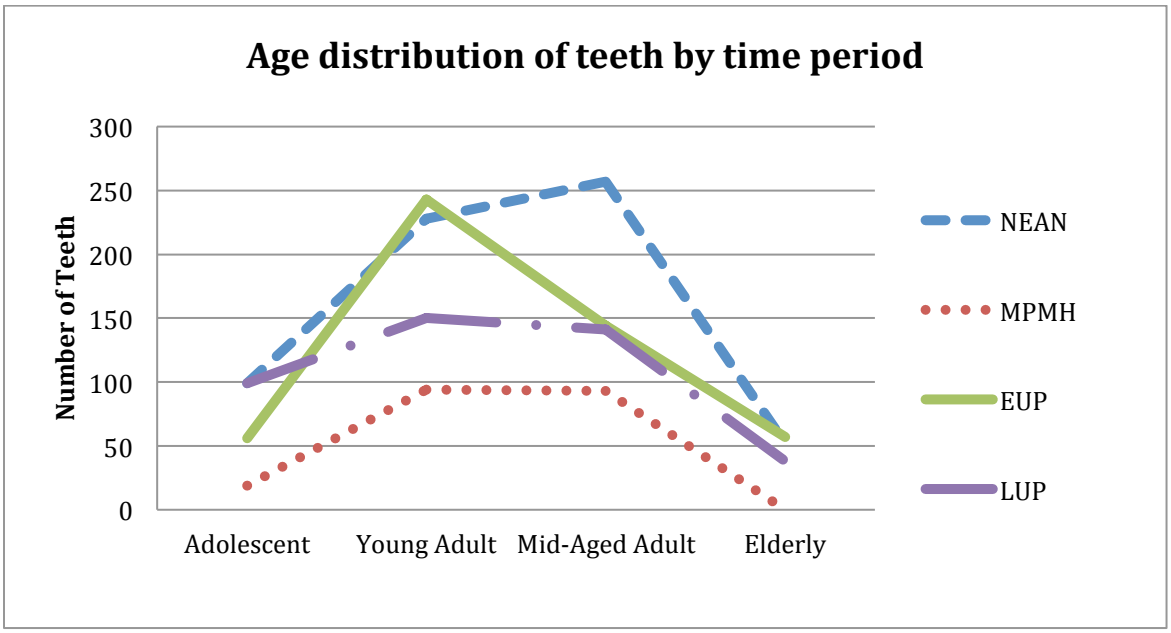


Figure 4.5: Age distribution of teeth by time period: The Neandertals are plotted both with and without the Sima de las Palomas individuals (p-value: 0.255)

Tooth type:

All tooth types are not equally susceptible to caries (Hillson, 2008). In fossils, this disparity may be further exaggerated as anterior teeth, which supposedly have fewer caries than posterior teeth, also have simpler root shapes that predispose them to postmortem loss (Hillson, 2008). The ratio of incisors to canines to premolars to molars should be approximately 2:1:2:3 (25%: 12.5%: 25%: 37.5%) reflecting their proportions in the mouth; however, in some samples outside the Late Pleistocene, third molar agenesis can be high as 30% of the population (Brothwell et al., 1963; Scott and Turner, 1997). The percentages of each tooth type here reflect this preservation bias in that there are significantly fewer incisors and more molars than expected (Incisors 20.4%: Canines 12.5%: Premolars 24.3%: Molars 42.7%) (Chi-square, p-value: 0.003).

Molars show the highest prevalence of carious lesions in this sample (Table 4.4), and this is statistically significantly different from the predicted values based on tooth type percentages

(Chi Square, p-value: 0.004). Modern and fossil studies have similar patterns in tooth type susceptibility (e.g., Klein and Palmer, 1941; Frayer, 1989; Macek et al., 2003; Hillson, 2008). Molars have occlusal surfaces with pits and fissures for bacteria to flourish in, they receive less cleaning tongue action than anterior teeth, and they are further from sublingual and submandibular salivary gland ducts. Most modern studies focus on the caries of permanent teeth in children, and the occlusal fissures and pits of permanent molars are the most susceptible to caries by far in children (Hillson, 2008). In adults who do not have access to dental care, caries on other teeth become more common, but the molars are still the most susceptible (Manji et al., 1989; 1991).

	Incisors	Canines	Anterior Teeth	Premolars	Molars	Posterior Teeth	**Total**
No. Carious Teeth	4 [6]	3	7 [9]	2	34 [37]	36 [39]	**43 [48]**
Total No. Adult Teeth	371 [380]	226 [231]	597 [611]	432 [437]	757 [774]	1189 [1211]	**1786 [1822]**
Percentage of tooth type w/ caries	1.1% [1.6%]	1.3% [1.3%]	1.2% [1.5%]	0.5% [0.5%]	4.5% [4.8%]	3.0% [3.2%]	**2.4% [2.6%]**

Table 4.4: Number of carious teeth organized by permanent tooth type. The total sums to 43 [48] because 4 [5] carious teeth were deciduous molars; Values in parentheses include published examples (Anterior vs. posterior, p-value: 0.295)

A previous study found caries only on molars in the Upper Paleolithic (Frayer, 1989), and the posterior caries "rate" was reported as 7 times that of the anterior caries "rate" in the Mesolithic. The anterior versus posterior caries prevalence is not nearly as divergent here (and non-significant, p-value: 0.295) with 2.1 times higher posterior caries prevalence (anterior: 1.5% vs. posterior: 3.2%).

Of the six incisors that did show caries, five were likely shoveled shaped, which means that they have a more crenulous lingual surface for cariogenic bacteria to thrive upon (Amud 1, probably the two Qafzeh 3 carious incisors, Kebara 27 (Tillier et al., 1995), and Skhul 2 (Sognnaes, 1956)). The Vindija modern human carious central incisor (22.2) does not have visible medial or lateral lingual tubercles, but it is heavily asymmetrically worn on the lingual side and another lateral incisor from the site (tooth #22.3) is mildly shoveled. This means that it is possible that all the carious incisors are shovel-shaped and would therefore support the hypothesis that oral pathology avoidance may be driving the selection for smaller teeth with less complex dental morphology over the Late Pleistocene and Holocene (Gibson and Calcagno, 1989; Calcagno and Gibson, 1991). Not all of the carious lesions on these incisors are on the lingual side; some are on the worn occlusal edge (Qafzeh 3, Skhul 2, and Amud 1). Shovel shaped incisors have much more surface area on their occlusal edges than chisel-shaped incisors though, and exposed dentin is more susceptible to demineralization than enamel. And once again, these incisors all hail from the Mediterranean region.

There are five carious deciduous teeth in this sample; however, they should not be viewed as inconsequential. In the Qafzeh 4 approx. 7 year old individual, there is a carious lesion on a deciduous first molar, which has a matching lesion on the neighboring deciduous canine (see Fig. 4.6). Deciduous caries can affect neighboring teeth even in the Pleistocene, suggesting that permanent teeth are also susceptible in these carious oral environments (Yi and Wang, 2002), especially considering the vulnerability of erupting teeth to demineralization. Multiple authors have stated that dental disease is an adult only disease before agriculture (Brabant and Brabant, 1962; Brabant, 1967; Meiklejohn et al., 1984; Frayer, 1989); however, this is not the case.

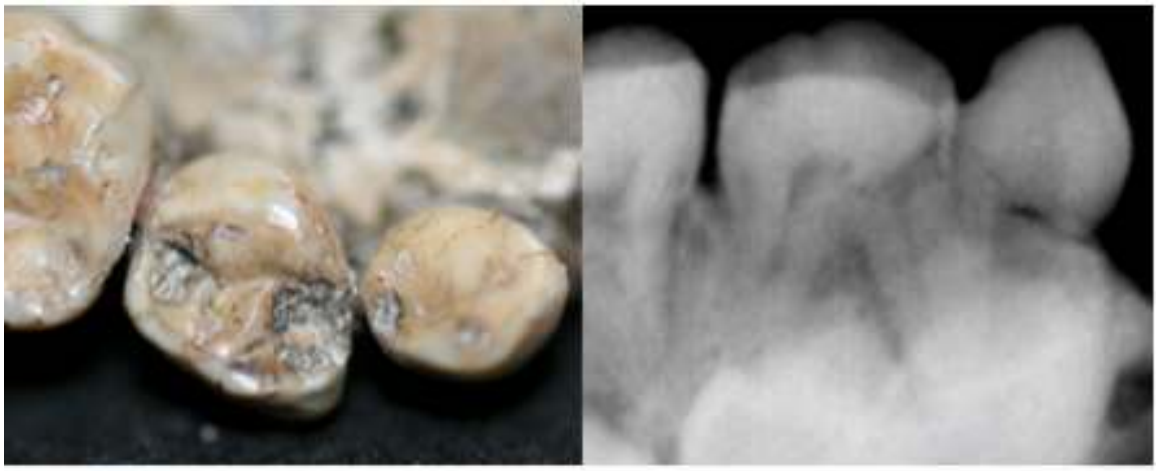


Figure 4.6: Qafzeh 4 left deciduous m^1 and deciduous canine caries. Left image is an occlusal photograph of the mesial deciduous m^1 lesion and the matching distal deciduous canine lesion. Right image is a radiograph. Note the radiolucency demonstrating a communication between the occlusal lesion and the pulp chamber of the deciduous m^1

Caries susceptibility also varies by upper and lower arcade. Saliva has an anti-bacterial effect (Dowd, 1999), but teeth in the lower arcade, with more exposure to saliva from proximity to salivary glands and gravity, are not less susceptible (Macek et al., 2003). 31 of the carious teeth are lower teeth—58.5% of the total lesioned teeth—and 22 are upper teeth. Out of a total of 997 mandibular teeth, 3.0% are carious, and of 853 maxillary teeth, 2.5% are carious. However this is not statistically significant from the predicted (Chi Square, p-value: 0.777). There are statistically more mandibular teeth than sampling error would predict, but the large sample size may be driving this p-value more than actual preservation bias (Chi-Square, p-value: 0.015). Previously studied Upper Paleolithic and Mesolithic samples showed the same pattern with 2.4% of Late Upper Paleolithic mandibular teeth having caries and 1.0% of maxillary teeth (Frayer, 1989) and mandibular caries outnumbering maxillary ones in the Epi-Paleolithic Jomon (Fujita, 2012).

Tooth Surface:

Carious lesions can form on any surface of the tooth, but certain locations are more favorable for cariogenic bacteria. It has been assumed that root caries would be more common in populations with high wear, such as Paleolithic ones, since both occlusal carious lesions would be worn away and the compensatory continuous or supra-eruption would expose the roots of the teeth (Katz et al., 1982; Kerr, 1990). However, this is not the case. In this sample, the occlusal surface was the most common location for lesions; followed by the buccal or lingual side of the crown (usually molar buccal or lingual pits); and then with equal percentages for the interproximal sides and cemento-enamel junction/ cervical region (see Table 4.5). There was only one example each of root caries (Vindija 22.7, see Fig. 4.7) and of a lesion so extensive that the initiation site was indeterminate in the original sample, plus the published root caries from Aubesier 12 (Lebel and Trinkaus, 2002). This pattern is statistically different from predicted values if one assumes any surface is equally likely (Chi-Square, p-value: 0.027). Low life expectancy may partially explain the low number of examples of root caries, as they are commonly an affliction of the elderly (Warren et al., 2000). This pattern matches Hillson's (2008) tooth type and location caries susceptibility pyramid with upper first molar occlusal fissures and lower buccal pits (side) being the most susceptible locations. Deciduous teeth follow a similar pattern with fissures of dm1s and buccal pits of dm2s being the most common sites (Evans and Lo, 1992; Skeie et al., 2006).

	Occlusal	Buccal/Lingual Side	Interproximal	Cervical	Root	Initiation Site Indeterminate
No. Carious Teeth	19 [20]	13 [16]	7 [8]	7	1 [2]	1
Percentage of total carious teeth	40.4% [37%]	27.7% [29.6%]	14.9% [14.8%]	14.9% [13%]	2.1% [3.7%]	2.1% [1.9%]

Table 4.5: Number of carious teeth organized by site of lesion. Total adds to 48 as one tooth, Les Rois no.23, had carious lesions on two different surfaces. Other teeth with multiple lesions had them on the same surface—typically occlusal—and were therefore still counted as one. Values in parentheses include published examples (p-value: **0.027**)

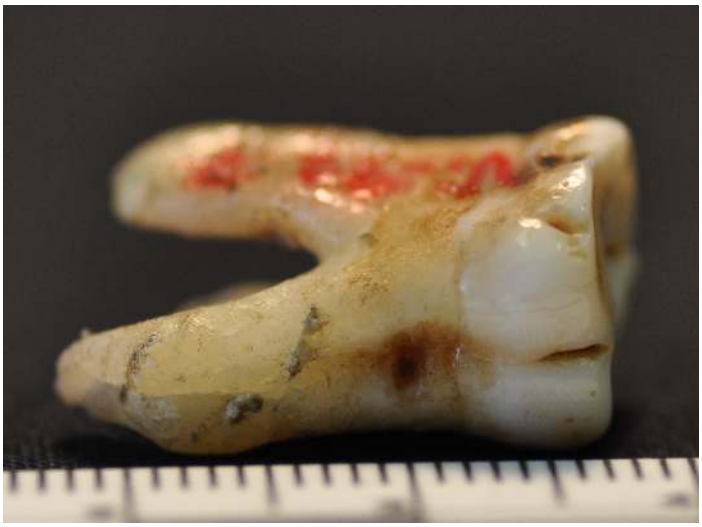


Figure 4.7: Root caries from Vindija Modern Human #22.7

Time Period:

Caries are recognized as being quite low in Neandertals, previously published as 0.3% of teeth before including the Sima de las Palomas specimens (Walker et al., 2011). The only previous attempts to quantify caries in the Late Pleistocene was an analysis geographically confined to Southwest France, which found only one example before the Magdalenian (i.e., Cro-Magnon 4) (Brennan, 1991)), and an analysis of Upper Paleolithic and Mesolithic Europeans (Frayer, 1989) (see Table 4.7; Brennan (1991) did not report percentages, just a list of lesions

and no tooth totals). The results of this study are presented in Table 4.6. The pattern over time in this study is the same as the previous studies (Frayer, 1989; Brennan, 1991): caries prevalence increases through time in Europe with the highest prevalence being in the Late Upper Paleolithic (Fisher's exact, p-value: 0.005). However the prevalences here are more than twice those for the Late Upper Paleolithic reported by Frayer (1989) (EUP: 0% vs. 1.9% (11 of 578); LUP: 1.5% vs. 3.6% (17 of 442)), and neither previous author included Middle Paleolithic modern humans from Southwest Asia in their samples.

	Neandertal	MP Moderns	EUP	LUP	Pooled Pleistocene Moderns
No. Carious Teeth	4 [8]	16 [17]	11	16 [17]	43 [45]
Total No. Teeth	659 [668]	206 [222]	578	426 [442]	1136 [1246]
Percentage of time period w/ caries	0.6% [1.2%]	7.8% [7.7%]	1.9%	3.8% [3.6%]	3.5% [3.6%]
No. Lesioned Alveoli	17	3	33	14	50
Percentage of time period w/ periapical lesions	2.5%	1.6%	5.0%	2.7%	3.6%

Table 4.6: Number of carious teeth and lesioned alveoli organized by time period and taxonomy; Values in parentheses include published examples (Caries: p-value: **0.005**; Periapical lesions: Chi-Square, p-value: 0.244)

The modern humans of the Middle Paleolithic have the highest per-tooth prevalence (7.7% (17 of 222)) (Table 4.6), more than twice that of any other group (note 43.8% of Middle Paleolithic modern human individuals have caries in this study). This sample did not include any Middle Paleolithic modern humans from East Asia or Africa, though caries has been previously identified from Zhirendong, South China (Lacy et al., 2012) and the Middle Pleistocene archaic human from Broken Hill (Kortizer and St. Hoyme, 1979; Peuch et al., 1980; Bartsiokas and Day,

1993; Lacy, n.d.). The Middle Paleolithic modern humans in this sample are only from Southwest Asia, and therefore their high caries prevalence may be related to regional factors rather than temporal/ taxonomic ones. Kebara and Amud are Neandertals also from Southwest Asia who have carious teeth. However, it cannot be ruled out that the Middle Paleolithic modern humans in general suffered from caries at a rate greater than any previous or following populations until the Neolithic. This was a population expanding across Asia out of Africa (Bar-Yosef, 1992; Templeton, 2002; Liu et al., 2010), and the dietary implications of this are unclear.

	This Study	Frayer, 1989	Other studies
Neandertal	0.6% [1.2%]	0%	0.5% (Walker et al., 2011), 0.48% (Lanfranco and Eggers, 2012)
MPMH	7.8% [7.7%]	-	5.3%[t] (Caselitz, 1998)
EUP	1.90%	0%	-
LUP	3.8% [3.6%]	1.50%	-
Mesolithic	-	2.60%	7% (Wells, 1975), 1.85% (Meiklejohn et al., 1988), 4%[t] (Caselitz, 1998)
Natufians	2.40%	-	6.4% (Eshed et al., 2006), 0.2-3.0% (Smith, 1972)
Point Hope (Ipiutak)	1.00%	-	* (Costa, 1980b)
Indian Knoll	8.10%	-	* (Leigh, 1925; Rabkin, 1943), <7% for Archaic foragers in North America (Larsen, 1997)
Recent Hunter-Gatherers	-	-	<10% (Lanfranco & Eggers, 2012), 4.18% (without Inuits, Caselitz, 1998)

Table 4.7: Comparison of this study's results (Caries percentage, per teeth present) with previous publications; Values in parenthesis include published examples. [t]This value includes caries and antemortem tooth loss as Caselitz (1998) assumed tooth loss was a product of caries; *None of the previous publications report this value, nor the necessary variables to compute it

With the Middle Paleolithic modern humans removed, the caries prevalence appears to increase slowly over time from Neandertals to Early Upper Paleolithic modern humans to Late Upper Paleolithic modern humans. The possible reasons for this are discussed below. This trajectory may continue as published Mesolithic caries prevalences generally increase further (Table 4.7), but there is also likely a regional component. When all modern humans in the Late Pleistocene are pooled, the prevalence of caries per-tooth is 3.6%, considerably higher than the Neandertal prevalence of 1.2%. The large difference between the Neandertal and Middle Paleolithic modern human caries prevalences (1.2% vs. 7.7%) suggests there is no justified reason to pool the Middle Paleolithic samples with respect to caries affliction. The Middle Paleolithic was diverse with respect to human diet and oral environment.

The periapical lesion pattern here is quite different from the caries pattern. Middle Paleolithic modern humans have the least prevalence of alveoli affected by periapical lesions (1.6%) and Early Upper Paleolithic moderns have the most (5.0%), but this pattern does not differ significantly from predicted (Chi-Square, p-value: 0.244). This also suggests that lesions in the Late Pleistocene are rarely of carious origin, as they do not pattern with caries; however, the Middle Paleolithic modern human sample has no elderly individual (Trinkaus, 2011). This may produce an artificially low lesion rate for the Middle Paleolithic modern humans. The elderly category has the most lesions, as lesions generally increase with age (see age category subsection below). Lesions were found to covary with periodontal disease, but not caries in the Late Pleistocene (see Chapter 7: Oral Health & Systemic Health).

Results by Region:

This sample is divided into three broad geographic regions: Atlantic Europe, Continental Europe, and the Northern Circum-Mediterranean. Individuals living in these different regions

likely experienced different climates and utilized different foodstuffs, and therefore these differing environments and diets likely manifested as differential caries prevalences. Figure 4.8 maps out individuals—but not number of lesioned teeth—with caries. Regardless of region, it is clear that caries are more common at lower latitudes than higher latitudes, especially in the Middle Paleolithic. Frayer (1989) points out that most of his Early Upper Paleolithic specimens were from above 45° N latitude and most of the Late Upper Paleolithic specimens were from below 45° N, and this could explain the higher caries prevalence in the Late Upper Paleolithic (45° N latitude is placed on the map). However, the clustering of Late Upper Paleolithic sites in southern Europe is a result of glacial expansions around 20,000 bp, and therefore the environment was not necessarily warmer. The relationship between caries and climatic period is further tested using Marine Isotope Stage (MIS) and faunal assemblage variables (see table 4.9 and Apprendix 1), showing there are more caries from temperate sites than cold ones (Cold: 1.9% (10 of 968) vs. Temperate: 3.7% (35 of 942)). The negative latitudinal cline with caries continues into the Mesolithic (Meiklejohn et al., 1988; Frayer, 1989; Lukacs and Pal, 1993; Fujita, 2012).

	Atlantic	Continental	Late Pleistocene Mediterranean	Pooled Mediterranean (incl. Natufians)
No. Carious Teeth	13	5	29 [35]	46 [52]
Total No. Teeth	731	411	727 [768]	1434
Percentage of region w/ caries	1.8%	1.2%	4.0% [4.6%]	3.2% [3.5%]
No. Lesioned Alveoli	31	21	15	23
Percentage of region w/ periapical lesions	3.88%	4.94%	1.82%	1.50%

Table 4.8: Number of carious teeth organized by region; Values in parentheses include published examples (Caries: p-value: **0.024**; Periapical lesions: p-value: 0.078)

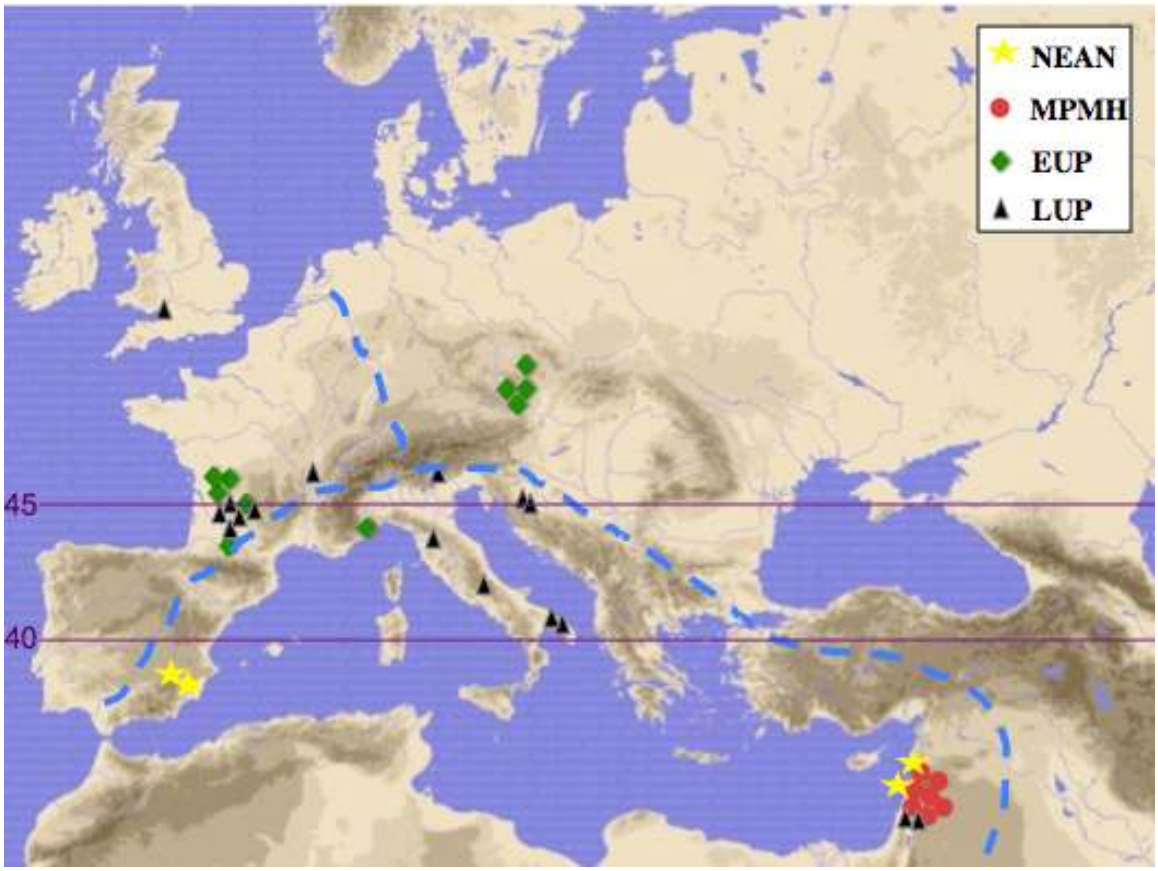


Figure 4.8: Map of individuals with caries in Western Eurasia: Locations are approximate (multiple individuals from the same site are represented by a wide grouping of dots). Dotted line indicates regional lines (Mediterranean, Atlantic & Continental); purple lines represent the 40°N and 45°N latitude lines

The numbers of carious teeth are presented by region in Table 4.8 and show a statistically significant pattern of many more caries in the Mediterranean region than predicted (Chi-Square, p-value: 0.024). One of the comparative samples, the Epi-Paleolithic Natufians, is also from Southwest Asia (Mediterranean region). Even when pooled with Paleolithic modern humans also from the Mediterranean region, the caries prevalence changes little (with versus without Natufians: Chi-Square, p-value: 0.362). The Continental and Atlantic regions caries prevalences are very similar, but the prevalence in the Mediterranean region is nearly twice that of the other two regions. This suggests the main dichotomy for caries is between the more southerly circum-

Mediterranean specimens and those specimens from further north in Europe. Many of these Mediterranean caries diagnoses cluster in Southwest Asia, but they are present across the whole upper Mediterranean region. There are few Early and Late Upper Paleolithic modern humans from Iberia available for study, but even the Neandertals—who rarely have caries—have caries in Spain along the Mediterranean (two from Sima de las Palomas and Banyoles 1) (Lalueza et al., 1993; Walker et al., 2011). All of the deciduous examples of caries are from the Mediterranean region as well (Qafzeh 4, Palomas 25, and Aubesier 5).

Periapical lesions appear have the opposite pattern, though it is non-significant (Chi-Square, p-value: 0.078). Once again the Atlantic and Continental regions are similar (3.9% and 4.9%), but they have twice (non-significantly) the lesions of the Mediterranean sample (1.8%). The higher Continental region value is likely related to periodontal disease, as this region also has the highest periodontal disease prevalence, though statistically insignificant (see Chapter 5). The Early Upper Paleolithic had the highest periapical lesion prevalence (5.0%), and many of the Continental specimens date to this period.

	Cool	Temperate
No of carious teeth	18	29 [35]
Total no. of teeth	968	901 [942]
% carious	1.9%	3.22% [3.72%]

Table 4.9: Number of carious teeth organized by climate; Values in parentheses include published examples (With published examples, p-value: **0.02**)

Sites were assigned to either cool or temperate climates based on MIS climatic research and/or faunal assemblages. 18 of the 53 (34%) carious lesions date to cool climatic periods and 35 (66.0%) of carious teeth are from temperate (warmer) climatic periods (see table 4.9). There

is no significant difference in caries prevalence by climate (Chi-square, p-value: 0.08) unless the published examples are added (p-value: 0.02). The great variance in data available per site made any more specific climatic assignment impossible.

Age Category:

	Adolescent	Young Adult	Mid-aged Adult	Elderly
No. Carious Lesion	11 [13]	19 [21]	17 [18]	0 [1]
Total No. Teeth	322 [324]	714 [731]	636 [652]	168 [174]
Percentage of age category w/ caries	3.4% [4.0%]	2.7% [2.9%]	2.7% [2.8%]	0% [0.57%]
Percentage of Total Carious Teeth	23.4% [24.5%]	40.4% [39.6%]	36.2% [34.0%]	0% [1.89%]
No. Lesioned Alveoli	2	18	15	21
Percentage of age category w/ periapical lesions	0.85%	2.32%	2.26%	7.87%

Table 4.10: Number of carious teeth and lesioned alveoli organized by age category (calculated by dental eruption and wear); Values in parentheses include published examples. (Caries: p-value: 0.333; Periapical lesions: p-value: **0.018**)

Caries increase in severity (size and penetration depth) over one's lifetime when there is no intervention (dentistry, dietary or otherwise), so one should not expect caries to be evenly distributed across a population. Table 4.10 details the distribution of carious teeth by age category in this sample. There is no pattern to the distribution of caries by age category though (Chi-Square, p-value=0.333). The highest percentage of carious teeth is in the adolescents (those aged between the eruption of M1 and M3) at 4.0%, followed by the young and mid-aged adults with similar prevalences of 2.9% and 2.8%, respectively. No elderly individuals had caries except Banyoles 1 (0.6%). The Point Hope sample also had the highest caries prevalence in the adolescent sample. Does this reflect the vulnerability of erupting dental enamel; differential diets

for the young; or attrition removing caries and the crenulations they flourish in as the individual ages?

Periapical lesions increase with age significantly (Chi-square, p-value: 0.018), which approximates the pattern seen in all the comparative samples as well. Periapical lesions are quite rare in Late Pleistocene adolescents, plateau between the young adults and mid-aged adults and then increase considerably in the elderly. Considering that there is only one example of elderly caries, these lesions' origins are likely periodontal disease, attrition-induced pulpal exposure, or trauma/breaks. Some individuals had alveoli, but no teeth, and therefore the aging technique used here could not be applied. There were 11 lesions in these un-aged individuals with 105 alveoli producing a periapical lesion prevalence of 10.5%. This is higher than any age group suggesting that the tooth loss in these individuals, whether ante- or postmortem, was likely related to the lesions, and this unaged sample is biased towards more lesions.

Caries Severity:

Caries were scored based on Hillson's (2001) scheme, which assigns most caries types (based on tooth type and location) a score between one and eight. A simple binary assignment of non-penetrant (enamel only, score 3) and penetrant (also affects dentin or pulp, scores 5-8) has been used elsewhere (Borgognini Tarli and Repetto, 1985; Frayer, 1989). Considering that the majority of caries are score 3 and score 5, the data are presented this way (Fig 4.9). Where scores 6-8 are appropriate (involves multiple surfaces and/or the pulp chamber), a designation of "severe" is made (see Table 4.11).

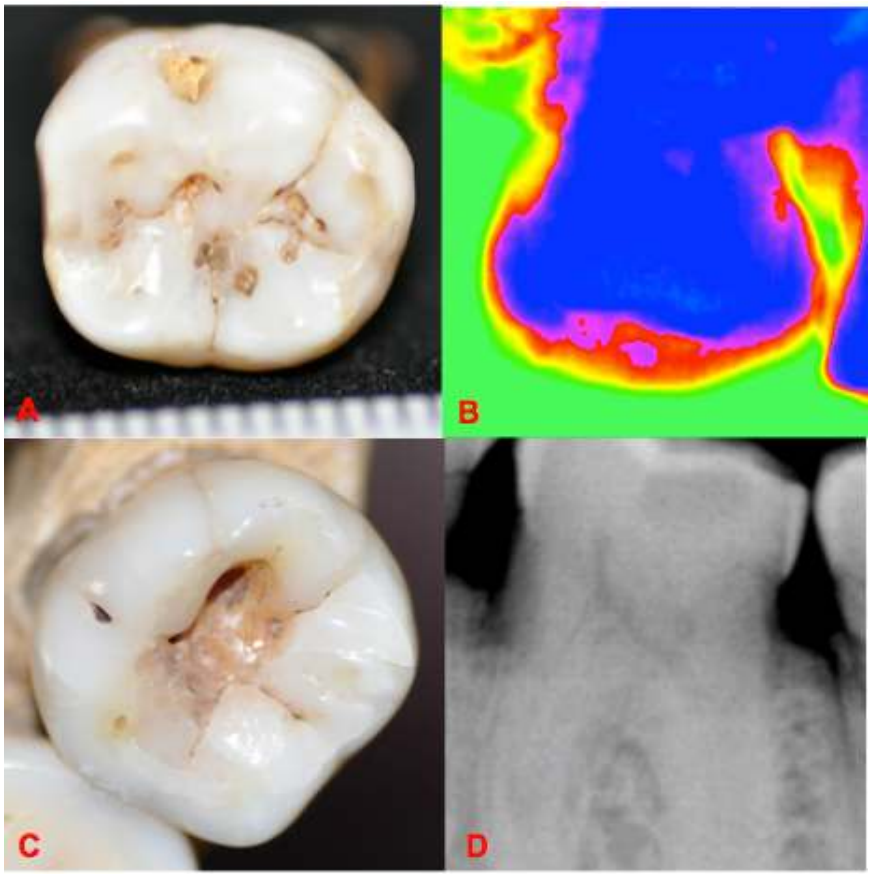


Figure 4.9: Images of non-penetrant and penetrant caries: A) Les Rois 23: Some occlusal caries are non-penetrant; B) Colorized radiograph of Ohalo 2 non-penetrant caries on M^3; C) Villabruna penetrant occlusal caries on M_3; D) Radiograph of Qafzeh 7 penetrant caries on M_2

Most Late Upper Paleolithic European caries in the earlier survey were non-penetrant (92.4%), but that dropped to 57.5% of carious lesions in the Mesolithic, meaning caries got more severe over time (Frayer, 1989). In this study, 64.2% (34 of 53) of carious teeth had non-penetrant lesions overall. Neandertals have the lowest prevalence of caries, but 62.5% (5 of 8) of their instances are penetrant. And conversely, Middle Paleolithic modern humans have the highest prevalence of caries, but the lowest percent of lesions that are penetrant (23.5%, or 4 of 17). However three of four examples of of penetrant lesions in the Middle Paleolithic modern humans were severe (i.e., affecting either the pulp chamber or multiple surfaces of the tooth).

The distribution of penetrant carious lesions over time is not significant though (Chi-Square, p-value: 0.802). Caries are not getting more severe over time in the total sample here.

	Neandertals	MPMHs	EUP	LUP
Non-penetrant (score 3)	1 [3]	13	7	10 [11]
Penetrant (scores 5-8)	3 [5]	3 [4]	4	6
Severe (6-8)	0	3	0	0
Percent Penetrant	75% [62.5%]	18.8% [23.5%]	36.4%	37.5% [35.3%]

	Atlantic	Continental	Mediterranean
Non-penetrant (score 3)	8	3	20 [23]
Penetrant (scores 5-8)	5	2	6 [9]
Severe (6-8)	0	0	3
Percent Penetrant	38.5%	40.0%	23.1% [28.1%]

	Adolescent	Young Adult	Mid-Aged Adult	Elderly
Non-penetrant (score 3)	7	13 [15]	11	0 [1]
Penetrant (scores 5-8)	4 [6]	6	6 [7]	0
Severe (6-8)	1	0	2	0
Percent Penetrant	36.4% [46.2%]	31.6% [28.6%]	35.4% [38.9%]	0%

Table 4.11: Caries severity organized by temporal group, region and age category; Values in parentheses include published examples. Chi-square: Temporal p-value: 0.802; Regional p-value: 0.922; Age p-value: 0.882

Caries severity was also organized by region and age category (Table 4.11). The Mediterranean region, which has the most examples of caries, has the lowest percentage of penetrant caries (23.1%) as compared to the Atlantic (38.5%) and Continental (40.0%) regions, which differ little (Chi-Square for all three regions, p-value: 0.922). Perhaps though caries are rarer further north, they are more severe when present. There is no pattern across age categories; the penetrant caries are equally spread across adolescents, young adults and mid-aged adults

(there is no caries in the elderly group, but Banyoles 1) (Chi-Square, p-value: 0.882). Carious lesions expand over a number of years, so one would expect the older age categories to have the most severe caries. These age categories are quite broad though (a decade or more). Lesions may have time to initiate, progress, and result in pulpal death within 10 years; or perhaps the quiescence phases of lesion development can last decades in this sample; or lesions are being erased by dental wear (Maat and van der Velde, 1987).

Results of Comparative Samples:

There are three comparative samples: Epi-Paleolithic Natufians (Nahel Oren, Mallaha, Kebara, El-Wad, Erq-el-Ahmar, and Hayonim), Archaic period Native Americans (Indian Knoll), and prehistoric Ipiutak Alaskan Natives (Point Hope). Their diets, mobility patterns and climates of residence all vary. The pattern of caries and lesions in these samples suggests that there is little similar about them.

Indian Knoll (Kentucky, ~5000 BP)

Overall caries and lesions prevalences suggest the Indian Knoll Native Americans have the highest caries prevalences (per-tooth or per-individual) and also a high lesion prevalence (highest per-individual). An early survey of their oral pathology reported a "low" frequency of 30% of skulls showing caries (Leigh, 1925). A later study reported that 21.3% of skulls showed some caries; however, skulls were chosen by their completeness for inclusion in the survey as well as a preference for young individuals with less dental wear (Rabkin, 1943). Therefore it is not surprising that the caries prevalence is lower when only younger, more complete specimens are surveyed—and methods have had 70 years to evolve. Leigh (1925) reports 150 osseous oral lesions in 66 crania but not the number of teeth affected or the distribution of lesions (it is highly unlikely that all of the skulls have lesions). 60.9% of the individuals having at least one lesion in

this survey is still quite a high prevalence though (16.0% of the teeth affected) (Table 4.12). A recent survey of American children reports an individual caries prevalence of 38.2% (Dye et al., 2012).

	Caries prevalence (per-tooth)	Caries Prevalence (per-individual)	Lesions (per-alveoli)	Lesions (per-individual)
Indian Knoll	8.1% (169 of 2089)	66.7% (50 of 75)	10.6% (222 of 2208)	64.0% (48 of 75)
Point Hope	1.0% (4 of 393)	8.7% (4 of 23)	7.2% (50 of 697)	60.9% (14 of 23)
Natufian	2.4% (17 of 707)	15.4% (8 of 52)	0.9% (8 of 896)	9.6% (5 of 52)

Table 4.12: Caries and lesion prevalence organized by tooth and by individual for each comparative sample

In Table 4.13, Indian Knoll caries prevalences increase with each age category until the elderly category where the prevalence drops (Statistically significant: Chi-Square, p-value: 0.0189). This is likely explained by high antemortem tooth loss in the elderly sample (see Chapter 6). Caries progresses until pulpal death or severe bony resorption from infection occurs, and the tooth is shed. The number of teeth lost before death from caries can be calculated with the "Caries Correction Factor" (Lukacs, 1995), but it assumes that at least some tooth loss is caused by caries (see Chapter 7: Oral Health & Systemic Health). Lesions increase consistently and significantly through the aging process (Chi-Square, p-value: $<<0.001$), and this supports the above hypothesis that the drop in caries in the elderly is a result of tooth loss from pulpal death and infection.

	Adolescent	Young Adult	Mid-Aged Adult	Elderly
Indian Knoll Caries	5.1%	7.4%	12.4%	5.2%
Indian Knoll Lesions	0.9%	6.4%	19.3%	24.4%
Point Hope Caries	2.8%	0.9%	0.7%	0%
Point Hope Lesions	0%	4.5%	19.2%	27.6%
Natufian Caries	0%	3.2%	1.1%	-
Natufian Lesions	0%	0.3%	2.4%	-

Table 4.13: Caries and lesion prevalence organized by age group for each comparative sample

Point Hope (Alaska, 100 BC-800 AD)

The Point Hope sample has the smallest sample size. They have the lowest caries prevalence of any comparative sample, but the highest lesion prevalence. Therefore it seems reasonable to assume these lesions are not of carious origins (unlike the Indian Knoll sample) (tested and confirmed in Chapter 7: Oral Health & Systemic Health). These Arctic peoples were caribou hunters, supplemented with marine mammals (namely seals) and fish and little vegetative matter (Larsen and Rainey, 1948). Because of this subsistence pattern, Arctic peoples have often been used as modern climatic and dietary analogies to Neandertals (*see discussion*). Costa (1980b) reports that caries prevalence peaked around the mid-aged adults and declined afterwards. This does not match the pattern here; caries prevalence does not change across age groups (Chi-Square, p-value: 0.722).

The lesion values here for Point Hope are also considerably higher than Costa's (1980b) values, which found the highest "abscess" prevalence to be in those aged 26-30 with 1.5% of individuals having lesions, compared to 60.9% overall here—a major difference. This is caused by a methods discrepancy though. Costa did not use radiographs and only included evidence of infection likely related to caries. As the caries prevalence is low, the lesion related to caries

prevalence is justifiably low as well. I did not make such a distinction. Costa (1982) also surveyed periodontal disease in a subsequent publication, which found high periodontal disease rates. As mentioned before, these lesions are indications of infection—but not likely of a carious origin—and this causes difficultly in comparing the results here with Costa's (1980b) survey. In Table 4.13, the lesion prevalence intensifies consistently and significantly with age in the Point Hope sample (Chi-square, p-value: 0.012).

Natufians (Israel, 13,100 - 9650 calibrated BC)

Previous Natufian research suggested an overall caries prevalence of 6.4% per-tooth (Eshed et al., 2006), much higher than what was found here, 2.4%. An older study reported a range of values per site ranging from 0.2% for Kebara to 3.0% for El Wad (Smith, 1972). However the Natufian sample is diverse including multiple sites, a range of several thousand years, rapidly changing mobility patterns and diet, and early animal commensalism. The El Wad and Kebaran material housed at the Peabody Museum at Harvard had few examples of caries; however, there is no published description of the preservation bias that led to which specimens were selected for this collection. The more fragmentary remains may not have been selected for inclusion, biasing the sample towards individuals with less pathology. El Wad and Kebara were not included in Eshed et al.'s (2006) survey. With all the various Natufian specimens pooled together, the caries prevalence peaks with young adults, although the differences are non-significantly (Chi-Square, p-value: 0.279).

Comparative Natufian values organized by age category were not available for caries or lesions, but periapical lesions by tooth were reported to not exceed 1.5% (Eshed et al., 2006). This approximates the value here of 0.9% in the Natufians. Periapical lesions also increased through the aging process in the Natufians (Eshed et al., 2006), a common pattern seen in this

study (though non-significantly here, Chi-Square, p-value: 0.153). There is no elderly individual present in the Natufian samples. This could be that the dental wear categories used for the other comparative samples and the Paleolithic do not work here (the Natufians had less dental wear), or people were not living long. Natufians did have a short life expectancy (24.6 years) (Eshed et al., 2004), but the elderly category here encompasses people approximately 40 and older—not particularly elderly by modern standards. Alternatively there could be preservation or curation biases in the samples with older individuals either not being buried, not preserving well, or excavators are not keeping them.. Many of these sites were excavated in the first half of the last century and excavator or curator bias cannot be ruled out.

Discussion:

There are some patterns in the prevalence of caries in the Late Pleistocene. Some of these have been suggested before, but no previous publication has suspected or demonstrated that caries prevalences would be twice that of previous publications (Frayer, 1989)—the only values available before now that were not based on literature searches. As a summary of the caries results major findings, it was shown that: (1) Late Pleistocene caries are most common on molars; (2) Caries are not limited to the adult population, at least in the Mediterranean region; (3) Most carious lesions are on occlusal surfaces, followed by the lingual/buccal sides and then interproximal and cervical surfaces; (4) Caries prevalence does not change significantly across age categories; (5) Caries increase over time from the Neandertals to the Early Upper Paleolithic to the Late Upper Paleolithic, with the exception that Middle Paleolithic modern humans have a considerably higher caries prevalence than the other groups; (6) Caries prevalence in the Mediterranean is twice that of regions further north; (7) Most carious lesions are not severe enough to endanger the pulp chamber, and penetrant lesions did not vary significantly over time,

region, or age category; and (8) though the comparative samples show a wide range of caries prevalences, when caries are rare, they do not change significantly over age categories (Point Hope and Natufians); where common, they increase over time until they result in tooth loss and caries prevalence decreases in the elderly (Indian Knoll). This patterning has implications for a number of variables.

The summary of periapical lesion major findings are as follows: (1) Periapical lesions are somewhat more common than caries in the Late Pleistocene, but they do not co-vary (see Chapter 7) and do not significantly pattern over time or region; (2) They increase significantly through the aging process in the Late Pleistocene sample and all the comparative samples (but not statistically for the Natufians (p-value: 0.153)); and (3) In the Indian Knoll and Point Hope samples, the per-individual prevalence of lesions is above 60%. The periapical lesions are rarely of carious origin (except at Indian Knoll, see Chapter 7), and therefore one must look elsewhere for their etiology.

Taxonomy:

Pooled modern humans have three times the caries prevalence of Neandertals (1.2% vs. 3.6%), and this disparity is further exaggerated if you compare Neandertals and modern humans in the Middle Paleolithic who have 6.4 times the prevalence (1.2% vs. 7.7%) (Table 4.6). However the regional data probably explain the caries patterning better than taxonomy without data on Middle Paleolithic moderns from Africa and Central and East Asia. The four Neandertal carious teeth in the sample are from Spain and Israel, plus the published examples from Israel (Kebara), Spain (Banyoles) and Southern France (Aubesier), and they are all along the Mediterranean. Neandertals in Southwest Asia were exploiting similar food resources to the

modern humans with the exception of the effects of climatic variation occurring between 110,000 ya and 50,000 ya in the region (Bar Yosef, 2004).

Many of the Early Upper Paleolithic and Late Upper Paleolithic modern humans sampled are from outside the Mediterranean region, and yet they still have more caries than Neandertals. These differences suggest that besides the higher caries prevalence around the Mediterranean for all hominin groups, there are still differences in caries prevalence between Neandertals and early moderns. Caries are strongly tied to diet in recent humans, and this suggests possible dietary differences between Neandertals and early modern humans. But other dietary research has shown only regional differences in diet, not necessarily taxonomic ones (Stiner, 1994; Hardy, 2010; Fiorenze et al., 2011; Henry, 2011; Trinkaus, 2013).

Neandertals also have fewer lesions than pooled modern humans (2.5% vs. 3.6%). Other examples of Neandertal oral lesions have been reported (e.g., the osteolytic lesion from Riparo Mezzena (Condemi et al., 2012)), but generally oral lesions are uncommon in the Late Pleistocene (relative to 9.0% of alveoli at Indian Knoll or 10.6% of alveoli at Point Hope). This may be related to short life expectancies or increased mortality risk from infection in the Late Pleistocene.

Time Period:

It is difficult to parse out taxonomy from time in this analysis as the only significant temporal overlap in Neandertals and early modern humans sampled is in the Middle Paleolithic. Caries increase over time in Europe, but Southwest Asia does not follow the pattern. There is evidence of increasing cultural heterogeneity (Bosinsky, 1990) and resource exploitation intensity and specialization (Grayson and Delpech, 2002; 2006; Drucker and Bocherens, 2004; Stiner and Kuhn, 2006) and decreasing mobility (Holt, 2003) from the Early Upper Paleolithic to

the Late Upper Paleolithic all over Western Eurasia. These were likely the result of increasing group-level foraging costs from population density increases coupled with decreasing environmental productivity and available home ranges. In this scenario, dietary shifts and increasing regional variability in diet are not surprising (Richards et al., 2001), and this could manifest as increased caries in groups intensifying their sugar consumption (e.g., honey, fruits). But this would also mean that changes over time in caries prevalence ultimately represent changes in diet.

And though caries is increasing by time period when Middle Paleolithic modern humans are removed, they do not increase directly and consistently over time. Rather there is a lot of variation within time periods, but the average per time period increases (Frayer, 1989). Frayer (1989) reported no significant correlation between ^{14}C dates and caries "rates", i.e., no gradual change. Rather there was a sudden shift around the Last Glacial Maximum. This pattern was also seen in stature studies. Stature did not decrease consistently over time between the Early Upper Paleolithic and Late Upper Paleolithic, but rather it was a step-wise change (Holt and Formicola, 2008; Meiklejohn and Babb, 2011). A doubling of the caries prevalence from the Early Upper Paleolithic to the Late Upper Paleolithic in this study (caries prevalence of 1.9% to 3.9%) may not be huge (and non-significant when only the two are compared; Chi-square, p-value: 0.298), but it is still substantial in the larger context of the Late Pleistocene (Chi-square, p-value: 0.005).

