INFECTIOUS DISEASE AS AN INDICATOR OF PHYSIOLOGICAL STRESS IN THE MIDDLE HOLOCENE CIS-BAIKAL

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By

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ABSTRACT

Two distinct hunter-fisher-gatherer cultures lived on either side of a Middle Neolithic (or MN; 7,000/6,800–6,000/5,800 B.P.) archaeological hiatus in the Cis-Baikal, Siberia, Russian Federation. The Kitoi occupied the region in the Early Neolithic (or EN; 8,000–7,000/6,800 B.P.) and the Isakovo-Serovo-Glazkovo (or ISG) occupied the region in the Late Neolithic (or LN; 6,000/5,800–5,200 B.P.) into the Early Bronze Age (or EBA; 5,200/5,000–3,400 B.P.; Weber et al. 2015). Both of these cultures buried their dead in formal cemeteries located adjacent to the shores of Lake Baikal and along the many rivers of the Cis-Baikal. Research concerning the levels of physiological stress in the two cultures has found that the Kitoi suffered from more frequent and severe episodes of physiological stress than did the ISG (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011).

A detailed non-destructive visual examination of the osteological remains of 250 hunter-fisher-gatherers from three cemeteries (Shamanka II, Lokomotiv, and Ust'-Ida I) from the middle Holocene Cis-Baikal was carried out to determine if non-specific infection-induced lesions occurred significantly more in those populations who were found to have been the most physiological stressed. An endoscope was used to examine individuals' middle ears and those sinuses that were unobservable to the human eye and were accessible through cracks in the skull; and a hand-held x-ray system was used to image individuals' mastoid processes. One question was asked of the presence/absence data: is the incidence of infection-induced lesions within the sample related to a) the type of infection, b) the sex of the individual, c) the age of the individual, d) the cemetery the individual came from, and/or e) the time period the individual lived in? To answer this question, binomial tests, chi-square tests, and generalized linear model logistic regression tests were conducted. These revealed the presence of statistically more lesions indicative of chronic non-specific infection in EN individuals, more specifically, in those from the cemetery of Lokomotiv, in males, and in those older than 20 years.

It was concluded that non-specific infection-induced lesions occurred more in those populations who were found to have been the most physiological stressed. This study is in line with our understanding of stress, provides a much more detailed view into the community health of the Kitoi and the ISG than was previously known, and explores the lifeways of these two cultures through a new lens.

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Chapter 1
Context and Research Objectives

1.0.0.0. Context and Research Objectives

1.1.0.0. Context

The Cis-Baikal, the region to the north and west of Lake Baikal in Siberia, Russian Federation (see Figures 1.1 and 1.2), is home to hundreds of middle Holocene (ca. 9,000–3,000 B.P.) cemeteries. Archaeological research in the region began over 100 years ago and was first truly synthesized by A. P. Okladnikov beginning in the 1950s (Okladnikov 1950, 1955, 1959, 1964). Since the mid-1990s, the Baikal Archaeology Project (now the Baikal-Hokkaido Archaeology Project, or BHAP), lead by Andrzej Weber and based out of the University of Alberta, Canada, has been taking a collaborative, international, and multidisciplinary approach to research in the region in an attempt to understand the life ways of northeast Asian middle Holocene hunter-fisher-gatherers.

1.1.1.0. The Geography and Ecology of the Cis-Baikal

Lake Baikal is the largest, deepest, and oldest freshwater lake in the world (UNESCO World Heritage Convention) and was formed in the rift between two diverging tectonic plates. Today, the lake is surrounded by coniferous and steppe forests and open steppe (Weber and Bettinger 2010b). Rolling hills and mountains surround the lake, the steepest of which are located to the northeast and southwest (UNESCO World Heritage Convention 2014). Due to its biodiversity and the endemic nature of a number of the plants and animals that inhabit it, Lake Baikal is an UNESCO World Heritage Site (UNESCO World Heritage Organization 2014). As would be expected, the lake and its surrounding regions are of interest geologically, climatologically, biologically, and archaeologically (e.g., the Baikal Drilling Project, the Baikal-Hokkaido Archaeology Project, and the Lake Baikal Dimensions of Biodiversity Project). The

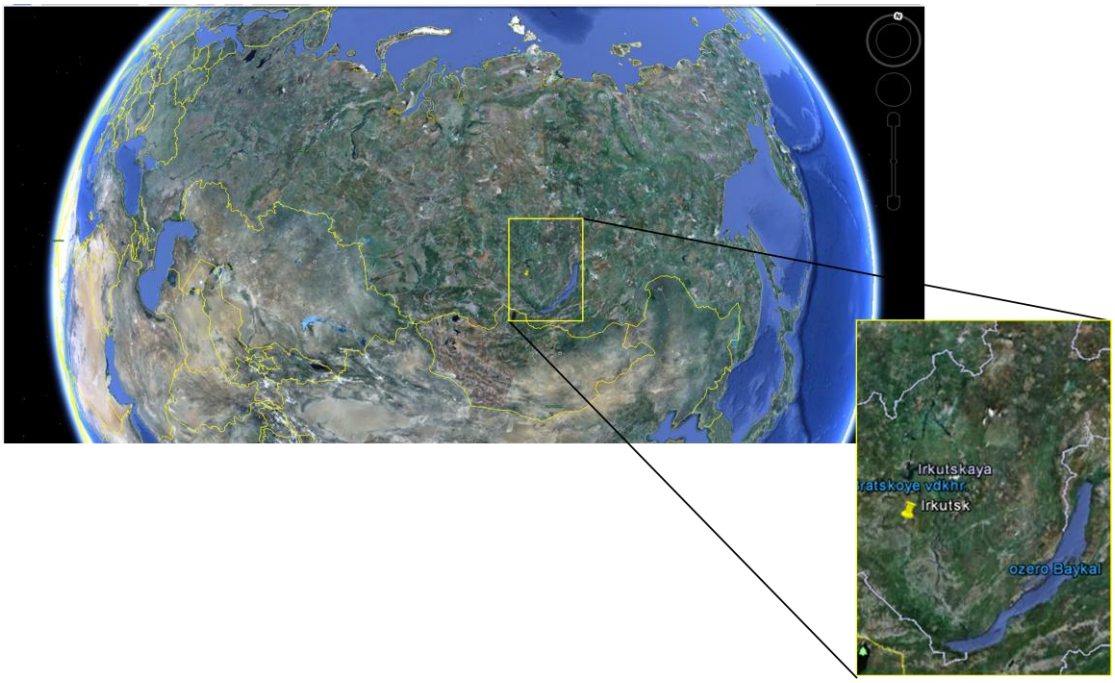

Figure 1.1: The Cis-Baikal as it is located within the Russian Federation (pictures from Google Earth).

Cis-Baikal can be divided into four microregions: the Angara River Valley (the region surrounding the Angara River, from Lake Baikal to the Ilim River), the upper Lena River Valley (the region surrounding the Lena River, from the mountains in the West to the Kirenga River, the Little Sea (or Ol'khon; the region between Ol'khon Island and the mainland, including Ol'khon Island and the smaller islands to its west), and South Baikal (the region from the east of the Selenga River delta to the southwestern tip of Lake Baikal; see Figure 2; Weber and Bettinger 2010b).

1.1.2.0. Culture History

Of interest to the BHAP are the hunter-fisher-gatherer populations that inhabited the region during a period of climatic and social transition: the Late Mesolithic (or LM; 8,800–8,000 B.P), the Early Neolithic (or EN; 8,000–7,000/6,800 B.P.), the Middle Neolithic (or MN; 7,000/6,800–6,000/5,800 B.P.), the Late Neolithic (or LN; 6,000/5,800–5,200 B.P.), and the Early Bronze Age (or EBA; 5,200/5,000–3,400 B.P.; Weber et al. 2015). In the MN, there was a cessation in the use of formal cemetery sites, though the presence of habitation data (such as lithics, ceramics, and fire pits) suggests that the region was still occupied (Weber and Bettinger 2010b). This MN occurrence is often referred to as a "hiatus," but it may more properly be called

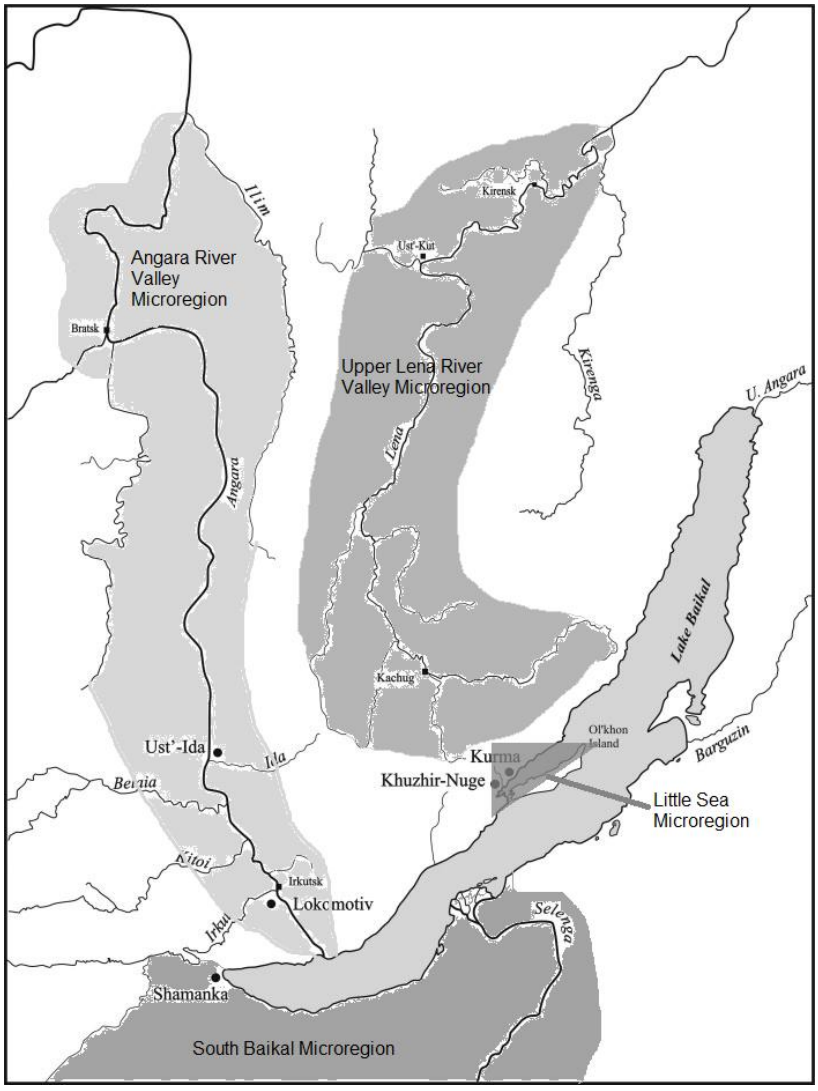

Figure 1.2: The microregions of the Cis-Baikal.

a cultural transition or a bio-cultural discontinuity. The connotation of "hiatus" alone may suggest that the region was devoid of human occupation during this time. Since this is not the case, it bears repeating that the MN represents a period in which no mortuary data exist. It is an archaeological blind spot, in which our primary source of data, cemetery sites, is not present. For the sake of consistency, however, the MN will be referred to here as a hiatus. Whether the hiatus represents the arrival of a distinct population and the departure of the first, or a cultural and demographic shift in the original population is one of the primary research concerns of the BHAP (Weber 1995; Weber et al. 2002 and 2010b). Some parts of this question have already begun to be answered.

The middle Holocene populations of the Cis-Baikal can be divided into two cultural and biological groups: the Kitoi culture (EN) and the Isakovo-Serovo-Glazkovo cultural complex or ISG (LN–EBA). Craniometric and aDNA data divide the middle Holocene populations biologically (Gerasimova 1992; Mamonova 1973, 1980, 1983; Mooder et al. 2003, 2005, 2010; Nasab et al. 2008; Schurr et al. 2010; Thomson 2006), while the material culture, mortuary patterns, health status, activity patterns, *etc.* divide the populations culturally (Weber 1995; Weber and Bettinger 2010b). How distinct these two populations are has recently been put into question by new genetic research that demonstrates some biological similarities between the two populations (Movsesian et al. 2014). Further research is necessary to synchronize these schools of thought.

1.1.2.1. The Biology of the EN and LN–EBA Populations

Recent mtDNA research not only seeks to understand the molecular differences between the EN and LN–EBA populations, but also to understand where they came from and, in the case of the EN Kitoi, where they went (Keyser-Tracqui et al. 2003; Mooder et al. 2006, 2010; Schurr et al. 2010; White and Bush 2010). The EN Kitoi are related most closely to the Kets and the Shors populations, and are biologically distinct from both the LN–EBA ISG and the present day populations of the Cis-Baikal (Derbeneva et al. 2002a, b; Mooder et al. 2005, 2006; Schurr et al. 2010). Today, the Kets live in the Upper and Middle Yeniseyi Valley, north of the Angara River Valley (Alekseenko 1999: 156) while the Shors live to the west of the Cis-Baikal (Movsesian et al. 2014). The ISG, on the other hand, are most closely related to both the northern Mongolian Egyin Gol population and the present day populations of the Cis-Baikal: the Evanks, Buryats, Tuvans, and Mongolians (Derbeneva et al 2002a, b; Keyser-Tracqui et al. 2003; Schurr et al. 2010). Their strong affinity with the east is underlined by their lack of genetic material from ancient West Eurasian populations (Schurr et al. 2010). In summary, the EN Kitoi and the LN–EBA ISG are very much biologically and culturally distinct populations with different adaptive strategies with regards to life in the Cis-Baikal. The extent to which the EN Kitoi interacted with the LN–EBA ISG across the MN and into the LN, however, is still unknown (Movsesian et al. 2014).

1.1.2.2. The Culture of the EN and LN–EBA Populations

Isotopic studies using both carbon and nitrogen isotopes obtained from bone collagen assert that both EN and LN–EBA populations exploited locally available game, fish, and plant

foods (Katzenberg et al. 2010). EN populations from the Angara River Valley and South Baikal micro-regions, however, consumed more fish than did LN–EBA populations (Katzenberg et al. 2009, 2010; Katzenberg and Weber 1999; Lam 1994; Weber and Bettinger 2010b; White and Bush 2010; Weber et al. 2010), while contemporary populations from the upper Lena River Valley and the Little Sea micro-regions relied more heavily on hunting game (and sealing in the Little Sea; Weber and Bettinger 2010b). Recent research indicates that only populations from the Angara River Valley show discernible signs of shifting from fishing to hunting terrestrial game during the EN to LN–EBA transition (Weber et al. 2011).

EN cemeteries are located along streams and rivers, and on the shores of Lake Baikal. It is hypothesized that EN settlements would have also followed this pattern (Weber and Bettinger 2010b). As such, and following further studies of carbon, nitrogen, and strontium isotopes from bone collagen, skeletal morphology, and grave goods, it has been suggested that EN individuals lived in larger but, more isolated populations than did LN–EBA individuals (Weber and Bettinger 2010b; Weber et al. 2002). They were irregularly situated around the Cis-Baikal and were concentrated largely on the Angara River and the southern tip of the lake in areas with easy access to aquatic resources (Haverkort et al. 2008, 2010; Katzenberg et al. 2010; Losey et al. 2008; Nomokonova et al. 2006, 2009a, b; Weber and Bettinger 2010b; Weber and Goriunova 2013; Weber et al. 2003). Due to their isolation from one another, they had a more heterogeneous archaeological culture than did those of the later LN–EBA (Weber and Bettinger 2010a, b).

Because of the localized concentrations of relatively large EN populations, local game would have become depleted within the immediate vicinity of the settlements, causing individuals to hunt farther away from home (Weber and Bettinger 2010b). This task appears to have been undertaken largely by males, as they tend to show greater osteological signs of having borne a heavier workload and engaged in more extensive travel when compared to females (Lieverse et al. 2007b, 2009, 2010, 2013, 2016; Stock et al. 2010). In addition, EN populations have more osteological evidence of poorer overall community health and greater levels of physiological stress than do those of the LN–EBA (Lieverse 2005, 2010; Lieverse et al. 2006, 2007b; Temple et al. 2014; Waters-Rist et al. 2011).

In the LN–EBA, there was a more even distribution of terrestrial game resources, human populations are believed to have been more evenly distributed across the Cis-Baikal, more mobile, and less isolated from one another, creating a more homogeneous archaeological culture

(Weber and Bettinger 2010a, b; Weber et al. 2002). Males are believed to have travelled less and overall, labour was more evenly distributed between the sexes (Lieverse et al. 2007b, 2009, 2010, 2013, 2016; Stock et al. 2010). In general, community health and physiological stress levels were also better in LN–EBA populations (Lieverse 2005, 2010; Lieverse et al. 2006, 2007b; Temple et al. 2014; Waters-Rist et al. 2011).

1.1.2.3. Physiological Stress in the EN and LN–EBA Populations

Stress episodes (including bouts of infection) can leave lesions on bones and teeth, allowing for such events to be studied on archaeological human remains. Each episode can impact different biological pathways, for which there are different micro- and macroscopic means of studying them. Physiological stress is the result of anything that makes a demand of the body, such as infection and nutritional deficiency. Factors such as population density, hygiene, dwelling conditions, and the local ecosystems can increase the likelihood that infection and poor nutrition make a demand of the body (Novak et al. 2009; Seyle 1973, 1976). The human body prioritizes and manages the functioning of its various systems—body temperature, bone growth, enamel formation, *etc.*—through homeostasis. Any outside entity that disrupts the body's homeostasis is called a "physiological stress." Such stress forces the body to prioritize the essential systems and stop the function of the non-essential systems in order to be able to deal with or weather the outside change (Goodman et al. 1988; Seyle 1973, 1976).

Isotopic Studies of Weaning Ages

Waters-Rist and colleagues (2011) analyzed the levels of stable nitrogen and carbon isotopes ($\delta^{15}N$ and $\delta^{13}C$) obtained from the collagen in bone samples taken from the proximal and distal metaphysis and the diaphysis of individuals aged from birth to 10 years. The goal of the study was to discern the weaning practices of EN and LN populations. It was found that EN populations breastfed infants exclusively until they were two years old, at which point weaning began for some as seen in the decrease in the number of infants being breastfed (62.5% of the observed population). Complete weaning occurred between the ages of 3.5 and 4.0 years old. Overall, there was a two year period (from 2.0 to 4.0 years old), in which there was variation within EN populations with regard to their weaning practices. LN populations, it was found, practiced exclusive breastfeeding until the infant was 1.0 year old. At this point, there was a two year weaning period and children were fully weaned by the time they were 3.0 years old. Weaned children and adults ate the same things, showing no preferential or differential treatment of young

children within either period (Katzenberg and Weber 1999; Katzenberg et al. 2009, 2010; Weber et al. 2002; Waters-Rist et al. 2011).

It has been suggested that more frequent periods of food stress in the EN as compared to the LN may have lead to the later and more variable weaning of EN infants (Lieverse et al. 2007a; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011). The death of some breastfeeding infants in the EN (Waters-Rist et al. 2011) may also suggest that the mothers themselves were under stress, as breastfeeding infants get their nutrition and immune resistance straight from their mothers (Maher 1992; Pelletier 2000). Within the EN population, isotopic signatures also point to the use of emergency breastfeeding (Waters-Rist et al. 2011), a practice reserved for times of nutritional stress when weaned infants are temporarily breastfed so as to protect them from starvation (Arifeen et al. 2001; Jakobsen et al. 2003). In the end, however, all that can be said for certain is that these two cultures, the EN Kitoi and the LN–EBA ISG, had unique weaning practices that may reflect cultural preference, or social or biological adaptations.

Long Bone Morphological Analysis

Research regarding skeletal growth patterns between EN and LN cemeteries was performed by Temple and colleagues (2014). In this study, the lengths of the femora and tibiae, the estimated body mass, and the maximum width of the femoral midshaft, cortex, and medullary cavity of all available EN and LN subadult individuals were measured and calculated. It was found that EN individuals had a shorter adult femoral length and a lower body mass in late infancy and early childhood than did those of the LN–EBA (Temple et al. 2014).

In comparing these results with those of the aforementioned isotopic study on breastfeeding and weaning ages, it was found that in the EN, the age at which weaning began corresponded to the age at which femoral growth and body mass were stunted (Temple et al. 2014). An infant's introduction to foreign infectious agents through supplementary food, the loss of passive resistance from their mother's milk, and the potential loss of adequate or balanced nutrition that occur during weaning can all contribute to a disruption in homeostatic regulation and the consequent stunting of an individual's growth (Berti et al. 2000; Cameron et al. 1998). Temple and colleagues also suggest that periods of food stress experienced throughout the EN may also have precipitated or contributed to the observed stunting (2014).

Dental Indicators of Physiological Stress

Dental indicators of physiological stress also support the presence of more frequent episodes of physiological stress in the EN. Linear enamel hypoplasia (or LEH), specifically, has been investigated. LEH is a developmental enamel defect, usually characterized by horizontal grooves perpendicular to the long axis of the tooth, which can occur on the outer surface of a tooth's enamel (Hillson 1996: 165). Such defects arise when severe stress or infection occur at the time of tooth development (Suckling 1989), causing ameloblasts to cease producing the matrix solution that later forms into enamel (Hillson 1996: 165–7). Since teeth form at known rates, LEH can be related back to the age at which the defects were formed (Reid and Dean 2000, 2006). Defects must be present on more than a couple adjacent teeth in order for them to be considered LEH and not the product of localized trauma or inflammation (Suckling 1989) and on those areas of teeth that relate to the same period of enamel formation (Reid and Dean 2000, 2006; Lieverse et al. 2007a; Waters-Rist 2011).

Three studies regarding the frequencies of LEH in the Cis-Baikal have been carried out by BHAP members (Lieverse et al. 2007a; Link 1999; Waters-Rist 2011). All have found higher frequencies of LEH in populations from the EN than from the LN–EBA. Such differences may reflect more frequent episodes of nutritional stress, or, specifically, seasonal resource scarcity in the EN and their lack of cultural buffers against such occurrences (Lieverse et al. 2007a; Waters-Rist 2011). Repetitive cases of LEH occured approximately within ten to twelve months of one another and may reflect seasonal resource shortages, such as in the months of late winter and early spring in the Cis-Baikal (Waters-Rist et al. 2011).

In interpreting her results, Waters-Rist (2011) cautions against the comparison of LEH patterns and weaning patterns, and states that the LEH patterns seen in EN and LN cemeteries do not match those seen in the respective cemetery's weaning patterns, and that teeth themselves do not provide an inclusive window through which to view a life's worth of stress events. Rather, those stress events that occurred while enamel was forming (fifth foetal month to eleven years old depending on the tooth) (Reid and Dean 2006) are the only ones that are observable (Waters-Rist 2011).

It is worth noting that the near lack (0.3%) of LEH in deciduous teeth, paired with the lack of rickets (the deformation of the lower long bones due to childhood osteomalacia) and osteomalacia (the softening of the bones caused by a deficiency in vitamin D, calcium, or

phosphorus) in all Cis-Baikal individuals, suggests that breastfeeding infants were receiving the proper levels of calcium and vitamin D from their mothers (Waters-Rist 2011). This is important to note in light of the deaths of EN breastfeeding infants that were noted above (Waters-Rist et al. 2011). The LEH lesions show, however, that the EN and EBA populations experienced more episodes of physiological stress in the years that the affected teeth represent than did the LN populations.

Summary

These studies of physiological stress in the Cis-Baikal present a diverse and complex picture of the environmental, biological, and social landscapes of the middle Holocene. The skeletal growth study concludes, and the isotopic study implies, that EN individuals experienced more frequent episodes of physiological stress than did those of the LN–EBA (Temple et al. 2014; Waters-Rist et al. 2011), while studies of LEH conclude that EN individuals experienced more, and more repetitive, episodes of physiological stress than did those of the LN–EBA (Lieverse et al. 2007a; Link 1999; Waters-Rist 2011). It is not possible to definitively state whether such episodes occurred as the result of nutritional stress and/or infection-induced stress. It is also important to bear in mind that numerous other factors, such as worldview, are invisible to us in the archaeological record and could affect such things as weaning practices and the spread of infection, respectively (Waters-Rist 2011).

1.1.3.0. Climate and Culture Change in the Middle Holocene

Climate change may have played a large role in the cultures' interactions within the region (Tarasov et al. 2007; White 2006; White and Bush 2010). Circa 9,000–7,000 cal. years B.P., the climate of the Cis-Baikal was at its warmest and wettest. This period coincides with the appearance and florescence of the Kitoi (Tarasov et al. 2007). This warm, wet climate and boreal forest vegetation later gave way to a major climatic shift in the MN: a cooler, dryer climate and an increase in steppe and meadow lands in the LN–EBA (Tarasov et al. 2007; White and Bush 2010). Both cultural periods are climatically stable, with the little-understood MN hiatus coinciding with climate shift (White and Bush 2010).

It has been hypothesized that the shift in climate from wet to dry may have altered the runoff patterns, streams, and rivers to such a point where the fisheries themselves were affected (While and Bush 2010), as has been seen to occur elsewhere around Lake Baikal and the world (Beamish 1995; Beamish and Bouillon 1993; Beamish et al. 2004; Chu et al. 2005; Daufresne et

al. 2003; Harley et al. 2006; Mathews and Marsh-Mathews 2003; Prokopenko et al. 2007; Xenopoulos et al. 2005). This change would not only have forced the local populations to change their subsistence practices, but the increase in steppe and forest-steppe microregions would have also lent itself more to herbivore migration and, in turn, hunting (Weber and Bettinger 2010b; White and Bush 2010).

How the Cis-Baikal relates to the greater pattern of middle Holocene human migration and why the LN–EBA populations appear to have been adapted to live in this geographically new region are all questions the BHAP is in the process of exploring. LN–EBA populations may have migrated from western Siberia or central Asia, as seen in their similar archaeological cultures and following the known dispersion of nomadic pastoralists, respectively (Mamonova 1983; Goriunova et al. 2004; Weber 1995; Weber et al. 2002). Alternatively, the ca. 8,000–7,000 B.P. (White and Bush 2010) southern departure of the East Asian summer monsoon may have stimulated a northern migration of populations from Mongolia and northern China into the Cis-Baikal, as supported by aDNA research and discussed previously in section 1.1.2.1. (An et al. 2000; C. Chen et al. 2003; F. Chen et al. 2003, 2006; He et al. 2004; Jiang et al. 2006; Rhodes et al. 1996; Shi et al. 2002; Tarasov et al. 2000). Climate change therefore, as seen in the movement of the East Asian summer monsoon and the general shift from warmer and wetter to cooler and dryer conditions, is, in part, a hypothesized explanation for the observed culture change of MN Cis-Baikal (White 2006; White and Bush 2010); as one culture's subsistence patterns began to change, another culture moved in to take its place.

1.2.0.0. Research Questions and Goals

1.2.1.0. Research Questions

As an indicator of quality of life, infection is both a reaction to physiological stress and a catalyst thereof (Wood et al. 1992; Allmäe and Limbo 2010; Novak et al. 2009; Roberts 2007; Lieverse 2010). Infection-induced lesions can be either non-specific or specific, and are characterized osteologically by osteoblastic and osteoclastic responses to inflammation resulting from the invasion of foreign bacteria, viruses, fungi, or parasites (more on this in Chapter 2; Murphy et al. 2008: 41). Previous research has not only noted the presence of a number of infectious indicators in the Cis-Baikal skeletal samples such as cribra orbitalia, porotic hyperostosis, periosteal lesions, and septic arthritis, but also the relatively low percentage of

individuals displaying signs of infectious disease in general (4.1% of observed individuals; Lieverse 2010). That the EN Kitoi experienced more physiological stress than did the subsequent LN–EBA ISG has also been noted (Antonova 2011; Lieverse 2010; Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011).

This study has one primary research question. Did non-specific infection-induced lesions occur significantly more in those populations who were found to have been the most physiologically stressed? This question can be refined to ask one pointed question of the data: is the incidence of infection-induced lesions within the sample related to a) the type of infection, b) the sex of the individual, c) the age of the individual, d) the cemetery the individual came from, and/or e) the time period the individual lived in? This will be discussed further in section 3.2.2.

Patterns that may emerge from the data will be compared to those aforementioned patterns of physiological stress. Should the presence/absence of infection-induced lesions mirror that of the physiological stress-induced lesions, then the primary research question can be answered positively. If not, then physiologically stressed populations did not also have higher levels of non-specific infection-induced lesions. In either case, the reasoning supporting each conclusion will be discussed and the findings will add to our broader understanding of the socio-cultural implications of physiological stress and infection in the face of human adaptation and cultural transition.

1.2.2.0. Research Goals

The BHAP's prerogative is to explore the lifeways of middle Holocene northeast Asian hunter-fisher-gatherers. In the Cis-Baikal, the nature of the MN hiatus is of great interest. The appearance of, and the MN disappearance of, the EN Kitoi, the subsequent appearance of the LN–EBA ISG, and the florescence of both within the Cis-Baikal are founding research areas for the project. In the same way that past research into physiological stress has augmented our understanding of the cultures of the middle Holocene Cis-Baikal and their interactions with the landscape and environment around them, this study also aims to augment our understanding of the interaction between the EN and LN–EBA populations of the Cis-Baikal and non-specific infection. This study aims to define the relationships between physiological stress and non-specific infection in the Cis-Baikal and to further our understanding of the life-ways of middle Holocene hunter-fisher-gatherers.

Should it be found that the patterns of infection mirror those of physiological stress, this would lend further support to the current understanding that EN individuals were generally suffering from more frequent episodes of stress than were the later LN–EBA individuals. It would also suggest that infection, itself, may have been (one of) the primary mechanism(s) behind the physiological stress. If not, then the fitness levels of each population and the robusticity of infection as an indicator of physiological stress in this context will be better understood in that I will have a window into the etiology of the physiological stress (not infectious) and the non-specific infections that occurred independently of physiological stress episodes.

Such research has a broad impact on our understanding of middle Holocene cultures. By better understanding the overall community health of the EN and LN–EBA cultures, we can better understand their lifeways and their experiences in their social, cultural, and environmental landscapes. How individuals of different age groups, sexes, time periods, and, ultimately, cemeteries, experienced such things helps us to understand, to some extent, how they perceived age, gender, time, and death.

Chapter 2
Infection and its Skeletal Indicators

2.0.0.0. Infection and its Skeletal Indicators

The archaeological literature contains scores of accounts and detailed descriptions of unusual skeletal lesions. When paired with the existing clinical literature, links can be made between the skeletal lesions we, as archaeologists, observe and the biological processes that created them. The following is a summary of the existing literature concerning the etiology and appearance of non-specific infection-induced lesions. Periostitis, osteomyelitis, periodontitis, caries, sinusitis, otitis externa and media, mastoiditis, trachoma, and cribra orbitalia and porotic hyperostosis (or COPH) will be discussed. First, however, an understanding of the human immune system, infection, and the biology of bone must be established.

2.1.0.0. Understanding Infections

2.1.1.0. The Immune System and Infection

Infection is defined as the invasion of a foreign bacterium, virus, fungus, or parasite (more specifically, a protozoa or a helminth) that results in an inflammatory immune response in the host (Murphy et al. 2008: 41). This immune response can be either innate or adaptive. An innate immune response will be triggered within minutes of the host's exposure to a pathogen, while an adaptive immune response will be triggered should the pathogen bypass or overwhelm the host's innate immune system (Murphy et al. 2008: 40).

When a pathogen gets past the body's primary defences, namely the skin or the mucosal lining of the respiratory and digestive tracts, or attaches itself to them, then it is identified as foreign by the natural killer (or NK) cells of the immune system and an innate immune response is triggered (Mahmoudi 2011: 1–5; McDade 2005; Murphy et al. 2008: 46–7). Innate immune responses consist of three basic components: opsonisation and phagocytosis, inflammation, and lysis (Mahmoudi 2011: 1–5; Murphy et al. 2008: 48–53). Opsonisation is the tagging of a

pathogen by an opsonin (a molecular component of the complement—the host of plasma and proteins synthesized as part of an immune responce; Mahmoudi 2011: 5), thus identifying the pathogen as the microorganism to be destroyed. Phagocytosis (the destruction of the pathogen) occurs when the host cell envelops the pathogen in a phagosome, which is then invaded by a membrane attach complement (or MAC) of enzymes that breakdown and destroy the pathogen. Such enzymatic destruction is called lysis. Inflammation aids these immune pathways via the dilation of the capillaries. Dilation eases the passage of fluids and proteins between the blood and the infected tissues, helps promote blood clotting (which forms a physical barrier between the pathogens and the rest of the body), and aids in the repair of damaged epithelia (DeWitte and Bekvalac 2011; Mahmoudi 2011: 1–5; McDade 2005; Murphy et al. 2008: 50; Weston 2008).

An adaptive immune response can occur alongside, or independently of, an innate immune response, and is more robust against persistent and aggressive pathogens than is an innate immune response (Murphy et al. 2008: 40, 323). In cases of adaptive immunity, inflammation still occurs, but this time it is B and T lymphocytes that produce antibodies and destroy the pathogen, respectively (Mahmoudi 2011: 5; McDade 2005; Murphy et al. 2008: 323). Adaptive immunity learns and remembers as it is introduced to new pathogens over the life of an individual, and it is this immunological memory that is exploited by vaccine technology. This memory allows for the immune system to trigger the appropriate B and T lymphocytes necessary to combat an infection (Murphy et al. 2008: 442).

Since all parts of the body are connected, either structurally or through the circulatory and lymphatic systems, a pathogen does not always affect the primary site of infection alone. Rather, both the pathogen and the cells of the immune system that respond to it can be carried to other parts of the body and infect and affect those tissues, respectively. Areas infected by pathogens in this way are called secondary infection sites.

Inflammation, for example, is triggered through the release of cytokines (proteins produced in responce to the presence of microbial antigens; Barksby et al. 2007; DeWitte and Bekvalac 2011; Mahmoudi 2011: 3–5; Weston 2008) which then circulate through the blood stream to all parts of the body. As a result, they are known to trigger inflammatory reactions in non-infected systems (Jimenez et al. 2012; Lauren et al. 2012; Persson 2012; Shay 2002; Salvi et al. 1997; Iacopino 1995). A localized infection, then, can have a systemic impact. Examples of cases will be discussed in detail throughout this chapter.

An immunological hypothesis from 1989, the hygiene hypothesis (Strachan 1989), states that exposure to different pathogens as a young child trains the body's naïve immune system to recognize the "genetic, social, geographic, cultural, and economic" (Kramer et al. 2013) context that it is in and primes it to react appropriately (Azad et al. 2013; Brown et al. 2013; Frei et al. 2014, 2012; Kondrashova et al. 2012; Kramer et al. 2013; Macia et al. 2011; Matsushima and Nagai 2012; Prokopakis et al. 2013; Strachan 1989; Taghipour et al. 2014; Wander et al. 2012). In this hypothesis, those who are not exposed to infectious agents as children, primarily those growing up in post-industrial, "sterile" environments, suffer from more autoimmune, atopic, allergic, and allergic asthmatic disorders than do those whose young immune systems were kick-started by contact with relevant infectious agents (Azad et al. 2013; Brown et al. 2013; Frei et al. 2014, 2012; Kondrashova et al. 2012; Kramer et al. 2013; Macia et al. 2011; Matsushima and Nagai 2012; Prokopakis et al. 2013; Taghipour et al. 2014; Wander et al. 2012).

Were the children of middle Holocene Cis-Baikal inhabitants raised in a sterile environment? Of course not, but it bears noting in a discussion of infection that not all pathogens are harmful. In fact, some may provide their hosts with resistance to other infectious agents and ailments (Abela and Fava 2013; Azad et al. 2013; Matsushima and Nagai 2012; Taghipour et al. 2014) while also priming their host's immune system for the rest of the host's life. So, while bacteria, viruses, fungi, and parasites can trigger an inflammatory immune response, they do not always do so.

2.1.2.0. Acute and Chronic Infection

Multiple factors contribute to the duration of an infection. An individual's age, sex, ethnicity, nutritional status, level of current and past physiological stress, *etc.* can all impact their immune response and their ability to survive an infection (Weston 2008). Should an infection be highly virulent and/or should the individual's immune response be weak, then the individual may acquire and or ultimately succumb to the infection relatively quickly. This type of infection is called "acute". On the contrary, should an infection be less virulent and/or the individual's immune response be robust, then the individual may survive, or survive for longer, to fight the infection. In these cases, such an infection is called "chronic". Individuals can die from either type of infection. It is the duration of the infection that identifies it as acute or chronic (Roberts and Manchester 2005: 7).

As per the Osteological Paradox (a theory that aims to explain what can and cannot be inferred from archaeological populations and why), the lesions that may result from acute infections are not usually visible on skeletal and dental tissues (Wood et al. 1992). Acute infections do not exist long enough in the living body for the skeleton to be involved in, or affected by, the immune response, because the host dies or recovers. In general, it is chronic infections that exist in the host's body long enough to affect their bone and consequently precipitate the creation of bony lesions. Barring cases where lesions were fully remodelled before the host's death, chronic infections are the only infections to create visible lesions on archaeological remains. In summary, the presence of an infection-induced lesion is not always indicative of an individual's death, but is, rather, indicative of the individual's physiological response to an infection over time (Wood et al. 1992).

2.1.3.0. Specific and Non-Specific Infection

This study is concerned with how infection—and non-specific infection in particular—affects the human skeleton. Infection-induced lesions can be either "specific" or "non-specific". Infections that create a host of bony lesions whose appearance and/or distribution observed on the bone are characteristic of individual diseases or conditions are referred to as "specific" (Buikstra and Ubelaker (ed.) 1994: 107; Mays 1998: 123–7; Roberts and Manchester 2005: 182–220). Non-specific infection-induced lesions, on the other hand, cannot be traced back to the pathological microorganism (or group of microorganisms) that precipitated the observed bony response. Rather, non-specific infections create a host of non-diagnostic bony lesions that can only be classified generally based on the type of bony response, destruction and/or formation, and the layer of bone tissue affected (Buikstra and Ubelaker (ed.) 1994: 107; Mays 1998: 123–7; Roberts and Manchester 2005: 167–8).

2.1.4.0. Changes in Bone

The bone deposition and destructive processes involved in lesion formation are abnormal, in that the formation of lesions is not something that would occur independently of a foreign stimulus (in this case, an immune response to a pathogen). That being said, bone destruction and formation is necessary for the growth and remodeling of the bone. Every year, 5–10% of the mineral component of the adult skeleton is replaced, with only 2–3% turnover of cortical bone being necessary to retain biomechanical strength (Kini and Nandeesh 2012). As such, bone destruction and depostition is not in itself indicative of pathological processes. Rather, abnormal

bone destruction and formation not in keeping with the structural maintenance of the skeleton is that which is termed "abnormal" and/or "pathological".

Bone destruction and formation are performed by osteoclasts and osteoblasts, respectively. Osteoclasts are a type of cellular macrophage and, as such, destroy bone in accordance with a triggered immune response (pathologically; Mahmoudi 2011: 2) and according to natural bone maintenance (regularly). They secrete an acidic substance to dissolve the mineral component (hydroxyapatite) of bone, while lysosomes (enzymes specifically suited for destroying the peptidoglycan cell walls of bacteria; Mahmoudi 2011: 1) destroy the bone's organic component (collagen; Steinbock 1976: 10).

Bone formation, on the other hand, is triggered by both osteoprogenitor cells, which differentiate into osteoblasts and can be found within the periosteum (the membrane that covers the outer surface of all bones with the exception of joint surfaces) and by osteocytes (which are embedded within the lacunae of bone matrix; Steele and Bramblett 1988:12). Osteoblasts are bone forming cells; they lay down osteoid (a protein substance that is a precursor to bone) and they are active in the primary mineralization process of said osteoid (Steele and Bramblett 1988:12). Osteocytes maintain bone; they are active in the secondary mineralization process of osteoid and in bone maintenance (Steele and Bramblett 1988:12).

It is the process of aggravating the periosteum and stimulating osteoclastic and osteoblastic activity at different times and in different places that is responsible for the abnormal bone proliferation discussed here (see below). The biological pathways outlined in the following section (2.1.0.0.) concerning the etiology of periostitis hold true for all the subsequent sections involving abnormal bone destruction and formation.

