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Point and Shoot

A Radiographic Analysis of Mastoiditis in Archaeological Populations from England's North-East

By

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Abstract

Despite how common, severe, and impactful mastoiditis is, it is understudied archaeologically. When it is, it requires destructive methods or access to large imaging equipment. This project developed and tested a new method of imaging the mastoid process using a hand-held X-ray system and diagnosing mastoiditis in human skeletal remains. The method was grounded in modern clinical practices, non-destructive, and accessible.

Adult individuals from two archaeological populations were studied: the Anglo-Saxon/Saxo-Norman population from Black Gate, Newcastle-upon-Tyne (7th–12th centuries CE) (n=263) and the Industrial Period population from St. Hilda's Church, Coronation Street, South Shields (*c.* 1750–1855 CE) (n=123). A preliminary analysis of Black Gate adults and non-adults (n=40) developed and refined the method. The prevalence of maxillary sinusitis (MS) and lower respiratory infection (LRI) was also studied.

A slight increase in adult mastoiditis amongst young adults (8/15, 53.3%; 8/13, 61.5%) compared to other age groups (0.0%–50.0%), and a significant difference in MS between senior adults (8/35, 22.9%) compared to young (8/13, 61.5%) and mature adults (19/40, 47.5%), likely reflected age-specific exposure to risk factors in the Black Gate population. Additionally, significant differences in the prevalence of LRI amongst those buried in plain (12/152, 7.9%) and coffin graves (7/28, 25.0%) may have reflected lifeway differences amongst the social classes. In the Coronation Street population, a significant difference in the prevalence of MS and LRI between females (17/22, 77.3%) and males (5/13, 38.5%) likely reflected gendered occupations and habits. In general, the Coronation Street population appeared frailer than the Black Gate population, likely reflecting the ubiquity and severity of some risk factors and the embodied effects of classism and sexism. The project achieved its aim: expanding the understanding of the epidemiology and etiology of mastoiditis and the lifeways of those living with mastoiditis in the context of public health.

Acknowledgements

CW: genocide (first paragraph)
I must first acknowledge that the discipline of archaeology has benefited from colonial research at the expense of Indigenous peoples around the globe. Particular to this project is the fact that some early mastoiditis studies used as participants Indigenous children who were stolen from their families and kept, against their will, in so-called residential "schools". The term school white-washes the horrors that took place in these institutions, for which the primary purpose was to assimilate Indigenous children into Canadian and American culture and "kill the Indian in the child." Thousands of children died in these schools. Children were murdered, tortured, abused, neglected, and malnourished. Some children died from suicide or accidentally while trying to escape. This was a genocide inflicted by the governments of Canada and the USA, at the hands of Christian missionaries and the Catholic and Protestant Churches. That the participants in these archaeological studies were held against their will, and underage, carries heavy negative ethical implications for their participation in these studies. At a minimum, they were unable to provide consent to be participants. I recognize that no one can adequately compensate for the atrocities suffered by these children, their families, their descendants, and their communities. I hope by recognizing these facts here, all those who read this thesis are forced to confront the issue, as I have been. I strongly encourage all my colleagues to donate, as I have, to a charity supported by the National Center for Truth and Reconciliation in Canada (**https://umanitoba.ca/community/giving/our-path-reconciliation-and-healing**); to read the report of the Truth and Reconciliation Commission (**https://nctr.ca/records/reports/**); to demand the Calls to Action be fulfilled (**https://ehprnh2mwo3.exactdn.com/wp-content/uploads/2021/01/Calls_to_Action_English2.pdf**); and to continue educating themselves about the legacy of residential schools and colonialism in archaeology.

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This project would not have been possible without the love and support of my parents: Dr. Georgina Purchase and Donald Purchase. They have always taught me to value education, to ask why and how, and to chase my dreams. When this dream seemed impossible to chase, they were there—always, all ways.

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This PhD is dedicated to
Dr. Emerson Coatsworth Ramsay Purchase
Grandpa, I did it!

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List of Abbreviations

AIC – Akaike information criterion

ALRI – Acute lower respiratory infection

AM – Adult mastoiditis

AOM/E – Acute otitis media/with effusion

BCE – Before Current Era

CE – Current Era

CO – Cribra orbitalia

COM/E – Chronic otitis media/with effusion

COPD – Chronic pulmonary disease

COVID-19 – Corona virus 2019

CSOM – Chronic otomastoiditis

CT – Computed tomography

DOHaD – Developmental Origins of Health and Disease Hypothesis

ENT – Ear, Nose, and Throat

GLM - General linear model

HIV – Human immunodeficiency virus

LEH – Linear enamel hypoplasia

LRI – Lower respiratory infection

LSH – Lytic and secondary hypocellularity

MAC – Mastoid air cells

MS – Maxillary sinusitis

NHS – National Health Service (United Kingdom)

OM/E – Otitis media/with effusion

PH – Porotic hyperostosis

PNS – Paranasal sinus

residual CM – Residual childhood mastoiditis

RSV – Respiratory syncytial virus

SARS – Severe acute respiratory syndrome

SARS-CoV-2 - Severe acute respiratory syndrome associated coronavirus 2

TB - Tuberculosis

UK – United Kingdom

URI – Upper respiratory infection

USA – United States of America

UNICEF – United Nations Children's Fund

VSRL – Visceral surface rib lesions

WHO – World Health Organization

Glossary of Terms

Akaike information criterion (AIC) - Demonstrates a model's predictive ability, where a lower AIC is more predictive than a larger AIC (Akaike 1974).

Atopy - A genetic predisposition to developing an allergic disease (Chadha and Chadha 2007; Hoover et al. 1997; Chang et al. 2018).

Cholesteatoma - "A mass formed by keratinizing squamous epithelium in the middle ear and/or mastoid, subepithelial connective tissue and by the progressive accumulation of keratin debris with/without surrounding inflammatory reaction" (Olszewska et al. 2015).

Cortex - Lamellar bone that surrounds the medullary cavity of all bones (White and Folkens 2005:40–6).

Dyspnea - Shortness of breath (Moore et al. 2008; Hay et al. 2017).

Empyema - Pockets of exudate buildup in the pleural cavity (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017).