Region:

The Mediterranean region has 2 to 3.8 times the prevalence of caries of the Continental and Atlantic regions (Mediterranean: 4.6% vs. Atlantic: 1.8% and Continental: 1.2%). This suggests a negative latitudinal pattern to caries prevalence. As latitude decreases, plant sugars increase (Kirschbaum, 2004; Zheng et al., 2009)—and by extension human dietary sugars. Plant

sugar levels are directly related to photosynthesis and the cumulative amount of daylight. A meta-analysis of hunter-gatherer diets found relatively consistent carbohydrate consumption between 40° North and South latitude, but significant decreases in hunter-gatherer carbohydrate consumption beyond 40° latitude (Ströhle and Hahn, 2011). The 40°N Latitude line was placed on Figure 4.7 for comparison, and all Middle Paleolithic examples of caries (Neandertal and modern human) are below 43°N (Aubesier and Banyoles are between 40°N and 43°N, but the faunal assemblages suggest they were always climatically temperate) (Lalueza et al., 1993; Lebel and Trinkaus, 2002a). The negative correlation between carbohydrate consumption and latitude mirrors Frayer's (1989) data, which found that latitude was negatively correlated with caries "rate", as well as other Mesolithic analyses in Europe (Meiklejohn et al., 1988), India (Lukacs and Pal, 1993), and Japan (Fujita, 2012). Surveys of extant humans often find more caries at northerly latitudes because dietary sugar supplementation—and the socioeconomics required for that access—tends to correlate directly with latitude (Dunning, 1953; Powell, 1983).

No survey of modern caries incidence makes an explicit connection between the Mediterranean region and increased caries, with the exception of some dietary components such as dates, figs, and carob consumption (*the diet of the Mediterranean region is explored below*) (Nelson et al., 1999). Bronze age Greeks and Cretans have more caries than contemporaneous Western Europeans, and this has been attributed to honey and dried fruit consumption (Angel, 1944; Carr, 1960; Wells, 1975). The Epi-Paleolithic site of Taforalt in Morocco has a caries prevalence of 5.9% according to Wells (1975), but that has since been changed to 51.2% of teeth in the Grotte des Pigeons cave at Taforalt (Humphrey et al., 2014), and further exploration of Late Pleistocene sites and their oral pathology along the Mediterranean is highly warranted.

However it is possible that this regional (Mediterranean-specific) pattern is actually a general latitudinal or climatic pattern.

The mineral content of local water sources also affects caries prevalence with certain minerals, namely fluoride, having an inhibitory effect on the development of caries. Much of the local fluoride content is randomly variable with local geology, but there are some overall geographic patterns, one of which is proximity to the ocean. Water sources closer to the ocean tend to have lower fluoride, and therefore some modern coastal communities have higher caries incidence (Dunning, 1953; Bang, 1964). The pattern did not bear out in a Mesolithic survey of Europe (Frayer, 1989), but fewer Neandertals fossils were found in close proximity to the ocean—however there are also plenty of Neandertal Middle Paleolithic archaeological sites along the coast (Finlayson, 2008; Stringer et al., 2008; Richards and Trinkaus, 2009). The circum-Mediterranean caries pattern here is therefore provocative in reference to dietary fluoride. Though temperate climate sites do show statistically more caries than cool climate sites, perhaps the Mediterranean pattern is further exaggerated by lower dietary fluoride dictating the relatively higher caries rate. An attempt to estimate distance from sites with carious teeth to contemporary coastlines would require additional study variables, as sea levels are not consistent over time. However the high mobility of Paleolithic peoples would be a confounding factor in this hypothesis.

Age:

Caries were present in all age categories here, but did not change with age. This could suggest younger individuals were eating more sweet foods, and as they are living in a high dental attrition environment, their carious lesions were removed as they aged. Newly erupted teeth are more vulnerable to caries formation as well (Hillson, 2008). This pattern was seen in a

longitudinal study of Nigerian children who did not have access to oral hygiene products, but their caries disappeared over time through attrition (Maat and van der Velde, 1987; Kubota et al., 1993). However the opposite pattern was observed in the Mesolithic; there was no caries in any individual who still had deciduous teeth (Meiklejohn et al., 1988). These authors argued that there was social differentiation where children were being denied access to these sweeter foods for any number of reasons. Perhaps the pattern here suggests that social differentiation had yet to arise in the Pleistocene, or children in the Mediterranean were given preferential access to foods like dates (there is evidence of their consumption at Shanidar (Henry et al., 2011))?

The Osteological Paradox may predict more caries in those who die younger though (Wood et al., 1992). Root caries, more common in the elderly in recent populations, were uncommon in the Late Pleistocene sample; most caries observed were occlusal and on relatively unworn teeth. This pattern may reflect poorer health in those who died young, or some differential diet along age lines. Weaning foods are often high in carbohydrates (Nout, 1993), and it is possible the older adults have less caries because their lesions were worn away, not because they were never present.

Diet:

Protein:

There has been a recent flourishing of dietary research focusing on the Late Pleistocene. Dietary nitrogen isotope analyses report the relative source of dietary proteins from the local tropic pyramid, and suggests early modern humans had greater diversity in their dietary protein sources than Neandertals (Richards and Trinkaus, 2009). The early modern human samples were variably utilizing fresh water fish, marine resources, and terrestrial herbivores, but the Neandertal samples are all consistently eating large amounts of terrestrial mammals (Richards et

al., 2000; 2001; Richards and Trinkaus, 2009). The archaeological data suggest though that modern humans were not utilizing totally novel sources of nutrition compared with Neandertals, but they were intensifying and specializing their use of food sources outside of large herbivores (Grayson and Delpech, 2002; 2006; Drucker and Bocherens, 2004; Stiner and Kuhn, 2006). Proteins and fats do not generally affect oral flora, but they raise the oral pH, which encourages calculus mineralization (Hillson, 1979). Sugars, especially sucrose, and processed (ground) starches do affect the oral flora because their digestion begins in the mouth (Hillson, 2008). Therefore this variance in protein sources should not directly affect caries prevalences, but it suggests larger patterns of increasing regional dietary differentiation in the Upper Paleolithic.

Dietary isotope analyses suggest Middle and Upper Paleolithic peoples are closer to top carnivores than omnivores in their isotopic signatures, and this may explain the low caries prevalences overall, at least in Neandertals relative to recent populations (Hillson, 2008; Richards and Trinkaus, 2009). High protein/low carbohydrate diets are associated with less caries and conversely low protein/high carbohydrate diets are associated with increased caries in recent humans (Hillson, 2008). Therefore osteological samples with low caries and high calculus are often reconstructed as eating diets high in protein and low in carbohydrates (e.g., Costa, 1980b; Bonsall et al., 1997). Late Pleistocene peoples were getting a large proportion of their calories from protein; however, the human body cannot live off protein alone. Protein metabolism costs the body water and calcium and produces high amounts of urea, putting costs on the liver, kidneys and bones. The diet must be subsidized with fats and/or carbohydrates to ensure protein poisoning does not occur (Cachel, 1997, but also see Speth et al., 1991).

Perhaps the pattern here of higher caries in the southern portion of the sample area reflects differential dietary solutions to avoiding protein poisoning while consuming a high

protein diet. Humans further north were focusing on fat consumption whereas people further south were using carbohydrates to the same end: maintaining a critical ratio of protein to fats or carbohydrates. This differential diet supplementation would therefore be reflected in higher caries prevalences for Mediterranean peoples (4.6% in the Mediterranean versus 1.6% for the pooled Continental and Atlantic regions). This caries cline continues into the Mesolithic with the circum-Baltic region having almost no caries (0.3%) to Mid-latitude Europe (2.3%) to Mediterranean Europe (9.2%) (Meiklejohn et al., 1988). The dietary isotope analyses assure us that protein consumption was incredibly high in Neandertals and Early Upper Paleolithic people, but to maintain the skeletal robusticity seen in these groups, calcium could not be endlessly sacrificed for protein metabolism. Shattered herbivore long bones from many sites across the Pleistocene suggest marrow extraction, a highly fatty food (Oatram, 2001). Cachel (1997) points to oil lamps from the Mid- to Late Upper Paleolithic as evidence of a decreasing reliance on dietary fat (i.e., fat could be spared from the diet for other uses). As individual-level foraging costs decreased from population expansion in the Late Upper Paleolithic, carbohydrate consumption likely increased across many regions leading to an increase in caries.

Carbohydrates:

Carbohydrate consumption in the Upper Paleolithic is suggested by a number of data types. Grindstones for processing wild grains have been found from Italy (Bilancino) to Russia (Kostenki) by 30,000 ya and are present at the site of Pavlov in the Czech Republic (Revedin et al., 2010), which also has an example of caries in Pavlov 1. Cook-stone technology ovens from Abri Pataud (France) to Tanegashima (Japan), also around 30,000 ya, suggest the consumption of carbohydrates including pre-biotic carbohydrates (Leach et al., 2006). The analysis of starch molecules preserved in dental calculus suggests Neandertals were eating nuts, grasses, and green

vegetables (Henry et al., 2011; Hardy et al., 2012), and that their diets varied regionally (Lev et al., 2005; Hardy and Moncel, 2011). There is also direct paleobotany evidence in the Middle Paleolithic of charred legumes and nuts from Kebara and Gibraltar (Barton, 2000; Lev et al., 2005); edible grass seeds from Amud (Madella et al., 2002); edible pulses from Abric Agut; hackberries from Mas des Caves (Rolland, 2004); and perhaps even tools for extracting edible inner bark of plants (Sandgathe and Hayden, 2003).

Not all carbohydrates are the same though. Various glucose polymers form starch molecules and their digestion begins in the mouth with amylase in the saliva (Lebenthal, 1987). Sucrose is the most carious of all sugar molecules, but there is no evidence of pure sucrose consumption until 500 BC (Galloway, 2000; Cordain et al., 2005). Fructose and glucose from fruits and honey were available with sucrose and are also especially carious. At the Late Upper Paleolithic Spanish cave art site of Altamira, honeycomb is depicted as well as possibly bees and honey collection ladders from 14,000 ya BP (Pager, 1976; Valladas et al., 1992). There are detailed honey depictions in Mesolithic cave art sites such as Bicorp (e.g., bees and hives, honey collecting) (Herńandez-Pacheco, 1924; Dams and Dams, 1977; Dams, 1978). Other sugary foods are grown in the Mediterranean today and whose consumption is associated with caries, such as dates (Nelson et al., 1999) and figs (Wells, 1975). Figs and carob were being consumed at the Mesolithic Italian site of Uzzo (Borgognini-Tarli and Repetto, 1985). Cooked starches can also be just as carious as a 10% sucrose solution (König, 2000), and Pleistocene peoples were almost certainly processing their starchy foods (see grindstones at 30 kya (Revedin et al., 2010)). There is little direct evidence of the consumption of any of these other highly sugary foods (fruits and tree gums) besides honey in the Pleistocene—other than the presence of caries.

Cariogenic bacteria in dental plaques must be present in the mouth for sugars to cause a major drop in oral pH (Stephan and Miller, 1943). Calculus (mineralized plaque) was quite common in Pleistocene peoples (Arensburg, 1996; personal observation). They were not practicing much oral hygiene to remove plaque and calculus, meaning if they did consume carbohydrates, the environment in the mouth was primed for a pH decrease (cariogenic bacteria were present in the mouths of Late Pleistocene peoples (Pap et al., 1995; Humphrey et al., 2014)). The patterning seen here where caries increase around the Mediterranean and in later time periods is certainly related in part to differential diets, likely including more carbohydrates. Meiklejohn et al. (1988) and Frayer's (1989) work also came to the conclusion that the increase in caries in the Late Upper Paleolithic and Mesolithic was related to increasing regional dietary variability.

Pleistocene versus Holocene:

The few previous analyses that examined caries prevalence in samples from Europe on both sides of the Holocene/Pleistocene divide have had mixed results: no difference between the Late Upper Paleolithic and Mesolithic (Frayer, 1989), or a quick decrease at the end of the Late Upper Paleolithic and recovering increase in the Mesolithic (Caselitz, 1998). By broadening the geographic and temporal view, it appears the story is much more complex. The Point Hope diet was likely the closest to European Middle Paleolithic and Early Upper Paleolithic peoples with a focus on large terrestrial herbivores, namely reindeer, and some supplementation with other animals and fish. The Neandertal caries prevalence of 0.6% (1.2%) and the Point Hope prevalence of 1.0% reaffirm this analogy. However the Point Hope sample has a higher lesion prevalence (9.0% vs. 2.5% in Neandertals). Neandertals and Point Hope peoples both experienced a high degree of dental wear and periodontal disease, so this is difficult to explain.

Perhaps the mortality risk associated with oral infection was ameliorated in the Point Hope sample through some behavioral aspect.

The Middle Paleolithic peoples have more caries (7.8%) than the Epi-Paleolithic ones (2.4%) in Southwest Asia. The Natufians were certainly consuming wild grains. A previously reported Natufian caries prevalence (6.4%) is closer to the Middle Paleolithic value here though (Eshed et al., 2006). Both the Natufians and Middle Paleolithic modern human samples are missing elderly individuals. The lesion per-alveoli prevalences are similar (Natufian: 0.9% vs. Middle Paleolithic moderns: 1.6%) and likely low due to low age profiles.

Indian Knoll peoples are described as pre-agricultural; however, they are also acknowledged as likely practicing some form of garden agriculture (Webb, 1946). This is a dietary pattern wholly unlike the Late Pleistocene, and that is reflected by both their caries and lesion prevalences, which differ markedly from the Pleistocene values (caries 8.1%; lesions 10.6%). They lived within the latitude range of the Mediterranean peoples, which perhaps explains the similar Middle Paleolithic modern humans caries value of 7.8%. The Middle Paleolithic modern humans had a lower lesion prevalence (1.6%) than Indian Knoll, likely related to their younger skewed age profiles; however it has been suggested that Middle Paleolithic modern humans had lower dental wear than European Neandertals and modern humans (Smith, 1977). The only consistent pattern over the Paleolithic and Holocene samples is that periapical lesions increase with increasing age (in all samples, but non-significantly in the Natufians). This is not surprising as lesions progress over time, and as one ages, there are continually more opportunities for bacteria to penetrate the periapical region (e.g., from caries, periodontal disease, pulpal exposure from severe attrition, tooth breaks, trauma).

Conclusion:

Though caries were not a major problem for Late Pleistocene peoples, they were more common than previously thought, especially for Middle Paleolithic modern humans. The increase in caries prevalence over time in Europe and the higher prevalence overall in the Mediterranean region suggest both regional and temporal variance in diet. Mediterranean peoples were likely eating foods with more sugars, especially sticky ones like fruit and honey, and by the Late Upper Paleolithic, similar diets were being adopted across Europe. Periapical lesions in the Late Pleistocene are rarely of carious origins and therefore should be assumed to be of another etiology. Also periapical lesions did not pattern over time or region, but did increase across age categories.

Chapter 5: Periodontal Disease

Introduction:

Periodontal disease is correlated with systemic health and diet (Garcia et al., 2001; Hujoel, 2009); however, it has never been thoroughly examined in populations outside the Holocene. Even when dental anthropologists refer to "ancient" periodontal disease analyses, they cite studies of the European Medieval period or the Pre-Colombian period of the Americas (e.g., Clarke, 1990; Fujita, 2012). Periodontal disease is known from *Australopithecus* (Ward et al., 1982; Ripamonti, 1988) including AL288-1 or "Lucy" (Shields, 2000), and therefore must have great antiquity in hominins. Occasionally periodontal disease examples have been presented for a Pleistocene region (Southwest France: Brennan, 1991) or analyzed with questionable methods (lesions and antemortem tooth loss used as a proxy for periodontal disease: Frayer, 1989), but never with tested methods from bioarchaeology on a large scale across prehistoric samples, though the need for such has been acknowledged (e.g., Fujita, 2012). Considering all the potential implications of periodontal disease (e.g., systemic inflammation, morbidity, carbohydrate consumption), it is surprising it has not been thoroughly researched for Pleistocene humans—despite alveolar bone not preserving as well as teeth. The sample here has been organized in the same way as the caries data: by time period/taxonomy, region, age category, and comparative samples. However the Pleistocene sample is smaller than the caries sample because alveolar bone needs to be present (a minimum of two alveoli). Therefore the Pleistocene sample contains 123 individuals—79 modern humans, 44 Neandertals (see Table 5.1 for temporal versus regional distribution). This does somewhat bias the sample towards specimens and sites with better preservation.

	Atlantic	Continental	Mediterranean	**Sum**
Neandertals	21	2	21	**44**
MPMH	-	-	10	**10**
EUP	11	21	3	**35**
LUP	17	2	15	**34**
Sum	**49**	**25**	**49**	**123**

Table 5.1: Temporal and regional distribution of periodontal disease sample: MPMH: Middle Paleolithic modern human; EUP: Early Upper Paleolithic; LUP: Late Upper Paleolithic

For data presented by individual, there are two diagnostic protocols. The first is scored for the most severe section of the mouth according to septa condition as long as at least two alveoli are affected. Lavigne and Molto (1995) also used the greatest CEJ-AC measurement per tooth in their analysis of complete specimens, giving a bioarchaeological precedent for focusing on the most severe diagnosis. The second method uses average CEJ-AC distance per individual (see Chapter 3: Materials and Methods). Despite the non-uniformity of periodontal resorption, a previous study found the average of a small selection of alveoli from an individual predicted overall average CEJ-AC distance (Shrout et al. 1990). This protocol is more applicable to dentistry methods. Other data are presented as raw values by tooth type, specific tooth, etc., and therefore no diagnostic protocol is required.

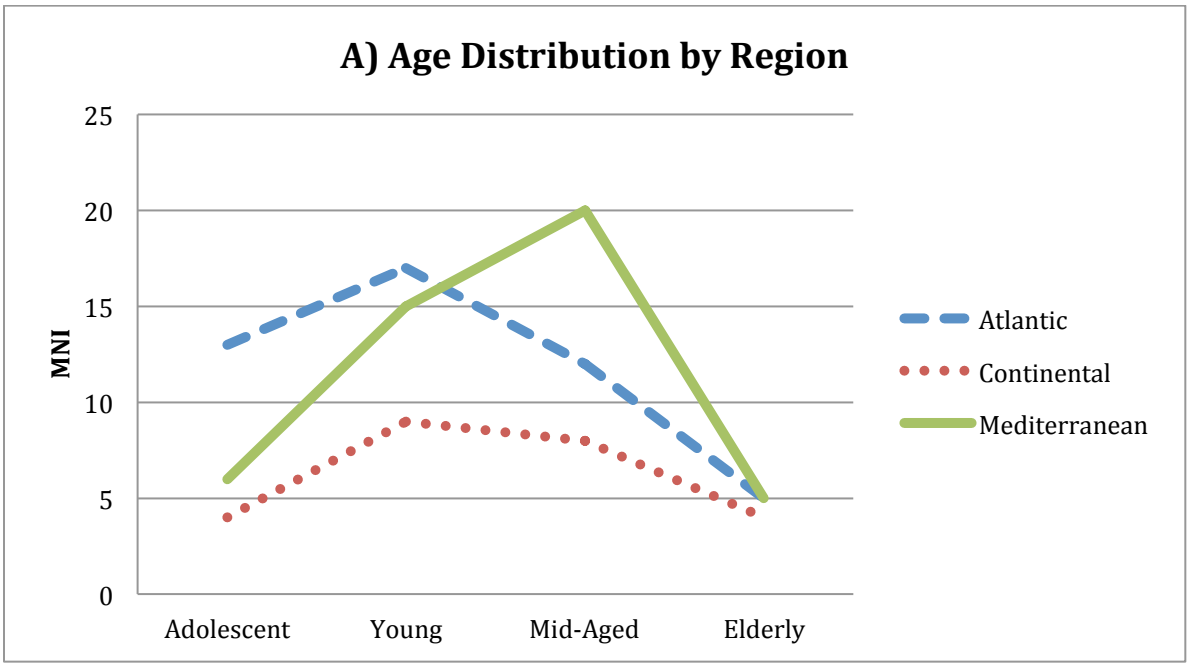
Results:

Age distribution:

Because periodontal disease is known to increase in presence and severity with age (Löe et al., 1992) as well as CEJ-AC distances potentially increasing with age for non-pathological reasons like continuous eruption (Varrela et al., 1995), it is important to make sure the variously defined groups do not have significantly different age distributions. Having a subsample skew

towards older individuals can make it artificially appear to have more periodontal disease. Because some fossils have no teeth preserved but the alveoli are in good enough condition to diagnoses periodontal disease, they are included in this study. However, they could not be assigned to an age category or given CEJ-AC measurements following the protocol here based on dental wear (this applied to five Late Pleistocene individuals). Therefore there are 118 individuals for which age could be assessed included in the analysis: 23 adolescents, 41 young adults, 40 mid-aged adults, 14 elderly. The age distributions of each region and time period are included in Figure 5.1.

Each time period and region has statistically indistinguishable age distributions (non-significant), with most individuals being either young or mid-aged adults with fewer adolescents and elderly individuals. The lack of elderly individuals in the Middle Paleolithic Modern Humans has not gone unnoticed by other researchers (Trinkaus, 2011); but since there are few individuals overall in this sample, it does not skew the pattern dramatically. The regions do not differ significantly by age distribution (Kruskal-Wallis, p-value: 0.23) nor do the temporal groups (p-value: 0.06; with MPMHs removed, p-value: 0.66). The Neandertals and EUP modern humans who straddle the Upper Paleolithic transition do not differ in age distribution (p-value: 0.77).



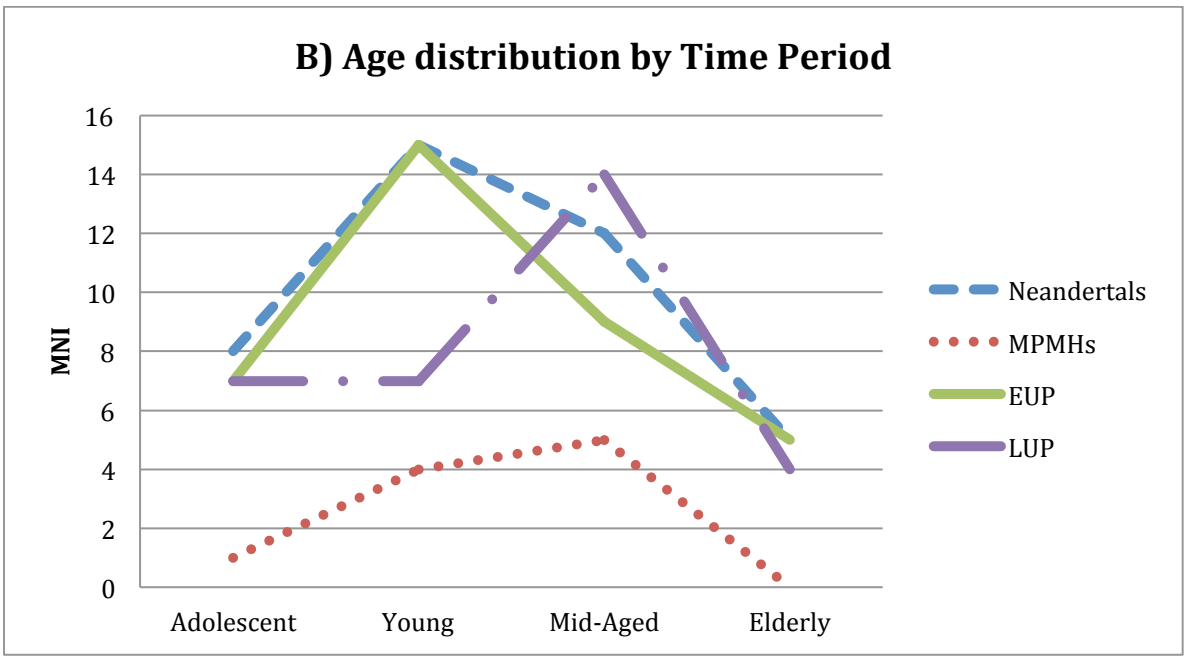


Figure 5.1: A) Age distribution of periodontal disease samples: A) by region (Kruskal-Wallis, p-value: 0.23); B) by time period (p-value: 0.66)

Maxillary vs. Mandibular:

The alveolar bone of the mandible and maxilla are not the same. The maxilla is more fragile in that the cortical bone is thinner because of the maxillary sinuses, and posterior maxillary teeth have more roots than mandibular ones (three-rooted molars are typical and double-rooted premolars are relatively common) (Scott and Turner, 1997). The mandible has thicker cortical bone, coupled with less complex dental roots. Other research has found periodontal disease to affect the upper and lower molars and lower incisors more than other teeth (Clarke et al., 1986); however continuous eruption also tends to be greater in the mandible (Glass, 1991; Appendix 3). So though the maxillary alveolar bone is more fragile, it appears to be less susceptible to increasing CEJ-AC distances from both alveolar bone loss from periodontal inflammation and continuous eruption. The mandible is more susceptible to periodontal disease, but also to continuous eruption, suggesting the researcher must be careful to differentiate the two in the mandible.

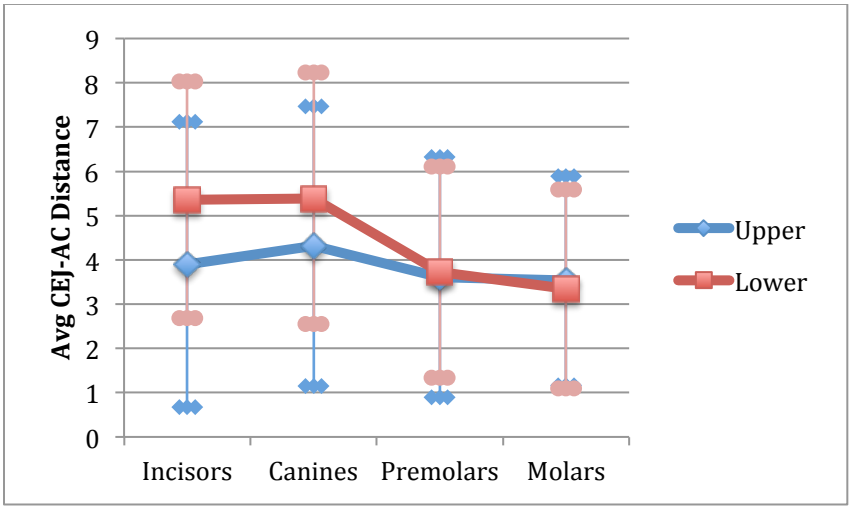


Figure 5.2: CEJ-AC average distances per tooth type organized by upper and lower teeth, with standard deviations (Using two sample mean comparison, p-values are as follows: incisors: <0.001; canines: 0.003; premolars: 0.701; molars: 0.470)

In this sample, CEJ-AC distances are greater in the mandible than the maxilla for anterior teeth, but not for posterior teeth (Fig. 5.2). This replicates Clarke and colleagues (1986) data for the incisors (i.e., more alveolar bone loss in the lower incisors than the upper ones), but not for the posterior teeth. There is no difference in CEJ-AC distance averages between the maxilla and mandible for the molars and premolars (but this was not tested as matched arcade pairs within individuals).

Time Period:

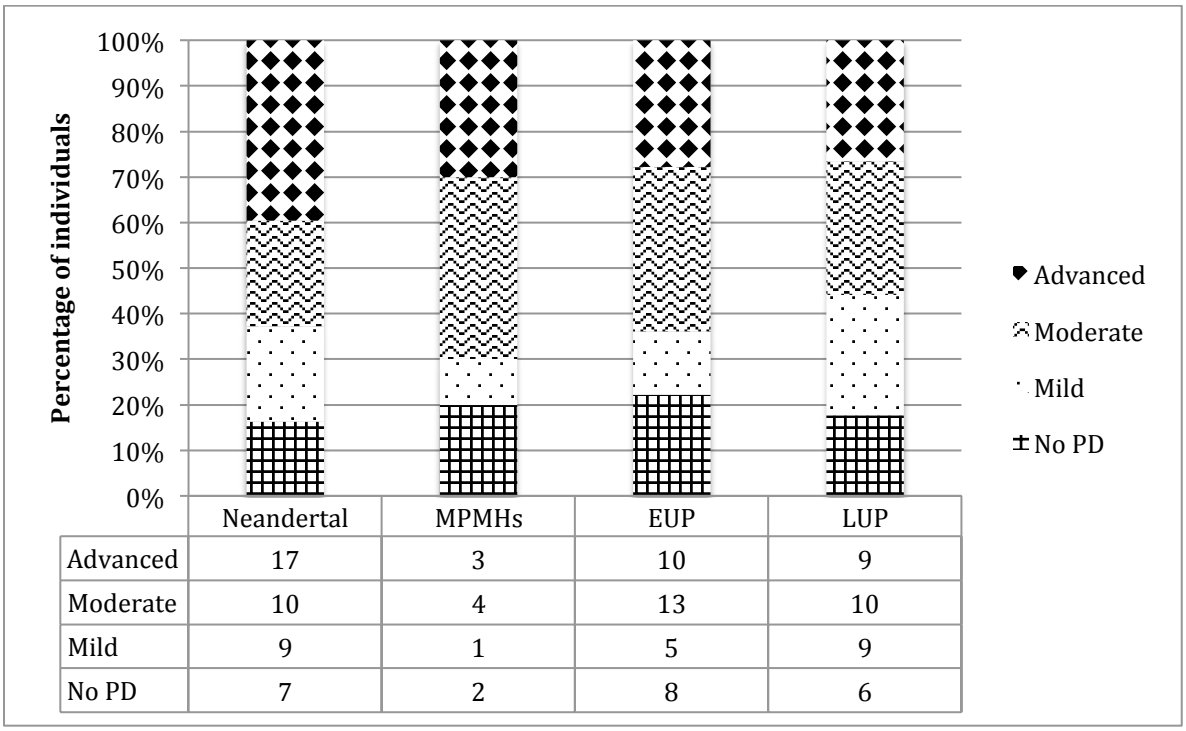
Because other research has found changes in health indicators over time in the Late Pleistocene of Europe, the null hypothesis here is that periodontal disease would follow the same pattern, i.e., the Early Upper Paleolithic is "healthier" than preceding and following groups. Based on both average most severe periodontal diagnosis score and average CEJ-AC distance per individual though, the score decreases consistently over time (Table 5.2). This pattern is not wholly consistent depending on how the data are further analyzed.

	Neandertal	MPMHs	EUP	LUP
Average Most Severe Periodontal Score	1.86	1.8	1.69	1.65
Average CEJ-AC Distance, mm	4.07	3.81	3.28	3.14

Table 5.2: Average most severe periodontal score per group (0=None, 1=Mild, 2=Moderate, 3=Advanced) and average CEJ-AC distance per individual per group

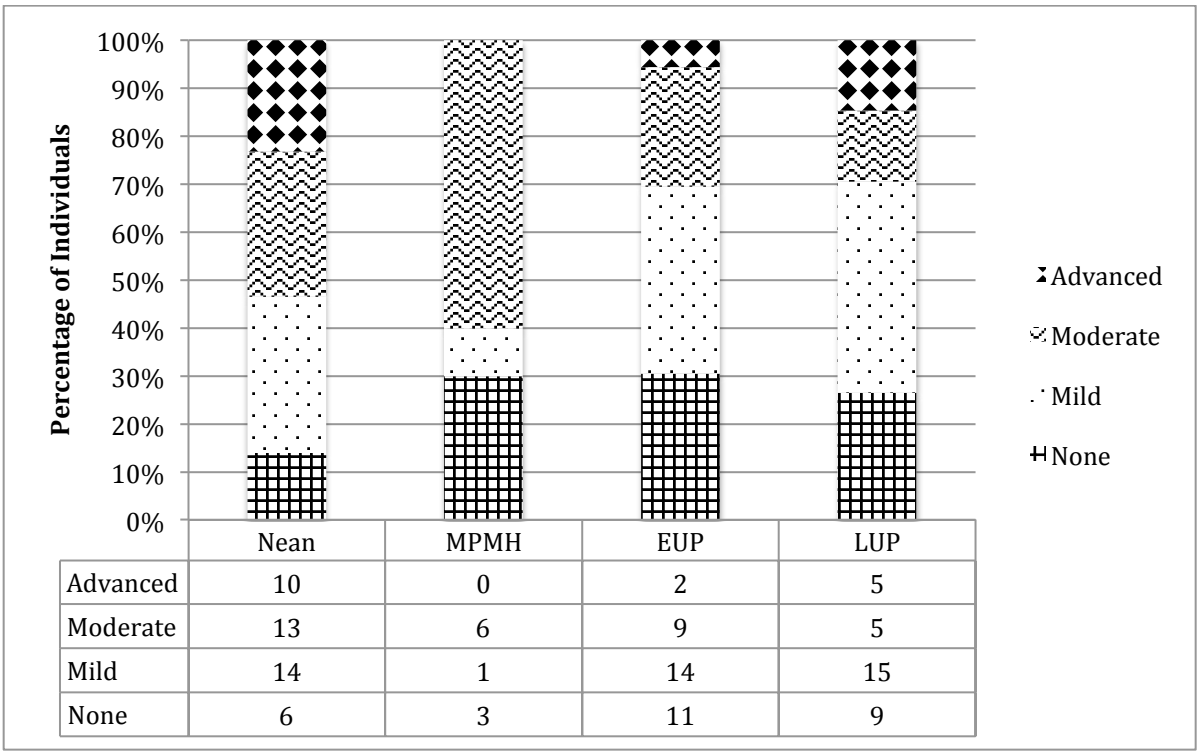
Using most severe diagnosis, the temporal/ taxonomic groups do differ statistically significantly (Kruskal Wallis, p-value: 0.033) (Fig. 5.3). Neandertals have more advanced periodontal disease than any of the modern human groups, and also fewer cases without disease than any of the modern human groups. The Late Upper Paleolithic group has less moderate and

advanced cases of periodontal disease than the Early Upper Paleolithic, suggesting improvement over time. There is a slightly higher percentage of Early Upper Paleolithic specimens with no periodontal disease than the Late Upper Paleolithic sample, but the Early Upper Paleolithic has more moderate and advanced diagnoses (Fig. 5.3). The percentage of "advanced" cases are those individuals whom had at least two alveoli with advanced disease or a localized infection, and this protocol automatically shifts the distribution towards more severe disease diagnoses.



	Neandertal	MPMHs	EUP	LUP
Advanced	17	3	10	9
Moderate	10	4	13	10
Mild	9	1	5	9
No PD	7	2	8	6

Figure 5.3: Percentage distribution of 4 ordinal periodontal disease severity diagnoses within each time period (Most severe score per individual) (p-value: 0.033)



	Nean	MPMH	EUP	LUP
Advanced	10	0	2	5
Moderate	13	6	9	5
Mild	14	1	14	15
None	6	3	11	9

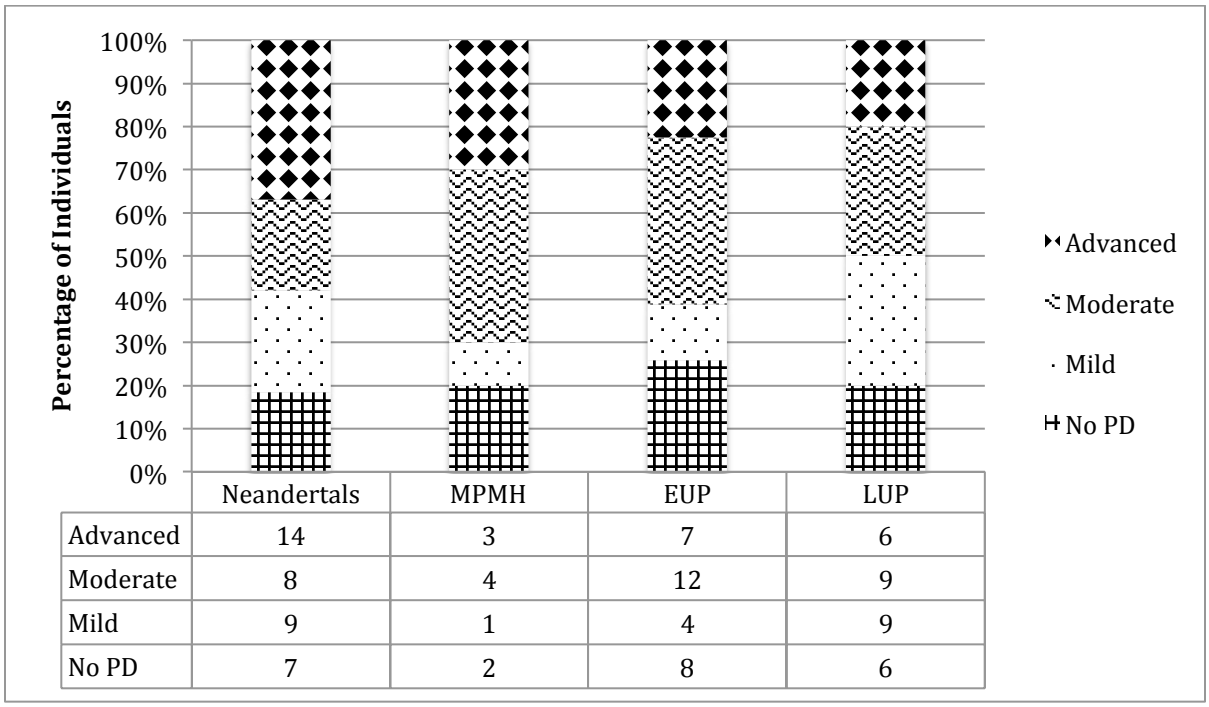
Fig. 5.4: Percent distribution of 4 ordinal periodontal disease severity scores based on average CEJ-AC distance per individual (p-value: 0.104)

The Late Upper Paleolithic modern humans have the least amount of advanced and moderate periodontal disease of any group. This is different from the pattern observed for other health and stress indicators, for which there was a decrease in health in the Late Upper Paleolithic relative to the Early Upper Paleolithic. Also the Late Upper Paleolithic sample is skewed slightly older than the other groups (but not significantly), which suggests periodontal health truly improved in the Late Upper Paleolithic (i.e., cases are less severe in spite of a slightly older sample). This could be related to a number of things, such as increased dietary diversity and therefore perhaps better nutrition in the Late Upper Paleolithic. The Neandertals compared with the pooled modern humans also show more periodontal disease, but not significantly (Kruskal-Wallis, p-value: 0.083). Neandertals have fewer periodontal disease free

cases as well as more advanced cases than the pooled modern humans. This suggests greater Neandertal morbidity using either presence/absence of disease or severity of disease.

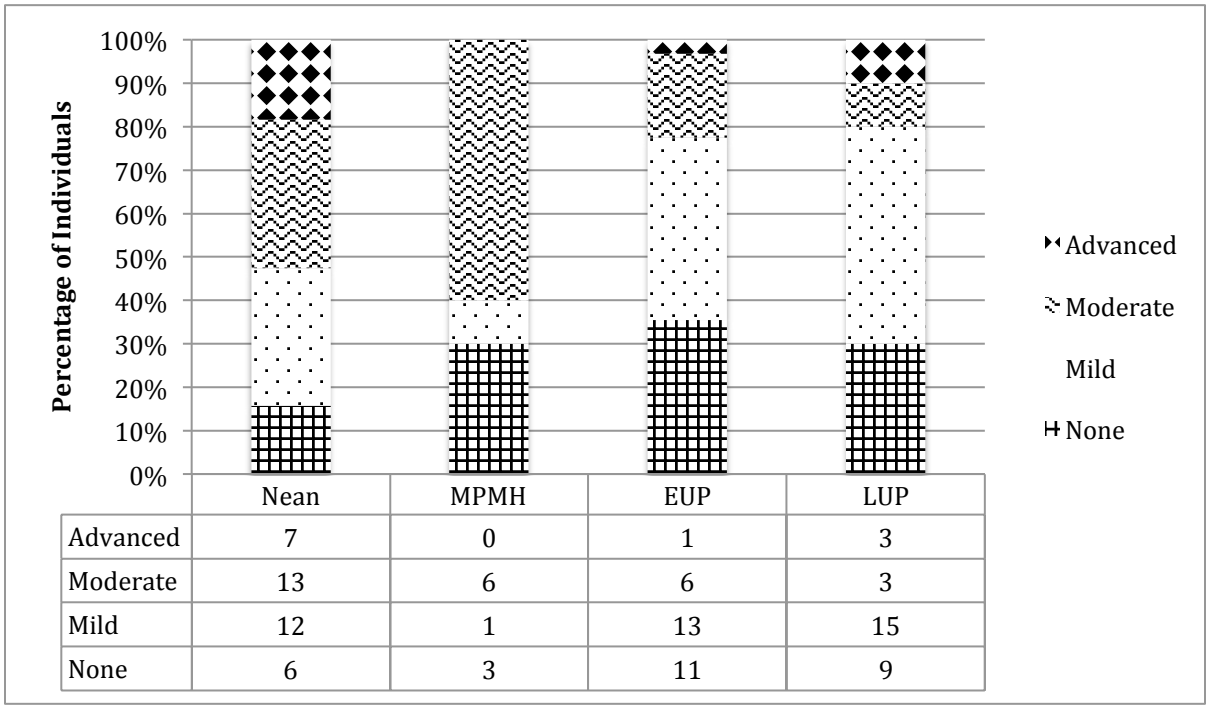
Based on average CEJ-AC distance, as opposed to most severe diagnosis, the pattern changes slightly and is no longer statistically significant (Kruskal-Wallis, p-value: 0.104) (Fig 5.4). Neandertals still have the most severe periodontal disease, but Early Upper Paleolithic modern humans have less periodontal disease diagnosis than with the other method, making them appear slightly healthier than Late Upper Paleolithic people. This may be biologically meaningful, in that it mirrors other stress indicator research.

Because there is no elderly individual in the Middle Paleolithic modern human sample, a further comparison is done with the elderly removed from all other groups (More severe septa diagnosis, Fig. 5.5; Average CEJ-AC diagnsos, Fig 6.6). This allows the Middle Paleolithic modern human groups to be more accurately compared to the rest. The pattern is still generally the same as with the elderly included: periodontal disease decreasing in severity over time for most severe septa diagnosis and statistically significant (Kruskal-Wallis, p-value: 0.0313); or an inflection in the Late Upper Paleolithic using average CEJ-AC distance diagnosis, but non-significant (p-value: 0. 0.169).



	Neandertals	MPMH	EUP	LUP
Advanced	14	3	7	6
Moderate	8	4	12	9
Mild	9	1	4	9
No PD	7	2	8	6

Fig 5.5: Percentage distribution of 4 ordinal periodontal disease severity diagnoses within each time period with the elderly individuals removed (Most severe score per individual) (p-value: 0.031)



	Nean	MPMH	EUP	LUP
Advanced	7	0	1	3
Moderate	13	6	6	3
Mild	12	1	13	15
None	6	3	11	9

Fig. 5.6: Percent distribution of 4 ordinal periodontal disease severity scores based on average CEJ-AC distance per individual with the elderly individuals removed (p-value: 0. 0.169)

Regardless of method, all of the groups show high prevalences of periodontal disease; completely periodontal disease free individuals range from 16.3% of the Neandertal sample to 22.2% of Early Upper Paleolithic samples (Fig. 5.3). So though Neandertals show greater morbidity, it is relative to other high morbidity samples. The Natufians (27.7%) and Indian Knoll (31.1%) have more periodontal disease free individuals, but Point Hope has none (i.e., everyone had at least some mild periodontal disease). Point Hope's age distribution is skewed older though (Table 5.3)

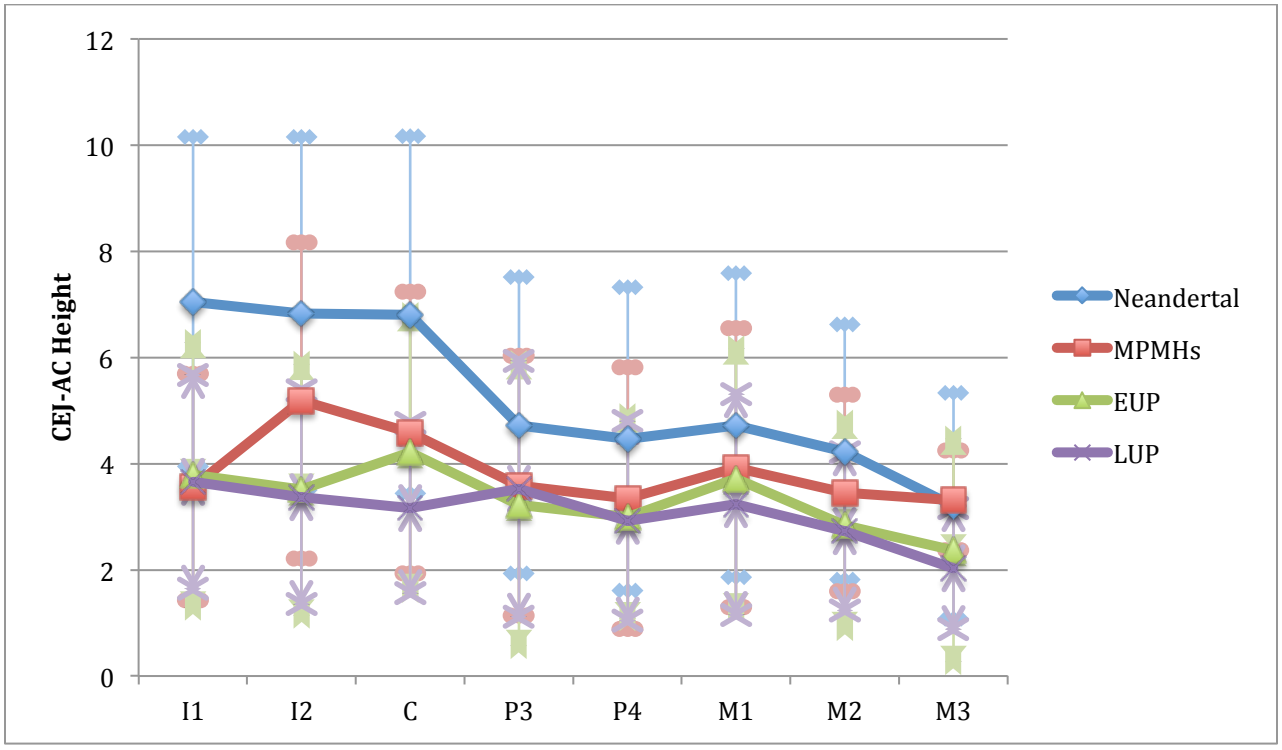


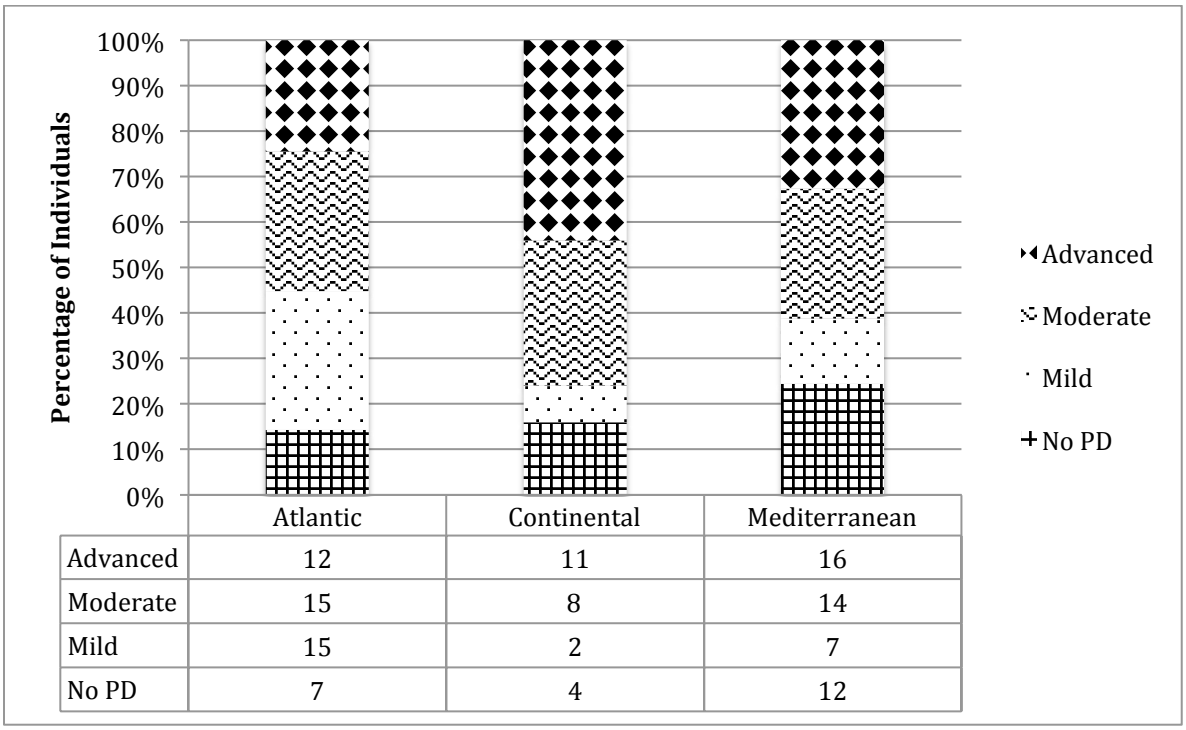
Figure 5.7: CEJ-AC distances per tooth organized by temporal/taxonomic group (standard deviations included)

Using CEJ-AC distances alone per tooth (right, left, upper and lower pooled) also show that Neandertals have considerably more alveolar bone loss than modern humans, especially in

anterior teeth (Fig. 5.7). The Neandertal CEJ-AC mean is more than a standard deviation above the CEJ-AC mean for the Early Upper Paleolithic and Late Upper Paleolithic for the anterior teeth. Using two-sample mean comparison, the Neandertal distribution and mean does not describe the modern human pooled samples distribution and mean for all tooth types (p-value range: 0.003 to <0.001) except the third molar (p-value: 0.074). This supports the most severe diagnosis protocol used above, which also found Neandertals have more periodontal disease, and modern humans cluster closer to one another (Fig. 5.3). The modern human pattern here also reflects the average periodontal score pattern (Table 5.2), with the mean alveolar bone loss per tooth decreasing over time for all teeth but the third premolars.