2.2.0.0. Understanding Non-Specific Infections

2.2.1.0. Periostitis

Periostitis refers to the non-specific proliferation of pathological subperiosteal new woven and/or healing lamellar bone over the cortex of the normal bone surface. Woven bone refers to the irregular pathological bone first laid down by the periosteum which can be subsequently remodelled into mature lamellar bone. The word periost*itis* implies that these lesions are caused by infections, but there are, in fact, many possible causes for the proliferation of periosteal lesions (Assis et al. 2011; Weston 2008). Rather, such lesions should be referred to as periost*osis*,

in which the infectious denotation is removed. However, for the sake of simplicity and consistency, and since the use of this language is a debate larger than the scope of this study, the word periostitis will be used here with the caveat that it does not always refer to a lesion with an infectious etiology.

Periostitis is just as much an indicator of physiological stress as it is an indicator of infection. In general, periostitis occurs most frequently in cases of infection (Ubelaker and Pap 1998). Infections, however, often occur in physiologically stressed or immunocompromised individuals (DeWitte and Bekvalac 2011). This does not mean that periostitis is solely an indicator of stress and/or infection, but it does mean that it can be. As a result, the etiology of periostitis is often difficult to discern. For the purpose of this study, it was considered the result of infection in the absence of indicators of other pathological conditions with the proviso that it may be the result of any number of other non-infectious conditions (see below).

Etiology

Periostitis is not caused by any one specific pathogen; rather it is the bony response to anything that agitates the periosteum. The breaking, tearing, stretching, inflammation, or touching of the periosteum as a result of trauma, mechanical changes, the growth of a tumor, ulcer, or granulation tissue, and/or the altered or new circulation of blood or exudate, *etc.* can cause the periosteum to react. Such irritations are perceived by the periosteum as a threat, causing osteoprogenitor cells to differentiate into osteoblasts (Assis et al. 2011; Boel and Ortner 2011; Cunha et al. 2009; White and Folkens 2005: 42–3), which in turn lay down woven bone over the existing lamellar surface in an attempt to protect it (Assis et al. 2011; Steinbock 1976: 13; Weston 2008). The aggravation of the periosteum precipitates a reaction common to all abnormal bone-forming lesions.

The following are the processes involved in the formation of an infection-induced periosteal lesion. In cases of inflammation, vasodilatation occurs and stimulates hyperemia (an increase in blood flow) and, as a result, hyperoxia (an increase in oxygen). Hyperoxia, in turn, stimulates osteoclastic function and the destruction of bone. The increased flow and accumulation of interstitial fluid (called an edema), contrarily, results in hypoxia (a decrease in oxygen), which stimulates osteoblastic functions and the creation of bone (Weston 2008). The type of bony lesion created depends on both the types of fluids involved in the inflammatory process and their ratios.

Once the pathogen has been killed, expelled, or has died, the irritating trauma or stress has ceased, or the unusual tissue has been reabsorbed, the periosteum and underlying bone undergo a healing process. Osteoclasts destroy all necrosed tissue and osteoblasts secret osteoid in the place of the bone that was destroyed. Over time, vascularization and mineralization of the osteoid occurs, excess fluids are reabsorbed, and the osteoid is converted into bone (Weston 2008; White and Folkens 2005: 43). Should an individual die at any point during this process, the state of the lesions (be it active, healing, or healed) can be observed osteologically.

As such, rather than being representative of the condition that aggravated the periosteum, periostitis is determined by the type of fluids present, the tissue(s) affected, the presence of other diseases, and the individual's age, sex, ethnicity, immune strength, and nutritional status (Weston 2008). The identification of the cause of a periosteal lesion requires macroscopic examination of the entire skeleton to see if the reaction is systemic or localized, and/or related to any other infection- or stress-induced lesions. Bone formation in muscle and ligament attachment sites can further complicate a diagnosis as disambiguating the new bone's presence based on pathology or mechanics is very difficult (Ubelaker and Pap 1998). The macroscopic study of the lesions themselves, the imaging of the lesions with an x-ray generatorraph, followed by differential diagnosis is required to diagnose periostitis as specifically as possible (Weston 2008).

Appearance

If an individual dies while their periosteum is still in the process of reacting to a perceived threat, the lesion will be "active". In general, woven bony striations parallel to the long-axis of the bone, fine grained pitting, and plaque-like bone formation characterize active periosteal lesions (Roberts and Manchester 2005: 172). On the contrary, if an individual dies after the periosteum is finished responding to a perceived threat and their body is in the process of remodelling the lesion, the observed lesion will be discernible as "healing". Such lesions will be composed more of mature lamellar bone than irregular woven bone. A mix of woven bone and lamellar bone may suggest a chronic infection (Mays 1998: 123). And finally, a healed lesion remains visible on the bone surface as an area of slightly thicker and more striated lamellar bone.

The appearance of periostitis can vary. As such, differential diagnoses based on lesion appearance are extremely important in furthering our understanding of lesion formation (DeWitte and Bekvalac 2011; Lambert 2002; Novak 2011). For example, diffuse lesions (those lesions that are ubiquitously present on the skeleton) can be characteristic of systemic infections while

localized lesions can be characteristic of focal infections, such as leg ulcers (Weston 2008). Barring the presence of specific markers, however, periostitis will remain a type of lesion with a non-specific etiology.

2.2.2.0. Osteomyelitis

Etiology

Osteomyelitis refers to the infection and subsequent inflammation of a bone's marrow cavity as the result of a primary, secondary, or tertiary infection. Primary infections involve the direct infection of the medullary cavity and occur as a result of trauma, burns, surgery, *etc.* (Aufderheide and Rodríguez-Martín 1998: 172; Ortner 2003: 181; Roberts and Manchester 2005: 171; Steinbock 1976: 60). Secondary infections involve the spread of a pathogen from an overlying area directly into the medullary cavity (Ortner 2003: 181). Finally, tertiary infections involve the hematogenous transfer of the pathogen from the primary site of infection to the medullary cavity via the bone's Haversian and Volkmann's canals. This is the most common cause of osteomyelitis as the bloodstream provides the medullary cavity with its major form of systemic contact (Aufderheide and Rodríguez-Martín 1998: 172; Ortner 2003; 181; Roberts and Manchester 2005: 169–72; Steinbock 1976: 60).

The long bone metaphyses are often the sites at which pathogens enter the medullary cavity. During childhood, the capillaries that enter the long bones at the metaphyses form a loop before entering. This allows pathogenic microorganisms to pool and gather, cutting off blood supply and causing the necrosis of the surrounding area (Steinbock 1976: 62). Also, those capillaries entering and exiting the bone do not contain active phagocytes which would aid in a pathogen's destruction if they were present (Hobo 1921). As a result, the metaphyses of children provide the ideal areas for the hematological transfer of an infection from a primary site of infection.

The throat, ears, sinuses, and chest are common regions from which infectious agents spread (Roberts and Manchester 2005: 169). Infectious agents responsible for osteomyelitis must be pyogenic, in that they must stimulate the production of exudate upon infection, because exudate formation leads to the bony lesions characteristic of osteomyelitis (Aufderheide and Rodríguez-Martín 1998: 172; Ortner 2003: 181; Steinbock 1976: 60). This often means that bacteria are responsible for these lesions, though viruses, fungi, and parasites can also cause osteomyelitis (Ortner 2003: 181). Most often, *Staphylococcus* bacteria is what causes

osteomyelitis (Ortner 2008: 195; Ortner 2003), with *Staphylococcus aureus* causing 90% of observed cases (Ortner 2003: 181; Steinbock 1976: 60). In cases involving infants, however, it is often *Streptococcus* bacteria involved (Robbins 1975; Steinbock 1976: 61).

Appearance

The processes involved in the formation of osteomyelitic lesions concern not only the inflammation of the medullary cavity, but also of both the bone and the periosteum (or periostitis; Aufderheide and Rodríguez-Martín 1998: 172; Ortner 2008: 195; Steinbock 1976: 60). These result from exudate formation and expulsion forming cloaca and sequestra, and bone destruction and creation forming involucra. Cloacae are abscesses for drainage of exudate that is building up within the medullary cavity. At least two cloacae should be present on a bone for the diagnosis of osteomyelitis, although this is not always the case (Ortner 2008: 195). The exiting substances create a fistula, or an abnormal opening, in the soft tissues and skin, allowing for the medullary cavity to drain continually (Aufderheide and Rodríguez-Martín 1998: 172).

Sequestra are sections of the infected bone that have necrosed as a result of either having their blood supply cut off by a cloaca or decreased by the pressure from exudate formation. These can either be ejected along with exudate through a cloaca, or can be destroyed by osteoclasts either fully or partially, depending on their size. As a result, the edges of sequestra are rounded with the occasional jagged sections formed by osteoclastic action (Ortner 2003: 182; Steinbock 1976: 66).

The entire diaphysis of a long bone can be made into a sequestrum. This occurs when exudate seeps through the cortex of a bone at the metaphysis, which is more porous than the cortex elsewhere, and lifts the periosteum off of the diaphysis. Functionally, this cuts off the blood supply to the bone and kills the diaphysis (Ortner 2003: 182).

Involucra are created through the stimulation of the periosteum via the two aforementioned processes. This creates an asymmetrical proliferation of bone around the diaphysis intended to maintain the bone's structural integrity. Involucra have a rough, highly vascularized appearance and are often punctured by cloacae (Ortner 2008: 195–6; Ortner 2003: 182–3; Roberts and Manchester 2005: 168–9). All three of these features are characteristic of osteomyelitis and can be present or absent depending on the processes involved in each particular case (Ortner 2008: 196).

Infections can also occur solely within the medullary cavity and show no signs on the exterior of the bone. Such lesions are called Brodie's abscesses and require a radiograph or sectioning to be detected (Aufderheide and Rodríguez-Martín 1998: 178; Resnick and Niwayama 1995: 2327; Steinbock 1976: 74–6). This occurs relatively rarely and will not be discussed here.

The appearance of osteomyelitis can also vary depending on the age of the individual affected. Osteomyelitis most often affects children between the ages of three and 12–15, though it can be present in all ages (Jaffe 1972 in Aufderheide and Rodríguez-Martín 1998; Aufderheide and Rodríguez-Martín 1998: 173; Steinbock 1976: 62). In cases of haematogenous (i.e., tertiary) infection, only one bone is usually infected in children, while adults usually have multiple infected bones (Ortner and Putschar 1981). In children, vessels carrying the blood supply to the diaphysis and metaphysis are different, limiting the contact between the two regions. These are also separated structurally by dense growth plates. As a result, the infection is limited to the diaphysis (Ortner 2003: 184). When osteomyelitis affects a bone in a child's hip or shoulder however, the infection can spread from epiphysis to epiphysis via the articular joint (Aufderheide and Rodríguez-Martín 1998: 173; Ortner 2003: 184). In these cases, as well as in cases of chronic osteomyelitis in adults, the infection can spread to adjacent bones and ankylosis, the pathological bony fusion of a joint, can occur (Aufderheide and Rodríguez-Martín 1998: 173 Ortner 2003: 184).

Different infectious pathways occur in infants than in adults and children. In infants, the infection can travel between bones and/or joints since bones at this time lack a dense growth plate that would stop this (Ortner 2003: 186–7). When the epiphysis of an infant's bone is infected, the infection can stop the growth of the bone in the direction of the epiphysis (Aufderheide and Rodríguez-Martín 1998: 173). Sequestra rarely occur in cases of infantile osteomyelitis as their developing cortical bone is very porous and allows for the accumulating exudate to seep out of the cortex without severally affecting the blood supply (Aufderheide and Rodríguez-Martín 1998: 173; Ortner 2003: 187).

Those bones affected by osteomyelitis vary depending on the cause and severity of the infection. Acute hematogenous osteomyelitis affects the femur, tibia, humerus, and radius the most frequently (in that order; Aufderheide and Rodríguez-Martín 1998: 172; Roberts and Manchester 2005; 172), while acute direct (i.e., primary) osteomyelitis affects the bones of the hands and feet most frequently (Ibáñez et al. 1984). Chronic osteomyelitis generally affects one

section of a long bone and is perpetuated by the presence of an infected piece of sequestrum, or the presence of a chronic infection in the adjacent soft tissue, joint, or blood (Steinbock 1976; Turek 1982). Chronic osteomyelitis can lead to Brodie's abcesses (previously discussed), Sclerosing Osteomyeitis of Garré (the localized or diaphyseal thickening of the cortex with a lack of other osteomyelitis indicators such as exudate formation), and pyogenic/septic arthritis (caused by the destruction of articular cartilage by proteolytic enzymes in the invading exudate; Aufderheide and Rodríguez-Martín 1998: 177–9; Steinbock 1976: 74–9).

Osteomyelitis has the propensity to create distinctive lesions that are identifiable based on the presence of its three characteristic features: cloacae, sequestra, and involucra. While the presence and/or appearance of these features vary in each case, active osteomyelitis is regularly identifiable. Since the pathogens that cause osteomyelitis are usually unknown in archaeological cases, it is classified as a non-specific infection-induced lesion.

2.2.3.0. Dental Disease

Teeth are comprised of both organic and inorganic structures. Three different substances form their structure: enamel, cementum, and dentin. Enamel is largely inorganic and acellular. It covers the crown of the tooth and provides a durable surface for mastication and a protective layer over top of the less-durable dentin (Hillson 1996: 148-9; Berkey and Shay 1992).

Dentin is the innermost layer of the tooth and surrounds the pulp cavity. It is both organic and inorganic and is composed of a series of tubules. Should wear or other processes on the incisal/occlusal (biting/chewing) surface destroy the enamel and expose the dentin, inflammation can spread through these tubules and into the pulp cavity (Oztunc et al. 2006; Nisengard et al. 1994). Dentin can also naturally re-grow, via the presence of odontoblasts (dentin-forming cells) within the surface of the pulp chamber, in an attempt to make up for occlusal wear or destruction due to caries or trauma (Hillson 1996: 182–5, 194; Pashley 1994). Sometimes, however, wear exceeds the speed at which dentin can reform, and the tooth can be nearly obliterated (for example, see Skinner et al. 1988; Hillson 1996: 182–5).

Cementum is also both organic and inorganic (Hillson 1996: 199) and covers the external surfaces of tooth roots. The roots of the teeth are sunken into the alveolar bone of the jaws. Ideally, the gingivae (or gums) that cover the alveolar bone meet the tooth at the cemento-enamel junction (or CEJ) and protect the cementum from external wear. It is also to the cementum that

the periodontal ligament attaches so as to secure the tooth into the bony socket (Hillson 1996: 198–9; Pashley 1991).

Saliva maintains the near neutral acidity of the mouth so as to limit microbial growth and protect the soluble components of the inorganic tissues in teeth (Nisengard et al. 1994). The sulci between the roots of the teeth and the gingivae also produce gingival crevice fluid, a plasma ultrafiltrate that keeps the sulcus clean (Nisengard et al. 1994). In some cases, the decreased production of saliva is what precipitates infection (Nisengard et al. 1994). Clinically, a decrease in saliva production in the elderly is a known risk factor for their acquiring dental diseases (Nisengard et al. 1994).

2.2.3.1. Periodontitis

There are two forms of dental disease that will be discussed here: periodontitis (or periodontal disease) and dental caries. First, periodontal disease refers to the infection of the marginal gingivae and/or the alveolar bone (Hillson 1996: 262; Soames and Southam 2005). It is the effect that periodontitis has on the alveolar bone that is of interest here.

Etiology

Over time, the buildup of oral biofilm, or plaque, (DeWitte 2012; Holt et al. 2000) on teeth allows for the incubation of various oral pathogens that cause periodontitis and/or caries (discussed below). *Actinobacillus actinomycetemcomitans*, *Porphyromonas gingivalis*, *Treponema denticola*, *Streptococcus*, and some herpes viruses are examples of those pathogens that can cause periodontitis (Hillson 1996: 262; Li et al. 2000; Slots 2004; van Winkelhoff and Slots 1999). Plaque can accumulate on the occlusal surfaces of teeth, where it is usually removed via mastication, in the areas between teeth, or in the sulci between teeth and the gingivae (Holt et al. 2000; Shay 2002).

There is some debate as to the effect of carbohydrates on bacterial plaque build up (Delgado-Darias et al. 2006; Wasterlain et al. 2011). In general it is accepted that soft diets high in carbohydrates, sugars, casein (a protein component of dairy; Hillson 1996: 254), fat (Kondo et al. 2014), and/or diets high in grit (as associated with tooth wear) lead to more caries and periodontitis than diverse diets high in proteins, fiber (Kondo et al. 2014), antioxidants (specifically, vitamins A, B, C, D, and E; Amaliya et al. 2007; American Dental Hygienists' Association 2012; Ehrlich 1994; Iwasaki et al. 2012; Kuzmanova et al. 2012; Otten at al. 2006; Rugg-Gunn and Nunn 1999; Salmeri 2012; Staudte et al. 2005; University of Maryland Medical

Center 2011; Yao and Fine 2012), and other micronutrients (Cohen and Armelagos 1984; Kelley et al. 1991; Larsen 1981, 1997: 79–80, 2002; Lopez et al. 2011; Molnar and Molnar 1985; Moore and Corbett 1971; Shaw 1962; Tayles et al. 2000; Walker and Erlandson 1986). It is important to note that periodontitis and caries can also arise independent of plaque buildup. As a result, the presence or absence of periodontitis (and caries) is not soley an indicator of an individual's infection level. Diet and hygein are also factors that play a role. Dental diseases are, however, infections and can impact the overall health of an individual.

Periodontitis begins as an infection of the gingivae, referred to as gingivitis. Eventually, the infection may spread to the bone or tooth, though some individuals have been known to live with massive plaque build-up for twenty years without gingivitis progressing to periodontitis (Baelum et al. 1986). This counters the common perception that gingivitis is the acute form of periodontitis (Hillson 1996: 262; Soames and Southam 2005). Rather, gingivitis can be chronic itself (Baelum et al. 1986). Should the infection spread from the gingivae to the periodontal ligament and to the alveolar bone, however, the infection would then be considered to be "periodontitis." Periodontitis leaves the tooth insecure and may lead to further infection and tooth loss.

Appearance

Clinically, periodontitis manifests itself as, and is diagnosed from, the recession of the gingiva, movement in the teeth, pain and/or ulceration in the gingiva, problems with occlusion, and the amount of accumulated plaque and/or calculus, which is mineralized dental plaque (Wasterlain et al. 2011). Osteologically, the destruction of alveolar bone is the characteristic symptom of periodontitis. Alveolar bone is reabsorbed, causing the distance between the alveolar bone and the cemento-enamel junction to increase.

A problem in diagnosing periodontitis on archaeological remains is the criteria with which to do it (Brothwell 1981; Davies et al. 1969; Karn et al. 1984; Kerr 1988, 1998; Levers and Darling 1983; Lukacs 1989). Criteria designed by Kerr (1988, 1998) use a difference of more than 3 mm between the alveolar crest (or AC) and the CEJ to diagnose periodontitis in dry bone. Now, however, it is recognized that teeth continue to erupt during adulthood so as to match tooth wear and maintain lower face height and occlusion (Clarke and Hirsch 1991; Westerlain et al. 2011). It has also been suggested that tooth sockets may begin to fill-in while, at the same time, the roots of teeth may undergo hypercementosis. These processes exudateh the teeth gradually

out of their sockets (Hillson 2000, 2005). As a result, measurements are still made from the AC and CEJ, but are not considered diagnostic in and of themselves. Rather, the interdental walls of the alveolar bone are scored based on their texture and architecture (Kerr 1998b, 1988). It is the macroscopic morphology of bone destruction (i.e., porosity) that is characteristic of periodontitis today, rather than the perceived extent of destruction (Kerr 1998a, 1988).

2.2.3.2. Caries

Etiology

Caries is the localized destruction of the tooth's enamel, dentin, and/or cementum caused by the acid produced by the bacteria harboured within dental plaque (Hillson 1996: 269). *Streptococcus mutans, S. oralis, S. milleri, S. salivarius, Actinomyces naeslundii, A. viscosus,* and lactobabilli bacteria are those most often responsible for creating carious lesions (Hillson 1996: 276). Caries can also occur as a result of, or in conjunction with, periodontitis (Hillson 1996; Roberts and Manchester 2005: 65). Compared to periodontitis, caries is far more common today, with 60–90% of children infected and close to 100% of adults affected worldwide (World Health Organization 2012). This compares to estimates of 15–20% of people from every population worldwide having periodontitis (World Health Organization 2012).

Appearance

Each tissue reacts differently to caries. If a carious lesion forms in the enamel, it begins through the process of demineralization. The enamel then proceeds to turn white and chalky, at which point it will dissolve and a cavity will form. If a lesion forms in the cementum, below the gingiva, an area of demineralization will first appear in the innermost layers of cementum. Over time, the lesion will erode the cementum and create a lesion perpendicular to the long axis of the tooth. The dentin responds to the demineralization and invasion of bacteria before the lesion reaches it by creating a zone of reparative secondary dentin. It lays down dentin over the ends of the tubules in an attempt to stop bacteria from spreading into the tubules. Should this not succeed, the bacteria will enter the dentin, following the tubules, and continue to demineralise the inorganic components of the tooth (Hillson 1996: 284–5).

If a carious lesion succeeds at reaching the pulp cavity, or if bacterial toxins penetrate the pulp cavity through the tubules of the dentin, then the pulp cavity will inflame. Inflammatory exudates accumulate and restrict blood flow within the cavity, causing localized and then systemic necrosis of the tooth. Exudate accumulation follows and can result in the formation of

an abscess through the periapical opening of the root into the surrounding alveolar bone (this lesion is called a periapical granuloma). Localized bone destruction ensues, but may go no further should the carious lesion close. If it does not, and bacteria continue to have access to the periapical granuloma, then a fistula will form and the exudate will drain. Generally, fistulae form on the buccal surface of alveolar bone, though they can also form lingually, or within the maxillary sinus or nasal cavity (see section 2.2.4.0. for more detail regarding sinusitis; Hillson 1996: 284–5).

2.2.4.0. Sinusitis

There are four pairs of sinuses in the human head: the maxillary sinuses, the frontal sinuses, the ethmoid sinuses, and the sphenoid sinuses (Martini et al. 2012). The maxillary, frontal, and ethmoid sinuses all open into the nasal cavity, while the sphenoid sinuses open into the nasopharynx (Evans 1994; Martini et al. 2012). The opening between the nasal cavity or nasopharynx and the sinus is called an ostium and allows for airflow between the respiratory system and the sinuses (Gwaltney 1996; Melén 1994; Wagenmann and Naclerio 1992).

The sinuses have a defensive function in the human body. They connect to the nose and nasopharynx directly and to the mouth, eyes, oropharynx, laryngopharynx, ears, and lungs indirectly. They are the first structures, apart from the nose and mouth, that inhaled pathogens and particles come into contact with when entering the human body (Merrett and Pfeiffer 2000). Ideally, when a breath is taken, pathogens and particulates in the air should become trapped in the mucosal lining of the sinuses' walls. The lining contains both antimicrobial and anti-inflammatory molecules (Kaliner 1992; Lindberg et al. 1997; Wright et al. 1994), while the sinuses themselves are filled with nitric oxide produced within the sinuses and nasal mucosa (Gwaltney 1996; Naraghi et al. 2007). In model conditions, the sinuses are sterile (Axelsson and Brorson 1973; Rantanen and Arvilommi 1973). Once pathogens and particles are trapped, they are ejected from the sinus(es) and, ultimately, the respiratory system, via the movement of the cilia that line the sinus(es)' walls (Evans 1994; Gwaltney 1996).

Etiology

Sinusitis, the inflammation of one or more sinus, is researched infrequently on human skeletal remains (Roberts and Manchester 2005: 174) either due to its unpopularity or, more likely, the difficulty inherent in accessing the sinuses noninvasively and non-destructively. Of particular interest in palaeopathology are the maxillary sinuses. While the frontal, ethmoid, and

sphenoid sinuses are also able to become infected, ancient cases of infection are more often reported regarding the maxillary sinuses (Haugen and Ramlo 1993). This is most likely due to the fact that they are relatively more accessible for observation in the dry bones than are the other three types. The ethmoid is also, itself, highly fragile, resulting in its partial or complete destruction in many archaeological cases.

Sinusitis can be caused by either a primary or secondary infection. For a primary sinus infection to occur, the pathogen must invade the sinus directly and disrupt the proper movement of air and mucus (Evans 1994; Merritt and Pfeiffer 2000). Viruses cause sinusitis the most often in modern clinical cases, followed by bacteria and allergens (Evans 1994; Gwaltney 1996; Sande and Gwaltney 2004). Fungi, structural blockage, trauma, hormone-induced mucosal edema, cystic fibrosis, cleft palate, and pre-existing syndromes such as Kartagener and Young's can also cause sinusitis (Andes et al. 2000; MacKay 1988; Gwaltney 1996). Sinusitis can also be caused secondarily by the abscessing of a tooth through its root and into the maxillary sinus, mechanical pressure exerted on the maxillary sinus from the force of chewing, complications from other infections such as TB, communicable infections, or oral surgery (Melén 1994; Melén et al. 1986; Shafer et al. 1974; Wright 1979).

The following immune response creates the bony lesions characteristic of sinusitis: the secretion of excess mucus and the creation of inflammatory mediators. Inflammatory mediators can cause a mucosal edema (the swelling of the mucus membrane) to form within the sinus, which functionally blocks the ostium. As a result, the previously inhaled pathogen is structurally stopped from being ejected from the sinus (Armentano et al. 1999; Lundberg 1980; Lundberg and Engquist 1983; Van Nostrand and Goodman 1976; Wright 1979; Yonkers 1992).

Should the ostium remain blocked, damage may then be done to, firstly, the epithelium and, secondly, to the underlying bone. First, an increase in pressure within the affected sinus due to mucus build-up causes the cilia to slow their movement (Norlander et al. 1994). Concomitantly, an increase in hypoxia within the sinus increases the acidity causing further harm to the cilia (Evans 1994). Finally, a secondary immune response is triggered and inflammatory cytokines are synthesized. These stimulate bone destruction within the affected sinus (Gowen 1994).

Should the ostium not clear, an abscess and fistula may form, allowing the exudate within the sinus to escape and the intra-sinus pressure to decrease. Generally, frontal sinus infections

abscess into the endocranium since the posterior bone wall of the sinus is usually thinner than the anterior wall. This inevitably results in meningitis and, usually, the death of the individual (Iwen et al. 1997). Should the fistula form through the anterior wall, however, the exudate will drain and the infection will heal (Becker et al. 1986). Only two such cases of anterior abscessing have been reported from archaeological populations, both originating from Barcelona (Armentano et al. 1999). Prolonged (i.e., chronic) sinusitis can lead to olfactory disorders (Fark and Hummel 2013), irreparable damage to the epithelial cells (Ohasi and Nakai 1983a, b), the creation of accessory ostia in the sinus cavity (Gwaltney 1996), the replacement of the mucosal lining by fibrous tissue (Hall and Coleman 1990), and the subsequent ubiquitous presence of a certain amount of exudate within the sinus (Hall and Coleman 1990).

Appearance

Chronic sinusitis, as with most infections, is the only form that elicits a bony reaction (Ortner and Putschar 1981). Clinically, sinusitis must persist for more than three months in order to be considered chronic rather than acute (Shapiro and Rachelefsky 1992), though there is an argument that any form of sinusitis that persists for longer than 7–10 days could be considered chronic, as acute forms related to rhinoviral infection show a marked lack of symptoms after this many days (Lindbaek et al. 1996). Sinusitis can persist due to the presence of other upper respiratory infections (Andes et al. 2000), dentigerous cysts (caused by the growth of a tooth within the maxillary sinus; Prabhu et al. 2009), allergies (Andes et al. 2000; Evans 1994), nasal septum deviation (Evans 1994), intranasal polyps (Evans 1994), cleft palate (Gwaltney 1996), mucociliary disorders (Gwaltney 1996), cystic fibrosis (Gwaltney 1996), having a compromised immune system (Gwaltney 1996), *etc.*

Osteologically, sinusitis presents as fine porosity, spicule-type bone formation that appears to have been stuck to the sinus wall, merged spicules/lobules that appear plaque-like or molten, remodelled spicules that appear to stand out from the sinus wall, bony cysts, and/or white pitted bone. White pitted bone may also appear on the external surface of the maxilla in the area overlying an infected sinus (Boocock et al. 1995; Merrett and Pfeiffer 2000). Research regarding sinusitis should increase regardless of the difficulties associated with its analysis, as it is a functional way of analyzing upper respiratory health and is an indicator of "endemic chronic respiratory distress" (Merritt and Pfeiffer 2000; Roberts 2007) within a population.

2.2.5.0. Infection of a Concha Bullosa

Etiology

An uncommon risk factor for maxillary, frontal, and ethmoid sinusitis is the infection of a concha bullosa. A concha bullosa is the abnormal asymptomatic pneumatisation of a turbinal, usually the middle turbinal, of the ethmoid bone (Al-Sebeih et al. 2014; Ariyürek et al. 1996; Bolger et al. 1991; Christmas et al. 2001; Clerico 1996; Messerklinger 1978). Turbinals are lined with the same epithelial tissues as are sinuses, and react in the same way to infection (Aygun and Zinreich 2010; Cohen and Matthews 2008). When healthy, they should drain into the nasal cavity (Zinreich 1992). Should a concha bullosa become blocked and infected, a cyst may form within it. This type of concha bullosa is known as a mucocele when blocked and a mucopyocele when secondarily infected (Bolger et al. 1991; Christmas et al. 2003; Cohen and Matthews 2008; Earwaker 1993; Hatipoğlu et al. 2005; Kennedy and Zinreich 1988; Kwiatkowska et al. 2011; Marianowski et al. 2002; Meloni et al. 1992; Zinreich et al. 1988). Mucocele and mucopyocele also commonly form within the sinuses (Cohen and Matthews 2008).

Appearance

Both mucopyocele and abnormally large concha bullosae can expand and fill part of the nasal cavity, altering the morphology of the surrounding bones and disrupting the regular drainage of mucus (Kwiatkowska et al. 2011; Shihada and Luntz 2012; Zinreich 1992). Should this occur, the ostia of the sinuses may become blocked, leading to sinusitis (Kwiatkowska et al. 2011), and local bone erosion within the nasal cavity (Cohen and Matthews 2008). In the clinical case reported by Cohen and Matthews (2008), the ethmoid sinuses were destroyed and the infected concha bullosa became continuous with the frontal sinus. Following the formation of a mucopyocele, the infection may travel through the bloodstream from the ethmoid sinus to the orbit of the eye. This secondary infection of the orbit can result in a supraorbital abscess (Christmas et al. 2007a, b). As such, both concha bullosae and mucopyoceles are abnormalities that should be observed and noted in the osteological record, as they may have impacted the sinuses, nasal cavity, and the eyes.

2.2.6.0. Otitis

The ear is composed of three anatomical sections: the outer ear, the middle ear, and the inner ear. Osteologically, the outer ear consists of the external acoustic meatus, which penetrates the temporal bone anterior to the mastoid process. It opens into the middle ear, which consists

osteologically of the tympanic cavity and the auditory ossicles. The bony roof of the tympanic cavity is called the tegmen tympani and it separates the middle ear from the endocranial space that houses the brain. The posterior and superior surface of the tympanic cavity connects with the mastoid air cells. Anteroinferiorly, the Eustachian tube runs from the middle ear, passes through the bony musculotubal canal, and opens into the nasopharynx. Osteologically, the musculotubal canal opens endocranially anterior to the carotid canal and posterior to the foramen spinosum. Approximately the final centimetre of the canal is formed by adjoining grooves in the adjacent temporal and sphenoid bones. The inner ear is the most medial section of the ear and opens into the middle ear via the oval and round windows. The middle and inner ear are contained within the petrous portion of the temporal bone (Martini et al. 2012: 152, 479–82).

In life, the outer ear is separated from the middle ear by the tympanic membrane (or ear drum) and the middle ear is separated from the inner ear by epithelium in the case of the round window and by the base of the stapes and the annular ligament in the case of the oval window. The chorda tympani nerve, which originates in the tongue and connects with the facial nerve, passes through the middle ear, while the tensor tympani muscle, which attaches to the malleus, passes through the musculotubal canal. Osteologically, a thin section of bone can be seen running the length of the canal and presumably separates the tensor tympani muscle from the rest of the space in the canal. Both the outer and middle ears are air-filled (the outer ear is filled with atmospheric air and the middle ear is filled with endogenous air produced from gasses dissolved within body water; Rădulescu 2013), while the inner ear is filled with endolymph and perilymph (fluids responsible for gauging balance and transmitting sound, respectively; Martini et al. 2012: 152, 479–82). The ear is a very complex structure with many delicate parts (bones, nerves, muscles, and blood vessels) and its infection can have a large impact on an individual's wellbeing, mortality, and morbidity (Gregg and Steele 1982; Mays and Holst 2006).

The ears are prone to outer, middle, and inner ear infections, also known as otitis externa, otitis media, and otitis interna, respectively. All three have different etiologies and morphological expressions within the soft tissues and bone, allowing for their differentiation in skeletal samples (Mays and Holst 2006). Both otitis externa and otitis media will be discussed here while otitis interna will not be. This research does not involve otitis interna due to the difficulty involved in observing the inner ear non-destructively (imaging of this area and the diagnosis of otitis interns

from an x-ray requires a proficiency I do not have and that is not disussed in the literature) and, as such, its etiology and appearance need not be discussed.

2.2.6.1. Otitis Externa

Etiology

Otitis externa is caused by the trapping and proliferation of a pathological microorganism in the outer ear canal. It is asymptomatic on its own (Mays and Holst 2006), but may lead to deafness should a cholesteatoma (an localized build-up of dead epithelial cells that have become infected with a low-grade pathological microorganism) form and proceed to infect and erode both the bone of the middle ear canal and the auditory ossicles (Anthony and Anthony 1982; Garin et al. 1997; Heilbrun et al. 2003; Holt 1992; Mann 1992; Mays and Holst 2006; Piepergerdes et al. 1980; Sismanis et al. 1986; Vrabec and Chaljub 2000). External ear infections commonly affect the elderly, potentially due both to the cerumen (or ear wax) produced by the ceruminous glands of the auditory canal becoming more adhesive with age and dead epithelial cells losing the ability to expel themselves from the auditory canal (Holt 1992). Ultimately, this results in the buildup of dead cells and the creation of a perfect environment in which to foster an infection and the formation of a cholesteatoma.

Appearance

Otitis externa is both a bone destroying and forming infection that can involve the external ear canal and the surrounding temporal bone (Mays and Holst 2006). Osteologically, its appearance is described as the presence of fine-grained pitting around the external auditory meatus (Mays and Holst 2006) and/or the resorption of the bone that forms the walls of the external auditory meatus (Mays and Host 2006). Bony complications of otitis externa may include osteomyelitis, abscess, mastoiditis, and otitis media (Mays and Holst 2006).

2.2.6.2. Otitis Media

Etiology

Clinically, otitis media is diagnosed as the inflammation of the middle ear cleft (the structures that attach to and make up the middle ear: the Eustachian tube, the tympanic cavity, and the mastoid air cells (or mastoiditis, see below; Rai 2014). Around 50% of cases are caused by *Streptococcus pneumonia* or *Haemophilus influenza* (Aufderheide and Rodríguez-Martín 1998: 253). Due to its direct connection with the nasopharynx via the Eustachian tube, the middle ear is very prone to infection, causing otitis media to be related to the general health of the

respiratory system (Flohr and Schultz 2009). As a result, the invasion of the inner ear by bacteria from the nasopharynx is the primary cause of the majority of middle ear infections, with fungal and viral aetiologies also existing (Boenninghaus and Lenarz 2005; Graham-Hodgson 1950).

The blocking of the Eustachian tube due to inflammation or debris results in the middle ear's inability to dispose of its dead epithelial cells by draining into the nasopharynx, or to maintain the proper air pressure in relation to that of the outer ear (Mays and Holst 2006). As a result, negative pressure builds up within the middle ear and a retraction pocket may form (Mays and Holst 2006). The dead epithelial cells that cannot be expelled then collect within the retraction pocket and form a cholesteatoma. The cholesteatomas may grow to involve and damage the tympanic membrane, and generally then precipitates both bone destruction and bone formation (Olszewska et al. 2004; Soldati and Mudry 2001; Sudhoff and Tos 2000).

Appearance

The inflammation of the mucosal lining of the middle ear, the accumulation of exudate within the tympanic cavity, and the creation of inflammatory cytokines can result in bone destruction and proliferation within the middle ear following those pathways discussed in section 2.1.0.0. (Mays and Holst 2006). In cases when the pressure build-up inside the middle ear becomes too large, the tympanic membrane will form a hole, allowing the exudate to drain out through the outer ear. Should this occur, the middle ear will then heal, though the hole in the tympanic membrane may not, allowing both bacteria and dead epithelial cells to enter the middle ear. This may once again lead to otitis media or the creation of a cholesteatoma, respectively (Aufderheide and Rodríguez-Martín 1998: 253). A cholesteatoma is bone destroying and presents on dry bone as a pit in the temporal bone and, occasionally, the destruction of the auditory ossicles (Mays and Holst 2006).

2.2.7.0. Mastoiditis

Etiology

Mastoiditis is the result of inflammation spreading from a primary site of infection (classically the middle ear, see above; Fleischer 1979), through the mucosal lining of the air filled diplöe, and into the diplöe of the mastoid process (Boenninghaus and Lenarz 2005; Fleischer 1979). The pathogens most often associated with mastoiditis in modern populations are *Streptococcus pneumoniae*, *Haemophilus influenzae*, *Branhamella catarrhalis*, and haemolytic *Streptococcus* (Bitar et al. 1996; Gliklich et al. 1996; Niv et al. 1998; Rosen et al. 1986). It is

important to note that secondary infections can also occur in the mastoid and petro-mastoid portions of the temporal bone via the haematopoietic transfer of an infection from its primary site through the emissary veins (Graham-Hodgson 1950).

Mastoiditis is considered both a common complication of acute otitis media (Groth et al. 2012) and a diagnostic feature of chronic otitis media (Rai 2014). Due to the propensity for infants and children to have otitis media, mastoiditis also often occurs at this age. This coincides with the growth of the mastoid portion of the temporal bone, meaning that mastoiditis can permanently damage the development and growth of the mastoid air cells (Aufderheide and Rodríguez-Martín 1998:253; Groth et al. 2012).

Clinically, mastoiditis requires both the mucosal lining and the bone itself to be inflamed before it is qualified as such (Fleischer 1979). If the infection lasts three weeks or less, it is considered "acute", and if it lasts more than three weeks, it is considered "chronic" (Chien et al. 2012). Both following and/or accompanying the creation of lesions within the mastoid, infection can spread elsewhere via bone erosion, thrombophlebitis (the swelling of a vein following the formation of a blood clot), periphlebitis (inflammation of the tissues surrounding a vein), and the existing anatomical pathways (such as the diploë; Minks et al. 2013).

Appearance

Pneumatisation (or the formation of air-filled cavities within bone) occurs when exudate builds up within the mastoid cavity and is unable to be released either through the Eustachian tube or via the perforation of the tympanic membrane. As a result, both the acidity and pressure within the mastoid cavity begins to increase, blood supply is diminished, the diplöic bone is gradually decalcified, and osteoclastic activity is triggered (Mafee et al. 1985; Vazquez et al. 2003). Mastoid abscessing can subsequently occur should the exudate follow the pathways of the emissary veins perforating the mastoid portion of the temporal bone, resulting in the creation of a subperiosteal abscess and the consequential proliferation of periostitis and the sagging of the superioposterior walls of the external auditory meatus (Djeric et al. 2014; Minks et al. 2013; Gliklich et al. 1996). This released exudate can then travel under the sternocleidomastoid muscle of the neck and collect in the parapharyngeal space and the mediastinum (the space in the thorax excluding the lungs), where it can cause further damage to other tissues and organs (Castillo et al. 1998; Dobben et al. 2000; Maroldi et al. 2001). While the abscess and periostitis would presumably be visible on the external surface of the mastoid process, the effects on the soft

tissues and organs would not be visible. Previously pneumatised diplöe can subsequently be filled by branching bone spicules when the body tries to repair the lesions (Fleischer 1979). Lesions can be seen radiographically (Boenninghaus and Lenarz 2005; Rai 2014), via magnetic resonance imaging (or MRI; Platzek et al. 2014), computed tomography (or CT; Chien et al. 2012; Djeric et al. 2014), radiography, and via the sectioning of the temporal bone (Boenninghaus and Lenarz 2005).