Epidemiology – The incidence and distribution of disease.

Epigenetics - The study of heritable changes in gene expression (Waddington 1942).

Etiology – The cause(s) of a disease.

Fetor oris - Bad breath (Kaimenyi 1985).

Frailty - A phenotype (Fried et al. 2001:M146; Gluckman et al. 2007) that dictates an individual's current and future inability to resist stress.

Glebe - Church land invested to a vicar or their incumbent (Merriam-Webster 2021).

Haemopoietic transfer - The transportation of an organism in the blood.

Health - A multidimensional concept. It refers to "a state of complete physical, mental[,] and social well-being and not merely the absence of disease or infirmity" (WHO 1946) with biological, social, and cultural factors (Marklien et al. 2016).

Hyposmia - The loss of the sense of smell (Gaines 2010).

Mean deviance - The residual deviance divided by the degrees of freedom. The mean deviance is close to 1 in a properly dispersed model (Crawley 2013; Mendenhall et al. 2001:581–24).

Medullary cavity - The inner shaft of all bones that is composed of cancellous/trabecular bone and is filled with red or yellow bone marrow (White and Folkens 2005:40–6).

Middle ear cleft - The middle ear, MAC, and Eustachian tube.

Odontalgia - Toothache (Clark 2006).

Ostium - The entrance into a sinus from the nasal cavity (Boocock et al. 1995).

Overdispersion - More variance in the data than was accounted for by the model. This is true when the residual deviance is larger than the degrees of freedom (Crawley 2013; Mendenhall et al. 2001:581–24).

Periosteum - A multi-layered tissue that covers the cortex of all bones (save at sesamoid bones and joint surfaces). Its inner layer is embedded with osteoblasts (Dwek 2010).

Pleurisy - The infection of the pleural cavity (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017).

Pleural effusion - Fluid buildup in the pleural cavity (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017).

Pneumatization - The aeration of the bone *via* the growth of interconnected, epithelium-lined air-filled cells (Mansour et al. 2013).

Pott's disease - Lytic lesions on the anterior portions of the thoracic and lumbar vertebrae and, ultimately, the collapse (kyphosis) and fusion of said vertebrae (Ortner 2003).

Sinus - A sinus is an air-filled cavity in the human head structurally connected to the respiratory system (White and Folkens 2005:75–126).

Stress/Physiological stress - Physical, nutritional, psychological, cultural, ecological, and other intrinsic and extrinsic risk factors that place pressure on the body.

Type 1 error - The rejection of a true null hypothesis (Crawley 2013; Mendenhall et al. 2001:343–5).

Chapter 1
Introduction

1.1 Introduction

This project concerns the creation of an accessible and non-destructive methodology to diagnose mastoiditis in human skeletal remains, and its application to an osteological dataset derived from archaeological excavations to shed a unique light on past human health. Mastoiditis is the infection of the mastoid air cells (MAC), or the bony air-filled cells that extend from the middle ear and into the mastoid process of the temporal bone (Bitar et al. 1996; Donaldson et al. 1992 Isaacson 2014). Due to its structural relationship with the middle ear, mastoiditis is considered a respiratory-related disease. In the present study, mastoiditis is examined in concert with two other respiratory-related diseases to better understand the health implications of certain environmental risk factors, over time, in the north-east of England. These diseases are maxillary sinusitis (MS), or the infection of the maxillary sinus[1] (Martini et al. 2012), and lower respiratory infection (LRI), or the infection of at least one structure in the lower respiratory system. As way of context for this study, this chapter introduces trends in palaeopathological study and how this research fits therein. Next, the history of palaeopathological research concerning mastoiditis is discussed and highlights the areas that would benefit from the development of an accessible and non-

[1] A sinus is an air-filled cavity in the human head structurally connected to the respiratory system (White and Folkens 2005:75–126).

destructive method of studying mastoiditis in human skeletal remains. Finally, knowing the novelty and value of this study, the aims and objectives of this project are presented. First, to facilitate further discussion of the palaeopathological context of the present study, four key terms must be defined: health, stress, frailty, and morbidity.

Health is a multidimensional concept. It refers to "a state of complete physical, mental[,] and social well-being and not merely the absence of disease or infirmity" (WHO 1946) with biological, social, and cultural factors (Marklien et al. 2016). Mindful that inferring mental and social well-being from physical evidence is impossible, paleopathologists must seek evidence for biological responses that offer proxies for health and interpret them appropriately, either as part of a wider health experience or by seeking alternative lines of evidence that offer insights into the social and cultural aspects of health experience.

Stress—or, more specifically, physiological stress—can leave evidence of a health experience on the skeleton that can be studied and interpreted paleopathologically. Specifically, there are physical, nutritional, psychological, cultural, ecological, and other intrinsic and extrinsic risk factors that place pressure on the body. Normally, the body maintains homeostasis by adapting to these pressures *via* allostasis. Survivorship of these pressures through adaptation is the desired outcome. When a pressure requires more bodily resources than allostasis can adapt to, the pressure disrupts homeostasis and is considered stress (Klaus 2014; Marklein et al. 2016; Reitsma and McIlvaine 2014). Such disruptions can cause bony lesions, specific or non-specific to the causative stress, to form on the skeleton.

Frailty is a phenotype (Fried et al. 2001:M146; Gluckman et al. 2007) that dictates an individual's current and future inability to resist stress. Physical decline affecting allostasis results in increased frailty and increased vulnerability to stressors (Marklein et al. 2016). In this way, individual frailty is constantly changing. Frailty is independent of age (Fried et al. 2001; Walston 2004) and confounded by "[b]iological, genetic, ecological, and sociocultural factors" (Marklein et al. 2016:219). At the population level, this is called morbidity (Marklein et al. 2016).

In context, the presence of a skeletal lesion, and its state (active, healing, and healed), in an archaeological population can indicate individual survivorship and adaptation, fragility, and morbidity in response to risk factors (Klaus 2014; Marklein et al. 2016; Matzelle et al. 2012; Reitsma and McIlvaine 2014). While this does not represent individual or population-level health as a whole, it is suggestive of a specific health experience pertaining to individual and population-level adaptation to, and survivorship of, risk factors within the larger context of both individual biology and the cultural and physical environment (Temple 2018; Temple and Goodman 2014).