Region:

The temporal pattern in periodontal disease presence and severity is not strong within modern human and neither is the regional pattern. Continental Europe has more severe periodontal disease than Atlantic Europe, but not significantly (Kruskal Wallis, p-value: 0.081 between the two; p-value: 0.123 for all three regions together). 44.0% of the Continental sample has advanced periodontal disease, compared to 32.7% in the Mediterranean and 24.5% in the Atlantic (Fig. 5.8).

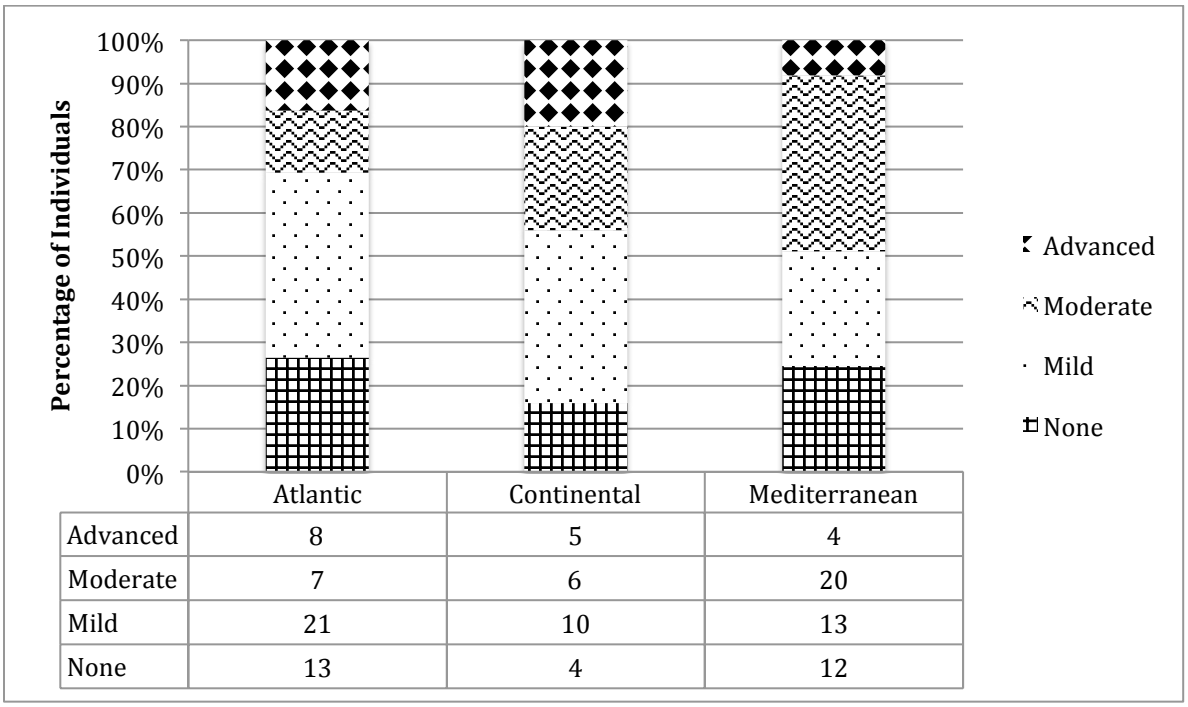


	Atlantic	Continental	Mediterranean
Advanced	12	11	16
Moderate	15	8	14
Mild	15	2	7
No PD	7	4	12

Figure 5.8: Percentage distribution of 4 ordinal periodontal disease severity scores per region based on most severe diagnosis (p-value: 0.123)

The Mediterranean pattern differs less from the Atlantic region than the Continental regional pattern does. But despite having more advanced periodontal disease than the Atlantic, the Mediterranean also has more individuals without any periodontal disease. The Mediterranean pattern is therefore somewhat bimodal, whereas the Atlantic region has its periodontal disease severity more evenly distributed. The regional pattern remains non-significant when using average CEJ-AC distance (Kruskal-Wallis, p-value: 0.198) (Fig. 5.9).

The CEJ-AC distances per tooth also show little difference between regions (Fig. 5.10). The CEJ-AC distance pattern across the dental arcade follows the temporal pattern (greater CEJ-AC distances in the anterior teeth and first molar), but does not separate out regional groups.



	Atlantic	Continental	Mediterranean
Advanced	8	5	4
Moderate	7	6	20
Mild	21	10	13
None	13	4	12

Figure 5.9: Percentage distribution of 4 ordinal periodontal disease severity scores per region based on average CEJ-AC distances per individual (p-value: 0.198)

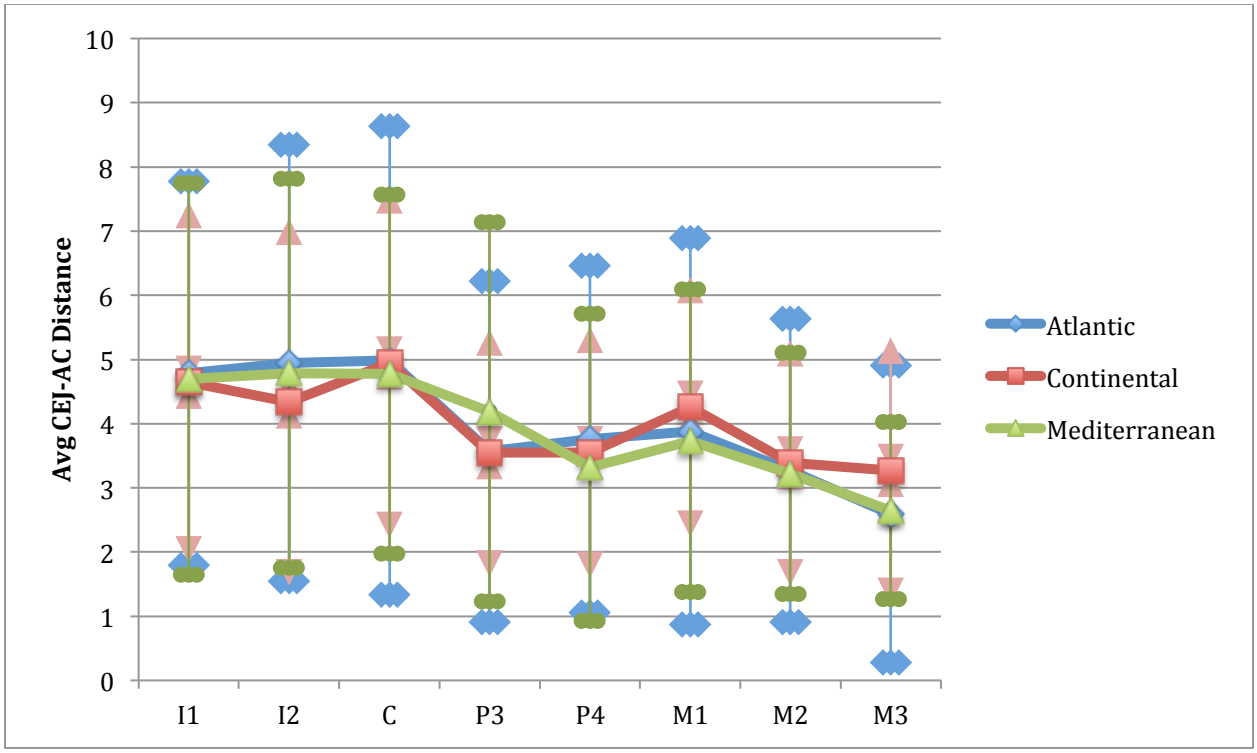
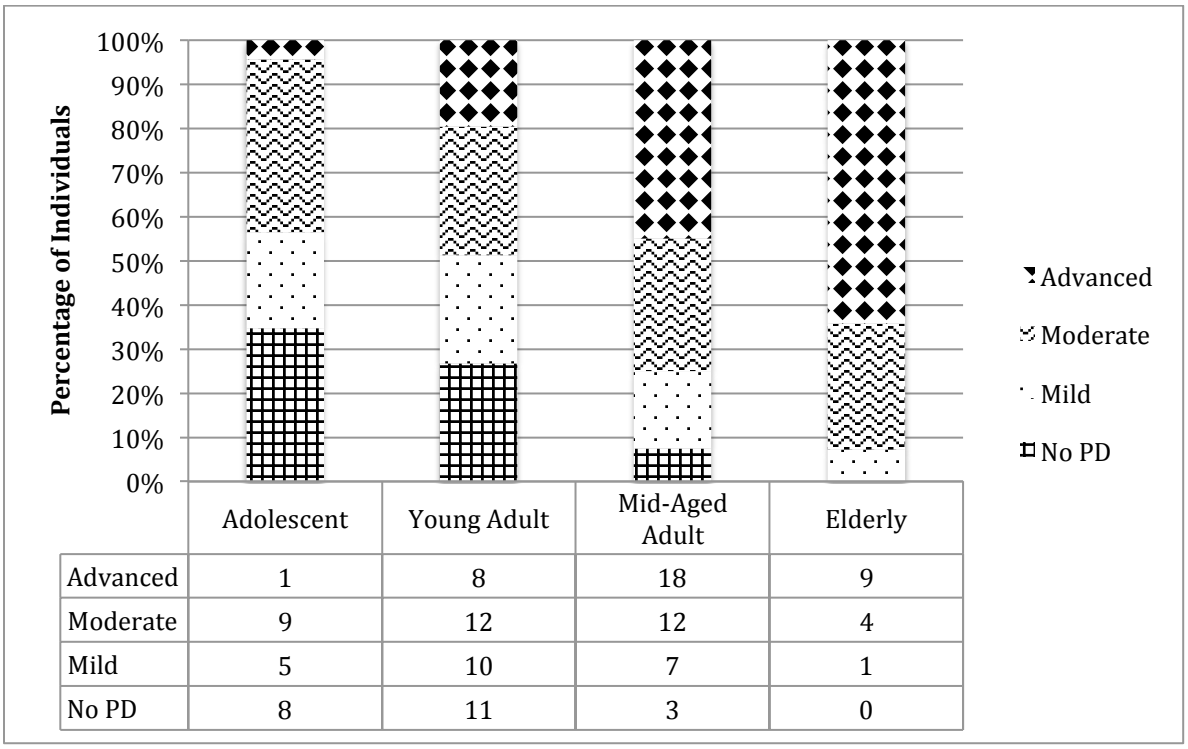


Figure 5.10: CEJ-AC distances per tooth organized by region (standard deviations included)

Age Category:

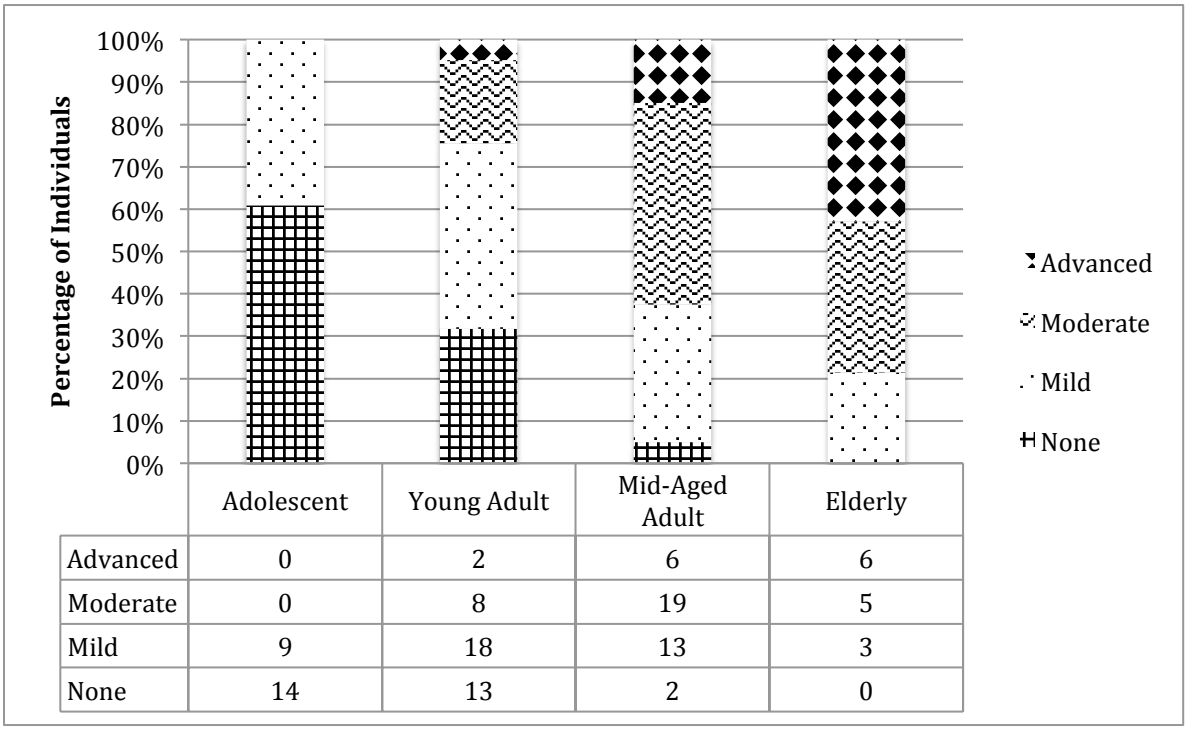
The distribution of periodontal disease severity across age categories is significant for both diagnostic protocols (Chi-square, p-value: 0.001), demonstrating a consistent increase in severity of disease prevalence and severity though age categories (Table 5.11 & 5.12). The percentage of individuals without periodontal disease decreases in each successive age category and the percentage of individuals with advanced periodontal disease increases. Few adolescents had advanced periodontal disease, though it was present in one individual, Laugerie-Basse 2. All elderly individuals—but Sunghir 1 with mild disease—had moderate or advanced periodontal disease. There are plenty of moderate cases in the adolescent group though. Localized aggressive periodontitis is not unknown from the fossil record; one of the Sterkfontein *Australopithecus africanus* juveniles (STS 24 & 24a) has it (reported as prepubertal periodontitis, Ripamonti, 1988). It is a rare condition, but when present, it progresses rapidly and severely and can produce early tooth loss (Page et al., 1983; Nibali et al., 2013).

Using average CEJ-AC distance per individual (Fig. 5.12), the pattern is the same with periodontal disease increasing in prevalence and severity through the aging process (Chi-square, p-value: <<0.001). Laugerie-Basse 2 is no longer diagnosed as having severe periodontal disease with this protocol.



	Adolescent	Young Adult	Mid-Aged Adult	Elderly
Advanced	1	8	18	9
Moderate	9	12	12	4
Mild	5	10	7	1
No PD	8	11	3	0

Figure 5.11: Percentage distribution of 4 ordinal periodontal disease severity diagnoses within each age category using most severe diagnosis (p-value: 0.001)



	Adolescent	Young Adult	Mid-Aged Adult	Elderly
Advanced	0	2	6	6
Moderate	0	8	19	5
Mild	9	18	13	3
None	14	13	2	0

Figure 5.12: Percentage distribution of 4 ordinal periodontal disease severity diagnoses within each age category using average CEJ-AC distance (p-value: <<0.001)

CEJ-AC distances increase through the aging process at all teeth (Fig. 5.13). Since the definition of the adolescent group includes that their third molar not be in full occlusion, the mean CEJ-AC distances for adolescent third molars is barely above zero. The elderly group shows a deviation from the general pattern across the dental arcade in that their first molars show more alveolar bone loss than any other tooth including the incisors. Other studies have found the first molar to be a common site for localized periodontal disease in adults (Clarke et al., 1986; Glass, 1991; Brown and Löe, 1993), which likely reflects the first molar's long time in the oral cavity (from age 6). It experiences more wear and therefore is at greater risk for pulpal exposure as well as potentially accumulating more dental calculus.

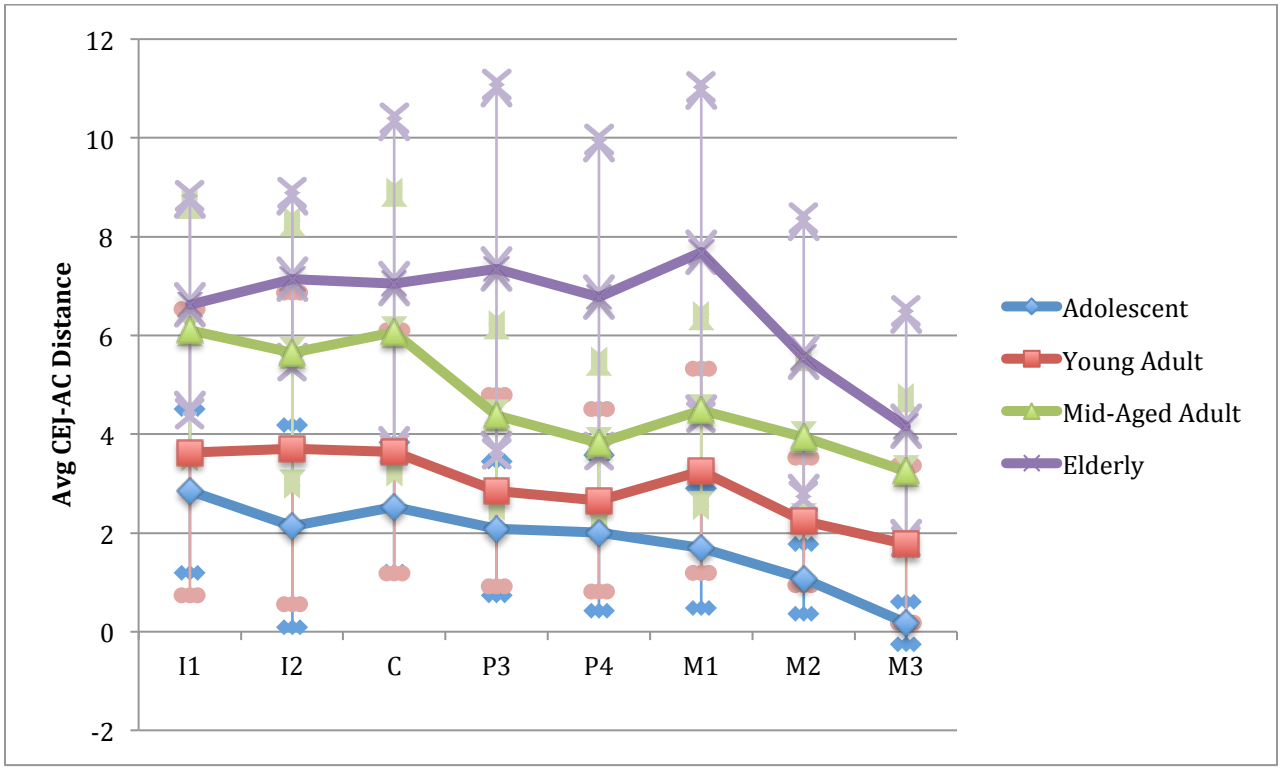


Figure 5.13: CEJ-AC distances by tooth organized by age category (standard deviations included)

Comparative Samples:

The small size of the Point Hope sample may affect the age distribution skewing older than the other two samples (Natufian: N=47; Point Hope: N=23; Indian Knoll: N=74), and therefore it is not surprising that it has much more severe periodontal disease than the other two samples (Table 5.4). However Costa's (1982) analysis of the periodontal status of the Point Hope sample had many more individuals, and also found a high rate of periodontal disease, especially severe in those over the age of 35. Every individual over 35 he examined had some level of periodontal disease, suggesting the Point Hope pattern here is not solely an artifact of sample size. The age distributions of the three samples do not differ significantly (Kruskal Wallis, p-value: 0.186), but the periodontal disease severity does between the comparative samples (p-value: 0.023).

The Natufian sample shows few individuals with advanced periodontal disease, and there is no moderate or advanced periodontal disease in the adolescent sample. But the life expectancy of Natufians peoples is low (Eshed et al., 2004), and there is no elderly individual in the sample. The Indian Knoll sample is intermediate in periodontal disease distribution between the other two samples, but part of this is related to the young skew of the Natufians and the older skew of the Point Hope sample (though insignificantly different). This is also a pattern in average periodontal disease value (using most severe diagnosis) with Point Hope > Indian Knoll > Natufians (Table 5.4). All three samples show increased periodontal disease prevalence and severity over the aging process (Fig. 5.14).

		None	Mild	Moderate	Advanced	Sum
Natufian	Adolescent	3	1	0	0	4
	Young Adult	7	11	10	2	30
	Mid-Aged Adult	3	4	4	2	13
	Elderly	0	0	0	0	0
	Sum	13	16	14	4	47
Point Hope	Adolescent	0	1	2	0	3
	Young Adult	0	1	4	0	5
	Mid-Aged Adult	0	0	4	7	11
	Elderly	0	0	1	3	4
	Sum	0	2	11	10	23
Indian Knoll	Adolescent	12	2	1	0	15
	Young Adult	8	7	11	2	28
	Mid-Aged Adult	3	3	8	9	23
	Elderly	0	2	1	5	8
	Sum	23	14	21	16	74

Table 5.3: Comparative sample periodontal severity totals (most severe septa diagnosis) organized by group and age category (Kruskal-Wallis p-values for each age series, Natufians: 0.047; Point Hope: 0.004; Indian Knoll: <<0.001)

	Natufians	Point Hope	Indian Knoll
Average Most Severe Periodontal Score	1.19	2.35	1.33

Table 5.4: Average most severe periodontal score per comparative sample (0=None, 1=Mild, 2=Moderate, 3=Advanced). Compare to Table 5.2 for the Late Pleistocene

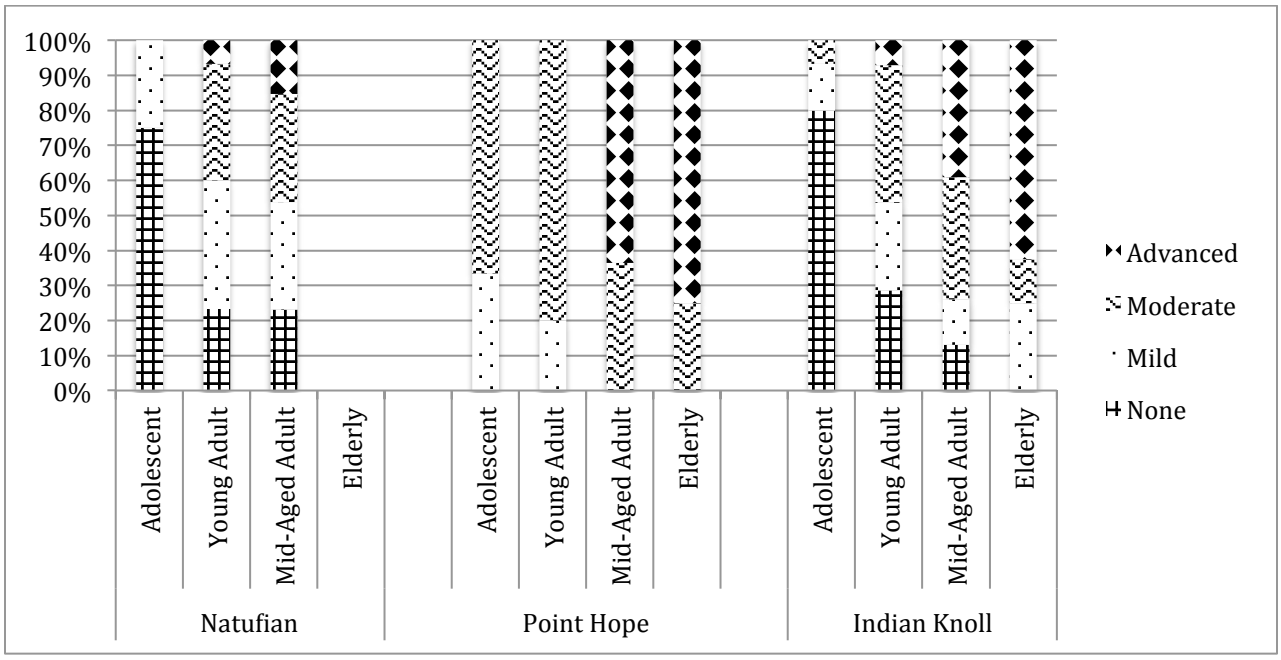


Figure 5.14: Comparative sample periodontal disease severity percentages (using most severe septa diagnosis) by age category

Discussion:

The summary of the major findings are as follows: 1) CEJ-AC distances and alveolar septa scores are moderately positively monotonically correlated (see Appendix 3); 2) alveolar bone loss is greater in the mandible than in the maxilla for anterior teeth, but comparable for the posterior teeth; 3) average periodontal score, most severe septa diagnosis, average CEJ-AC distance per individual and average CEJ-AC distance per tooth all demonstrate that Neandertals had more advanced cases of periodontal disease and more general alveolar bone loss than modern humans; 4) within modern humans without Neandertals, the periodontal disease decreases slightly over time using most severe diagnosis (Kruskal Wallis, p-value: 0.0248), but not does not statistically change using average CEJ-AC distance per individual (Kruskal-Wallis, p-value: 0.123); 5) regions do not differ statistically significantly, but perhaps it is biologically

significant that the Continental sample is more severely affected by periodontal disease; 6) periodontal disease increases in prevalence and severity across age categories by most severe septa diagnosis, average CEJ-AC distance per individual, and average CEJ-AC distance per tooth for Late Pleistocene and Holocene samples; 7) Point Hope has the most severe periodontal disease, but also the oldest age distribution; 9) the Natufians have the least severe periodontal disease, but also the youngest age distribution; 10) Indian Knoll is intermediate in age distribution and periodontal disease prevalence and severity between the three comparative sample. Overall periodontal disease was quite common in the Pleistocene, and most of the differences between groups relate to severity and not presence/ absence of the disease.

Previous research:

Clarke and co-authors claimed that periodontal disease in "ancient" skeletal samples is over-assessed (Clarke et al., 1986; Clarke, 1990), but their values for hunter-gatherers are likely extreme underestimations. They report that 76 - 99.9% of pre-modern humans have no periodontal disease (Clarke et al., 1986); however, as Lavigne and Molto (1995) pointed out, they also reported that their samples have large amounts of dental calculus, which means they were unlikely to be periodontal disease free (others also question Clarke et al's (1986) conclusions (e.g., Oztunc et al., 2006)). Overall, only 18.7% of the sample here shows no evidence of periodontal disease (23.6% when using average CEJ-AC distance). Table 5.5 compares the values found here with other published examples.

	This Study	Other Studies*
Neandertal	83.7% / 86.0%	-
MPMH	80.0% / 70.0%	-
EUP	77.8% / 69.4%	42.9% (Holt & Formicola, 2008)
LUP	82.3% / 73.5%	12% (Holt & Formicola, 2008)
Mesolithic	-	81.5% (Wittwer-Backofen & Tomo, 2008)
Natufians	72.30%	36.4% (Eshed et al., 2006)
Point Hope (Ipiutak)	100%	96.9%[1] (Costa, 1982)
Indian Knoll	68.9%	[2](Leigh, 1925)

Table 5.5: Comparison between the periodontal disease presence values presented here (most severe septa diagnosis/ average CEJ-AC) and previous publications. [1]Methods for diagnosis vary considerably. [t]Calculated from values available in the publication. [2]Periodontal disease is described, but no values are given

Clarke's main point that many diagnoses of periodontal disease in skeletal remains are actually large CEJ-AC distances caused by pulpal infections is a valid one. Lesions of a pulpal origin do not have to be periapical, but can be anywhere along the alveolus or alveolar crest (Goldman and Schilder, 1988). Septa conditions were recorded here to correct for continuous eruption, but they cannot correct for infections. The inflammatory response from a pulpal lesion can also occur in conjunction with periodontal disease (Seltzer et al., 1963; Bender and Seltzer, 1972). Clarke's (1990: et al., 1986) supposition that periodontal disease is not an ancient disease seems unreasonable given the results presented here, and the fact that his periodontal disease prevalences for modern populations are also considerably lower than those presented in dentistry texts (i.e., between 30-60% of people in many extant, Westernized groups (Löe et al., 1992; Oliver et al., 1998; Hugoson et al., 2008)) Clarke (1990) also interpreted Costa's (1982) data as proving his point that periodontal disease was less severe in the past than it is today, but Costa's data show everyone over age 35 having periodontal disease.

Other studies have found similarly high periodontal disease prevalences, such as at Assos, Turkey (4th century BC) where 62% of individuals over 15 and 85% of individuals over 30 had periodontal disease (Oztunc et al., 2006). Unfortunately few anthropological assessments study periodontal disease directly, and fewer report actual disease prevalence because they use an idiosyncratic metric [e.g., Topić et al. (2012) reports a ratio of the averaged buccal CEJ-AC distances per individual to the averaged interdental CEJ-AC distance; Fujita (2012) reports caries and antemortem tooth loss suggesting that where caries are low, tooth loss is caused by periodontal disease; Frayer (1989) reports antemortem tooth loss and abscesses as periodontal disease]; or the sample size is too small (e.g., Lavigne and Molto, 1995).

Time Period:

It has been assumed that periodontal disease has generally gotten worse over time until the introduction of oral hygiene techniques (Gold, 1985); however, this survey rejects this hypothesis. Periodontal disease is not only ancient (Fujita, 2012), but it appears to have actually improved over time in the Late Pleistocene and into the Holocene (Wittwer-Backofen and Tomo, 2009). Periodontal disease has a complex etiology beyond diet, and it does not likely pattern with agricultural intensification in the Holocene as caries does (Larsen, 1995). The more advanced cases of periodontal disease in Neandertals versus early modern humans does have some implications for differential morbidity, especially around the Upper Paleolithic transition. What was going on in Neandertal populations—diet, health, or stress-wise—that was producing more advanced cases of periodontal disease as compared with the early modern humans that came after? Even though a definitive answer cannot be found, there are some serious health and demographic consequences of these periodontal disease severity differences. The Neandertals appear to be less "healthy" than the modern humans who followed them. Other research has

supported this with dental enamel hypoplasias (Ogilvie et al., 1989; Brennan, 1991; Skinner, 1996; Guatelli-Steinberg et al., 2004), Harris lines (Brennan, 1991), fluctuating dental asymmetry (Barrett et al., 2012; Willman, 2014) and other aggregated stress/health indicators (Brennan, 1991; Holt and Formicola, 2008; Trinkaus, 2013). Therefore this result is not be surprising.

The Early Upper Paleolithic sample not being healthier than the Late Upper Paleolithic sample is contrary to some existing stress research. The above studies looking at health changes over time found the Late Upper Paleolithic showed a small inflection where health generally declined relative to the Early Upper Paleolithic (Brennan, 1991; Holt and Formicola, 2008). Though Holt and Formicola (2008) reported periodontal disease decreasing from 42.9% of the EUP to 12% of the LUP based on published descriptions (Table 5.5), this runs counter to their other examples. The climate became colder and preferable environments contracted around the Last Glacial Maximum (Dennel, 1983; Gamble, 1986; Straus, 1995); this likely resulted in increasing stress on the Late Upper Paleolithic population. However, this is not found here, reflected in periodontal disease based on most severe diagnosis, but perhaps with average CEJ-AC distance (it is statistically non-significant). Were individuals practicing better oral hygiene in the Late Upper Paleolithic? There is plenty of evidence of tooth pick grooves from the whole of the Pleistocene (Formicola, 1988), but do they increase in the Late Upper Paleolithic relative to the Early Upper Paleolithic? No data is currently available. It is certainly an old habit (Hlusko, 2003; Lozano et al., 2013), but it may not represent the use of effective oral hygiene. Increased dietary breadth is also associated with improved oral health in modern humans (Lopez et al., 2011), but Late Upper Paleolithic people were not necessarily exploiting a larger breadth of food items, just intensifying that exploitation (Grayson and Delpech, 2002; 2006; Drucker and

Bocherens, 2004; Stiner and Kuhn, 2006). Oral biodiversity changed over time with cariogenic species dominating in the last few centuries (Adler et al., 2013), but this has not been explored systematically at the time depth of the Late Pleistocene.

The Osteological Paradox should also be weighed in the interpretation of these results (Wood et al., 1992). Perhaps the slight decrease in periodontal disease in the Late Upper Paleolithic coupled with increasing demographic stress represents an increase in periodontal disease-related, infection-related, or systemic disease-related mortality. There is not a statistical age profile difference between the Early Upper Paleolithic and Late Upper Paleolithic (see Figure 5.1 (Kruskal-Wallis, p-value: 0.564)), but perhaps those with moderate periodontal disease are dying before it can advance in severity in the Late Upper Paleolithic?

Region:

Though it is not statistically significant, there appears to be a mild regional pattern with more severe cases of periodontal disease in Continental Europe relative to the Atlantic and Mediterranean regions. This could reflect dietary differences (discussed below), a latitudinal effect, or some other health or climate-related difference. A latitudinal effect is likely to also be related to diet as it may be with caries. The Continental individuals all lived in cold environments without Maritime climatic amelioration, which would likely place stress on the population; the Point Hope sample from Alaska (cold, but Maritime) also shows high periodontal disease prevalence and severity. Is the increased periodontal disease the result of cold stress or other climate-related variables? From Figure 5.15, there does not appear to be a clear pattern with latitude. If anything, there is an east-west cline with the more advanced cases in the east. The cluster of moderate and severe periodontal disease in the Czech Republic sites is driving this

difference, so perhaps there is a localized source of health problems that does not reflect latitude *per se* or the wider Continental region.

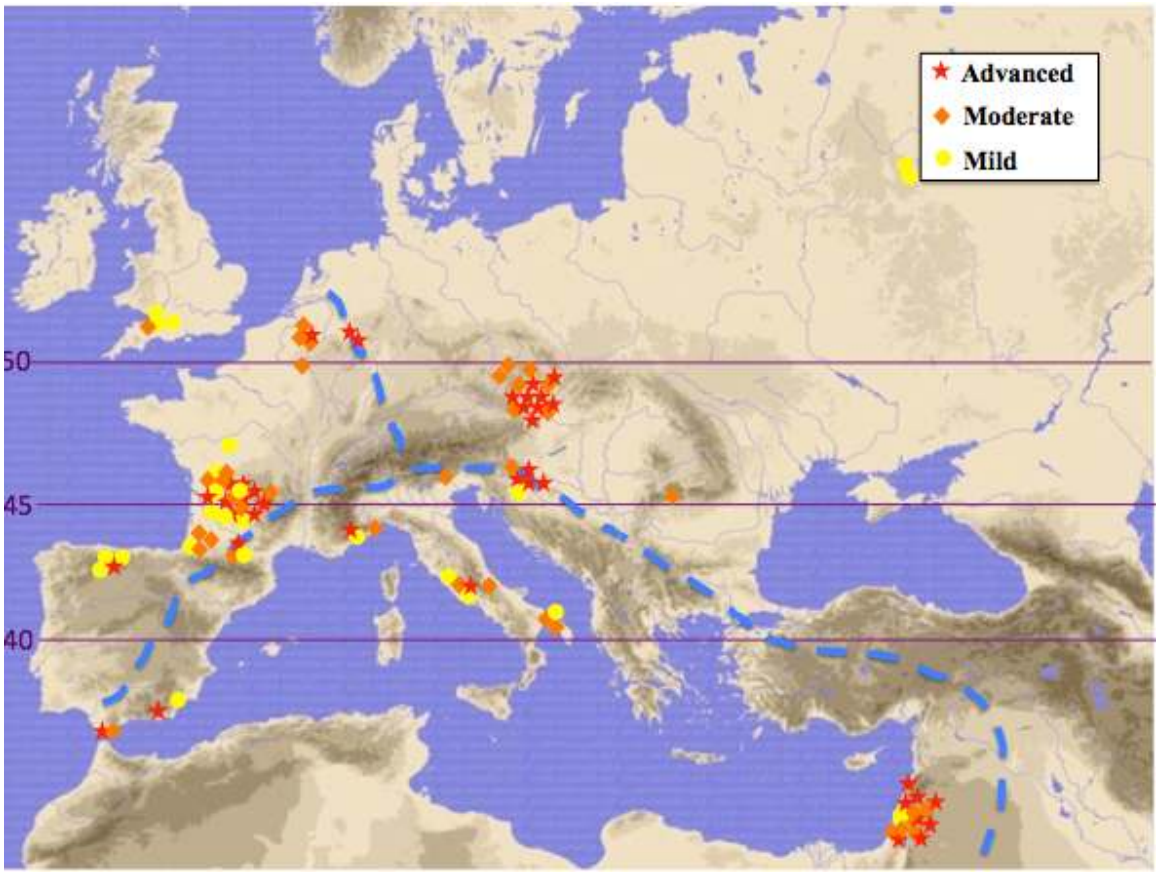


Figure 5.15: Distribution of mild, moderate, and severe periodontal disease with respect to latitude (locations are approximate, especially where there are a number of individuals from one site)

The Mediterranean region has a higher percentage of individuals without periodontal disease than the other two groups, but also more advanced cases than the Atlantic region. This bimodal pattern is difficult to explain. Perhaps if individuals do develop periodontal disease, it progresses to an advanced case more rapidly than in the Atlantic region? In chapter seven, the covariance of pathologies will be tested, as caries are also most common in the Mediterranean.

The Middle Paleolithic modern humans of Southwest Asia do not have any elderly individuals, and yet 80% of the individuals have at least mild periodontal disease somewhere in their dental arcade (Fig 5.3). Since periodontal disease prevalence and severity increases with age, this is a high rate of periodontal disease for a sample without elderly individuals. Since there is also plenty of caries (by Pleistocene standards) in the Middle Paleolithic modern human samples, this may suggest a larger poor oral health trend. Other authors have also noted periodontal disease from other Southwest Asian fossil sites (Kebara (Tillier et al., 1989; 1995; Vandermeersch et al., 1994); Qafzeh (Tillier et al., 2004)).

Age:

Periodontal disease increasing in prevalence and severity through the aging process appears to be a common trend in other archaeological studies (e.g., Loë et al., 1986; Neely et al., 2001; Ronderos et al., 2001; Oztunc et al., 2006); however modern studies only confirm this in samples of populations that do not have access to oral hygiene (Ånerud et al., 1979; Eke et al., 2012). In samples from developed countries, disease prevalence and severity percentages appears to be consistent through age classes (Ånerud et al., 1979). Costa (1982) found an increase in those over the age of 35 relative to those below 35, but with his more precise aging of specimens, he did not find a consistent increase with age.

Life expectancy certainly plays some role here, but in which direction? The poor oral health of Jomon peoples has been attributed to the overall rapid physiological aging relative to modern populations that the Jomon experienced because of bodily stresses (Fujita, 2012). But if oral pathologies such as periodontal disease increase with age and people are dying young, should one not expect them to have less oral pathology (Osteological paradox: Wood et al., 1992)? Low antemortem tooth loss in the Assos sample despite high periodontal disease

prevalence was attributed to short life expectancy, i.e., periodontal disease did not have time to progress to tooth loss (Oztunc et al., 2006). Life expectancy was certainly low in the Late Pleistocene, related to population instability and the demands of their mobile lifestyle (Trinkaus, 2011). Low life expectancy coupled with high periodontal disease suggests rather high morbidity then. Life expectancy did not improve in the Early Upper Paleolithic relative to the Middle Paleolithic, so the mild alleviation in periodontal disease severity may reflect a small decrease in population stress and morbidity, but not enough to lengthen life expectancy. Trinkaus (2011) used a young versus old (over ~40 years old) dichotomy for his mortality analysis and doing something similar here, the young groups over time differ little from one another (Fisher (small sample sizes), A) p-value: 0.7997; B) p-value: 0.116) (Fig. 5.16). But the older groups (pooled Mid-Aged and Elderly) insignificantly appear to show less periodontal disease presence and severity through time (A) p-value: 0.370; B) 0.209). Though it is statistically insignificant (there are a number of zero cells), it may be biologically meaningful. This suggests that perhaps the improvement in health in the Upper Paleolithic is focused on older individuals. The morbidity of Late Upper Paleolithic peoples at older ages may be less than comparably aged Neandertals, even if mortality risk is the same. This is an avenue that should be explored further for other stress indicators.

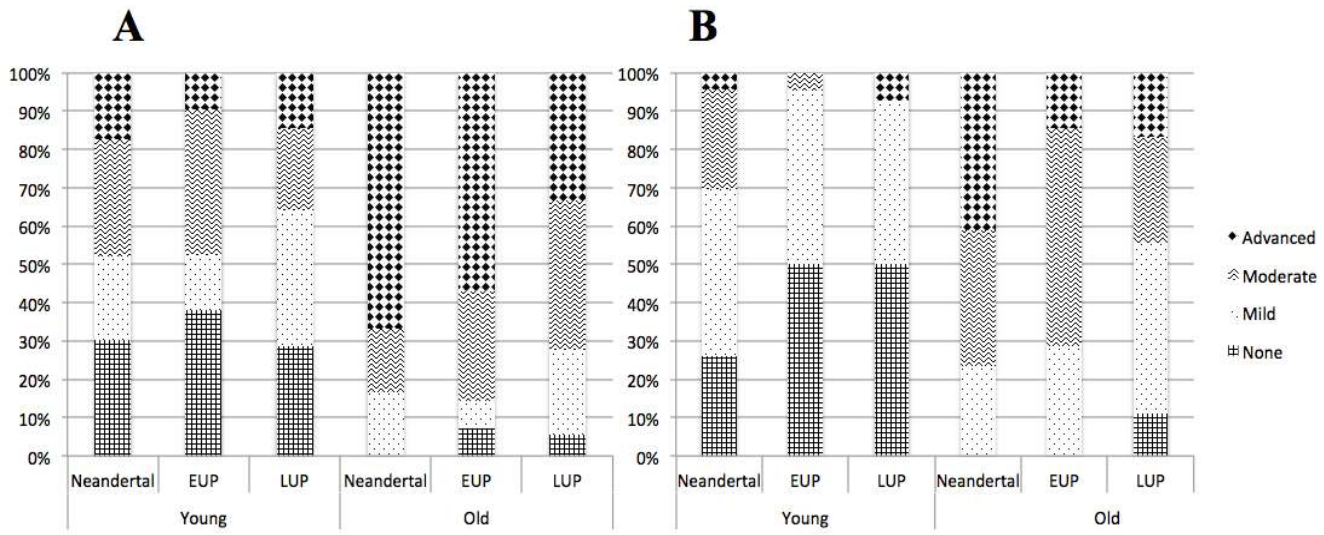


Figure 5.16: Young vs. Old periodontal disease severity (A.) Most severe; B.) Average CEJ-AC) for the Late Pleistocene

This also brings up the question: Do humans become more susceptible to periodontal disease with age, or does the increase with age reflect increasing exposure to sources of inflammation (Oztunc et al., 2006)? Ånerud et al. (1979) found Sri Lankan laborers with no oral hygiene showed a small increase in periodontal disease with age, but even the youngest group (around 17 years old) had extensive dental calculus. Does this suggest that the sources of inflammation were always there, and the individuals were becoming more susceptible to their effects over time? Unfortunately dental calculus is rather fragile and is likely missing from many specimens, so this hypothesis cannot be tested with this sample. The problem of calculus underestimation has affected other studies as well (e.g., Costa, 1982; Oztunc et al., 2006). Chronic periodontal disease can progress slowly or in bursts of activity (Molnar and Hildebolt, 1991), and the increase with age may be a general characteristic of the disease.

Pleistocene vs. Holocene:

The Natufians maintain the Pleistocene trajectory and have a lower mean periodontal disease value than the Late Upper Paleolithic (Table 5.5), but the other comparative samples do not show lessened periodontal disease with time. The Indian Knoll average is below any of the Pleistocene values and the Point Hope value is larger than any of the Pleistocene values. The Point Hope population is skewed older though, and perhaps a larger sample would look more like the Neandertals. This is not an unlikely hypothesis given their diet, cold environment, and strenuous lifestyle. There is no definitive Holocene hunter-gatherer pattern here though; they are quite divergent from one another. Therefore it appears that into the Holocene, differences in diet and health increase between groups. To examine temporal trends in the Holocene, one needs to look regionally or even locally. Temporal patterns no longer hold at a Continent-wide scale.