2.2.8.0. Auditory Exostoses

Etiology

Auditory exostoses, which are asymptomatic benign bone tumors that form at the external auditory meatus, are stimulated by aggravation of the periosteum of the external auditory canal. The skin overlying the canal is relatively thin and the periosteum is close to the surface (Hutchinson et al. 1997; Özbek 2012), causing the periosteum to be easily affected by any external stimuli. In association with the direct aggravation of the periosteum is the vasoconstriction that occurs following exposure to cold. This can also stimulate the creation of auditory exostoses by putting pressure on the periosteum, causing the osteoblastic reaction that creates the exostoses (Fabiani et al. 1984; Filipo et al. 1982). Auditory exostoses are not caused by infection. Rather, their presence may help explain the context of other infection-induced bone changes such as otitis and mastoiditis. For example, an ear infection precipitated by swimming may be suggested by the co-occurrence of an auditory exostoses and otitis externa.

The etiological mechanisms behind auditory exostoses are incompletely understood. It is generally agreed that they are either aquatic activity markers or cold stress markers (Deleyiannis et al. 1996; Fabiani et al. 1984; Filipo et al. 1982; Kemink and Graham 1982; Kennedy 1986; Kroon et al. 2002; Umeda et al. 1989) that can be used, in conjunction with other evidence, as indicators of aquatic subsistence strategies and cold stress in archaeological populations (Ascenzi and Balistreri 1975; Crowe et al. 2010; Frayer 1988; Godde 2010; Hutchinson et al. 1997; Kennedy 1986; King et al. 2010; Manzi et al. 1991; Okumura et al. 2007; Özbek 2012; Wang et al. 2005; Wong et al. 1999). Godde (2010) names auditory exostoses as "the most prominent environmentally induced trait" since it is commonly agreed that they are the result of the aggravation of the periosteum within the external ear canal by water colder than 19^{o}C, wind chill, trauma, and/or cold atmospheric temperatures. Biological, chemical, or mechanical stimuli, and genetic susceptibility are also believed to precipitate the creation of auditory exostoses. In light of

our current limited understanding of their cause, however, all discussions will have to be prefaced with this list of hypothesized risk factors (Berry 1975; Hrdlička 1935; Hutchinson et al. 1997; Godde 2010; Okumura et al. 2007; Özbek 2012).

Appearance

Auditory exostoses are benign bone tumors made of dense lamellar bone that form 70% of the time near the posterior edge of the external auditory meatus, 30% of the time either near the superior edge or on the floor of the meatus, and rarely on the roof of the meatus (Wang et al. 2005). This pathological condition is usually bilateral and multiple exostoses can form in the same meatus, sometimes fully blocking off the canal (Godde 2010; Hutchinson et al. 1997; Hyams et al. 1988; Katayama 1998; Kennedy 1986; Okumura et al. 2007; Sheehy 1982; Steinbock 1976: 332; Tommaseo et al. 1997).

2.2.9.0. Trachoma

Etiology

Trachoma, or trachomatous dacryoadentitis, results from a direct or indirect infection of the lacrimal gland with *Chlamydia trachomatis* (Bird et al. 2003; Mabey and Fraser-Hurt 2001; World Health Organization 2014). Direct infections most often occur via person-to-person contact or potentially via flies, which act as vectors for carrying the bacteria. Indirect infections, on the other hand, occur when the bacteria spreads from a primary site of infection to the eye via the ductules and lymphatics of the orbit (Aufderheide and Rodríguez-Martín 1998: 251; Emerson et al. 1999; Webb 1990; World Health Organization 2014).

Lesions result from the swelling of the conjunctival lining (the inner lining of the eyelids adjacent to the eyeball) and the tarsal plate (the cartilaginous plates within the eyelids, which may abscess). This progression causes the eye to swell shut or nearly shut (Strano and Font 1976; Webb 1990), and subsequently leads to entropion (the shortening and bending of the upper eyelid; Mabey and Fraser-Hurt 2001), trichiasis (the backwards growth of the eyelashes towards the eye; Mabey and Fraser-Hurt 2001; World Health Organization 2014), and the inflammation and scaring of the cornea from inadequate tear film and scratching from the eyelashes (Mabey and Fraser-Hurt 2001; Munoz and West 1997; Strano and Font 1976; World Health Organization 2014). If trachoma is allowed to progresses fully, the individual will, in all likelihood, go blind in the infected eye (Strano and Font 1976; Webb 1990). Bacterial infections secondary to trachoma

may arise as a result of the drying of the conjunctival lining and further add to the damage (Strano and Font 1976).

There are two forms of trachoma: acute and chronic. Acute trachoma infections usually occur in children, below the ages of 10–15 years old, with blindness following the initial acute infection 15–20 years later due to the damage to the cornea (Ladany and Sarov 1985). Re-infection can then occur and may lead to chronic trachoma (Ladany and Sarov 1985; Mabey and Fraser-Hurt 2001). This is often determined by the degree of an individual's *C. trachomatis* loading, the duration of their last infection, and individual variation (Bobo et al. 1997).

Appearance

The bony lesions that result from chronic trachoma are relatively unique in their morphology from others that occur in the eye orbit. They generally form in the lacrimal fossa of the superior orbital surface (Euber et al. 2007; Webb 1990). Lesions usually comprise of one or two round or oval perforations in the cortex and the destruction of the trabecular bone within (Euber et al. 2007; Webb 1990). Lesions are usually no larger than 12 mm wide and 4 mm deep, they never perforate completely through the frontal bone of the orbit, the surrounding bone is usually porous and/or discoloured, and the margins of lesions are well defined (Webb 1990). Since clinicians rarely deal with dry bone, it is in the theoretical hands of paleopathologists to link these suspected lesions with trachoma and to report on all the details of their finds (Aufderheide and Rodríguez-Martín 1998: 251).

2.2.10.0. Cribra Orbitalia and Porotic Hyperostosis

Cribra orbitalia and porotic hyperostosis (or COPH) are small porotic lesions that appear on the roofs of the orbits and on the cranial vault, respectively. Porotic hyperostosis appears most often on the parietal, frontal, and occipital bones, but there is some confusion as to the degree of involvement of the later two (Aufderheide and Rodríguez-Martín 1998: 348; Buikstra and Ubelaker (ed.) 1994: 115; Roberts and Manchester 2005: 229). Cribra orbitalia appears on the anterolateral portion of the orbital roofs (Aufderheide and Rodríguez-Martín 1998: 349).

Etiology

In 1885, porotic hyperostosis was first identified and named by Welcker, and in 1966 Angel separated and named cribra orbitalia (Angel 1966; Welcker 1885). Today, porotic hyperostosis is also known as spongy hyperostosis, cribra cranii, and symmetrical osteoporosis (Ortner 2003: 55). Cribra orbitalia can occur separately from porotic hyperostosis, though the

latter is rarely seen without the involvement of the orbits (Aufderheide and Rodríguez-Martín 1998: 349; Lallo et al. 1977; Roberts and Manchester 2005: 230). This pattern has left many researchers believing that cribra orbitalia is the result of a more sensitive process than is porotic hyperostosis (Aufderheide and Rodríguez-Martín 1998: 350; Lallo et al. 1977; Stuart-Macadam 1989a ; Wiggins 1991). Porotic hyperostosis and cribra orbitalia are often considered separately (Lallo et al. 1977; Wiggins 1991), though their relationship is acknowledged and continues to be researched (Lallo et al. 1977).

To what these lesions are responding is still incompletely understood. The dominant theory has been, and generally continues to be, that these lesions are caused by iron-deficiency anemia (El-Najjar et al. 1976; Stuart-Macadam 1989a, b; 1992: 151–6; Taylor 1985; Ubelaker 1992). Iron is crucial in the creation of the hemoglobin in red blood cells, with 90% of the iron from previous red blood cells being recycled and reused to form new red blood cells (Provan et al. 2015: 579). Hemoglobin is primarily responsible for the transfer of oxygen (Provan et al. 2015: 575–7; Roberts and Manchester 2005: 226; Steyn et al. 2014). Iron can be diminished due to genetic factors, trauma, chronic disease, menstruation, pregnancy, nutrition, *etc.* (Provan et al. 2015: 578–80; Steyn et al. 2014). When stores of iron are diminished, and the lifespan of red blood cells are cut in half as a result, it is theorized that the body tries to compensate for this by creating more red blood cells. Red blood cells are created within red bone marrow which is located, among other areas of the skeleton, in the diplöe of the cranial vault. As a result, it is theorized that the body stimulates the growth of the diplöe during times of iron deficiency in order to create more red blood cells (Steyn et al. 2014).

It is just this relationship, however, that remains controvercial. Iron deficiency anemia may also be the result of iron binding by the body in an adaptive immune response to the presence of an infectious disease (Ong et al. 2006). This is the reason why COPH can now be considered to be potential indicators of infection (Goodman and Rose 1990; Goodman et al. 1988; Kent 1986; Reinhard 1988; Steinbock 1976; Stuart-Macadam 1988, 1989b, 1990, 1991, 1992a; Weinberg 1992; Williams and Nesse 1991). Infectious organisms require iron in order to reproduce. As a result, when an inflammatory response is triggered by the immune system, free iron is bound to proteins such as ferritin in order to withhold the iron from the organism (Thurnham et al. 2010). As a result, the organism is more likely to die. The state of anemia that this places the body into is a function of this immune response and is called “anemia of

inflammation" or "anemia of chronic disease" (Aufderheide and Rodríguez-Martín 1998: 349; Jurado 1997; Miller 2016; Weinberg 1984; 1992). This immune response and our understanding of it are not perfect (see Holland and O'Brien 1997; Miller 2016; Pasvol and Abdalla 1999: 1552; or Stuart-Macadam 1992a: 159–60 for example), but it expands the current discussion from one of stress and nutrition, to one of general health and is in line with the type of new research required for us to understand the etiology of these lesions.

An influential paper by Phillip Walker and colleagues (2009) argues that COPH is the result of vitamin B_9 and B_{12} deficiencies that cause megaloblastic anemia and the expansion of the cranial diplöe or of vitamin C deficiencies that cause scurvey and sub-periosteal bleeding. The basis for this argument is body's decreased ability to produce mature red blood cells when iron stores are reduced. Their discussion is very compelling and forces paleopathologists to examin their understanding of COPH—a discussion, they note, that does not occur in the clinical literature and that must, then, occur within the relm of archaeology (2009). That said, summarizing and deliberating upon the full range etiological mechanisms behind COPH lesions is beyond the scope of this work, the focus of which is non-specific infection. Presenting all the arguments, though, remains essential to understanding the lesions here and to moving the larger discussion forward.

Appearance

Both cribra orbitalia and porotic hyperostosis result from the same bony processes: the initial thinning of the external cortex; hematopoietic marrow hyperplasia and the vertical reorganization and expansion of the diplöe; the pressure atrophy and perforation of the remaining external cortex and its subsequent obliteration; and, finally, the further thickening and perpendicular growth of the diplöe, that causes the vault to be thicker in the regions of the lesions. Radiographically, the diplöe take on the appearance of sun rays emanating from the internal cortex, also referred to as a "hair-on-end" appearance (Aufderheide and Rodríguez-Martín 1998; Ortner 2003: 55; Steinbock 1976). The inner cortex is very rarely affected (Aufderheide and Rodríguez-Martín 1998: 348–9, Guillen 1992: 162–3; Ortner 2003: 55, 102–3).

The porosity that lesions such as these create on the external cortex at first appear macroscopically to be no more than isolated areas of fine grained pitting (Ortner 2003: 103), and then becomes more identifiable as irregular pitting, some of which is converged, creating pores up to 2mm in diameter (Ortner 2003: 55). This gives the cranial vault or orbits the appearance of

coral (Wright and Chew 1998) or pumice stone (Ortner 2003: 55). A single lesion can be irregular in its appearance, though the lesions as a whole are generally symmetrical and bilateral (Aufderheide and Rodríguez-Martín 1998: 348; Roberts and Manchester 2005: 230) and, in the case of cribra orbitalia, appear in both orbits more often than in just one (Aufderheide and Rodríguez-Martín 1998: 349).

2.2.11.0. Summary

Periostitis, osteomyelitis, periodontal disease, caries, sinusitis, the infection of a concha bullosa, otitis externa and media, mastoiditis, trachoma, and COPH are all non-specific infections that leave distinct lesions on the human skeleton characteristic of the immune response responsible for their creation. An analysis of such infectious indicators allows for paleoepidemiological reconstructions regarding past populations and for an analysis of the mortality and morbidity patterns that would have resulted from such lesion frequencies. Paleopathological studies communicate information based on both the health of the population as a whole and of the subgroups that compose the population. Interactions among these subgroups and between these subgroups and the infections they had all communicate information regarding the life ways of the population as a whole. This study aims to do just that.

Chapter 3
Materials and Methods

3.0.0.0. Materials and Methods

3.1.0.0. Materials

There exist throughout the Cis-Baikal both relatively large and small middle Holocene cemeteries. This research is concerned with three of the large cemeteries: Shamanka II (n=155), Lokomotiv (n=99), and Ust'-Ida I (n=67). The Shamanka II cemetery is located on the southwestern tip of Lake Baikal in the South Baikal microregion; Lokomotiv is located at the confluence of the Angara and Irkut Rivers in the Angara Valley microregion; and Ust'-Ida I is located at the confluence of the Angara and Ida Rivers, also in the Angara River Valley microregion (see Figure 1.2).

Shamanka II and Lokomotiv represent the Early Neolithic (or EN) population, while Ust'-Ida I represents the Late Neolithic to Early Bronze Age (or LN–EBA) populations. This is, however, a generalization. More specifically, Shamanka II contains burials dating to the EN Kitoi culture (n=155), the EBA Glazkovo culture (n=10), and the Iron Age (n=1); and Ust'-Ida I contains burials from the LN Isakovo-Serovo culture (n=48) and the EBA Glazkovo culture (n=19). In this study, only individuals from Shamanka II who were dated to the EN were included in the study. Finally, Lokomotiv contains no temporal anomalies. All of the burials therein are from one time period and culture: the EN Kitoi.

Of the 321 individuals represented by these sites, I studied 250 of them. Individuals were considered for the study if they could be distinguished as those of a single individual (i.e., comingled remains could be separated out into distinct individuals). Due to time constraints during data collection, only one third of the individuals from Lokomotiv (n=32/99) were studied and could be considered for analysis. The sample of middle Holocene burials considered in this study can be broken down by sex as follows: 61 females, 101 males, and 88 individuals whose sex was not able to be determined. In addition, it can be broken down by age as follows: 34

individuals less than four years old, 59 individuals between four and 20 years old, 142 individuals between 20 and 50 years old, 14 individuals older than 50 years old, and one individual whose age was not able to be determined. Sex and age data were provided by Dr. Angela Lieverse and reflected the combined efforts of a number of researchers (Link and Lieverse worked with the individuals from Lokomotiv and Ust'-Ida I; Haverkt, Faccia, Waters-Rist, Yahdati, Antonova, and Lieverse worked with the individuals from Shamanka II; Link 1996; Lieverse 2005; Lieverse et al. in press). Sex and age assessment was largely based on the standards laid out by Buikstra and Ubelaker (ed. 1994: 16–21). Individuals identified as probable females and probable males were pooled with females and males, respectively, for analysis. Table 3.1 provides a detailed breakdown regarding the distribution of the sexes and age groups within each of the cemeteries.

Table 3.1: Demographic profile of the sampled Shamanka II, Lokomotiv, and Ust'-Ida I cemeteries (data from Lieverse 2005; Lieverse et al. in press; Link 1996).

Cemetery	Age Group (in years old)		Sex		Pooled Sex	
Shamanka II	Less than 4	20	Female	23	Female	37
	4–20	25	Male	61	Male	69
	20–50	98	Probable Female	14	Unobservable	45
	More than 50	7	Probable Male	8		
	Unobservable	1	Unobservable	45		
Lokomotiv	Less than 4	3	Female	12	Female	12
	4–20	6	Male	13	Male	13
	20–50	21	Probable Female	0	Unobservable	7
	More than 50	2	Probable Male	0		
	Unobservable	0	Unobservable	7		
Ust'-Ida I	Less than 4	11	Female	11	Female	12
	4–20	28	Male	19	Male	19
	20–50	23	Probable Female	1	Unobservable	36
	More than 50	5	Probable Male	0		
	Unobservable	0	Unobservable	36		

Excavation first occurred at the site of Shamanka II in 1962 and 1965 when A. V. Tivanenko unearthed three graves. The site was then revisited in 1998 and 1999 when A. V. Kharinskii and G. V. Turkin excavated an additional six graves (Turkin and Kharinskii 2004: 124–58). The remainder of the site (61 graves representing 159 individuals) was excavated

between 2000 and 2008 by the Baikal Archaeological Project under the supervision of V. I. Bazaliiskii. One grave was known to have been destroyed by erosion at that time (Bazaliiskii and Weber 2005: 16–21).

The site of Lokomotiv has not been entirely excavated, and of what has been, many graves have been destroyed (Bazaliiskii 2010: 65). Lokomotiv was discovered in 1897 when two graves were accidentally uncovered and destroyed during the construction of the Trans-Siberian Railway (Ovchinnikov 1904: 67–71). In 1927, five graves were excavated, followed by 21 graves between 1946 and 1959 (Khoroshikh 1966: 84–93; Okladnikov 1974: 35–45). Fifty-nine graves were finally excavated between 1980 and 1997 by the Irkutsk State University Laboratory of Archaeology and Ethnography (Bazaliiskii and Savelyev 2003). In total, these 87 graves represent 99 individuals.

Excavation at the site of Ust'-Ida I began in the 1980s and then recommenced in the mid-1990s, at which point the remainder of the site was excavated (Goriunova and Novikov 2010: 252; Link 1999; Weber et al. 2002). Today, the remains from Ust'-Ida I are housed at Irkutsk State University in Irkutsk, Russian Federation, as are most of those from Shamanka II and Lokomotiv.

3.2.0.0. Methodology

3.2.1.0. Laboratory Methods

Data were collected over a two month period (the periodontitis data were collected by my colleague, Megan Clarke; see below) while visiting Irkutsk State University, Russian Federation. I was granted permission to conduct a non-destructive systematic macroscopic examination of the skeletons from Shamanka II, Lokomotiv, and Ust'-Ida I. Bones were examined macroscopically in natural light using the naked eye. A magnifying glass (8x) was occasionally used to better study the morphology of a lesion. An endoscope and hand-held x-ray machine were used to examine bones for otitis, sinusitis, or mastoiditis (discussed below). When a lesion was observed, its presence was recorded (at the level of the lesion). After examining the entire skeleton, if a type of lesion was not present or was unobservable (as defined below), it was recorded as such at the level of the individual (for further reference, see my dataframe in Appendix A).

The importance of analyzing as many skeletal elements as possible was paramount in order to obtain a sample size that adequately represented the middle Holocene populations.

Unfortunately, there is no consensus in the literature as to the number of bones that have to be present in order for an individual to be considered observable for a pathological condition. Many researchers determine how they will discriminate between sufficient and insufficient preservation and intactness of bones according to a set of criteria tailored specifically to the type of infection that they are researching. In her comprehensive study concerning the etiology of sinusitis for example, Charlotte Roberts (2007: 797) considered one sinus a representative sample size and did not discount an individual if only one was observable. Similarly, Anne Kennleyside (2003) considered the presence of over two thirds of long bones sufficient to analyze the relationship between infection and trauma. For studies involving periostitis, it has been common for one tibia to be all that is required in order for an individual to be considered observable (e.g., Da-Gloria et al., 2011; DeWitte and Bekvalac 2011) because this is the element most often affected by the condition (Klaus, 2014; Ortner, 2003: 209; Roberts and Manchester, 2005: 172-173; Weston, 2008).

For each type of infection-induced lesion included in this study, a different set of criteria specific to that lesion type—indicating which and how many bones had to be present—was used to determine whether or not it was observable on a given individual (see below). In all cases, skeletal elements were considered observable only if they were at least one third complete and their surface features discernible with either the naked eye or with the aid of an endoscope or x-ray generatorraph. According to this, the number of bones that could be observed differed from the number that would be present under ideal preservation conditions. For example, when collecting data concerning mastoiditis, if over two thirds of the mastoid process was absent and/or obscured, then the element would be considered unobservable and would be recorded as such in the dataframe.

If a diagnostic lesion was discerned on any bone, then it was considered observable and the condition as present regardless of what other elements were recovered or how complete they were. Data integrity was maintained by excluding lesions that may have resulted from conditions other than infection. For example, if periostitis was visible on a radius adjacent to an incompletely healed fracture, it was not recorded since the periosteal lesion could not be definitely considered the result of an infection unrelated to the traumatic event. This is an effective way to eliminate some sources of error in the data (Keenleyside, 2003; Novak et al., 2009).

For periostitis, individuals were considered observable if at least one third (or four) of their twelve large long bones (the left and right femora, tibiae, fibulae, humei, radii, and ulnae) were present and observable according to the criteria listed above (i.e., at least one third present with discernible surface features). Since periostitis can present on single bones, bilaterally, or systemically (Ortner, 2003: 211; Roberts and Manchester, 2005: 173; Weston, 2008), requiring at least one third of the large long bones to document observability runs the risk of missing some isolated cases, but is likely to catch most bilateral and systemic ones while still representing the population as a whole.

Periostitis was considered to be present if bony striations parallel to the long-axis of the bone or plaque-like bone formations were visible overlying the existing cortex (Roberts and Manchester 2005: 172), and were independent of muscle attachment sites (as these lesions are likely to reflect other etiological mechanisms, particularly mechanical ones; see DeWitte and Bekvalac 2011 and Papazoglou-Manioudaki et al. 2010, for example) and traumatic lesions (such as fractures; see Belcastro et al. 2007 and Da-Gloria et al. 2011, for example). For periosteal lesions composed more of irregular (new) woven bone than of regular (mature) lamellar bone (separate from the original, healthy cortex), the lesion was recorded as "active". If a lesion was composed more of regular lamellar bone than of irregular woven bone, then the lesion was recorded as "healing". "Chronic" conditions were recorded if a mix of both types of bone was present (Mays 1998: 123) and "healed" lesions were recorded if only regular lamellar bone was present (Weston 2008). A number of researchers (e.g., Belcastro and colleagues 2007; Buckley 2000; Da-Gloria and colleagues 2011; DeWitte and Bekvalac 2011; Peck 2013; Ubelaker and Pap 1998) diagnosed periosteal lesions similarly in their studies. If an observable individual did not exhibit these lesions, then the condition was considered to be absent.

Documentation of osteomyelitis was focused on, but was not limited to, the examination of the long bones, but since these lesions appear most often on the long bones (Aufderheide and Rodríguez-Martín 1998: 172; Ortner 2003; 181; Roberts and Manchester 2005: 169–72; Steinbock 1976: 60–2), at least one third of the 12 large long bones had to be present and discernible for an individual to be considered observable for the condition. A diagnosis for osteomyelitis required at least one of the following to be present independent of a traumatic lesion: a cloaca, a sequestrum, or an involucrum (Aufderheide and Rodríguez-Martín 1998: 172; Ortner 2003: 195). Traditionally, all three features have to be present for a diagnosis of

osteomyelitis to be made, but Ortner (2003: 195) warns that this is often not the case (see section 2.2.0.0. for more details; Ortner 2008: 196). Thus, if any one of these features was observed, then osteomyelitis was considered to be present. If these features were not recorded on an observable individual, then osteomyelitis was considered to be absent.

An individual was considered to be observable for trachoma when at least one third of the left or right orbital portion of the frontal bone was present. Trachomatous lesions were diagnosed as one or more well defined round or oval perforations in the cortex of the orbital surface of the frontal bone with destroyed trabecular bone within (Euber et al. 2007; Webb 1990; see section 2.9.0.0. for more detail; Webb 1990). When these lesions were not discernible on observable individuals, then trachoma was documented as absent.

For cribra orbitalia and porotic hyperostosis (or COPH), the diagnostic bones or features considered had to be at least one third complete and discernible for the individual to be deemed observable for the lesion. For cribra orbitalia, the orbital surfaces of the frontal bone were considered (similar to trachoma, see above), and, for porotic hyperostosis, the frontal, parietal, and occipital bones were considered. Only one of these features/bones had to be present and at least one third of its surface discernible for an individual to be considered observable for cribra orbitalia or porotic hyperostosis, respectively. While these lesions were described separately, they were documented together in the dataframe.

COPH were considered to be present when the outer cortex of the diagnostic bones or features showed signs of thinning and had irregular and/or converging areas of pitting (Ortner 2003: 103 and 55), that were independent of muscle attachment sites. These lesions could range from having the appearance of subtle pitting similar to that of coral (Wright and Chew 1998) to more pronounced porosity similar to pumice stone (Ortner 2003: 55; for greater detail, see section 2.2.10.0.). How thin the outer cortex appeared could only be judged macroscopically with the human eye (and was noted in the comments section of the dataframe), but the morphology of the diplöe could not be examined at all because cross sections of the skulls could not be taken. These limitations meant that a definitive diagnosis of anemia-related porotic hyperostosis could not be made. This will be discussed further in the following chapter. When these lesions were not visible on observable individuals, COPH was documented as absent.

The observability of sinusitis (and otitis media, see below) depended often on the use of the endoscope. This was a flexible endoscope (SuperEyes Y001 5mm) with a light of two

different strengths that plugged into my computer using a USB. A camera at its end provided a live image to my computer of what it was seeing. The hardware on the endoscope allowed for digital pictures to be taken in real time using the camera.

When access to a sinus was available but was too small or obstructed for it to be observed with the human eye alone, then the documentation of sinusitis was dependent on the use of the endoscope. Often, the sinuses were accessed by first threading the endoscope through the nasal cavity then through fractures in a wall of the sinus (see Figure 3.1). When the bones of the skull were disarticulated and large fractures existed in the walls of the sinuses, the sinuses were accessed directly via these fractures (i.e., without the use of the endoscope). Individuals were observable for sinusitis when at least one third of one sinus cavity was present and discernible, either directly (e.g., through a fracture in the cranium) or via the endoscope.

Figure 3.1: the use of the endoscope to view the right maxillary sinus via the nose and a fracture in the medial wall of the right sinus.

Sinusitis was considered to be present when spiculated, lobulated, or plaque-like bone formation, a bony cyst, and/or white highly pitted bone appeared inside the sinus cavity (see 2.4.0.0. for more detail regarding the lesion's appearance; Boocock et al. 1995; Merrett and Pfeiffer 2000). When these lesions were not visible on observable individuals, sinusitis was considered to be absent.

The infection of a concha bullosa was observed when at least one (left or right) inferior nasal concha and the vomer were present and at least one third of each was complete. When this was the case, such an infection was documented as present when the nasal concha was abnormally large, in so much as the abnormal shape of the nasal concha affected the regular morphology of the vomer. If the nasal conchae present appeared to be a typical size and/or the vomer was strait, then the infection was recorded as absent.

For otitis externa, one third of the walls of an external auditory meatus of either temporal bone had to be present and discernible for the individual to be considered to be observed for this lesion. Otitis externa was considered present if both the following conditions were observed: 1) fine-grained pitting was present around the external auditory meatus (Mays and Holst 2006) and 2) there was destruction (appearing as divots) visible on the walls of the external auditory meatus (Mays and Host 2006). If neither of these features was observed, then the lesion was documented as absent.

When individuals' outer and middle ears were clear of sediment, or when sediment was present but could be removed without damaging the integrity of the bone, then the endoscope was used to observe the middle ears. If at least one third of one (left and/or right) middle ear was present and discernible, then the individual was considered to be observable for otitis media. When viewing the middle ear, the endoscope was inserted through the outer ear canal (see Figure 3.2) and into the middle ear just past where the tympanic membrane would have been in life. From this vantage point, approximately one third the middle ears could be seen.

Chronic otitis media is often associated with the formation of a cholesteatoma, mastoiditis, and the destruction of the auditory ossicles (Aufderheide and Rodríguez-Martín 1998: 253–4; Roberts and Manchester 2005: 177–8). Otitis media was documented as present if a large (approximately 3mm wide) destructive pit was observed within the middle ear. Such pits form as the result of a cholesteatoma (Mays and Holst 2006). Mastoiditis was recorded separately, but otitis externa, otitis media, and mastoiditis were pooled during statistical analysis. It was noted in the comments section if the auditory ossicles (if present) were deformed in any way. If no abnormal bone loss was noted in the middle ear and if the auditory ossicles appeared normal (if present), then the condition was documented as absent.

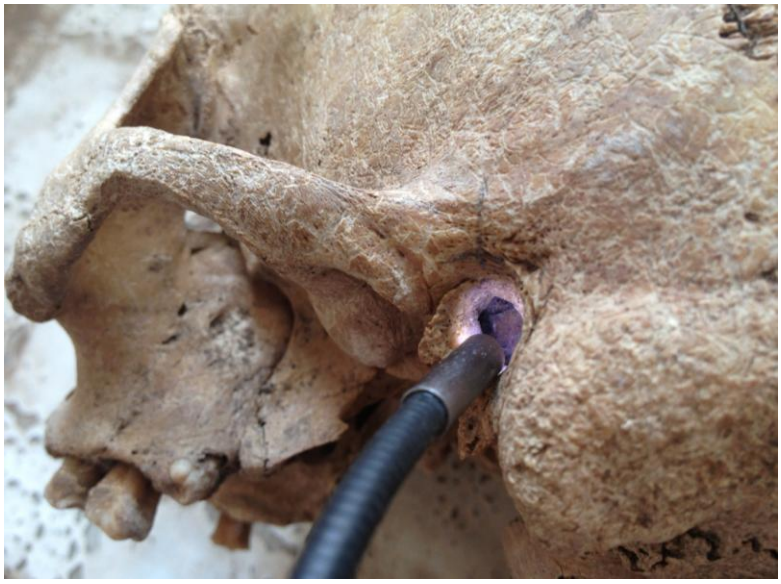

Figure 3.2: How the endoscope was inserted in to the outer ear canal in order to be able to view the middle ear.

Mastoiditis was observed either macroscopically, if the mastoid process was fractured in such a way as to grant visual access to the air cells (see Figure 3.3), or by the use of the NOMAD Pro Hand-Held X-ray System (Aribex, Provo, Utah) and Dr. Suni Plus Intraoral Digital Light Sensor (SUNI Medical Imaging Inc., San Jose, California). The x-ray system is designed for field use and is safe to use. Its sensor plugged into a computer via a USB and its software produced a digital radiograph that could be saved and exported for future use. A mastoid process was considered unobservable when less than one third of it was present or discernible, or when the individual being considered was so young (approximately <10 years old) that the mastoid process was not developed enough to be radiographed (see below).

For a diagnostic radiograph to be taken, the sensor had to be secured to the lateral surface of the mastoid process using modelling clay that remained out of the field of view and the NOMAD Pro had to be positioned as directly opposite to the sensor as possible (the range between the two varied depending on whether or not the temporal bone was articulated with the rest of the cranium; see Figures 3.4 and 3.5). Taking a radiograph as such created an image in which the entire portion of the mastoid that projected beyond the natural curve of the temporal bone was visible.

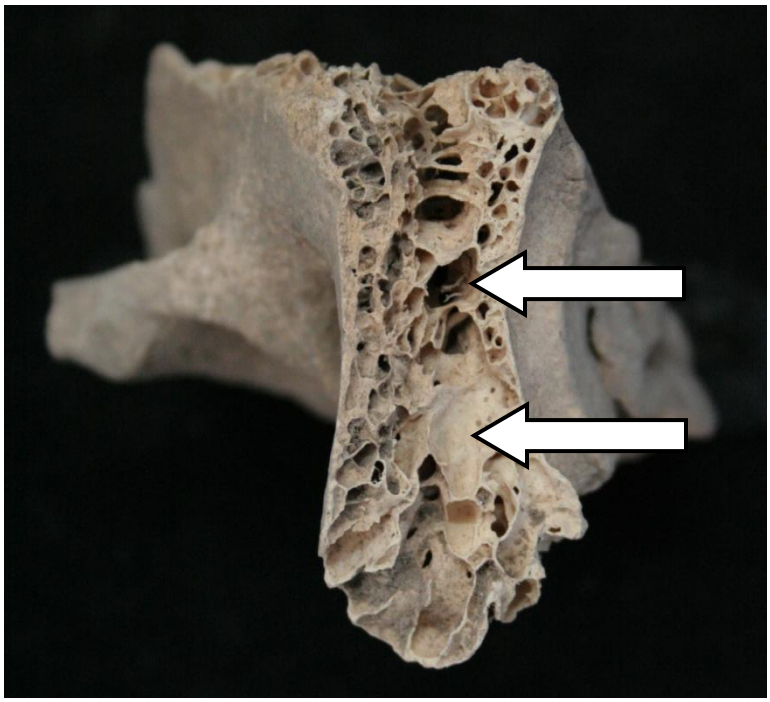

Figure 3.3: Ust'-Ida I 14.1, a left mastoid fractured postmortem to reveal pneumatised diplöe (indicated by arrows).

Figure 3.4: How the sensor was attached to the lateral surface of a mastoid using modelling clay.

Figure 3.5: How the sensor was attached to a mastoid using modelling clay when the temporal bone was disarticulated from the rest of the cranium.

In cases of immature development, a radiograph could not be taken because the mastoid process did not project far enough away from the temporal bone for the sensor to be secured to its lateral side. Securing the sensor on the interior surface of the bone opposite the mastoid process and taking the radiograph from the inferior surface of the mastoid and through the portion of the temporal bone superior to it was not an adequate solution to this problem. This produced too poor of an image for the mastoid air cells to be examined because the bones superior to them also appeared in the image, obscuring the morphology of the air cells.

Mastoiditis was documented as present if at least one third of the mastoid air cells were pneumatised (meaning that the mucous-lined air cells, or diplöe, within the mastoid process were destroyed by the spreading of a purulent infection) and/or if bone proliferation was visible inside the air cells (see Figure 3.6; Fleischer 1979; Mafee et al. 1985; Vazquez et al. 2003). If these lesions were not visible on an observable individual, then mastoiditis was documented as absent.

The superior, inferior, and posterior walls of an external auditory meatus are the structures that were observed for auditory exostoses. One third of one (left or right) auditory exostoses had to be present for an individual to be considered observable for this condition. The anterior wall was not considered, as external auditory exostoses more commonly form on the posterior and

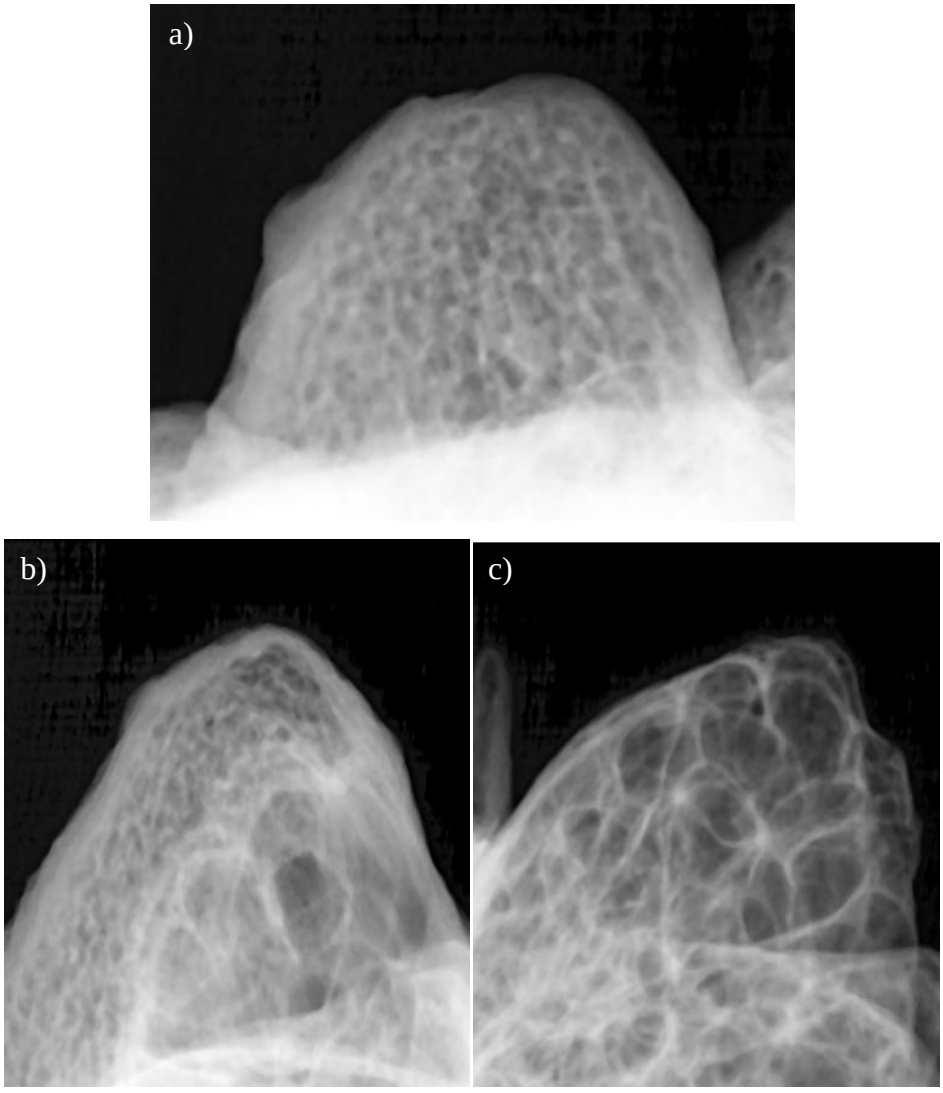

Figure 3.6: Images from the NOMAD Pro x-ray generatorraph: a) Shamanka II 24.1, right, no mastoiditis (a healthy mastoid with relatively homogeneous diplöe); b) Shamanka II 63.1, right, mastoiditis (diplöe beginning to be pneumatised); c) Shamanka II 49.1, left, mastoiditis.

superior walls (Wang et al. 2005). Auditory exostoses were considered to be present when a bone tumour made of dense lamellar bone was discernible on or in the external auditory meatus (Godde 2010; Hutchinson et al. 1997; Hyams et al. 1998; Katayama 1998; Kennedy 1986; Okumura et al. 2007; Sheehy 1982; Steinbock 1976: 332; Tommaseo et al. 1997; Wang et al. 2005). This bone tumour had to obscure the external auditory meatus to some extent and be

differentiated from an irregular bony surface in order for it to be recorded. If this was not discernible, then the lesion was documented as absent.

Caries was recorded tooth-by-tooth for each individual. When a tooth was present with at least two thirds of its surface features preserved (different from the criteria for other lesions), then it was considered observable. An individual was considered observable for caries if over one third (i.e., seven or more) of their posterior teeth (molars and premolars) were present and documented or if a carious lesion was discernible on any tooth regardless of other teeth or their preservation. It is common in the literature for caries to be considered as a proportion (or percentage) of observable teeth exhibiting carious lesions (Belcastro et al. 2007; Cucina and Tiesler 2003; Delgado-Darias et al. 2005; Klaus and Tam 2010; Nelson et al. 1999; Wasterlain et al. 2009; Willis and Oxenham 2013). Since this study analyzed lesions at the level of the individual, rather than the tooth, the presence of seven or more observable posterior teeth was decided upon as a measure of caries observability because it is these teeth that most often exhibit carious lesions (with a modern, western diet in adults; Hillson 1996: 280). Similarly, Mark Hubbe and colleagues (2012) included in their study only those individuals who had 25% of their teeth present and discernible.

Caries appears as a dental tissue-destroying pit that can be observed on any surface of a tooth (Hillson 1996: 284–5). These destructive processes are described in greater detail in Section 2.2.3.0 but, for the purpose of diagnosis, it is simply the presence of a destructive pit in the dental tissue(s) caused by an antemortem chemical process that was considered here. When this lesion was not present on observable individuals, then caries was documented as absent.