1.2 Approaches to Palaeopathology

The following sub-section provides discipline-specific context for this project. It presents four key approaches to palaeopathological study that have influenced the conceptualization, study, and interpretation of health in this project. These approaches are as follows: the palaeopathological interpretation of physiological stress from skeletal evidence; multidisciplinary approaches to interpreting stress; the life history approach to interpreting stress; and conceptualizing stress *via* the osteological paradox.

Interpreting Physiological Stress from Skeletal Evidence

Paleopathologists reconstruct the health of past populations through the analysis, contextualisation, and comparison of physical remains, most often, human skeletons (Kyle et al. 2020; PPA 2020). Paleopathologists diagnose disease from human skeletal remains to better understand the epidemiology and etiology of diseases throughout history, and to interpret population and individual health experiences. A wide range of specialised methodologies suited to the preservation of the remains, the specific bones and forms of lesions being examined are employed to rigorously test hypotheses. In general, the identification of disease in the skeleton is reliant on the fact that bone is a living structure in the living human body (Roberts and Manchester 2007:7–11). It contains bone-destroying (osteoclast) and bone-forming (osteoblast) cells that work in tandem to maintain healthy bone structures and to harvest the minerals stored in bone as they are needed by the rest of the body. On average, the human skeleton is fully remodelled every ten years (USDHHS 2004:16). Pathological stimuli (such as physiological stress) can trigger unusual osteoclast and/or osteoblast activity by decreasing and/or increasing the oxygen supply to (a) bone(s), respectively (Roberts and Manchester 2007:7–11). The proportion of osteoclast/-blast activity varies depending on the causative pathological stimuli resulting in a range of identifiable skeletal manifestations of disease. The bony response to both nutritional and infection-induced stress are introduced here. The former is discussed because it can create certain bony lesions that are near-/permanent, and therefore comparable in their significance to those resulting from mastoiditis. The later is introduced because it is the focus of this project. The permanence of some mastoiditis lesions is discussed in section 1.3.

Nutritional stress (a form of physiological stress) occurs when an individual does not consume, or have metabolic access to, the necessary quantities of nutrients (vitamins, minerals, fats, proteins, calories, *etcetera*) required to maintain homeostasis (Roberts and Manchester 2007:221–5). This can occur for numerous reasons, such as seasonal resource scarcity,

abstinence, parasitic infection, or metabolic disease. The human body responds differently to nutritional stress depending on which nutrient(s) is/are lacking. When it is lacking iron, for example, the marrow cavity in the bones of the skull (especially the frontal, parietals, and occipital) expand externally, to make room for additional red blood cell creation. This expansion leaves a distinctive porotic, hair-on-end-like lesion on the external lamellar bone visible in a macroscopic analysis (Aufderheide and Rodríguez-Martín 1998; Roberts and Manchester 2007:229–34). Lesions such as this, which form on the orbital surfaces of the frontal bone, are called *cribra orbitalia* (CO), while those that form on the other bones of the skull are called *porotic hyperostosis* (PH).

Examples of indicators of generic physiological stress during development are Harris lines (Roberts and Manchester 2007:75–7) and linear enamel hypoplasia (LEH) (Roberts and Manchester 2007:240–2). These indicators are caused by episodes of stress—often, nutritional stress—that divert resources away from growth to the allostatic response. As a result, growth is stunted, and lines are formed in the bone (Harris lines) and/or dental enamel (LEH) in places that correspond to the age of the individual at the time of the stress episode.

LEH and (due to its slow remodelling process) CO are near-/permanent indicators of childhood infection that remain visible on the skeleton. Consequently, they enable paleopathologists to interpret health experiences from a time other than that at which a skeletal individual died (e.g., Kyle et al. 2016; Gowland 2015; Larsen 2015; Orellana-González et al. 2020; Primeau et al. 2018, 2019; Redfern and DeWitte 2011; Roberts and Manchester 2005; Yaussy et al. 2016). For example, Orellana-González *et al.* (2020) analysed the frequency and age-of-onset of LEH in a transitional Neolithic sample from Liguria, Italy, and found that LEH presence likely reflected physiological stress triggered by a developmental disturbance around the time of weaning; and that early physiological stress had a negative correlation with age at death and a positive correlation with the total number of defects. Thus, they concluded that weaning was a stressful period in Neolithic children's lives that had critical frailty implications. Death in early childhood (1–5 years) is a WHO parameter for assessing environmental conditions in developing countries (WHO 2020b). Likewise, Orellana-González *et al.* (2020) concluded that the local ecology negatively impacted the weaning processes of individuals in Neolithic Liguria and/or that the population practiced unsuccessful survivorship strategies to compensate for stressors in their environment. In this way, LEH, and CO can inform a discussion of individual and population health over time, and the impact of early-life stress on later-life frailty.

Infection refers to the pathological invasion of the body by a bacteria, virus, fungi, parasite, or helminth (Murphy et al. 2008:41; Roberts and Manchester 2005:167). The immune system

often reacts by inflammation of the area of infection and/or by surrounding the infectious organism in exudate (Roberts and Manchester 2005:164–20). As a result, the oxygen supply to the bone(s) at the site of the immune response can be altered, triggering an osteoblast or osteoclast reaction.

There are three sites of infection that can affect bone: at the periosteum[2], cortex[3], and medullary cavity[4]. And each site reacts differently to pathological stimulation: called sub-periosteal bone formation, osteitis, and osteomyelitis, respectively. In studying these bony lesions, paleopathologists can extrapolate the causative immune response and infer what (type of) infection triggered the response. Those lesions that can be diagnosed to the level of the causative infectious organism are called specific infections; while those that can be diagnosed generically based on the lesion type(s) and the bone(s)/bony structure(s) involved are called non-specific infections. This project deals with three types of non-specific infections: mastoiditis, maxillary sinusitis (MS), and lower respiratory infection (LRI). The later two are introduced here and the former—being the primary focus on this research—is introduced in more detail in section 1.3.