Diet:

What an individual eats affects his/her oral cavity in two ways: the diet directly interacts with the dentition and oral flora; and the nutrition derived from their diet has systemic affects on both dental development and lifelong alveolar and mucosal health. Therefore nutrition has a generally constructive influence on teeth, while diet is destructive (through wear and acidity) (König, 2000). But the relationship between diet and other oral soft tissues is less direct than it is with dental tissues, with diet modifying oral tissues through nutrition (Schifferle, 2009).

Costa (1982) hypothesized that generalized mild periodontal disease with localized severe periodontal disease reflects a high protein/fat diet. The Point Hope Ipiutak peoples ate a diet dominated by caribou, fish and seals (Rainey, 1941; 1971; Larsen and Rainey, 1948), and the Late Pleistocene diet was also likely high in animal proteins and fats. Dental calculus is deposited during alkaline periods in the oral cavity, which is caused by protein consumption—as

opposed to acidic oral conditions caused by carbohydrate consumption (Hillson, 1979; Meiklejohn et al., 1988). The presence of dental calculus was recorded with this analysis, but it is a gross underestimation due to poor preservation. Isotope analyses of Pleistocene fossils also confirm that the diet was dominated by protein from vertebrates (Richards et al., 2008; Richards and Trinkaus, 2009). This could suggest similar dietary etiology for the high periodontal disease prevalence of Point Hope and Late Pleistocene peoples, namely high protein consumption and dental calculus.

It has been assumed that dietary carbohydrates were low in the diets of Late Pleistocene peoples (Cordain et al., 2005), but the previous caries chapter as well as new archaeological and calculus data have called that into question. In modern humans, dietary carbohydrates can have an inflammatory effect on oral soft tissues within a matter of weeks, but the systemic effects will not be felt for years (Hujoel, 2009). This is why epidemiologists have argued for periodontal disease and caries as a so-called "warning bell" for heart disease, diabetes, and other systemic diseases to come if dietary interventions are not taken. The high periodontal disease prevalence in the Late Pleistocene, taken with the caries data, could be used to argue for greater carbohydrate consumption than has been reconstructed. The relationship between the amount of carbohydrates consumed and the severity of caries and periodontal disease is not direct (König, 2000), so I would not argue Neandertals were eating more carbohydrates than Early Upper Paleolithic modern humans because of greater advanced periodontal disease prevalence. It should be weighed with other data. Hujoel (2009) hypothesized that high caries and periodontal disease indicated carbohydrate consumption from a young age, and lower caries and high periodontal disease would be associated with carbohydrate consumption in adults only. If this

prediction is correct, it could be used to argue for changing differential food access for children in the Late Pleistocene.

A number of other dietary components have relationships with periodontal disease that could have implications for the Pleistocene though. Omega 3 fatty acids are anti-inflammatory and are found to ameliorate the effects of periodontal disease (Kesavalu et al., 2006; 2007). These are found in fish oils, and there is evidence for the consumption of both salt water and fresh water fish in different Upper Paleolithic modern humans from isotope studies (Richards et al., 2001). This may also contribute to why Mediterranean and Atlantic individuals generally had less severe periodontal disease than the land-locked Continental sample. Conversely fish consumption is also sometimes associated with a lot of dietary grit and attrition, which can accelerate dental wear, pulpal exposure, and potentially periodontal inflammation (Oztunc et al., 2006). The Point Hope sample was likely eating large amounts of fish, at least seasonally, but everyone over the age of 35 had at least mild periodontal disease (Costa, 1982).

Low blood serum levels of vitamin D are linked to periodontal disease, which has implications for Late Pleistocene peoples living at more northerly latitudes. Humans produce vitamin D with exposure to UV radiation, which can be difficult to come by in the north due to both the oblique angle with which the sun's rays hit the planet at higher latitudes, and in cold areas, people cover their skin with clothing. Individuals living beyond 50° latitude have the strongest selection for vitamin D_3 synthesis (Jablonski and Chaplin, 2000). Vitamin D is also generally anti-inflammatory, anti-bacterial, and anti-vital (Wintergerst et al., 2007; Garcia et al., 2011). Finding high prevalences of periodontal disease in Pleistocene peoples living at northerly clines therefore possibly suggests they may not have been getting the optimal amount of vitamin D. Periodontal disease is mapped with respect to latitude in Fig. 5.13, but there is no clear

pattern. There is only one known example of possible rickets in the Late Pleistocene, Arene Candide (Formicola, 1995)—but possibly Sunghir 3 (Trinkaus et al., 2014; but see Ortner, 2003)—suggesting severe hypovitaminosis D was uncommon, but does not rule out insufficient levels of circulating Vitamin D.

The inflammation caused by obesity is also associated with periodontal disease (Schifferle, 2009). There is evidence that at least some people may have been seasonally obese in the Upper Paleolithic based on the anatomical correctness of obese Venus figures (Trinkaus, 2005). Many of the individuals from the Czech Early Upper Paleolithic exhibit moderate to severe periodontal disease, and there are a number of obese Venus figurines known from the Continental region (e.g., Willendorf, Dolní Věstonice, Kostenki, Moravany, Gagarino), but they also appear across Eurasia (Svoboda, 2008). Perhaps the cluster of caries in this subgroup along with the advanced periodontal disease and corpulence of the local Venus figures could be used to argue for seasonal excess in foods, namely carbohydrates. Further, a recent genomic study found alleles of Neandertal origin in recent human associated with type 2 diabetes, a disease also associated with obesity and periodontal disease (Sankararaman et al., 2014).

Periodontal disease was quite common in the Late Pleistocene, even for adolescents. Once they reached mid-life and beyond, moderate to severe periodontal disease becomes ubiquitous. Frayer (1989) suggested that the high amounts of "alveolar disease" in the Upper Paleolithic meant many individuals were not at optimal masticatory efficiency as well as needing some social care or special dietary/culinary techniques well in advance of death. The high prevalence of periodontal disease suggests though that a "special" diet was not required. Either this was common enough to affect the cultural preparation of food or people literally "grinned and bore it".

Smoke Inhalation:

A future direction to explore is the connection between smoke inhalation and periodontal disease in fossil humans. The association between cigarette smoking and periodontal disease is well established in recent humans (e.g., Bergström, 1989; 2004; Albandar et al., 2000; Kinane and Chestnutt, 2000). Late Pleistocene humans often inhabited caves, and it is possible they were regularly subjected to smoky environments. Analysis of dental calculus from El Sidron suggests evidence of wood smoke inhalation or the consumption of smoked foods (Hardy et al., 2012). Barrel-shaped chests, ubiquitous in Neandertals (Smith FH, 1976; Franciscus and Churchill, 2002), are a symptom of emphysema in recent humans (Pierce and Ebert, 1958). The inhabitants of Point Hope constructed small homes with central open fires (Rainey, 1941; Daifuku, 1952) and were therefore also likely subjected to frequent smoke inhalation. Other researchers have suspected smoke inhalation as a factor in Late Pleistocene life (Platek, 2002; Størmer and Mysterud, 2007), and though this theory needs more support, the periodontal disease data presented here could contribute to it.

Overall Health:

There are a number of systemic diseases associated with periodontal disease; however, periodontal disease is too common to be used as a diagnostic tool for any one specific disease. Periodontal disease can be weighted with other evidence to make hypotheses about Late Pleistocene health and morbidity though. Periodontal disease is associated with cardiac disease, likely related to systemic inflammation (Slavkin and Baum, 2000; Meurman et al., 2004). Pleistocene peoples were certainly eating a large amount of dietary proteins and fats, but would that result in higher blood cholesterol and cardiac disease in an active population? A high cholesterol diet is associated with periodontal disease in modern peoples (Schifferle, 2009). In a

population with low life expectancy, there may be little selection against diseases like heart disease that do not have an effect until later in life. But with more than 80% of the Late Pleistocene sample having mild periodontal disease to advanced periodontitis, there was certainly increased morbidity in the samples. At least for those individuals with advanced alveolar bone loss and assumed soft tissue inflammation, there would have been health implications, though exactly what those were is unclear.

Conclusion:

All Late Pleistocene subgroups showed relatively high prevalence of periodontal disease and severity increased with age, with the Neandertals showing the most advanced cases. This suggests relatively high morbidity for these groups, but early modern humans in general show a moderate improvement in periodontal disease prevalence and severity relative to Neandertals. This decrease in morbidity did not result in increased life expectancy for Upper Paleolithic modern humans, but perhaps was enough to give them a small demographic advantage over the Neandertals in the Upper Paleolithic transition.

Chapter 6: Antemortem Tooth Loss

Introduction:

Antemortem tooth loss has not been systemically analyzed for the Pleistocene. The few individuals who lost many teeth before death (e.g., Late Pleistocene La Chapelle-aux-Saints 1 and Guattari 1&2, Middle Pleistocene Aubesier 11, Early Pleistocene Dmanisi D3444/D3900) have been debated in the context of societal/conspecific care, with much disagreement (Rowlett and Schneider, 1974; Tappan, 1985; Lebel et al., 2001; DeGusta, 2002; 2003; Lebel and Trinkaus, 2002a; Lordkipanidze et al., 2005; Hublin, 2009; Spikins et al., 2010). However focusing the discussion on such extreme conditions, though severe for those living with them, does not adequately characterize the broader prevalence of tooth loss for Pleistocene individuals. It is highly unlikely that the only individuals experiencing tooth loss were missing a third or more of their teeth. There must be intermediate conditions, but this has not been explored for early modern humans. The only survey of Neandertals analyzed their antemortem tooth loss in the context of chimpanzees and recent humans and with a sample size of 26 individuals (Gilmore, 2011; n.d.). Gilmore's conclusion is that Neandertals are closer to chimpanzees in their antemortem tooth loss prevalence than recent modern human groups. But to understand the Upper Paleolithic transition, Neandertals need to be compared with those who came directly after them: Upper Paleolithic modern humans.

Antemortem tooth loss is reported as the number of teeth missing before death over the total number of alveoli observed. Postmortem loss of teeth is quite common, but it has no meaning beyond a study of taphonomic processes. Identifying antemortem loss is not always straightforward, as some alveoli show either some in-filling (osteoblastic activity), but the alveolus is not completely healed (and therefore it is possible a small portion of the tooth root is

still present); or there was an infection and the alveolus is obliterated (osteoclastic activity), but the tooth may have been held in by soft tissue. So though tooth loss is a binary state, the interpretation of the alveolar bone's condition must acknowledge gradations (Gilmore, 2013). To address this issue, definitive examples of antemortem tooth loss are reported followed by a number in parentheses, which indicates the pooled value of definitive and probable cases of antemortem tooth loss. This can also be interpreted as the first value is a conservative estimate and the second value a more liberal one. Similar methods have been employed elsewhere (Gilmore, 2013). The number of individuals with at least one tooth missing antemortem is also reported, following the above method of a conservative value, followed by the pooled definitive and probable cases. This value has less meaning in that specimens can be represented by between one and 32 alveoli, and one missing tooth and an edentulous individual are considered the same—which they are not.

Sometimes it can be difficult in older individuals to determine whether a tooth was lost long ago, or if it was never present. Dental agenesis, or the failure of a dental bud to form or progress to an adult tooth, is common for the third molar in recent humans, but can happen to any tooth with fourth premolars and maxillary lateral incisors being most common after third molars (Polder et al., 2004; Scott and Turner, 1997). Third molar agenesis is assumed to be much less common in Late Pleistocene individuals, but this has not been quantified. I include all observed instances of dental agenesis in this chapter as they can be confused with tooth loss, especially for third molars in older individuals. One individual, Malarnaud 1, is an adolescent Neandertal with bilateral second lower incisor agenesis, but all of the other Late Pleistocene examples are of third molar agenesis. There are three cases of unilateral lateral incisor agenesis from Indian Knoll in addition to third molar agenesis.

Intentional removal is also difficult to differentiate from "natural" tooth loss and is known from many societies, especially hunter-gatherer groups (e.g., Cook, 1981; Kangxin and Nakahashi, 1996; Scott and Turner, 1997; Lukacs, 2007 Humphrey and Bocaege, 2008; Bocquentin, 2011; Temple et al., 2011). However definitive examples of dental ablation have not been presented for the Late Pleistocene, with some Epi-Paleolithic exceptions (i.e., Humphrey and Bocaege, 2008; Bocquentin, 2011). All examples seen here were assumed to be natural tooth loss and not examples of dental ablation.

Results:

Time period:

	Neandertal	MPMHs	EUP	LUP	Total
No. of teeth missing AM	34 [40]	1 [2]	4 [17]	38 [43]	76 [101]
No. of individuals with AMTL	4 [5]	1	3 [11]	6 [9]	13 [25]
Total Alveoli	689	184	750	531	2070
% of alveoli with some evidence of AMTL	4.9% [5.8%]	0.5% [1.1%]	0.5% [2.4%]	7.2% [8.1%]	3.7% [4.9%]
% of individuals with any AMTL	7.4% [9.3%]	9.1%	7.3% [26.8%]	16.7% [25.0%]	9.4% [18.0%]
No. of agenetic teeth	2	0	2	11	15
No. of individuals with agenesis	1	0	2	8	11
% of individuals with at least one agenetic tooth	1.9%	0%	4.9%	22.2%	7.9%

Table 6.1: Distribution of antemortem tooth loss and agenesis across temporal/taxonomic groups; values in parentheses are definitive plus probable cases (AMTL per alveolus: p-value: <<0.001)

Neandertals show considerably more antemortem tooth loss than Middle Paleolithic and Early Upper Paleolithic modern humans, who are roughly equivalent for definitive examples per-alveolus (Table 6.1) (Definitive and definitive plus probable cases: Chi-Square, p-value:

<<0.001). This result has some major health and behavioral/cultural implications discussed below. The Middle Paleolithic modern humans have no elderly individuals, which likely makes antemortem tooth loss appear more rare. In the age category section below, antemortem tooth loss is considerably more prevalent in the elderly category over the younger three. Tooth loss is cumulative over one's life, so this is not surprising and has been reported elsewhere (Müller et al., 2007). The Early Upper Paleolithic specimens have a similar age distribution to the Neandertals though (see Chapter 5: Periodontal disease), so the difference in antemortem tooth loss prevalence is large and striking. Neandertals have statistically significantly more tooth loss per-alveolus, and this suggests both behavioral and health causal differences producing more tooth loss in Neandertals as opposed to Early Upper Paleolithic modern humans, as well as a greater need in Neandertal groups to care for those who had lost some of their teeth. Neandertals also had more severe periodontal disease than Upper Paleolithic modern humans, so the covariance of these pathologies is explored in the following chapter. The Late Upper Paleolithic peoples have more tooth loss per-alveolus than any of the preceding groups (7.2% [8.1%]). For modern humans, there is stark difference between the Late Upper Paleolithic and those that came before, which may suggest a relaxing of mortality risk associated with tooth loss. The few available published tooth loss values relevant for this study are presented in Table 6.2.

	This Study: indiv.	Frayer, 1989: indiv	This Study: alveolus	Other Studies: alveolus
Neandertals	7.4% [9.3%]	-	4.9% [5.8%]	4.8% (Brabant and Twiesselmann, 1964)
MPMH	9.1%	-	0.5% [1.1%]	-
EUP	7.3% [26.8%]	7.10%	0.5% [2.4%]	-
LUP	16.7% [25%]	17.60%	7.2% [8.1%]	7.8%* (Wells, 1975)
Mesolithic	-	21.50%	-	-
Natufians	16.9% [18.9%]	-	3.1% [3.6%]	3.7% (Eshed et al., 2006)
Point Hope (Ipiutak)	50%	-	16.9% [17.6%]	15% (Costa, 1980a)
Indian Knoll	32%	-	5.40%	-
Hunter-Gatherers	-	-	-	2.0-41.6% (Wells, 1975)

Table 6.2: Comparison of this study's results (tooth loss per-alveolus and per individual percentages) with previous publications; *presented as a value for the "Paleolithic"

The per-individual values tell a much different story than the per-alveolus values, making tooth loss appear much more common in the Early Upper Paleolithic (7.3% [26.8%]) (Table 6.1). In the Early Upper Paleolithic, most individuals with tooth loss are only missing one or two teeth. By comparison in the Neandertals, some individuals are missing half or more of their teeth. For the definitive cases of tooth loss, the temporal pattern generally holds for the per-individual values set by the per-alveolus values, but it is to a much lesser degree. In the Middle Paleolithic modern human sample from this study, only one tooth is missing, but there is an N of 11, inflating the per-individual prevalence to 9.1%.

Region:

	Atlantic	Continental	Mediterranean	Pooled Mediterranean (incl. Natufians)
No. of teeth missing AM	37 [46]	14 [24]	26 [32]	54 [64]
No. of individuals with AMTL	5 [9]	3 [8]	6 [9]	15 [19]
Total Alveoli	799	509	846	1742
% of alveoli with some evidence of AMTL	4.6% [5.6%]	2.8% [4.7%]	3.1% [3.8%]	3.1% [3.7%]
% of individuals with any AMTL	8.3% [15.0%]	11.5% [30.8%]	10.7% [16.1%]	13.8% [17.4%]
No. of agenetic teeth	10	2	3	9
No. of individuals with agenesis	7	2	2	6
% of individuals with at least one agenetic tooth	11.7%	8.0%	3.6%	5.5%

Table 6.3: Distribution of antemortem tooth loss and agenesis across regions of Western Eurasian; values in parentheses are definitive plus probable cases (Definitive per-alveolus AMTL: p-value: 0.374; Definitive plus probable: p-value: 0.422)

There is little difference in antemortem tooth loss prevalence between regions (Table 6.3) (Definitive cases: Chi-square, p-value: 0.374; Definitive plus probable: p-value: 0.422). The age distribution of each region (for specimens with preserved alveolar bone) was tested in the periodontal disease chapter, and found to be not significantly different. This suggests that region and the variables related to it (e.g., environment, climate, diet) play little role in the presentation of tooth loss in Late Pleistocene humans. This also suggests that the temporal and taxonomic differences in antemortem tooth loss are not artifacts of some underlying regional pattern.

Age category:

Antemortem tooth loss is known to increase through the aging process. It is not only cumulative, but the alveolar bone and periodontal ligaments weaken over time, further

accelerated by periodontal disease (Copeland et al., 2004; Müller et al., 2007; but see Papapanou et al., 1991). And once it reaches an advanced degree, processing of food in the mouth becomes difficult. This is fixed with dentures and other implants in modern, urban/industrial societies, but tooth loss has more impact in societies without access to dental care (e.g., Neely et al., 2005). Therefore it is not unusual to see here that elderly individuals have the most tooth loss (Both definitive and definitive plus probable cases: Chi-square, p-value: $<<0.001$). What is surprising is that tooth loss prevalence per-alveolus does not appear to be gradual; it is less than 1% in all age categories and then jumps to 23.6-26.6% in the elderly sample (Table 6.4). Other studies have shown an acceleration in tooth loss rate over time, but not to this degree (Norderyd and Hugoson, 1998). Certainly a few individuals are tipping the scale, namely La Chapelle-aux-Saints 1, Guattari 1 & 2, Oberkassel 1, and those missing the majority of their teeth are almost automatically assumed to be elderly. Wear is accelerated on the few teeth remaining by focusing chewing on a reduced surface area, ensuring they will be categorized as elderly (based on wear alone) (Hillson, 2008). However there is little evidence to assume that these individuals are not older by Late Pleistocene standards, i.e., over approximately 35-40 years of age; their cranial and postcranial remains confirm the age assessment (La Chapelle-aux-Saints 1 (Trinkaus, 1985); Oberkassel 1 (Henke, 1986); Guattari 1&2 (Mallegni, 1995)).

	Adolescent	Young Adult	Mid-Aged Adult	Elderly	Unaged
No. of teeth missing AM	1 [1]	7 [13]	5 [13]	64 [72]	0 [3]
No. of individuals with AMTL	1 [1]	3 [6]	3 [7]	7 [10]	0 [3]
Total Alveoli	237	739	717	267	162
% of alveoli with some evidence of AMTL	0.40%	0.9% [1.8%]	0.7% [1.8%]	21.4% [24.1%]	0% [1.9%]
% of individuals with any AMTL	4.0%	6.5% [13.0%]	7.0% [16.3%]	46.7% [66.7%]	0% [7.3%]
No. of agenetic teeth	2	6	4	3	0
No. of individuals with agenesis	1	5	3	2	0
% of individuals with at least one agenetic tooth	4.0%	10.9%	7.0%	13.3%	0%

Table 6.4: Distribution of antemortem tooth loss and agenesis across age category; values in parentheses are definitive plus probable cases (AMTL per-alveolus: p-value: <<0.001)

Comparative Samples:

	Natufians	Point Hope	Indian Knoll
No. of teeth missing AM	28 [32]	118 [123]	119
No. of individuals with AMTL	9 [10]	12	24
Total Alveoli	896	697	2193
% of alveoli with some evidence of AMTL	3.1% [3.6%]	16.9% [17.6%]	5.40%
% of individuals with any AMTL	16.9% [18.9%]	50%	32%
No. of agenetic teeth	5	12	10
No. of individuals with agenesis	4	7	7
% of individuals with at least one agenetic tooth	7.70%	30.40%	9.30%

Table 6.5: Distribution of antemortem tooth loss and agenesis across comparative samples; values in parentheses are definitive plus probable cases (per-alveolus AMTL: p-value: <<0.001)

The Point Hope comparative sample shows more antemortem tooth loss (16.9% (17.6%)) than the other two samples, Natufians and Indian Knoll (Table 6.5) (Chi-square, p-value:

<<0.001). This may further strengthen the argument for an analogy between Neandertals and Arctic peoples; however, the Neandertal tooth loss prevalence (4.9% (5.8%)), though higher than Middle Paleolithic and Early Upper Paleolithic modern humans, is within the range of other modern groups including the Natufians (3.1% (3.6%)) and Indian Knoll (5.4%). The Point Hope prevalence is especially high, but likely driven in part by high periodontal disease prevalence and an age distribution skewed towards older individuals. Costa (1980a) found high antemortem tooth loss rates for the Point Hope peoples, especially the Ipiutak (15.0%), which are the only sub-group from Point Hope included for this study here. A strong relationship between number of teeth lost and age was also found (Costa, 1980a). The elderly individuals in my study have slightly less tooth loss than the mid-aged adults in the Point Hope sample, and this may reflect mortality risk associated with tooth loss (Table 6.6) (p-value: <<0.001). Those whom made it to the elderly category were slightly healthier and had more teeth than those whom died younger, invoking the Osteological Paradox (Wood et al., 1992). Costa's (1980a) data also showed a small decrease in antemortem tooth loss in the over age 46 group, suggesting that the pattern here is not just a result of small sample size.

Natufian					
	Adolescent	Young Adult	Mid-Aged Adult	Elderly	Unaged
No. of teeth missing AM	0	6 (7)	19 (21)	0	2 (3)
No. of individuals with AMTL	0	3 (4)	4	0	1
Total Alveoli	54	584	243	0	15
% of alveoli with some evidence of AMTL	0%	1.0% (1.2%)	7.8% (8.6%)	0%	13.3% (20.0%)
No. of agenesised teeth	0	6	0	0	0
No. of individuals with agenesis	0	4	0	0	0
% of individuals with at least one agenetic tooth	0%	13.30%	0%	0%	0%

Point Hope				
	Adolescent	Young Adult	Mid-Aged Adult	Elderly
No. of teeth missing AM	0	9	84 (87)	25 (27)
No. of individuals with AMTL	0	1	7	4
Total Alveoli	86	154	329	127
% of alveoli with some evidence of AMTL	0%	5.80%	25.5% (26.4%)	19.7% (21.3%)
No. of agenesised teeth	0	6	5	1
No. of individuals with agenesis	0	3	3	1
% of individuals with at least one agenetic tooth	0%	60%	27.30%	25%

Indian Knoll				
	Adolescent	Young Adult	Mid-Aged Adult	Elderly
No. of teeth missing AM	0	7	60	52
No. of individuals with AMTL	0	4	12	8
Total Alveoli	432	857	628	172
% of alveoli with some evidence of AMTL	0%	0.80%	9.60%	30.20%
No. of agenesised teeth	1	5	3	1
No. of individuals with agenesis	1	4	1	1
% of individuals with at least one agenetic tooth	6.70%	14.30%	13.00%	12.50%

Table 6.6: Distribution of antemortem tooth loss and agenesis across age categories for all three comparative samples; values in parentheses are definitive plus probable cases (All samples per-alveolus AMTL, p-value: <0.002)

The Natufians' lower prevalence in this study (3.1% [3.6%]) is partially the result of the lack of elderly individuals, but this matches prior estimates (3.7% from Eshed et al., 2006). Tooth loss prevalences increase through the first three Natufian age categories (Chi-square, p-value: <0.002). The Indian Knoll sample also shows tooth loss increasing over the aging process, up to 30.2% in the elderly sub-sample (p-value: <<0.001). All the data from this study and that available from the literature (see Table 6.2) suggest that for individuals over approximately the age of 40 in forager societies, the loss of a quarter or more of the teeth is relatively common.

Agenesis:

Third molar agenesis increases over time in *Homo* as faces become more orthognathic and the length of the dental arcade shortens (Wu and Xianglong, 1996). The only two Neandertal agenetic teeth are from Malarnaud, and they are lower lateral incisors. The Neandertals and Middle Paleolithic modern humans show no example of third molar agenesis (Table 3.1). The Early Upper Paleolithic has two individuals (both from the Czech Republic), each missing one third molar, and there are many more examples in the Late Upper Paleolithic (8 individuals or 22.2% of individuals) (Table 6.1). By the Late Holocene, groups range in third molar agenesis rates from 0.2- 36% (Brothwell et al., 1963) and range in the comparative samples here from 7.7-30.4% (Table 6.5), putting the Late Upper Paleolithic modern human value from this study within the range of recent Europeans. This pattern represents a cranial morphological trajectory over time in facial flatness and posterior dental reduction and therefore is not an independent trait. Excluding third molars, agenesis has a much lower prevalence, but it is not rare (3.9-5.5% of individuals in Western populations (Thierry et al., 2007); 2.2-10.1% globally (Polder et al., 2004)). Also there are rare exceptions all the way back to the Early Pleistocene (*Homo erectus* Omo 75-14a (Wallace, 1977)).

Agenesis has been described as being common in some Continental samples (i.e., Dolní Věstonice) suggesting a small, closely genetically related group (Alt et al., 1997; Hillson, 2006); however, agenesis appears to be more common in the Atlantic region than the Continental region (Table 6.3). There are few Late Upper Paleolithic individuals in the Continental sample, and therefore this regional pattern is likely the indirect result of the temporal pattern in agenesis. Agenesis is not expected to be correlated with age (Table 6.4). It occurs during growth and development and persists through the rest of life.

Discussion:

The major findings of this chapter include that: 1) tooth loss was significantly lower in Early Upper Paleolithic modern humans relative to Neandertals and Late Upper Paleolithic peoples; 2) tooth loss did not pattern regionally in the Late Pleistocene; 3) tooth loss increases significantly over the aging process for both Late Pleistocene humans and the comparative samples; and 4) tooth loss differed significantly for the comparative samples with Point Hope having the most tooth loss. These findings have implications for behavior and health in foragers of the Late Pleistocene and Holocene.

Taxonomy & Social Care:

There is one previous study that assessed Neandertal tooth loss (Gilmore, 2011; n.d.). However it's conclusion—that Neandertals were not necessarily practicing modern human behaviors, such as the care of the disabled, because of their antemortem tooth loss prevalence—is called into question with these results. Gilmore (2011) assumes that since her data show Neandertals have a "rate" of tooth loss (3.7% teeth lost per individual) closer to chimpanzees (2.0% teeth lost per individual) (who do not provide special care for those with missing teeth) than recent humans (7.8% teeth lost per individual) (who assumedly do supplement the diet of

those who are missing teeth), then Neandertals *ipso facto* did not provide care or process the food of those with tooth loss. This thesis found that Neandertals had 3.4% [4%] of teeth lost antemortem per individual and pooled Upper Paleolithic modern humans had 2.2% [4.1%], suggesting that taxonomy places little role (Definitive cases: One sample T-test, p-value: 0.213; definitive plus probable cases: One sample T-test, p-value: 0.956). Her work did find the Neandertal tooth loss rate to be intermediate between chimpanzees and modern humans if she assumed a faster development rate in Neandertals than modern humans (Gilmore, n.d.), but that assumption is problematic (i.e., if dental development is faster (Smith et al., 2007), is whole body aging also accelerated?).

Neandertals in this study show more antemortem tooth loss per-alveolus than the contemporary Middle Paleolithic modern humans or the Early Upper Paleolithic humans that follow them, and the modernity of Upper Paleolithic humans is not in question. Therefore if Neandertals have more antemortem tooth loss than the definitively "modern" humans who replaced them, why would one interpret Neandertals to be incapable of or unwilling to care for their disabled? There are even other Neandertal individuals with tooth loss known from the literature that were not included in the sample (e.g., Krapina 59 (Lebel and Trinkaus, 2002a)).

This is also brings into question how far back should the possibility of conspecific care be extended? There are many examples of extensive tooth loss or oral impairment from the Middle Pleistocene (e.g., Broken Hill, Arago 21, Sima de los Huesos 1, Aubesier 11) and Early Pleistocene (Dmanisi D3444/D3900) from around the Old World. This care could include softening and processing food by one individual to provision another, and this has broader implications about social structure, care for the diabled, centralized food sharing, and

communication (both communicating pain and need for help, as well as the social need to keep older individuals around who can communicate their knowledge) (Tilley, 2012).

Many examples of congenital abnormalities and trauma have been used to argue for a great antiquity in conspecific care (Sima de los Huesos 14, Shanidar 1, Qafzeh 12), but extensive antemortem tooth loss has been the most discussed (Degusta, 2002; 2003; Hublin, 2009; Lebel and Trinkaus, 2002a; Lebel et al., 2001). The debate is polarized with those suggesting tooth loss requires no special treatment (Dettwyler, 1991), pointing to evidence of its presence in non-human primates (Tappan, 1985; Degusta, 2002; 2003; see Hublin, 2009). But chimpanzees do provision one another, and yet there are few wild examples of chimpanzees living long while missing a third or more of their teeth (though chimpanzees have shorter life expectancies than later *Homo*) (Lovell, 1990; Lebel and Trinkaus, 2002a).

This debate has major implications for behavior and cognition, but it has ignored the basic pathological/biological question: how prevalent is tooth loss in Pleistocene humans? As presented in the introduction to this chapter, these papers have only discussed extensive oral impairment and not tooth loss in general. The present study attempts to elucidate this issue around the Upper Paleolithic transition, not the whole Pleistocene; though, the focus in the literature has generally been around Neandertals and early modern humans. An emphasis on advanced tooth loss, ignoring the larger oral health of these individuals, has lead to conjectural narratives for both sides of the debate. In this study, there are prevalences for tooth loss available across Western Eurasia, elucidating the issue of tooth loss more broadly in the Late Pleistocene and providing concrete data for those interested in the bioarchaeology of compassion (e.g., Tilley, 2012).

Time Period:

Perhaps the decrease in antemortem tooth loss prevalence per-alveolus in Early Upper Paleolithic humans relative to Neandertals suggests an increase in tooth loss-related mortality in the Early Upper Paleolithic, as opposed to the interpretation that they were loosing fewer teeth because of differential behavior. Neandertals also showed more severe periodontal disease than Early Upper Paleolithic modern humans, perhaps reflecting the osteological paradox (Wood et al., 1992): Were Neandertals less healthy than Early Upper Paleolithic modern humans because they had more periodontal disease and tooth loss; or were Neandertals healthier because they survived despite periodontal disease and tooth loss? The major inflection in tooth loss prevalence in the Late Upper Paleolithic likely reflects both an evolution in behavior and a relaxing of tooth loss-related mortality risk (e.g., teeth-as-tools, social care, diet). Cultures were becoming more complex and social divisions in labor were arising in the Late Upper Paleolithic (e.g., Villotte et al., 2010). The result may have been both a place in society for elderly individuals with few teeth and other individuals to assist in the preparation of their processed diet. The debate erupts over the assessment of these behaviors outside *Homo sapiens sensu stricto*, specifically relevant for Neandertals in this study.

Relative to recent modern human rates of tooth loss (2.0- 41.6% (Wells, 1975); 3.1-17.6% comparative samples here), the low Early Upper Paleolithic prevalence per-alveolus (0.5% (2.4%)) is actually more surprising than the higher Neandertal prevalence (4.9% (5.8%)). The question may more logically be: why were Early Upper Paleolithic modern humans not losing their teeth? Tooth loss is assumed to be higher in populations eating abrasive diets causing increased dental wear, pulpal exposure, tooth fractures, periapical lesions, and consequent tooth loss (Wells, 1975); however these factors were experienced by all Pleistocene groups. Tooth loss

has been demonstrated to decrease with the transition to agriculture from foraging, with the main cause of antemortem tooth loss shifting from attrition to caries (Anderson, 1968). Gilmore (n.d.) argues that the high variance in antemortem tooth loss rates in modern human groups suggests that modern human behavior reduces tooth loss-related mortality. Does this then mean that the low prevalence of tooth loss in Early Upper Paleolithic humans derives from higher tooth loss-related mortality caused by a lack of mediating (modern) behaviors? Or did they truly have better oral health than Neandertals or Late Upper Paleolithic peoples? Though Early Upper Paleolithic modern humans have less advanced periodontal disease than Neandertals, their oral health is by no means "good" when compared with recent humans (Hildebolt and Molnar, 1991; Oliver et al., 1998; Hugoson et al, 2008). Perhaps Early Upper Paleolithic modern humans were using their teeth less as tools than Neandertals, and this alleviated the physical stress on the dentition that produced pulpal exposure, trauma, and tooth loss (Wallace et al., 1975; Smith, 1983). The low prevalence of tooth loss in Early Upper Paleolithic peoples, whose age distribution is not statistically different from the Neandertals and Late Upper Paleolithic peoples, remains somewhat enigmatic and is likely related in part to some behavioral (less use of teeth-as-tools?) or morbidity difference.

Region:

The lack of difference between regions for antemortem tooth loss prevalence suggests that other variables associated with region are likely not affecting tooth loss. This would include environment, climate, and available diet. If diet is not contributing to tooth loss, only health and behavioral differences remain. This is rather provocative when taken with the temporal pattern suggesting more tooth loss in Neandertals than Early Upper Paleolithic modern humans. A previous study suggested less tooth loss in Southwest Asia relative to Europe for the Middle

Paleolithic (Neandertals versus Middle Paleolithic modern humans), but no regional difference in the Upper Paleolithic (Smith P, 1976), and this study supports that. Though the focus in the literature on antemortem tooth loss has been on the behavioral responses to those already experiencing major tooth loss by other members of their groups, there are likely other behavioral differences that are producing tooth loss. Something about Neandertal health and behavior is either producing more tooth loss than Early Upper Paleolithic modern humans or ameliorating the negative mortality affects of tooth loss, and this is likely independent of climate or climatically-dictated dietary variables. There is no regional pattern in tooth loss identified in here.

Age Category:

Not surprisingly, tooth loss is highest in the Late Pleistocene elderly sample, but the increase with age is not linear. The adolescent prevalence is low with only one antemortem missing tooth, and there is no difference between the young adult and mid-aged adult groups per-alveolus (1.8%). Nearly all the tooth loss occurs in the elderly group, and this pattern is repeated in the comparative samples. Also the entire elderly sample showed at least mild periodontal disease, and 64.3% had advanced periodontal disease. Periodontal disease is a risk factor for tooth loss (Neely et al., 2005 and many others); however, it has been argued that periodontal disease is not causing much tooth loss in Pleistocene humans (Clarke et al., 1986). The covariance of these two pathologies is further tested in the next chapter. Considering that the members of the oldest age category here—based on dental wear—were likely not elderly by modern standards (40-55 years old), this could suggest some oral health-related mortality is occurring, but within the context of larger health decline.

Health and diet:

The causal relationship between dietary variables and tooth loss in recent humans is not well researched (e.g., Eklund and Burt, 1994). What is clear though is that once extensive tooth loss has occurred, there is an effect on the individual's subsequent diet (Joshipura et al., 1996). Hard and tough foods are difficult to process orally, so the individual begins to favor soft foods and fat and carbohydrate intake goes up and fiber goes down (Joshipura et al., 1996). As caries were increasing in the Late Upper Paleolithic relative to earlier periods in Europe, likely from increased carbohydrate consumption, this would also indicate that softer foods were socially available for those with antemortem tooth loss. Even if tooth loss in the Late Upper Paleolithic is not a result of caries, both pathologies point to a dietary pattern where tooth loss was less of a handicap than before.

Having fewer than 20 teeth (out of 28, not including third molars) is directly correlated with increased mortality in recent humans, even when other variables are controlled for, including diabetes, caries, periodontal disease, coronary artery disease, etc. (Padilha et al., 2008). This suggests it is not common risk factors driving this relationship. The relationship between tooth loss and mortality is further supported by other tooth loss research showing a small increase in the number of original teeth per individual in the oldest age category in a cross sectional and longitudinal study in modern Swedes, which generally showed a decrease in number of teeth through each decade (Norderyd and Huguson, 1998). The oldest individuals were likely healthier (i.e., lived longer) and had more teeth than those who had died in earlier decades. The Point Hope sample here, and surveyed by Costa (1980a), showed the same pattern. It seems poor health can cause tooth loss and vice versa, and therefore good oral health as represented by number of teeth present is a good indicator of lowered mortality risk.

Agenesis:

Dental agenesis is not a disease state, but it is included here as: 1) it can be confused with antemortem tooth loss; and 2) it has never been systemically analyzed for the Late Pleistocene. The only available values for third molar agenesis in the Upper Paleolithic are 3.9%, contrasted with 0.6% in the Mesolithic from Brothwell et al. (1963). Values were higher here with 4.9% of individuals in the Early Upper Paleolithic and 22.2% in the Late Upper Paleolithic. As faces become flatter in the hominin lineage, teeth have also become smaller (Frayer, 1978; Brace et al., 1987; Calcagno and Gibson, 1991). But this dental reduction is not always enough to alleviate space issues, and third molar agenesis became much more common in recent groups (Bermúdez De Castro, 1989; Mattheeuws et al., 2004) as has third molar impaction (Gibson and Calcagno, 1993)) There is also an individual-level positive relationship between smaller teeth and third molar agenesis (Brook, 1984). The pattern found here where third molar agenesis increases in frequency though the Late Pleistocene with most of it occurring in the Late Upper Paleolithic confirms what had already been assumed. Agenesis of other teeth is more likely related to genetic and environment interactions than general lack of space (tooth development is a threshold trait) (Pinho et al., 2010), but third molar agenesis is also associated with agenesis of other teeth (Garn et al., 1963). It is a complicated relationship, and there is a large amount of variance in the expression of this trait in modern samples, ranging from nearly none to over a third of individuals in a sample showing agenesis of at least one third molar (Brothwell et al., 1963).

There was only one example from the Late Pleistocene of agenesis of teeth other than the third molars, i.e., the symmetrical lower lateral incisor agenesis of the Malarnaud, Neandertal. Lateral incisor agenesis is rare in the mandible and twice as common in females (Stamatiou and

Symons, 1991), who tend to have smaller faces, at least in modern humans (agenesis overall is 1.37 times more common in recent modern females (Polder et al., 2004)). These trends are also present in the comparative sample data: the Indian Knoll sample had two unilateral cases of upper lateral incisor agenesis and one unilateral case of lower lateral incisor agenesis. Lateral incisor agenesis frequencies in recent groups range from 0.2%-2.1% of individuals (Brothwell et al., 1963).

There are other reported cases of third molar agenesis from the Pleistocene outside Western Eurasia (e.g., Early Pleistocene African Omo 75-14b (Wallace, 1977), Middle Pleistocene Chinese Lantian Mandible (Wu and Zeng, 1996), the Chinese Liujiang maxilla (Liu and Zeng, 1996)), suggesting sporadic cases are a feature of human dentition. It is the high number of cases in the Late Upper Paleolithic continuing into the Holocene that reflects a change in facial morphology and tooth size.

Additional Note on impacted teeth:

Dental malocclusion and impaction were not a primary focus of this study. Dolní Věstonice 3 was described as having asymmetrical lower third molar agenesis along with Dolní Věstonice 16 (Hillson, 2006), perhaps suggesting a familial relationship amongst those buried at Dolní Věstonice (Alt et al., 1997). Radiographs from this study show that Dolní Věstonice 3 actually has a third molar, but because of its bony impaction, it cannot be seen externally (see Fig. 6.1). Impacted third molars are quite common in recent populations (25% of third molars present: Scherstén et al., 1989). In the Late Pleistocene, impacted third molars are already known from Dolní Věstonice 15, Cro-Magnon 4 (Hillson, 2006), and Cap Blanc (Dunsworth, 2007), and now Dolní Věstonice 3, all Upper Paleolithic modern humans. The presence of the retromolar space in Neandertals makes an impacted third molar unlikely, but it is known from Krapina

(Wolpoff, 1979) and also early modern human Zhoukoudian Upper Cave 101 (Liu, 1997) and *Australopithecus* (STS52b and KNM-WT17400 (Gibson and Calcagno, 1993)). It should now be noted that Dolní Věstonice 3 is not a case of third molar agenesis.

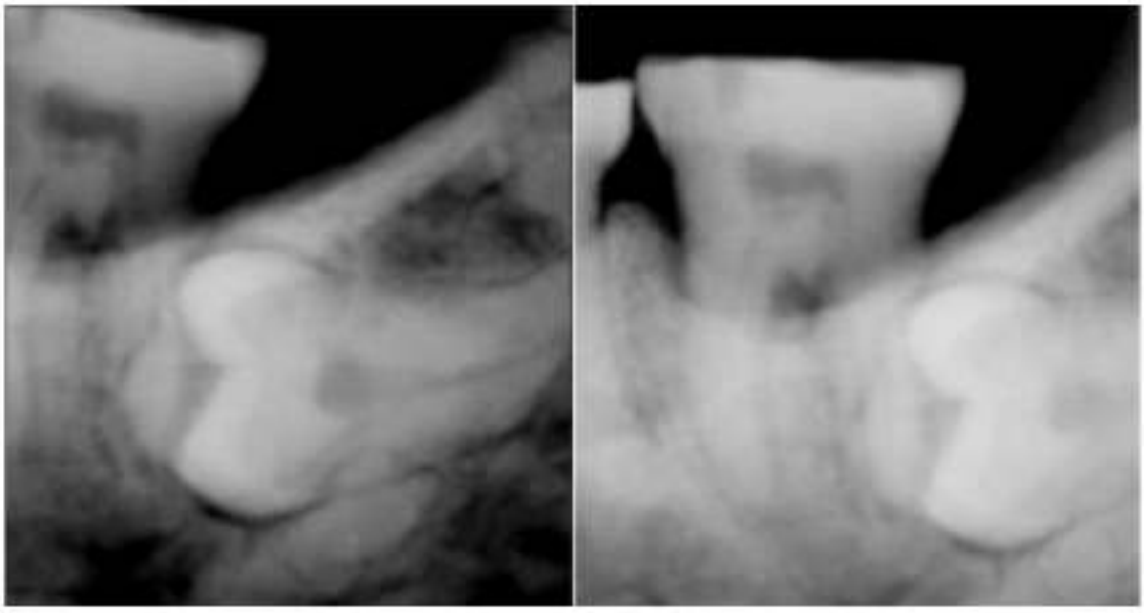


Figure 6.1: Impacted left lower third molar from Dolní Věstonice 3, two views and exposures

Chapter 7: Oral Health & Systemic Health

Introduction:

As caries, periapical lesions, periodontal disease, and antemortem tooth loss are being considered in the aggregate to approximate oral health for this study, it is relevant to understand whether these pathologies co-vary within individuals. This is done using Spearman's rho rank correlation scores (Table 7.1), reported here along with the number of tested pairs and p-values. The results of this inquiry line are presented to contribute to the larger discussion of oral health, systemic health, and morbidity in the Late Pleistocene of Western Eurasia. The results of an overall morbidity score between each temporal group similar to Brennan's (1991) method are also reported. The previous results chapters' conclusions considered with the co-variance data can be used to inform our understanding of the role region and its correlates (namely climate and diet), culture and taxonomy, temporal change, and overall health affect and are affected by oral health. Though previous chapters contain discussion sections relevant for each individual pathological analysis, this chapter attempts to bring these discussions together to test the project hypotheses and understand health more broadly in the context of the Late Pleistocene.

Pathology covariance results:

Late Pleistocene only		Caries	Lesions	Tooth loss: Definitive	Tooth loss: +Probable
Tooth loss	Lesions	0.1436	--		
	Definitive cases	-0.0448	0.1058	--	
	+ Probable cases	-0.0732	**0.3245****	--	--
	Periodontal D.	0.1124	**0.3470****	**0.2249***	**0.3423****
Pooled Samples		Caries	Lesions	Tooth loss: Definitive	Tooth loss: +Probable
Tooth loss	Lesions	**0.3235[1]****	--		
	Definitive cases	-0.0122	**0.3938****	--	
	+ Probable cases	0.0157	**0.5174****	--	--
	Periodontal D.	0.0446	**0.3599****	**0.3319****	**0.4258****

Table 7.1: Table of Spearman's rho values for co-variance of pathologies: Periodontal disease severity scores and % of affected teeth per individual for caries, lesions and antemortem tooth loss (Bolded values are statistically significant with *p-value: 0.01, **p-value: 0.001); [1]this value is being driven by the Indian Knoll sample; it is non-significant when Indian Knoll is removed

Caries and Periapical Lesions:

The majority of Late Pleistocene caries are non-penetrant (34 of 53 examples, or 64.2%); therefore the non-significant and only slightly positive relationship between percent carious teeth and percent lesioned alveoli per individual is expected (124 individuals in the Late Pleistocene preserved both teeth and alveoli; Spearman's rho: 0.14, p-value: 0.11) (Table 7.1). Carious lesions were not progressing far enough to affect the pulp chamber and produce periapical lesions. When the Natufian and Point Hope samples are included, the relationship is still non-significant (N=192, Spearman's rho: 0.12, p-value: 0.09). This suggests that periapical lesions in all of these samples are generally not caused by caries. In agricultural samples and recent skeletal populations, periapical lesions are generally considered to be of carious origin (e.g., Lucas et al., 2010), but this assumption does not appear to be valid for these pre-agricultural groups. The

observed alveolar lesions are more likely to be of dental attrition (Kieser et al., 2001) or periodontal disease origins (see results below). The Early Upper Paleolithic sample had the most lesions (5.0% of alveoli), but the least caries of modern humans (1.9% of teeth), and conversely the Middle Paleolithic modern humans had the most caries of any group (7.7% of teeth), but the least percentage of lesions (1.6% of alveoli). Part of this may be related to age. The Middle Paleolithic modern humans have no elderly individual and lesions increase with age, but caries do not. However the lack of matching patterning between the two pathological conditions over regional and temporal samples or within individuals suggests they are independent variables.

The Indian Knoll sample was not pooled with the other sub-samples, as it did not follow the same pattern. The people of Indian Knoll practiced early garden agriculture and heavily processed gathered items such as acorns (Leigh, 1925). Percent carious teeth and percent of alveoli with periapical lesions were significantly and somewhat positively correlated for the Indian Knoll sample (N=74, Spearman's rho: 0.31; p-value: <0.01). When Indian Knoll is included with the other samples, it overwhelms the pattern and artificially makes the relationship between caries and periapical lesions appear stronger for the whole sample (N=266, Spearman's rho: 0.32; p-value: <<0.01). Grouping was only done for subsequent comparisons when the relationships between pathological conditions were similar for all sub-groups.