A summary of the diagnostic features and/or bones observed for each lesion type can be seen in Table 3.2. Further written and photographic documentation of each lesion supplemented data collection. This was done in order to ensure the consistency of lesion identification while collecting data and to aid in the subsequent differential diagnoses of those lesions that were unusual and/or unique and required a greater deal of attention. Photos were taken using an 8 megapixel Canon digital SLR with a 35 mm lens and were shot onto a black felt background in natural light. Descriptions of the lesions, along with scores based on the Skeletal Pathology Code Key summarized by Buikstra and Ubelaker (ed. 1994), were recorded in a description and comments section, respectively, of my dataframe. Photographs were saved in digital folders along with the endoscope images and radiographs.

Table 3.2: The bones or features required for an individual to be considered observable for each type of lesion (*teeth must be 2/3 present).

Lesion Type	The Bones or Features that Must be at least 1/3 Present and Discernible for an Individual to be Considered Observable
Periostitis	4 large long bones
Osteomyelitis	4 large long bones
Cribra Orbitalia	1 orbital surface of the frontal bone
Porotic Hyperostosis	1 frontal, parietal, or occipital bone
Caries	7 premolars and/or molars*
Periodontitis	1 maxillae or mandible
Maxillary Sinusitis	1 maxillary sinus
Frontal Sinusitis	Frontal sinus(es)
Ethmoid Sinusitis	Ethmoid sinuses
Sphenoid Sinusitis	Sphenoid sinus(es)
Otitis Externa	Walls of 1 external auditory meatus
Otitis Media	1 middle ear
Auditory Exostoses	Superior, inferior, and posterior walls of 1 external auditory meatus
Trachoma	1 orbital surface of the frontal bone

Further written and photographic documentation of each lesion supplemented data collection. This was done in order to ensure the consistency of lesion identification while collecting data and to aid in the subsequent differential diagnoses of those lesions that were unusual and/or unique and required a greater deal of attention. Photos were taken using an 8 megapixel Canon digital SLR with a 35 mm lens and were shot onto a black felt background in natural light. Descriptions of the lesions, along with scores based on the Skeletal Pathology Code Key summarized by Buikstra and Ubelaker (ed. 1994: 114–5), were recorded in a description and comments section, respectively, of my dataframe. Photographs were saved in digital folders along with the endoscope images and radiographs.

Data regarding the presence/absence of periodontitis were collected by my colleague, Megan Clarke, while we were both visiting Irkutsk State University. She collected the data as a supplement to her Master's thesis and allowed me to use them as a part of mine. The remainder of this section pertains to her methodology.

There are two factors that can be considered when identifying periodontitis: morbidity and activity. Morbidity (or the amount of resorbed alveolar bone) is considered by many to be an

accurate indicator of the presence of periodontitis (e.g., DeWitte 2012; Eshed et al. 2006; Lavigne and Molto 1995; Ramfjord 1959). To observe morbidity, a measurement is made from the CEJ of a tooth to the alveolar crest (AC) of the alveolar bone surrounding the tooth. If the distance between the two is greater than 2mm, the alveolar bone is said to have receded as the result of periodontitis (a measurement of 0–2mm being accepted as "healthy"; Lavigne and Molto 1995; Pattison and Pattison 1992; Gargiulo et al. 1961; Ritchie and Orban 1953). There does exist some debate as to which surfaces of a tooth to measure. Davies and colleagues (1969) only measure mesial and distal surfaces, for example, whereas Lavigne and Molto (1995) measure six surfaces: the mesial/lingual, lingual, distal/lingual, medial/buccal, buccal, and distal/buccal.

There are some problems with studying periodontitis using morbidity, however. First, the alveolar crest is highly variable and changes in its appearance can be natural rather than pathological. Second, teeth can continue to erupt into adulthood in an attempt to match dental wear and attrition. And facial growth can occur into adulthood, obscuring the CEJ-AC measurement. Third, alveolar bone is naturally thin and, as a result, is highly prone to post-mortem breakage (Clarke and Hirsch 1991; Hildebolt and Molnar 1991). Continuous eruption was an obscuring factor in the Cis-Baikal samples as dental wear was severe in these populations (pers. comm. Megan Clarke; Lieverse et al. 2007a).

Activity refers to the presence/absence of inflammation as determined by alveolar bone loss and porosity (Clarke and Hirsch 1991; DeWitte and Bekvalac 2011; Lieverse et al. 2007a). Porosity, however, is not always indicative of periodontitis as it may arise as a result of a pulpal infection and not gingivitis or periodontitis. If porosity does arise as a result of gingivitis, gingivitis does not always lead to periodontitis and periodontitis is not always preceded by gingivitis (Hildebolt and Molnar 1991; Clarke and Hirsch 1991).

To avoid overestimating the presence of periodontitis, Kerr (1988, 1989, 1998) developed a system that looks at the form and texture of the interdental walls of the alveolar bone in order to diagnose and score periodontitis. Oztunc and colleagues (2006) modified Kerr's system, while Costa (1982) developed a similar system to score and diagnose periodontitis. The first system scores periodontitis on a four-level system rather than Kerr's six (Oztunc et al. 2006); and the second system looks at the cross sectional shape of the septum and the presence of porosity (Costa 1982).

The methodology used here involves combining different portions of the aforementioned methodologies in an attempt to overcome the problems discussed in each. A two-step system resulted: diagnosis and severity. First, diagnosis involved combining Costa's (1982) descriptions of septal shape and porosity with Oztunc and colleagues' (2006) scoring system. The result was a four level scoring system: 1) normal (the cortical surface is continuous and uninterrupted by foramina or grooves, i.e., there is no porosity and the shape of the interdental septum is convex at the incisors and grades to flat at the molars); 2) gingivitis (the septal form is characteristic of its region; the cortical surface shows a range of foramina and/or small to large grooves); 3) periodontitis (a breakdown of septal contour, i.e., a concave shape, is visible; pores with a sharp, ragged texture or honeycomb effect may be present along the surface; and all defects are rounded); and 9) unobservable (antemortem resorption or postmortem damage make the septum or crest unobservable). Periodontitis was only considered to be present if a score of 3 was given to the septum. To avoid the problems regarding porosity previously discussed, porosity without septal changes are noted (score 2), but are not considered to be indicative of periodontitis.

Second, severity was recorded for all those septa that were scored as being indicative of periodontitis (score 3) by measuring from the CEJ to the AC using a dental probe. This provided additional information regarding the severity of periodontitis via the destruction of the alveolar bone. A seven level scoring system was used here: 0) 0–1.99mm; 1) 2–2.99mm; 2) 3–4.99mm; 3) 5–6.99mm; 4) 7–9.99mm; 5) 10+ mm; and 9) unobservable. Those margins that exhibited a depth greater than 2mm but were not given a score of 3 may represent continuous tooth eruption or facial growth. Attrition and dental wear were not systematically recorded, but severe wear was noted in a comments section of Clarke's dataframe (pers. comm. Megan Clarke; Lieverse et al. 2007a).

When incorporating Clarke's periodontal data with my dataframe, the data concerning the diagnosis of periodontitis, or its presence/absence, were of interest to me. All those individuals who were given at least one score of 3 were considered to have periodontitis and this was recorded in my dataframe at the level of the individual. If at least one third of an individual's interdental septa (as they would have been present in life) was recorded as observable (scores of 1 or 2) and periodontitis was not observed (score of 3), then periodontitis was considered to be absent for that individual. If over one third of an individual's interdental septa were unobservable (score of 9) and periodontitis was not observed (score of 3), then the individual was considered to

be unobservable for periodontitis (i.e., one third of the interdental septa had to be present for the tooth to be considered observable for periodontitis). In this way I was able to incorporate Clarke's data with my own so as to test for patterns regarding dental health in the middle Holocene sample.

3.2.2.0. Analytical Methods

To refresh, my dataframe (or spreadsheet; Appendix A) includes the following information: each individual's identification number, sex, age at death, and the cemetery and time period from which they came. Data regarding the sex and age of each individual were obtained from Dr. Angela Lieverse, who previously determined the age and sex of each of the individuals included in this study. Data regarding sex, age, cemetery, and time period were used as explanatory variables, while the incidence values of the lesion types, which were recorded as binomial presence/absence data were used as response variables. The use of binomial presence/absence data is well documented by other authors (DeWitte and Bekvalac 2011; Keenlysìde 2003; Roberts 2007) and serves as a method for calculating the frequency of infection within and between populations (see below).

The purpose of this study is to see if the populations that were found to have had more physiological stress also exhibited more non-specific infection-induced lesions. The incidence of each type of infection-induced lesion amongst the explanatory variables (age at death, sex, cemetery, and time period) was graphed and studied. Subsequently, these patterns were compared to those patterns of physiological stress documented by previous researchers (Lieverse 2010; Lieverse et al. 2007a; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011).

The research question that was posed from the data is as follows: is the incidence of infection-induced lesions within the sample related to a) the type of infection, b) the sex of the individual, c) the age of the individual, d) the cemetery the individual came from, and/or e) the time period the individual lived in? From this question, two statistical questions were posed. First, are the numbers of individuals with infection-induced lesions significantly different across populations or across sub-groups (e.g., age groups or sexes) within each population? This question draws upon the previous research of BHAP members revealing that there are more indicators of physiological stress on individuals from the EN (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011) and indicators of physical stress on males (Lieverse et al. 2013; 2009). Second, which levels (e.g., males or females) of which

explanatory variables (e.g., sex) are predictive of a positive response variable (i.e., the presence of a lesion of a certain type)?

Organizing the Data

Before these questions could be answered, the data had to be organized. To aid in the process of analysis and comparison, I pooled the individuals' ages documented by Dr. Angela Lieverse into age groups as follows: less than 4 years, 4–19 years, 20–49 years, and over 50 years. This was done so as to create a level of consistency amongst the ages, to facilitate statistical analysis, and to facilitate later analysis and interpretation. The first two age groups were created to facilitate comparison between my infection data and the breastfeeding data previously collected by Waters-Rist and colleagues (2011). They found that EN populations were weaned between the ages of 3.5 and 4.0 years old, and LN individuals were weaned by the age of 3.0 years old. As such, dividing the ages based on the fourth year of life permited for one age group to contain, more or less, all those individuals who are not yet weaned, while these other three groups contain those who have been weaned.

Presence data, which were originally collected at the level of the lesion (considered observable if one third, or two thirds for caries, of the diagnostic surface was visible; see above) in order to capture the greatest level of detail possible and to aid in the potential differential diagnosis of lesions with obscure etiologies, were then pooled to the level of the individual for statistical analyses (as previously discussed, see Figure 3.2). This was done because, for the purpose of the research question, the presence or absence of lesions was more important than the number of lesions.

Two new groups of lesions ("Upper Respiratory Infection" and "Otitis") were then created at the level of the individual by pooling data. "Upper Respiratory Infection" was created by pooling the presence/absence data from each type of sinusitis lesion recorded (frontal, maxillary, ethmoid, and sphenoid) and the data from the infection of a concha bullosa. This was also done with the present/absent results for otitis externa, otitis media, and mastoiditis to create the response variable "Otitis". In pooling the data, the new lesion types were considered "present" if at least one of the original variables had been recorded as "present". The new lesion types were considered to be "absent" if none of the original variables had been recorded as "present" and at least one of the original variables had been recorded as "absent" regardless of if the other variables were recorded as "observable" or not. The new lesion types were only

considered to be “unobservable” if all the original variables had been recorded as “unobservable”. Just one variable recorded as “present” or “absent” was required for the individual to be recorded as “present” or “absent”, respectively, in order to represent the individual as a whole whenever information was available.

These new response variables were created in order to focus more clearly on these two areas of interest: the upper respiratory system and the ears. Infection-induced lesions in one of these areas are indicative of an infection in the larger system. For example, maxillary sinusitis is indicative of an upper respiratory infection. The fact that one of these more specific types of lesions may not have been observable or may not have exhibited a lesion indicative of an upper respiratory or ear infection was not of interest in this study if another bone in the system did exhibit a lesion. By condensing these data, I obtained the level of detail I required for statistical analysis and reduced both ambiguity and sources of error within my data.

Statistical analysis included binomial tests and generalized linear model logistic regressions. All of these tests were run using the open source software, R. Chapter 4 outlines the results of these tests in detail and Chapter 5 discusses the results in relation to the research question.

Proportions

First, to better understand the distribution of the lesions that were documented, incidence data were calculated for each lesion type. For each condition, this was expressed as a percentage of observable individuals who exhibited one or more lesions. The dataframe was then subdivided and these incidence values were recalculated by subgroup (e.g., individuals by sex, sex by time period, age group, age group by time period, and time period). These percentages can be easily compared between the different categories and represent the distribution of the data across time and space.

Binomial Tests

In order to answer the statistical question, binomial tests for equality of proportions were performed. Binomial tests are a type of proportion test designed to determine if proportions from two different populations are significantly different from one another or if they could have occurred through chance alone (Crawley 2013: 365). This type of test was chosen because the data being used were binomial (present/absent). Binomial tests are also robust against small sample sizes, which occasionally occurred here. The proportions of each lesion type were

analyzed by time period, sex, sex in each time period, age group, and age group in each time period so as to determine which, if any, of these sub-populations presented with more infection-induced lesions than others. Each test was run with a significance level of 95%.

As for the chi-square tests, no statistical corrections were made here to stop errors related to repeated tests. While binomial tests relating to individual lesion types were often compared within sub-populations (e.g., males), they were also compared to other sub-populations (e.g., males *vs.* females). As a result, no corrections were made at the level of the sub-population.

Chi-Square Tests for Co-occurrence

Chi-square tests were conducted to expand on the results of the first statistical question by developing an understanding of the relationships among the lesions themselves, when controlling for age, sex, time period, and cemetery. This was done by using the same raw frequency data used to calculate the binomial tests. Chi-square tests used categorical and numerical (or frequency) data to test the null hypothesis that these lesions did not co-occur any more than would be expected through chance alone.

In order to perform a chi-square test, contingency tables were created to structure the frequency data as follows. The x-axis contained the number of observable individuals who exhibited, and did not exhibit, the first lesion being tested (e.g., periostitis) and the y-axis contained the number of individuals who exhibited, and did not exhibit, the second lesion being tested (e.g., periodontitis). As a result, cell XY contains the number of individuals who have both lesions recorded as present; cell xY contains those individuals who have the first lesion recorded as absent and the second as present; cell Xy contains those from the EN who have the first lesion recorded as present and the second as absent; and cell xy contains those who have both lesions recorded as absent (see Table 3.3). This creates a 2x2 table in which the Xy cell contains the number of times, for example, both periostitis and periodontitis were present in the same individual (i.e., the number of times periostitis and periodontitis co-occur); cell xY contains the number of times periostitis was absent and periodontitis was present in the same individual; cell Xy contains the number of times periostitis was present and periodontitis was absent in the same individual; and cell xy contains the number of times both lesions were recorded as absent in the same individual (see Table 3.4).

Traditionally, chi-square tests are done by hand using the formula (**3.1**) $X^2=(\Sigma(o-e)^2)/e$, in which *o* represents the observed frequency and *e* represents the expected frequency. However, I

used the open source software, R. R calculates the critical value of chi-squared, the test statistic, the p-value, and the degrees of freedom for each test. The degrees of freedom (traditionally calculated as *df=(number of rows in the probability table–1)x(number of columns in the probability table–1)*, and the probability value can then be used to calculate the critical value of chi-square test. The probability value tells us the probability that the observed frequency is due to chance alone. Five percent (or α=0.05) is the standard value used to reduce the chances of committing a Type 1 error (or the rejection of a true null hypothesis).

Table 3.3: How the contingency tables for the chi-squared tests were constructed.

	X	**x**
Y	XY	xY
y	Xy	xy

Table 3.4: An example of a contingency table used to test if periostitis and periodontitis co-occur significantly more than would be expected through chance alone.

	Periostitis Present	**Periostitis Absent**
Periodontitis Present	77	9
Periodontitis Absent	33	13

If the test statistic is larger than the critical value, then the null hypothesis is rejected and the alternate hypothesis (that any co-occurrance cannot be explained through chance alone and is statistically significant) can be accepted. If the test statistic is smaller than the critical value, then the null hypothesis cannot be rejected and chance alone may explain the co-occurrences between them (Crawley 2013: 367–9; Mendenhall et al. 2001: 633–4).

Logistic Regressions

Generalized linear model (or GLM) logistic regressions were conducted in order to answer the second statistical question. This test was chosen because it is robust for categorical explanatory variables, non-homogeneity, and non-normality. This was necessary since the response variables (the lesion types) are binomial and the explanatory variables (Age Groups, Sex, Cemetery, and Time Period) are categorical. Neither the classic tests for outliers, homogeneity, and normalcy could be performed on the response variables, nor could the classic

tests for outliers or homogeneity be performed on the explanatory variables. As such, a GLM robust enough to deal with these sorts of data was required.

It is important to note, for those unfamiliar with logistic regressions, that the first level of each explanatory variable is taken by the model to be the intercept and, as such, there is no p-value associated with them in the results tables of Chapter 4. It is the intercept to which the other levels in an explanatory variable are compared to when assessing their significance in the model. The intercept is where the graph crosses (or intercepts) the y-axis when the data are modeled by the logistic regression and it is this model that allows us to assess if an explanatory variable or its level is predictive of the response variable.

R uses the following equation to model the data: (**3.2**) $\hat{y}_i = b_o + b_1X_{i1} + b_2X_{i2} + ... + b_pX_{ip}$ ($\hat{y}$ is the response variable, b_o is the intercept, and b_pX_{ip} are the parameters—more specifically, b_p are the explanatory variables, and X_{ip} are the dummy variables calculated to model categorical variables as continuous variables on the graph). Once the data are modeled, a response variable can be predicted for any point in the data set or for any explanatory variable.

Models were created using one response variable and one explanatory variable at a time (the dataframe was too large to perform multivariate statistics without crashing R) in order to test the null hypothesis that the presence of a lesion is associated with 1) the sex of the individual; 2) the age of the individual; 3) the cemetery from which the individual came; and 4) the time period from which the individual came. Overdispersion (when the data is not binomial, when the explanatory variables do not contribute any information to explaining the data, and/or when the residual deviance is inflated; Crawley 2013) was always monitored by comparing the null and residual degrees of freedom of each model (i.e., the residual deviance was smaller than the degrees of freedom in each model). Some models were found to be overdispersed. These models show that the variables included therein do not predict for the amount of variation in the data (i.e., there are other variables that, also, account for the variation in the data).

Models were created first to test to see which explanatory variables contributed to a positive response variable and second to test which levels contributed to a positive response variable. Specifically, a linear regression was used to analyze the relationships among the explanatory variables and the response variables. In order to do this, two rounds of tests were performed. This first round tested the effect of the explanatory variables on the response variables and used the entire data frame. The second round tested the effect of the *levels* of the explanatory

variables on the response variable. In order to test the effects of the *diagnostic* levels (such as males and females rather than those classified as "unidentifiable sex"), the non-diagnostic levels were deleted. When running logistic regressions using sex as the explanatory variable, for example, individuals whose sex is recorded as "unidentifiable" were deleted, allowing only those individuals who are known to be male or female to be tested. These non-diagnostic levels were included in the previous logistic regressions because they represent individuals included in the sample whose effects on the presence of a lesion is important, regardless of if they are of a known sex or a known age. When considering the effects of the levels alone, however, the non-diagnostic levels were removed.

For each round of tests, the entire dataframe (minus the non-diagnostic levels for the second round of tests) was used, followed by the dataframe as divided into the time periods (EN and LN–EBA) and the cemeteries (Shamanka II, Lokomotiv, and Ust'-Ida I). This was done in order to compare the effects of the explanatory variables and the levels through both time (EN and LN–EBA) and space (Shamanka II, Lokomotiv, and Ust'-Ida I).

This methodology was designed to provide insight into the nature of culture change in the middle Holocene and to shed further light on the observed decrease in physiological stress in the LN–EBA. The data collection phase was an opportunity for the detailed and systematic documentation of all infection-induced lesions to an extent not previously seen involving the Cis-Baikal individuals, while the analysis phase was an opportunity for the further exploration of the relationships between infection and physiological stress.

Chapter 4
Results

4.0.0.0. Results

4.1.0.0. Minimum Estimates of Incidence

The first statistical question in this study asks whether or not the proportions of individuals with infection-induced lesions differed significantly across populations or across sub-groups (e.g., age groups or sexes) within each population. This was tested using binomial statistics and run on the open source software R. The p-values for each test are listed in the tables below. Those tests that produced a significant p-value (when assessed at α=0.05) are highlighted in grey and are italicized. Those tests that produced nearly significant (or between 0.060 and 0.099) p-values are also highlighted in grey, but are not italicized.

To refresh, the null hypothesis for each binomial test was that there is no statistically significant difference between the two groups (e.g., males vs. females). If the test statistic was larger than the critical value, then the null hypothesis was rejected and the alternate hypothesis, that there is a significant difference between the two groups, was accepted. If the test statistic was smaller than the critical value, then I failed to reject the null hypothesis.

4.1.1.0. Incidence by Lesion Type

There were 250 individuals included in this study. Table 4.1 and Figure 4.1 show the presence and observability of each lesion type. For ease of comparison, the data were turned into percentages (observable individuals affected by each lesion type) for all of the figures so as to facilitate comparisons among the categories. Raw data are available in the tables. This is how all subsequent data in this section are presented. In descending order, those types of lesions that were the most frequently observed in the skeletal population are as follows: otitis (99.4%), periostitis (76.1%), upper respiratory infections (70.6%), periodontitis (65.9%), cribra orbitalia and/or

Table 4.1: Summary of lesion incidence in each pathological category (all individuals pooled).

Lesion Type	Incidence, X(n)
Periostitis	169 (222)
Osteomyelitis	1 (172)
COPH	80 (161)
Caries	9 (124)
Periodontitis	91 (138)
Upper Respiratory Infection	89 (126)
Otitis	161 (162)
Auditory Exostoses	2 (169)
Trachoma	0 (166)

X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis.

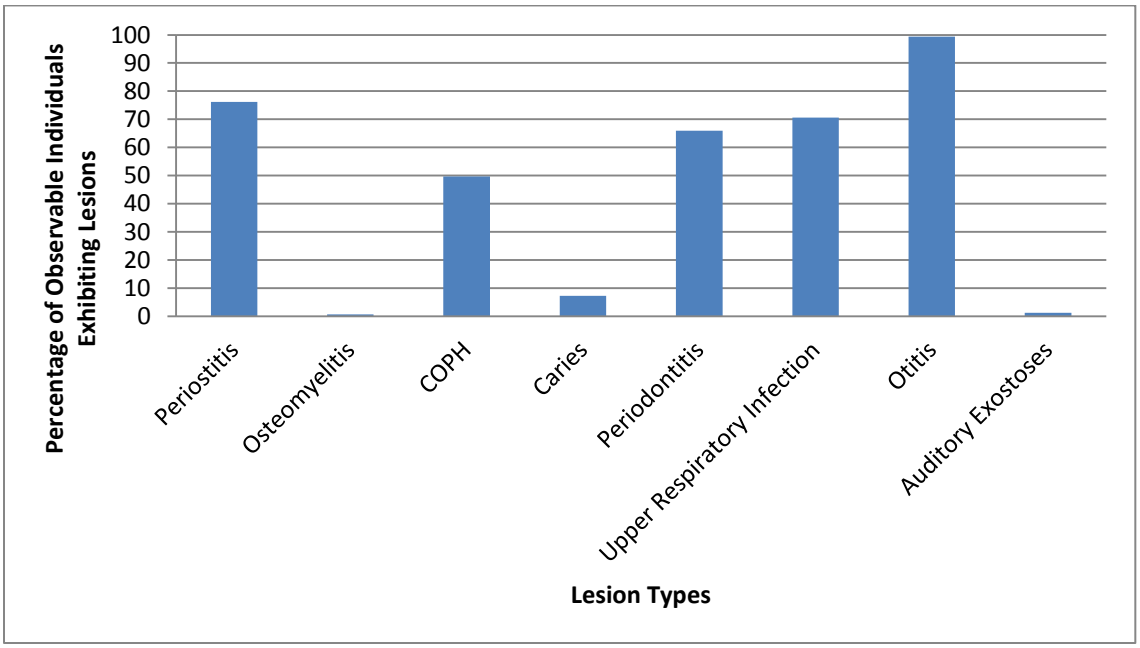

Figure 4.1: Lesion incidence for all individuals (pooled).

porotic hyperostosis (or COPH; 49.7%), caries (7.3%), auditory exostoses (1.2%), and osteomyelitis (0.6%). Trachoma was completely absent in the sample. Otitis was almost ubiquitous within the sample. Periostitis, upper respiratory infections, and periodontitis were also very common, with more than half of the sample exhibiting characteristic lesions. COPH appeared in almost half the sample. Caries and auditory exostoses appeared in less than ten

percent of the individuals. And finally, osteomyelitis and trachoma were extremely rare and absent, respectively. Since trachoma was not documented at all, it was excluded from analyses.

4.1.2.0. Incidence by Time Period

Data were divided by time period: Early Neolithic (or EN) and Late Neolithic to Early Bronze Age (or LN–EBA). There were 183 EN individuals and 67 LN–EBA individuals included observed. When comparing the EN and the LN–EBA (see Table 4.2), there were proportionally more individuals with each type of lesion in the EN (though not all were significant; see Figure 4.2). Caries was the only exception to this trend; it was more common in the LN–EBA. Five and a half percent of observable individuals from the EN had lesions, compared to 12.1% of observable individuals from the LN–EBA.

The largest disparities between the proportions of lesions in each period came from COPH (61.9% vs. 16.3%), and URI (70.7% vs. 40.4%), periodontitis (72.4% vs. 45.5%), and periostitis (80.0% vs. 63.5%), in each case the condition being significantly more common among EN individuals (see Table 4.2). This general trend of higher lesion incidence in the EN held true for all other pathological conditions except caries. The difference between the number of caries in each period was, itself, not significant (p-value=0.3869). This was not an artefact of its sample size, as it was comparable to those of the other lesions tested (n=124).

4.1.3.0. Incidence by Cemetery

Second, the data were divided by cemetery: Shamanka II, Lokomotiv, and Ust'-Ida I. From the Shamanka II cemetery, 151 individuals were studied from Lokomotiv, 32; and from Ust'-Ida I, 67. As explained in Chapter 2, the 32 individuals from Lokomotiv represented only a third of those individuals available to study.

The general trend in the data was for there to be a higher incidence of lesions in the EN cemeteries of Shamanka II and Lokomotiv than in the LN–EBA cemetery of Ust'-Ida I (see Table 4.3 and Figure 4.3). There were more periodontal lesions in Shamanka II cemetery than any other; more periosteal, COPH, and upper respiratory infections in Lokomotiv; and more carious lesions in Ust'-Ida I. There was an equal amount (100.0%) of otitis lesions in both EN cemeteries. The trend for caries was once again contrary to the general trend.

The binomial tests (see Table 4.3) showed that there are significant, or almost significant, differences in the proportions of periosteal and COPH lesions among the three cemeteries. The difference between the number of COPH lesions in the EN cemeteries and the LN–EBA

Table 4.2: Summary of lesion incidence in each pathological category (all individuals pooled by time periods) and results (p-values) listed for each binomial test.

	Incidence, X(n)		P-Values
Lesion Type	**EN**	**LN–EBA**	**EN vs LN–EBA**
Periostitis	136 (170)	33 (52)	*0.02369*
Osteomyelitis	1 (139)	0 (33)	1
COPH	73 (118)	7 (43)	7.806^{-7}
Caries	5 (91)	4 (33)	0.3869
Periodontitis	76 (105)	15 (33)	*0.008376*
Upper Respiratory Infection	70 (99)	19 (47)	*0.0008917*
Otitis	114 (114)	47 (48)	0.6545
Auditory Exostoses	2 (122)	0 (47)	0.9289
Trachoma	0 (121)	0 (45)	NA

Significant and nearly significant tests are highlighted in grey, and significant tests are italicized; X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis.

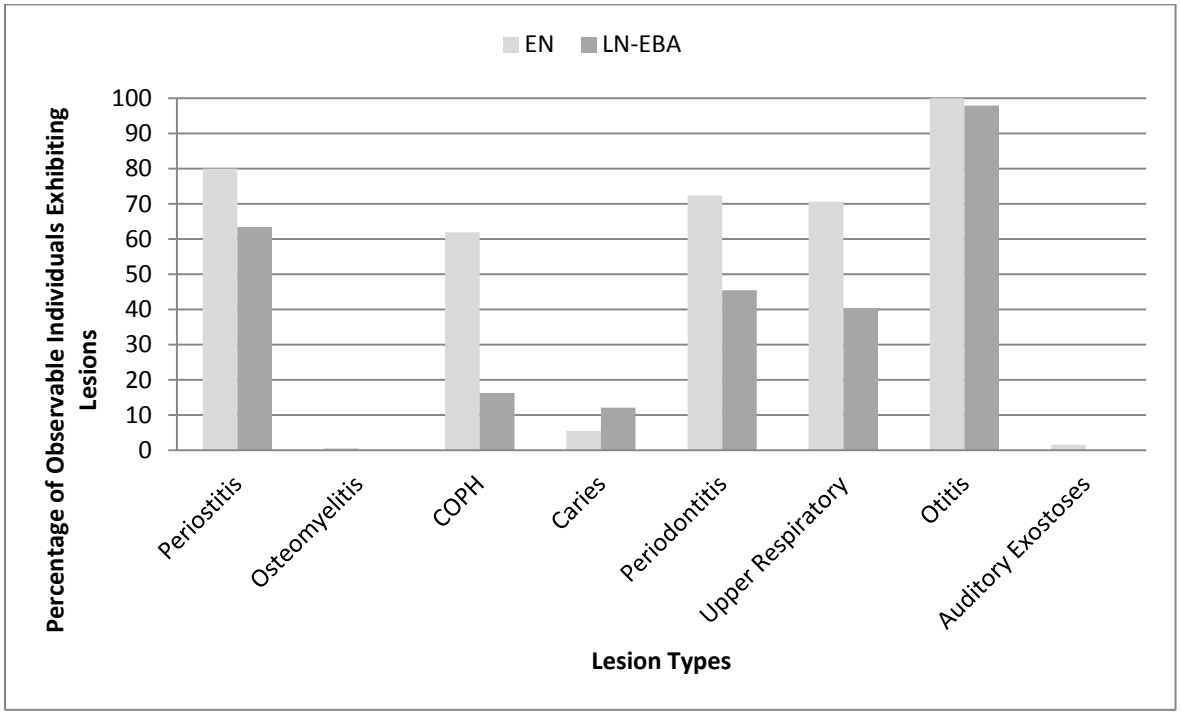

Figure 4.2: Lesion incidence by time period.

Table 4.3: Summary of lesion incidence in each pathological category (all individuals pooled by cemetery) and results (p-values) listed for each binomial test.

	Incidence, X(n)			P-Values		
Lesion Type	**SHA**	**LOK**	**UID**	**SHA vs LOK**	**SHA vs UID**	**LOK vs UID**
Periostitis	110 (139)	26 (31)	33 (52)	0.07281	*0.04178*	0.08298
Osteomyelitis	1 (112)	0 (27)	0 (33)	1	1	NA
COPH	53 (93)	20 (25)	7 (43)	0.06135	2.042^{-5}	8.608^{-7}
Caries	5 (69)	0 (22)	4 (33)	0.4463	0.6607	0.2437
Periodontitis	62 (82)	14 (23)	15 (33)	0.2571	*0.003844*	0.3876
URI	51 (77)	19 (22)	19 (47)	0.1178	*0.0008917*	*0.0009151*
Otitis	88 (88)	26 (26)	47 (48)	NA	0.7574	1
Auditory Exostoses	2 (97)	0 (25)	0 (47)	1	0.8165	NA
Trachoma	0 (95)	0 (26)	0 (45)	NA	NA	NA

Significant and nearly significant tests are highlighted in grey (sub-populations that are involved in significant and nearly significant tests more than once are highlighted in dark grey), and significant tests are italicized; X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis; URI is Upper Respiratory Infection; SHA is Shamanka II; LOK is Lokomotiv; UID is Ust'-Ida I.

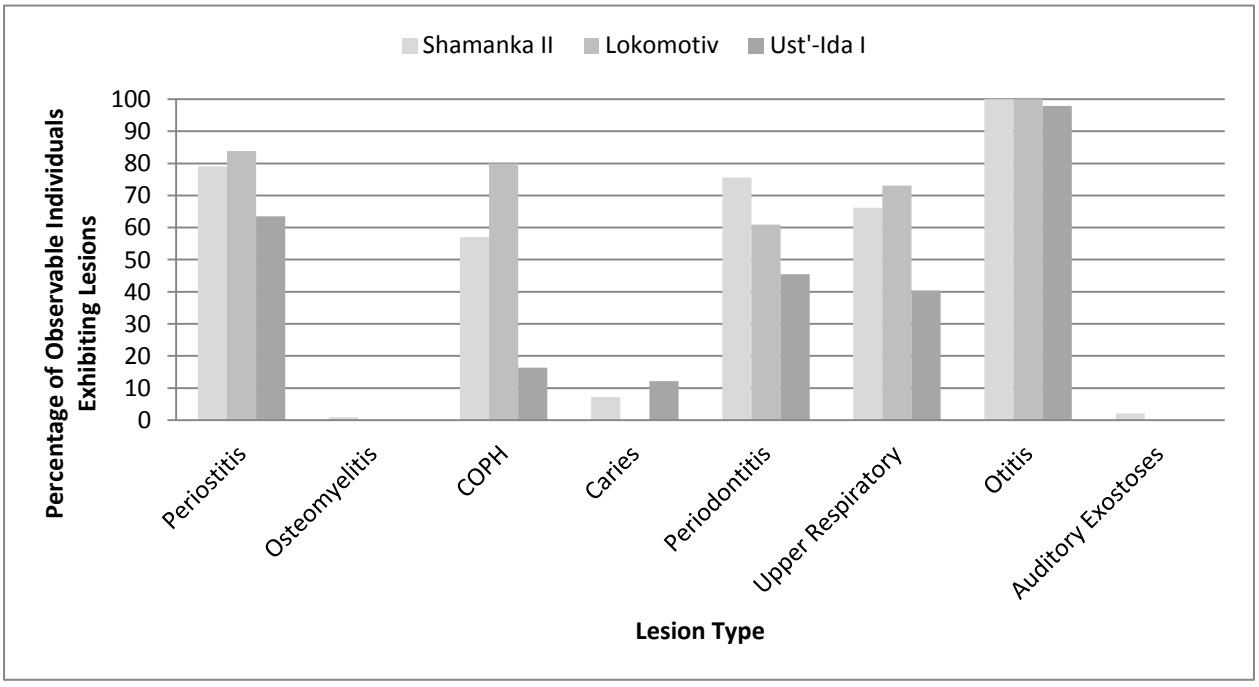

Figure 4.3: Lesion incidence by cemetery.

cemetery was highly significant (p-value=≤0.009). For periodontitis, there were significantly more lesions in the EN cemetery of Shamanka II than in the LN–EBA cemetery, Ust'-Ida I. For upper respiratory infection, there were significantly more lesions in the EN cemetery or Lokomotiv and nearly significantly more in the EN cemetery of Shamanka II compared to the LN–EBA cemetery of Ust'-Ida I. As for caries, it was clear that all those from the EN came from Shamanka II.

4.1.4.0. Incidence by Sex

Third, the data were divided by sex: male and female. There were 101 males and 61 females recorded in the sample (see Table 4.4 and Figure 4.4). Proportionally, there was a higher incidence of lesions in males, save for upper respiratory infection-induced lesions and auditory exostoses, than in females. It was interesting to note that all of the cases of caries occurred in males and that all the cases of auditory exostoses occurred in females.

The difference between the proportions of periosteal and COPH lesions on males and females was nearly significant and significant, respectively (see Table 4.4 and Figure 4.4). Both these lesion types followed the general pattern in the sample. The difference between the proportions of upper respiratory infection-induced lesions and auditory exostoses (exceptions to the pattern) in males and females were not statistically significant.

4.1.4.1. Incidence by Sex and Time Period

Fourth, the data were divided by sex and time period: En males (n=82), LN–EBA males (n=19), EN females (n=49), and LN–EBA females (n=12; see Table 4.5 and Figure 4.5). It was noted that males had a higher proportion of lesions than did females during both the EN and the LN–EBA, with the exception of auditory exostoses, upper respiratory infection, and otitis. Auditory exostoses were only present in EN females, upper respiratory infections occurred proportionally more in EN females than in LN–EBA males, and otitis occurred equally in EN males and females (100%). None of these tests were significant, however.

The proportions of periosteal lesions in EN males versus in LN–EBA males and of COPH in EN males versus in EN females were significantly different. These tests supported the general trend in the data for the EN individuals to have proportionatly more lesions than LN–EBA individuals, and males to have proportionally more lesions than females, respectively. Neither of the exceptions to this trend (upper respiratory infection and otitis in the EN) were significant. These exceptions, then, did not affect our understanding of the general trend in the data, but they

were of interest themselves for the information that they lent to our understanding of upper respiratory infection and COPH, in particular.

Table 4.4: Summary of lesion incidence in each pathological category (all individuals pooled by sex) and p-values listed for each binomial test.

	Incidence, X(n)		P-Values
Lesion Type	**Males**	**Females**	**Males vs Females**
Periostitis	87 (99)	41 (55)	0.05849
Osteomyelitis	0 (87)	0 (38)	NA
COPH	48 (68)	18 (37)	*0.0443*
Caries	6 (62)	0 (31)	0.1792
Periodontitis	61 (72)	27 (38)	0.146
Upper Respiratory Infection	42 (55)	23 (27)	0.5246
Otitis	63 (63)	30 (31)	0.7159
Auditory Exostoses	0 (69)	1 (41)	0.7915
Trachoma	0 (69)	0 (38)	NA

Significant and nearly significant tests are highlighted in grey, and significant tests are italicized; X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis.

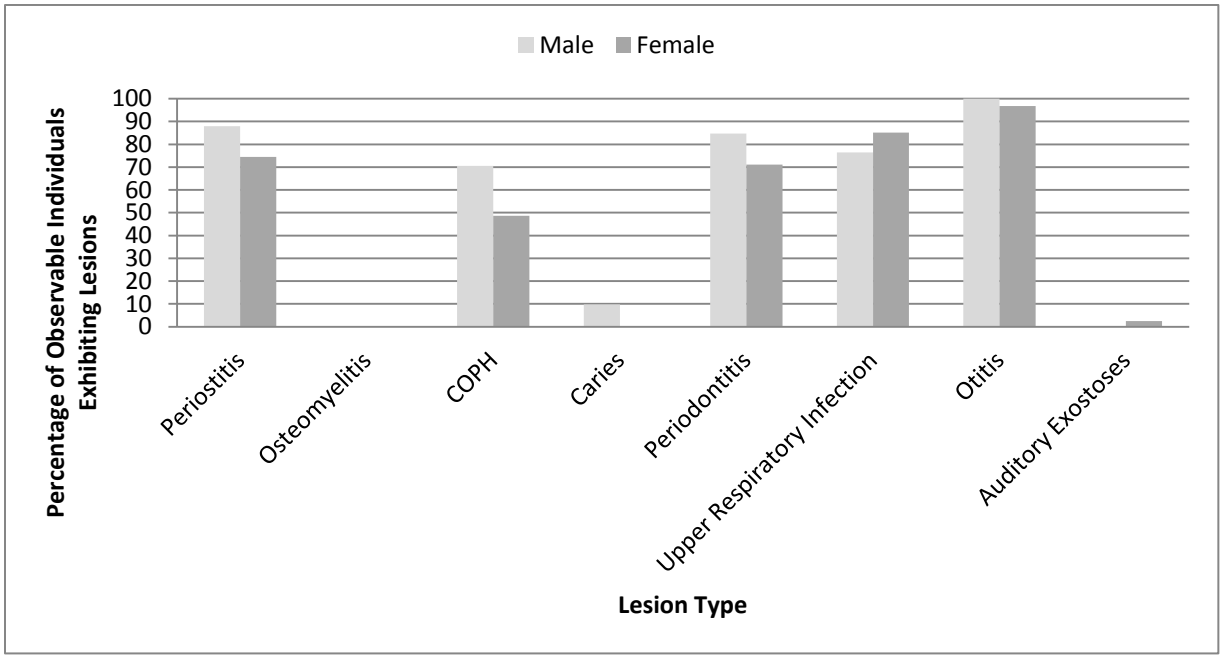

Figure 4.4: Lesion incidence by sex.