There are two maxillae in the human cranium and, therefore, two maxillary sinuses. One or both of these can become infected either primarily through the inhalation of a pathological organism (Evans 1994; Gwaltney 1996; Sande and Gwaltney 2004), or secondarily through the transfer of a pathogen odontogenicly or from an upper or LRI (Boocock et al. 1995; Roberts 2007; Van de Vijer 2018). If the organism is not expelled naturally, the immune system responds by increasing mucus production in the sinus, surrounding the pathogen in exudate, and/or inflaming the sinus (Evans 1994; Gowen 1994; Norlander et al. 1994). Debris, inflamed tissues, mucus, or exudate may block the ostium[5] and stop the sinus from naturally emptying, further inflaming the sinus, and damaging the soft tissues therein. As a result, MS can appear archaeologically as bone destruction (often observed as pitting in the floor of the sinus) or bone formation (often observed as sub-periosteal bone formation on the walls and floor of the sinus) (Boocock et al. 1995).

Sinusitis is recognised paleopathologically as an indicator of environmental risk factors, such as air pollution and overcrowded housing (see Table 1.1). The reported population-level

[2] The periosteum is a multi-layered tissue that covers the cortex of all bones (save at sesamoid bones and joint surfaces). Its inner layer is embedded with osteoblasts (Dwek 2010).
[3] The cortex is lamellar bone that surrounds the medullary cavity of all bones (White and Folkens 2005:40–6).
[4] The medullary cavity is the inner shaft of all bones that is composed of cancellous/trabecular bone and is filled with red or yellow bone marrow (White and Folkens 2005:40–6).
[5] An ostium is the entrance into a sinus from the nasal cavity (Boocock et al. 1995).

prevalence of sinusitis varies in the palaeopathological literature; reflective of the disparate environments studied (e.g., the Sudan or the Netherlands) (Davies-Barrett 2018; Davies-Barrett et al. 2021b; Casna and Schrader 2021) and the populations' varying exposures to environmental risk factors. To better understand the effect that environment and activity have had on human health, Roberts (2007) encouraged further research into populations from diverse global sites to increase the breadth of knowledge concerning factors that increase a population's risk of developing sinusitis.

Table 1.1 Previous studies that analysed the prevalence of maxillary sinusitis and the populations they examined.

Work Cited	Time Period	Region/Country	Site(s)
Boocock et al. 1995	Late medieval (12th–17th centuries)	England	Chichester
Casna and Schrader 2021	Post-medieval (1500–1850CE)	Netherlands	Arnhem
Collins 2019	Medieval (872–1552CE)	Iceland	Hofstaðir, Keldudalur, Skeljastaðir, and Skiðuklaustur
Davies-Barrett 2018	Kerma Classique period (1750–1500BCE)	Sudan	4-L-2
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-88
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-100
	Meroitic and Post-Meroitic (c.300BCE–600CE)	Sudan	3-Q-33
	Post-Meroitic	Sudan	3-O-1
	Post-Meroitic	Sudan	4-M-53
	Medieval	Sudan	3-J-23
	Post-Meroitic or Early Christian periods	Sudan	3-J-18
	Neolithic	Sudan	R12

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Davies-Barrett 2018	Kerma (2500–2050BCE)	Sudan	P37
	Meroitic (c.200BCE–200CE), Post-Meroitis (c.400–700CE), and Medieval (c.800–1200BCE)	Sudan	Gabati
	Medieval	Sudan	Soba East
Davies-Barrett et al. 2021a	Late Intermediate Period (1000–1476CE)	Peru	Pachacamac
Davies-Barrett et al. 2021b	Neolithic (6000–3100BCE)	Sudan	R12
	Kerma (2500–1500BCE)	Sudan	P37
	Kerma (2500–1500BCE)	Sudan	Fourth Cataract and Kerma Classic sites
	Meroitic (300BCE–1500CE) to Post-Meroitic (350–550CE)	Sudan	Fourth Cataract and Meroitic/Post-Meroitic sites
	Meroitic (300B–1500CE) to Post-Meroitic (350–550CE)	Sudan	Kawa
	Meroitic (300B–1500CE) to Medieval (550–1500CE)	Sudan	Gabati
Davies-Barrett et al. 2021b	Medieval (550–1500CE)	Sudan	3-J-23
	Medieval (550–1500CE)	Sudan	3-J-18
	Medieval (550–1500CE)	Sudan	Soba East
Digangi and Sirianni 2017	19th century	USA	Monroe County Almshouse, New York
Geber 2016	1845–1852CE	Ireland	Kilkenny Union Workhouse
Gowland et al. 2018	c.1711–1857CE	England	Coach Lane, North Shields
	Predominantly 19th century	England	Fewstone, North Yorkshire
Gregg et al. 1981	Mid-14th century	USA	Crow Creek Site, South Dakota

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Krenz-Niedbała & Łukasik 2016a	Medieval (10th–14th centuries)	Poland	Cedynia
	Early-modern (14th–17th centuries)	Poland	Słaboszewo
Lewis 2002	950–1500CE	England	Wharram Percy
	950–1550CE	England	St. Helen-on-the-Walls
	850–1100CE	England	Raunds Furrells
	1729–1859CE	England	Christ Church, Spitalfields
Lewis et al. 1995	Late medieval (until 14th–19th centuries)	England	Wharram Percy
	Late medieval (1100–1600CE)	England	St. Helen-on-the-Walls
Merrett and Pfeiffer 2000	c.1440CE	Canada	Uxbridge Ossuary, Ontario
Panhuysen et al. 1997	Medieval (600–800CE)	Netherlands	Boschstraat, Maastricht
	Medieval (450–950CE)	Netherlands	Servaas, Maastricht
	Medieval (1250–1600CE)	Netherlands	Nunnery, Maastricht
Purchase 2016	Early Neolithic (8000–6800BP)	Russia	Shamanka II, Siberia
	Late Neolithic (6000/5800–5200BP) to Early Bronze Age (5200/5000–3400BP)	Russia	Lokomotiv and Ust'-Ida I, Siberia
Roberts 2007	1550–1675CE	USA	Hardin Village, Kentucky
	1500–1600CE	USA	Aleutian Islanders
	"Late Woodland" (800–1100CE)	USA	Bluff Mounds, Illinois
	"Archaic" (4570+/-75 years–350+/-60 years)	USA	Indian Knoll, Kentucky