Caries and Antemortem Tooth Loss:

In the Late Pleistocene sample, the variables "percent carious teeth" and "percent of alveoli showing evidence of antemortem tooth loss per individual" are independent for the definitive cases (N=124, Spearman's rho: -0.04; p-value: 0.62), and also for the definitive cases plus the probable cases of tooth loss (Spearman's rho: -0.07; p-value: 0.42). When the Natufian and Point Hope samples are included with the Late Pleistocene sample, the pattern is still the

same for the definitive cases (N=192, Spearman's rho: -0.01; p-value: 0.87) and definitive plus probable cases (Spearman's rho: -0.04; p-value: 0.60). With all the comparative samples (including Indian Knoll), the sign switches, but the interpretation is the same (Definitive plus probable cases: N=266, Spearman's rho: 0.02; p-value: 0.80) (Table 7.1). The relationship is generally negative, but always near zero and non-significant, i.e., there is no relationship between percent carious teeth and percent of alveoli with antemortem missing teeth per individual. Therefore caries are not associated with much tooth loss in any of these groups. The results are even slightly negative, related to age; there was only one example from the literature (Banyoles) of an elderly individual with an example of caries in the Late Pleistocene. Tooth loss was greatest by an order of magnitude in the elderly category relative to the other age groups.

Recently erupted teeth are most vulnerable to demineralization and caries, and wear likely removes many carious lesions (Caries-Attrition Competition: Maat and van der Velde, 1987; but see Meiklejohn et al., 1992). Because the aging of these specimens was done using dental wear scores, the relationship between caries and antemortem tooth loss is likely also confounded by dental attrition. Attrition removes caries, but can also expose the pulp chamber and cause infection or inflammation and tooth loss (Kieser et al., 2001). Therefore the elderly individuals, i.e., those with the most dental wear, having fewer caries and much tooth loss is predictable under the caries-attrition competition model and likely driving the relationship—or lack thereof—between caries and antemortem tooth loss. Tooth loss before agriculture was not uncommon, and it has been suggested that this loss was the result of attrition; with agriculture, tooth loss rates decreased within the same regions and are likely the result of caries where tooth loss does occur (Anderson, 1968). The results here further support that proposed shift in that the pre-agricultural groups here have much tooth loss in the elderly, but few caries.

The Caries Correction Factor estimates the percentage of antemortem missing teeth caused by caries (Lukacs, 1995). This should hypothetically account for one of the issues of the osteological paradox: where what is preserved may only represent the health status of individuals when they died. Therefore caries may be underestimated, and this should be accounted for. However in this sample, there appears to be no relationship between antemortem tooth loss and caries. The dip in the elderly caries prevalence despite the general caries increase trajectory over age categories for the Indian Knoll sample is likely partially a result of caries progressing to pulpal death and tooth loss in the elderly; however the Spearman's rho changes little when Indian Knoll is pulled out on its own (N=74, Spearman's rho: -0.05; p-value: 0.64). These teeth with small carious lesions are being lost for other reasons, likely attrition, trauma, and periodontal disease.

Antemortem Tooth Loss & Periapical Lesions

Severe periapical lesions can result in tooth loss if they are persistent. Individuals in sub-recent and Neolithic-era populations even attempted on occasion to extract teeth to alleviate the discomfort of infection and inflammation (Jackson, 1914; Brothwell, 1959; Zias and Numeroff, 1986; Forshaw, 2009). For the Late Pleistocene only sample, the relationship is only slightly positive and non-significant for the percentage of definitive cases of antemortem tooth loss and percent of alveoli with periapical lesions per individual (N=136, Spearman's rho: 0.11; p-value: 0.22), but the relationship is more positive and significant when the probable cases of tooth loss are added (Spearman's rho: 0.32; p-value: <<0.01). With the comparative samples included, both definitive cases (N=259, Spearman's rho: 0.39; p-value: <<0.01) and definitive plus probable cases of tooth loss (Spearman's rho: 0.52; p-value: <<0.01) have a positive and significant relationship with periapical lesions (Table 7.1). Correlations for the Late Pleistocene and pooled

samples strengthen when probable cases are added. This is because cases of severe lesions are also likely diagnosed as possible, but not definitive examples of antemortem tooth loss (perhaps perimortem?) depending on the amount of bony support still present in the alveolus. Therefore there is some redundancy in those diagnoses, though one would expect these pathologies to be positively correlated either way. Whether caries, attrition, or periodontal disease is causing periapical lesions, they can produce tooth loss if they are chronic and expansive. Another study of the Late Pleistocene also confirms the co-variance of "abscesses" and tooth loss, at least for the Early Upper Paleolithic, suggesting common risk factors like trauma and heavy attrition (Frayer, 1989).

Caries and Periodontal Disease:

Periodontal disease severity score and percent of carious teeth per individual are slightly positively, but non-significantly correlated for the Late Pleistocene sample (N=112, Spearman's rho: 0.11; p-value: 0.24) and even less correlated when the comparative samples are included (N=254, Spearman's rho: 0.04; p-value: 0.48), especially considering that Point Hope had the highest prevalence of periodontal disease and the least caries. The lack of relationship between periodontal disease and caries in these samples may be related to non-dietary causes of periodontal disease. If carbohydrate consumption can cause both caries and periodontal disease, but these pathologies are not co-varying in individuals, they likely do not share an etiology in these samples. High periodontal disease and low caries prevalence in a sample could still indicate carbohydrate consumption if it is restricted to adults, who are less susceptible to the formation of new carious lesions (Hujoel, 2009). Because periodontal disease severity decreased over time in Europe, especially for the older age categories, but caries increased over time, one would not anticipate these pathologies to be strongly correlated. A strong relationship between

caries and "alveolar disease" was lacking in previous assessments of the Upper Paleolithic and Mesolithic (Frayer, 1989), and Brothwell and colleagues (1963) considered caries to be a "minor factor" in alveolar disease for the Pleistocene. However certain species of oral flora are associated with both periodontal disease and caries in recent samples (Loesche, 1986; Löe, 2000), and the two pathologies are associated with one another in modern dentistry practice, especially root caries and periodontal disease (Ravald and Hamp, 1981). Few root caries were observed in this study.

Periapical Lesions and Periodontal Disease:

For the Late Pleistocene, periodontal disease severity score (most severe septa diagnosis) and "percent of alveoli affected by periapical lesions per individual" are somewhat positively correlated (N=117, Spearman's rho: 0.35; p-value: <<0.01) and the pattern strengthens when the comparative samples are included (with only Point Hope and Natufians, N=185, Spearman's rho: 0.47; p-value: <<0.01; with all comparative samples, N=257, Spearman's rho: 0.36; p-value: <<0.01). The data were tested with and without the Indian Knoll sample since many of the lesions in the Indian Knoll sample were likely of carious origin (see above), and the Spearman's rho value is lower when the Indian Knoll sample is included. Food production at Indian Knoll was not like the other samples (Leigh, 1925). This suggests that indeed many lesions are related to periodontal disease and not caries for pre-agricultural groups (caries and periapical lesions were not significantly correlated except at Indian Knoll). However because the relationship is not stronger, many lesions may be attributable to another cause, perhaps wear or oral trauma; or there may be a strong mortality risk associated with periapical lesions. Also the causality is two-way for these pathological conditions: the inflammation from a lesion can induce alveolar

resorption; and periodontal inflammation and alveolar destruction can create conditions that allow bacteria to enter the alveolus.

Antemortem Tooth Loss & Periodontal Disease

For the Late Pleistocene, periodontal disease severity scores and percent of definitive cases of tooth loss per individual are somewhat positively and significantly correlated (N= 117, Spearman's rho: 0.22; p-value: 0.01), as are periodontal disease severity scores and definitive plus probable cases of tooth loss (Spearman's rho: 0.34; p-value: <<0.01). The pattern further strengthens when the comparative samples are included (definitive cases (N=259, Spearman's rho: 0.33; p-value: <<0.01) and definitive plus probable cases (Spearman's rho: 0.43; p-value: <<0.01)) (Table 7.1). This suggests that some tooth loss in pre-agricultural groups (Late Pleistocene and Holocene) is caused by periodontal disease—and nearly none caused by caries.

Previous research hypothesized that tooth loss in the Pleistocene could not be attributed to periodontal disease, but more likely attrition and trauma (Clarke et al., 1986; Kerr, 1994). This position was supposedly supported by results showing a decrease in tooth loss with agriculture as the cause shifts from attrition to caries (Anderson, 1968). Though the Point Hope sample has high wear, high periodontal disease prevalence, and high tooth loss prevalence, any causality between periodontal disease and tooth loss for this sample was also dismissed (Costa, 1982). But other researchers presume a stronger relationship between periodontal disease and tooth loss is possible in the fossil record (Scott and Turner, 1988) and it is assumed to be so in the dental literature (McLeod et al., 1997; Nibali et al., 2013). The Assos skeletal remains had much periodontal disease, but tooth loss was rare. This was explained as being attributed to short life expectancy where the individuals did not have enough time for their periodontal disease to progress to tooth loss (Oztunc et al., 2006). There is likely much multicollinearity between

periapical lesions caused by attrition and periodontal disease producing tooth loss in this sample. Running all the pathologies but caries through a regression is now done to attempt to control for these confounding factors.

All pathologies:

Antemortem tooth loss percentage per individual (definitive plus probable) in the Late Pleistocene can be predicted from a regression equation of age, periodontal disease severity, and percent of lesioned alveoli per individual (see Equation 7.1). This regression shows that for each increase in age category, there is a 4% increase in alveoli with evidence of tooth loss per individual *ceteris paribus*, and for each increase in periodontal disease severity score, alveoli with evidence of tooth loss increases by 2% per individual *ceteris paribus* (periapical lesions are not a significant coefficient value). This suggests that the dental wear (age) and periodontal disease more strongly predict tooth loss than periapical lesions and the relationship between lesions and antemortem tooth loss identified above may then be a result of multicollinearity. And even with age/wear held constant, periodontal disease severity score still predicted tooth loss. In an ANCOVA with tooth loss as the dependent variable and time period, region, and age as the independent variables, only the age variable (p-value: <<0.01) significantly predicted tooth loss in the Late Pleistocene model (p-value: <<0.01).

$$\%\, AMTL\ = .\mathbf{04}(Age) + .14\ (\%lesioned\ alveoli) + .\mathbf{02}(Periodontal\ disease\ severity) - \mathbf{0.09}(constant)$$

Equation 7.1: Regression using age category (1-4), percentage of lesioned alveoli per individual and periodontal disease severity score (0-3) to predict percentage of teeth lost antemortem (bolded coefficients were statistically significant; r^2=0.20, p-value: <<0.01)

Using Brennan's (1991) equation (3.1, see Chapter 3: Methods) to calculate the weight and directionality of health changes, there is little overall oral health change pattern here (Table 7.2). From Neandertals to Early Upper Paleolithic peoples, two pathological conditions increase (caries and periapical lesions), but two decrease (periodontal disease and antemortem tooth loss) with a score of 2.67. From Middle Paleolithic to Early Upper Paleolithic modern humans, one pathology increases (periapical lesions), one pathology decreases (caries) and the other two prevalences are stagnant (periodontal disease and antemortem tooth loss) for a score of 1.33. From the Early Upper Paleolithic to the Late Upper Paleolithic modern humans, two pathologies increase (caries and antemortem tooth loss) and one decrease (lesions) and one has an inconsistent pattern (periodontal disease) with a score of 2.67. Considering that the highest possible score here would be a 5.33, the health changes overall between these temporal groups are minimal. The trajectories per pathology are compelling, but conflictory when taken together as "overall health". However caries is unlikely to have a strong systemic effect unless severe—though diet itself can—and the other three pathological conditions are more strongly linked to morbidity and mortality. The differing implications for diet and health are discussed further.

	Caries	Lesions*	Periodontal Disease	Tooth loss	Value
Neandertals to EUP	↑	↑	↓	↓	2.67
MPMHs to EUP	↓	↑	No change	No change	1.33
EUP to LUP	↑	↓	No change	↑	2.67

Table 7.2: Table of pathology prevalence directionality between time periods (MPMHs: Middle Paleolithic modern humans; EUP: Early Upper Paleolithic; LUP: Late Upper Paleolithic); *insignificant

Summary:

Caries stands alone and is not significantly correlated with any of the other pathologies, except within the Indian Knoll sample. It is also the only pathology that does not increase with age/ dental wear. The other pathological conditions—periodontal disease, periapical lesions, and antemortem tooth loss (whether only definitive cases or with the definitive and probable cases pooled)—are all positively and significantly correlated with each other for the Late Pleistocene and comparative samples (except for definitive tooth loss and periapical lesions for the Late Pleistocene sample) and also increase with age. Therefore one of the contributing factors in the co-variance of these pathologies is the age of the individual, related to either cumulative exposure risk including dental attrition or increasing susceptibility to disease. Since age is calculated by dental wear for these individuals, increases in periapical lesions and antemortem tooth loss with age is related to dental attrition's contribution to the formation of periapical lesions and tooth loss. However periodontal disease could also be contributing to both periapical lesions and tooth loss in these samples; all of the variables but caries are highly correlated with one another. Because wear can increase alveolar crest to cemento-enamel junction distances through continuous eruption without disease present in samples with high dental attrition, the "most severe" periodontal disease diagnoses were produced with interdental septa condition scores. Hopefully this avoided a situation where all of these pathologies are correlated with one another because they are all correlated with dental wear. Regression shows that even when holding age/ dental wear constant though, periodontal disease and tooth loss are still positively correlated.

Project Hypotheses:

This dissertation project was designed around four basic null hypotheses presented in the introductory chapter. These concern differences in oral health, ergo morbidity, between the variously defined subgroups. Here the results of this project are discussed as they pertain to each of the four hypotheses and within the context of the larger issues surrounding systemic health, diet, and quality of life.

$H1_0$: There are no significant differences between Late Pleistocene groups and Holocene pre-agricultural comparative samples: This project had only three comparative samples to represent the whole of Holocene hunter-gatherers. However the initial pattern suggests that for oral health, there is not a major shift from the Late Pleistocene to the Holocene when the Agricultural Revolution is removed from consideration, but rather regional differences become stronger. The Epi-Paleolithic Natufians actually have slightly fewer caries, periapical lesions, periodontal disease diagnoses, and antemortem tooth loss than Late Upper Paleolithic modern humans, though life expectancy was very low for Natufians (Eshed et al., 2006) and most pathologies increase with age. The Point Hope sample is similar to Neandertals for caries, but Point Hope has even higher periodontal disease diagnoses, and percent of alveoli affected by periapical lesions and antemortem tooth loss than any Late Pleistocene group. The Point Hope diet was high in fat and protein, similar to the Late Pleistocene, but the age distribution skews older. This may explain the increase in pathologies correlated with age, but similar caries prevalence. The increases in regional heterogeneity in the Holocene with respect to subsistence strategy may be driving these subtle trends, but it does not seem that the Holocene is wholly more or less healthy than the Pleistocene with the exception of increasing life expectancy in the Mesolithic of Europe (Wittwer-Backofen and Tomo, 2008) and at Point Hope (Dabbs, 2009). Including Mesolithic

European samples in the future can further refine this hypothesis. Initial surveys of available Mesolithic samples found higher caries prevalence than the Upper Paleolithic (15.2% of adult teeth, as compared with 3.8% in the Late Upper Paleolithic) and roughly consistent periodontal disease diagnosis (81.5% of adults, as compared to 82.4% in the Late Upper Paleolithic) (Wittwer-Backofen and Tomo, 2008).

The peoples of Indian Knoll were practicing early forms of agriculture, and this is likely reflected in their higher caries prevalence (8.1%, similar to the Middle Paleolithic modern humans though, 7.7%) and much higher lesion prevalence (10.6%). The lower periodontal disease diagnoses, 68.9% of individuals, suggests they were slightly healthier, though life expectancy is not longer for the Indian Knoll peoples than Paleolithic peoples (Johnston and Snow, 1961). Tooth loss prevalence does not differ in the Indian Knoll sample from the Late Pleistocene, but the co-variance tests suggest that the cause of this tooth loss shifted from periodontal disease and attrition to caries and attrition. Where subsistence patterns shift in the Holocene, oral health differs from the Pleistocene, but otherwise it does not appear that oral health increases or decreases dramatically or consistently in the Holocene. Other sociocultural changes must take place first. Therefore this hypothesis is not rejected.

$H2_0$: There are no significant differences between the Middle Paleolithic (Neandertals and modern humans) and Early Upper Paleolithic: There were two Middle Paleolithic samples considered here: Middle Paleolithic modern humans from Southwest Asia and Middle Paleolithic Neandertals from Europe and Southwest Asia (the few Initial Upper Paleolithic Neandertal remains were not available for this study). From Table 7.2, there is not a concrete pattern from the Middle Paleolithic to the Early Upper Paleolithic. Periapical lesions increased in the Early Upper Paleolithic relative to both Middle Paleolithic modern humans and Neandertals, but there

was no change in periodontal disease severity and antemortem tooth loss prevalence between Middle Paleolithic and Early Upper Paleolithic modern humans.

Earlier analyses of stress indicators on both sides of the Upper Paleolithic transition found that stress decreased in the Early Upper Paleolithic relative to Neandertals (dental enamel hypoplasias (Ogilvie et al., 1989; Brennan, 1991; Skinner, 1996; Hillson and Bond, 1997; Teschler-Nicola et al., 2006; Doboş et al., 2010), stature (Holliday, 1995)). Early Upper Paleolithic peoples have slightly more caries (non-significant when published examples are included in the sample) and less advanced periodontal disease and tooth loss than Neandertals. Recent summaries of the Upper Paleolithic transition have dialed back the contrast between Early Upper Paleolithic modern humans and Neandertals, recognizing that the differences are minimal with Early Upper Paleolithic modern humans reducing their use of teeth-as-tools and other uses of anatomy for manipulation as well as reducing stress levels (Trinkaus, 2013). Otherwise this shift is subtle and not due to some hypothetical overwhelming technological, biological or resource advantage in the favor of modern humans (Trinkaus, 2013). The results here could be used to support this conclusion in that caries prevalence, perhaps representing diet, changes little, but periodontal disease and tooth loss, representing systemic health and mortality risk, alleviate. Others have also declared that periodontal disease was high in Neandertals, but without citation or data (Lanfranco and Eggers, 2012). It is unclear whether population density in the Early Upper Paleolithic was lower than it had been previously (Morin, 2008) or higher (Mellars and French, 2011); therefore it is further unclear which direction population stress was driven as environmental stress increased (Lambeck and Chappell, 2001). The decrease in tooth loss and mild alleviation of periodontal disease suggests some social aspect was combatting the effects of increasing environmental stress though.

Rarely are Middle Paleolithic modern humans explicitly compared with Early Upper Paleolithic modern humans, but they both show lower levels of dental enamel hypoplasias as compared with Neandertals or Late Upper Paleolithic modern humans (Skinner, 1996; Buzhilova, 2000; Tillier et al., 2004; Teschler-Nicola et al., 2006; Trinkaus et al., 2006b; Doboş et al., 2010), mirroring the static levels of tooth loss and periodontal disease between the two groups observed here. Therefore if tooth loss and periodontal disease are assumed to represent systemic health, there is little difference between modern humans in the Middle Paleolithic and Early Upper Paleolithic. The major difference in caries prevalence (MPMH: 7.7%; EUP: 1.9%) likely reflects regional dietary variation in that Middle Paleolithic modern humans included here are exclusively from Southwest Asia along the Mediterranean, and the Early Upper Paleolithic peoples cover a wide latitudinal range in Europe, but not Southwest Asia. Once again, this verifies the hypothesis that the major shift in modern human health and behavior occurs later on in the Upper Paleolithic, not at the Middle to Upper Paleolithic transition (Holt and Formicola, 2008; Trinkaus, 2013). Hypothesis two is rejected for Neandertals to Early Upper Paleolithic modern humans, but only rejected in references to caries and lesions for the Middle Paleolithic modern humans to Early Upper Paleolithic.

$H3_0$: There are no significant differences between the Early Upper Paleolithic and the Late Upper Paleolithic: This hypothesis is firmly rejected for all oral pathologies except periodontal disease. In the Late Upper Paleolithic—as compared with the Early Upper Paleolithic—caries, tooth loss, and agenesis (not a pathology) increase, and lesions decrease. This reflects the demographic and subsistence shifts occurring around the Last Glacial Maximum including the decrease of group territory sizes caused by population density increases (Mellars, 1985; Jochim, 1987) and the contraction of productive environments and decreased resource reliability in

Western Eurasia (Dennel, 1983; Gamble, 1986; Straus, 1995; Holt et al., 2000). This resulted in increasing foraging costs for groups (Stiner, 2001) and decreasing mobility (Holt, 2003), coupled with increasing regional cultural diversity (Bosinsky, 1990) and specialized resource exploitation camps (Straus 1986; 1990). In the context of a degrading environment, human groups were attempting to intensify and specialize resource extraction as well as differentiate themselves from other groups in both ecological and cultural niches. This resulted in dietary change producing increased carious lesions for some groups and increasing cultural and group cohesion (Bar-Yosef, 2007), which may have reduced mortality risk from tooth loss. Lesion percentage decreases, and this could be because carious lesions were still mostly non-penetrant and therefore not affecting the pulp chamber (but this is a consistent pattern across the Late Pleistocene), or dental attrition is less severe (but there are no dental wear differences between the Early Upper Paleolithic and Late Upper Paleolithic at least in immature individuals (Skinner, 1997)).

Greater intra-individual variance in alveolar condition in the Early Upper Paleolithic makes it difficult to compare periodontal disease severity with the Late Upper Paleolithic. Based on CEJ-AC averages, there is a slight increase in periodontal disease severity in the Late Upper Paleolithic compared with the Early Upper Paleolithic (not statistically significant, but perhaps biologically meaningful), but with most severe septa diagnosis per individual, the Late Upper Paleolithic decreases from the Early Upper Paleolithic. Considering that a number of other stress indicator analyses have found a slight, but definitive increase in stress in the Late Upper Paleolithic relative to the Early Upper Paleolithic, this seems like the most likely interpretation: focusing on the increase in average CEJ-AC distance diagnoses in the Late Upper Paleolithic. But others have reported a decline in periodontal disease presence (no severity was reported) between the Early Upper Paleolithic and Late Upper Paleolithic (Brennan, 1991), which may

also reflect the decrease in periapical lesions observed. Dental enamel hypoplasias increased (Brennan, 1991) and stature and body mass decreased as well in the Late Upper Paleolithic (Frayer, 1980; Holt and Formicola, 2008). Perhaps there was selection for smaller overall body size (Formicola and Holt, 2007), but it could reflect declining nutrition's effect on growth and development (Formicola and Giannecchini, 1999), also seen in increasing caries prevalence. Whether periodontal disease severity increases or decreases in the Late Upper Paleolithic, the shift is slight relative to the other pathologies that support the rejection of the null hypothesis.

$H4_0$: There are no significant differences between the three identified regions of western Eurasia*:* The pathology with the strongest regional pattern is caries, rejecting the null hypothesis. There is a clear latitudinal cline. In the Middle Paleolithic, there are no caries above 44°N latitude (Aubesier 5 and 12 from Southern France are the most northerly examples). In the Upper Paleolithic, modern humans colonize the North regardless of climatic cycle (van Andel and Davies, 2003). Caries prevalence inches northward with them, but continues to focus and intensify around the Mediterranean. The confounding factor for Western Eurasia is that the Mediterranean Sea dominates the southern portion of the continent here. Is this a latitudinal cline or an ocean-proximity cline? The Mediterranean's coastline shifted throughout history, but the sea was never completely dry during the Late Pleistocene (Vesica et al., 2000). However portions of what is now Maritime Atlantic Europe were not consistently oceanfront in the past (e.g., Atlantic France, Northern Spain, Southern England) (Donn et al., 1962).

Therefore was the warmer environment of Mediterranean Europe producing vegetal resources higher in sugars; or was it the access to water from the Mediterranean affecting the plants; or the decreased groundwater fluoride levels produced by ocean proximity? Some Mediterranean regions are and have been somewhat dry (Robinson et al., 2006), and there is no

evidence of increased water access for plants increasing their sugars (drought actually concentrates sugars (Chaves et al., 2002)); therefore it was not likely that the water itself was affecting the plants. Perhaps it was a combination of both of the other factors in that individuals' teeth were developing without much dietary fluoride and their diets contained more sugar (Epi-Paleolithic caries in Mediterranean Morocco was extensive (Humphrey et al., 2014)). The Late Pleistocene diet was high in protein regardless of region, so these regional differences may have been small, producing small increases in caries prevalence in the Mediterranean. For the Late Pleistocene, the Mediterranean caries prevalence is more than double that of the other two regions (Mediterranean: 4.6%; Atlantic: 1.8%; Continental: 1.2%). Mediterranean Europe recovered faster from cold intervals in the Late Pleistocene and was consistently temperate and usually wet (van Andel, 2003), and longer hours of daylight and a warm and wet climate produce higher levels of sugars in plants (Kirschbaum, 2004; Zheng et al., 2009). Relative to other parts of the world, all of Europe generally has low groundwater fluoride levels, and wet environments have even lower fluoride levels than arid ones because of aquifer dilution (Brunt et al., 2004). Therefore many Western Eurasian peoples did not have the protective benefits of fluoride in their diets, and Mediterranean ones had access to plants with higher sugar contents than peoples living further north. Greater caries prevalence is unsurprising in this context.

Conversely, there is half the prevalence of periapical lesions along the Mediterranean (1.8%, or 1.5% when the Natufians are included) than further North (Atlantic, 3.9%: Continental, 4.9%). This further confirms the lack of relationship between caries and periapical lesions in the Late Pleistocene. An explanation for this pattern has yet to be identified. It could be related to some introduced dietary grit causing dental attrition further north or lack of vitamin D from reduced sun exposure. Other studies have linked oral infections to carbohydrate consumption in

skeletal remains (Larsen, 1997), which is the opposite of what is seen here considering dietary carbohydrates are inversely related to latitude (Ströhle and Hahn, 2011).

Whether using most severe septa diagnosis or CEJ-AC average per individual, Continental Europe has non-significantly more severe periodontal disease than the other two regions. This could mean there was no regional difference in health; or it could indicate that there was a slight increase in periodontal disease severity in Continental Europe, but it is not strong enough with this sample size to be statistically confirmed. Low circulating vitamin D can cause periodontal disease (Hennig et al., 1999; Garcia et al., 2011) and for Early Upper Paleolithic modern humans (which dominate the Continental sample) recently arriving from further south, Vitamin D insufficiency may have been an issue. But there is no definitive example of rickets or osteomalacia from the Late Pleistocene (Skinner, 1996), suggesting severe vitamin D deficiency was not a persistent problem. There may have been some yet unexplained environment or biocultural phenomenon in Continental Europe during the Late Pleistocene causing decreased health relative to the rest of Western Eurasia (Seasonal obesity? (Coleman, 1998; Trinkaus, 2005)); or a broader pattern of decreased health over all of Europe (Smoke Inhalation? (Platek et al., 2002; Størmer and Mysterud, 2007)). There was no difference by region for antemortem tooth loss.

Overall health and environment discussion:

Systemic Health:

Both periodontal disease (Destefano et al., 1993; Jansson et al., 2002; Dewitte and Bekvalac, 2010) and tooth loss (Padilha et al., 2008) are associated with increased mortality risk in living humans and recent skeletal samples. Considering that periodontal disease and tooth loss are both correlated with each other as well as with periapical lesions (infection) in the samples

examined here, the reconstruction of oral health in these fossil humans has implications for life expectancy and mortality risk. People did not live long in general in the Late Pleistocene (Trinkaus, 1995; 2011; Caspari and Lee, 2004), and the elderly age category here contained individuals approximately aged 40 and over (Smith, 1984). The elderly individuals had the most severe periodontal disease, the most teeth missing antemortem, and the most alveoli affected by periapical lesions. The only pathology that did not affect the elderly the most severely was caries.

The Middle Paleolithic modern humans sampled here contained no elderly individual (Trinkaus, 2011) and had the most caries, the least lesions, the least severe periodontal disease based on average CEJ-AC distance, and the least antemortem tooth loss. Are modern humans in Southwest Asia in the Middle Paleolithic living shorter lives; or is this preservation bias? The Middle Paleolithic modern human sample was not living long enough for their oral disease to reach severe states, but if this is the true demographic structure, this was a stressed population (Trinkaus, 2011). None of the Southwest Asia Neandertals sampled here were elderly either (The Shanidar Neandertals were not available for this study). Based on their oral health alone, one may surmise that this sample was healthier than the rest, but without elderly individuals, that interpretation is likely incorrect.

Previous research did not find a difference in mortality distribution between Neandertals and Upper Paleolithic modern humans in Europe (Trinkaus, 2011), so the oral health differences observed here were not producing mortality differences. The morbidity differences are real though. The relationship between oral health and systemic health are well established in recent humans from a number of different research lines (Slavkin and Baum, 2000; Garcia et al., 2001; Meurman et al., 2004; Williams et al., 2008; Cullinan et al., 2009; Hujoel, 2009), and this

relationship is assumedly the same in the past. Periodontal disease is considered to be a good indicator of general health and socioeconomic status in skeletal remains (Minotti, 2003; Dewitte and Bekvalac, 2010).

Neandertals had more tooth loss and more severe periodontal disease than Early Upper Paleolithic modern humans. Neandertals were then suffering from greater morbidity than their direct ecological competitors, and this may have contributed to their shrinking demographics around the arrival of modern humans and ultimate disappearance (Sørensen, 2011; Bonquet-Appel and Degioanni, 2013). Periodontal disease is also correlated with negative pregnancy outcomes (Cohen et al., 1969; Garcia et al., 2001; Lieff et al., 2004; Shetty et al. 2010), a major factor in demographic differences between groups. Though Early Upper Paleolithic modern humans also have high levels of periodontal disease, their slight improvement in periodontal health and tooth loss over Neandertals may have been enough to improve reproductive fitness and contribute to their ascendancy in Western Eurasia.

Research on other indicators of health from Late Pleistocene skeletal remains can be used to verify the patterns seen here in oral pathologies in the Late Pleistocene. Dental enamel hypoplasias (Ogilvie et al., 1989; Brenna, 1991; Skinner, 1996; Buzhilova, 2000; Guatelli-Steinberg et al., 2004; Teschler-Nicola et al., 2006; Trinkaus et al., 2006; Doboş et al., 2010), Harris lines (Brennan, 1991), trauma (Berger and Trinkaus, 1995; Trinkaus 2005b; Trinkaus, 2013), and infection (Dastugue, 1967; Oliva, 2000; Lebel and Trinkaus, 2002a; Trinkaus, 2005a; Vercellotti et al., 2008) all show a decrease in the Early Upper Paleolithic as compared with Neandertals (Holt and Formicola, 2008; Trinkaus, 2013). The pattern is more conflictory for the Early Upper Paleolithic to Late Upper Paleolithic with respect to other stress indicators (Holt and Formicola, 2008; Trinkaus, 2013), and this is also the pattern here. Average CEJ-AC distance

increases in the LUP, but most severe periodontal disease diagnosis per individual decreases. Percent of lesioned alveoli decrease, but percent of teeth lost antemortem and carious increases. Diet was certainly shifting between the Early and Late Upper Paleolithic along with the environment, but it is unclear to what extent health did as well.

Subsistence:

Diet is strongly tied to caries and dental attrition, but also periodontal disease and infections in systemic ways, and these can all result in tooth loss. Historically the main focus of research on Late Pleistocene diet has been on large terrestrial mammals through zooarchaeological surveys. These large packages of protein and fat were certainly a major component of any Paleolithic human groups' subsistence and they dominate archaeological assemblages. Only more recently have paleoanthropologists begun to focus on small mammals (Stiner et al., 1999; Stiner, 2001), fish (Richards et al., 2001), birds (Hardy and Moncel, 2011; Peresani et al., 2011; Finlayson et al., 2012) and vegetal dietary resources (Hardy et al., 2001; Lev et al., 2005; Revedin et al., 2010; Hardy and Moncel, 2011; Henry, 2011; Henry et al., 2011). There is little dietary shift at the Upper Paleolithic transition (Stiner, 1994; Hardy, 2010; Fiorenza et al., 2011; Henry, 2011; Trinkaus, 2013); but diet does vary regionally (Fiorenza et al., 2011; Henry, 2011). The more noticeable subsistence shift in the middle of the Upper Paleolithic does not reflect improved food acquisition techniques though, but increased hunting pressures on the larger animals resources forcing humans to shift to other sources (Stiner et al., 1999; Richards and Trinkaus, 2009; Trinkaus, 2013). Wholly new items are not necessarily added to the diet, but there is intensification and specialization on resources previously exploited only on occasion, e.g., turtles, lagomorphs, bivalves (Straus, 1987; Grayson and Delpech, 2002; Stiner and Kuhn, 2006; Morin, 2008).

The caries data support the hypothesis that modern humans shifted from fats to carbohydrates in the Late Upper Paleolithic as the supplement of choice to avoid protein poisoning (Cachel, 1997). Humans must consume either fats and/or carbohydrates with protein, or they risk calcium depletion and protein poisoning. This increase in caries is therefore the result of a sociocultural phenomenon and not a biological one (Caselitz, 1998). Carbohydrates were not absent from earlier diets (*Homo erectus* got perhaps 50% of its calories from carbohydrates (Wrangham, 2009)), but the poor energy trade-off of collecting fruits, berries, and honey (all high in sugars) became more necessary as the availability of large packages of calories (large terrestrial mammals) became more ecologically expensive. Modern human expansion was largely influenced by shifting ecozones (Trinkaus, 2013), and biocultural adaptations were necessary for their success in any climatic zone. One of these was resource flexibility reflected in increasing caries prevalence, especially for those further south, who had available resources in their environment with high amounts of sugar.

Neandertals may not have been able to shift their resource exploitation strategies as easily. Neandertals show more dental enamel hypoplasias post-weaning, suggesting higher resource instability once maternal buffering is removed (Ogilvie et al., 1989; Hillson and Bond, 1997; Trinkaus, 2013). Large mammal resources also fluctuate greater and more frequently in colder climates (Morin, 2008), suggesting population crashes in reindeer and other mammals could results in population crashes for humans dependent on those resources without fallback dietary supplements (Monge and Mann, 2007; Dennell et al., 2011; Sørensen, 2011). Though periodontal disease can be the result of carbohydrate consumption (Hujoel, 2009), high periodontal disease and tooth loss in Neandertals more likely reflects higher physiological stress related to high mobility as well as dietary instability.

Quality of life:

Understanding morbidity in the past is a worthy endeavor to elucidate ecological competition between groups and sample life expectancies. However it can also inform a biocultural approach to exploring stress, survival, and qualify of life for these Late Pleistocene groups (Trinkaus and Svoboda, 2006; Trinkaus, 2013). How did oral pathologies affect these individuals' lives, and what did they do to mediate unpleasant symptoms? A recent case study of a Neandertal from Spain with periodontal disease also showed evidence of toothpick grooves (Lozano et al., 2013). This individual may have been picking at his inflamed interdental septa with a sharpened piece of wood or bone or attempting to remove food or plaque caught in the interproximal spaces between his teeth. The interpretation provided for the presence of both of these conditions (periodontal disease and tooth pick grooves) suggested Neandertals were practicing a medical solution to a bodily discomfort. Toothpick grooves have been documented as far back as *Australopithecus* (Ungar et al., 2001), so it is unlikely that they represent a novel innovation to oral hygiene problems in Neandertals. But it does suggest that oral inflammation affected the individual to the point where he/she sought remedy (Lozano et al., 2013). Tooth pick grooves were recorded when noted during the data collection portion of this project, and therefore a broader context to periodontal disease and one potential mediating behavior can be explored in the future.

Extensive tooth loss and generalized gingival inflammation has been used to argue for societal care in the past (Lebel et al., 2001; Lebel and Trinkaus, 2002a; Lordkipanidze et al., 2005; Hublin, 2009). Post-tooth loss was not painful state for individuals, but it may have compromised their masticatory efficiency (Hublin, 2009). Abscessed teeth can be very agonizing though and also produce mechanical instability during mastication (Hublin, 2009). This suggests

that perhaps it is more problematic for the individual just before a tooth is lost, when it is unstable and more painful, than after the tooth is lost and inflammation subsides. The loss of the tooth is likely a relief as there is much evidence for dental extractions in the Neolithic and in sub-recent groups for reasons other than aesthetics (Jackson, 1914; Brothwell, 1959; Zias and Numeroff, 1986; Forshaw, 2009). Those with abscessed and loose teeth perhaps required more provisioning and care than those who have lost the problem teeth (McLeod et al., 1997).

Periodontal disease is now demonstrated to be common in the Late Pleistocene. It seems unlikely that everyone with advanced periodontal disease required extensive special food preparation and care when 40% of Neandertals had advanced periodontal disease somewhere in their dental arcades. There would more likely be cultural innovations in food preparation to avoid oral discomfort for everyone than special treatment for two out of five members of every group. Tooth loss is generally confined to the elderly age category for every temporal group though, so it was considerably less widespread and perhaps necessitated—and received—special food preparation. Considering that elderly individuals were likely left behind when they could no longer keep up with the mobility of the group (Trinkaus, 2013), they may have been expected to keep up with the diet of the group as well.

Oral health was not stellar in the Upper Paleolithic. Many previous authors assert that oral health was good, perhaps because caries and malocclusion are rare (e.g., Holt and Formicola, 2008), but no one had attempted to survey the tooth loss, oral infection, and periodontal disease status of these individuals. Oral pathological conditions did affect the lives of those who suffered from them in the Late Pleistocene (e.g., Lozano et al., 2013), but considering how common periodontal disease was (81.3% of all individuals surveyed in the Late Pleistocene had at least mild periodontal disease), especially for the older individuals (94.4% of all Late

Pleistocene Mid-Aged or Elderly individuals had at least mild periodontal disease), individuals were afflicted equally.

Summary:

Regardless of taxonomic designation, the changes over time in oral health in the Pleistocene are relevant for our understanding of health today. Entire subfields of medical research are devoted to understanding the effects of modern diet, lifestyle, and environment on human health and mortality. A baseline of pathology prevalences before the introduction of these variables is vital to interpreting their effect on modern human health. The vast majority of human evolution occurred when humans were practicing hunter-gatherer subsistence and living at low population densities across the Old World. Without understanding oral pathological conditions in the Late Pleistocene, modern oral health has no context. The research here suggests that periodontal disease, especially in older cohorts, actually lessened through the Late Pleistocene, at least between Neandertals and modern humans.

Caries increase through time in Europe. This likely reflects dietary changes occurring further south, namely tempering high protein consumption with carbohydrates in the diet as opposed to fats, moving northward over time initially with modern human expansion into Europe, and then along with shifting ecozones as the Last Glacial Maximum waned. This trajectory likely continued into the Holocene (Wittwer-Backofen and Tomo, 2008), but not consistently as seen with low caries in the Natufian sample here. Tooth loss also increases within the Upper Paleolithic and may reflect decreasing mortality risk associated with the pathology from increasing social complexity.

Finally the hypotheses of this thesis were framed to test whether oral health could provide another avenue of data on the causes of the Upper Paleolithic transition. It does appear that

periodontal disease and antemortem tooth loss prevalences were slightly lower in the Early Upper Paleolithic relative to Neandertals, but differences are generally small. They may have been enough to contribute to the larger ecological advantages that modern humans exercised over Neandertals, ultimately producing demographic expansion in modern humans and contraction in Neandertals. Hypotheses presented elsewhere that the major shift in the biology and culture in the Late Pleistocene does not take place at the Middle to Upper Paleolithic transition, but between the Early and Late Upper Paleolithic, are further supported with these data. Especially since diet has such a strong relationship with oral health—as opposed to the health of any other part of the bony skeleton—and dietary shifts were occurring most strongly around the Last Glacial Maximum. Changes in oral health in the Late Pleistocene reflect both known changes in health and demography as well as diet and culture, validating the use of oral health as a proxy for systemic/ overall status for fossil humans.

Chapter 8: Conclusion

Casual observations of oral pathology in fossil remains "tempts one to conclude that the same kind of dental diseases affected ancient as well as modern humans" (Tillier et al., 1995: 191), and this survey confirms Tillier and colleagues' suspicion. Though the prevalence of pathology may change, all the same oral diseases of recent humans are present in the fossil record. Rare and idiosyncratic pathologies were not explored in this paper, though they were observed and recorded during data collection for future analysis. The focus of this thesis has been on the major scourges of modern dentistry: caries, oral lesions and infections, periodontal disease, and tooth loss. Caries may have increased in prevalence dramatically with the advent of agriculture (Larsen, 1995), but they were certainly present in Late Pleistocene hunter-gatherers, and an increasing trend in prevalence was initiated well in advance of agriculture (see Humphrey et al. (2014) as well). The caries of the Late Pleistocene are rarely severe though—baring earlier examples such as Broken Hill. And because these carious lesions rarely progressed to pulpal involvement, caries are not correlated with periapical lesions or tooth loss within individuals, nor do they follow the temporal or regional patterns of the other pathologies examined.

Periodontal disease severity may have alleviated through the Late Pleistocene, but overall prevalence remained high in all groups (73.5- 86% of individuals have at least mild periodontal disease) relative to modern, dentistry analyses where individuals assumedly have access to oral hygiene (e.g., 56% of surveyed Swedes in 2003 has some level of periodontal disease (Hugoson et al, 2008); 40% of Americans had more than 3mm of alveolar loss (Oliver et al., 1998); 27.1% of Americans have periodontal disease (Eke et al., 2012)). Periodontal disease is not a recent phenomenon, nor is it exclusively the result of modern behaviors.

Tooth loss was actually higher in Neandertals than Early Upper Paleolithic modern humans. Previous attempts to dismiss the likelihood of Neandertals using "modern" mediating behaviors to deal with antemortem tooth loss by comparing them with primates or recent humans are invalidated (e.g., Degusta, 2002; Gilmore, n.d.). Neandertals were able to survive more tooth loss than incontrovertibly modern humans in Western Eurasia on both sides of the Middle to Upper Paleolithic transition. This morbidity data should not be used to argue for the less-than-modern status of Neandertals. Tooth loss was also not caused by caries (they do not co-vary), but was significantly predicted by age/ dental wear and periodontal disease status.

The one pathology that went against previous assessments of health was the increase in periapical lesions in the Early Upper Paleolithic relative to both Neandertals and Late Upper Paleolithic modern humans. This could be a function of higher dental wear and survival in spite of oral infection for Early Upper Paleolithic peoples, but the temporal pattern was not statistically significant. Periapical lesions do not co-vary with caries and therefore are likely caused by attrition, trauma, or periodontal disease.

Three of the four research hypotheses were rejected in some way; a Holocene versus Pleistocene contrast could not be identified independent of region. Early Upper Paleolithic peoples were healthier than the preceding Neandertals with the exception of the prevalence of periapical lesions (Hypothesis 2). Late Upper Paleolithic peoples had more tooth loss and caries than the Early Upper Paleolithic as well as having higher average CEJ-AC distances per individual reflecting the major climatic shifts occurring around and after the Last Glacial Maximum and their affect on diet, social structure, and behavior (Hypothesis 3). Caries followed a negative latitudinal cline that relaxed through time (Hypothesis 4). The comparative samples' oral health reflected their regional origin and subsistence patterns, but did not demonstrate a

major shift from the Pleistocene pattern until the initial adoption of agriculture, represented by Indian Knoll, which practiced early garden agriculture (Hypothesis 1 not rejected). The Holocene samples represent a continuation of the trajectory from the Late Upper Paleolithic of increasing regional heterogeneity. Ultimately this research has implications for our understanding of dietary and health changes through time from both an anthropological and broader medical perspective.

Importance of this research within and outside anthropology

The field of anthropology has long been concerned with the interplay between human biology and behavior. These interests include a variety of topics currently included within either cross-cultural medical anthropology for living populations—situated within larger ethnology—or paleopathology of Holocene past human populations within bioarchaeology. Both of these fields have experienced a recent renaissance, providing insight into the complex dynamics of human health, economy, subsistence, and social structure. At the same time, the subfield of paleoanthropology has become increasingly focused on the Late Pleistocene, which saw the emergence and eventual establishment of modern humans across the Old World. Much of this paleoanthropological research has been concerned with the populational processes involved in modern human evolution and migration, which has implications for assessing recent human biological and cultural diversity. But there has also been a growing concern with assessing possible differential ecological, technological, or demographic mechanisms that led to the "dominance" of modern humans over late archaic humans. This has been addressed in terms of possible dietary, functional anatomical, and other parameters, but only secondarily in terms of possible shifts in the morbidities and life histories of these past populations.

Within the context of these debates, this project contributes to our understanding of the evolutionary dynamics leading to the origins and establishment of modern humans across the

globe through the lens of oral health. Drawing from previous research in medical anthropology and paleopathology, this survey used established methods and questions of morbidity and differential health in recent humans and applied these to Late Pleistocene humans. This furthers the debate on how and why modern humans expanded into Western Eurasia despite Neandertal occupation. Incorporating a biocultural paradigm to the interpretation of the resultant data allows for new explanations of both modern human ascendancy and diversity. Past studies have found little difference between early modern humans and Neandertals for some variables previously thought to differentiate them (e.g., diet: Richards and Trinkaus, 2009; Henry et al., 2011; mortality: Trinkaus, 2011), and this study provides a new example of how modern humans ecologically out-competed Neandertals with decreased morbidity as represented by oral health.

A cross-cultural perspective permits these paleontological results to be taken beyond human origins studies by establishing an pre-agricultural oral health baseline before the widespread (known) use of oral inhalants and intoxicants (e.g., tobacco, betel-nut, coca), and comparing it with oral health statistics in recent groups. The Holocene samples included here also present another test of the applicability of "recent" hunter-gatherers as comparative models. The high prevalence of caries and periodontal disease in some comparative samples contrast strongly with studies of recent hunter-gatherer groups showing caries and periodontal disease to be relatively uncommon (e.g., Wells, 1975; Caselitz, 1998). Oral health and morbidity in the Late Pleistocene has implications beyond paleoanthropological debates and can elucidate differential health and its effects on modern cultural and physiological diversity for the broader health and social science community.

Assessing oral health in Holocene and Late Pleistocene hunter-gatherers gives a general global health evaluation from which to compare health levels in non-industrial populations

today. Oral health is correlated with overall health and is especially important for maternal and childhood health issues, i.e., periodontal disease is associated with low fetal birth weight and preterm birth, childhood oral infections can adversely affect the eruption of the permanent dentition, etc., and maternal and childhood health issues are a major focus in global public health appraisals. The Late Pleistocene sample takes this analysis of pre-industrial oral health deeper into human history. These comparisons are important for dentists worldwide to provide a baseline of oral pathology prevalences in humans, especially for populations eating non-industrial diets. Caries and periodontal disease affliction rates increase with decreasing socio-economic status (Hobdell et al., 2003). A prehistoric standard provides perhaps a better comparison for low socioeconomic status individuals in developing countries besides using dentistry studies of high socioeconomic status individuals in developed countries as the assumed baseline for "normal" oral health.