Table 4.5: Summary of lesion incidence in each pathological category (all individuals pooled by sex and time period) and results (p-values) listed for each binomial test.

	Incidence, X(n)				P-Values			
	Males		Females		Males vs Females		EN vs LN–EBA	
Lesion Type	EN	LN–EBA	EN	LN–EBA	EN	LN–EBA	M	F
Periostitis	74 (81)	13 (18)	36 (46)	5 (9)	1	0.665	0.06419	0.3117
Osteomyelitis	0 (75)	0 (12)	0 (35)	0 (3)	NA	NA	NA	NA
COPH	44 (59)	4 (9)	17 (32)	1 (5)	0.06504	0.17394	0.1456	0.3697
Caries	4 (53)	2 (9)	0 (26)	0 (5)	0.3726	0.7327	0.443	NA
Periodontitis	52 (61)	9 (11)	23 (33)	4 (5)	0.1278	1	1	1
URI	37 (48)	5 (7)	21 (24)	2 (3)	0.4611	1	1	0.9237
Otitis	53 (53)	10 (10)	25 (25)	5 (6)	NA	0.7897	NA	0.4304
AE	0 (59)	0 (10)	1 (36)	0 (5)	0.8019	NA	NA	1
Trachoma	0 (59)	0 (10)	0 (33)	0 (5)	NA	NA	NA	NA

Significant and nearly significant tests are highlighted in grey, and significant tests are italicized; X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis; URI is Upper Respiratory Infection; AE is Auditory Exostoses.

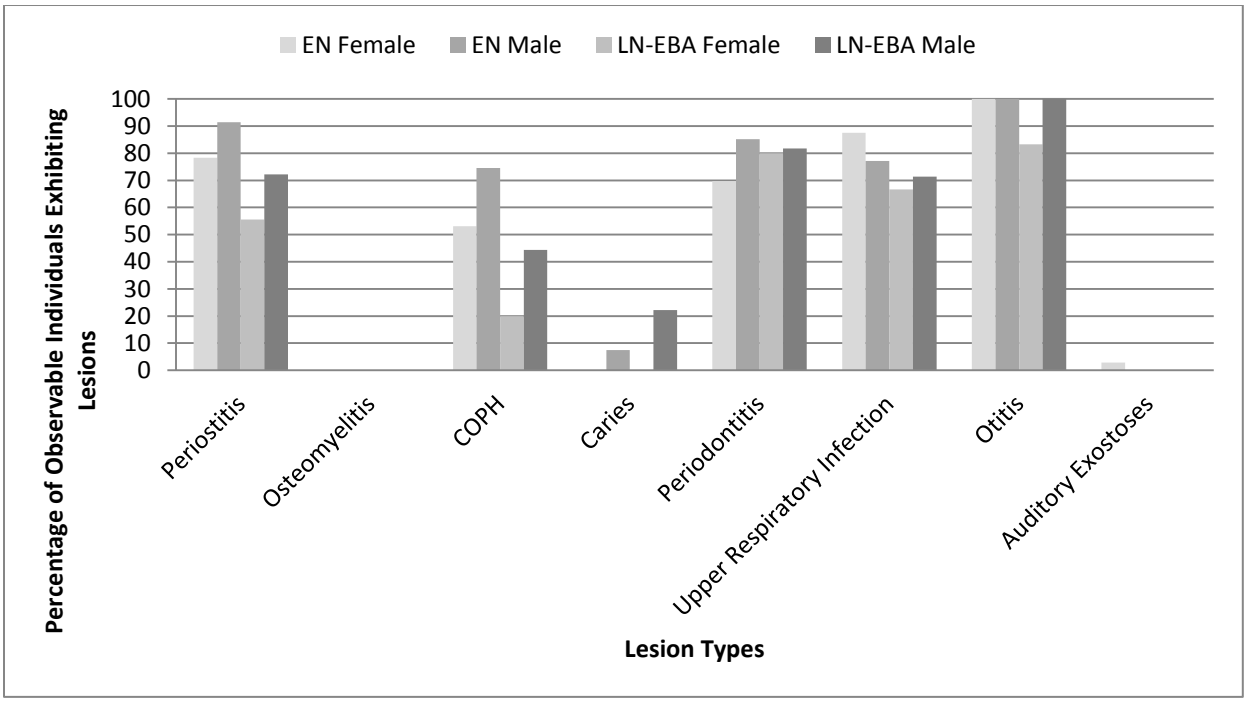

Figure 4.5: Lesion incidence by sex and time period.

4.1.5.0. Incidence by Age Group

Fifth, the data were divided by age group: <4 years (n=20), 5–19 years (n=25), 20–49 years (n=98), and +50 years (n=7). There was one individual whose age could not be determined (see Table 4.6 and Figure 4.6). In general, the proportion of individuals with each lesion type increased with age until the 20–49 year age group, after which point it decreased again.

Caries, periodontitis, and otitis did not follow this pattern, however. Caries incidence increased with age, but the proportion of individuals 20–49 years old with caries was low compared to individuals 5–19 or 50+ years old. The proportion of individuals with periodontitis did not drop off after 49 years old, but, rather, it increases. Finally, with the exception of individuals 20–49 years old, 100% of those observable for otitis exhibited indicative lesions. None of these exceptions varied drastically from the general trend in the data and, infact, are all consistent with increasing lesion incidence with advancing age.

Eight tests examining the difference in the proportion of individuals with lesions of each type by age group were significant (see Table 4.6). Three involved the proportion of periostitis

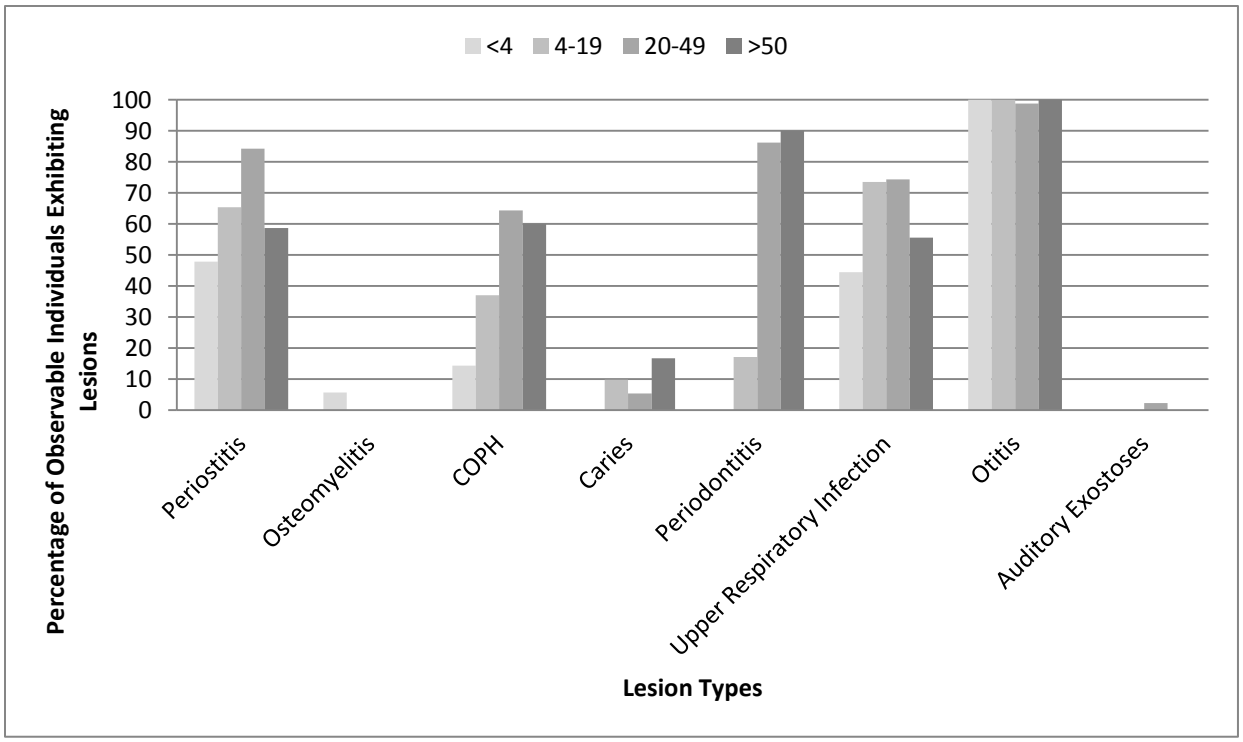

Figure 4.6: Lesion incidence by age group (in years at death).

Table 4.6: Summary of lesion incidence in each pathological category (all individuals pooled age group, indicated in years at death) and results (p-values) listed for each binomial test.

	Incidence, X(n)			
Lesion Type	**<4 y.o.**	**5–19 y.o.**	**20–49 y.o.**	**+50 y.o.**
Periostitis	11 (23)	34 (52)	112 (133)	12 (14)
Osteomyelitis	1 (18)	0 (39)	0 (103)	0 (12)
COPH	3 (21)	17 (46)	54 (84)	6 (10)
Caries	0 (1)	4 (41)	4 (76)	1 (6)
Periodontitis	0 (0)	7 (41)	75 (87)	9 (10)
URI	4 (9)	25 (34)	55 (74)	5 (9)
Otitis	23 (23)	45 (45)	84 (85)	9 (9)
Auditory Exostoses	0 (24)	0 (46)	2 (89)	0 (10)
Trachoma	0 (24)	0 (47)	0 (85)	0 (10)

Significant and nearly significant tests are highlighted in grey (sub-populations that are involved in significant and nearly significant tests more than once are highlighted in dark grey), and significant tests are italicized; X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis; URI is Upper Respiratory Infection; y.o. is years old.

	P-Values					
	<4 y.o. vs			**5–19 y.o. vs**		**20–49 y.o. vs**
Lesion Type	**5–19 y.o.**	**20–49 y.o.**	**+50 y.o.**	**20–49 y.o.**	**+50 y.o.**	**+50 y.o.**
Periostitis	0.2397	*0.00024*	*0.03733*	*0.00875*	0.2636	1
Osteomyelitis	0.6893	0.3216	1	NA	NA	NA
COPH	0.1111	*0.00011*	*0.02795*	*0.00498*	0.3232	1
Caries	1	1	1	0.5928	1	0.8121
Periodontitis	1	1	1	1.26^{-13}	4.578^{-5}	1
URI	0.2092	0.1395	1	1	0.5248	0.4275
Otitis	NA	1	NA	1	NA	1
Auditory Exostoses	NA	1	NA	0.785	NA	1
Trachoma	NA	NA	NA	NA	NA	NA

(<4 years versus 20–19 years and versus +50 years; and 5–19 years versus 20–49 years), three involved the proportion of COPH (<4 years versus 20–19 years and versus +50 years; and 5–19 years versus 20–49 years), and two involved the proportion of periodontitis (5–19 years versus 20–49 years and versus +50 years). Of these significant tests, the three involving periostitis and COPH were testing the same age groups. What these tests show is that there was a significant difference in the proportions of individuals exhibiting these lesions from the <4 year and 5–19 year to the >20 year age groups. In other words, these lesions increased in frequency significantly between subadult and adulthood. Between the 20–49 year and +50 year age groups, however, there was no significant difference in the proportion of lesions. Caries and otitis, however, remained exceptions to the general trend.

4.1.5.1. Incidence by Age Group by Time Period

Sixth, the data were divided by age group and time period: EN and LN–EBA <4 years, EN and LN–EBA 5–19 years, EN and LN–EBA 20–49 years, and EN and LN–EBA +50 years (see Table 4.7 and Figure 4.7). Even when divided by time period, the proportions of lesions present in the sample still increased by age. The only exceptions to this trend arose from the proportion of otitis-induced lesions in the LN–EBA, and auditory exostoses and osteomyelitis in the EN. Otitis was an exception to the trend once more because it was present in all sub-populations 100% of the time with the exception of in LN–EBA 20–49 year olds, where 90.9% of the people exhibited these lesions. Auditory exostoses and osteomyelitis were exceptions because only one sub-population exhibited each kind of lesion: EN 20–49 year olds in the case of auditory exostoses and EN 4–19 year olds in the case of osteomyelitis.

In general, there were also more individuals with lesions in the EN than in the LN–EBA. This was a trend in the data that appeared when the data were divided only by time period (see above) and this held true here. Exceptions to this trend occured in the younger age groups for periostitis, caries, periodontitis, and upper respiratory infection. Of the binomial tests, two were significant (see Table 4.7): those testing the difference between the proportions of EN and LN–EBA 5–19 year olds with periostitis and 20–49 year olds with COPH. The proportions of individuals exhibiting these lesions increased with age, but these proportions were significantly smaller in the LN–EBA populations than in the EN populations.

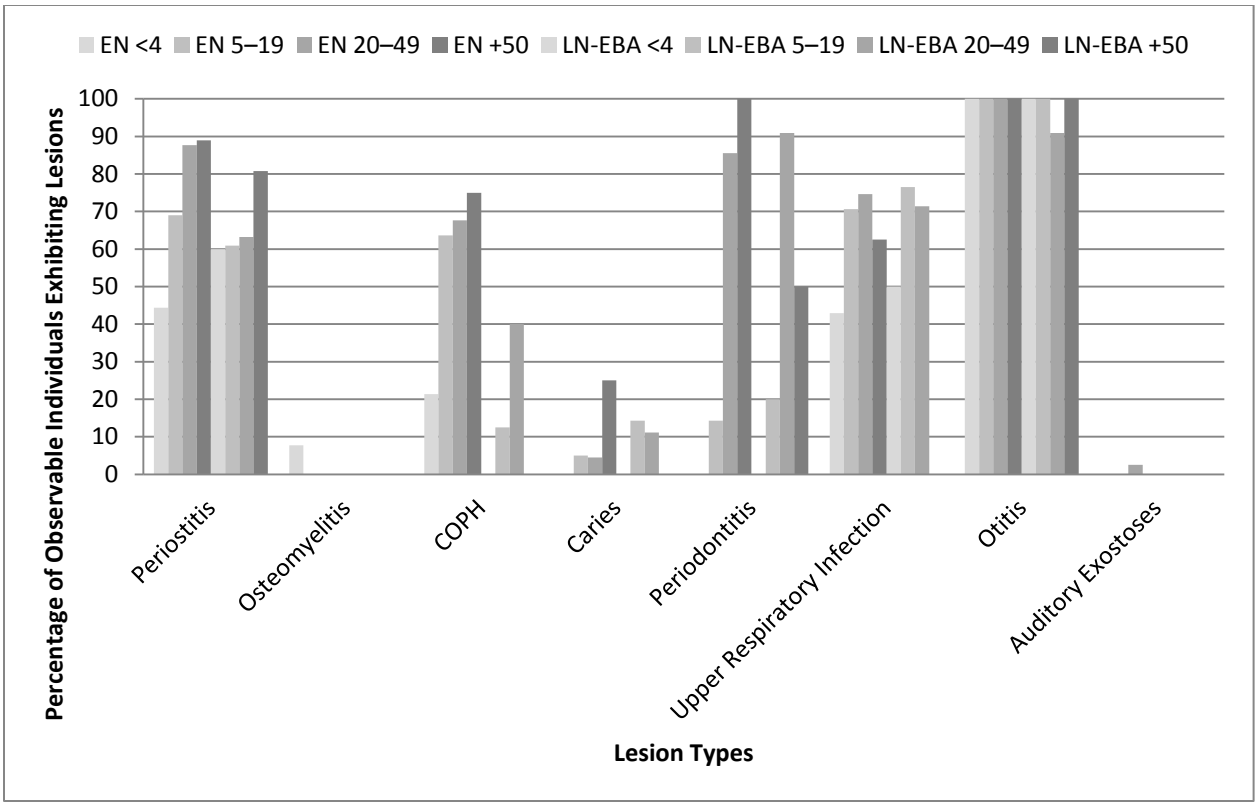

Figure 4.7: Lesion incidence by age group (in years at death) and time period.

4.2.0.0. Chi-Square Tests: Co-occurring Lesions

Table 4.8 presents the results for the chi-square tests for co-occurrence (see Appendix A for the raw data). The null hypothesis was rejected when testing for the following co-occurring lesions: periostitis and COPH, periostitis and periodontitis, periostitis and upper respiratory infections, COPH and upper respiratory infections, and COPH and otitis. For these tests, the lesions being tested co-occurred more often than would have been expected though chance alone. The test statistic was 3.841459 for each test and each test resulted in one degree of freedom. Three tests involving otitis could not be performed because two or more of the cells in the contingency table contained "0" (i.e., zero individuals were recorded as absent for both otitis and periostitis, and zero individuals were recorded as absent for otitis and present for periostitis). Tests such as these provided meaningless results and, therefore, were calculated as "not applicable" by the stats software, R.

Those lesions that co-occurred with other lesions the most were periostitis (three co-occurrences), COPH and upper respiratory infection-induced lesions (each with two significant

Table 4.7: Summary of lesion incidence in each pathological category (all individuals pooled by age group, indicated in years at death and time period) and results (p-values) listed for each binomial test (EN data vs LN–EBA data).

	Incidence, X(n)							
	EN				LN–EBA			
Lesion Type	**<4 y.o.**	**5–19 y.o.**	**20–49 y.o.**	**+50 y.o.**	**<4 y.o.**	**5–19 y.o.**	**20–49 y.o.**	**+50 y.o.**
Periostitis	8 (18)	20 (29)	100 (114)	8 (9)	3 (5)	14 (23)	12 (19)	4 (5)
Osteomyelitis	1 (13)	0 (24)	0 (93)	0 (9)	0 (5)	0 (15)	0 (10)	0 (3)
COPH	3 (14)	14 (22)	50 (74)	6 (8)	0 (7)	3 (24)	4 (10)	0 (2)
Caries	0 (0)	1 (20)	3 (66)	1 (4)	0 (1)	3 (21)	1 (9)	0 (2)
Periodontitis	0 (0)	3 (21)	65 (76)	8 (8)	0 (0)	4 (20)	10 (11)	1 (2)
URI	3 (9)	12 (17)	50 (67)	5 (8)	1 (2)	13 (17)	5 (7)	0 (1)
Otitis	14 (14)	19 (19)	74 (74)	7 (7)	9 (9)	26 (26)	10 (11)	2 (2)
Auditory Exostoses	0 (15)	0 (20)	2 (79)	0 (8)	0 (9)	0 (26)	0 (10)	0 (2)
Trachoma	0 (16)	0 (22)	0 (75)	0 (8)	0 (8)	0 (25)	0 (10)	0 (2)

	P-Values			
Lesion Type	**<4 y.o.**	**5–19 y.o.**	**20–49 y.o.**	**+50 y.o.**
Periostitis	0.9124	0.752	*0.01738*	1
Osteomyelitis	1	NA	NA	NA
COPH	0.5083	*0.001025*	0.1751	0.2586
Caries	NA	0.6347	0.9748	1
Periodontitis	NA	0.9435	0.9871	0.4292
URI	1	1	1	0.9056
Otitis	NA	NA	0.2667	NA
Auditory Exostoses	NA	NA	1	NA
Trachoma	NA	NA	NA	NA

Significant and nearly significant tests are highlighted in grey, and significant tests are italicized; X is the number of individuals affected; n is the total number of individuals observed; COPH is Cribra Orbitalia and Porotic Hyperostosis; URI is Upper Respiratory Infection.

co-occurrences and one nearly significant co-occurrence), periodontitis (one significant co-occurrence and one nearly significant co-occurrence), and otitis (one significant co-occurrence and one nearly significant co-occurrence). When the significance requirement was refined to 99.99995% (or α=0.00005), then I was left with the two most significant co-occurrences: COPH with each periostitis and otitis.

Table 4.8: Results of Chi-square tests.

Co-occurring Lesions	TS	P-Value
Periostitis and COPH	*19.6022*	*9.536^{-6}*
Periostitis and Caries	0.003	0.9565
Periostitis and Periodontitis	*6.8336*	*0.008946*
Periostitis and Upper Respiratory	*5.042*	*0.02474*
Periostitis and Otitis	NA	NA
COPH and Caries	0.0415	0.8386
COPH and Periodontitis	2.9457	0.08611
COPH and Upper Respiratory	*9.8215*	*0.001725*
COPH and Otitis	*80.00*	*$<2.2^{-16}$*
Caries and Periodontitis	0.6276	0.4286
Caries and Upper Respiratory	0.0698	0.7916
Caries and Otitis	NA	NA
Periodontitis and Upper Respiratory	0.9967	0.3181
Periodontitis and Otitis	NA	NA
Upper Respiratory and Otitis	3.4312	0.06398

Significant and nearly significant tests are highlighted in grey, and significant tests are italicized; TS is test statistic; COPH is Cribra Orbitalia and Porotic Hyperostosis; NA refers to those tests where otitis occurred 100% of the time along with the other lesion making it unnecessary to test their co-occurrence.

Of those lesions that co-occurred highly significantly, periostitis and COPH were regularly significant in the binomial tests and the dispersion of these lesions consistently followed the trends in the data. In other words, these lesions were often exhibited on individuals from the same sub-populations: EN males between the ages of 20 and 49 years old who were buried in Lokomotiv (i.e., they co-occurred). Binomial tests involving periodontitis were also regularly significant, but these lesions occurred more commonly on EN individuals over 50 years old who were buried in Shamanka II. This variation to the general trend explained the less-significant co-occurrence of periodontitis and periostitis.

The co-occurrence of some of these lesions may be an artifact of their frequent occurrence within the sample in general. Otitis-induced lesions, for example, were present in nearly everyone (99.4% of the sample). This was also true, though to a lesser extent, for periostitis (76.1% of the sample), upper respiratory infection-induced lesions (70.6% of the sample), and periodontitis (65.9% of the sample), all of which occurred in the majority of the sample.

4.3.0.0. Logistic Regressions: the Effect of the Sub-Populations

Generalized linear model logistic regressions were run in order to help answer the second statistical question: which sub-populations are predictive of a positive response variable. Each test created a model that assessed the significance of the sub-populations (e.g., male and female) as predictors of the presence of a response variable (e.g., periostitis). Those tests that resulted in significant sub-populations rejected the null hypothesis and accepted the alternate hypothesis: the presence of a lesion is determined by the sub-population(s) from which an individual came. The models were created using only the *diagnostic* sub-populations. Non-diagnostic sub-populations, such as "unidentifiable sex" and "unidentifiable age," were deleted from the dataframe before the tests were run. The meaning of these tests in the context of the Cis-Baikal will be discussed in Chapter 5. Significance was assessed at 95% (α=0.05). Summarized results are included in Table 4.9 below, while full results tables can be found in Appendix B.

Most of the trends seen in the binomial tests remained true here; males, those aged 20–49 years, and those from the EN were significant, or in this case, contributed the most variance to the data. In the entire data set, Shamanka II and Ust'-Ida I were never significant if the intercept (Lokomotiv) was not. Interestingly, while Lokomotiv contained higher lesion incidence in general than did Shamanka II, the data from Shamanka II contained more variation than did those from its EN partner. Like the data from Lokomotiv, those from Ust'-Ida I/the LN–EBA were highly homogeneous. Only the periodontitis data contributed variation to the Ust'-Ida I data. In other words, there was very little variability *within* Lokomotiv and Ust'-Ida I, making these cemeteries, themselves, predictive of the presence of the lesions (i.e., if an individual was from Lokomotiv or Ust'-Ida I, the probability of having a lesion was high or low, respectively, regardless of your sex or age).

Every lesion type formed at least one significant model with the exception of osteomyelitis, which only formed models that were not significant. Many of these significant models remained significant in the EN cemeteries. In contrast, no models (with the exception of two models involving periodontitis) remained significant in the LN–EBA. This, too, is consistent with the results of the binomial tests, in that it is the EN cemeteries that contributed the most variance to the data.

4.4.0.0. Summary and Answering the Research Question

The binomial tests support all but one of the trends visible in the data. There were comparatively more infection-induced lesions in the EN than in the LN–EBA, and in males than in females both in the entire sample population, as well as in the EN and LN–EBA separately. There were proportionally more infection-induced lesions in those aged 20–49 years than in any other age group (or, more broadly, lesion incidence increased with age up to 49 years old), and this holds true in the EN and the LN–EBA. And, there were comparatively more infection-induced lesions in the EN cemeteries of Shamanka II and Lokomotiv than there were in the LN–EBA cemetery of Ust'-Ida I. More specifically, there were proportionally generally more individuals with infection-induced lesions in Lokomotiv than Shamanka II. There were some exceptions to these trends, the most consistent of which came from the data involving caries. Finally, the binomial tests did not support the trend that there were comparatively more lesions in individuals under the age of 50 in the LN–EBA than in the EN (this was a large age range and sample sizes varied depending on the type of lesion being examined). In spite of this, four lesion types follow this trend, making it worth noting.

Every lesion present in the middle Holocene sample co-occurred significantly with at least one other lesion type, with the exception of caries, which did not co-occur. Otitis; COPH, upper respiratory infection and periostitis; and otitis and periodontitis co-occurred with other lesions the most, in descending order. Otitis co-occurred with every other lesion, making this relationship impossible to test statistically. Of these co-occurrences, those between COPH and periostitis, and COPH and otitis were highly significant (α=0.00005). These relationships form a network as illustrated in Figure 4.8.

In light of these results, we can answer the first statistical question in the affirmative. The number of individuals with infection-induced lesions *is* significantly different across populations and sub-groups. The binomial tests illustrated the changes that occured in the

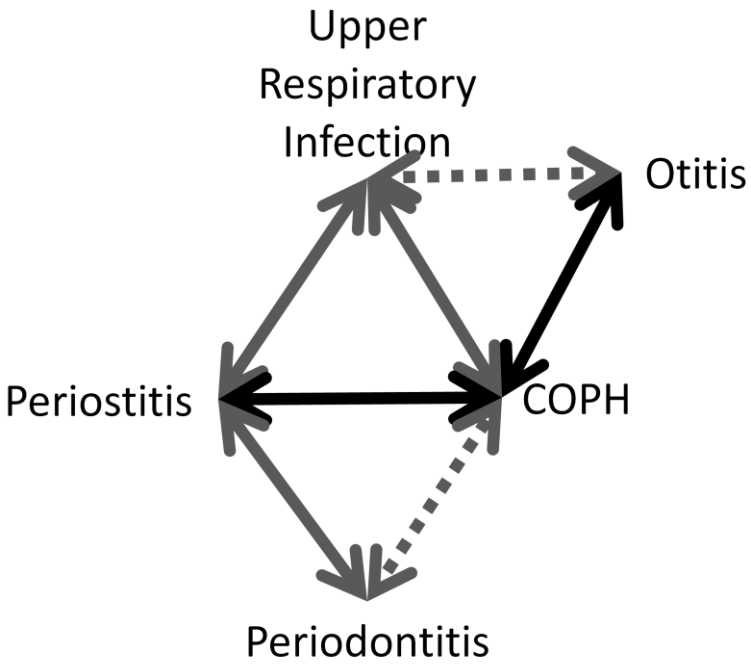

Figure 4.8: An illustration of the co-occurring lesions in the sample. Significant co-occurrences are illustrated by solid lines and nearly significant co-occurrences are illustrated by dashed lines; highly significant co-occurrences are illustrated by black lines.

incidence of lesions across the sub-populations and the chi-square tests illustrated the relationships between the lesions themselves (i.e., co-occurrence) in-light of these patterns.

The logistic regression tests highlighted the variability within the entire sample, the time periods, and the cemeteries, and showed which sub-populations were predictive of positive response variables in the data. These tests revealed that the EN period contributes much of the variance to the entire data set, with the variance therein coming mostly from Shamanka II. In Shamanka II, the youngest three age groups were generally predictive of the presence of a positive response variable, and one or both sexes were predictive of the presence of periodontitis and auditory exostoses. Both Lokomotiv and Ust'-Ida I were generally themselves predictive of a positive response variable, with very little variance coming from said data sets. In Lokomotiv,

the variance came from the intercept (or female) in the sex and COPH test, while in Ust'-Ida I, the variance came from the intercept (or <4 year old) and 20–49 year old age groups in the age and periodontitis test.

In answer to the second statistical question, at some point, every sub-population was predictive of a positive response variable in the sample. The extent to which a sub-population was predictive depended on the type of lesion being considered and the time period and cemetery that the individual was from. Age and sex had more of a bearing on an individual's probability of having a lesion in the EN and at Shamanka II, while the sub-populations had less of an effect in the LN–EBA/Ust'-Ida I and at Lokomotiv.

The logistic regression tests highlighted which sub-populations contributed significantly to the variance *within* a sample, while the binomial tests *compared* two samples and highlighted which sub-populations had a higher incidence of a lesion. The only time the results of the two tests overlapped are when the binomial tests compared sub-populations within the entire sample, and the sexes within the EN and the LN–EBA. Those sub-populations that were found to contain significantly higher incidence of infection-induced lesions by the binomial tests were also found to be predictive of the presence of a positive response variable by the logistic regressions (with one exception, which will be discussed below). In other words, those sub-populations that had lesions that occured significantly more within them were also predictive of the presence of said lesion. Intuitively, this makes sense. Those sub-populations with higher lesion incidence would lend a lot of the variability to the model as a function of being so different (here "different" in terms of being the level with the highest proportion of lesions). As a result, they were predictive of the presence of said lesion.

One exception existed. In the binomial tests, there were significantly more periodontal lesions in individuals from Shamanka II than those from Ust'-Ida I, but Shamanka II was not a significant sub-population in the logistic regression test for periodontitis and cemetery. That the binomial tests found no significant difference between the proportion of individuals with periosteal lesions in Shamanka II and Lokomotiv can explain this exception. While there were comparatively more individuals in Shamanka II than in Ust'-Ida I with periodontal lesions, there were not so many more as to make Shamanka II predictive, as proven by there being a statistically similar amount of individuals with periodontal lesions in Lokomotiv.

Two other incidences are worth noting. The logistic regressions highlighted males as being predictive of periodontal lesions and 20–49 year olds as being predictive of upper respiratory infection-induced lesions, but the binomial tests did not highlight these sub-populations as having proportionally more individuals with these lesions than others. Figures 4.4 and 4.6, however, demonstrate that these sub-populations follow the trends seen in the data. Therefore, even though these sub-populations were not found to be significant in the binomial tests, the logistic regression tests were sensitive enough to highlight their effects within the data.

This increased sensitivity, along with the different focus of the logistic regression tests, can also explain the presence of significant intercepts for logistic regression tests involving lesion types (caries, otitis, and auditory exostoses) that were never found as having proportionally more individuals with lesions in the binomial tests. Logistic regression tests are more sensitive to differences *within* samples and the differences between their findings and those of the binomial tests can be explained by this. There was variation within the entire sample with regard to the presence of caries, otitis, and auditory exostoses but, compared to the variation found in other lesion types, it was minor.

In light of these two answers, I can answer the research question in the affirmative. The incidence of infection-induced lesions within the sample *is* related to the type of infection, the sex and age of the individual, the cemetery the individual came from, and the time period the individual lived in. Each of these factors affected an individual's probability that he/she would exhibit a non-specific infection-induced lesion. The effects of these factors varied among sub-populations, with some having very little variability within them. In general, it was males aged 20–49 years, from the EN cemetery of Shamanka II who had the highest probability of having non-specific infection-induced lesions on their skeletons. The lesions most commonly observed in the sample were otitis, periostitis, upper respiratory infection, periodontitis, and COPH.

Chapter 5
Discussion

5.0.0. Discussion

The presence of non-specific infection-induced lesions in the sample broadly mirrors the summarized population data concerning stress in the middle Holocene Cis-Baikal. Here I discuss the similarities and the differences between the patterns of physiological stress, as published in the literature (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011), and non-specific infection in the sample, and I make some general inferences regarding community health and activities of these individuals. First, I will discuss each lesion type individually (its potential etiological mechanisms, its relation to the physiological stress data, *etc.*). Certain lesions types—specifically osteomyelitis and trachoma—will not be discussed here because they were not observed or were only observed once or twice in the sample. Caries and auditory exostoses will be discussed along with periodontitis and otitis, respectively. Suffice to say that the absence, or near absence, of many of these lesions was neither unusual for the geographic location that these hunter-fisher-gatherers lived in or the genetic populations themselves (Aufderheide and Rodríguez-Martín 1998: 251; Berry 1975; Emerson et al. 1999; Godde 2010; Hrdlička 1935; Hutchinson et al. 1997; Okumura et al. 2007; Özbek 2012; Webb 1990), nor for the presence of other associated lesions (discussed more below). Second, I will discuss the presence of the infection-induced lesions in the sample as a whole and its relationship to the physiological stress data in general.

5.1.0. Non-Specific Infection and Physiological Stress in the Sample

The species of pathogen behind a non-specific infection-induced lesion cannot, by definition, be known (see Section 2.0.0.0.), but general discussions concerning the risk factors for these lesions can communicate much concerning a population's past lifeways. The following is a

discussion concerning the presence of five of the lesion types investigated here and their relationship(s) with the physiological stress data gathered by other Baikal-Hokkaido Archaeology Project members.

5.1.1. Periostitis

Periosteal lesions (i.e., those bony lesions that occur beneath the periosteum) were present in over three-quarters of the sample (76.1%). They occurred significantly more frequently in the EN Kitoi than in the LN–EBA Isakovo-Serovo-Glazkovo (or ISG); significantly more in Lokomotiv than in Shamanka II and Ust'-Ida I; significantly more in males than in females; and significantly more in those older than 20 years compared to those younger, regardless of time period. Of these sub-populations, the 20–49 year old age group was the most predictive sub-population regarding the presence of periostitis in Shamanka II. In both Lokomotiv and Ust'-Ida I, however, age and sex had less influence on the presence of a lesion. Finally, periostitis co-occurred with cribra orbitalia and porotic hyperostosis (or COPH), periodontitis, and upper respiratory infection.

To refresh, periostitis is caused by aggravating the periosteum. Aggravation can be caused by trauma, mechanical changes, the growth of a tumor, ulcer, or neoplasm, the formation of granulated tissue, or the altered flow of blood or exudate (Assis et al. 2011; Boel and Ortner 2011; Cunha et al. 2009; White and Folkens 2005: 42–3). While it is impossible to speculate on the etiology of the majority of these periosteal lesions, it is safe to assume that all of the periosteal lesions were not caused by a single mechanism. That said, I cannot rule out any one of these mechanisms as the cause of some of these lesions. The etiology of periostitis is non-specific.

Similarly, many of these mechanisms can be the result of inflammatory processes associated with physiological stress (Klaus 2014). I must consider that infections often occur in physiologically stressed or immunocompromised individuals and infection is, itself, a form of physiological stress (DeWitte and Bekvalac 2011). In fact, periostitis is more often the result of an infection than of other forms of stress (Steckel and Rose 2002; Ubelaker and Pap 1998).

While periostitis was considered to be the result of infection specifically in this study (see section 2.1.0.0.), this does not mean that other forms of physiological stress can be ruled out as the cause of some, if not many, of the lesions observed. In fact, the processes behind the formation of periosteal new bone are not understood well enough yet to definitively state

whether physiologically stressed individuals can form new bone or not (Weston 2008). While researchers such as Weston (2012) argue, for example, that new bone formation would not be a priority during a stress event and that systems more critical to maintaining homeostasis would be the focus, others (e.g., Ragsdale and Lehmer 2012) maintain that any form of inflammation associated with a physiological stress event can aggravate the periosteum and release enough chemicals to stimulate the formation of periosteal new bone. Many researchers have considered the presence of periosteal lesions to be a sign of a physiologically stressed population, even though the etiology of the lesions themselves are not always known, because they occur in populations of physiologically stressed individuals (Buckley 2000; Da-Gloria et al. 2011; DeWitte and Bekvalac 2011; Novak 2011). As a result, the lesions documented here may have been caused by any number of stimuli (infectious, stressful, traumatic, *etc.*). I will discuss those causes with an infectious etiology and will leave the other causes to future discussions.

That the Early Neolithic (or EN) Kitoi were more physiologically stressed than the Late Neolithic to Early Bronze Age (or LN–EBA) Isakovo-Serovo-Glazkovo (or ISG) has been suggested by previous Baikal-Hokkaido Archaeological Project (or BHAP) members (Lieverse et al. 2007a; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011). Higher rates of linear enamel hypoplasia (or LEH; Lieverse et al. 2007a; Link 1999; Waters-Rist 2011), shorter adult femoral length, lower body mass in late infancy and early childhood (Temple et al. 2014), as well as the death of breastfeeding infants in the EN (Waters-Rist et al. 2011), all suggest that the EN Kitoi underwent frequent and severe episodes of physiological stress. While the specific etiology behind the lesions cannot be known, the presence of periostitis supports the notion that the EN Kitoi were more physiologically stressed than were the LN–EBA ISG. Should some of the periosteal lesions have been caused by infections, then these infections would have served as another kind of stress for the individuals to weather.

The presence of significantly more periosteal lesions on individuals from Lokomotiv, on males, and on those older than 20 years suggests that these individuals may also have been more physiologically stressed than their counterparts. Periostitis also significantly co-occurred with COPH, periodontitis, and upper respiratory infection. The presence of multiple infection-induced lesions in the same individuals would imply a high rate of physiological stress for the individual. This, however, will be discussed more below.

5.1.2. Cribra Orbitalia and Porotic Hyperostosis (COPH)

COPH was visible on the bones of half of the individuals in the sample (49.7%). Of these individuals, significantly more were from the EN than from the LN–EBA. Of these EN individuals, significantly more of them were from the Lokomotiv cemetery than from Shamanka II. Other than periostitis, COPH is the only lesion type that is present significantly more in males than in females. This holds true in the EN and, though not significantly so, in the LN–EBA. COPH was visible on the skeletal remains of those aged 20–49 years significantly more often than those of any other age, and this remained true when the sample was divided by time period. The lesion appeared significantly more frequently on EN individuals between the ages of 5 and 19 years than on those of the same age from the LN–EBA. These patterns were reflected in the results of the logistic regressions. Here, the LN–EBA and Ust'-Ida I were predictive of the presence of COPH, while age was predictive of the presence of COPH in Shamanka II and sex was predictive of the presence of COPH in Lokomotiv. These patterns and results reflected common trends in the rest of the data. This was supported by the chi-square tests, which showed that COPH co-occurred with more other lesions than did any of the other lesions (periostitis, periodontitis, upper respiratory infection, and otitis).

Iron deficiency anemia is classically considered to be the cause of COPH lesions (El-Najjar et al. 1976; Stuart-Macadam 1989a, b; 1992: 151–6; Taylor 1985; Ubelaker 1992), but the mechanisms that lead to this deficiency are still hotly debated (El-Najjar et al. 1976; Stuart-Macadam 1989a, b; 1992: 151–6; Taylor 1985; Ubelaker 1992). These include blood loss (e.g., via trauma, menstruation, or parasitic infection), iron-deficient nutrition, or self-imposed anemia (via the immune response to an infection).

Megaloblastic anemia and sub-periosteal bleeding resulting from scurvy may also cause COPH lesions (Walker et al. 2009). Like iron-deficiency anemia, these conditions result from nutritional deficiencies: B_9 and B_{12} for megaloblastic anemia and vitamin C for scurvy (Walker et al. 2009). The primary source of B_9 and B_{12} is animal products (Ambroszkiewicz et al. 2006). We know from various studies (Katzenberg et al. 2010; Katzenberg and Weber 1999) that the Kitoi and ISG both relied on fish and mammal meat along with other plant-based foods, so an etiology related to a B_9 and B_{12} deficiency would appear unlikely. Furthermore, plants, along with raw meat, organ meats, and the stomach contents of herbavores, can provide sufficient vitamin C to satisfy an individual's nutritional requirements (Ambroszkiewicz et al. 2006; Walker et al. 2009). It remains possible, however, that the individuals included in the sample

cooked their meat (consequently denaturing the vitamin C) and/or ate few vitamin C-rich plants, at least seasonally. Regardless, because the focus of this thesis is non-specific infection rather than nutrition *per se*, the remainder of this section will focus on iron deficiency, which can have an infectious origin.

We cannot rule out any of these mechanisms or risk factors, but we can make some broad inferences based on the generally-accepted etiologies published in the clinical literature. A discussion on trauma, menstruation, and genetics is beyond the scope of this research, but suffice to say that they are all risk factors that could have exacerbated blood loss and/or anemia within the populations. Since infection and/or nutrition are more likely to account for these lesions than are other factors, the following discussion will focus on these mechanisms.