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
	500–750CE	Sudan	S and R cemetery, Kulubnarti
	18th–19th centuries	England	Christchurch, Spitalfields
	Late 1500s–early 1800s CE	USA	Site in South Dakota
Schultz et al. 2007	14th century	USA	Grasshopper Pueblo, Arizona
Shapland et al. 2015	Late medieval	England	Barton-upon-Humber
	Late medieval	England	St. Mary Spital, London
	Late medieval	England	St. Oswald's Priory, Gloucester
	Late medieval	England	St. Helen-on-the-Walls and Fishergate House, York
Van de Vijver et al. 2018	Late medieval (12th–14th centuries CE)	Belgium	St. Rombout's cemetery
	Post-medieval (15th–16th centuries CE)	Belgium	St. Rombout's cemetery
	Post-medieval (17th–18th centuries CE)	Belgium	St. Rombout's cemetery

The lower respiratory system is composed of the trachea, bronchi, bronchioles, and alveoli. Some LRI create lesions on the visceral surface(s) of the rib(s) which overly the site of the inflammatory immune response (Davies-Barrett et al. 2019). There are both non-specific and specific types of LRI: such as, pneumonia and tuberculosis (TB), respectively (FIRS 2017; NHS 2018d). The differential diagnosis of specific LRI often requires the analysis of bones other than the ribs (e.g., Holloway et al. 2011). While complex, the palaeopathological study of LRI, like that of MS, lends itself well to the study of physiological stress and risk factors in archaeological samples, as it can indicate the presence of environmental risk factors for LRI (see Table 1.2).

Table 1.2 Previous studies that analysed the prevalence of lower respiratory infection and the populations they examined. Works marked with an * are cited in Roberts *et al.* (1998) and unavailable otherwise.

Work Cited	Time Period	Region/Country	Site(s)
Aufderheid et al. 2002	350BCE–500CE	Chile	AZ-75 cemetery, Azapa Valley (sample)
Boulter et al.*	Post-medieval (18th–19th centuries)	England	Newcastle Infirmary at the Fourth, Newcastle-upon-Tyne
Boylston and Addingham 1991*; Wiggins et al. 1993*	Anglo-Saxon (8th–10th centuries)	England	ACH90 and Castledyke, Barton-upon-Humber
Boylston and Roberts 1996*; Roberts 1989*	Roman (4th century CE)	England	Kempston, Bedfordshire and 76 Kingsholm, Gloucester
Chundun 1991*, 1992*	Late medieval (12th–16th centuries)	England	Chichester Medieval Hospital, Chichester and St. Giles Hospital, Brough
Cooper et al. 2016	Early medieval (610–670CE)	Switzerland	Courroux
Davies-Barrett 2018	Kerma Classique period (1750–1500BCE)	Sudan	4-L-2
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-88
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-100
	Meroitic and Post-Meroitic (c.300BCE–600CE)	Sudan	3-Q-33
	Post-Meroitic	Sudan	3-O-1
	Post-Meroitic	Sudan	4-M-53
	Medieval	Sudan	3-J-23
	Post-Meroitic or Early Christian periods	Sudan	3-J-18

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Davies-Barrett 2018 continued	Neolithic	Sudan	R12
	Kerma (2500–2050BCE)	Sudan	P37
	Meroitis (c.200BCE–200CE), Post-Meroitis (c.400–700CE) and Medieval (c.800–1200BCE)	Sudan	Gabati
	Medieval	Sudan	Soba East
Davies-Barrett et al. 2021	Late Intermediate Period (1000–1476CE)	Peru	Pachacamac
Davies-Barrett et al. 2019	Medieval (c.500–1500CE)	Sudan	3-J-23
	Meroitic and Post-Meroitic (c.300BCE–600CE)	Sudan	3-Q-33
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-2
Kelley and Micozzi 1984	20th century	USA	Hammann-Todd Osteological Collection
Lambert 2002	Prehistoric Puebloan (1075–1280CE)	USA	Ute Mountain (5MT8651, 5MT9541, 5MT9924, 5MT9942, 5MT9943, and 5MT10010), Colorado
Lewis 2016	Medieval (900–1550BCE)	England	104 urban sites (primary data, Archaeological Data Service, and (un)published reports)
Lewis 2016	Medieval (900–1550BCE)	England	47 rural sites (primary data, Archaeological Data Service, and (un)published reports)

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Mariotti et al. 2015	Late 19th–early 20th century	Italy	Certosa cemetery of Bologna
Mays et al. 2002	Medieval (10th–16th centuries)	England	Wharram Percy
Nicklisch et al. 2012	Linear Pottery Culture (5400–4800BCE)	Germany	Halberstadt, Derenburg, and Karsdorf in Saxony-Anhalt
Pedersen et al. 2019	Medieval	Denmark	Ribe
	Early-modern	Denmark	Ribe
Raff et al. 2006	Pre-colonial	USA	Schild population, Illinois
Roberts et al. 1994	1910–1940	USA	Terry Collection
Santos and Roberts 2001	1904–1936	Portugal	Coimbra Identified Skeletal Collection (sample)
Santos and Roberts 2006	1904–1936	Portugal	Coimbra Identified Skeletal Collection (sample)
Teschler-Nicola et al. 2015	Early-modern	Austria	Gars/Thunau
Van de Vijver et al. 2018	Late medieval (12th–14th centuries)	Belgium	St. Rombout's cemetery
	Late medieval (15th–16th centuries)	Belgium	St. Rombout's cemetery
	Industrial (17th–18th centuries)	Belgium	St. Rombout's cemetery
Western and Bekvalac 2020	Pre-Industrial	England	Various site in London
	Pre-Industrial	England	Various site outside London
	Industrial	England	Various sites in London
	Industrial	England	Various sites outside London

The study of MS and LRI in archaeological populations can reveal otherwise hidden facets of human health. However, larger conceptual frameworks are necessary to structure the interpretation of palaeopathological study to understand their implications for lived experience. Thus, the remainder of this section explores three approaches to palaeopathological research that influenced the study and interpretation of health and lived experience in this project.