This project's results and conclusions also further evaluate the popular "Paleo-fantasy" movement in Western popular culture, which fetishizes the supposedly superior health and diet of Pleistocene peoples (Zuk, 2013). Besides the gross misinterpretation of the dietary habits of Paleolithic humans by this movement, this study shows that Pleistocene individuals were not the paragons of health to which one should aspire. Well-occluded teeth do not define good oral health.

Furthermore oral health has already been debated within the developing field of evolutionary medicine, but only as a hypothetical in respect to the Pleistocene (Williams and Nesse, 1991). This perspective advocates viewing oral health within the lens of the interaction of multiple organisms (humans and their oral flora) over evolutionary time and selection acting on the variation in the immunological responses of individuals to physiological and external forces

(see Supplement A in Lukacs (2008) for a summary). This thesis provides concrete data with which to test the hypotheses generated within evolutionary medicine. A study of human oral health over the last 120,000 years therefore has global implications for understanding current human oral pathology and general health for those addressing it from either an academic or clinical perspective.

Conclusion:

Caries, periodontal disease, oral lesions, and tooth loss are not only a consequence of an agricultural diet. The so-called "original affluent society" (Sahlins, 1968), Pleistocene hunter-gatherers, also suffered from oral diseases, including pathologies that affected systemic health, chewing efficiency, and the comfort of the individual. However these pathologies' prevalence in the human population were not consistent; they shifted over time and geography in response to changes in population structure, behavior, and environment, along with other previously studied indicators of health and stress. A contributing factor of the demographic success of modern humans was their improved health relative to Neandertals (less periodontal disease and tooth loss, but also less dental enamel hypoplasias, *cribia orbitalia*, Harris lines, etc. (Brennan, 1991; Formicola and Holt, 2008; Trinkaus, 2013)); however the cause of this improved health—better hunting technology, demographics, subsistence strategy—has been explored elsewhere. Modern humans were also not static, and as the environment of Europe changed rapidly in response to the Last Glacial Maximum, human populations changed too. This is reflected in increasing caries prevalence in the Late Upper Paleolithic from shifting diets, and increasing antemortem tooth loss prevalence, perhaps from social cohesion alleviating the mortality risk associated with tooth loss. This survey of oral health elucidates the underlying shifting patterns of diet and behavior in the Late Pleistocene and their effect on the health of the individual and their population.

Ultimately poor oral health is a warning indicator for suboptimal diet and declining systemic health, and my hope is that this study provides a new insight into the demographic competition amongst human groups over the last 120,000 years in Western Eurasia.

References:

Abbott PV. 2004. Classification, diagnosis, and clinical manifestations of apical periodontitis. Endodont Top 8:36.

Adatia A. 1975. Dental caries and periodontal disease. In: DP Burkitt. HC Trowell, editors. Refined Carbohydrates Foods and Disease. London: Academic. p 251-277.

Adler DS, Bar-Oz G, Belfer-Cohen A, Bar-Yosef O. 2006. Ahead of the game. Middle and Upper Palaeolithic hunting behaviors in the southern Caucasus. Curr Anthropol 47:89-118.

Adler CJ, Dobney K, Weyrich LS, Kaidonis J, Walker AW, Haak W, Bradshaw CJ, Townsend G, Sołtysiak A, Alt KW, et al. 2013. Sequencing ancient calcified dental plaque shows changes in oral microbiota with dietary shifts of the Neolithic and Industrial revolutions. Nat. Genet. 45:450–455

Al-Shammari K, Al-Khabbaz AK, Al-Ansari JM, Neiva R, and Wang H-L. 2005. Risk indicators for tooth loss due to periodontal disease. J Periodontol 76:1910-1918.

Albandar JM, Streckfus CF, Adesanya MR, Winn DM. 2000. Cigar, pipe, and cigarette smoking as risk factors for periodontal disease and tooth loss. J Periodontol 71:1874-1881.

Albert RM, Lavi O, Estroff L, Weiner S. 1999. Mode of occupation of Tabun Cave, Mt. Carmel, Israel during the Mousterian period: a study of the sediments and phytoliths. J Archaeol Sci 26:1249-1260.

AlQahtani SJ, Hector MP, Liversidge HM. 2010. Brief Communication: The London Atlas of Human Tooth Development and Eruption. Am J Phys Anthropol 142:481-490.

Alt KW, Pichler S, Vach W, Klíma B, Vlček E, Sedlmeier J. 1997. Twenty-five thousand-year-old triple burial from Dolní Věstonice: An ice-age family? Am J Phys Anthropol 102:123-131.

Ambrose, SH. 2001. Paleolithic technology and human evolution." Science 291:1748-1753.

Anderson JE. 1968. Late Paleolithic skeletal remains from Nubia. Nubia Prehistory 2:996-1040.

Ånerud ÅG, Löe H, Boysen H, Smith M. 1979. The natural history of periodontal disease in man." J Periodont Res 14:526-540.

Angel JL. 1944. A racial analysis of the ancient Greeks: An essay on use of morphological types. Am J Phys Anthropol 2:329-376.

Angel JL. 1981. History and development of paleopathology. Am J Physical Anthropol 56:509-515.

Aranguren B, Becattini R, Lippi MM, Revedin A. 2007. Grinding flour in Upper Paleolithic Europe (25 000 years bp). Antiquity 81:845–855.

Arensburg B. 1996. Ancient dental calculus and diet. Hum Evol 11:139-145.

Arensburg B, Bar-Yosef O. 1973. Human remains from Ein Gev I, Jordan Valley, Israel. Paléorient 201-206.

Armitage GC. 2004. Periodontal diagnoses and classification of periodontal diseases. Periodontol 2000 34:9-21.

Astuti P. 2002. Due nuovi ciottoli dipinti rinvenuti nei livelli epigravettiani e mesolitici di Grotta Continenza: nota preliminare. Rivista di scienze preistoriche 52:321-325.

Bagramian R, Garcia-Godoy F, Volpe AR. 2009. The global increase in dental caries. A pending public health crisis. Am J Dent 22:3–8.

Bahrami G, Vaeth M, Kirkevang LL, Wenzel A, Isidor F. 2008. Risk factors for tooth loss in an adult population: a radiographic study. J Clin Periodontol 35:1059-65.
Baker JL, Olsen LW, Sørensen TIA. 2008. Weight at birth and all-cause mortality in adulthood. Epidemiol 19:197-203.
Bang G. 1964. A comparison between the incidence of dental caries in typical coastal populations and inland populations with particular regard to the possible effect of a high intake of salt water fish. Odont. Tidskr 72:12-43.
Bar-Yosef O. 1992. The role of western Asia in modern human origins. Phil Transact Royal Soc London B Bio Sci 337:193-200.
Bar-Yosef, O. 1994. The contributions of Southwest Asia to the study of the origin of modern humans. In: Nitecki NH, Nitecki DV, editors. Origins of anatomically modern humans. Springer US p 23-66
Bar-Yosef O. 2002. The upper paleolithic revolution. Ann Rev Anthropol 2002:363-393.
Bar-Yosef O. 2004. Eat what is there: Hunting and gathering in the world of Neanderthals and their neighbours. Intl J Osteoarchaeol 14:333-342.
Bar-Yosef O. 2007. The archaeological framework of the Upper Paleolithic revolution. Diogenes 54:3-18.
Bar-Yosef O, Kuhn SL. 1999. The big deal about blades: laminar technologies and human evolution Am Anthropologist 101:322-338.
Bardsley PF. 2008. The evolution of tooth wear indices. Clin Oral Investigat 12:15-19.
Barrett CK, Guatelli-Steinberg D, Sciulli PW. 2012. Revisiting dental fluctuating asymmetry in neandertals and modern humans. Am J Phys Anthropol 149:193-204.
Barroso Ruíz C, de Lumley H. 2006. La Grotte du Bocquete de Zafarraya. Consejería de Cultura, Sevilla.
Barton NE. 2000. Mousterian hearths and shellfish: late Neanderthal activities on Gibraltar. In: Stringer CBE, Barton NE, Finlayson C, editors. Neanderthals on the Edge. Oxbow Books
Bartsiokas A, Day DH, 1993. Lead poisoning and dental caries in the Broken Hill hominid. J Hum Evol 24:243-249.
Begg PR. 1954. Stone Age man's dentition. Am J Orthodon 40: 298-312.
Belfer-Cohen A, Davidzon A, Goring-Morris AN, Lieberman D, Spiers M. 2004. Nahal ein gev I: A late upper palaeolithic site by the sea of Galilee, Israel. Paléorient 25-45.
Bender IB, Seltzer S. 1972. The effect of periodontal disease on the pulp." Oral Surg Oral Med Oral Pathol 33:458-474.
Berger TD, Trinkaus E. 1995. Patterns of trauma among the Neandertals. J Archaeol Sci *22*:841-852.
Bergström J. 1989. Cigarette smoking as risk factor in chronic periodontal disease. Communit Denti Oral Epidemiol 17:245-247.
Bergström J. 2004. Tobacco smoking and chronic destructive periodontal disease. Odontol 92:1-8.
Bietti A, Manzi G. 1991. Proposal for a new denomination of the Monte Circeo hominids. In: Bietti A, Manzi G (eds). The Fossil Man of Monte Circeo. Fifty Years of Studies on the Neandertals in Latium. Quaternaria Nova 1 (proceedings of the homonymous symposium; Sabaudia, October 1989) pp 47-48.
Bocherens H, Billou D, Mariotti A, Toussaint M, Patou-Mathis M, Bonjean D, Otte M. 2001. New Isotopic evidence from dietary habits of Neandertals from Belgium. J Hum Evol

40:497-505.
Bocherens H. 2009. Neanderthal dietary habits: review of the isotopic evidence. In: Hublin JJ, Richards MP, editors. The evolution of hominin diets Springer Netherlands, p 241-250.
Bocquentin F. 2011. Dental Extractions and Regional Identity in Natufians. Tuba-Ar-Turkish Acad Sci J Archaeol 14:261-270.
Bocquet-Appel JP, Degioanni A. 2013. Neanderthal demographic estimates. Curr Anthropol 54:S202-S213.
Bocquet-Appel JP, Demars PY, Noiret L, Dobrowsky D. 2005. Estimates of Upper Palaeolithic meta-population size in Europe from archaeological data. J Archaeol Sci, 32: 1656-1668.
Bonsall C, Lennon R, McSweeney K, Stewart C, Harkness D, Boronean V, Bartosiewicz L, Payton R, Chapman J. 1997. Mesolithic and Early Neolithic in the Iron Gates: A Paiaeodietary Perspective. J Eur Archaeol 5:50-92.
Borgognini Tarli SM, Fornaciari G, Palma di Cesnola A. 1980. Restes humains des niveaux gravettiens de la Grotte Paglicci (Rignano Garganico) : contexte archéologique, étude anthropologique et notes de paléopathologie [Italian] Bull Memoir Societ d'Anthropol Paris. 7:125-152.
Borgognini Tarli S. Repetto E. 1985. Diet, dental features and oral pathology in the Mesolithic samples from Uzzo and Molara caves (Sicily). Papers in Italian Archaeology. Oxford: British Archaeological Reports (International Series) p 245.
Bosinsky G. 1990. Les civilisations de la préhistoire: Les chasseurs du Paléolithique Supérior. Paris: Masson et C.
Brabant H. 1967. Paleostomatology. In: Brothwel DR, Sandison AT, editors. Disease in Antiquity. Springfield: CC Thomas, p 538-550.
Brabant H, Brabant H. 1962. Contribution à l'étude de la paleopathology des dents et des maxillairse. La denture en Belgique à l'époque néolithique. Bull Inst Res Sci Nat Belg 38:1-32.
Brabant H, Twiesselmann H. 1964. Observations sur l'evolution de la denture permanente en Europe Occidentale. Bull Groupement Int Rech Scient Stomatologie 7:11-84.
Brace CL. 1964. The probable mutation effect. Am Naturalist 98:453-455.
Brace CL, Rosenberg KR, Hunt KD. 1987. Gradual change in human tooth size in the late Pleistocene and post-Pleistocene. Evol 705-720.
Brennan MU. 1991. Health and disease in the Middle and Upper Paleolithic of Southwestern France: A bioarchaeological study. PhD Thesis. New York University.
Bridges PS. 1991. Degenerative joint disease in hunter–gatherers and agriculturalists from the southeastern United States. Am J Phys Anthropol 85:379-391.
Broderick AH. 1948. Early Man: a Survey of Human Origins. London: Hutchinson.
Brothwell, DR. 1959. Teeth in earlier human populations. Proc Nutri Soc 18: 59-65.
Brothwell DR. 1963. The macroscopic dental pathology of some earlier human populations. In: Brothwell DR, editor. Dental Anthropology. New York: MacMillan, p 271-288.
Brothwell DR. 1972. Digging up bones. London: Trustees of the British Museum.
Brothwell DR, Carbonell VM, Goose DH. 1963. Congenital absence of teeth in human populations. In: Brothwell DR, Ed. Dental Anthropology. Oxford: Pergamon Press, p 179-190.
Brown LJ, Löe H. 1993. Prevalence, extent, severity and progression of periodontal disease.

Periodontol 2000 2:57-71.
Brown P, Sutikna T, Morwood MJ, Soejono RP. 2004. A new small-bodied hominin from the Late Pleistocene of Flores, Indonesia. Nature 431:1055-1061.
Brunet M, Fronty P, Sapanet M, de Bonis L, Viriot L. 2002. Enamel hypoplasia in a pliocene hominid from Chad." Connective Tiss Res 43:94-97.
Brunt R, Vasak L, Griffioen J. 2004. Fluoride in groundwater: probability of occurrence of excessive concentration on global scale. IGRAC.
Buikstra JE, Cook DC. 1980. Palaeopathology: an American account. Ann Rev Anthropol 9:433-470.
Buikstra JE, Ubelaker DH, eds. 1994. Standards for data collection from human skeletal remains. Fayetteville: Arkansas Archaeological Survey.
Bullen JJ. 1981. The significance of iron in infection. Rev Infect Dis 3:1127–1138.
Buzhilova AP. 2000. The analysis of anomalies and indicators of physiological stress in non-mature Sunghir individuals (in with English summary). In: Alexeeva TI, Bader NO, Munchaev RM, Buzhilova AP, Kozlovskaya MV, Mednikova MB, editors. Homo Sungirensis. Upper Palaeolithic Man: ecological and evolutionary aspects of the investigation. Moscow: Scientific World. p 302-314.
Cachel S. 1997. Dietary Shifts and the European Upper Paleolithic Transition. Curr Anthropol 38:579-603.
Caglar E, Kusca OO, Sandalli N, Ari I. 2007. Prevalence of dental caries from tooth wear in a Byzantine population (13th c. A.D.) from northwest Turkey. Archs Oral Biol 52:1136-1145.
Calcagno J, Gibson K. 1991. Selective Compromise: evolutionary trends and mechanisms in hominid tooth size. In: Kelley MA, Larsen CS, editors. Advances in dental anthropology. New York: Wiley Liss. p 59-76.
Carr HG. 1960. Some Dental Characteristics of the Middle Minoans. Man 60:119-122.
Carter JT. 1928. The Teeth of Rhodesian Man. In: Pycraft WP, Smith GE, Yearsley M, Carter JT, Smith RA, Hopwood AT, Bate DMA, Swinton WE. Rhodesian Man and Associated Remains. London: British Museum (Natural History), p 64-65.
Caselitz, P. 1998. Caries-Ancient Plague of Humankind. In: Alt, K.W., Rösing, F.W. & Teschler-Nicova, M. (eds.) Dental Anthropology. Fundamentals, Limits, and Prospects. New York: Springer Verlag, p 203-226.
Caspari R, Lee SH. 2004. Older age becomes common late in human evolution. Proc Nat Acad Sci 101:10895-10900.
Cassidy, CM. 1972. A comparison of nutrition and health in pre-agricultural and agricultural Amerindian skeletal populations. PhD dissertation. University of Wisconsin-Madison.
Chaves MM, Pereira JS, Maroco J, Rodrigues ML, Pereira Pinto Ricardo C, Osorio ML, Carvalho I, Faria T, Pinheiro C. 2002. How plants cope with water stress in the field Photosynthesis and growth. Annals Botany 89:907-916.
Churchill SE. 1998. Cold adaptation, heterochrony, and Neandertals." Evol Anthropol 7: 46-60.
Clarke NG. 1990. Periodontal defects of pulpal origin: evidence in early man. Am J Phys Anthropol 82:371-376.
Clarke NG, Hirsch RS. 1991. Physiological, Pulpal, and Periodontal Factors Influencing Alveolar Bone. In: Kelley MA, Larsen CS, editors. Advances in Dental Anthropology. New York: Wiley-Liss p 13-31.

Clarke NG, Carey SE, Srikandi W, Hirsch RS, Leppard PI. 1986. Periodontal disease in ancient populations. Am J Phys Anthropol 71:173-183.
Clement AJ. 1956. Caries in the South African ape-man: some examples of undoubted pathological authenticity believed to be 800,000 years old. Brit Dent J 101:4-7.
Cohen MN, Armelagos GJ, editors. 1984. Paleopathology at the Origins of Agriculture. New York: Academic Press
Cohen D, Friedman L, Shapiro J, Kyle G. 1969. A longitudinal investigation of the periodontal changes during pregnancy. J Periodontol 40:563-570.
Colman, E. 1998. Obesity in the Paleolithic era? The Venus of Willendorf. Endocr Prac 4:58-59.
Commission on Oral Health, Research, and Epidemiology. 1982. An epidemiological index of developmental defects of dental enamel (DDE Index). Internat Dent J 32:159-167.
Conard, NJ, ed. 2006. When Neanderthals and modern humans met. Tübingen: Kerns.
Condemi S, Tardivo D, Foti B, Ricci S, Giunti P, Longo L. 2012. A case of an osteolytic lesion on an Italian Neanderthal jaw. Comptes Rendus Palevol, 11:79-83.
Cook DC. 1981. Koniag Eskimo tooth ablation: was Hrdlicka right after all? Curr Anthropol 159-163.
Copeland LB, Krall EA, Brown LJ, Garcia RI, Streckfus CF. 2004. Predictors of tooth loss in two US adult populations. J Pub Health Dent 64:31-37.
Cordain, L., Eaton, S.B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B.A., O'Keefe, J.H., Brand-Miller, J., 2005. Origins and evolution of the Western diet: health implications for the 21st century. Am. J. Clin. Nutri. 81, 341-354.
Corruccini RS, Jacobi KP, Handler JS, Aufderheide AC. 1987. Implications of tooth root hypercementosis in a Barbados slave skeletal collection. Am J Phys Anthropol 74:179-84.
Costa RL. 1977. Dental Pathology and related factors in archeological Eskimo skeletal samples from Point Hope and Kodiak Island, Alaska. Doctoral Dissertation, University of Pennsylvania.
Costa RL. 1980a. Age, sex, and antemortem loss of teeth in prehistoric Eskimo samples from Point Hope and Kodiak Island, Alaska. Am J Phys Anthropol 53:579-587.
Costa RL. 1980b. Incidence of caries and abscesses in archeological Eskimo skeletal samples from Point Hope and Kodiak Island, Alaska. Am J Phys Anthropol 52:501-514.
Costa RL. 1982. Periodontal disease in the prehistoric Ipiutak and Tigara skeletal remains from Point Hope, Alaska. Am J Phys Anthropol 59:97-100.
Cucina A, Tiesler V. 2003. Dental caries and antemortem tooth loss in the Northern Peten area, Mexico: a biocultural perspective on social status differences among the Classic Maya. Am J Phys Anthropol. 122:1–10.
Cucina A, Vargiu R, Mancinelli D, Ricci R, Santandrea E, Catalano P, Coppa A. 2006. The necropolis of Vallerano (Rome, 2nd–3rd century AD): an anthropological perspective on the ancient Romans in the Suburbium. Int J Osteoarchaeol. 16:104–117.
Cucina A, Cantillo CP, Sosa TS, Tiesler V. 2011. Carious lesions and Maize Consumption Among the Prehistoric Maya: An Analysis of a Coastal Community in Northern Yucatan. Amer J Phys Anthropol 145:560-567.
Cullinan MP, Ford PJ, Seymour GJ. 2009. Periodontal disease and systemic health: current status. Aust Dent J Supp 54:S62–9.
d'Errico F, Sánchez Goñi MF. 2003. Neandertal extinction and the millennial scale climatic

variability of OIS 3. Quatern Sci Rev 22:769-788.
d'Errico F, Banks WE, Vanhaeren M, Laroulandie V, Langlais M. 2011. PACEA geo-referenced radiocarbon database. PaleoAnthropol 2011:1-12.
Dabbs, GR. 2009. Health and Nutrition at Prehistoric Point Hope, Alaska: Application and Critique of the Western Hemisphere Health Index. Doctoral Dissertation, University of Michigan.
Daifuku H. 1952. The pit house in the old world and in native North America. Am Antiq 18:1-7.
Dams LR. 1978. Bees and honey collecting scenes in the Mesolithic rock art of Eastern Spain. Bee World 59:45-53.
Dams M, Dams LR. 1977. Spanish rock art depicting honey gathering during the Mesolithic. Nature 268:228-230.
Danenberg PJ, Hirsch RS, Clarke NG, Leppard PI, Richards LC. 1991. Continuous tooth eruption in Australian aboriginal skulls. Am J Phys Anthropol 85:305-312.
Dastugue J. 1967. Pathologie des hommes fossiles de l'Abri de Cro-Magnon. Anthropologie 71:479-492.
Dawson H, Brown KR. 2013. Exploring the relationship between dental wear and status in late medieval subadults from England. Am J Phys Anthropol. 150:433–41.
Dean MC, Stringer CB, Bromage TG. 1986. Age at death of the Neanderthal child from Devil's Tower, Gibraltar and the implications for studies of general growth and development in Neanderthals." Am J Phys Anthropol 70:301-309.
De Castro JMB. 1989. Third molar agenesis in human prehistoric populations of the Canary Islands. Am J Phys Anthropol 79:207-215.
Degusta D. 2002. Comparative skeletal pathology and the case for conspecific care in Middle Pleistocene hominids. J Archaeol Sci 29:1435-1438.
DeGusta D. 2003. Aubesier 11 is not evidence of Neanderthal conspecific care. J Hum Evol 45:91-94.
Delgado-Darias T, Velasco-Vázquez J, Arnay-de-la-Rosa M, Martín-Rodríguez E, González-Reimers E. 2006. Calculus, periodontal disease and tooth decay among the prehispanic population from Gran Canaria. J Archaeol Sci. 33:663–670.
Dennell, R.W., 1983. European Economic Prehistory: A New Approach. London and New York: Academic Press.
Dennell RW, Martinón-Torres M, Bermúdez de Castro JM. 2011. Hominin variability, climatic instability and population demography in Middle Pleistocene Europe. Quatern Sci Rev 30:1511-1524.
Dentamerica Inc. 18320 Beford Circle, Industry, CA 91744 USA
DeStefano F, Anda RF, Kahn HS, Williamson DF, Russell CM. 1993. Dental disease and risk of coronary heart disease and mortality. Brit Med J 306:688.
Dettwyler KA. 1991. Can paleopathology provide evidence for "compassion"? Amer J Phys Anthropol 84:375-384.
DeWitte SN, Bekvalac J. 2010. Oral health and frailty in the medieval English cemetery of St Mary Graces. Am J Phys Anthropol. 142:341–54.
Dewitte SN, Bekvalac J. 2011. The association between periodontal disease and periosteal lesions in the St. Mary Graces cemetery, London, England A.D. 1350-1538. Am J Phys Anthropol. 146:609–18.
Dias G, Tayles N. 1997. Absess cavity: a misnomer. Int J Osteoarchaeol 7:548-554

Doboş A, Soficaru A., Trinkaus E. 2010. The Prehistory and Paleontology of the Pestera Muierii, Romania. Études et Recherches Archéologiques de l'Université de Liège 124:1–122.
Dolan TA, Gooch BF, Bourque BL. 1991. Associations of self-reported dental health and general health measures in the Rand Health Insurance Experiment. Commun Dentr Oral Epidem 19:1-8.
Donn WL, Farrand WR, Ewing M. 1962. Pleistocene ice volumes and sea-level lowering. J Geol. 1962:206-214.
Dowd FJ. 1999. Saliva and dental caries. Dent Clinics North Am 43:579.
Drucker D, Bocherens H. 2004. Carbon and nitrogen stable isotopes as tracers of change in diet breadth during Middle and Upper Palaeolithic in Europe. Int J Osteoarchaeol 14:162-177.
Drucker D, Bocherens H, Bridault A, Billiou D. 2003. Carbon and nitrogen isotopic composition of red deer (*Cervus elaphus*) collagen as a tool for tracking palaeoenvironmental change during the Late-Glacial and Early Holocene in the northern Jura (France). Palaeogeo, Palaeoclimatol, Palaeoecol 195:375-388.
Dunning JM. 1953. The influence of latitude and distance from seacoast on dental disease. J Dent Res 32:811–829.
Dunsworth HM. 2007. Human Origins 101. Los Angelos: ABC-CLIO.
Duray SM. 1992. Enamel defects and caries etiology: an historical perspective. J Paleopathol Monographic Publications 2:307-319.
Dye BA, Thornton-Evans G. 2010. Trends in oral health by poverty status as measured by Healthy People 2010 objectives. Pub health Reports, 125:817.
Dye BA, Arevalo O, Vargas CM. 2010. Trends in paediatric dental caries by poverty status in the United States, 1988–1994 and 1999–2004. Int J Paediat Dentr, 20:132-143.
Eke PI, Dye BA, Wei L, Thornton-Evans GO, Genco RJ. 2012. Prevalence of Periodontitis in Adults in the United States: 2009 and 2010. J Dent Res 91:914-920.
Eklund SA, Burt BA. 1994. Risk factors for total tooth loss in the United States; longitudinal analysis of national data. J Pub Health Dentis 54: 5-14.
El Zaatari S, Grine F, Ungar P, Hublin JJ. 2011. Ecogeographic variation in Neandertal dietary habits: evidence from occlusal molar microwear texture analysis. J Hum Evol. 61:411–424.
Eshed V, Gopher A, Gage TB, Hershkovitz I. 2004. Has the transition to agriculture reshaped the demographic structure of prehistoric populations? New evidence from the Levant. Am J Phys Anthropol 124:315-329.
Eshed V, Gopher A, Hershkovitz I. 2006. Tooth wear and Dental Pathology at the Advent of Agriculture: New Evidence from the Levant. Am J Phys Anthropol 130:145-159.
Eshed V, Gopher A, Pinhasi R, Hershkovitz I. 2010. Paleopathology and the origin of agriculture in the Levant. Am J Phys Anthropol 143:121-133.
Evans RW, Lo E. 1992. Effects of School Dental Care Service in Hong Kong-primary teeth. Commun Dent Oral Epidemiol 20:193-195.
Fabbri,PF, Mallegni F. 1988. Dental anthropology of the Upper Palaeolithic remains from Romito cave at Papasidero (Cosenza, Italy)/ Anthropologie dentaire du Paléolithique supérieur, Grotte del Romito à Papasidero (Cosenza, Italie). Bulletins et mémoires de la Société d'anthropologie de Paris 5:163-177.
Farmer P, Kim JY, Kleinman A, Basilico M. 2013. Reimagining global health. An

introduction. Berkeley: University of California Press.
Fields M, Herschaft EE, Martin DL, Watson JT. 2009. Sex and the agricultural transition: Dental health of early farming females. J Denti Oral Hygiene 1:42-51.
Finlayson, C. (2008). On the importance of coastal areas in the survival of Neanderthal populations during the Late Pleistocene. *Quaternary Science Reviews*, *27*(23), 2246-2252.
Finlayson C, Brown K, Blasco R, Rosell J, Negro JJ, Bortolotti GR, Finlayson G. 2012. Birds of a feather: Neanderthal exploitation of raptors and corvids. PloS ONE. 7: e45927.
Fiorenza L, Benazzi S, Tausch J, Kullmer O, Bromage T, Schrenk F. 2011. Molar Macrowear Reveals Neanderthal Eco-Geographic Dietary Variation. PLoS ONE. 6:e14769
Formicola V. 1988. Interproximal grooving of teeth: additional evidence and interpretation. Curr Anthropol 29:663-671.
Formicola V. 1995. X-linked hypophosphatemic rickets: A probable upper paleolithic case. Am J Phys Anthropol 98:403-409.
Formicola V. 2007. From the Sunghir Children to the Romito Dwarf. Curr Anthropol 48:446-453.
Formicola V, Holt BM. 2007. Resource availability and stature decrease in Upper Palaeolithic Europe. J Anthropol Sci 85:147-155.
Formicola V, Holt BM, 2008. I Resti Umani del Paleolitico Superiore della Liguria Occidentale: Una Sintesi sui Risultati degli Studi Recenti. Bulletin du Musée d'anthropologie préhistorique de Monaco. 2008: 41-48.
Formicola V, Repetto E. 1989. The dentition of the Cro-Magnon type (Grotte des Enfants 4). Bull Mus Anthropol Préhist Monaco 3251-62.
Forshaw RJ. 2009. The practice of dentistry in ancient Egypt. Brit Dent J 206:481-486.
Foucher P, San Juan C, Valladas H, Clottes J, Bégouën R, Giraud JP. 2002. De nouvelles dates 14C pour le gravettien des Pyrénées. Bulletin de la Société Préhistorique Ariège-Pyrénées 56:35-44.
Franciscus RG, Churchill SE. 2002. The costal skeleton of Shanidar 3 and a reappraisal of Neandertal thoracic morphology. J Hum Evol 43:303-356.
Frayer DW. 1978. The evolution of the dentition in Upper Paleolithic and Mesolithic Europe. University of Kansas: University of Kansas Publications in Anthropology.
Frayer DW. 1980. Sexual dimorphism and cultural evolution in the late Pleistocene and Holocene of Europe. J Hum Evol 9:399-415.
Frayer DW. 1981. Body size, weapon size, and natural selection in the European Upper Paleolithic and Mesolithic. Am Anthropol 8:57-73.
Frayer DW. 1989. Oral pathologies in the European Upper Paleolithic and Mesolithic. In: Hershkovitz I ed. People and Culture in Change: Proceedings of the Second Symposium on Upper Palaeolithic, Mesolithic and Neolithic Populations of Europe and the Mediterranean Basin. 255-282.
Fujita H. 2012. Periodontal Diseases in Anthropology. In: Manaki K, editor. Periodontal Diseases–A Clinician's Guide p 279-294.
Galloway JH. 2000. Sugar. In: Kiple KF, Ornelas KC, editors. The Cambridge world history of food. Vol 1. Cambridge: Cambridge University Press p 437– 49.
Gambier D, Valladas H, Tisnérat-Laborde N, Arnold M, Bresson F. 2000. Datation de

vestiges humains présumés du Paléolithique supérieur par la méthod du Carbone 14 en spectrométrie de masse par accélétateur (Accelerator mass spectrometry radiocarbon dates of human remains from Upper Paleolithic). Paleo 12:2010-2212.
Gamble C. 1986. The Paleolithic settlement of Europe. Cambridge: Cambridge University Press.
Garcia RI, Henshaw MM, and Krall EA. 2001. Relationship between periodontal disease and systemic health. Periodontol. 2000 25:21–36.
Garcia MN, Hildebolt CF, Miley DD, Dixon DA, Couture RA, Anderson Spearie CL, Langenwalter EM, Shannon WD, Deych E, Mueller C, Civitelli R. 2011. One-year effects of vitamin D and calcium supplementation on chronic periodontitis. J Periodontol 82:25-32.
Garn SM, Lewis AB, Kerewsky RS. 1963. Third molar agenesis and size reduction of the remaining teeth. Nature 200, 488 – 489.
Gendron R, Grenier D, Maheu-Robert L. 2000. The oral cavity as a reservoir of bacterial pathogens for focal infections. Microbes Infect. 2:897–906.
Gibson KR, Calcagno JM. 1989. A critique of the «increasing population density effect». Hum Evol 4:515-523.
Gibson KR, Calcagno JM. 1993. Brief communication: possible third molar impactions in the hominid fossil record. Am J Phys Anthropol 91:517-521.
Gift H, Atchison K. 1995. Oral Health , Health , and Health-Related Quality of Life. Med. Care 33: NS57-NS77.
Gilead I. 1988. The Upper Palaeolithic to Epi-Palaeolithic transition in the Levant. Paléorient, 1988:177-182.
Gilmore C. 2011. Does antemortem tooth loss in Homo neanderthalensis indicate human-like behavior? Evidence from a comparative approach. "Abstracts of the PaleoAnthropology Society 2011 Meetings." PaleoAnthropology 2011: A13.
Gilmore CC. 2013. A comparison of antemortem tooth loss in human hunter-gatherers and non-human catarrhines: Implications for the identification of behavioral evolution in the human fossil record. Am J Phys Anthropol 151:252-264.
Gilmore CC. n.d. A comparative framework for the interpretation of antemortem tooth loss in Neandertals.
Glass GB. 1991. Continuous eruption and periodontal status in pre-industrial dentitions. Int J Osteoarchaeol 1:265-271.
Gluckman PD, Beedle A, Hanson MA. 2009. Principles of evolutionary medicine. Oxford: Oxford University Press.
Gold SI. 1985. Periodontics: The Past. J Clin Periodont 12:79-97.
Goldman HM, Schilder H. 1988. Regeneration of attachment apparatus lost due to disease of endodontic origin. J Periodont, 59:609-610.
Goldman HM, Isenberg G, Shuman A. 1976. The gingival autograft and gingivectomy. J Periodont 47:586-589.
Goodman AH, Armelagos GJ. 1988. Childhood stress and decreased longevity in a prehistoric population. Am Anthropol 90:936-944.
Goodman AH, Armelagos GJ. 1989. Infant and childhood morbidity and mortality risks in archaeological populations. World Arch 21:225-243.
Goring-Morris AN. 2002. Epipaleolithic. In: Peregrin PN, Ember M, Eds. Encyclopedia of Prehistory. New York: Facts on File pp 122-126.

Gravina B, Mellars P, Ramsey CB. 2005. Radiocarbon dating of interstratified Neanderthal and early modern human occupations at the Chatelperronian type-site. Nature 438:51-56.
Grayson DK, Delpech F. 2002. Specialized early Upper Palaeolithic hunters in southwestern France? J Archaeol Sci 29:1439-1449.
Grayson DK, Delpech F. 2003. Ungulates and the Middle-to-Upper Paleolithic transition at Grotte XVI (Dordogne, France). J Archaeol Sci 30:1633-1648.
Grayson DK, Delpech F. 2006. Was there increasing dietary specialization across the Middle-to-Upper Paleolithic transition in France. In: Conard N, editor. When Neanderthals and modern humans met Tübingen: Kerns p 377-417.
Grine FE, Gwinnett AJ, Oaks JH. 1990. Early hominid dental pathology: Interproximal caries in 1.5 million-year-old *Paranthropus robustus* from Swartkrans. Archs Oral Bio 35:381-386.
Guatelli-Steinberg D. 2004. Analysis and significance of linear enamel hypoplasia in Plio-Pleistocene hominins. Am J Phys Anthropol 123:199-215.
Guatelli-Steinberg D, Larsen CS, Hutchinson DL. 2004. Prevalence and the duration of linear enamel hypoplasia: A comparative study of Neandertals and Inuit foragers. J Hum Evol 47:65-84.
Guatelli-Steinberg D, Buzhilova AP, Trinkaus E. 2011. Developmental Stress and Survival among the Mid Upper Paleolithic Sunghir Children: Dental Enamel Hypoplasias of Sunghir 2 and 3. Am J Phys Anthropol 23:421-431.
Halcrow SE, Harris NJ, Tayles N, Ikehara-Quebral R, Pietrusewsky M. 2013. From the mouths of babes: Dental caries in infants and children and the intensification of agriculture in mainland Southeast Asia. Am J Phys Anthropol. 150:409–20.
Hardy BL. 2010. Climatic variability and plant food distribution in Pleistocene Europe: Implications for Neandertal diet and subsistence. Quatnernary Sci 29:662-679.
Hardy BL, Moncel MH. 2011. Neandertal Use of Fish, Mammals, Birds, Starchy Plants and Wood 125-250,000 Years Ago. PLoS ONE 6:8.
Hardy BL, Kay M Marks AE, Monigal K. 2001.Stone tool function at the paleolithic sites of Starosele and Buran Kaya III, Crimea: Behavioral implications. Proc Nat Acad Sci 98: 10972-10977.
Hardy K, Buckley S, Collins MJ, Estalrrich A, Brothwell D, Copeland L, García-Tabernero A, Garcia-Vargas S, de la Rasilla M, Lalueza-Fox C, Huhuet R, Bastir M, Sanramaria D, Madella M, Wilson J, Fernandez Cortes A, Rosas A. 2012. Neanderthal medics? Evidence for food, cooking, and medicinal plants entrapped in dental calculus. Naturwissenschaften 99:617-626.
Heim JL, Granat J. 1995. La mandibule de l'enfant néandertalien de Malarnaud (Ariége). Une nouvelle approche anthropologique par la radiographie et la tomodensitométrie. Anthropol Prehist. 106:79–96.
Heim JL. 1976. Les hommes fossiles de La Ferrassie. Arch Inst Paleontol Hum. 35:1-331.
Henke W. 1986. Die magdalénienzeitlichen Menschenfunde von Oberkassel bei Bonn. Bonner Jahrbücher 186:317-366.
Hennig BJ, Parkhill JM, Chapple LL, Heasman PA, Taylor JJ. 1999. Association of a vitamin D receptor gene polymorphism with localized early-onset periodontal diseases. J periodont, 70:1032-1038.
Henry AG. 2011. Plant foods and the dietary ecology of Neanderthals (Abstract). Am J Phys

Anth Supp 144:160-161.
Henry GA, Piperno D. 2008. Plants in Neanderthal diet: Plant microfossil evidence from the dental calculus of Shanidar III (abstract). PaleoAnthropol 2008:A11-A12.
Henry AG, Brooks AS, Piperno DR. 2011. Microfossils in calculus demonstrate consumption of plants and cooked foods in Neanderthal diets (Shanidar III, Iraq, Spy I and II, Belgium). Proc Nat Acad Sci 108:486-491.
Henshilwood CS, Marean CW. 2003. The origin of modern human behavior. Curr Anthropol 44:627-651.
Herńandez-Pacheco E, Obermaier H. 1915. La mandíbula neandertaloide de Bañolas. Junta para la Ampliacion de Estudios e Investigaciones Científicas, Comisión de Investigaciones Paleontológicas y Prehistóricas.
Herńandez-Pacheco E. 1924. Las pinturas prehistoricas de Las Cuevas de la Araña (Valencia). Comisíon de investigaciones paleontológicas p 34.
Hildebolt CF. 1987. Methods for studying geochemical-dental-disease interrelationships, including the assessment of periodontal disease by digital imaging. Doctoral Dissertation, Washington University in Saint Louis.
Hildebolt CF, Molnar S. 1991. Measurement and Description of Periodontal Disease in Anthropological Studies. In: Kelley MA, Larsen CS editors. Advances in Dental Anthropology. New York: Wiley-Liss, p 225-240.
Hillson SW. 1979. Diet and Dental Disease. World Archaeol 11:147-162.
Hillson SW. 1996. Dental Anthropology. Cambridge University Press: Cambridge.
Hillson SW. 2000. Dental Pathology. In: Biological Anthropology of the Human Skeleton. Eds MA Kaztenberg, SR Saunders. Wiley-Liss: New York 249-286.
Hillson SW. 2001. Recording dental caries in archaeological human remains. Int J Osteoarch 11:249-289.
Hillson SW. 2006. Dental Morphology, Proportions, and Attrition. In: Trinkaus E, Svoboda J. editors. Early modern human evolution in Central Europe: the people of Dolní Věstonice and Pavlov. Oxford: Oxford University Press p 179-223.
Hillson SW. 2008. The current state of dental decay. In: Irish JD, Nelson G. editors. Technique and Application in Dental Anthropology. Cambridge Studies in Biological and Evolutionary Anthropology. Cambridge: Cambridge University Press, p 111-135.
Hillson S, Rose JC. 2012. The first dental pathologists and paleopathologists. In: Buikstra J, Roberts C, editors. The global history of paleopathology: pioneers and prospects. Oxford: Oxford University Press p 619 - 631.
Hillson SW, Parfitt SA, Bello SM, Roberts MB, Stringer CB. 2010. Two hominin incisors teeth from the middle Pleistocene site of Boxgrove, Sussex, England. J Hum Evol 59:493-503.
Hlusko LJ. 2003. The Oldest Hominid Habit? Experimental Evidence for Toothpicking with Grass Stalks. Curr Anthropol 44:738-741.
Hobdell MH, Oliveira ER, Bautista R, Myburgh NG, Lalloo R, Narendran S, Johnson N. 2003. Oral diseases and socio-economic status (SES). Brit Dent J 194:91-96.
Holliday TW. 1995. Body size and proportions in the Late Pleistocene Western Old World and the origins of modern humans. Doctoral Dissertation. University of New Mexico.
Holliday TW. 1997. Body proportions in Late Pleistocene Europe and modern human origins. J Hum Evol 32:423-447.
Holliday TW. 1999. Brachial and crural indices of European Late Upper Paleolithic and

Mesolithic humans. J Hum Evol 36:549-566.
Holt BM. 2003. Mobility in Upper Paleolithic and Mesolithic Europe: evidence from the lower limb. Am J Phys Anthropol 122:200-215.
Holt BM, Formicola V. 2008. Hunters of the Ice Age: The Biology of Upper Paleolithic People. Yrbk Phys Anthropol 51:70-99.
Holt BM, Mussi M, Churchill SE, Formicola V. 2000. Biological and cultural trends in Upper Paleolithic Europe. Riv Antropol 78:179-192.
Hovers E, Belfer-Cohen A. 2006. "Now you see it, now you don't" –Modern Human Behavior in the Middle Paleolithic. In: Hovers E, Kuhn SL. Editors. Transitions Before the Transition: Interdisciplinary Contributions to Archaeology. New York: Springer p 295-304.
Hovers E, Horwitz LK, Bar-Yosef DE, Cope-Miyashiro C. 1988. The site of Urkan-E-rub IIa: a case study of subsistence and mobility patterns in the Kebaran period in the lower Jordan Valley. J Israel Prehist Soc 21:20-48.
Hublin JJ, Spoor F, Braun M, Zonneveld F, Condemi S. 1996. A late Neanderthal associated with Upper Palaeolithic artefacts. Nature 381:224-226.
Hublin JJ. 2009. The prehistory of compassion. Nature 106:6429-6430.
Hugoson A, Sjödin B, Norderyd O. 2008. Trends over 30 years, 1973–2003, in the prevalence and severity of periodontal disease. J Clin Periodont 35:405-414.
Hujoel P. 2009. Dietary carbohydrates and dental-systemic diseases. J Dent Res. 88:490–502.
Humphrey LT, Bocaege E. 2008. Tooth evulsion in the Maghreb: chronological and geographical patterns. Afri Archaeol Rev 25:109-123.
Humphrey LT, De Groote I, Morales J, Barton N, Collcutt S, Ramsey CB, Bouzouggar A. 2014. Earliest evidence for caries and exploitation of starchy plant foods in Pleistocene hunter-gatherers from Morocco. Proc Nat Acad Sci USA 111:954-959.
Ioannidou E, Swede H, Dongari-Bagtzoglou A. 2011. Periodontitis predicts elevated C-reactive protein levels in chronic kidney disease. J Dent Res. 90:1411–5.
Jablonski NG, Chaplin G. 2000. The evolution of human skin coloration. J Hum Evol 39: 57-106.
Jackson, JW. Dental mutilations in Neolithic human remains. J Anat Physiol 49:72.
Jamieson LM, Paradies YC, Gunthorpe W, Cairney SJ, Sayers SM. 2011. Oral health and social and emotional well-being in a birth cohort of Aboriginal Australian young adults. BMC public health 11:656.
Janković I, Karavanić I, Ahern JCM, Brajković D, Lenardić JM, Smith FH. 2006. Vindija Cave and the modern human people of Europe. Collegium Antropologicum 30:457-466.
Jansson L, Lavstedt S, Frithiof L. 2002. Relationship between oral health and mortality rate. J Clin Periodontol. 29:1029–34.
Jelinek AJ, Farrand WR, Haas G, Horowitz A, Goldberg P. 1973. New excavations at the Tabun Cave, Mount Carmel, Israel, 1967-1972: a preliminary report. Paléorient 1:151-183.
Jenkins WM, Kinane DF. 1989. The 'high risk' group of periodontitis. Br Dent J 167:168-171.
Jochim M. 1987. Late pleistocene refugia in Europe: The Pleistocene Old World. New York: Plenum Press, 317-331.
Johanson DC, White TD, Coppens Y. 1982. Dental remains from the Hadar Formation,

Ethiopia: 1974–1977 collections. Am J Phys Anthropol 57:545-603.
Johnston FE, Snow CE. 1961. The reassessment of the age and sex of the Indian Knoll skeletal population: demographic and methodological aspects. Am J Phys Anthropol 19:237-244.
Joshipura KJ, Willett WC, Douglass CW. 1996. The impact of edentulousness on food and nutrient intake. J Am Dent Assoc, 127:459-467.
Kaifu Y. 2000. Was Extensive Tooth Wear Normal in Our Ancestors? Anthropol Sci 108:371-385.
Kaifu Y, Kasai K, Townsend GC, Richards LC. 2003. Tooth wear and the "design" of the human dentition: a perspective from evolutionary medicine. Am J Phys Anthropol Suppl 37:47–61.
Kangxin H, Nakahashi T. 1996. A comparative study of ritual tooth ablation in ancient China and Japan. Anthropol Sci 104:43-64.
Karn KW, Shockett HP, Moffitt WC, Gray JL. 1984. Topographic classification of deformities of the alveolar process. J Periodontol 55: 336-340
Katz RV, Hazen SP, Chilton NW, Mumma RD. 1982. Prevalence and intraoral distribution of root caries in an adult population. Caries Res 16:265-271.
Keenleyside A. 1998. Skeletal evidence of health and disease in pre-contact Alaskan Eskimos and Aleuts. Am J Phys Anthropol. 107:51–70.
Kerr DA. 1962. Relations between periodontal disease and systemic disease. J Dent Res 41:302-304.
Kerr NW. 1988. A method for assessing periodontal status in archaeologically derived material. J Paleopathol 2: 67-78.
Kerr NW. 1990. The prevalence and pattern of distribution of root caries in a Scottish Medieval Population. J Dent Res 69:857-860.
Kerr NW. 1991. Prevalence and natural history of periodontal disease in Scotland- the mediaeval period (900-1600 AD). J Periodontal Res 26: 346-354.
Kerr NW. 1994. Prevalence and natural history of periodontal disease in a London, Spitalfields, population (1645–1852 AD). Arch Oral Biol 39:581-588.
Kesavalu L, Vasudevan B, Raghu B, Browning E. Dawson D, Novak JM, Ebersole JL. 2006. Omega-3 fatty acid effect on alveolar bone loss in rats. J Dent Res 85:648-652.
Kesavalu L, Bakthavatchalu V, Rahman MM, Su J, Raghu B, Dawson D, Fernandes G, Ebersole JL. 2007. Omega-3 fatty acid regulates inflammatory cytokine/mediator messenger RNA expression in Porphyromonas gingivalis-induced experimental periodontal disease. Oral microbiol immunol 22:232-239.
Kieser JA, Kelsen A, Love R, Herbison PGP, Dennison KJ. 2001. Periapical lesions and dental wear in the early Maori. Int J Osteoarchaeol 4:290–297.
Kinane DF, Chestnutt IG. 2000. Smoking and periodontal disease. Crit Rev Oral Bio Med 11:356-365.
King, W. 1964. The reputed fossil man of the Neanderthal. *Quart J Sci* 1:88-97.
Kirschbaum MUF. 2004. Direct and indirect climate change effects on photosynthesis and transpiration. Plant Biology 6:242-253.
Kiyak HA, Mulligan K. 1987. Studies of the relationship between oral health and psychological well-being. Gerodontics 3:109-112.
Klein H, Palmer CE. 1941. Studies on Dental Caries XII. Comparison of the Caries Susceptibility of the Various Morphological Types of Permanent Teeth. J Dent Res