Seasonal resource scarcity is cited as being the possible cause of the annual stress episodes suffered by the EN Kitoi (Lieverse et al. 2007a; Temple et al. 2014; Waters-Rist 2011). While these episodes appear to have been severe enough to leave their marks on the skeletons, I cannot say if they placed the hunter-fisher-gatherers into a state of iron deficiency. Furthermore, the EN relied more on fishing than did the LN–EBA of the Angara River Valley (Katzenberg et al. 2009, 2010; Katzenberg and Weber 1999; Lam 1994; Weber et al. 2011 and 2010a; Weber and Bettinger 2010b). This is not to say, however, that the EN Kitoi did not hunt terrestrial game like their LN–EBA counterparts. In fact, more recent literature supports the interpretation that there were fewer differences between the respective diets of the EN and LN–EBA than previously thought (Weber et al. 2011).

Should I assume for a moment, though, that the EN Kitoi relied less on hunting and more on fishing than did the LN–EBA ISG, then I can examine what this difference may have meant to the Kitoi. More zooarchaeological remains of whitefish (*Coregonus lavaretus baicalensis*) exist in the middle Holocene Cis-Baikal deposits than those of any other fish species (Losey et al. 2012; Nomokonova et al. 2010). That, paired with the relative accessibility of the species suggests to Losey and his colleagues that this species was the primary fishing prey of the Kitoi and ISG (2012). Other fish species such as perch (*Perca spp.*) and grayling (*Thymallus thymallus*) may have played equal, or arguably greater (Katzenberg et al. 2012), rolls in diet (Losey et al. 2012), but it will be whitefish that I examine here due to its presence in the zooarchaeological sample. The iron content in whitefish is 0.4 mg per 100 g (SELF Nutrition Data 2014a). Moose (*Alces alces*) and deer (*cervus*), in comparison contain 3.2 and 3.4 mg of

iron per 100 g of raw meat, respectively (SELF Nutrition Data 2014b, c). Females of child bearing age require between 16 and 20 mg of iron a day according to Health Canada (Health Canada 2011). There is approximately eight times as much iron in raw moose and deer meat as there is in whitefish. It would take eating approximately 4.0 kg of whitefish, 500 g of raw moose meat, or 470 g of raw deer meat every day for a woman of childbearing age to satisfy her iron requirements. Game meat such as caribou, for example, is documented as providing enough iron to satisfy the nutritional requirements of hunter-fisher-gatherers in northern Canada (MacKay and Orr 1988; Sharma et al. 2009). It follows, then, that the ISG hunters of deer and moose may have had a better chance of satisfying their iron requirements than would have had the Kitoi if this nutritional difference existed. The fact remains, however, that we cannot say for certain how much the Kitoi were hunting game compared to the ISG and, so, this argument remains speculative.

Equally noteworthy are the weaning practices of the Kitoi and the ISG in understanding the presence of COPH in those EN individuals younger than four years. Health Canada suggests that mothers should breastfeed their infants exclusively for the first six months of life. After this point, the infant's pre-birth iron stores are depleted and their nutritional requirements cannot be met by breast milk alone, so supplementary/weaning foods are required (Healthy Active Living Committee and the Canadian Pediatric Society 2002). This follows a developmental increase in the use of iron that occurs between an infant's second and sixth month following birth (Fomon et al. 2000). While both the Kitoi and the ISG breastfed their children longer than six months, the ISG appear to have introduced weaning foods earlier (at one year of age versus the Kitoi's two years of age; Waters-Rist et al. 2011). The nutritional requirements of the Kitoi between the ages of 0.5–3.5/4.0 years old may not have been met compared to the somewhat shorter weaning period (0.5–3.0 years old) in the life of an ISG infant.

Furthermore, if their mothers were iron-deficient during pregnancy, the pre-birth iron stores of infants would also have been low and the mother's breast milk would have been iron-deficient (Betke 1970; Palkovich 1984). During pregnancy a woman's iron demands increases by 1000 mg (Bolt and Tchernia 1999; Halvorsen 2000) and they lose from 0.1 mg of iron per day (non-pregnant adult average) to 7.5 mg of iron per day at the peak of iron use (Milman 2006). This can be exacerbated if the mother is an adolescent, as subsdult women have higher developmental iron requirements than do older women (Beard 2000) or if the mother had a short

interval between this and her previous pregnancy (Miller 2016). Food stress within the population paired with the demands of pregnancy put mothers at greater risk for nutrition-induced anemia than their non-pregnant counterparts. While an argument for nutrition-induced anemia may be somewhat speculative, it is worth presenting if only to highlight a gap in our understanding. It is also important in light of recent clinical research stating that iron deficiency during infancy can cause problems during neurodevelopment (Beard and Connor 2003; Lozoff et al. 1987; Walter et al. 1989).

A discussion concerning infection-induced anemia is perhaps less precarious than one concerning nutrition-induced anemia. It has been hypothesised that the Kitoi lived in larger, more sedentary groups, while the ISG lived in smaller, more nomadic ones (Weber and Bettinger 2010b). As a result, factors such as population density, community hygiene, and the local ecosystem may have negatively affected members of the Kitoi culture more than they would have those of the ISG. The ISG did not stay in one place very long and, as such, they were able to leave behind their waste (food scraps, human and animal excrement, *etc.*) and any undesirable (e.g., boggy, snowy, barren, *etc.*) location. The Kitoi, on the other hand, were less mobile and, as a result, could have lived in closer proximity to one another and their waste for longer periods of time. They also hunted farther afield than did the ISG, suggesting that they either first cleared the local area of game and were then forced to hunt elsewhere, or that they chose to do so (Weber and Bettinger 2010b).

With higher sedentism and aggregation comes an increased risk for contracting and transmitting viral, bacterial, and parasitic infections (Christofides et al. 2003; Kent 1986). In section 2.10.0.0, I introduced the concept of iron-withholding. This is an adaptive immune response that attempts to stunt the reproduction of parasites, bacteria, and viruses by withholding the iron they require to reproduce. Iron-withholding functionally places the body into a manufactured state of iron-deficiency, one result of which may be COPH (Aufderheide and Rodríguez-Martín 1998: 349; Jurado 1997; Weinberg 1984; 1992).

Both Lieverse (2005) and Waters-Rist and colleagues (2014) have documented cases of calcified hydatid cysts found within the graves of EN females (one from Shamanka II, Grave 23; Waters-Rist et al. 2014; and two from Lokomotiv, Grave 25; Lieverse 2005). Analysis on the former cyst suggests that it was created during an infection of a parasitic tapeworm, *Echinococcus granulosis* (Waters-Rist et al. 2014). Such parasitic infections occur when an

individual ingests moose or reindeer meat contaminated with the parasite, or when they ingest other food stuffs or water containing contaminated feces, or via direct transmission from an infected dog, wolf, moose, or reindeer to a human (Sergiev and Ozeretskovskaya 2003). In their article, Waters-Rist and colleagues note that such an infection is often indicative of a close association between humans and dogs (Ovodov et al. 2011; Sablin and Khlopachev 2002; Waters-Rist et al. 2014) and that dog burials were the most prevalent during the EN in the Cis-Baikal (Losey et al. 2013; Waters-Rist et al. 2014). While these cysts are both documented in females, my data indicate significantly more incidences of COPH in males than in females. This suggests that individuals from the middle Holocene Cis-Baikal suffered from multiple types of infections (not just *Echinococcus granulosis*) that may have placed them into states of self-imposed iron-deficiency anemia. Sample sizes are small here, but the trend in the data may suggest that parasitic infections were both present in the sample and, arguably for *Echinococcus granulosis* infections, more common in the EN than in the LN–EBA. This supports the interpretation that the COPH lesions observed in the sample may have been due to infection-induced iron-deficiency anemia rather than (or in addition to) nutrition-induced iron-deficiency anemia.

In the last twenty years, other researchers have documented cases of potential infection-induced anemia in various populations. In the southwestern United States, the early Archaic Anasazi suffered from chronic iron deficiency. It has been argued that this was a result of viral, bacterial, and parasitic infections, and not the result of an iron-poor diet (Holland and O'Brien 1997; Kent 1986). Similar studies have been done on communities from northern Canada (northern Ontario and Nunavut) and from South Africa (in the Griqua, Khoe, and "Black" African communities) and they too have concluded that the iron-deficiency seen there was the result of infection rather than nutritional deficiencies (Christofides et al. 2005). Studies on more modern populations also find links between anemia and factors such as population density, hygiene, dwelling conditions, and infection (e.g., 19th century Fayette, Michigan and 21st century Mexico; Faulkner et al. 2000 and Cattaneo et al. 2009, respectively).

The sharp increase in individuals with COPH after weaning (i.e., in the 4–19 year age group) in both time periods, and the significant difference between the number of individuals with COPH between the EN and LN–EBA suggests an individual's (especially a EN individual's and, according to the logistic regression tests, those from Shamanka II) risk of developing iron-

deficiency anemia increased once they were weaned. Exposure to new foodstuffs and the loss of their mother's passive immunity all open a weaned infant up to new potentials for infection (Maher 1992; Pelletier 2000). That these infections occurred is strongly suggested by the presence of COPH in the sample. The presence of lesions in those less than 4 years old is not anomalous. Anasazi infants also presented with cribra orbitalia (Kent 1986). In fact, on archaeological human remains, COPH is seen in infants and children more frequently than in adults (Aufderheide and Rodríguez-Martín 1998: 349; Stuart-Macadam 1989a, b) and its presence is associated with an increase in mortality (Huss-Ashmore et al. 1982).

Finally, there are comparatively low proportions of individuals with COPH lesions amongst the LN–EBA ISG. These lesions are also absent among those less than 4 years and those older than 50 years. Risk factors for anemia-induced lesions appear to have been less common during this time than they were in the EN. The smaller and more mobile groups that the ISG lived in may have limited the risks that population density, community hygiene, and the local ecosystem (via their ability, as more mobile foragers, to leave behind undesirable locations) bring in terms of contracting an infection, while their hunting may have provided them with more iron-rich foods. The fewer indicators of physiological stress, such as short femoral length, low body mass, and linear enamel hypoplasia (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011), may be indicative of lower overall morbidity during infancy and childhood for the ISG. While all discussions remain speculative, they help to build our understanding of the helath of these individuals and open the general discussion to other possabilities.

5.1.3. Periodontitis

Periodontitis was present in over half of the sample (65.9%), with significantly more individuals exhibiting lesions in the EN than in the LN–EBA. Unlike the general trend, there are more individuals from Shamanka II with periodontitis than from Lokomotiv, and significantly more than from Ust'-Ida I. The difference between the proportions of individuals with periodontal lesions is not significant between the sexes, though males have more in the entire sample and by time period (just barely in the LN–EBA). In the entire sample, the number of individuals with periodontitis increases significantly after 20 years of age regardless of time period. Unlike any of the other lesions, the presence of periodontitis is variable within Ust'-Ida I and consistent within Lokomotiv. In Ust'-Ida I, its presence is dependent upon age, while age

and sex have no statistically significant bearing on the presence of the lesion in Lokomotiv, in spite of the overall increase in the presence of periodontitis after 20 years of age. In the EN, all the variability comes from Shamanka II, in which both age and sex are predictive of the presence of a lesion. Finally, periodontitis only co-occurs with periostitis and COPH.

Periodontitis is caused by the build up of oral biofilm, or plaque, (DeWitte 2012; Holt et al. 2000) on teeth, which allows for the incubation of oral pathogens that cause the disease. Given my results, it follows that there would be more dental calculus (or calcified dental plaque) on the teeth of individuals from the EN and, specifically, on those from Shamanka II. In their work on dental health indicators in middle Holocene hunter-fisher-gatherers from the Cis-Baikal, Lieverse and colleagues (2007a) found more dental calculus on the teeth of individuals from Shamanka II than on those from the other cemetery they studied (Khuzhir-Nuge XIV, an EBA site from the Little Sea microregion). More recently, Megan Clarke, who collected the periodontitis data used here, found more calculus deposits on those from Shamanka II than on those from Lokomotiv, Ust'-Ida I, and Khuzhir-Nuge XIV (2015: 90–111). The difference in severity was significant between Shamanka II and Ust'-Ida I for all sub-populations save for subadults (<20 years of age), where the difference existed even though it was not significant (Clarke 2015: 98–111). Clarke also found significantly more calculus on males than on females in all cemeteries, save for on those from Lokomotiv, and more on young adults (20–35 years old) than on old adults (>35 years old; Clarke 2015: 90–98).

The dental calculus data align in many ways with the periodontitis data. Individuals from Shamanka II have more dental calculus and periodontitis than do those from other cemeteries, and young adults have more dental calculus and periodontitis than older adults (for my data, this is only true in the LN–EBA). While the incidence of dental calculus on individuals from Lokomotiv and Ust'-Ida I varied by age and sex, that from Ust'-Ida I was significantly lower than that from Shamanka II for both adult males and females (Clarke 2015: 82–97). In other words, the incidence of both periodontitis and dental calculus generally decreased from Shamanka II to Lokomotiv to Ust'-Ida I.

Clarke found that males had a higher dental calculus severity than did females. The notable exception to this trend was in individuals from Lokomotiv, where females had more dental calculus than males, though not significantly so (2015: 75–82). This suggests that individuals buried in Lokomotiv may have divided food along gender lines differently than those

buried in Shamanka II and Us'-Ida I, and may help to explain some of the variation in the Lokomotiv data.

Research by Lieverse and colleagues (2007a) found more incidences of periodontitis in individuals from Shamanka II than from Lokomotiv, and more incidences from Ust'-Ida I than from Lokomotiv. The first half of these results is consistent with both my data concerning periodontitis and Clarke's concerning dental calculus, but the second half is not. Both Lieverse and her colleagues, and Clarke were collecting and analyzing dental calculus in different ways and to different degrees. Clarke's work was, arguably, designed to be more detailed than that of Lieverse. But the fact that Lieverse's and colleagues' methodology uncovered this discrepancy in the EN cemeteries is consistent with other aspects of Clarke's work concerning the inter-cemetery sex-based differences previously discussed (2015: 75).

While these sex-based differences in Lokomotiv appear to have affected the amount of dental calculus on the teeth of males and females, there was more periodontitis present in individuals from Lokomotiv than there was in those from the LN–EBA. In other words, the infection load of males remained higher than that of females from Lokomotiv in spite of them having less dental calculus. Perhaps their stress level affected their robusticity against these dental pathogens. Both Lieverse and colleagues (2007a), and Clarke (2015: 34) make a point of discussing the different post-excavation strategies employed with the EN and the LN–EBA remains in that dental calculus was often removed during the cleaning of the Lokomotiv and Ust'-Ida I remains making it difficult to later access its frequency within the population. This may account for some of the differences in calculus frequencies observed and must be mentioned here, too.

One other notable exception is worth discussing. Clarke found that young adults generally had more dental calculus than older adults. She attributed this to dental wear and using the mouth as a tool, all of which may have removed calculus from tooth surfaces (Clarke 2015: 82). My LN–EBA data are consistent with this; the amount of periodontitis decreases after 50 years of age. In the EN, however, the amount of periodontitis increased in those older than 50 years. This may be an artifact of small sample sizes, since there were only eight and two individuals sampled from the EN and LN–EBA, respectively, who were older that 50 years (see Table 7 in section 4.1.5.1.). Antemortem tooth loss was also seen to increase with age by Lieverse and colleagues (2007a) in the same sample. This may also account for the decrease in

periodontitis after fifty years of age because following tooth loss, periodontitis in the surrounding gingeva and bone will recover. Since periodontitis is known clinically to increase with age (Hillson 1996: 263; Oztunc et al. 2006; Wasterlain et al. 2011), this decrease after the age of 50 suggests that individuals of these ages may have lost many of their teeth due to severe periodontitis, thus preventing Clarke from observing the lesions in reference to the teeth.

Populations can often be classified as caries formers or calculus formers (Hillson 1979). The Kitoi and ISG, then, appear to be calculus formers. In each time period, sex, age group, and cemetery, there was more periodontitis than there was caries within the sample. Caries was absent (or nearly absent) in the sample, similar to previous studies (Lieverse 2005, Lieverse et al., 2007). Four different scenarios can explain the low incidence of caries. The first two reflect a change in the pH of the mouth. First, the data may reflect a diet high in protein and low in carbohydrates, which promotes a higher oral pH and calculus formation (Hillson 1979). Second, the mineral content of the water in the region may have also promoted a high pH within the mouth and the mineralization of dental plaque (i.e., calculus; Lieverse 1999). Third, dental wear—and antemortem tooth loss for that matter—as noted within the sample by Lieverse and her colleagues (2007), may also account, at least in part, for the low frequency of caries because localized areas of demineralization can be destroyed by dental wear before they can expand. Fourth, the genetic makeup of the populations may have lent itself to saliva with high levels of calcium and phosphate (both linked to calculus formation; Dawes 1970). It is most likely that the first scenario played the most substantive role in the formation of dental calculus rather than caries in the sample. Previous research has demonstrated that the Kitoi and ISG cosumed a diet in which animal and fish proteins were the main elements (Katzenberg and Weber 1999; Katzenberg et al. 2012; Lam 1994). This sort of pattern is also consistent with other northern hunter-fisher-gatherer populations (Cordain et al. 2000). Regardless of the cause(s) for the presence of dental calculus and absence of caries within the sample, however, this distinction is visible and highlights this etiological dichotomy.

Periodontitis is different from the other infection-induced lesions examined here since it reflects other mechanisms (e.g., plaque and calculus formation, dental wear, *etc.*) more directly than it reflects a population's infection load. While antemortem tooth loss was not investigated here, as Lieverse and colleagues noted (2007a), antemortem tooth loss was usually the result of severe periodontitis. Periodontitis remains an important lesion to study, in spite of its irregular

etiology, since chronic periodontitis is a risk factor for many other diseases such as Type 2 diabetes mellitus (or T2DM; Jimenez et al. 2012; Macedo et al. 2014), preterm births and preeclampsia (Conteras et al. 2006), rheumatoid arthritis (or RA; Lauren et al. 2012; Persson 2012), pneumonia (Shay 2002; Yoneyama et al. 2002), cardiovascular disease (Azarpazhooh and Tenenbaum 2012; Geerts et al. 2004; Hokamura and Umemura 2010; Radwan-Oczko et al. 2014; Shay 2012), and cerebrovascular disease (Shay 2002). As such, individuals from Shamanka II (and to a somewhat lesser extent, Lokomotiv), males, and adults may have all been at greater risk of developing other complications associated with their periodontitis than their counterparts.

5.1.4. Upper Respiratory Infection (URI)

The upper respiratory infection category is comprised of the merged binary results of four types of sinusitis and the infection of the concha bullosa (see section 3.2.2.). These lesions were present in well over half of the sample (70.6%). This percentage decreased significantly over time and varied significantly by cemetery. By cemetery, URI lesions were significantly more common in Shamanka II and Lokomotiv than in Ust'-Ida I, with the condition being more common at Lokomotiv than Shamanka II (but not to a significant extent). Unlike many of the other lesions documented here, females more frequently exhibited lesions indicative of upper respiratory infection than did males. When divided by time period, this remained true for the EN population, but not for the LN–EBA. None of these sex-based differences were statistically significant. Lesion incidence increased with age and was the most common in those between the ages of four and 50. This remained true in both time periods. In general, age, sex, cemetery, and time period were predictive of URI lesions and this remained true in the EN. Within each cemetery, nothing else was predictive of the presence of upper respiratory infection, making the cemeteries themselves the predictive variables there. Despite its exception to the general sex-based trend in the sample, upper respiratory infection co-occurred with periostitis, COPH, and otitis.

Sinusitis can be caused by a primary or secondary infection (Evans 1994; Merritt and Pfeiffer 2000). Most commonly, viruses are the cause of these, followed by bacteria (Evans 1994; Gwaltney 1996; Sande and Gwaltney 2004). Many mechanisms can lead to sinusitis, an example of which is the abscessing of a maxillary tooth infection through its root and into the maxillary sinus (Melén 1994; Melén et al. 1986; Shafer et al. 1974; Wright 1979). In fact, five different individuals exhibited maxillary sinusitis that could be attributed to (or was exacerbated

by) a periapical tooth abscess: Shamanka II Burials 17.1, 34.1, 48.1, 58.1, and 71.1. In general, however, the etiology of the lesions noted in the sample was non-specific.

The EN populations may have been at a higher risk of developing sinusitis than were the ISG. As was suggested in section 5.1.2.0, the larger and more sedentary settlements that the Kitoi are believed to have lived in (Weber and Bettinger 2010b) may have resulted in more opportunities for the transfer of communal pathogens, including those responsible for upper respiratory infections. Other risk factors such as immune suppression, chronic mucosal edemas, and/or hypersecretion resulting from allergies or other upper respiratory infections (Albalak 1997; D'Souza 1997; Ellegård 1996; Evans 1994; Gwaltney 1996; Headland 1989; Melén et al. 1986; Pérez-Padilla et al. 1996) may have also been common for the Kitoi, these as a result of their higher levels of physiological stress and, as we have seen here, infection in general.

Factors within the natural and cultural environments of the Cis-Baikal may have also had a hand in the rate of upper respiratory infection within both the EN and LN–EBA populations. Cold winds, as seen throughout Siberia in the fall and winter (Klemm et al. 2013), elevate the risk of developing sinusitis. Swimming, which may have occurred in these water-focused cultures (as per their placement of burials around the lake and rivers, and their use of aquatic resources for subsistence), is also a risk factor of sinusitis (Albalak 1997; D'Souza 1997; Ellegård 1996; Evans 1994; Gwaltney 1996; Headland 1989; Melén et al. 1986; Pérez-Padilla et al. 1996; Shafer et al. 1974; Wright 1979). Finally, chronic exposure to wood smoke, as may have been present in the built environments of the Kitoi and ISG, is considered to be one of the largest risk factors for the development of sinus infections in the world today and is the risk factor most widely discussed in the archaeological literature (e.g., Albalak 1997; Brachman 1990; de Koning et al. 1985; D'Souza 1997; Ellegård 1996; Headland 1989; Lewis et al. 1995; Merritt and Pfeiffer 2000; Pérez-Padilla et al. 1996; Wright et al. 1994).

In the pre-industrial era, wood smoke would have been the most common pollutant in built environments (Roberts 2007). It is likely that wood, being readily available throughout the middle Holocene due to the presence of taiga and steppe forests all around the Cis-Baikal (Weber and Bettinger 2010b), would have been the fuel of choice for both the Kitoi and the ISG. Indoor wood fires in poorly ventilated spaces would have especially exposed the people to these pollutants and this chronic smoke inhalation would have put the populations at high risk of developing sinusitis. Wood smoke contains noxious gasses and particulates that both cause

respiratory distress and decrease the local humidity. These result in the paralysis of the cilia within the sinuses and the decreased permeability of the ciliated epithelium, respectively (Albalak 1997; Brachman 1990; de Koning et al. 1985; D'Souza 1997; Ellegård 1996; Headland 1989; Lewis et al. 1995; Merritt and Pfeiffer 2000; Pérez-Padilla et al. 1996; Wright et al. 1994). Reactive oxygen species (or ROS, aka free radicals) within wood smoke particulates can precipitate inflammation within the epithelium and cause the swelling of the skin around an ostium, consequently blocking the entrance and exit of the sinus and precipitating sinusitis (Merritt and Pfeiffer 2000; Halliwell and Cross 1994).

There is very little archaeological evidence concerning the dwellings of the Kitoi and the ISG (Weber and Bettinger 2010b), though some Russian literature on the subject does exist (Emel'ianova 2006; Emel'ianova and Kharinskii 2008; Emel'ianova et al. 2009) and concerns the presence of middle Holocene pit houses within the Cis-Baikal. More recently, the discovery of pit-house remains in northern Baikal has been discussed at a BHAP workshop (personal communication, Lieverse 2015). Pit houses from other geographic regions have been widely researched in the archaeological literature and this work suggests that these structures were poorly ventilated (e.g., Kivett and Metcalf 1997). For example, among the prehistoric Upper Republican peoples of Nebraska, United States of America, pit houses contained only one entrance: an opening in the center of the roof that allowed access down into the dwelling via a ladder (Kivett and Metcalf 1997). Such a built environment would not allow for enough air circulation to remove the aggregating gasses and particulates from the central fires (Raphaël et al. 1997; Roberts 2007; Sundman and Kjellström 2013).

In many cultures, females spend more time indoors than do males. Domestic activities such as cooking, meat and hide processing, sewing, *etc.* are all commonly carried out indoors, particularly in cold environments (Raphaël et al. 1997; Roberts 2007; Sundman and Kjellström 2013). As a result, it is females who typically suffer from wood smoke-induced sinusitis (e.g., Panhuysen et al. 1997; Roberts 2007). Kitoi males are believed to have borne a heavier physical activity load (hunting far afield, *etc.*), while Kitoi females are believed to have borne less (Lieverse et al. 2013, 2016), and it may have been these indoor domestic activities that were keeping the females from more physically-stressful activities.

This pattern can be seen in the EN data. I would then expect, however, that females from Shamanka II (and all individuals from Shamanka II) would have presented with a higher

incidence of lesions than those from Lokomotiv, as Lieverse and colleagues (2013; 2016) found Lokomotiv to be unique in that individuals there consistently exhibited more severe entheseal changes than did those from the other sites examined. This was not the case with my data. Here, there was more sinusitis present at Lokomotiv than at Shamanka II, though this difference was not statistically significant. In the logistic regression models, sex was not a predictive variable for the presence of sinusitis in any cemetery, though it was in the EN. Clearly, physical activity and sinusitis patterns are not mutually exclusive. Since there is no biological predisposition for one sex or the other to contract sinusitis (Merritt and Pfeiffer 2000), it is exposure to environmental risk factors and the presence of chemical and structural changes within the body that influence the incidence of sinusitis within populations (Evans 1994; Merritt and Pfeiffer 2000). As a result, it is likely that females from the EN were exposed to these risk factors more often than were EN males, and that the EN populations in general (particularly those from Shamanka II) were exposed to these risk factors more often than were the LN–EBA populations.

Finally, the dispersal of sinusitis by age is what would be expected in the sample. While the hypertrophy of the tonsils or adenoids can lead to sinusitis in children (Albalak 1997; D'Souza 1997; Ellegård 1996; Evans 1994; Gwaltney 1996; Headland 1989; Melén et al. 1986; Pérez-Padilla et al. 1996) and can help explain the high percentage of those younger than four years with chronic sinusitis, I would generally expect to see lesions primarily in adults. This is because children are not done growing and, as a result, the bone in their sinuses is remodelling faster than in that of adults. Rapid bone turnover more easily masks the condition in infants and children than in adults (Enlow 1990), as is seen in the sample.

5.1.5. Otitis

Otitis is unusual in that almost every individual examined presented with some indicator of otitis (present in 99.4% of the sample): otitis externa, otitis media, and/or mastoiditis. All of the EN individuals examined had otitis, as did all but one LN–EBA individual. This individual was a female between aged 20–49 years. Otitis co-occured with every lesion type save upper respiratory infection, which it co-occurred with nearly significantly. While the frequency of otitis is unusual in the context of these cemeteries, its frequency is not entirely unusual in the context of other non-antibiotic using populations. Flohr and Schultz found that 83.4% of the early medieval skeletons they examined had lesions associated with mastoiditis (2009), while Avenstorp and colleagues found that between 5.8% and 55.6% of modern Greenland children

they surveyed had some form of otitis media (2016). These examples show that otitis may be quite common in populations that do not have, or that have limited, access to antibiotics as was the case for the populations examined here.

The etiology and spread of these three indicators of otitis can be linked. Otitis externa is caused by the trapping and proliferation of a pathological microorganism in the outer ear canal (Anthony and Anthony 1982; Garin et al. 1997; Heilbrun et al. 2003; Holt 1992; Mann 1992; Mays and Holst 2006; Piepergerdes et al. 1980; Sismenis et al. 1986; Vrabec and Chaljub 2000). Otitis externa can spread to the middle ear and cause otitis media and otitis media can spread to the mastoid process and cause mastoiditis. More often than not, otitis media and mastoiditis are caused by *Streptococcus pneumonia* or *Haemophilus influenza* infections (Aufderheide and Rodríguez-Martín 1998: 253; Rosen et al. 1986; Bitar et al. 1996; Gliklich et al. 1996; Niv et al. 1998). Both of these bacteria are contagious and can be transmitted via the spittle produced when coughing or sneezing, or (in the case of *H. influenza*) the touching of a contaminated surface (Centers for Disease Control and Prevention 2014a, b). Both these bacteria cause otitis media, mastoiditis, and sinusitis among other things (Center for Disease Control and Prevention 2014a, b; Fleischer 1979). It makes sense, then, that otitis and upper respiratory infections co-occur within the sample.

Otitis and mastoiditis can also be caused by upper respiratory infection due to their structural connection via the Eustachian tube. Almost the entire sample suffered from otitis externa, otitis media, and/or mastoiditis (99.4%), while more than half of the sample showed signs of having had chronic upper respiratory infections (70.6%). As a result, it may be possible that more than half of the middle ear and mastoid infections could have been caused by *S. pneumonia* or *H. influenza* subsequent to the bacteria travelling from the nasopharynx up the Eustachian tube and into the middle ears.

Genetic factors may have also played a part in the high incidence of otitis (particularly otitis media and mastoiditis) in these two populations. The length of an individual's Eustachian tube can affect the chance of regularly developing otitis media (Mays and Holst 2006). Some northern populations, such as the Canadian, Alaskan, and Greenland Inuit, have short Eustachian tubes as children, which predisposes them to suffer from higher rates of otitis than non-Inuit children (Dallaire et al. 2006). These acute infections frequently develop into chronic infections that can result in hearing impairment (Baxter 1999; Bluestone 1998; Dallaire et al. 2006; Julien

et al. 1987; Thérien 1988). Lavallée and Bourgault (2000), for example, report higher incidence of hearing problems among Quebec Inuit females than among Quebec Cree and southern Quebec females. Before their fifth birthdays, Inuit children commonly contract acute otitis media, upper and lower respiratory tract infections, gastro-intestinal infections, and coetaneous infections (Dallaire et al. 2006). The populations of the middle Holocene Cis-Baikal were comprised of various haplogroups (Mooder et al. 2006, 2010; Schurr et al. 2010). While distinct, it is possible that all were predisposed to otitis.

There is a large variety of pathological micro-organisms that can cause otitis externa, making its etiology difficult to diagnose. More informative, perhaps, is the most common activity associated with trapping micro-organisms in the outer ear canal: swimming (Centers for Disease Control and Prevention 2014; 2015; Dorevitch et al. 2012; Piercefield 2011; Springer and Shapiro 1985). In fact, otitis externa is often called "swimmer's ear" for this reason. Pathological micro-organisms can get trapped when an individual leaves their outer ear canal(s) wet by not drying or draining them following swimming (Centers for Disease Control and Prevention 2015) or when an individual swims often and the constant exposure to water removes the protective, wax coating of the outer ear canal (Springer and Shapiro 1985). If left untreated, this acute infection can turn into a chronic problem, which would be visible osteologically.

Water was likely a central part of life for the Kitoi and ISG. The Kitoi and, perhaps to a lesser extent, the ISG of the Angara River Valley were dependant on fish as a primary resource (Weber and Bettinger 2010b). In their 2009 paper, Lieverse and colleagues hypothesized that the pattern of large muscle attachments sites on Kitoi individuals' upper limb bones was caused by manually propelling watercraft (Lieverse et al. 2009). Perhaps these middle Holocene cultures did use boats, but what about swimming? The water in the region is notoriously cold, and yet, it would seem counterintuitive to think that these peoples' lives were centered (geographically and subsistence-wise) around water and that they did not swim. That otitis externa was so prevalent amongst the Kitoi and the ISG of all ages and both sexes suggests that perhaps they did.

What is interesting, in light of the pervasiveness of otitis externa, is the almost complete lack of auditory exostoses documented in the sample. External auditory exostoses are believed to be the result of cold stress, be that exposure to water or air colder than 19^{o}C (Deleyiannis et al. 1996; Fabiani et al. 1984; Filipo et al. 1982; Kemink and Graham 1982; Kennedy 1986; Kroon et al. 2002; Scrivener 1981; Umeda et al. 1989), or mechanical or chemical stimuli (Godde 2010;

Okumura et al. 2007; Özbek 2012; Hrdlička 1935; Berry 1975; Hutchinson et al. 1997). Swimming in cold water should have precipitated these lesions, according to many researchers (e.g., Ascenzi and Balistreri 1975; Crowe et al. 2010; Frayer 1988; Godde 2010; Hutchinson et al. 1997; Kennedy 1986; King et al. 2010; Manzi et al. 1991; Okumura et al. 2007; Özbek 2012; Wang et al. 2005; Wong et al. 1999), so their absence may suggest that something else was occurring. Similarly, the cold atmospheric temperatures of the Cis-Baikal, particularly in the winter months, should also have triggered the formation of external auditory exostoses.

There is no definitive explanation for the absence of these lesions, but I propose two possible ones. First, the Kitoi and the ISG may have both employed the use of clothing or other items that protected their ears from cold, be that from cold water or air. Second, the Kitoi and ISG may not have been genetically predisposed to the formation of external auditory exostoses. Whatever the reason for the absence of these lesions, external auditory exostoses do not lend more information to our understanding of otitis in the middle Holocene Cis-Baikal.

Chronic otitis and mastoiditis can lead to a host of complications. Tinnitus (or the hearing of sounds that do not exist) is the mildest complication. The destruction of the auditory ossicles, the tympanic membrane, or the cochlear branch of the auditory nerve can lead to hearing impairment and loss. Associated intra-cranial complications such as septicemia and osteomyelitis may lead to meningitis or cerebritis (the inflammation of the brain) and even death. And inflammation and the associated erosion of bone can cause the destruction of the stylomastoid foramen of the facial nerve and the paralysis of the ipsilateral side of the face (Aufderheide and Rodríguez-Martín 1998: 253; Bayazit et al. 2002; Go et al. 2000; Mays and Holst 2006; Minks et al. 2013; Palma et al. 2014; Rai 2014). In some cases involving endocranial complications and mastoid abscessing, the cerebrospinal fluid that coats the brain can begin to drain through the mastoid and out of the abscess, leaving the brain in great danger of being damaged (Minks et al. 2013). The presence of each of these conditions could provide us with further insight into the lifeways of the middle Holocene inhabitants of the Cis-Baikal and would be important areas for future research.

5.1.6. Summary

The etiological mechanisms and risk factors of these infection-induced lesions, as well as the patterns of their distribution, communicate a lot in terms of the community health and lifeways of the Kitoi and the ISG: activities undertaken, apparel worn, foods eaten, communities

lived in, *etc.* Below is a summary of the possible etiological mechanisms and risk factors for each type of lesion that was observed in the sample (Table 5.1).

Table 5.1: Summary of likely etiological mechanisms and risk factors for each type of lesion observed in the sample.

Lesion Type	Etiological Mechanism(s)	Risk Factors
Periostitis	Inflammation of and/or trauma to the overlying soft tissue	Chronic infection, inflammation, direct trauma, *etc.*
Cribra Orbitalia and Porotic Hyperostosis	Nutrition- or infection-induced iron-deficiency anemia, megaloblastic anemia, or sub-periosteal bleeding	Infection, nutritional deficiency, genetic susceptibility, population density, dwelling conditions, or local ecosystem
Periodontitis	Inflammation of the overlying gingiva	Oral hygiene or presence of micronutrients, fat, grit, carbohydrates, sugars, or casein in the diet
Upper Respiratory Infection	Inflammation of the overlying soft tissue and/or an increase in pressure within the sinus	Otitis, living in close proximity to other people, or cold stress
Otitis	Inflammation of or trauma to the overlying soft tissue and/or and increase in pressure within the structure of the ear	Upper respiratory infection, short Eustachian tube, cold stress, or swimming

Many of these infections are associated with one another in terms of their etiologies (be they shared or related). Upper respiratory infections can lead to otitis media and vice versa. Infection, in general can precipitate COPH. Periodontitis can spread inflammatory cytokines around the body and give rise to a host of disorders, as explained above. And sinusitis and otitis externa can be brought about by swimming. That many of these lesions were observed in the same individuals reflects how many males, adults, and EN individuals presented with infection-induced lesions, and the ways in which those lesions are related epidemiologically.

5.2.0. Larger Implications

Considering these non-specific infection data and the physical stress data collected previously by BHAP members, three connections between the two have emerged: nutrition, activity, and habitation. How each of these themes relates to the infection data is the subject of

the rest of this chapter and I will draw upon the etiologies of the infection-induced lesions as discussed above.

Nutritional stress experiences by the Kitoi may have resulted in an increase in their morbidity and mortality. As discussed in section 1.2.2, resource scarcity in the late winter and early spring may account for the more severe episodes of physiological stress seen in the EN than seen in the LN–EBA (Lieverse et al. 2007a; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011). These episodes affected the growth of young individuals, as seen in the patterns of linear enamel hypoplasia (LEH; Lieverse et al. 2007a; Link 1999; Waters-Rist 2011), femoral lengths, and the body mass in infants and children (Temple et al. 2014), and affected the mortality of infants being breastfed by stressed mothers (Waters-Rist et al. 2011). Nutritional stress in childhood and later weaning can affect the mortality and morbidity of individuals in the long-term, since stress events that occur in childhood can increase the risk of death in later adulthood. This can occur through epigenetic pathways or through the failure to prime the immune system properly (e.g., via the absence of a healthy microbiome in the gut), all of which makes the individual more frail and susceptible to infection and other future stress episodes (Cameron 2002; Klaus 2014; Saunders and Hoppa 1993; Watts 2011). Kitoi individuals were more physiologically stressed as infants (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011) and, as a result, were possibly more susceptible to infection and other forms of stress in their later years.

The breastfeeding habits of the Kitoi and ISG probably contributed to their immune function. The duration of the weaning process for the EN Kitoi (from 0.5 to 1.0 year; Waters-Rist et al. 2011) could have decreased the survivorship potential of these toddlers, as they had a shorter period of time in which to build up their resistance to foreign infectious agents while being buffered from harsher infections via their mother's resistance as compared to the LN–EBA ISG (Habicht et al. 1986; López-Alarcón et al. 1997). The use of emergency breastfeeding as well as the death of breastfeeding infants (Waters-Rist et al. 2011) suggests that outside stress, most likely seasonal nutritional stress, was an ubiquitous fact of life in the EN that had both short- and long-term consequences.

That there were more infection-induced lesions in the Kitoi populations than in the ISG suggests that they suffered from more chronic infections than did the ISG populations. These were not necessarily life-threatening infections, but chronic infections are both the result and

catalyst of physiological stress (DeWitte and Bekvalac 2011). Infection incidence increases with advancing age in both the EN and the LN–EBA (small sample sizes in those older than fifty years old prohibited assumptions from being made for these older individuals). In both populations, the individuals with the most robust immune systems often survived later, with their skeletons showing more signs of the chronic infections, while individuals with less-robust immune systems died younger and before their skeletons could be affected by an infection. For these individuals, death may have come during or shortly following a stress event.

As for sex, under ideal conditions in which all age groups and sexes have equal access to food and other resources, females clinically exhibit a stronger immune response and have a lower risk of early mortality than do males (Ackerman 2006; Bouman et al. 2005; Ghazeeri et al. 2011; Gleicher and Barad 2007; Rubtsov et al. 2010). That females have a biological buffer to the deleterious effects of physiological stress helps to explain why males in the sample generally have more signs of infection-induced lesions on their skeletons than did females. This is not to say that females in the sample were not stressed, they were, but males appear to have suffered from more of the physical effects.

Biology, as well as activity-related stress, can explain why males in the sample had more infection-induced lesions than females. Changes in the entheses and patterns of osteo-arthritis show that males from the EN engaged in more/different types of physical activity than did EN females and individuals from the LN–EBA (Lieverse 2010; Lieverse et al. 2007b, 2009, 2013, 2016; Stock et al. 2010). Males, as a result, bore much of the physical burden in the EN. Following this pattern, there were more periostitis lesions in both the EN and in males. As previously discussed, I cannot rule out that some of these lesions were the result of soft tissue trauma or local infections. It is possible that this trauma may have been sustained while the males were working (e.g., hunting, fishing, travelling *etc.*).