Multidisciplinary Approaches to Palaeopathology

Working in a multidisciplinary manner has been encouraged in most academic fields (Cooke et al. 2020; Mays 2018; Snoddy et al. 2020). It reduces the precipitation of disciplinary biases and incorrect assumptions (Snoddy et al. 2020); expands skill sets; fosters innovation and self-reflection; challenges the status quo; and creates new knowledge, robust and relevant outputs, and more research possibilities and opportunities (Cooke et al. 2020). In palaeopathology, multidisciplinarity has challenged the traditional classification of disease and formed a more holistic understanding of health (Temple 2018; Temple and Goodman 2014; Van Schalik et al. 2014). For example, palaeopathology adopted the concept of physiological stress from epidemiology (Goodman et al. 1988; Klaus 2014; Raoult and Drancourt 2008); and both the expanded definition of disease that now includes any symptomatic condition that causes physiological stress (e.g., obesity) (Van Schalik et al. 2014) and the recognition that multiple diseases in combination (or comorbidities or multimorbidities) can act as a chronic condition (Charlson 1987; Tinetti et al. 2012) from clinical medicine.

Paleopathologists have been accused of having an insufficient understanding of the microscopic processes involved in the response to disease at the cell, tissue, and organ level; and, thus, they are often encouraged to work with clinicians (DeWitte and Stojanowski 2015; Mays 2018, 2012; Snoddy et al. 2020; Van Schaik et al. 2014; Wood et al. 1992). Similarly, Simon Mays (2018) encourages paleopathologists to adopt a comparative and/or biological—rather than a statistical—approach to the interpretation of health from skeletal lesions, as the statistical approach emphasises pattern matching and ignores the intersectionality of the many factors involved in bony lesion presentation. Since many of the earliest archaeologists were medical doctors (Hrdlička 1918; Mays 2018), such an approach is not new; but is, rather, being revitalised by multidisciplinary work. Today, such work benefits from the nuanced anthropological approach of the paleopathologists which centers the lived experience of individuals and populations studied.

Scientific advances of the past century can also be credited for reinvigorating interest in the palaeopathological applications of clinical and other scientific methods and theories. For example, technological advances in X-ray and CT scan hardware and software (Collins and Jónsson 2010; Mays 2012; Primeau et al. 2018, 2019), as well as studies into the roles of hormones, genetics, and molecular signaling pathways in bony lesion formation (Gosman 2012; Klaus 2014; Klein 2000; Weston 2012), allowed for an enhanced interpretation of health experiences based on lesion presence and appearance (e.g., Zhang et al. 2020).

To illustrate, the Developmental Origins of Health and Disease Hypothesis (DOHaD) (Waterland and Michels 2007) applies epigenetic[6] concepts to palaeopathological study to explain the multigenerational impacts of physiological stress on gene expression (Klaus 2014). These changes can occur even before birth (Roseboom et al. 2001; Temple 2018) and reflect the body's interactions with its environment. Since these changes are heritable, they can be expressed in subsequent generations and leave descendants well or maladapted to their environments (Gowland 2015; Klaus 2014; Temple 2018; Yaussy et al. 2016). This research has profound implications for both medical science and palaeopathology, as it redefines the implications of physiological stress to last multiple lifetimes *via* a "heritable phenotypic legacy" (Gowland 2015:536).

Also, microscopic studies into the bony reactions to stress have revealed that the state (active, healing, or healed) of a bony lesion is critical to interpreting frailty and morbidity. The state of a lesion reflects the disease process involved at the time of death (DeWitte 2014a; DeWitte and Stojanowski 2015; Mays 2012, 2018; Snoddy et al. 2020; Van Schaik et al. 2014; Wood et al. 1992); and the proportion of active *versus* inactive lesions in a sample may be indicative of the average force of the causative stressor (Sołtysiak 2015). For those affected by rheumatoid arthritis, for example, new bone formation is promoted during the final phase of inflammation and the healing process (Matzelle et al. 2012). Thus, for rheumatoid arthritis, sub-periosteal bone formation is indicative of inflammation and, more specifically, recovery and survivorship (Klaus 2014; Matzelle et al. 2012). Contrarily, other studies have found that active sub-periosteal bone formation related to infection is more common on the bones of individuals who succumbed to said infection or a concomitant stressor (DeWitte 2014a, b; DeWitte and Wood 2008; Mays et al. 2002; Novak and Šlaus 2010; Usher 2000). In these instances, active sub-periosteal bone formation is indicative of high frailty levels and morbidity. Thus, the interpretation of a sub-periosteal lesion can vary based on these causative factors. Further molecular studies are necessary to elucidate the relationships between sub-periosteal bone formation and specific diseases. Both this and the DOHaH Hypothesis are active fields of study, with clinical and archaeological contributions.

The Life History Approach to Palaeopathology

The life history approach moves beyond the realm of clinical reductionism to naturalise health experiences within the context of an individual's lived experience over the course of their entire life. Here, population health is assessed by comparing the presence of bony

[6] Epigenetics is the study of heritable changes in gene expression (Waddington 1942).

lesions (often called frailty markers in such reports) to potentially causative risk factors present in the environment (be they natural, domestic, political, or social) (Marklein et al. 2016).

Frailty markers can be divided into two types: those that are representative of adult or childhood physiological stress. Any lesion resulting from physiological stress is a frailty marker, but the most commonly researched in adult skeletons following the life history approach include active sub-periosteal lesions (e.g., Marklein et al. 2016; Redfern and DeWitte 2011; Yaussy et al. 2016); dental caries (e.g., Redfern and DeWitte 2011); osteomyelitis (e.g., Marklein et al. 2016); CO (Gowland 2015; Redfern and DeWitte 2011; Yaussy et al. 2016); PH (Redfern and DeWitte 2011); and lesions indicative of tuberculosis (e.g., Redfern and DeWitte 2011). There are four well-researched permanent lesions indicative of childhood physiological stress: LEH and CO (Kyle et al. 2016; Gowland 2015; Larsen 2015; Orellana-González et al. 2020; Primeau et al. 2018, 2019; Redfern and DeWitte 2011; Roberts and Manchester 2005; Yaussy et al. 2016), and height, as represented by limb length and/or vertebral height (Gowland 2015; Newman 2016).