20:203-216.
Kluger MJ. 1991. Fever: role of pyrogens and cryogens. Physiol Rev 71:93-127.
König KG. 2000. Diet and Oral Health. Int Dent J 50:162-174.
Kortizer, R.T., St. Hoyme, L.E., 1979. Extensive caries in early man circa 110,000 years before present. J. Am. Dent. Assoc. 99, 642-643.
Krueger KL. 2011. Dietary and Behavioral Stratgies of Neandertals and anatomically modern humans: evidence from anterior dental microwear texture analysis. Doctoral Dissertation, University of Arkansas.
Krueger KL, Ungar PS. 2012. Anterior dental microwear texture analysis of the Krapina Neandertals. Central Eur J Geosci 4:651-662.
Kubota K, Olayimika Hollist N, Oluniyi Olusile A, Yonemitsu M, Minakuchi S, Watanabe H, Ohsawa K, Ohnishi M, Ono Y, Oladosu Ajayi-Obe S. 1993. Joint epidemiological longitudinal dental survey in Nigeria, especially in comparison with that of Japanese. Bulletin Tokyo Med Dent Univers 40:59.
Lacruz RS, Ramirez Rozzi F, Bromage TG. 2005. Dental enamel hypoplasia, age at death, and weaning in the Taung child. South Afri J Sci 101:567.
Lacy SA, Wu XJ, Jin CZ, Qin DG, Cai YJ, Trinkaus, E. 2012. Dentoalveolar Paleopathology of the early modern humans from Zhirendong, South China. Int J Paleopathol. 2:10-18.
Lacy, n.d. Case Study: The oral pathologies of the Broken Hill (Kabwe) 1 cranium.
Lacy SA, Bailey S, Delage C. *In preparation.* A newly recognized Neandertal individual represented by dental remains at Les Fadets (Lussac-les-Châteaux, Vienne, France).
Lai PY, Seow K, Tudehope DI, Rogers Y. 1997. Enamel hypoplasia and dental caries in very-low birthweight children: a case-controlled, longitudinal study. Pediat denti 19:42-49.
Lalueza, C, Pérez-Pérez A, Chimenos E, Maroto J, Turbón D.1993. Estudi radiografici microscopic de la mandıbula de Banyoles: pathologies i estat de conservacio . In: Maroto J editor. La Mandibula de Banyoles en el Context dels Fossils Humans del Pleistocene. Centre d'Investigacions Arquelógiques de Girona Série Monográfica p.135–144.
Lambeck K, Chappell J. 2001. Sea level change through the last glacial cycle. Science 292:679–686.
Lanfranco LP, Eggers S. 2010. The usefulness of caries frequency, depth, and location in determining cariogenicity and past subsistence: A test on early and later agriculturalists from the Peruvian coast. Am J Phys Anthropol 143:75-91.
Lanfranco LP, Eggers S. 2012. Caries through time: an anthropological overview. Contemporary approach to dental caries. InTech: Rijeka, 3-34.
Larsen CS. 1995. Biological changes in human populations with agriculture. Ann Rev Anthropol 1995:185-213.
Larsen CS. 1997. Bioarchaeology: Interpreting Behavior From the Human Skeleton Cambridge: Cambridge University Press.
Larsen CS. 2006. The agricultural revolution as environmental catastrophe: implications for health and lifestyle in the Holocene. Quatern Int 150:12-20.
Larsen CS, Shavit R, Griffin MC. 1991. Dental caries evidence for dietary change: an archaeological context. In: Kelley MA, Larsen CS, editors. Advances in Dental Anthropology. New York: Wiley-Liss, 179-202

Larsen H, Rainey F. 1948. Ipiutak and the Arctic Whale-Hunting Culture: Anthropological Paper 42. American Museum of Natural History: New York.
Lavigne SE, Molto JE. 1995. System of Measurement of the Severity of Periodontal Disease in Past Populations. Internat J Osteoarch 5:265-273.
Leach JD, Gibson GR, Loo JV. 2006. Human evolution, nutritional ecology and prebiotics in ancient diet. Biosci microflora 25:1-8.
Lebel S, Trinkaus E. 2002a. Middle Pleistocene human remains from the Bau de l'Aubesier. J Hum Evol, 43:659-685.
Lebel S, Trinkaus E. 2002b. Short Note. A Carious Neandertal Molar from the Bau de l'Aubesier, Vaucluse, France. J Archaeol Sci 29:555-557.
Lebel S, Trinkaus E, Faure M, Fernandez P, Guerin C, Richter D, Mercier N, Valladas H, Wagner GA. 2001. Comparative Morphology and Paleobiology of Middle Pleistocene Humans Remains from the Bau de l'Aubesier, Vaucluse, France. Proc Nat Acad Sci USA. 98:11097-11102.
Lebenthal E. 1987. Role of salivary amylase in gastric and intestinal digestion of starch. Digestive Diseases Sci 32:1155-1157.
Leigh RW. 1925. Dental pathology of Indian tribes of varied environmental and food conditions. Am J Phys Anthropol 8:179-199.
Lev E, Kislev ME, Bar-Yosef O. 2005. Mousterian vegetal food in Kebara Cave, Mt. Carmel. J Archaeol Sci 32: 475-484.
Li Y, Wang W. 2002. Predicting caries in permanent teeth from caries in primary teeth: an eight-year cohort study. J Dent Res 81:561-566.
Liebe-Harkort C. 2012. Exceptional rates of dental caries in a scandinavian Early Iron Age population-A study of dental pathology at Alvastra, Östergötland, Sweden. Int J Osteoarchaeol. 22:168–184.
Lieff S, Boggess K, Murtha A, Jared H, Madianos P, K M, Beck J, and Offenbacher S. 2004. The oral conditions and pregnancy study: Periodontal status of a cohort of pregnant women. J Periodontol 75:116-126.
Lieverse A, Link D. 2007. Dental health indicators of hunter–gatherer adaptation and cultural change in Siberia's Ciskal Baikal. Am J Phys Anthropol 339:323–339.
Lindly JM. 1990. Symbolism and Modern Human Origins. Curr Anthropol 31: 233-261.
Liu W, Zeng X. 1996. The degeneration of third molars and its significance to human evolution. Acta Anthropologica Sinica 15:185-199.
Liu W, Jin CZ, Zhang YQ, Cai YJ, Xing S, Wu XJ, Cheng H, Edwards RL, Pan WS, Qin DG, An ZS, Trinkaus E, Wu XZ. 2010. Human remains from Zhirendong, South China, and modern human emergence in East Asia. Proc Nat Acad Sci 107: 19201-19206.
Löe H, Anerud A, Boysen H, Morrison E. 1986. Natural history of periodontal disease in man. J Clin Periodontol 13:431-440.
Löe H. 2000. Oral hygiene in the prevention of caries and periodontal disease. Int Dent J 50:129-139.
Löe H, Ånerud Å, Boysen H. 1992. The Natural History of Periodontal Disease in Man: Prevalence, Severity, and Extent of Gingival Recession. J Periodontol 63:489-495.
Loesche WJ. 1986. The identification of bacteria associated with periodontal disease and dental caries by enzymatic methods. Oral microbio immuno 1:65-70.
Loesche WJ. 1996. Association of the oral flora with important medical diseases. Curr

Opinion in Periodontol 4:21-28.
Lopez B, Garcia-Vazquez E, Dopico E. 2011. Dental indicators suggest health improvement associated with increased food diversity in modern age spain. Hum Ecol 39:527-534.
Lordkipanidze D, Vekua A, Ferring R, Rightmire GP, Agusti J, Kiladze G, Mouskhelishvili A, Nioradze M, Ponce de Leon MS, Tappan M, Zollikofer CPE. 2005. Anthropology: The earliest toothless hominin skull. Nature 434:717-718.
Lovejoy CO. 1985. Dental wear in the Libben population: its functional pattern and role in the determination of adult skeletal age at death. Am J Phys Anthropol 68:47–56.
Lovell NC. 1990. Patterns of Injury and Illness in Great Apes. Washington, DC: Smithsonian Institution Press.
Lovell NC. 1991. An evolutionary framework for assessing illness and injury in nonhuman primates. Am J Phys Anthropol Supp 34: 117-155.
Lozano M, Eulàlia Subirà M, Aparicio J, Lorenzo C, Gómez-Merino G. 2013. Toothpicking and Periodontal Disease in a Neanderthal Specimen from Cova Foradà Site (Valencia, Spain). *PloS one* 8:e76852.
Lucas S, Sevin A, Passarius O, Esclassan R, Crubezy E, Grimoud AM. 2010. Study of dental caries and periapical lesions in a mediaeval population of the southwest France: differences in visual and radiographic inspections. HOMO-J Compar Hum Bio 61:359-372.
Lukacs JR. 1995. The 'Caries Correction Factor': a new method of calibrating dental caries rates to compensate for antemortem loss of teeth. Internat J Osteoarch 5:151-156.
Lukacs JR. 2007. Dental trauma and antemortem tooth loss in prehistoric Canary Islanders: prevalence and contributing factors. Int J Osteoarchaeol 17:157-173.
Lukacs JR. 2008. Fertility and Agriculture Accentuate Sex Differences in Dental Caries Rates. Curr Anthropol. 49:901–914.
Lukacs JR. 2011. Sex differences in dental caries experience: clinical evidence, complex etiology. Clin Oral investigat15:649-656.
Lukacs JR, Pal JN. 1993. Mesolithic subsistence in north India: inferences from dental attributes. Curr Anthropol 745-765.
Maat GJR, van der Velde EA. 1987. The caries-attrition competition. Int J Anthropol 2: 281-292.
Macek MD, Beltran-Aguilar ED, Lockwood SA, Malvitz DM. 2003. Updated comparison of the caries susceptibility of various morphological types of permanent teeth. J Pub Health Denti 63:174-182.
Madella M, Jones MK, Goldberg P, Goren Y, Hovers E. 2002. The exploitation of plant resources by Neanderthals in Amud Cave (Israel): the evidence from phytolith studies. J Archaeol Sci 29:703-719.
Mai LL, Young Owl M, Kersting MP. 2005. The Cambridge Dictionary of Human Biology and Evolution. 1st ed. Cambridge: Cambridge University Press.
Maki JM. 2013. The Biomechanics of Spear Throwing: An Analysis of the Effects of Anatomical Variation on Throwing Performance, with Implications for the Fossil Record. Doctoral Dissertation. Washington University.
Mallegni F. 1991. On the possibility that the Circeo II mandible may have belonged to the same individual as the Circeo 1 cranium. In: Piperno M, Scichilne G, editors. The Circeo 1 neandertal skull studies and documentation. Instituto Poligrafico e Zecca delo Stato p 391-408.

Mallegni F. 1995. The teeth and the periodontal apparatus of the Neandertal mandibles from the Guattari Cave (Monte Circeo, Lazio, Italy). Zeitschrift für Morphologie und Anthropologie 329-351.
Manji F, Fejerskov O, Baelum V. 1989. Pattern of dental caries in an adult rural population. Caries Res 23:55-62.
Manji F, Fejerskov O, Nagelkerke NJD, Baelum V. 1991. A random effects model for some epidemiological features of dental caries. Commun Denti Oral Epidemiol 19:324-328.
Mann A, Vandermeersch B, Delagnes A, Tournepiche JF. 2007. Restes humains fossiles des niveaux moustériens d'Artenac (Charente). C R Palévol 6:581-589.
Marcenes WS, Sheiham A. 1992. The relationship between work stress and oral health status. Soc Sci Med 35:1511-1520.
Marean CW, Kim SY. 1998. Mousterian large mammal remains from Kobeh Cave. Behavioral implications for Neanderthals and early modern humans. Curr Anthropol 39:S79-S113.
Marin V, Hrvoje B, Mario S, Zeljko D. 2005. The frequency and distribution of caries in the mediaeval population of Bijelo Brdo in Croatia (10^{th}-11^{th} century). Archs Oral Biol 50:669-680.
Marin V, Kristina P, Amila Z, Marjana K, Mario N, Mario S, Hrvoje B. 2012. Periodontal diseases at the transition from the late antique to the early mediaeval period in Croatia. Arch Oral Biol. 57:1362–76.
Márquez-Grant N. 2009. Caries correction factors applied to a Punic (6th-2nd BC) population from Ibiza (Spain). Bull Int Assoc Paleodontology 3:20–29.
Mashoto KO, Astrom AN, Skeie MS, Masalu JR. 2010. Socio-demographic disparity in oral health among the poor: a cross sectional study of early adolescents in Kilwa district, Tanzania. BMC Oral Health 10:7.
Mattheeuws N, Dermaut L, Martens G. 2004. Has hypodontia increased in Caucasians during the 20th century? A meta-analysis. Eur J Orthodont 26:99-103.
McBrearty S, Brooks AS. 2000. The revolution that wasn't: a new interpretation of the origin of modern human behavior. J Hum Evol 39:453-563.
McCorriston J, Hole F. 1991. The ecology of seasonal stress and the origins of agriculture in the Near East. Am Anthropol 93:46-69.
McDougall I, Brown FH, Fleagle JG. 2005. Stratigraphic placement and age of modern humans from Kibish, Ethiopia. Nature. 433:733-736.
Mcilvaine BK, Reitsema LJ. 2013. A look at the literature: Recent developments and long-term trends in the interpretation of skeletal stress markers and ancient health (abstract) Am J Phys Anthropol Supp S56:194-195
McKee JK. 1984. A genetic model of dental reduction through the probable mutation effect. Am J Phys Anthropol 65:231-241.
McLeod DE, Lainson PA, Spivey JD. 1997. Tooth loss due to periodontal abscess: a retrospective study. J Periodont 68:963-966.
Mieklejohn C, Babb J. 2011. Long bone length, stature and time in the European Late Pleistocene and Early Holocene. In: Pinhasi R, Stock JT, editors. Human Bioarchaeology of the Transition to Agriculture p 151-175.
Meiklejohn C, Schentag C, Venema A, Key P. 1984. Socioeconomic change and patterns of pathology and variation in the Mesolithic and Neolithic of Western Europe: some suggestions. In: Cohen, Armelagos, editors. Paleopathology at the Origins of

Agriculture p 75-100.
Meiklejohn C, Baldwin J, Schentag C. 1988. Caries as a probable dietary marker in the Western European Mesolithic. In: Diet and Subsistence: Current Archaeological Perspectives p 273-279.
Meiklejohn C, Wyman JM, Schentag CT. 1992. Caries and attrition: Dependent or independent variables? Intl J Anthropol 7:17-22.
Mellars PA. 1985. The ecological basis of social complexity in the Upper Paleolithic of southwestern France. In: Prehistoric hunter-gatherers: The emergence of cultural complexity p 271-297.
Mellars P. 1996. The Neanderthal legacy: an archaeological perspective from Western Europe. Princeton: Princeton University Press.
Mellars P, French JC. 2011. Tenfold Population Increase in Western Europe at the Neandertal–to–Modern Human Transition. Science 333:623-627.
Meurman JH, Hämäläinen P. 2006. Oral health and morbidity-implications of oral infections on the elderly. Gerodontology 23:3-16.
Meurman JH, Sanz M, Janket SJ. 2004. Oral Health, Atherosclerosis, and Cardiovascular disease. Crit Rev Oral Bio Med 15:403-413.
Migliorati CA, Madrid C. 2007. The interface between oral and systemic health: the need for more collaboration. Clin. Microbiol. Infect Supp. 13:11–6.
Miles AEW. 1963. Dentition in the assessment of individual age in skeletal material. In: Brothwell DR, editor. Dental Anthropology. Pergamon: Oxford, p 191-209.
Minotti NA 2003. Parodontaler Knochenschwund zu Beginn des 19. Jahrhundrerts. Bull Schweizerischen Geschellschaft Anthropologie 9:59-86.
Molnar, S. 1971. Human tooth wear, tooth function and cultural variability." Am J Phys Anthropol 34:175-189.
Molnar P. 2008. Dental Wear and Oral Pathology: Possible Evidence and Consequences of Habitual Use of Teeth in a Swedish Neolithic Sample. Am J Phys Anthropol 136:423-431.
Molnar S, Molnar I. 1985. The Incidence of enamel hypoplasia among the Krapina Neandertals. Am Anthropol 87:536-549.
Molnar S, Hildebolt C, Molnar IM, Radovic J, Gravier M. 1993. Hominid Enamel Thickness: I. The Krapina Neandertals. Am J Phys Anthropol 92:131-138.
Monge J, Mann A. 2007. Paleodemography of Extinct Hominin Populations. In: Henke HCW, Tattersall I. Handbook of paleoanthropology. Berlin: Springer p 673-700.
Montgomery, P.Q., Williams, H.O.L., Reading, N. Stringer, C.B., 1994. An Assessment of the Temporal Bone Lesions of the Broken Hill Cranium. J Archaeol. Sci. 21, 331-337.
Morin E. 2008. Evidence for declines in human population densities during the early Upper Paleolithic in western Europe. Proc Nat Acad Sci 105:48-53.
Müller F, Naharro M, Carlsson GE. 2007. What are the prevalence and incidence of tooth loss in the adult and elderly population in Europe? Clin Oral Implants Res 18:2-14.
Nadel D, Hershkovitz I. 1991. New subsistence data and human remains from the earliest Levantine Epipalaeolithic. Curr Anthropol 32:631-635.
Nagar Y, Hershkovitz I. 2004. Interrelationship between various aging methods, and their relevance to palaeodemography. Hum Evol 19:145-156.
Nadel D, Grinberg U, Boaretto E, Werker E. 2006. Wooden objects from Ohalo II (23,000 cal BP), Jordan Valley, Israel. J Hum Evol 30:644-662.

Nariyama M, Shimizu K, Uematsu T, Maeda T. 2004. Identification of chromosomes associated with dental caries susceptibility using quantitative trait locus analysis in mice. Caries Res 38:79-84.
Neely AL, Holford TR, Löe H, Ånerud Å, Boysen H. 2001. The natural history of periodontal disease in man. Risk factors for progression of attachment loss in individuals receiving no oral health care. J Periodontol 72:1006-1015.
Neely AL, Holford TR, Löe H, Ånerud Å, Boysen H. 2005. The natural history of periodontal disease in humans: risk factors for tooth loss in caries-free subjects receiving no oral health care. J Clinical Periodontol 32:984-993.
Nesse RM, Williams GC. 1998. Evolution and the origins of disease. Scientific American 279:58-65.
Niewoehner WA. 2001. Behavioral inferences from the Skhul/Qafzeh early modern human hand remains." Proc Nat Acad Sci 98:2979-2984.
Nelson GCC, Lukacs JRR, and Yule P. 1999. Dates, caries, and early tooth loss during the Iron Age of Oman. Am J Phys Anthropol. 108:333–43.
Newman H. 1998. Do not confuse the bone loss of periodontitis with root exposure due to continuous eruption. Factors affecting the lifespan of the human dentition in Britain prior to the seventeenth century. Brit Dent J 184:229-229.
Nibali L, Farias BC, Vajgel A, Tu YK, Donos N. 2013. Tooth loss in Aggressive Periodontitis: A systematic review. J Dent Res 92:868-875.
Norderyd O, Hugoson A. 1998. Risk of severe periodontal disease in a Swedish adult population. Journal of clinical periodontology 25:1022-1028.
Nout MJR. 1993. Processed weaning foods for tropical climates. Int J Food Sci Nutri 43:213-221.
Oliver RC, Brown LJ, Löe, H. 1998. Periodontal diseases in the United States population. J Periodont, 69:269-278.
O'Connell JF. 2006. How did modern humans displace Neanderthals? Insights from hunter-gatherer ethnography and archaeology.In: Conard N. editor. When Neanderthals and modern humans met. Tübingen: Kern p 43-64.
O'Reilly PG, Claffey NM. 2000. A history of oral sepsis as a cause of disease. Periodontol 2000, 23:13-18.
Ogden A. 2008. Advances in the Paleopathology of Teeth and Jaws. In: Advances in Human Paleopathology. Eds R Pinhasi, S Mays. New York: Wiley-Liss. p 283-307.
Ogilvie MD, Curran BK, Trinkaus E. 1989. Incidence and patterning of dental enamel hypoplasia among the Neandertals. Am J Phys Anthopol 79:25-41.
Olejniczak AJ, Smith TM, Feeney RNM, Macchiarelli R, Mazurier A, Bondioli L, Rosas A, Fortea J, Rasilla M de la, Garcia-Tabernero A, Radovčić J, Skinner MM, Toussaint M, Hublin JJ. 2008. Dental tissue proportions and enamel thickness in Neandertal and modern human molars. J Hum Evol 55:12-23.
Oliva M. 2000. The Brno II Upper Paleolithic burial. In: Roebroeks W, Mussi M, Svoboda J, Fennema K, editors. Hunters of the golden age. The Mid Upper Paleolithic of Eurasia (30,000-20,000 BP) Leiden: Leiden University Press p 143-153.
Oliver RC, Brown, LJ, Löe H. 1998. Periodontal diseases in the United States population. J Periodontol 69:269-278.
Ortner DJ. 2003. The Identification of Pathological Conditions in Human Skeletal Remains, 2nd edition. New York: Academic Press

Ortner DJ, Putschar WG. l985. Identification of Pathological Conditions in Human Skeletal Remains." Smithsonian Contributions to Anthropology 28.
Orschiedt J. 2002. Datation d'un vestige humain provenant de La Rochette (Saint Léon-sur-Vézère, Dordogne) par la méthode du carbone 14 en spectrométrie de masse. PALEO Revue d'archéologie préhistorique 14:239-240.
Outram, Alan K. 2001. A new approach to identifying bone marrow and grease exploitation: why the "indeterminate" fragments should not be ignored. J Archaeol Sci 28:401-410.
Oxenham MF, Matsumura H. 2008. Oral and Physiological Paleohealth in Cold Adapted Peoples: Northeast Asia, Hokkaido. Am J Phys Anthropol 135:64-74.
Oxenham, MF, Nguyen LC, Nguyen KT. 2006. The oral health consequences of the adoption and intensification of agriculture in Southeast Asia. Bioarchaeology of Southeast Asia 43:263.
Oztunc H, Yoldas O, Nalbantoglu E. 2006. The periodontal disease status of the historical population of Assos. Int J Osteoarchaeol, 16:76-81.
Padilha DMP, Hilgert JB, Hugo FN, Bós ÂJG, Ferrucci L. 2008. Number of teeth and mortality risk in the Baltimore Longitudinal Study of Aging. J Gerontol A: Bio Sci Med Sci 63:739-744.
Page RC, Bowen T, Altman L, Vandesteen E, Ochs H, Mackenzie P, Osterberg S, Engel LD, Williams BL. 1983. Prepubertal Periodontitis. I. Definition of a Clinical Disease Entity. J Periodontol 54:257-271.
Pager, H., 1976. Cave paintings suggest honey hunting activities in Ice Age times. Bee World. 57:9-14.
Palubeckaitė Ž, Jankauskas R, and Boldsen J. 2002. Enamel hypoplasia in Danish and Lithuanian Late Medieval/Early Modern samples: a possible reflection of child morbidity and mortality patterns. Int J Osteoarchaeol. 12:189–201.
Pap I, Tillier AM, Arensberg B, Weiner S, Chech M. 1995. First scanning electron microscope analysis of dental calculus from Europeans Neanderthals: Subalyuk (Middle Paleolithic, Hungary). Bull Mem Soc Anthropol Paris 7:69-72.
Papapanou PN, Lindhe J, Sterrett JD, Eneroth L. 1991. Considerations on the contribution of ageing to loss of periodontal tissue support. J Clin Periodontol 18:611-615.
Pascoe L, Seow W. 1994. Enamel hypoplasia and dental caries in Australian aboriginal children: prevalence and correlation between the two diseases. Pediatr Dent 16:193-193.
Pechenkina EA, Benfer RA, Zhijun W. 2002. Diet and health changes at the end of the Chinese Neolithic: the Yangshao/Longshan transition in Shaanxi province. Am J Phys Anthropol 117:15-36.
Peresani M. 2008. A new cultural frontier for the last Neanderthals: The Uluzzian in northern Italy." Curr Anthropol 49:725-731.
Peresani M, Fiore I, Gala M, Romandini M, Tagliacozzo A. 2011. Late Neandertals and the intentional removal of feathers as evidenced from bird bone taphonomy at Fumane Cave 44 ky BP, Italy. Proc Nat Acad Sci 108:3888-3893.
Perzigian AJ. 1976. The dentition of the Indian Knoll skeletal populations: odontometrics and cusp number. Am J Phys Anthropol 44:113-121.
Perzigian, AJ. 1977. Fluctuating dental asymmetry: variation among skeletal populations. Am J Phys Anthropol 47:81-88.
Peterson J. 2002. Sexual revolutions: gender and labor at the dawn of agriculture. Vol. 4.

Rowman Altamira.
Pierce JA, Ebert RV. 1958. The barrel deformity of the chest, the senile lung and obstructive pulmonary emphysema. Am J Med 25:13-22.
Pinhasi R, Stock JT, editors. 2011. Human bioarchaeology of the transition to agriculture. Chichester: Wiley-Blackwell.
Pine CM, ten Bosch JJ. 1996. Dynamics of and diagnostic methods for detecting methods for small carious lesions. Caries Res 30:381-388.
Pinho T, Maciel P, Lemos C, Sousa A. 2010. Familial aggregation of maxillary lateral incisor agenesis. J Dent Research 89:621-625.
Platek SM, Gallup Jr, GG, Fryer BD. 2002. The fireside hypothesis: was there differential selection to tolerate air pollution during human evolution? Med Hypothes 58:1-5.
Polder BJ, Vant Hof MA, Van der Linden FP, Kuijpers-Jagtman AM. 2004. A meta-analysis of the prevalence of dental agenesis of permanent teeth. Commun Dent Oral Epidemiol 32:217-226.
Poole DFG, Tratman EK. 1978. Post-Mortem changes on human teeth from late upper Palaeolithic/Mesolithic occupants of an English limestone cave. Arch Oral Bio 23: 1115-1120.
Pottier C. 2005. Le Gravettien moyen de l'abri Pataud (Dordogne, France): le niveau 4 et l'éboulis 3/4. Etude technologique et typologique de l'industrie lithique. Doctoral dissertation, Museum National d'Histoire Naturelle.
Powell A, Shennan S, Thomas MG. 2009. Late Pleistocene demography and the appearance of modern human behavior. Science 324:1298-1301.
Powell RN. 1983. Geographic effects on dental caries prevalence and tooth loss in Australia. Commun Dent Oral Epidemiol 11:242-245.
Pradel L. 1959. Le niveau de Noailles d'Oreille-d'Enfer, Les Eyzies-de-Tayac (Dordogne). Bulletin de la Société préhistorique de France 56:228-235.
Prieto JL. 2005. Hallazgos paleopatológicos en la mandíbula SDR-7-8 del Sidrón. In: Montes-Barquín R, Lasheras-Corruchaga JA editors. Neandertales Cantábricos, Estado de la Cuestión. Monografías del Museo de Altamira 20, Santillana del Mar p 397-403.
Puech PF, Albertini H, Mills NTW. 1980. Dental Destruction in Broken-Hill Man. J Hum Evol 9:33-39.
Rabkin S. 1943. Dental conditions among prehistoric Indians of Kentucky. J Dent Res 22:355-366.
Rabinovich R, Hovers E. 2004. Faunal analyses from Amud Cave: preliminary results and interpretations. Int J Osteoarchaeol 14:287-306.
Rainey FG. 1941. The Ipiutak Culture at Point Hope, Alaska. Am Anthropol 43:364-375.
Rainey FG. 1971. The Ipiutak Culture: Excavations at Point Hope, Alaska. New York: Addison-Wesley.
Ramfjord SP. 1967). The periodontal disease index (PDI). J Periodont 38:602-610.
Ramirez Rozzi FV, d'Errico F, Vanhaeren M, Grootes PM, Kerautret B, Dujardin V. 2009. Cutmarked human remains bearing Neandertal features and modern human remains associated with the Aurignacian at Les Rois. J Anthropol Sci 87:153-185.
Rautemaa R, Lauhio A, Cullinan MP, Seymour GJ. 2007. Oral infections and systemic disease--an emerging problem in medicine. Clin Microbiol Infect. 13:1041–7.
Ravald N, Hamp SE. 1981. Prediction of root surface caries in patients treated for advanced periodontal disease. J Clin Periodont 8:400-414.

Revedin A, Aranguren B, Becattini R, Longo L, Marconi E, Lippi MM, Skakun N, Sinitsyn A, Spiridonova E, Svoboda S. 2010. Thirty thousand-year-old evidence of plant food processing. Proc Nat Acad Sci 107:18815-18819.
Richards MP, Trinkaus E. 2009. Isotopic evidence for the diets of European Neanderthals and early modern humans. Proc Nat Acad Sci 106:16034-16039.
Richards MP, Pettitt PB, Trinkaus E, Smith FH, Paunović M, Karavanić I. 2000. Neanderthal diet at Vindija and Neanderthal predation: the evidence from stable isotopes. Proc Nat Acad Sci 97:7663-7666.
Richards MP, Pettitt PB, Stiner MC, Trinkaus E. 2001. Stable isotope evidence for increasing dietary breadth in the European mid-Upper Paleolithic. Proc Nat Acad Sci 98:6528-6532.
Richards MP, Taylor G, Steele T, McPherron SP, Soressi M, Jaubert J, Orschiedt J, Mallye JB, Rendu W, Hublin JJ. 2008. Isotopic dietary analysis of a Neanderthal and associated fauna from the site of Jonzac (Charente-Maritime), France. J Hum Evol 55:179-185.
Ripamonti U. 1988. Paleopathology in Australopithecus africanus: A suggested case of a 3-million-year-old prepubertal periodontitis. Am J Phys Anthropol 76:197-210.
Rivals F, Schulz E, Kaiser TM. 2009. Late and middle Pleistocene ungulates dietary diversity in Western Europe indicate variations of Neanderthal paleoenvironments through time and space. Quartern Sci Rev 28:3388-3400.
Robinson JT. 1952. Some hominid features of the ape-man dentition. J Dent Assoc South Afr 7:102-113.
Robinson SA, Black S, Sellwood BW, Valdes PJ. 2006. A review of palaeoclimates and palaeoenvironments in the Levant and Eastern Mediterranean from 25,000 to 5000 years BP: setting the environmental background for the evolution of human civilisation. Quartern Sci Rev, 25:1517-1541.
Rolland N. 2004. Was the emergence of home bases and domestic fire a punctuated event? A review of the Middle Pleistocene record in Eurasia. Asian Perspectives 43:248-280.
Ronderos M, Pihlstrom BL, Hodges JS. 2001. Periodontal disease among indigenous people in the Amazon rain forest. J Clin Periodontol. 28:995–1003.
Rosas A, Martínez-Maza C, Bastir M, García-Tabernero A, Lalueza-Fox C, Huguet R, Eugenio Ortiz J, Ramóm J, Soler V, de Torres T, Martínez E, Cañaveras JC, Sánchez-Moral S, Cuezva S, Lario J, Santamaría D, de la Rasilla M, Fortea J. 2006. Paleobiology and comparative morphology of a late Neandertal sample from El Sidron, Asturias, Spain. Proc Nat Acad Sci USA 103:19266-19271.
Rose J, Ungar P. 1998. Gross dental wear and dental microwear in historical perspective. In: Alt KW, FW Rösing, M Teschler-Nicola, editors. Dental Anthropology. Vienna: Springer p 349-386.
Rose JC, Armelagos GJ, Lallo JW. 1978. Histological enamel indicator of childhood stress in prehistoric skeletal samples. Am J Phys Anthropol 49:511-516.
Rowlett RM, Schneider MJ. 1974. The material expression of Neanderthal child care. In: Richardson M, ed. The Human Mirror. Baton Rouge: Louisiana State University Press, pp. 41-58.
Rozzi FVR, Bermudez de Castro JM. 2004. Surprisingly rapid growth in Neanderthals. Nature 428:936-939.
Ruff CB, Trinkaus E, Walker A, Larsen CS. 1993. Postcranial robusticity in Homo. I:

Temporal trends and mechanical interpretation. Am J Phys Anthropol 91:21-53.
Ruff CB. 1994. Morphological adaptation to climate in modern and fossil hominids. Am J Phys Anthropol 37:65-107.
Ruff CB, Holt BM, Trinkaus E. 2006. "Who's afraid of the big bad Wolff?:"Wolff's law" and bone functional adaptation." Am J Phys Anthropol 129:484-498.
Sahlins M. 1968. "Notes on the original affluent society." Man the hunter: 85-89.
Samuelson G, Grahnen H, Arvidsson E. 1971. An epidemiological study of child health and nutrition in a northern Swedish county: VI. Relationship between general and oral health, food habits, and socioeconomic conditions. Am J Clin Nutri 24:1361-1373.
Sandgathe DM, Hayden B. 2003. Did Neanderthals eat inner bark? Antiquity 77:709-718.
Sankararaman S, Mallick S, Dannemann M, Prüfer K, Kelso J, Pääbo S, Patterson N, Reich D. 2014. The genomic landscape of Neanderthal ancestry in present-day humans. Nature, Advance View.
Sattenspiel L, Harpending H. 1983. Stable Populations and Skeletal Age. Am Antiquity 48: 489-498.
Schersten E, Lysell L, Rohlin M. 1989. Prevalence of impacted third molars in dental students. Swed Dent J 13:7.
Schifferle RE. 2009. Periodontal disease and nutrition: separating the evidence from current fads. Periodont 2000 50:78-89.
Schultz AH. 1956. The occurrence and frequency of pathological and teratological conditions and of twinning among non-human primates. Primatologia 1:965-1014.
Schwartz JH, Tattersall I. 2003. The Human Fossil Record, Volume II. New York: Wiley-Liss.
Scott, EC. 1979. Dental wear scoring technique. Am J Phys Anthropol 51:213-217.
Scott GR, Turner CG II. 1988. Dental Anthropology. Ann Rev Anthropol 17:99-126
Scott GR, Turner CG II. 1997. The anthropology of modern human teeth: dental morphology and its variations in recent human populations. Cambridge: Cambridge University Press.
Seltzer S, Bender IB, Ziontz M. 1963. The interrelationship of pulp and periodontal disease. Oral Surg Oral Med Oral Pathol 16:1474-1490.
Sergi S. 1974. Il Cranio Neandertaliano del Monte Circeo (Circeo I). Rome: Accademia Nazionale dei Lincei.
Shackelford LL. 2005. Patterns of geographic variation in the postcranical robusticity of Late Upper Paleolithic humans. Doctoral Dissertation. Washington University
Shang H, Trinkaus E. 2010. The early modern human from Tianyuan Cave, China. College Station: Texas A&M University Press.
Shen G, Wu Z, Wang Q, Tu H, Feng Y-X, Zhao J-X. 2013. Mass spectrometric U-series dating of Huanglong Cave in Hubei Province, central China: Evidence for early presence of modern humans in eastern Asia." J Hum Evol 65:162-167.
Shetty M, Shetty PK, Ramesh A, Thomas B, Prabhu S, Rao A. 2010 Periodontal disease in pregnancy is a risk factor for preeclampsia. Acta Obstet Gynecol Scand. 89:718-21.
Shrout MK, Hildebolt CF, Vannier MW, Province M, Vahey EP. 1990. Periodontal Disease Morbidity Quantification. I. Optimal Selection of Teeth for Periodontal Bone Loss Surveys. J Periodontol 61:618-622.
Simek JF. 1986. A Paleolithic sculpture from the Abri Labattut in the American Museum of Natural History collection. Curr Anthropol 402-407.

Skeie MS, Raadal M, Strand GV, Espelid I. 2006. The relationship between caries in the primary dentition at 5 years of age and permanent dentition at 10 years of age–a longitudinal study. Int J Paediatric Denti 16:152-160.
Skinner M. 1996. Developmental Stress in Immature Hominines from Late Pleistocene Eurasia: Evidence from enamel Hypoplasia. J Archaeol Sci 23:833-852.
Skinner M. 1997. Dental Wear in Immature Late Pleistocene Europeans Hominines. J Archaeol Sci 24:677-700.
Skinner MF, Sperber GH, 1982. Atlas of Radiographs of Early Man. Alan R. Liss, Inc., New York.
Slavkin HC, Baum BJ. 2000. Relationship of dental and oral pathology to systemic illness. J Am Med Assoc 284:1215-1217.
Smith BHH. 1984. Patterns of molar wear in hunger-gatherers and agriculturalists. Am J Phys Anthropol. 63:39–56.
Smith FH. 1976. The Neandertal remains from Krapina: A descriptive and comparative study. Univ Tennessee Dep Anthropol Rep Invest 15:1–359.
Smith FH. 1983. Behavioral interpretations of changes in craniofacial morphology across the archaic/modern Homo sapiens transition. In: Trinkaus E, editor. The Mousterian Legacy. Oxford: British Archaeological Reports International Series p 141–163.
Smith P. 1970. Dental morphology and pathology in Natufians: The dental evidence for dietary specialization. Unpublished Ph.D. dissertation, University of Chicago
Smith P. 1972. Diet and attrition in the Natufian. Am J Phys Anthropol 37:233-238.
Smith P. 1976. Dental pathology in fossil hominids: what did Neanderthals do with their teeth? Curr Anthropol 17:149-151.
Smith P. 1977. Selective pressures and dental evolution in hominids. Am J Phys Anthropol 47:453-458.
Smith P, Peretz B. 1986. Hypoplasia and health status: a comparison of two lifestyles. Hum Evol 1:535-544.
Smith TM, Toussaint M, Reid DJ, Olejniczak AJ, Hublin JJ. 2007. Rapid dental development in a middle Paleolithic Belgian Neanderthal. Proc Nat Acad Sci 104:20220-20225.
Smith TM, Tafforeau P, Reid DJ, Pouech J, Lazzari V, Zermeno JP, Guatelli-Steinberg D, Olejniczak AJ, Hoffman A, Radovčić J, Makaremi M, Toussaint M, Strnger C, Hublin JJ. 2010. Dental evidence for ontogenetic differences between modern humans and Neanderthals. Proc Nat Acad Sci 107:20923-20928.
Soficaru A, Doboş A, Trinkaus E. 2006. Early modern humans from the Peştera Muierii, Baia de Fier, Romania. Proc Nat Acad Sci USA 103:17196-17201.
Sognnaes RF. 1956. Histological evidence of developmental lesions in teeth originating from Paleolithic, prehistoric, and ancient man. Am J Pathology 32:547-577.
Sołtysiak A. 2012. Comment: Low dental caries rate in Neandertals: The result of diet or the oral flora composition? HOMO-J Compar Hum Biol 63:110-113.
Sørensen, B. 2011. Demography and the extinction of European Neanderthals. J Anthropol Archaeol 30:17-29.
Speth JC, Tchernov E. 2003. Paleoclimatic and behavioral implications of Middle Paleolithic tortoise use at Kebara Cave (Israel). In: Patou-Mathis M, Bocherens H (Eds). Le Rôle de l'Environnement dans les Comportements des Chasseurs-Cueilleurs Préhistoriques. Oxford: BAR International Series 1105, pp 9-21.
Speth JD, Widdowson EM, Oftedal OT, Foley RA, Van Soest P. 1991. Protein Selection and

Avoidance Strategies of Contemporary and Ancestral Foragers: Unresolved Issues [and Discussion]. Phil Trans Royal Soc London B: Bio Sci 334:265-270.
Spikins PA, Rutherford HE, Needham AP. 2010. From homininity to humanity: Compassion from the earliest archaics to modern humans. Time Mind 3:303-325.
Stamatiou J, Symons AL. 1991. Agenesis of the permanent lateral incisor: distribution, number and sites. J Clin Pedia Denti 15:244.
Starling A, and Stock J. 2007. Dental indicators of health and stress in early Egyptian and Nubian agriculturalists: a difficult transition and gradual recovery. Am J Phys Anthropol 528:520–528.
Steckel RH. 2003. Research project: A history of health in Europe from the late Paleolithic era to the present. Econo Hum Biol 1:139-142.
Steegmann AT, Cerny FJ, Holliday TW. 2002. Neandertal cold adaptation: physiological and energetic factors." Am J Hum Biol 14:566-583.
Stephen RM, Miller BF. 1943. A quantative method for evaluating physical and chemical agents which modify production of acids in bacterial plaques on human teeth. J Dent Res 22:45-51
Stiner MC. 1994. Honor Among Thieves. A Zooarchaeological Study of Neandertal Ecology. Princeton: Princeton Univesity Press.
Stiner MC. 2001. Thirty years on the "Broad Spectrum Revolution" and paleolithic demography. Proc Nat Acad Sci 98:6993-6996.
Stiner MC, Kuhn SL. 2006. Changes in the 'connectedness' and resilience of Paleolithic societies in Mediterranean ecosystems. Hum Ecol 34:693-712.
Stiner MC, Munro ND, Surovell TA, Tchernov E, Bar-Yosef O. 1999. Paleolithic population growth pulses evidenced by small animal exploitation. Science 283:190-194.
Størmer FC, Mysterud I. 2007. Cave smoke: Air pollution poisoning involved in Neanderthal extinction? Med Hypothes 68:723-724.
Straus LG. 1986. Once more into the breach: Solutrean chronology. Munibe 38:35-38.
Straus LG, editor. 1986. The End of the paleolithic in the Old World. British Archaeological Reports.
Straus LG. 1987. Hunting in late Upper Paleolithic Western Europe. In: Nitecki MH, Nitecki DV. The Evolution of Human Hunting. Springer US p 147-176.
Straus LG. 1995. The upper paleolithic of Europe: An overview. Evol Anthropol 4:4-16.
Stringer C. 2002. Modern human origins: progress and prospects." Philo Trans Royal Soc B: Biol Sci 357:563-579.
Stringer, 2012. Lone survivors: how we came to be the only humans on earth. New York: MacMillan.
Stringer, Chris, and Clive Gamble. 1993. In search of the Neanderthals: Solving the puzzle of human origins. London: Thames and Hudson.
Stringer CB, Finlayson JC, Barton RNE, Fernández-Jalvo Y, Cáceres I, Sabin RC, Rhodes EJ. 2008. Neanderthal exploitation of marine mammals in Gibraltar. Proc Nat Acad Sci 105:14319-14324.
Ströhle A, Hahn A. 2011. Diets of modern hunter-gatherers vary substantially in their carbohydrate content depending on ecoenvironments: results from ethnographic analysis. Nutr Res 31, 429-435.
Svoboda J. 2008. Petřkovice: On shouldered points and female figurines. Academy of Sciences of the Czech Republic, Institute of Archaeology at Brno.