If males were undertaking more physically intense activities than were the females, then the question "what were they doing?" logically comes next. I do not wish to suggest that work was divided along gender lines in such a black and white manner, but it is important to note that females did exhibit a higher incidence of lesions suggestive of upper respiratory infection than did males in the EN, though not significantly so. This may have been the result of chronic exposure to wood smoke, as is popularly hypothesized (e.g., Panhuysen et al. 1997; Rajpandey 1984; Roberts 2007), suggesting that much of the work females did could have been inside

poorly ventilated dwellings. Thus, the answer to this question may become clearer; the Kitoi females were working indoors more than were men.

The distribution of Kitoi and ISG populations across the Cis-Baikal may have also resulted in significantly more individuals from the EN suffering from chronic infections than their LN–EBA counterparts. While also contributing to increased physiological stress among males and seasonal resource scarcity in the EN, the larger, more sedentary settlements of the Kitoi likely increased the likelihood of transmitting pathogens from person-to-person (Christofides et al. 2003; Kent 1986). Each of these factors could have lead to more instances of infection (and chronic infection-induced lesions such as periostitis and COPH) within the Kitoi populations.

In summary, differences in nutrition, activity, and habitation conditions may account for much of the variability in the presence of non-specific infections documented here. Frequent episodes of nutrition-based physiological stress in the early lives of Kitoi infants and children may have resulted in an increase in the mortality of said individuals (especially for the biologically more-susceptible males). Higher activity levels in males and lower activity levels in females in the EN may have resulted in an increase in periostitis and sinusitis, respectively. Finally, the more concentrated settlements of the Kitoi may have lead to an increase in contagious infection and, in turn, physiological stress, in EN individuals.

The etiological mechanismss behind these lesions are varied, complex, and involve risk factors and relationships that cannot easily be summarized or clearly labelled. These are non-specific infections and, while common themes may emerge from this examination, their complete etiologies will, for now, remain unknown. The aim of this discussion was not to define the factors that lead to the observed lesions, but to discuss the infectious pathways that are often associated with them and to relate these back to the physiological stress data previously gathered.

Chapter 6
Summary and Conclusions

6.0.0. Summary and Conclusions

6.1.0. Summary

This research was designed to answer the following research question of whether or not non-specific infection-induced lesions occurred more in those populations who were found to have been the most physiologically stressed. In order to do this, skeletal remains from 250 individuals from the middle Holocene Cis-Baikal were macroscopically and non-destructively examined for ten indicators of non-specific infection: periostitis, osteomyelitis, cribra orbitalia and porotic hyperostosis (or COPH), caries, periodontitis, upper respiratory infection (maxillary, ethmoid, frontal, and sphenoid sinusitis, and the infection of a concha bullosa), otitis (otitis externa and interna, and mastoiditis), auditory exostoses, and trachoma.

Two statistical questions were constructed in order to answer the research question. 1) Are the numbers of individuals with infection-induced lesions significantly different across populations or across sub-groups, e.g., age categories or sexes, within each population? This was answered in two ways. First, a series of binomial tests were carried out in which the proportion of individuals from one group who exhibited each lesion type was compared to the proportion of individuals from another group who exhibited the same type of lesions (e.g., comparing the number of females and males with periostitis). Second, chi-square tests were performed in order to see if any of the lesion types co-occurred within the sample. 2) Which levels of which explanatory variables are predictive of a positive response variable, i.e., the presence of a lesion of a certain type? This was answered by running logistic regression tests in order to discover the influence of each explanatory variable (i.e., age, sex, cemetery, and time period) on the presence of a non-specific lesion.

Some underlying trends emerged from the results of the statistical tests. There were generally more non-specific infection-induced lesions in adult males from Lokomotiv than any

other sub-population. The binomial tests showed that these patterns were statistically significant for 2–4 lesion types at a time (most consistently involving periostitis, COPH, and periodontitis) and the chi-square tests confirmed the co-occurrence of these lesions types along with upper respiratory infection and otitis. The logistic regression tests showed greater variation in the Early Neolithic (or EN) cemetery of Shamanka II than in either the EN cemetery of Lokomotiv or the Late Neolithic to Early Bronze Age (or LN–EBA) cemetery of Ust'-Ida I with much of this variation coming from the aforementioned sub-populations (particularly from those individuals older than 20 years).

These trends are consistent with research on disease and physiological stress previously done by Baikal-Hokkaido Archaeology Project (or BHAP) members (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011). The nutrition, activities, and dwellings of the EN Kitoi and LN–EBA Isakovo-Serovo-Glazkovo (or ISG) may have all played significant roles in determining the patterns of infection and stress that can be seen on their skeletons today (see section 5.2.0.0.). Each of these trends elaborates on our understanding of the relationship between infection and physiological stress in some way. In other words, the results of this research suggest that, yes, those populations who were the most physiologically stressed were also those populations who had the most non-specific infection-induced lesions.

Previous research undertaken by Angela Lieverse (2005, 2010) showed that only 4.1% of observed Cis-Baikal individuals presented with infection-induced lesions. My data build on this study, so the fact that I observed a higher incidence of lesions is not surprising. From the beginning, my research was designed to be more detailed, using invasive (but non-destructive) tools in order to observe lesion types that could not be seen with the naked eye. As a result, my research bolsters our understanding of non-specific infection in the middle Holocene Cis-Baikal and will allow future researchers to investigate the cultures to an extent not possible previously.

My research underlines the biological and cultural differences between the two populations, and furthers our understanding of both. In particular, my research explores some risk factors for infection hitherto not discussed (e.g., swimming) and considers types of infection-induced lesions not previously observed (e.g., otitis and mastoiditis). As a result, I believe my research has achieved its goals of defining the relationships between non-specific infection and physiological stress within the sample and of exploring the cultural implications of these findings.

6.1.1. Future Research

The worth of my research can be measured not only in what it has achieved, but also in what it has revealed and highlighted as possibilities for future research. Indeed, the wide varieties of lesions documented here have provided some novel insights into the middle Holocene cultures of the Cis-Baikal that can guide future investigations. Six areas in particular are worth discussing.

First, the study of the Eustachian tube and its relationship with upper respiratory and middle ear infections has rarely been considered in the archaeological literature. Study of the musculotubal canal, the bony foramen through which the Eustachian tube passes, should not be ignored simply due to the obscurity of the area. Non-destructive examinations can be carried out involving the inferior opening of the canal and the bone within its vicinity. From my own observation, the bone anteromedial to the inferior opening of the canal often exhibits pathological pitting (see Figure 6.1). It is my belief that an analysis concerning the co-occurrence of lesions in this area and lesions indicative of otitis media would reveal much concerning the etiological relationship between nasopharyngeal infections and otitis media (Boenninghaus and Lenarz 2005; Flohr and Schultz 2009; Graham-Hodgson 1950). Similarly, a study concerning Eustachian tube length via the length of the musculotubal canal would also be enlightening concerning the high percentage of individuals with otitis within the sample and the etiological relationship found by previous researchers between the two (e.g., Dallaire et al. 2006; Mays and Holst 2006). Then comparing these findings to those of other individuals researching northern populations would help to define the relationship between Eustachian tube length and the risk and complications of developing otitis media.

Second, I could find no archaeological or clinical literature concerning the healthy (non-pathological) versus pathological appearance of the bony middle ear. Much literature exists concerning the appearance of the soft tissue within the middle ear and its response to infection (e.g., Aufderheide and Rodríguez-Martín 1998: 253; Mays and Holst 2006), but I could find no literature that could tell me what a "healthy" bony middle ear should look like and what changes are then indicative of chronic otitis media. I checked all my diagnoses of otitis media against the presence of mastoiditis as I was pooling my otitis data and I found that only a few

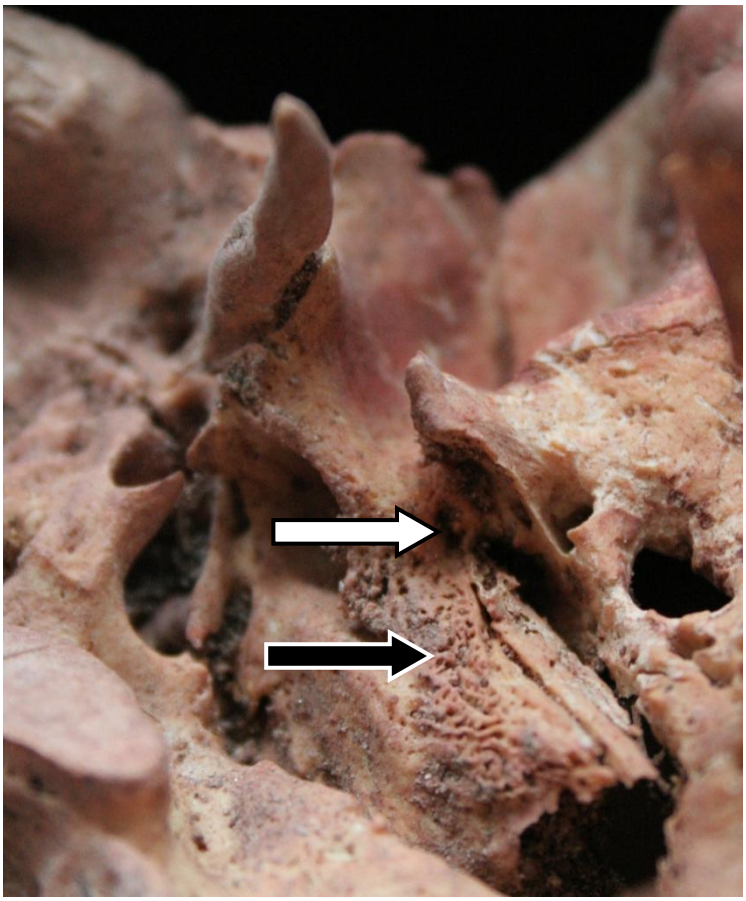

Figure 6.1: Pitting, indicated by a black arrow, antero-medial to the right musculotubal canal, indicated by a white arrow, on the basicrania of Shamanka II Individual 75.1. Barings: right styloid process visible in upper left quarter of photo and foramen magnum in bottom left corner of photo.

recorded cases of otitis media did not correspond with a diagnosis of mastoiditis. As a result, I am confident in my diagnoses of otitis media. That said, a study investigating otitis media in bone-based archaeological samples would benefit future researchers, with my own findings providing a place to start. Not only does this lack of literature represent a gap in our understanding, but it also highlights inconsistencies in terms of how paleopathologists diagnose otitis media. Such a study should either be clinical in nature, involve a modern bone collection, or, if done with archaeological specimens, should be done in conjunction with an examination of mastoiditis or chronic otitis externa in order to establish the presence of chronic otitis within individuals. Benchmarks need to be established so that paleopathologists can confidently make diagnoses. In this study, I was fortunate that the majority of my otitis media cases were

associated with either otitis externa or mastoiditis so that I could be confident in making a general diagnosis of "otitis."

Third, a study of the progression of otitis and mastoiditis, and the associated complications as observed via a visual, non-destructive examination of the basicrania of individuals within the middle Holocene populations would help us to understand how these individuals may have been affected by their conditions. Since 99.4% of my sample exhibited lesions indicative of chronic otitis, how many of these individuals then suffered from other complications such as facial paralysis, jaw pain, and hearing loss? For example, in my examination of the left middle ear of Shamanka II individual 54.1, I observed that the incus was attached to a bony tumor (see Figure 6.2). Was this anomaly associated with an infection and did it impair hearing in the left ear? These are the types of questions that I feel would lead to meaningful pictures of these individuals' lives should these complications be examined in more detail.

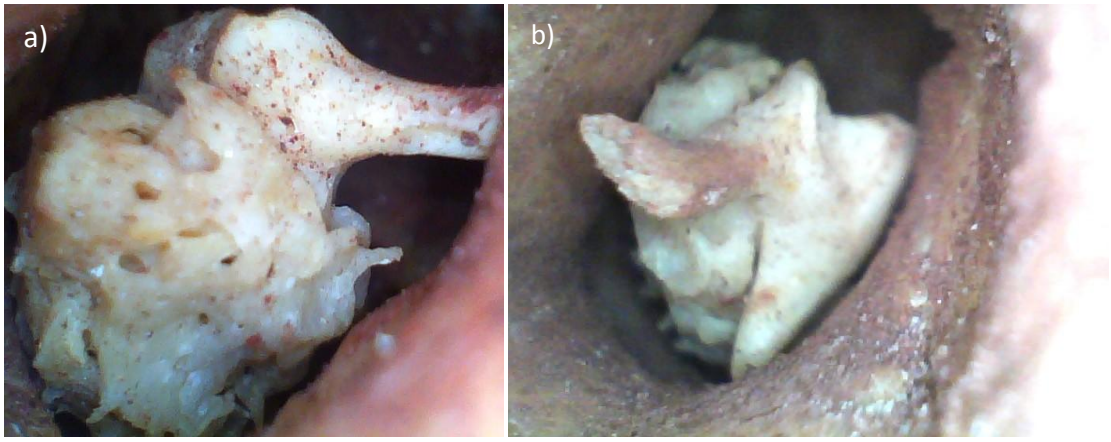

Figure 6.2: bony tumor attached to the incus of Shamanka II individual 54.1. Picture "a" shows the tumor and how it attaches to the incus, and picture "b" shows the incus.

Fourth, the large percentage of individuals with otitis-indicative lesions, and otitis externa in particular, has led me to wonder whether some of these external ear infections were brought on by swimming. The clinical literature names swimming as the leading risk factor for developing otitis externa today (Centers for Disease Control and Prevention 2014; 2015; Dorevitch et al. 2012; Piercefield 2011; Springer and Shapiro 1985). In fact, this infection is often referred to as "swimmer's ear." Though I found little evidence of external auditory

exostoses in the sample, the absence of these lesions may not signal the absence of swimming within the populations (see Section 5.1.5.0.). Rather, the middle Holocene individuals may not have been genetically prone to developing external auditory exostoses or they may have worn some form of ear protection while out in the cold and/or while swimming. In any event, we should remain open to the possibility of swimming as an occasional or even regular activity done by middle Holocene occupants of the Cis-Baikal. While the water is cold, the locals today demonstrate that swimming in Lake Baikal and its tributaries is possible and even fun.

The fifth area that is of interest requires our attention to shift from observing the ears and mastoid processes to observing the frontal and parietal bones. The etiology/ies of the observed cribra orbitalia and porotic hyperostosis (or COPH) lesions requires more in-depth attention than could be given here. An analysis of parasite load in northern hunter-fisher-gatherer populations could be done, as well as a study concerning the viability of the presence of various parasites in the region. Knowing what parasites could have lived in the region on either side of the shift in climate as well as what other, similar populations suffered from could allow us to narrow down the types of parasites that may have infected the middle Holocene populations observed here. Knowing what parasites the Kitoi and ISG may have lived with would also help us to understand what their day-to-day lives may have looked like. Would the populations have had to live in close quarters with animals for them to regularly have become infected with a parasite? Would they have suffered irritation and discomfort in the mornings because of pin worms? Humanizing information such as this can build on my presence-absence data to paint a much more life-like picture of their lives.

Sixth, and finally, more work needs to be done in order to understand where and in what kinds of dwellings the Kitoi and ISG lived. Due to our general lack of data, BHAP researchers have generally assumed that cemetery populations represent living populations and settlements (Weber and Bettinger 2010b), but much less work has been done to study or excavate the smaller cemeteries and lone burials around the Cis-Baikal or to examine the occupation sites in the region. Logistics and funding are, of course, the limiting factors. Dr. Angela Lieverse and Hugh McKenzie (MacEwan University, Edmonton) however, are endeavouring to change this. They have created a project whose focus it is to excavate and analyze some of the smaller cemeteries in the Cis-Baikal. Occupational sites, however, remain little examined and little understood, largely limiting our ability to interpret the lifeways of these hunter-fisher-gatherers. Small

cemetery sites, occupation sites, and especially dwellings should be the focus of fieldwork-based projects going forward. Where, in what, and with how many other people did the Kitoi and the ISG live? Such questions need answers. Only then can we have fruitful discussions concerning sinusitis, overcrowding, *etc.*

These six areas for future research became apparent over the course of my analysis. In the second and sixth cases, the absence of information either limited or hindered my ability to interpret the results in my discussion (Chapter 5). The other four cases intrigued me as I worked and caused me to ask further questions. It is my hope that others are as curious as I am about these questions and will venture to answer them.

6.2.0. Concluding Remarks

Seasonal episodes of severe nutritional stress (as suggested by previous researchers; Lieverse et al. 2007a; Waters-Rist 2011; Waters-Rist et al. 2011) appear to have left the Kitoi more susceptible to various forms of infection, which then placed them under further physiological stress. These seasonal episodes of stress do not appear to have been as common or pronounced among the LN–EBA ISG (Lieverse et al. 2007a; Link 1999; Temple et al. 2014; Waters-Rist 2011; Waters-Rist et al. 2011). In comparison to the Kitoi, the ISG may have been better buffered (culturally and/or biologically) from the physiological stresses associated with seasonal food shortage or may have experienced fewer shortages overall. Risk factors such as population density, un-hygienic dwelling conditions, oral hygiene, swimming, a lack of ear protection from water or cold, *etc.* (see Table 5.1 in section 5.1.6.0.) would have placed those with weaker immune systems (i.e., those who were or had been physiologically stressed) at greater risk of contracting an infection than those who had more robust immune systems (i.e., those who were not physiologically stressed). The Kitoi appear to have been both more stressed and exposed to more of these risk factors than the ISG.

This research adds much to our understanding of the cultures that existed on either side of the middle Neolithic hiatus in the Cis-Baikal: their physiological stress levels, their overall community health, and some of their activities. By better understanding each, we can better recognize how they themselves may have perceived age, sex, time, illness, health, and death. In addition, this thesis has successfully demonstrated that the presence of non-specific infection-

induced lesions reflects the presence of physiological stress within the population, and vice versa.

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APPENDIX A: Dataframe

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
4	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	P	U
6	1	B	M	SH	EN	P	A	A	P	P	P	A	U	A	P	A	U	P	P	P	P	A
7	1	C	F	SH	EN	P	A	A	P	A	A	A	A	A	A	A	P	P	P	P	A	A
8	1	C	M	SH	EN	P	A	A	P	A	U	U	U	U	U	A	P	P	P	P	P	A
10	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	P	U
11	1	B	F	SH	EN	A	A	A	P	U	U	U	U	A	U	A	P	P	P	P	A	A
11	2	C	M	SH	EN	P	U	A	A	A	U	U	U	A	U	A	P	P	P	P	P	U
12	1	C	U	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	P	A
13	1	C	F	SH	EN	P	U	A	A	A	A	A	A	A	A	A	U	A	U	U	U	U
13	2	C	M	SH	EN	A	U	A	A	U	U	U	U	A	U	A	U	P	P	P	P	U
13	3	B	F	SH	EN	P	A	A	P	P	U	A	U	A	P	A	P	P	P	P	P	A
14	1	C	M	SH	EN	P	A	A	P	A	A	A	A	A	A	A	P	P	P	P	P	A
14	2	C	F	SH	EN	P	A	A	U	P	U	U	U	U	P	A	P	P	P	P	A	A
15	1	C	M	SH	EN	P	A	A	A	P	P	P	U	A	P	A	P	P	P	P	P	A
16	1	C	U	SH	EN	P	A	A	P	U	U	A	U	U	U	A	P	P	P	P	U	U
17	1	C	M	SH	EN	P	A	A	P	P	P	P	U	U	P	A	P	P	P	P	P	A
17	2	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
18	1	C	M	SH	EN	A	A	U	U	U	U	U	U	U	U	U	U	U	P	P	U	U
19	1	C	M	SH	EN	P	A	A	P	P	P	A	A	A	P	A	P	P	P	P	A	A
20	1	C	M	SH	EN	U	U	A	P	A	U	P	U	A	P	A	P	P	U	U	P	U
20	2	C	F	SH	EN	U	U	A	P	P	U	A	A	A	P	A	P	P	P	P	P	U
20	3	C	F	SH	EN	U	U	A	A	A	A	A	A	A	A	A	P	P	P	P	U	U
21	1	C	M	SH	EN	P	A	A	P	P	A	P	P	U	P	A	U	P	U	P	P	P
21	2	C	M	SH	EN	P	A	A	P	P	P	A	A	A	P	A	P	P	P	P	A	A
21	3	B	U	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	A	A
22	1	C	M	SH	EN	P	A	A	P	P	U	U	P	A	P	A	A	P	U	P	P	A
23	1	C	M	SH	EN	P	A	A	A	A	A	A	A	A	A	A	U	A	U	U	P	U
23	2	C	F	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
23	3	C	U	SH	EN	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
23	4	C	U	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
23	5	C	U	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
24	1	C	M	SH	EN	P	A	U	U	U	U	U	U	A	U	A	U	P	P	P	P	A
24	2	B	U	SH	EN	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
25	1	C	F	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
25	2	B	U	SH	EN	A	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
25	3	C	F	SH	EN	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
26	1	C	F	SH	EN	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
26	2	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
26	3	B	U	SH	EN	P	A	A	A	A	U	U	U	U	U	A	U	P	U	P	A	A
26	5	B	U	SH	EN	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
27	1	C	M	SH	EN	P	A	A	A	A	A	A	A	A	A	A	U	A	U	U	U	U
27	2	C	M	SH	EN	A	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
27	3	A	U	SH	EN	U	U	A	A	U	U	U	U	A	U	A	U	P	A	P	U	U
27	4	A	U	SH	EN	A	U	U	A	U	U	U	U	U	U	A	U	P	P	P	U	U
28	1	A	U	SH	EN	P	A	A	A	A	U	A	U	A	A	A	U	P	U	P	U	U
29	1	C	M	SH	EN	P	A	A	P	P	P	P	U	U	P	A	P	P	U	P	P	A
30	1	C	M	SH	EN	P	A	A	P	P	P	U	A	U	P	A	P	P	U	P	P	A
31	1	B	U	SH	EN	P	A	A	P	A	A	A	U	U	A	A	U	P	P	P	U	U
32	1	C	M	SH	EN	P	A	A	P	P	P	U	A	A	P	A	U	P	U	P	P	A
33	1	C	M	SH	EN	P	A	U	U	U	P	U	U	U	P	U	U	U	U	U	P	A
34	1	C	M	SH	EN	P	A	A	P	P	U	U	A	A	P	A	P	P	P	P	P	A
35	1	C	M	SH	EN	A	U	A	A	A	U	U	U	U	U	A	U	P	P	P	P	A
36	1	C	M	SH	EN	P	U	A	P	P	U	U	U	U	P	A	P	P	U	P	P	A
37	1	B	U	SH	EN	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
38	1	A	U	SH	EN	P	A	A	P	U	U	U	U	U	U	A	P	P	P	P	U	U
39	1	C	M	SH	EN	P	A	A	P	U	P	U	U	A	P	A	P	P	P	P	P	A
40	1	A	U	SH	EN	A	A	A	A	A	A	A	A	U	A	A	U	A	U	U	U	U
41	1	C	M	SH	EN	P	A	A	A	P	P	P	A	U	P	A	P	P	P	P	P	A
42	1	C	F	SH	EN	P	A	A	P	A	U	U	U	A	U	A	P	P	A	P	P	A
42	2	D	F	SH	EN	P	A	A	A	A	A	A	A	A	A	A	U	A	U	U	P	U
43	1	C	F	SH	EN	P	A	U	U	U	U	U	U	U	U	A	U	P	U	P	U	U
44	1	D	M	SH	EN	A	A	A	A	A	A	A	A	A	A	A	P	P	P	P	P	U
44	2	C	U	SH	EN	P	U	A	P	P	A	A	A	A	P	P	P	P	U	P	U	U
45	1	C	M	SH	EN	P	A	A	P	U	A	A	A	A	A	A	P	P	U	P	A	A
46	1	C	M	SH	EN	P	A	A	A	A	A	A	A	A	A	A	P	A	P	P	P	A
47	1	C	F	SH	EN	P	A	A	A	U	U	U	A	A	U	A	P	A	P	P	A	A
48	1	D	M	SH	EN	P	A	A	P	A	A	A	A	A	A	A	P	P	U	P	P	A
48	2	A	U	SH	EN	A	A	A	A	A	A	A	A	A	A	A	U	A	U	U	U	U
48	4	C	U	SH	EN	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
48	5	C	U	SH	EN	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
49	1	B	M	SH	EN	P	A	A	P	P	P	A	A	A	P	A	P	P	P	P	A	A
50	1	C	M	SH	EN	P	A	A	A	A	U	U	U	A	U	A	P	P	P	P	P	A

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
50	2	C	M	SH	EN	P	A	A	P	A	A	A	A	A	A	A	P	A	P	P	P	A
50	3	C	M	SH	EN	P	A	A	A	A	A	A	A	A	A	A	U	A	U	U	U	U
51	1	C	M	SH	EN	P	A	A	P	A	A	A	A	A	A	A	U	P	P	P	P	A
52	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	P	P	U	P	U	U
52	2	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
53	1	C	M	SH	EN	P	A	A	P	A	A	P	U	A	P	A	P	P	P	P	P	A
53	2	D	M	SH	EN	P	A	A	P	P	A	A	U	A	P	A	P	P	P	P	P	U
54	1	B	F	SH	EN	P	A	A	P	P	P	P	A	A	P	A	P	P	P	P	A	A
55	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
55	2	B	U	SH	EN	P	A	U	U	A	U	U	U	U	U	U	U	P	U	P	A	A
56	1	B	U	SH	EN	P	A	A	P	A	P	U	A	U	P	A	P	P	P	P	U	U
56	2	B	U	SH	EN	P	P	A	A	A	U	U	U	A	U	U	U	U	U	U	A	A
57	1	C	F	SH	EN	P	U	A	A	A	A	A	A	A	A	A	U	A	U	U	U	U
57	2	C	F	SH	EN	P	U	A	P	P	U	U	A	A	P	A	P	P	P	P	P	A
58	1	C	M	SH	EN	P	A	A	P	P	P	A	A	A	P	A	P	P	U	P	P	P
59	1	C	M	SH	EN	P	A	U	U	P	U	U	P	U	P	U	U	U	U	U	U	A
59	2	B	F	SH	EN	A	A	A	A	A	A	A	A	A	A	A	U	A	U	U	A	U
60	1	D	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
60	2	C	F	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	P	A
61	1	C	F	SH	EN	P	A	A	A	P	U	P	A	A	P	A	P	P	P	P	P	A
61	2	C	M	SH	EN	P	A	A	P	P	P	P	P	A	P	A	P	P	P	P	P	A
61	3	A	U	SH	EN	U	U	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U
62	1	C	F	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
62	2	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
62	3	C	F	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
62	4	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
62	5	D	M	SH	EN	P	A	A	P	A	P	U	P	A	P	A	P	P	U	P	P	A
63	1	C	M	SH	EN	P	A	A	A	P	P	U	U	A	P	A	P	P	P	P	P	A
63	2	C	M	SH	EN	P	A	A	P	P	P	P	A	U	P	A	P	P	P	P	P	A
63	3	A	U	SH	EN	P	U	A	A	A	P	A	A	A	P	A	U	P	U	P	U	U
64	1	C	M	SH	EN	P	A	A	P	P	A	A	A	U	P	A	P	P	P	P	P	A
64	2	B	U	SH	EN	A	A	A	A	A	A	A	A	A	A	A	U	A	U	U	U	P
65	1	D	M	SH	EN	P	A	A	P	P	P	U	U	A	P	A	P	P	P	P	P	P
66	1	C	F	SH	EN	A	A	A	A	A	A	A	A	A	A	A	U	A	U	U	P	A
66	2	A	U	SH	EN	P	A	A	A	P	A	A	P	A	P	A	P	P	P	P	U	U
67	1	B	U	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	A	A
68	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
69	1	C	F	SH	EN	P	A	A	A	P	U	U	A	A	P	A	P	P	P	P	P	A
69	2	C	F	SH	EN	P	A	A	P	P	P	U	A	U	P	A	P	P	U	P	P	A
69	3	A	U	SH	EN	P	A	A	P	A	A	A	A	U	A	A	U	P	U	P	U	U
70	1	C	M	SH	EN	P	A	A	P	A	P	U	U	A	P	A	P	P	P	P	P	U
71	1	C	M	SH	EN	P	A	A	P	P	U	U	U	U	P	A	P	P	P	P	P	U
72	1	A	U	SH	EN	P	U	A	P	U	U	U	U	U	U	A	U	P	U	P	U	U
73	1	B	F	SH	EN	P	A	A	A	P	U	U	A	A	P	A	P	P	P	P	A	A
74	1	B	M	SH	EN	P	A	A	P	A	U	U	U	A	U	A	P	P	U	P	A	A
75	1	C	M	SH	EN	P	A	A	P	A	A	A	A	A	A	A	P	P	P	P	P	A
76	1	C	M	SH	EN	P	A	A	P	P	A	A	A	A	P	A	P	P	P	P	P	P
77	1	C	F	SH	EN	P	A	A	P	P	A	A	A	A	P	A	P	P	P	P	P	A
78	1	B	F	SH	EN	U	U	A	A	P	P	P	A	U	P	U	U	U	U	U	P	U
78	2	C	F	SH	EN	A	U	U	U	P	U	U	U	U	P	A	P	P	P	P	P	U
78	3	C	M	SH	EN	A	U	A	A	P	U	P	A	U	P	A	P	P	P	P	P	A
78	4	C	F	SH	EN	A	U	U	U	U	U	U	U	U	U	A	P	P	P	P	U	U
78	5	C	F	SH	EN	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
79	1	C	F	SH	EN	P	A	A	A	A	A	A	A	A	A	A	U	A	U	U	P	U
80	1	A	U	SH	EN	A	A	U	U	U	U	U	U	U	U	A	U	P	P	P	U	U
81	1	A	U	SH	EN	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
82	1	A	U	SH	EN	P	U	A	A	U	U	U	U	U	U	A	U	P	U	P	U	U
83	1	C	M	SH	EN	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
83	2	C	F	SH	EN	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
85	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	A
86	1	B	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
86	2	C	U	SH	EN	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
87	1	A	U	SH	EN	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
88	1	B	U	SH	EN	A	A	A	P	A	U	A	A	U	A	A	P	P	P	P	A	A
89	1	A	U	SH	EN	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
90	1	B	M	SH	EN	P	A	A	P	A	P	P	A	A	P	A	P	P	P	P	A	A
91	1	A	U	SH	EN	A	A	A	U	U	U	U	U	U	U	U	U	P	A	P	U	U
92	1	B	U	SH	EN	P	U	A	P	A	P	A	A	U	P	A	P	P	P	P	A	A
93	2	C	F	SH	EN	P	A	A	P	U	U	U	U	P	P	A	P	P	P	P	P	A
94	1	A	U	SH	EN	A	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
95	1	A	U	SH	EN	A	A	A	A	U	U	U	U	U	U	A	A	P	U	P	U	U
96	1	C	U	SH	EN	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
96	2	C	F	SH	EN	P	A	A	A	A	A	A	A	P	P	A	P	P	P	P	P	A
98	1	N	U	SH	EN	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
99	1	A	U	SH	EN	A	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
104	1	C	F	SH	EN	A	A	A	A	A	U	U	U	U	U	P	P	P	P	P	P	A
108	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
108	3	C	M	SH	EN	P	A	A	A	A	U	U	U	U	U	A	A	P	P	P	P	A
112	1	C	M	SH	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
L3	1	A	U	L	EN	A	A	A	A	P	U	U	U	U	P	A	U	P	U	P	U	U
L4	1	C	F	L	EN	P	A	A	P	U	U	U	U	U	U	A	P	P	U	P	P	A
L6	1	C	M	L	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
L7	1	C	F	L	EN	P	A	U	U	U	P	U	U	U	P	A	P	P	P	P	P	A
L8	1	C	M	L	EN	P	A	A	P	A	A	P	U	U	P	A	A	P	U	P	A	A
L10	1	C	M	L	EN	P	A	A	P	P	U	P	U	U	P	A	P	P	U	P	A	A
L10	2	C	M	L	EN	P	A	A	P	P	U	U	U	U	P	A	P	P	U	P	P	A
L11	1	D	M	L	EN	P	A	A	P	P	U	P	U	U	P	A	P	P	U	P	P	A
L12	1	C	F	L	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
L13	1	C	M	L	EN	P	A	A	P	U	U	P	U	U	P	U	P	U	U	P	P	A
L14	3	B	U	L	EN	P	U	A	P	A	U	A	U	U	U	A	P	P	P	P	A	A
L15	1	C	M	L	EN	P	A	A	P	U	U	A	U	U	U	A	P	P	U	P	P	A
L16	1	C	M	L	EN	P	A	A	P	A	A	A	A	U	A	A	P	P	U	P	P	A
L18	1	D	F	L	EN	P	A	A	P	U	P	A	U	U	P	A	P	P	U	P	P	U
L20	1	C	F	L	EN	P	A	A	P	P	P	A	A	U	P	A	P	P	U	P	P	A
L23	1	C	M	L	EN	P	A	A	P	P	U	U	U	U	P	A	P	P	U	P	P	A
L27	1	B	M	L	EN	A	A	A	A	A	P	A	U	U	P	A	P	P	U	P	A	A
L28	1	C	F	L	EN	P	A	A	P	P	U	A	U	U	P	A	P	P	U	P	A	A
L33	1	C	M	L	EN	P	A	A	P	A	P	P	P	U	P	A	P	P	U	P	P	A
L36	1	C	F	L	EN	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
L38	2	C	F	L	EN	P	A	A	A	P	P	P	P	U	P	A	P	P	U	P	P	A
L43	1	B	U	L	EN	P	U	A	P	U	P	U	A	U	P	A	P	P	U	P	U	U
L43	2	C	F	L	EN	P	A	A	P	U	P	A	U	U	P	A	P	P	U	P	A	A
R3	1	C	M	L	EN	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
R5	1	A	U	L	EN	A	A	A	U	U	U	U	U	U	U	A	U	P	U	P	U	U
R7	2	B	F	L	EN	P	A	U	U	U	A	U	U	U	U	U	U	U	U	U	U	U
R9	1	B	U	L	EN	A	A	A	P	A	A	U	A	U	A	A	P	P	U	P	A	A
R11	1	C	F	L	EN	P	A	A	P	P	A	A	P	U	P	A	P	P	U	P	P	A
R11	2	A	U	L	EN	U	U	A	A	U	A	U	U	U	U	U	U	U	U	U	U	U
R12	1	B	U	L	EN	A	A	A	A	U	A	P	U	U	P	A	P	A	U	P	A	A
R14	1	C	M	L	EN	P	A	A	P	A	A	A	U	U	A	A	P	P	U	P	P	A

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
R15	1	C	F	L	EN	P	U	A	P	P	A	U	U	U	P	A	U	P	U	P	A	A
1	1	C	F	UI	LE	U	U	A	A	A	A	A	A	A	A	A	A	A	A	A	U	U
3	1	B	U	UI	LE	U	U	U	A	U	U	U	U	U	U	A	A	U	P	P	U	U
3	2	A	U	UI	LE	U	U	U	U	U	U	U	U	U	U	A	U	A	P	P	U	U
4	1	A	U	UI	LE	P	A	A	A	A	A	U	U	U	U	A	P	P	P	P	U	U
5	1	B	U	UI	LE	A	A	A	A	P	A	A	A	A	P	A	P	P	A	P	A	A
6	1	C	M	UI	LE	P	A	A	A	A	A	A	U	U	A	A	P	P	P	P	P	A
7	1	C	M	UI	LE	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
8	1	B	U	UI	LE	P	U	A	P	P	A	A	U	U	P	A	P	P	U	P	U	A
9	1	B	U	UI	LE	P	U	A	A	A	A	P	P	A	P	A	P	P	P	P	A	A
10	1	B	U	UI	LE	P	U	A	A	A	A	A	A	A	A	A	P	P	P	P	A	P
11	1	C	F	UI	LE	P	U	U	U	P	U	U	U	U	P	U	P	P	U	P	P	A
12	1	C	M	UI	LE	P	A	A	P	U	U	A	U	U	U	A	P	P	U	P	P	A
14	1	B	M	UI	LE	U	U	A	U	A	P	A	U	U	P	A	P	P	U	P	P	P
15	1	B	U	UI	LE	A	A	A	A	A	A	P	U	U	P	A	U	P	P	P	U	U
16	1	C	M	UI	LE	A	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
16	2	C	M	UI	LE	P	A	A	P	P	P	P	U	U	P	A	P	P	P	P	P	P
17	1	A	U	UI	LE	U	U	A	A	U	U	U	U	U	U	A	U	P	P	P	U	U
18	1	B	U	UI	LE	U	U	A	A	U	U	U	U	A	U	A	P	P	P	P	A	P
19	1	C	M	UI	LE	P	A	A	P	P	P	A	U	U	P	A	U	P	U	P	P	A
20	1	C	M	UI	LE	A	A	A	P	U	U	U	U	U	U	A	P	P	P	P	A	A
20	2	C	F	UI	LE	U	U	A	A	U	U	U	U	A	U	A	P	P	U	P	P	A
21	1	A	U	UI	LE	U	U	A	U	A	U	A	U	U	U	A	P	P	P	P	U	U
21	2	B	U	UI	LE	U	U	A	A	U	U	A	U	A	U	A	P	A	U	P	U	A
22	1	B	F	UI	LE	P	U	A	A	U	U	U	U	A	U	A	P	P	P	P	A	A
23	1	B	U	UI	LE	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
24	1	B	F	UI	LE	P	A	A	P	P	P	P	U	A	P	A	U	P	U	P	P	A
25	1	A	U	UI	LE	A	A	A	A	P	A	A	U	U	P	A	U	P	U	P	U	U
25	2	B	U	UI	LE	A	A	A	A	P	P	A	U	U	P	A	P	P	P	P	A	A
25	3	B	U	UI	LE	P	A	A	A	A	A	P	U	U	P	A	U	P	U	P	A	A
26	1	B	U	UI	LE	A	U	A	U	A	A	A	A	A	A	A	P	P	P	P	A	A
26	2	A	U	UI	LE	P	A	A	A	U	U	U	U	U	U	A	U	P	P	P	U	A
26	3	A	U	UI	LE	P	A	A	A	A	A	A	U	U	A	A	P	P	P	P	U	U
26	4	B	U	UI	LE	P	A	A	A	A	U	U	U	U	U	A	P	P	P	P	A	A
26	5	B	U	UI	LE	P	U	A	A	A	A	A	A	U	A	A	P	P	P	P	A	A
28	1	C	U	UI	LE	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
29	1	D	M	UI	LE	P	U	A	A	A	A	A	A	U	A	A	P	P	U	P	A	A

Burial	Individual	Age Group	Pooled Sex	Cemetery	Time Period	Periostitis	Osteomyelitis	Trachoma	COPH	Sinusitis Maxilla	Sinusitis Sphenoid	Sinusitis Frontal	Sinusitis Ethmoid	Cholesteatoma	Upper Resp	Auditory Exostoses	Mastoiditis	Otitis Externa	Otitis Media	Otitis	Periodontitis	Caries
30	1	D	F	UI	LE	A	U	A	A	U	U	U	U	U	U	A	U	P	P	P	P	A
31	1	B	U	UI	LE	P	U	A	A	P	A	A	A	A	P	A	P	P	P	P	A	A
32	1	B	U	UI	LE	P	A	A	A	A	U	A	U	U	U	A	P	P	P	P	A	A
33	1	B	U	UI	LE	P	A	A	A	P	P	A	A	A	P	A	P	P	P	P	P	A
33	2	B	U	UI	LE	A	A	A	P	P	P	P	P	A	P	A	P	P	P	P	A	U
36	1	A	U	UI	LE	A	A	A	A	U	A	A	U	U	U	A	U	P	U	P	U	U
36	2	C	F	UI	LE	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
38	1	C	M	UI	LE	P	A	A	A	P	P	A	A	U	P	A	P	P	U	P	P	U
39	1	C	F	UI	LE	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
40	1	C	F	UI	LE	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
40	2	A	U	UI	LE	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
41	1	C	M	UI	LE	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
42	1	D	F	UI	LE	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
43	1	C	M	UI	LE	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
44	1	B	U	UI	LE	A	U	A	A	U	U	A	U	U	U	A	U	P	U	P	A	A
44	2	B	U	UI	LE	A	A	A	A	A	A	A	U	U	A	A	U	P	U	P	U	U
44	3	B	U	UI	LE	A	A	A	A	U	U	U	U	U	U	A	P	P	U	P	A	A
45	1	C	M	UI	LE	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	P	U
46	1	A	U	UI	LE	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
47	1	C	M	UI	LE	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
48	1	C	M	UI	LE	A	U	A	A	U	U	U	U	A	U	A	P	P	U	P	P	A
49	1	C	F	UI	LE	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
51	1	C	M	UI	LE	P	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
52	1	D	F	UI	LE	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
53	1	B	U	UI	LE	P	A	A	A	U	U	A	U	U	U	A	P	P	U	P	P	A
53	2	B	U	UI	LE	P	A	A	A	A	A	U	U	P	P	A	U	P	P	P	U	U
54	1	D	M	UI	LE	P	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
55	1	A	U	UI	LE	U	U	A	A	U	U	U	U	A	U	A	U	P	P	P	U	U
55	2	B	M	UI	LE	A	A	U	U	U	U	U	U	U	U	U	U	U	U	U	U	U
56	1	C	M	UI	LE	P	A	A	A	P	U	U	U	U	P	A	P	P	P	P	P	A
56	2	B	U	UI	LE	P	A	A	A	P	A	A	U	U	P	A	P	P	U	P	A	A

L in Burial refers to Lokomotiv burials. R in Burial refers to Lokomotiv-Raisotet burials (not considered here). Age Groups A is <4 years old. Age Group B is 5–19 years old. Age Group C is 20–49 years old. Age Group D is +50 years old. M is Male. F is Female. U is Undetermined for

Pooled Sex and Unobservable for data. SH is Shamanka II. L is Lokomotiv. UI is Ust'-Ida I. EN is Early Neolithic. LE is Late Neolithic to Early Bronze Age. P is present. A is absent. All lightly shaded columns are those that are pooled to become those darker shaded columns: Upper Respiratory Infection and Otitis.