Children are sensitive to stress and "embody the impacts of social processes" (Penny-Mason et al. 2014:162); and, thus, they provide insight into the environment and lived experiences of a population. The study of permanent indicators of childhood physiological stress can shed light on the risk factors present in the past environment that had an impact on frailty and morbidity; while the study of frailty markers in non-adult skeletal remains provide a sensitive indicator of the risk factors present in the environment at the time of individual death. In context, the study of childhood frailty markers allows for the interpretation of morbidity, and, ultimately, health experience in a population over time.

While the presence of frailty markers can represent resilience and increased frailty; their absence can represent good health, an absence of physiological stress, or extreme frailty (Kyle et al. 2018; Marklein et al. 2016). This is the mortality-morbidity paradox (Marklein et al. 2016), in which absence of evidence is not evidence of absence. In other words, the absence of a frailty marker may represent extreme frailty, rather than health, as the individual succumbed to the physiological stress event before their bone had time to react. The interpretation of such absence is dependent upon context.

Many studies demonstrate the benefits of the life history approach (e.g., Marklein et al. 2016; Mays 2010; Orellana-Gonzalez et al. 2020; Wilson 2014; Yaussy et al. 2016). To illustrate, we highlight some of the work of DeWitte *et al.*. A series of six publications explored the health of Londoners from the 11th to 16th centuries with special attention paid to the health impacts of the Black Death (14th century) (DeWitte 2010, 2014a,b; 2015; DeWitte and Hughes-Morey 2012; DeWitte and Wood 2008).

First, using statistical modelling, they analysed the age-at-death distributions of 13th century skeletal populations and found that the populations' health was declining, as evidenced by increased mortality rates (DeWitte 2015). Next, analysing the age and sex distribution of LEH, PH, CO, tibial sub-periosteal bone formation, and femur and tibia length using the Gompertz–Makeham and Usher models and Fisher's exact test for stature, they found that the Black Death was selective (DeWitte and Hughes-Morey 2012; DeWitte and Wood 2008). Those of short stature, especially men who experienced previous episodes of physiological stress and who were, consequently, frailer, were more likely to die of the plague than women, less frail men, and those of either biological sex of an average height (DeWitte 2010; DeWitte and Hughes-Morey 2012; DeWitte and Wood 2008). Finally, using Kaplan-Meire survival analysis (DeWitte 2014a) and transition analysis (DeWitte 2014b) to study the age distribution of sub-periosteal bone formations (here, classified as active, healed, or mixed) in pre- and post-epidemic cemeteries, they found significantly higher survivorship among those with healed lesions than those with active or no lesions (DeWitte 2014a); and significantly more lesions in the post-, rather than pre-, epidemic sample and in older, rather than younger, individuals (DeWitte 2014b). In sum, surviving episodes of physiological stress in the decades leading up to the Black Death made individuals frailer and increased their chances of dying from the epidemic. Males also had a biological disadvantage for surviving the Black Death (see also Klein 2000; Yaussy et al. 2016). In the years that followed the epidemic, Londoners were healthier, as the least-frail survived the plague. The demographics also shifted, with more women alive than men. The social, cultural, and further health implications of the latter are discussed by Lewis (2016). By employing a life history approach, DeWitte *et al.* were able to tease-out multiple types of data from multiple samples to broaden the understanding of the lived experience of 11th to 16th century Londoners and reveal how the Black Death reshaped British society.

The Osteological Paradox

The most impactful publication of the modern period was, arguably, that of Wood *et al.* (1992) (Sołtysiak 2015). The osteological paradox, as it has become known, outlined three paleopathologically overlooked problems inherent to the discipline: demographic nonstationarity, selective mortality, and hidden heterogeneity in risk. Each problem has precipitated innovative thinking and novel methods from the palaeopathological community (see also Cohen et al. 1994; DeWitte and Stojanowski 2015; Sołtysiak 2015; Wilson 2014). The remainder of this section explores these problems, how the approaches discussed above responded to them, and how this has shaped palaeopathology today.

Firstly, demographic nonstationarity refers to the fact that *age*-at-death distributions in nonstationary populations (those affected by migration and with an inconsistent age distribution or age-specific fertility, mortality, or growth rate) reflect fertility, rather than mortality, contrary to the term's connotation. Wood *et al.* (1992) deemphasised demographic nonstationarity as a problem to be addressed by paleopathologists, as it had been thoroughly researched by demographers. Instead, they highlighted the importance of studying selective mortality and hidden heterogeneity in risk. For the purposes of this chapter, the same is done here. Demographic nonstationarity has, however, been dealt with archaeologically, especially by those concerned with periods of transition (e.g., Bocquet-Appel 2011; Page et al. 2016).

Secondly, the problem of selective mortality refers to the invisible age-specific factors that influenced mortality in a skeletal population (Wood et al. 1992). Skeletal samples, regardless of size, only reveal those who died at each age, not all those at risk of death at each age. Thus, samples are biased towards those lesions that increase the risk of death at each age; and the frequency of active lesions at each age overestimates the frequency of deaths associated with the condition within the population (or proportional mortality). Proportional mortality rates are incomparable between populations, as mortality due to one cause within one population reflects the combined impact of multiple unique and population-specific individual risk factors. Thus, the comparative use of aggregate single-cause mortality data is limited to within the population at best. This phenomenon also means that modern mortality rates are not directly comparable to those of archaeological populations.

Thirdly, hidden heterogeneity in risk refers to the unknown factors that affect individual frailty and, in turn, affect population-level mortality (Wood et al.1992). These factors can be genetic, socioeconomic, microenvironmental, or temporal. As with the problem of selective mortality, the influence of unknown factors means that aggregate mortality data cannot be compared between populations if the frailty distribution within populations are unknown.