Tappen NC. 1985. The dentition of the “old man” of La Chapelle-aux-Saints and inferences concerning Neandertal behavior. Am J Phys Anthropol 67: 43-50.
Tattersall I. 1999. *The Last Neanderthal*. New York: Westview Press.
Tayles N, Domett K, Halcrow S. 2009. Can dental caries be interpreted as evidence of farming? The Asian experience. 2009:162-166.
Temple DH, Kusaka S, Sciulli PW. 2011. Patterns of social identity in relation to tooth ablation among prehistoric Jomon foragers from the Yoshigo site, Aichi prefecture, Japan. Int J Osteoarchaeol 21:323-335.
Templeton A. 2002. Out of Africa again and again. Nature 416:45-51.
Teschler-Nicola, M, Czerny C, Oliva M, Schamall D, Schultz M. 2006. Pathological alterations and traumas in the human skeletal remains from Mladeč. In: Teschler-Nicola, editor. Early Modern Humans at the Moravian Gate Vienna: Springer p 473-489.
Theirry M., Granat J, Vermelin L. 2008. Dental agenesis: etiology, evolution and treatment options.
Tilley L. 2012. The bioarchaeology of care. The SAA Archaeol Rec 12:39-41.
Tillier AM, B. Arensberg B, Duday H. I989. La mandibule et les dents du Neanderthalien de Kebara (Homo 2), Mont Carmel, Israel. Paleorient 15:39-58.
Tillier AM, Arensburg B, Rak Y, Vandermeersch B. 1995. Middle Palaeolithic dental caries: new evidence from Kebara (Mount Carmel, Israel). J Hum Evol 29:189-192.
Tillier AM, Duday H, Arensburg B, Vandermeersch B. 2004. Dental pathology, stressful events, and disease in Levantine early anatomically modern humans: evidence from Qafzeh. In: Goren-Inbar N, Spth JD. Human paleoecology in the Levantine Corridor. Oxbow Books, p 135-148.
Tomczyk J. 2012. Comments on Soltysiak's paper:“Comment: Low dental caries rate in Neandertals: The result of diet or the oral flora compositions? HOMO-J Compar Hum Biol. 63:311-314
Topić B, Raščić-Konjhodžić H, Cižek Sajko M. 2012. Periodontal disease and dental caries from Krapina Neanderthal to contemporary man - skeletal studies. Acta Med Acad. 41:119–30.
Torrent, JMU. 1997. Història de l'odontologia. Barcelona: Edicions Universitat.
Toussaint M, Semal P, Pirson S. 2011. Les Néandertaliens du bassin mosan belge: bilan 2006-2011. Le Paléolithique moyen en Belgique. Mélanges Marguerite Ulrix-Closset. Bulletin de la Société belge d'études Géologiques et Archéologiques Les Chercheurs de la Wallonie, hors série. 4:149-196.
Trevathan WR. 2007. Evolutionary Medicine. Annu Rev Anthropol. 36:139–154.
Trinkaus E. 1983. The Shanidar Neandertals. New York: Academic Press
Trinkaus E. 1985. Pathology and the Posture of the La Chapelle-aux-Saints Neandertal. Am J Phys Anthropol 67:19-41.
Trinkaus E. 1995. Neanderthal Mortality Patterns. J Arch Sci 22:121-142.
Trinkaus E. 2005a. Early modern humans. Annu Rev Anthropol. 34:207-30.
Trinkaus E. 2005b. History of Disability: Pleistocene Period. Encyclopedia of Disability.
Trinkaus E. 2011. Late Pleistocene adult mortality patterns and modern human establishment. Proc Nat Acad Sci 108:1267-1271.
Trinkaus E. 2013. The Paleobiology of Modern Human Emergence. In: Smith FH, Ahern JC. The Origins of Modern Humans: Biology Reconsidered. New York:John Wiley &

Sons, p 393-434.
Trinkaus E, Buzhilova AP. 2012. The death and burial of Sunghir 1. Internat J Osteoarch 22:655-666
Trinkaus E, Pinilla B. 2009. Dental caries in the Qafzeh 3 Middle Paleolithic modern human. Paléorient 35:69-76.
Trinkaus E, Shipman P. 1993. The Neandertals: Changing the image of mankind. New York: Jonathan Cape.
Trinkhaus E, Svoboda J. Editors. 2006. Early Modern Human Evolution in Central Europe: The People of Dolní Věstonice and Pavlov. Oxford University Press.
Trinkaus E, Churchill SE, Ruff CB. 1994. Postcranial robusticity in Homo. II: Humeral bilateral asymmetry and bone plasticity. Am J Phys Anthropol 93:1-34.
Trinkaus E, Smith RJ, Lebel S. 2000. Dental caries in the Aubesier 5 Neandertal primary molar. J Archaeol Sci 27:1017-1021.
Ttinkaus E, Bailey SE, Zilhão J. 2001. Upper Paleolithic human remains from the Gruta do Caldeirão, Tomar, Portugal. Revista Portuguesa de Arqueologia, 4:5-17.
Trinkaus E, Hillson SW, Franciscus RG, Holliday TW. 2006. Skeletal and Dental Paleopathology. In: Trinkaus E, Svoboda J. editors. Early modern human evolution in Central Europe : the people of Dolní Věstonice and Pavlov. Oxford: Oxford University Press.
Trinkaus E, Moldovan O, Bîlgăr A, Sarcina L, Athreya S, Bailey SE, Rodrigo R, Mircea G, Higham T, Bronk Ramsey C, van der Plicht J. 2003. An early modern human from the Peştera cu Oase, Romania. Proc Nat Acad Sci 100:11231-11236.
Ungar PS, Grine FE, Teaford MF, Pérez-Pérez A. 2001. A review of interproximal wear grooves on fossil hominin teeth with new evidence from Olduvai Gorge. Archiv Oral Biol 46:285-292.
Valladas H, Cachier H, Maurice P, de Quirost FB, Clottes J, Cabrera Valdes V, Uzquiano P, Arnold M. 1992. Direct radiocarbon dates for prehistoric paintings at the Altamira, El Castillo and Niaux caves. Nature 357:68-70.
Van Andel TH, Davies W. 2003. Neanderthals and modern humans in the European landscape during the last glaciation: archaeological results of the Stage 3 Project. McDonald Institute for Archaeological Research monographs.
Vandermeersch B. 1981. Les Hommes Fossiles de Qafzeh (Israël). Paris: CNRS.
Vandermeersch B, Arensberg B, Tillier AM, Rak Y, Weiner S, Spiers M. 1994. Middle Paleolithic dental bacteria from Kebara, Israel. C R Acad Sci Paris 319:727-731.
Varrela TM, Paunio K, Wouters FR, Tiekso J, Söder P-Ö. 1995. The relation between tooth eruption and alveolar crest height in a human skeletal sample. Arch Oral Biol 40:175-180.
Vercellotti G, Alciati G, Richards MP, Formicola V. 2008. The Late Upper Paleolithic skeleton Villabruna 1 (Italy): a source of data on biology and behavior of a 14.000 year-old hunter. J Anthropol Sci 86:143-163.
Vesica PL, Tuccimei P, Turi B, Fornós JJ, Ginés A, Ginés J. 2000. Late Pleistocene paleoclimates and sea-level change in the Mediterranean as inferred from stable isotope and U-series studies of overgrowths on speleothems, Mallorca, Spain." Quatern Sci Rev 19:865-879.
Villa G, Giacobini G. 1996. Neandertal teeth from alpine caves of Monte Fenera (Piedmont, Northern Italy): Description of the remains and microwear analysis. Anthropologie

34:55-67.
Villotte S, Churchill SE, Dutour OJ, Henry-Gambier D. 2010. Subsistence activities and the sexual division of labor in the European Upper Paleolithic and Mesolithic: Evidence from upper limb enthesopathies. J Hum Evol 59:35-43.
Walker M, Johnsen S, Rasmussen SO, Popp T, Steffensen JP, Gibbard P, Hoek W, Lowe J, Andrews J, Björck S, Cwynar LC, Hughen K., Kershaw P, Kromer B, Litt T, Lowe DJ, Nakagawa T, Newnham R, Schwander J. 2009. Formal definition and dating of the GSSP (Global Stratotype Section and Point) for the base of the Holocene using the Greenland NGRIP ice core, and selected auxiliary records. J Quaternary Sci 24:3–17.
Walker MJ, Gibert J, Sánchez F, Lombardi AV, Serrano I, Gómez A, Eastham A, Ribot F, Arribas A, Cuenca A, Gibert L, Albadalejo S, Andreu JA. 1999. Excavations at new sites of early man in Murcia: Sima de las Palomas del Cabezo Gordo and Cueva Negra del Estrecho del Río Quípar de la Encarnación. Hum Evol 14:99-123.
Walker MJ, Zapata J, Lombardi AV, Trinkaus E. 2011. New evidence of Dental Pathology in 40,000 Year Old Neandertals. J Dent Res 90:428-432.
Walker PL, Dean G, Shapiro P. 1991. Estimating Age from Tooth Wear in Archaeological Populations. In: Kelley MA, Larsen CS editors. Advances in Dental Anthropology. New York: Wiley-Liss p 169-178.
Wallace JA. 1977. Gingival eruption sequences of permanent teeth in early hominids. Am J Phys Anthropol 46:483-493.
Wallace JA, Barrett MJ, Brown T, Brace CL, Howells WW, Koritzer RT, Sakura H, Stoukal M, Wolpoff MH, Žlábek K. 1975. Did La Ferrassie I use his teeth as a tool?[and comments and reply]. Curr Anthropol 1975:393-401.
Wang X, Shaffer JR, Weyant RJ, Cuenco KT, DeSensi RS, Crout R, McNeil DW, Marazita ML. 2010. Genes and Their Effects on Dental Caries May Differ between Primary and Permanent Dentitions. Caries Res 44:277-284.
Ward SC, Johanson DC, Coppens Y. 1982. Subocclusal morphology and alveolar process relationships of hominid gnathic elements from the Hadar Formation: 1974–1977 collections. Am J Phys Anthropol 57:605-630.
Warinner C. 2013. Ancient DNA and the Metagenomics of Disease *(*abstract). Am J Phys Anthropol Supp S56:284-285.
Warinner C, Matias Rodrigues JF, Vyas R, Trachsel C, Shved N, Grossmann J, Radini A, Hancock Y, Tito RY, Fiddyment S, Speller C, Hendy J, Charlton S, Luder HU, Salazar-Garcia DC, Eppler E, Seiler R, Hansen LH, Samaniego Castuita JA, Barkow-Oesterreicher S, Teoh KY, Kelstrup CD, Olsen JV, Nanni P, Kawai T, Willersley E, von Mering C, Lewis CM Jr, Collins MJ, Gilbert MTP, Rühli F, Cappellini E. 2014. Pathogens and host immunity in the ancient human oral cavity. Nature Genetics 46:336-344.
Warren JJ, Cowen HJ, Watkins CM, Hand JS. 2000. Dental caries prevalence and dental care utilization among the very old. Journal Am Dent Assoc 131:1571-1579.
Wasterlain SN, Cunha E, Hillson S. 2011. Periodontal disease in a Portuguese identified skeletal sample from the late nineteenth and early Twentieth Centuries. Am J Phys Anthropol. 145:30-42.
Watson JT, Fields M, Martin DL. 2010. Introduction of agriculture and its effects on women's oral health." Am J Hum Biol 22:92-102
Watt ME, Lunt DA, Gilmour WH. 1997. Caries prevalence in the permanent dentition of a

mediaeval population from the Southwest of Scotland. Archs Oral Biol 42:601-620.
Weaver TD. 2003. The shape of the Neandertal femur is primarily the consequence of a hyperpolar body form. Proc Nat Acad Sci 100:6926-6929
Webb WS. 1946. Indian Knoll site, Oh 2, Ohio County, Kentucky. University of Kentucky Reports in Archaeology and Anthropology 4: 115-365.
Wells C. 1975. Prehistoric and historical changes in nutritional diseases and associated conditions. Progress Food Nutri Sci 1:729-779.
White R, Arts N, Bahn PG, Binford LR, Dewez M, Dibble HL, Fish PR. Rethinking the Middle/Upper Paleolithic Transition [and Comments and Replies]. Curr Anthropol 1982:169-192.
White TD. 1988. The comparative biology of "robust" Australopithecus: clues from context. In: Grine F, editor. Evolutionary History of the 'Robust' Australopithecines. New Brunswick: Transaction Books p 449-484.
White TD, Asfaw B, DeGusta D, Gilbert H, Richards GD, Suwa G, and Howell FC. 2003. Pleistocene *Homo sapiens* from Middle Awash, Ethiopia. Nature 423:742-747
Whittaker DK, Molleson T, Daniel AT, Williams JT, Rose P, Resteghini R. 1985. Quantitative assessment of tooth wear, alveolar-crest height and continuing eruption in a Romano-British population. Arch Oral Biol 30:493-501.
Whittaker DK, Griffiths S, Robson A, Roger-Davies P, Thomas G, Molleson T. 1990. Continuing tooth eruption and alveolar crest height in an eighteenth-century population from Spitalfields, east London. Arch Oral Biol 35:81-85.
Wiebe CB, Putnins EE. 2000. The periodontal disease classification system of the American Academy of Periodontology-an update. J Canad Dent Assoc 66:594-599.
Williams G, Nesse R. 1991. The Dawn of Darwinian Medicine. Q Rev Biol 66:1–22.
Williams RC, Barnett AH, Claffey N, Davis M, Gadsby R, Kellett M, Lip GYH, Thackray S. 2008. The potential impact of periodontal disease on general health: a consensus view. Curr Med Res Opin 24:1635–1643.
Wintergerst ES, Maggini S, Hornig DH. 2007. Contribution of selected vitamins and trace elements to immune function. Ann Nutri Metabolism 51:301-323.
Winters HD. 1974 Introduction to the New Edition. In: Webb WS, ed. Indian Knoll. Knoxville: University of Tennessee Press, pp. v-xxvii.
Wittwer-Backofen U, Tomo N. 2008. From health to civilization stress? In search of traces of a health transition during the early Neolithic in Europe. In: Bocquet-Appel JP, Bar-Yosef O, editors. The Neolithic Demographic Transition and its Consequences. Dordrecht: Springer p 501-538.
Wolpoff MH. 1979. The Krapina dental remains. Am J Phys Anthropol 50:67-113.
Wood JW, Milner GR, Harpending HC, Weiss KM, Cohen MN, Eisenberg LE, Hutchinson D Jankaukas R, Cesnys G, Katzenberg MA, Lukacs JR, McGarth JW, Roth EA, Ubelaker DH, Wilkinson RG. 1992. The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples [and Comments and Reply]. Curr Anthropol 33:343-370.
Wood JW, Milner GR, Harpending HC, Weiss KM, Cohen MN, Eisenberg LE, Hutchinson DL. Jankauskas R, Česnys G, Katzenberg MA, Lukacs JR, McGrath JW, Roth EA, Ubelaker DH, Wilkinson RG. The osteological paradox: problems of inferring prehistoric health from skeletal samples [and comments and reply]. Curr Anthropol 33:343-370.

Worthman C, Kohrt B. 2005. Receding horizons of health: biocultural approaches to public health paradoxes. Soc Sci Med 61:861–78
Wrangham, R., 2009. Catching Fire: How Cooking Made Us Human. New York: Basic Books.
Wu XJ, Schepartz LA, Liu W, Trinkaus E. 2011. Antemortem trauma and survival in the late Middle Pleistocene human cranium from Maba, South China. Proc Nat Acad Sci 108:19558-19562.
Wu L, Xianglong Z. 1996. The degeneration of third molars and its significance to human evolution. Acta Anthropologica Sinica 15:185-199.
Wu L, Zeng X. 1996. The degeneration of third molars and its significance to human evolution. Acta Anthropologica Sinica 15:185-199.
Wu XJ, Xing S, Trinkaus E. 2013. An Enlarged Parietal Foramen in the Late Archaic Xujiayao 11 Neurocranium from Northern China, and Rare Anomalies among Pleistocene Homo. PloS one 8:e59587.
Zheng J, Yang B, Tuomasjukka S, Ou S, Kallio H. 2009. Effects of latitude and weather conditions on contents of sugars, fruit acids, and ascorbic acid in black currant (Ribes nigrum L.) juice. J Agricul Food Chem57:2977-2987.
Zias J, Numeroff K. 1986. Ancient dentistry in the Eastern Mediterranean: a brief review. Israel Exploration J 65-67.
Zuk M. 2013. Paleofantasy: What Evolution Really Tells Us about Sex, Diet, and How We Live. New York: WW Norton & Company.

Appendix 1: Site descriptions

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
Neandertals						
Belgium	Goyet	River Samson, near Namèche, Mozet, Namur	Mousterian	Cold, Mammoth	3	Oakley et al., 1971; Toussaint et al., 2011
	La Naulette	River Lesse, near Dinant, Hulsonniaux, Namur	150,000	Cold, Mammoth	1	Oakley et al., 1971; Toussaint et al., 2011
	Spy	Betche-aux-Rotches, Spy, Namur	OIS 3	Cold, Mammoth	1,2	Oakley et al., 1971
Croatia	Vindija	Donja Voća, Croatia	42,000 BP	Temperate		Janković et al., 2006
Czech Republic	Kulna	Near Sloup village, 35 km N Brno	45,660 (+2,850-2,200)BP	Cold, Mammoth	1	Jelínek & Orvanová, 1999
	Ochoz	Near Ochoz village	OIS 4	Cold	1	Jelínek & Orvanová, 1999
England:	Boxgrove	Dover, England	Early Middle Pleistocene, MIS 13/12	Cold	2,3	Hillson et al., 2010
France:	La Chapelle-aux-Saints	40 km SE of Brive, Corrèze	Würm II, Mousterian	Cold, Rangifer	1	Oakley et al., 1971
	Les Fadets	Lussac-les-Chateaux	Mousterian	Cold	1-4	Lacy et al., *in prep*
	La Ferrassie	40 km SE of Périgueux	OIS 4	Cold, Wholly Rhino	1,2	Gambier & Houet, 1993

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	La Quina	Gardes-le-Pontaroux, 25 km S of Angoulême, Charente	OIS 4-3	Cold, Rangifer	5	Oakley et al., 1971
	Malarnaud	Arize, Montseron, NE of St Girons, Ariège	Possibily Riss-Würm	Temperate	1	Oakley et al., 1971
	Monsempron	Monsempron, 25 km NE of Villeneuve-Sur-Lot	OIS 4	Cold, Rangifer	2,3,4	Oakley et al., 1971
	Montmaurin	La Niche, Montmaurin, 19 km N of Saint-Gaudens, Haute-Garonne	Possibly Mindel-Riss	-	1	Oakley et al., 1971
	Petit-Puynoyen	Puymoyen, 5 km S of Angoulême, Charente	OIS 3	Cold, Rangifer	1,2,3,4	Oakley et al., 1971
	Regourdou	Montignac sur Vézère, 50 km E of Périgueux. Dordogne	45500 +/- 1800 BP	Cold	1	Oakley et al., 1971; d'Errico et al., 2011
Germany	Neanderthal	Neander Valley, 12km E of Düsseldorf	38,000-44,000 BP	Cold	2, new teeth	Orschiedt, 2000
Gibraltar	Genista	W side of Windmill Flats at S end of Gibraltar	Upper Pleistocene	Temperate	1	Oakley et al., 1971
	Devil's Tower	North Front, 350 m E-SE of Forbes' Quarry	30,000 BP	Temperate	Gibraltar 2	Oakley et al., 1971; d'Errico et al., 2011
	Forbes' Quarry	W end of North Front, Gibraltar	Würm?	Temperate	Gibraltar 1	Oakley et al., 1971
Israel	Amud	Northwest Sea of Galilee	OIS 3	Warm, Gazelle	1	Rabinovich & Hovers, 2004
	Kebara	Mount Carmel, Israel	OIS 4/3	Warm, Gazelle	-	Speth & Tchernov, 1998

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	Tabun	Mount Carmel, Israel	OIS 6/5	Temperate	-	Jelinek et al., 1973; Albert et al., 1999
Italy	Archi	Archi, Reggio Calabria	>40,000 BP	Temperate	1	Orban, 1988
	Castel di Guido	20 km NW of Rome	Riss	Temperate	4	Orban, 1988; Mallegni
	Ciota Ciara & Ciutarun	Monte Fenera, Borgosesia, Piedmont, NW Italy	Mousterian	Cold, Alpine	1-4	Villa & Giacobini, 1996
	Caverna dell Fate	4 km NE of Finale Ligure, Savona	75,000-82,000 BP	Temperate	2,3	Giacobini et al., 1984; Orban, 1988
	Fossellone	San Felice Circeo, Latina	25380 +/- 1060 BP	Temperate	3	Bietti & Manzi, 1991; d'Errico et al., 2011
	Guattari	300 m SE of San Felice Circeo, Latina	Late Würm I/Early Würm II	Temperate	1,2,3, loose teeth	Oakley et al., 1971
	Saccopastore	River Aniene, 3.5km from Porta Pia, Rome	OIS 5e	Temperate	1,2	Oakley et al., 1971
	San Bernardino	Colli Berici, near Mossano, Vicenza	Würm	Temperate		Oakley et al., 1971
Spain	Lezetxiki	Lezetxiki, Mondragón, Guipuzcoa	OIS 3	Temperate	1,2	Baldeón, 1987; Orban, 1991; d'Errico & Sánchez Goñi, 2003
	Palomas	Murcia, SE Spain	Late Middle/Early Upper Paleolithic	Temperate	Many	Walker et al., 1999

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	Cova Negra	near Xátiva, Valencia	28900 +/- 5600 BP	Temperate	1-6	Arsuaga et al., 1989; Orban, 1991; Walker et al., 1999; d'Errico et al., 2011
	El Sidron	Malagá, Spain	43,000 BP	Temperate	1-6, 1-3 adolescent	Rosas et al., 2006
	Zafarraya:	Zafarraya, 35 km NE of Málaga	OIS 3: 33,500 years BP	Temperate	2	Orban, 1991; Barroso Ruiz & de Lumley, 2006
Middle Paleolithic Modern Humans						
Israel	Skhul:	Mount Carmel	OIS 5: 130,000 - 100,000 years BP	Warm, Gazelle	5	Schwartz & Tattersall, 2003
	Qafzeh:	near Nazareth, Israel	OIS 5: 120,000 - 90,000 years BP	Temperate, Gazelle	Many	Vandermeersch, 1981; Schwartz & Tattersall, 2003
Early Paleolithic Modern Humans						
Austria	Miesslingtal	Spitz, Lower Austria	Post Würm II	Cold, Rangifer	1	Oakley et al., 1971
	Willendorf	23 km N of Willendorf Station, Lower Austria	41700 +/- 3700 BP	Cold, Mammoth	1	Oakley et al., 1971; d'Errico et al., 2011
Czech Republic	Brno	Center of Town, Brno	28550 +/- 320 BP	Cold	Brno 2	Jelínek & Orvanová, 1999; d'Errico et al., 2011
	Dolní Věstonice	Near town of Dolní Věstonice	26,640+-110 BP	Cold	3,13,14,15, 16, loose teeth	Jelínek & Orvanová, 1999, Klíma 1995

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	Mladeč	Mladeč village	34930 +/- 520 BP	Cold	Full cranium, loose teeth, 8 5457, Lautsch 2	Jelínek 1987, Jelínek & Orvanová, 1999; d'Errico et al., 2011
	Pavlov	Pavlov hills, near town of Dolní Věstonice	26,620+-230 BP	Cold, Mammoth	1,2,3, loose teeth	Klíma & Kukla, 1963; Jelínek & Orvanová, 1999;
	Předmostí	W part of Předmostí	~26,000 BP (based on Pavlov dates)	Cold, Mammoth	Whole & Hemi mandible (both given # A 17 088)	Jelínek & Orvanová, 1999
France	Abri Labatut	Labatut, Castelmerle Valley	25,000 and 18,000 years BP	Cold	1,2	Oakley et al., 1971; Simek, 1986
	Abri Pataud	Les Eyzies-de-Tayac	26,900-25,500 BP	Cold	1	Oakley et al., 1971; Movius 1963; Pottier, 2005
	Les Battuts	River Aveyron, 22 km E-NE of Montauben, Penne	Recent Würm	Cold	1	Gambier & Houet, 1993
	Blanchard	Blanchard, Castelmerle	Early Upper Paleolithic	-	1	Oakley et al., 1971
	Brassempouy	35km S-SE of Mont-de-Marsan, Brassempouy	28,000-32,000 BP	Cold	-	Gambier & Houet, 1993
	Castanet	Castanet, Castelmerle valley, 10 km SW of Montignac, Sergeac, Dordogne	Würm III	Cold, Rangifer	1	Oakley et al., 1971

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	Cro-Magnon	Les Eyzies-de-Tayac	27,680 +/- 270 yr bp	Cold, Rangifer	1 to 4	Oakley et al., 1971
	Grotte des Abeilles	Montmaurin site, 19 km N of Saint Gaudens Massif of Lespugue, Montmaurin	Recent Würm	Cold	1,2,3	Gambier & Houet, 1993
	Lachaud	Terrasson, 18 km W of Brive, Dordogne	OIS 3	Cold, Rangifer	Found 1980	Oakley et al., 1971
	Oreille d'Enfer	Val d'Enfer, Les Eyzies-de-Tayac	Würm?	Cold	1	Pradel, 1959; Oakley et al., 1971
	La Rochette	Saint Léon sur Vézère, 10 km SW of Montignac, Dordogne	23.630 ± 130 B.P.	Cold	1	Oakley et al., 1971; Orschiedt, 2002
	Les Rois	River Boëme, Mouthiers, 13 km S of Angoulême	OIS 3: 30,000 to 28,000 years BP	Cold, Mammoth	Many	Oakley et al., 1971; Ramirez Rozzi et al., 2009
	La Tannerie	Lussac-les-Chateaux	18,020±270	Cold	1-3	Straus, 1986
	La Vachons	River Boëme, Voulgezac, 14 km S of Angoulême	Würm III	Cold	1	Oakley et al., 1971
Italy	Baousso de Torre (Grimaldi)	Destroyed cave 6th from W, Balzi Rossi, 5 km W of Ventimiglia	Late Pleistoc.	Temperate	-	Oakley et al., 1971
	Barma Grande	5th of W, 5 km W of Balzi Rossi	24,800 +/- 800 BP	Temperate	2,3,4 (or 3,4,5)	Oakley et al., 1971; d'Errico et al., 2011
	Caviglione (Grimaldi)	4th from W, 5 km W of Ventimiglia	Late Pleistoc.	Temperate	1	Oakley et al., 1971
Romania	Muierii	Baia de Fier, Romania	35 ka cal BP	Temperate	1	Soficaru et al., 2006

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	Oase	Southwestern Carpathian Mountains, Romania	34,000–36,000 ^{14}C years B.P	Temperate	1,2	Trinkaus et al., 2003
Spain	Aitzbitarte III	Guipuzcoa, Spain	25 000 et 23 000 BP	Temperate	1	Foucher et al., 2002
Late Paleolithic Modern Humans						
Croatia	Romualdo	near Rovinj, Istria	Würm III	Temperate	1	Oakley et al., 1971
	Sandalja	4 km NE of Pula, Southern Istria	12,320+-100 BP	Cold, Rangifer	2	Oakley et al., 1971
	Vindija	Donja Voća, Croatia	OIS 2: 22,000 - 17,500 years BP	Temperate, but Rangifer present	Many	Janković et al., 2006
England	Gough's Cave	Cheddar Gorge, Somerset	11,900-12,800 BP	Cold, Rangifer	1,4,6,86,87, 87 253, loose teeth	Orban, 1990
	Tornewton	Torbryan Valley, Devonshire	Late Upper Paleolithic	Cold, Rangifer	1	Orban, 1990
France	Bois-Ragot	Near Lussac-les-Châteaux	11,000 BP	Cold	2	Gambier & Houet, 1993
	Bruniquel	Aveyron valley, 22 km E-NE of Montauban, Tarn Penne	11,750+-300 BP	Cold, Rangifer	1	Oakley et al., 1971; Barket et al., 1969
	Chancelade	Beauronne river, Chancelade, 6 km NW of Périgueux, Dordogne	Würm IV	Cold, Rangifer	1	Oakley et al., 1971
	Font de Gaume	1 km from Les Eyzies-de-Tayac	Beginning of recent Würm	Cold	-	-
	Fourneau du Diable	20 km NW of Périgueux	Recent Würm	Cold	1,2,3,5	Gambier & Houet, 1993

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	La Greze	6 km from Les Eyzies-de-Tayac	Recent Würm to Holocene	Cold	1	Gambier & Houet, 1993
	La Gravette	Couze River, 18 km E-SE of Bergerac, Dordogne	Würm III	Cold, Rangifer	1	Oakley et al., 1971
	Isturitz	Saint-Germain-la-Rivière	OIS 3/2	Cold, Rangifer	None of the #s match	Gambier & Houet, 1993
	Lalinde	Lalinde, 15 km E Bergerac, Dordogne	12,540 BP	Cold	1	Oakley et al., 1971; d'Errico et al., 2011
	Laugerie-Basse	Les Eyzies-de-Tayac	OIS 2: 15,000 to 12,000 years BP	Cold, Rangifer	1	Oakley et al., 1971; Gambier et al., 2000
	Les Peyregues	2.5 km NE of Cabrerets, Orgnac	13,020+-140 BP	Cold	-	Gambier & Houet, 1993; Allard, 1992
	Limeuil	30 km E of Bergerac, Limeuil	11,720 BP	Cold	3	Gambier & Houet, 1993; d'Errico et al., 2011
	Lussac les Chateau	Lussac les Châteaux	Beginning Würm IV	Cold, Rangifer	2, 5?	Oakley et al., 1971
	La Madeleine:	4 km N-NE of Les Eyzies-de-Tayac	12,070-12,750 BP	Cold	4	Gambier & Houet, 1993
	Le Morin	Le Moustelate, 50 km E of Bordeaux	Recent Würm	Cold	1	Gambier & Houet, 1993
	Moulin Neuf	Canodonne valley, Saint-Quentin-de-Baron	14,280-13,570	Cold	2	Gambier & Houet, 1993; Lenoir, 1983
	Pech de la Boissiere	7 km SE of Sarlat, Carsac	Recent Würm	Cold	2	Gambier & Houet, 1993
	La Piscine	Montmorillon, Vienne	Recent Würm	Cold	Un-numbered	Gambier & Houet, 1993

Temporal Group: Country	Site	Location	Date(s)	Temperate/ Cold	Specimens Examined	Citations
	Roc de Combe Capelle	Couze River, 20 km E of Bergerac	Recent Würm	Cold	3	Gambier & Houet, 1993
	Saint Germain la Rivière	River Dordogne, 10 km from Libourne, Saint-Germain-la-Rivière	15,300+-410 BP	Cold, Rangifer	4,8-16	Gambier & Houet, 1993; Lenoir et al., 1991
Germany	Oberkassel	Oberkassel, 4 km SE of Bonn	1: 11,570+-100 BP; 2: 12,180+-110 BP	Cold	1,2	Orschiedt, 2000
Israel	Ein Gev	East Sea of Galilee	13.750 BC	Temperate	1	Arensburg & Bar-Yosef, 1973
	Nahal Ein Gev	East Sea of Galilee	Late Upper Paleolithic	Temperate	1	Belfer-Cohen et al., 2004
	Ohalo	Near the Sea of Galilee	OIS 2: 23,500 to 22,500 years BP	Warm, Gazelle	1,2	Nadel & Hershkovitz, 1991; Nadel et al., 2006
Italy:	Continenza	Trasacco L'Aquila, Italy	11,500 +- 120 BP	Temperate	4-6	Astuti, 2002
	Romanelli	5 km S of Lecce, Puglia	Late Würm, 10,000-11,000 BP	Temperate	1,4-8, loose teeth	Oakley et al., 1971
	Tagliente	Near Stallavena, Valpantena, Verona	Würm	Temperate	1	Oakley et al., 1971
	Villabruna:	Belluno, Italy	12,150 BP	Temperate	1	D'Errico et al., 2011
Luxembourg	Oetrange	Grotte de Schleid, 8 km E of Luxembourg	Würm	Cold, Mammoth	1	Oakley et al., 1971
Spain	Nerja	Nerja village, Málaga	Epi-paleolithic?	Temperate	Pepita	Garcia Sanchez, 1982; Orban, 1991

Appendix 2: Pathological Diagnoses (alphabetical order)

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
Abri Labatut	EMH	A	E	Y								2	1
Abri Pataud	EMH	A	E	Y		UM2	0	2				16	1
Abri Sur Cure 27	EMH	A	E	Y			0	0				3	1
Aitzbitarte III	EMH	A	E	2 A, 1 E								3	3
Amud (1?)	Nean	M	N	P	UI2	UI2	2	3				32	1
Archi	Nean	M	N	A			0	0				5	1
Baousso de Torre 1 adult	EMH	A	E	Y								4	1
Baousso de Torre 3 immature	EMH	A	E	A								2	1
Barma Grande 2	EMH	M	E	Y	LM2		0	1				30	1
Barma Grande 3	EMH	M	E	Y			1	0				12	1
Barma Grande 4	EMH	M	E	E		UP3	2	3	2	2		18	1
Blanchard	EMH	A	E	A								3	2
Bois-Ragot	EMH	A	L	Y	LM2							1	1
Boxgrove	Pre-Nean	A	N	Y								2	1
Brassempouy	EMH	A	E	Y								4	1
Brno II	EMH	C	E	E		LM1	3	3	0	1		6	1
Bruniquel (Abri Lafaye)	EMH	A	L	P	LM2		1	3				30	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
Bruniquel 539	EMH	A	L	P			1	1	0	0	1	1	1
Castanet 1935-1-1	EMH	A	E	E								1	1
Castel di Guido 4	Pre-Nean	M	N	P		2						2	1
Caverna dell Fate adult	Nean	M	N	E			1	2				1	1
Caverna dell Fate child	Nean	M	N	A			0	0				1	1
Caviglione	EMH	A	E	P								32	1
Chancelade	EMH	A	L	E		2	3	3	17	19	2	9	1
Ciota Ciara 2	Nean	M	N	Y								1	1
Ciota Ciara 3	Nean	M	N	Y								1	1
Ciutarun 1 (formerly Fenera 4)	Nean	M	N	Y								1	1
Continenza 4	EMH	M	L	P	UM		1	2				8	1
Cro Magnon 1	EMH	A	E	?		LI2, LP3, LP3, LM1, LM2, UM1, UP3, UP4						0	1
Cro Magnon 2	EMH	A	E	Y								2	1
Cro Magnon 3	EMH	A	E	?								0	1
Cro Magnon 4	EMH	A	E	P	LM2	UP3	2	3				5	1
Cueva Negra	Nean	M	N	P								6	1
Dolni Vestonice	EMH	C	E	Y	LM		1	2				26	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
13													
Dolni Vestonice 14	EMH	C	E	Y			1	2				30	1
Dolni Vestonice 15	EMH	C	E	Y	LM3		1	2				30	1
Dolni Vestonice 16	EMH	C	E	E		2UM1, UP4, UI2, UI1, UI2, UM1, UM2, UM3	2	3			1	28	1
Dolni Vestonice 3	EMH	C	E	P			2	3				31	1
DV loose teeth	EMH	C	E	A, Y								5	2
Ein Gev	EMH	M	L	P		LM1	1	2	0	1		2	1
El Sidron Adolescente 1	Nean	A	N	A			0	0				13	1
El Sidron Adolescente 2	Nean	A	N	A								20	1
El Sidron Adolescente 3	Nean	A	N	A								12	1
El Sidron Adulto 1	Nean	A	N	P			2	1				27	1
El Sidron Adulto	Nean	A	N	Y		LM3,	1	3				23	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
2						LM1, LC, LI2							
El Sidron Adulto 3	Nean	A	N	Y			1	1				11	1
El Sidron Adulto 4	Nean	A	N	Y			1	0				23	1
El Sidron Adulto 5	Nean	A	N	Y			0	1				25	1
El Sidron Adulto 6	Nean	A	N	Y								15	1
Font de Gaume FG1 et 2	EMH	A	L	A								2	1
Fossellone	Nean	M	N	Y			0	1				2	1
Fourneau du Diable	EMH	A	L	Y			1	1				8	2
Genista	Nean	A	N	A								1	1
Gibralter 1	Nean	M	N	E		UP4, UC	3	3	3	3		9	1
Gibralter 2	Nean	M	N	A			0	2	1	1		7	1
Gough's Cabe 86	EMH	A	L	Y			0	1	1	1	1	13	1
Gough's Cave 1	EMH	A	L	Y	UM1	U5	1	2	5	5		20	1
Gough's Cave 4	EMH	A	L	A			0	0				1	1
Gough's Cave 6	EMH	A	L	P		LM2	2	1	0	0	2	1	1
Gough's Cave 87 253	EMH	A	L	Y			1	0			1	2	1
Gough's Cave	EMH	A	L	A			0	1				11	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
87?													
Gough's Cave loose teeth	EMH	A	L	Y								5	1
Goyet	Nean	A	N	Y			2	2				2	1
Grotte des Abeilles MNP 1989-5-2	EMH	A	E	A	LM1							3	1
Grotte des Rois 1955-148	EMH	A	E	A			1	2				8	1
Guattari 1	Nean	M	N	E?					16	16		0	1
Guattari 2	Nean	M	N	P					1	3		1	1
Guattari 3	Nean	M	N	P			2	3				11	1
Guattari 4	Nean	M	N	Y								3	1
Isturitz 111-1936	EMH	A	E	Y		2U	1	2			1	5	3
Isturitz 1950-10-2	EMH	A	E	Y			0	1				2	1
Isturitz 1950-11-1	EMH	A	E	E		LM1	2	2	0	1		3	1
Isturitz 1950-4-1	EMH	A	E	P		LM1	1	2	0	1		7	1
Kebara 2	Nean	M	N	P			2	3				16	1
Kulna maxilla	Nean	C	N	A			1	2				4	1
La Chapelle Aux Saints	Nean	A	N	E		UC	3	3	13	16		3	1
La Chaud 3 1980-6	EMH	A	E	Y			0	0				6	1
La Ferrassie 1	Nean	A	N	E		LP4,	3	3				32	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
						LP3, LC, LI2							
La Ferrassie 2	Nean	A	N	P			3	3				8	1
La Ferrassie MNP 1934-2-1-to 3	N	A	N	Y								3	1
La Gravette 1993-10-1	EMH	A	E	A								1	1
La Greze 1949-2-1	EMH	A	L	P								1	1
La Madeleine Child	EMH	A	L	A	dcC							13	1
La Naulette	Nean	A	N	?			3	3				0	1
La Piscine	EMH	A	L	A			1	2				1	1
La Quina 5	Nean	A	N	P			3	1				25	1
La Rochette	EMH	A	E	A, Y								9	2
La Tannerie	EMH	A	L	A								5	1
La Vachons	EMH	A	M	P								3	1
Lachaud 1980-8-1	EMH	A	E	A			1	2				14	1
Lalinde MNP 1930-1-2	? See brennan 1991	A	L	P	UM1	LM1	0	1				5	1
Laugerie-Basse	EMH	A	L	A	LM2		1	3				26	1
Le Morin	EMH	A	L	A	M3	UM2						4	1
Les Battuts 122	EMH	A	E	P								1	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
ab													
Les Fadets	Nean	A	N	A								4	1
Les Peyrugues 12.A	EMH	A	L	Y								8	1
Les Peyrugues 7 et 9	EMH	A	L	A								3	1
Les Peyrugues Carre 12 A	EMH	A	L	P								13	1
Les Rois 11-13	EMH	A	E	Y								5	1
Les Rois B 1955-148	EMH	A	E	A			0	1				2	1
Les Rois loose teeth	EMH	A	E	Y	3							24	3
Lexetxiki	Nean	A	N	A								2	1
Limeuil 1924-2-113	EMH	A	L	?			1	0				0	1
Lussac les Chateau 123-13	EMH	A	L	E								1	1
Malarnaud	Nean	A	N	A			1	2			2	1	1
Miesslingtal	EMH	C	E	A			0	0				10	1
Mladec 8 5457	EMH	C	E	P		UM1	2	3	0	2		4	1
Mladec cranium	EMH	C	E	Y		UM2, UM3	1	3	0	4		4	1
Mladec Lautsch II	EMH	C	E	Y			2	3	1	2		4	1
Mladec loose	EMH	C	E	Y								3	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
teeth													
Monsempron 1953-1	Nean	A	N	Y			3	3				10	4
Monsempron Individual 3	Nean	A	N	A			1	1					
Montmaurin	Pre-Nean	A	N	Y			0	0				6	1
Moulin Neuf MN 1 et 2	EMH	A	L	A								3	2
Muierii cranium	EMH	C	E	P			1	2				8	1
Nahal Ein Gev	EMH	M	L	P			2	2				13	1
Neandertal	Nean	C	N	P								2	1
Nerja	EMH	M	L	Y			0	1			2	30	1
New Neandertal loose teeth	Nean	C	N	A, P								5	2
Oase cranium (1)	EMH	C	E	Y			0	0				6	1
Oase mandible (2)	EMH	C	E	P			1	0				5	1
Oberkassel female	EMH	C	L	Y		UM1	3	3			1	20	1
Oberkassel male	EMH	C	L	E		LP4	3	3	12	12		14	1
Ochoz mandible	Nean	C	N	P			3	3				15	1
Oetrange 1	EMH	A	L	P			1	2	1	1		3	1
Ohalo 1	EMH	M	L	P	LM2		1	3				11	1
Ohalo 2	EMH	M	L	P	UM3	LM1	1	3	2	2		30	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
Oreille d'Enfer	EMH	A	E	?		LP3			0	1		0	1
Palomas 59	Nean	M	N	Y			1	0				5	1
Palomas collection	Nean	M	N	A, Y, P								76	37
Pavlov 1	EMH	C	E	P	LM3	UC	3	3				26	1
Pavlov 2	EMH	C	E	P			2	2				7	1
Pavlov 3	EMH	C	E	P			2	3				4	1
Pavlov loose teeth	EMH	C	E	A, P								22	2
Pech de la Boissiere 1934-1&2	EMH	A	L	Y								2	2
Petit-Puynoyen 1976-29 (Individual 1)	Nean	A	N	Y			1	2				15	1
Petit-Puynoyen 1976-29 (Individual 2)	Nean	A	N	A			0	2				4	1
Petit-Puynoyen 1976-29 (Individual 3)	Nean	A	N	Y			1	1				2	1
Predmosti hemi mandible	EMH	C	E	Y		UM3	1	2				5	1
Predmosti whole mandible	EMH	C	E	Y	LM1, LM2	LI2, LC	1	2	0	1		9	1
Qafzeh 11	EMH	M	M	Y	LM1		0	2				26	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
Qafzeh 3	EMH	M	M	P	1		2	2				10	1
Qafzeh 4	EMH	M	M	A	P3, P4, C, P4	UC	0	0				19	1
Qafzeh 4b loose teeth	EMH	M	M	Y								3	1
Qafzeh 5	EMH	M	M	Y			0	1				11	1
Qafzeh 6	EMH	M	M	P			2	2	1	2		12	1
Qafzeh 7	EMH	M	M	P	UM2, LM2		2	3				31	1
Qafzeh 8	EMH	M	M	Y			1	2				9	1
Qafzeh 9	EMH	M	M	Y	LM1, LM3		2	3				32	1
Qafzeh H4	EMH	M	M	Y	M3							8	1
Regourdou	Nean	A	N	P			3	3				16	1
Roc de Combe Capelle 1943-1-2	Nean	A	L	A			0	1				2	1
Romanelli (no?)	EMH	M	L	P			3	2			1	4	1
Romanelli 2	EMH	M	L	P			0	0				1	1
Romanelli 29	EMH	M	L	Y	LM2		0	0				3	1
Romanelli 4	EMH	M	L	P			2	1				1	1
Romanelli 5697	EMH	M	L	E			2	2				6	1
Romanelli loose teeth	EMH	M	L	A, Y, P	LM3							15	3
Romualdo	EMH	M	L	A								2	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
Saccopastore 1	Nean	M	N	E			1	2				5	1
Saccopastore 2	Nean	M	N	P		UP3	1	1				11	1
Saint Germain la Riviere (IPH)	EMH	A	L	E			2	3	0	1		8	1
Saint Germain le Riviere (MNP)	EMH	A	L	A								1	1
Saint Germain loose teeth	EMH	A	L	A	C							14	2
San Bernardino	Nean?	M	N	E, Y								3	1
Sandalja loose teeth	EMH	M	L	A, P								8	2
Skhul 5	EMH	M	M	P		LM1						31	1
Skhul 6	EMH	M	M	Y								5	1
Skhul 7	EMH	M	M	P			2	0				6	1
Spy 1	Nean	A	N	P			1	2				19	1
Spy 2	Nean	A	N	Y			2	2				25	1
Tabun	Nean	M	N	Y			2	3				31	1
Tabun D loose teeth	Nean	M	N	A, P								13	2
Tabun i	Nean	M	N	A			1	0				2	1
Tabun ii	Nean	M	N	A, Y, P	UM2							7	3
Tabun misc series iii	Nean	M	N	A								6	1
Tagliente loose teeth	EMH	M	L	A								2	1

Specimen	Taxon.	Reg.	Time	Age	Caries	Lesions	PD: Average CEJ-AC	PD: Most Severe	# teeth DEF AMTL	# teeth def + prob AMTL	Age-nesis	# teeth	MNI
Tornewton	EMH	A	L	P								1	1
Villabruna	EMH	M	L	Y	LM3		1	2				29	1
Vindija 11.39	Nean	M	N	Y			2	0				4	1
Vindija 11.40	Nean	M	N	Y			2	0				1	1
Vindija 11.41	Nean	M	N	?		M1						0	1
Vindija 11.42	Nean	M	N	?			3	3				0	1
Vindija 11.43	Nean	M	N	?		1						0	1
Vindija 11.44	Nean	M	N	?			2	1				0	1
Vindija 11.45	Nean	M	N	P		LM1	2	3				4	1
Vindija 11.46	Nean	M	N	Y			2	3				1	1
Vindija loose teeth	Nean	M	N	Y								7	1
Vindija MH 21.18	EMH	M	L	?			3	3	0	1		0	1
Vindija MH 21.20	EMH	M	L	A			1	2				3	1
Vindija MH loose teeth	EMH	M	L	A, P	UM							8	2
Willendorf II	EMH	C	E	?					0	1		0	1
Zafarraya mand	Nean	M	N	P		LM3	2	3		1		13	1

Abbreviations:
Region: A: Atlantic; C: Continental; M: Mediterranean
Time: N: Neandertal; M: Middle Paleolithic Modern Humans; E: Early Upper Paleolithic; L: Late Upper Paleolithic
Age: A: Adolescent: Y: Young Adult; P: Mid-Aged Adult; E: Elderly
PD: 0: No periodontal disease; 1: Mild; 2: Moderate; 3: Advanced

Appendix 3: Correlation Testing: CEJ-AC and septa score

Is there a correlation between the two forms of periodontal disease diagnostic data collected: CEJ-AC distances (CEJ-AC) and septum condition scores? Both represent alveolar bone loss, but describe it in different ways. The septum scores are based on two values, presence/absence of porosity and shape—convex, flat, and concave—of the interdental septum (adapted from Costa, 1982). Costa (1982) assigned combined ordinal ranking scores: no porosity with either convex and flat shape is considered free of periodontal disease; "osteoporosis" with convex shape is mild periodontal disease; "osteoporosis" with flat shape is moderate; and "osteoporosis" with concave shape was advanced. "No porosity with a concave shape" was not an option in his study; however, it was occasionally observed here. Adolescents who still had erupting teeth were most likely to have healthy alveolar bone without a convex shape (Costa (1982) concurs). I assigned each of these a ranked numerical score (see Table A3.1).

Score	Description
1	No porosity, convex shape, generally healthy alveolar bone, no PD
2	No porosity, flat or concave shape, but otherwise healthy, no definitive PD
3	Porosity, convex shape, early, mild PD
4	Porosity, flat shape, advancing moderate PD
5	Porosity, concave shape, advanced severe PD

Table A3.1: Ranking of interdental septum shape

These numerical scores are used to test the correlation between interdental septum condition (ordinal) and average cemento-enamel junction to alveolar crest distance measurements (continuous) using Spearman's rank correlation. I tested it by tooth to avoid

inter-correlation between data points from the same individuals and both the Late Pleistocene and Comparative sample data was used. 31 of 32 Spearman's rho were significant and varied between 0.6034 (p-value <0.0001) to 0.2996 (p-value=0.0180) (see Table A3.2 and A3.3 for full values). Only one Spearman's rho value, upper left central incisor, was low (0.1400) and insignificant (p-value= 0.4606). This suggests that generally CEJ-AC distances and septum scores are positively monotonically correlated. The lack of a stronger correlation is an artifact of the fact that there are only 5 options for septa condition, but many more options for the continuous CEJ-AC measurements (0.0 mm to over 20.0 mm where there is a lesion). They cannot be perfectly correlated.

The measurements also tend to be more strongly correlated in posterior than anterior teeth. This may reflect greater preservation damage in anterior alveoli of fossils. These tests only contain individuals whose teeth AND alveoli were present and in relatively good condition. Teeth lost from severe periodontal disease or infection are not represented as well as postmortem loss, common in anterior teeth with their less complex root shapes. Also correlation scores are generally higher in the maxilla than the mandible, possibly suggesting there is less continuous eruption in the maxilla than the mandible (Glass, 1991).

Upper Left Teeth	Rho	P-value	N	Upper Right Teeth	Rho	P-value	N
LM^3 (16)	0.56	<<0.01	43	RM^3(1)	0.64	<0.01	30
LM^2 (15)	0.49	<<0.01	67	RM^2(2)	0.37	<0.01	59
LM^1 (14)	0.45	<<0.01	71	RM^1 (3)	0.51	<<0.01	64
LP^4 (13)	0.32	0.01	67	RP^4 (4)	0.58	<<0.01	62
LP^3 (12)	0.43	<<0.01	61	RP^3 (5)	0.60	<<0.01	58
LC (11)	0.36	<0.01	61	RC (6)	0.37	<0.01	61
LI^2 (10)	0.60	<<0.01	39	RI^2 (7)	0.45	0.01	34
LI^1 (9)	0.14	0.46	30	RI^1 (8)	0.54	<0.01	30

Table A3.2: Spearman's Rank Order Correlation values for Septa Condition scores and CEJ-AC distances: Upper Teeth

Lower Left Teeth	Rho	P-value	N	Lower Right Teeth	Rho	P-value	N
LM_3 (17)	0.31	0.03	47	RM_3 (32)	0.38	0.02	39
LM_2 (18)	0.50	<<0.01	77	RM_2 (31)	0.46	<<0.01	70
LM_1 (19)	0.42	<<0.01	77	RM_1 (30)	0.53	<<0.01	80
LP_4 (20)	0.37	<0.01	59	RP_4 (29)	0.60	<<0.01	67
LP_3 (21)	0.30	0.02	62	RP_3 (28)	0.38	<0.01	59
LC (22)	0.33	0.02	50	RC (27)	0.35	0.01	60
LI_2 (23)	0.33	0.02	51	RI_2 (26)	0.30	0.03	54
LI_1 (24)	0.40	0.01	46	RI_1(25)	0.38	0.01	42

Table A3.3: Spearman's Rank Order Correlation values for Septa Condition scores and CEJ-AC distances: Lower Teeth