APPENDIX B: Logistic Regression Test Results

	Entire Sample	EN	Shamanka II	Lokomotiv	LN–EBA/ Ust'-Ida I
Response Variables	**Significant Variables**				
Periostitis	Intercept	Intercept	Intercept	Intercept	Intercept
	4–19 y. o.	4–19 y. o.	4–19 y. o.	4–19 y. o.	4–19 y. o.
	20–49 y. o.	*20–49 y. o.*	*20–49 y. o.*	20–49 y. o.	20–49 y. o.
	+50 y. o.	*+50 y. o.*	+50 y. o.	+50 y. o.	+50 y. o.
	Intercept	*Intercept*	Intercept	Intercept	Intercept
	Male	*Male*	Male	Male	Male
	Intercept	*Intercept*			
	Shamanka II	Shamanka II			
	Ust'-Ida I				
	Intercept				
	EN				
Osteomyelitis	Intercept	Intercept	Intercept		
	4–19 y. o.	4–19 y. o.	4–19 y. o.		
	20–49 y. o.	20–49 y. o.	20–49 y. o.		
	+50 y. o.	+50 y. o.	+50 y. o.		
	Intercept	Intercept	Intercept		
	Male	Male	Male		
	Intercept	Intercept			
	Shamanka II	Shamanka II			
	Ust'-Ida I				
	Intercept				
	EN				
COPH	*Intercept*	*Intercept*	Intercept	Intercept	Intercept
	4–19 y. o.	*4–19 y. o.*	*4–19 y. o.*	4–19 y. o.	4–19 y. o.
	20–49 y. o.	*20–49 y. o.*	*20–49 y. o.*	20–49 y. o.	20–49 y. o.
	+50 y. o.	*+50 y. o.*	+50 y. o.	+50 y. o.	+50 y. o.
	Intercept	Intercept	Intercept	Intercept	Intercept
	Male	*Male*	Male	Male	Male
	Intercept	*Intercept*			
	Shamanka II	*Shamanka II*			
	Ust'-Ida I				
	Intercept				
	EN				
Caries	Intercept	*Intercept*	*Intercept*		Intercept
	4–19 y. o.	4–19 y. o.	4–19 y. o.		4–19 y. o.
	20–49 y. o.	20–49 y. o.	20–49 y. o.		20–49 y. o.

	Entire Sample	EN	Shamanka II	Lokomotiv	LN–EBA/ Ust'-Ida I
Response Variables	**Significant Variables**				
	+50 y. o.	+50 y. o.	+50 y. o.		+50 y. o.
	Intercept	Intercept	Intercept		Intercept
	Male	Male	Male		Male
	Intercept	Intercept			
	Shamanka II	Shamanka II			
	Ust'-Ida I				
	Intercept				
	EN				
Periodontitis	*Intercept*	*Intercept*	*Intercept*	Intercept	*Intercept*
	4–19 y. o.	4–19 y. o.	4–19 y. o.	4–19 y. o.	4–19 y. o.
	20–49 y. o.	*20–49 y. o.*	*20–49 y. o.*	20–49 y. o.	*20–49 y. o.*
	+50 y. o.	+50 y. o.	+50 y. o.	+50 y. o.	+50 y. o.
	Intercept	*Intercept*	*Intercept*	Intercept	Intercept
	Male	Male	Male	Male	Male
	Intercept	Intercept			
	Shamanka II	Shamanka II			
	Ust'-Ida I				
	Intercept				
	EN				
Upper Respiratory Infection	Intercept	Intercept	Intercept	Intercept	Intercept
	4–19 y. o.	4–19 y. o.	4–19 y. o.	4–19 y. o.	4–19 y. o.
	20–49 y. o.	20–49 y. o.	20–49 y. o.	20–49 y. o.	20–49 y. o.
	+50 y. o.	+50 y. o.	+50 y. o.	+50 y. o.	+50 y. o.
	Intercept	*Intercept*	Intercept	Intercept	Intercept
	Male	Male	Male	Male	Male
	Intercept	*Intercept*			
	Shamanka II	Shamanka II			
	Ust'-Ida I				
	Intercept				
	EN				
Otitis	Intercept				Intercept
	4–19 y. o.				4–19 y. o.
	20–49 y. o.				20–49 y. o.
	+50 y. o.				+50 y. o.
	Intercept				Intercept
	Male				Male
	Intercept				

	Entire Sample	EN	Shamanka II	Lokomotiv	LN–EBA/ Ust'-Ida I
Response Variables	**Significant Variables**				
	Shamanka II				
	Ust'-Ida I				
	Intercept				
	EN				
Auditory Exostoses	Intercept	Intercept	Intercept		
	4–19 y. o.	4–19 y. o.	4–19 y. o.		
	20–49 y. o.	20–49 y. o.	20–49 y. o.		
	+50 y. o.	+50 y. o.	+50 y. o.		
	Intercept	*Intercept*	*Intercept*		
	Male	Male	Male		
	Intercept	Intercept			
	Shamanka II	Shamanka II			
	Ust'-Ida I				
	Intercept				
	EN				

Models involving the age groups are coloured orange (listed first); those involving the sexes are coloured blue (listed second); those involving the cemeteries are coloured purple (listed third); and those involving the time periods are coloured green (listed fourth). Significant and nearly significant sub-populations are highlighted in dark grey, and significant sub-populations are italicized.

Appendix C: Images of Lesion Types

Figure C.1: active periostitis, anterior view of the right tibia, Lokomotiv, Burial 23, Individual 1.

Figure C.2: chronic periostitis, lateral view of the anterior crest on the left tibia, Shamanka II, Burial 86, Individual 2.

Figure C.3: chronic periostitis, lateral view of the left tibia, Lokomotiv, Burial 7, Individual 1.

Figure C.4: healing periostitis, medial view of left tibia, Shamanka II, Burial 52.

Figure C.5: healed periostitis, lateral view of the anterior crest of the left tibia, Lokomotiv, Burial 8, Individual 1.

Figure C.6: a) periodontitis and potential apical fistulae, inferior view of the left maxilla, Lokomotiv, Burial 38, Individual 2; b) periodontitis, inferior view of the right maxilla, Lokomotiv, Burial 3, Individual 1.

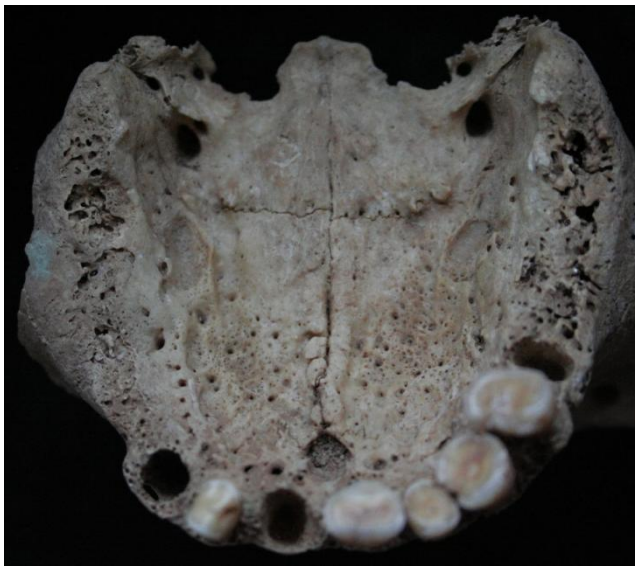

Figure C.7: periodontitis, inferiod view of the maxillae, Shamanka II, Burial 44, Individual 1.

Figure C.8: cribra orbitalia, left orbital surface of frontal bone, Lokomotiv, Burial 5, Individual 1.

Figure C.9: cribra orbitalia, anteroinferior view of the left orbial surface of the frontal bone, Ust'-Ida I, Burial 20, Individual 1.

Figure C.10: cribra orbitalia, inferior view of the orbital surface of the frontal bone, Lokomotiv, Burial 14, Individual 3.

Figure C.11: active porotic hyperostosis, superolatoral view of the right parietal bone, Shamanka II, Burial 13, Individual 3.

Figure C.12: healed porotic hyperostosis, posterolatoral view of the left parietal, Shamanka II, Burial 75, Individual 1.

Figure C.13: healed porotic hyperostosis, posterior view of the occipital, Ust'-Ida I, Burial 16, Individual 2.

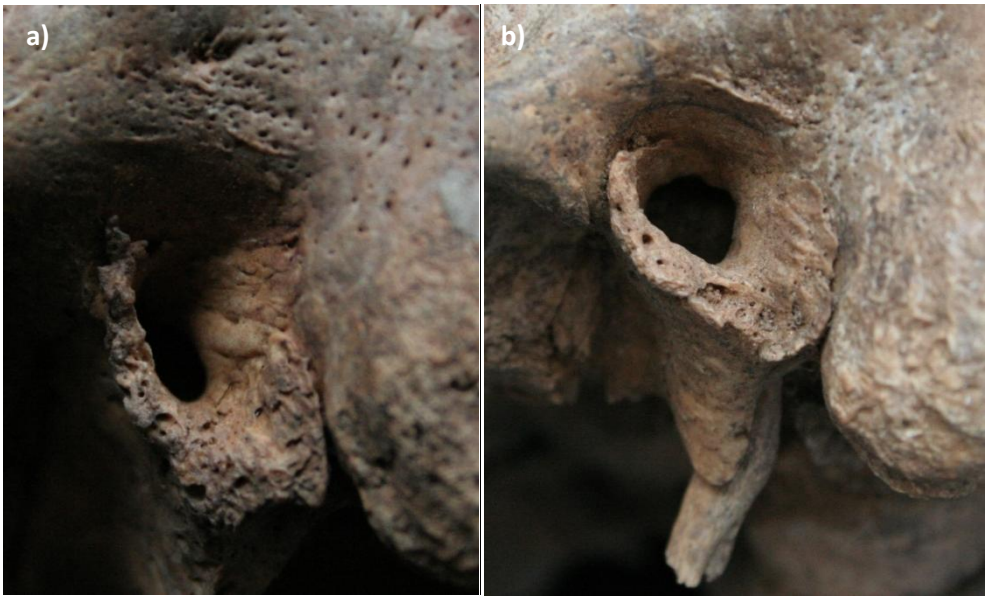

Figure C.14: a) chronic otitis externa, lateral view of the external auditory exostose of the left temporal bone, Lokomotiv, Burial 15, Individual 1; b) healing otitis externa, lateral view of the external auditory exostose of the left temporal bone, Lokomotiv, Burial 14, Individual 11.

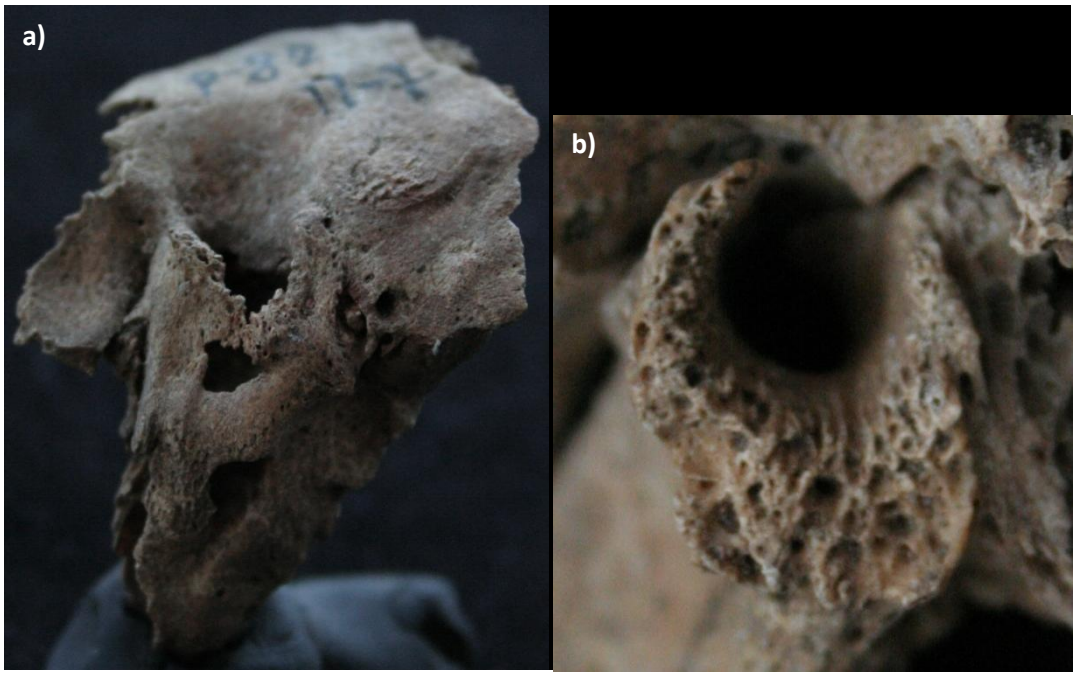

Figure C.15: a) active otitis externa, inferiolateral view of the petrous portion and external auditory exostose of the left parietal bone, Lokomotiv, Burial 5, Individual 1; b) active otitis externa, lateral view of the external auditory exostose of the left frontal bone, Shamanka II, Burial 107.

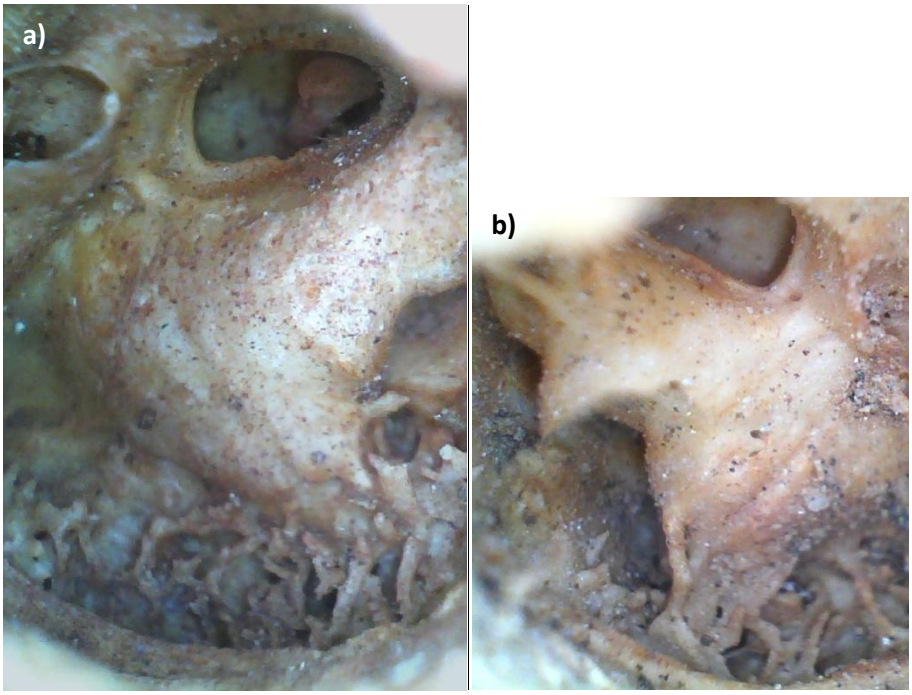

Figure C.16: a) active otitis media, three endoscopic images combined digitally, Shamanka II, Burial 14, Individual 2; b) active otitis media, two endoscopic images combined digitally, Lokomotiv, Burial 14, Individual 3.

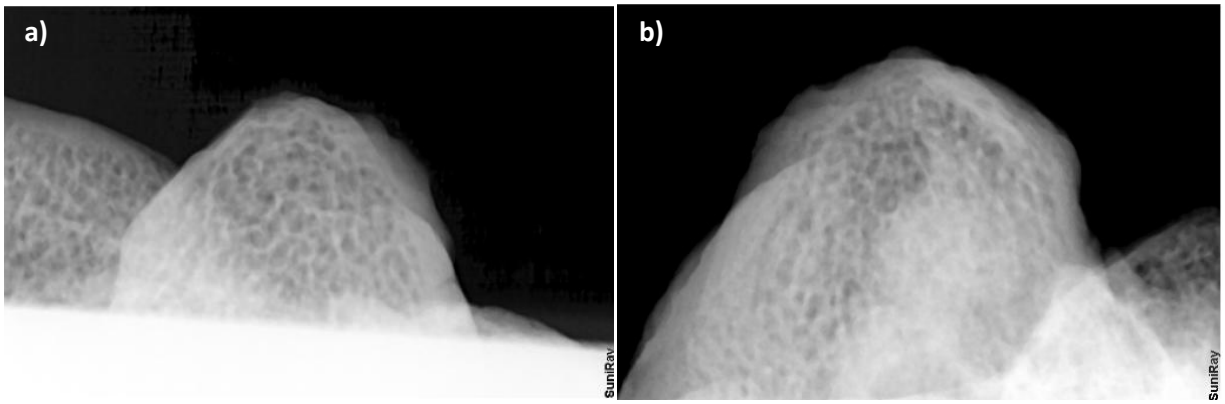

Figure C.17: a) Healthy, medial view of the mastoid of the left temporal bone, Shamanka II, Burial 24, Individual 1; b) Healthy, medial view of the mastoid of the right temporal bone, Shamanka II, Burial 48, Individual 1.

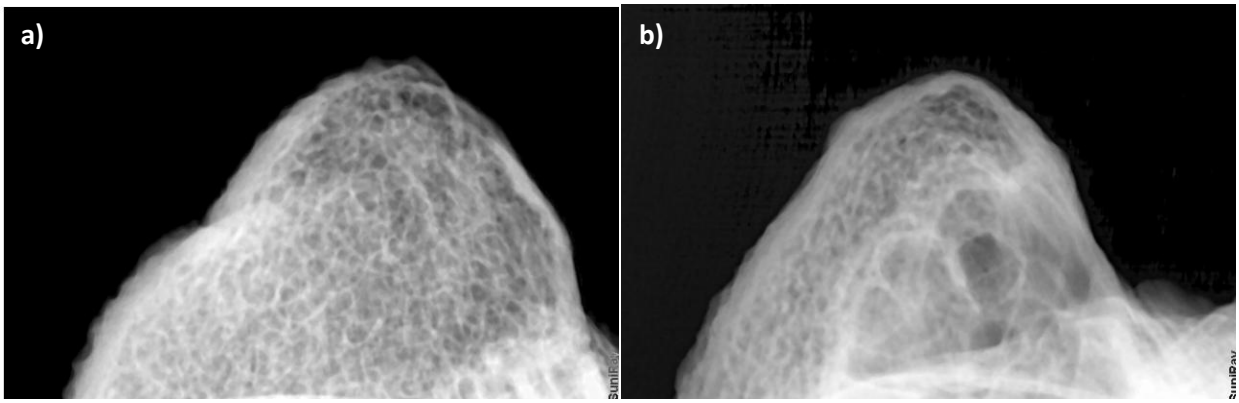

Figure C.18: Healthy, medial view of the mastoid of the right temporal bone, Shamanka II, Burial 50, Individual 2; b) Mastoiditis, medial vew of the mastoid of the right temporal bone, Shamanka II, Burial 63, Individual 1.

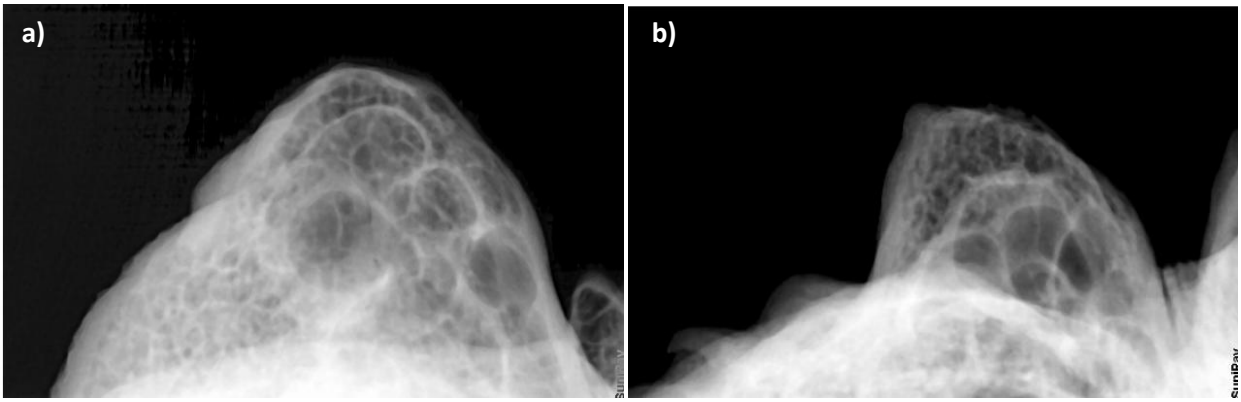

Figure C.19: a) Mastoiditis, medial view of the mastoid of the right temporal bone, Shamanka II, Burial 62, Individual 5; b) Mastoiditis, medial view of the mastoid of the left temporal bone, Ust'-Ida I, Burial 16, Individual 2.

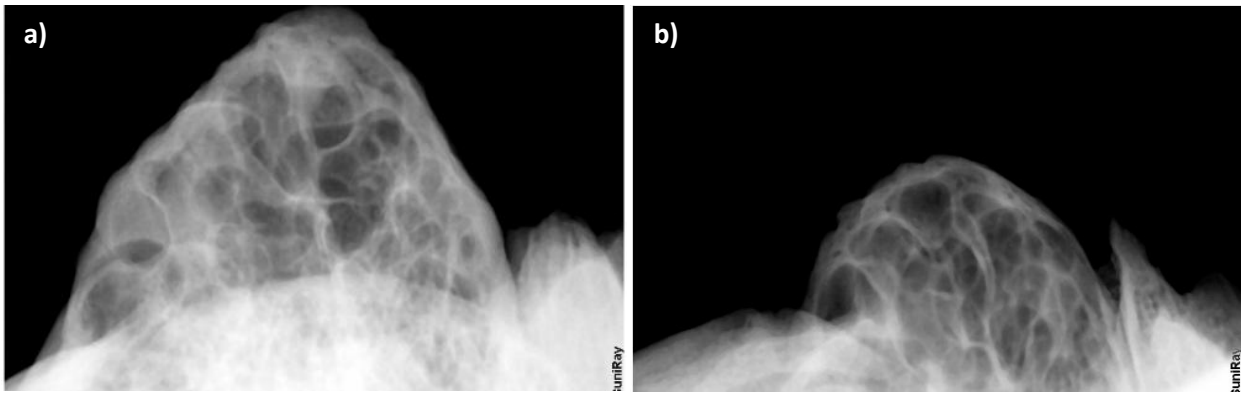

Figure C.20: a) Mastoiditis, medial view of the mastoid of the right temporal bone, Shamanka II, Burial 11, Individual 2; b) Mastoiditis, medial view of the mastoid of the right temporal bone, Shamanka II, Burial 13, Individual 3.

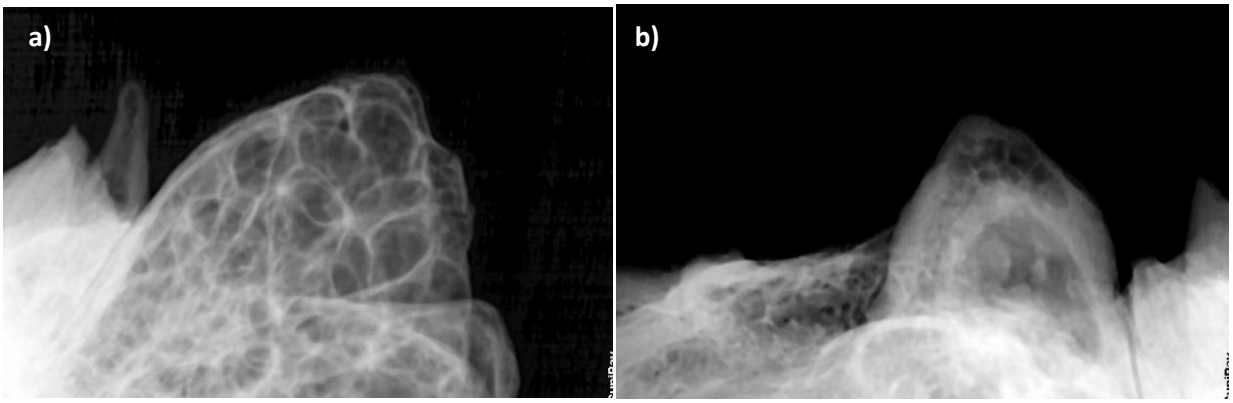

Figure C.21: a) Mastoiditis, medial view of the mastoid of the left temporal bone, Shamanka II, Burial 49, Individual 1; b) Mastoiditis, media view of the mastoid of the right temporal bone, Shamanka II, Burial 15, Individual 1.

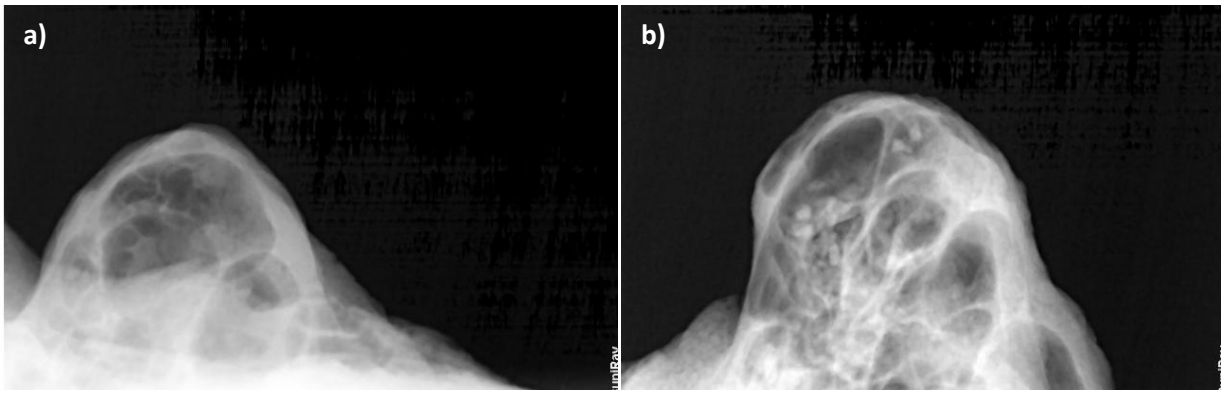

Figure C.22: a) Mastoiditis, medial view of the mastoid of the left temporal bone, Shamanka II, Burial 34, Individual 1; b) Mastoiditis, medial view of the mastoid of the left temporal bone, Shmanka II, Burial 39, Individual 1.

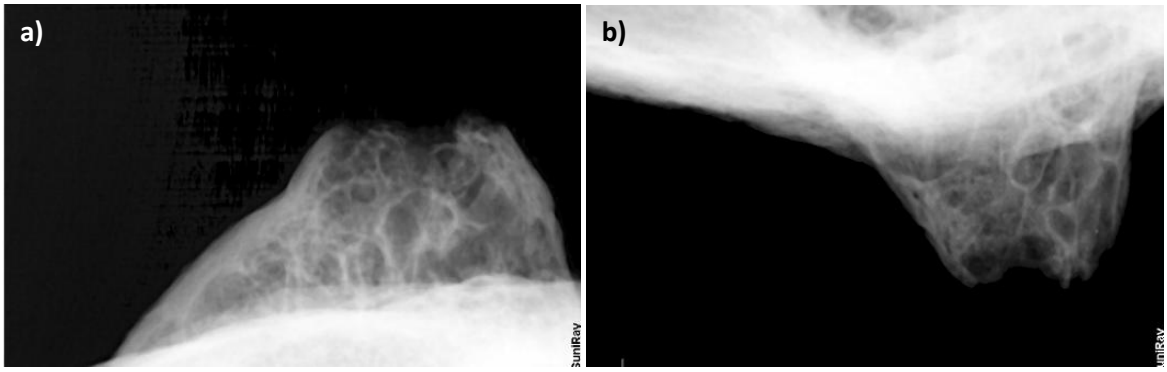

Figure C.23: a) Mastoiditis, medial vew of the mastoid of the right temporal bone, Shamanka II, Burial 20, Individual 1; b) Mastoiditis, medial view of the mastoid of the right temporal bone, Shamanka II, Burial 103, Individual 1.

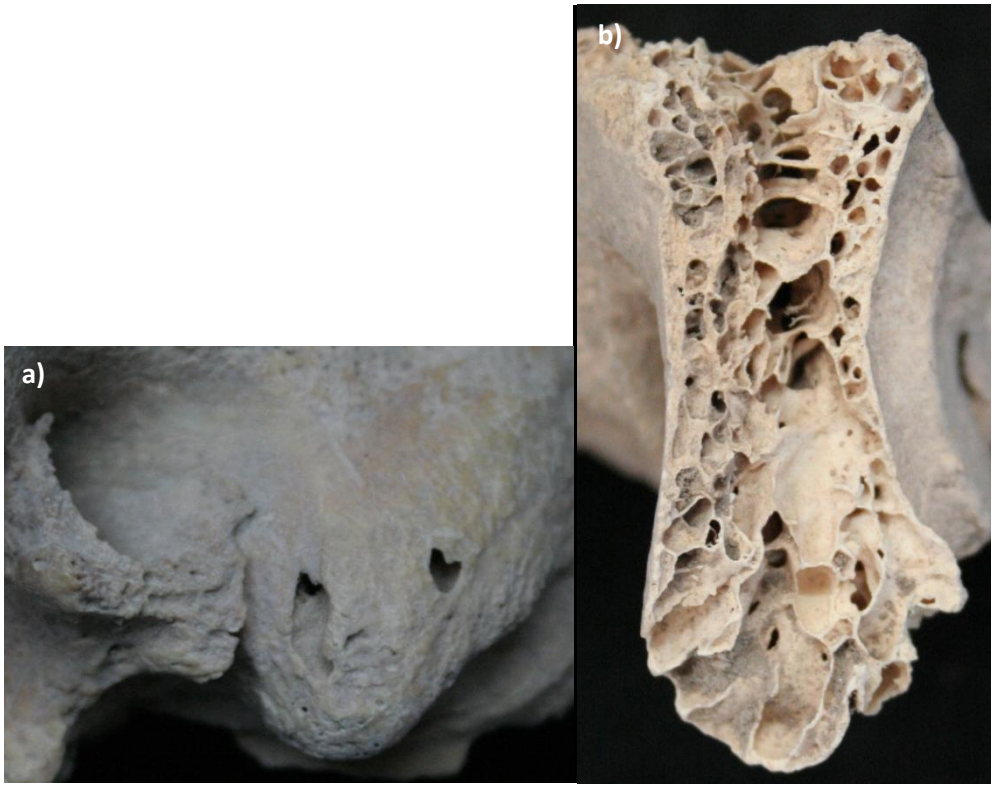

Figure C.24: mastoiditis indicated by two abscesses, anterolateral view of the external auditory meatus and mastoid of the left temporal, Ust'-Ida I, Burial 56, Individual 2; b) mastoiditis, posterior view into a fractured mastoid of the left temporal bone, Ust'-Ida I, Burial 14, Individual 1.

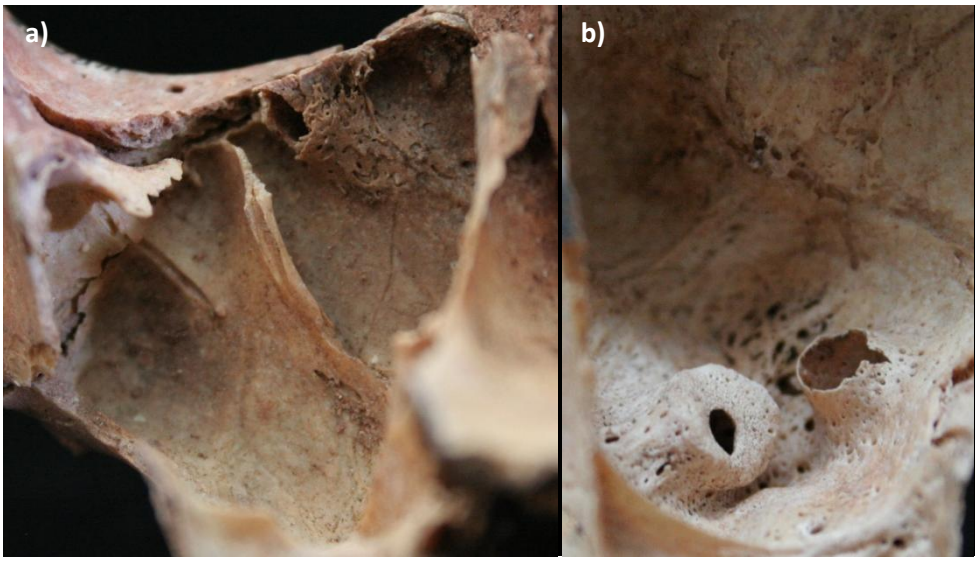

Figure C.25: a) active mastoid sinusitis, medial view into the fractured left mastoid, Shamanka II, Burial 78, Individual 1; b) active maxillary sinusitis and apical fistulae, posterior view into fractured maxillary sinus of the right maxilla, Shamanka II, Burial 32.

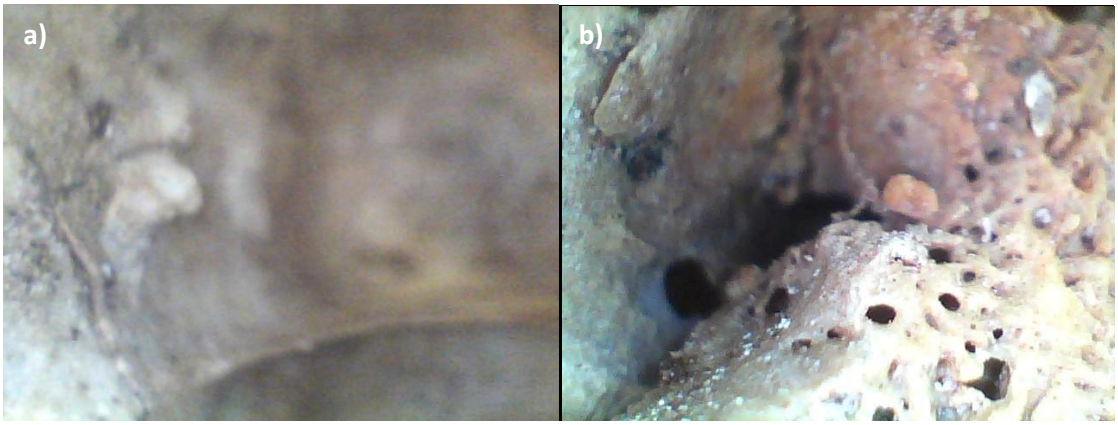

Figure C.26: a) healing maxillary sinusitis, endoscopic image taken within left maxillary sinus, Shamanka II, Burial 16, Individual 1; b) active frontal sinusitis, endoscopic image taken within the frontal sinus, Shamanka II, Burial 53, Individual 2.

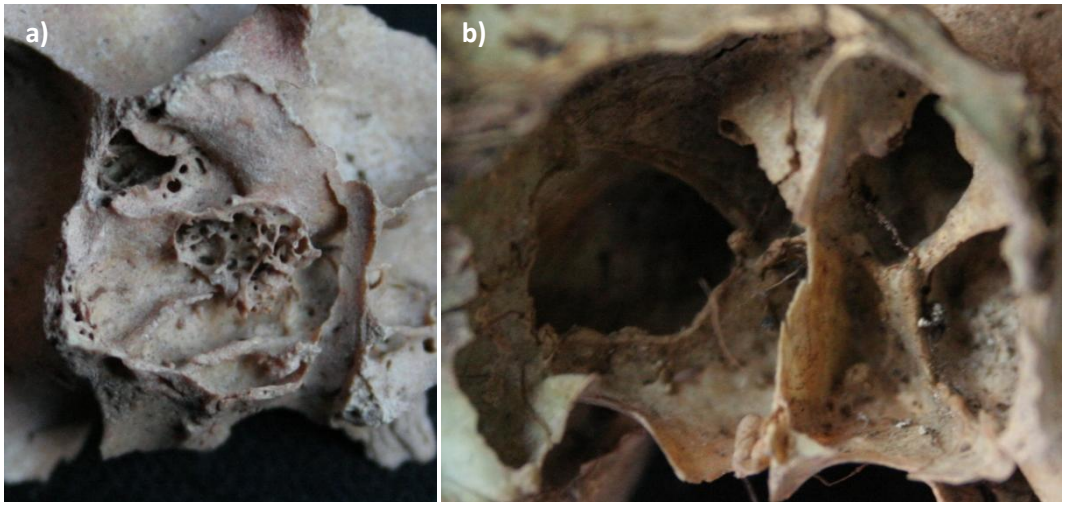

Figure C.27: a) active sphenoid sinusitis, view of left portion of the fractured sphenoid sinus, Lokomotiv, Burial 20, Individual 1; b) healing sphenoid sinusitis, posteriolateral view into the fractured sphenoid sinus, Shamanka II, Burial 111.

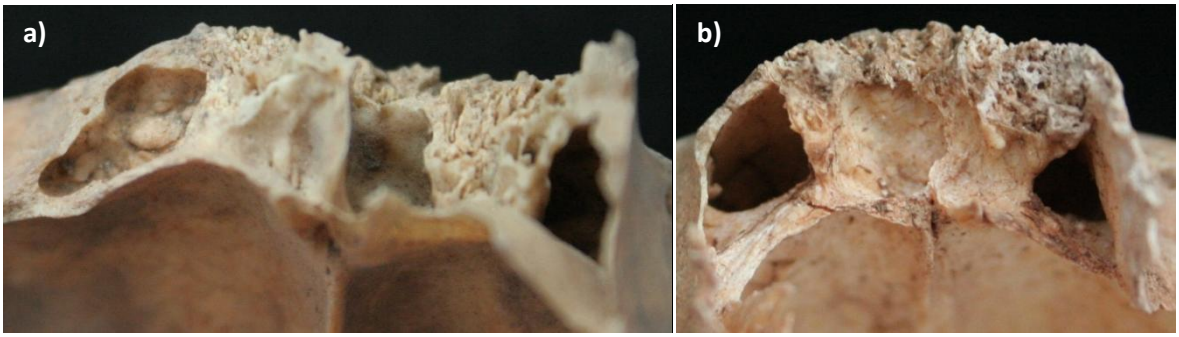

Figure C.28: a) healing frontal sinusitis, inferior view into the fractured frontal sinus of the frontal bone, Ust'-Ida I, Burial 16, Individual 2; b) healing frontal sinusitis, inferior view into the frontal sinus of the frontal bone, Shamanka II, Burial 78, Individual 3.