Those paleopathologists who have operationalized the problems of the osteological paradox have challenged the ways in which health is conceptualized, studied, and interpreted (e.g., Kyle et al. 2018). Both the multidisciplinary and the life history approaches have been used in this way to address the problem of hidden heterogeneity in risk and selective mortality, respectively. As illustrated in the examples above, taking a multidisciplinary approach to the study of physiological stress revealed that stress can be multi-generational (Gowland 2015; Klaus 2014; Yaussy et al. 2016); therefore, the frailty of

each individual in a population is as diverse as their ancestors' lived experiences. This revealed a layer of heterogeneity previously unknown to palaeopathology.

Additionally, the work of DeWitte *et al.* (DeWitte 2010, 2014a, b; 2015; DeWitte and Hughes-Morey 2012; DeWitte and Wood 2008) underlined the multi-generational impacts that diverse health experiences had on the frailty and survivorship of individuals living (or not) either side of the Black Death. Their work highlighted the impact of hidden heterogeneity of risk in the survivorship potential of individuals who contracted the disease and how the epidemic was selective in killing those who were male and/or the frailest. Those who survived often being female and/or the least frail, created a population with a unique morbidity profile incomparable to that of other contemporary populations.

These diverse approaches to palaeopathological study showcase the depth and breath of knowledge that can be amassed regarding experiences of health in the past. Thus, this project follows suit. It approaches the study of health from a clinical and life history perspective to both innovatively and accurately address its aim and meet the challenges raised by the Osteological Paradox.

1.3 An Introduction to the Palaeopathological Study of Mastoiditis

In this section, mastoiditis and otitis media (OM) are defined, and the strengths and limitations of the palaeopathological study of mastoiditis are introduced. These concepts are discussed in detail in Chapters 2 and 3; but they are introduced here to offer context for the project's aim and objectives.

OM is the infection of the middle ear cleft[7] (Rai 2014) with, most often today, *Streptococcus pneumoniae* or *Haemophilus influenzae* (World Health Organization 2018); and mastoiditis is the infection of the temporal bone air cells, chiefly the mastoid air cells (MAC) (Vázquez Fernandez et al. 2018). Mastoiditis is the most common complication of acute OM (Vázquez Fernandez et al. 2018; Groth et al. 2012); is diagnostic of chronic OM (Rai 2014); and can occasionally arise as a secondary infection *via* the haemopoietic transfer[8] of a pathogen from a primary infection site elsewhere in the body to the temporal bone air cells (Graham-Hodgson 1950). Both OM and mastoiditis are common, sometimes fatal, infections prevalent in children (NHS 2019b; Paul and Wilkinson 2012; Wilson et al. 2017).

Palaeopathological studies have regularly supported the clinical literature in finding that children (birth to three years) rather than adults (Homøe et al. 1994; Krenz-Niedbała and

[7] The middle ear cleft refers to the middle ear, MAC, and Eustachian tube.

[8] Haemopoietic transfer refers to the transportation of an organism in the blood.

Łukasik 2016b; Schultz et al. 2007), and males rather than females (Daniel et al. 1988; Flohr and Schultz 2009a; Flohr et al. 2009), are more likely to suffer from mastoiditis. Other palaeopathological studies have highlighted that OM can be reflective of general respiratory health due to its structural relationship, *via* the Eustachian tube, with the nasopharynx (Flohr and Schultz 2009); and those environmental factors, such as air pollution and overcrowding, are risk factors for developing mastoiditis (Lynnerup et al. 2000; Primeau et al. 2018; Titche et al. 1981; Qvist and Grøntved 2001; Zhang et al. 2020). In this way, the presence of mastoiditis in a sample can be indicative of specific biological or environmental risk factors in the past.

Significantly, the palaeopathological study of mastoiditis revealed that primary hypocellularity (caused by the stunting of temporal bone air cell growth during childhood) is *permanently* indicative of an episode of childhood mastoiditis and leaves residual evidence on the adult skeleton, called residual childhood mastoiditis (residual CM) here (Flohr et al. 2009; Primeau et al. 2018, 2019; Purchase 2016; Purchase et al. 2019; Zhang et al. 2020). Thus, residual CM—specifically, primary hypocellularity—falls within the narrow ranks of LEH, CO, and height as a permanent lesion indicative of childhood infection. Additionally, small, immature air cells cannot adequately buffer against changes in air pressure within the middle ear and, thus, primary hypocellularity increases frailty by increasing individual risk of future mastoiditis and OM (Koç et al. 2003; Sadé 1992).

As such, researchers can take a life history approach to the study of mastoiditis by examining incidences of mastoiditis as proxies for the presence of risk factors (such as air pollution and overcrowding) (Lynnerup et al. 2000; Primeau et al. 2018; Titche et al. 1981; Qvist and Grøntved 2001; Zhang et al. 2020) in the environment over the course of a lifetime; and study the frailty effects of residual CM on adult health (Primeau et al. 2018, 2019). As primary hypocellularity is permanent, its presence/absence in a population also more accurately reflects the disease burden than other infection-induced lesions, as the latter remodel over time and primary hypocellularity does not. Finally, studying archaeological cases of mastoiditis can advance the clinical understanding of mastoiditis, by revealing how the infection progresses to its ultimate conclusion in untreated individuals.

While there is much that can be learned from the palaeopathological study of mastoiditis, only a handful of bioarchaeologists have studied mastoiditis, though it has increased, in English, in the last ten years (see Table 1.3). This has been attributed to such lesions being difficult to visualise and interpret (Mays and Holst 2006; Qvist and Grøntved 2001; Zhang et al. 2020). The mastoid air cells are internal structures, encased in the temporal bone. So, to visualise them, they require destructive sectioning (Flohr and Schultz 2009a,b; Primeau et al.

2018, 2019) or specialized imaging: such as X-ray (Aufderheide and Rodríguez-Martín 1998:253; Flohr and Schultz 2009a; Homøe and Lynnerup 1991; Homøe et al. 1996; Lynnerup et al. 2000; Purchase 2016; Purchase et al. 2019) or CT (Collins and Jónsson 2010; Flohr and Schultz 2009a,b; Homøe et al. 1992; Primeau et al. 2018, 2019; Zhang et al. 2020). CT-scanning is the standard clinical tool for both the diagnosis and assessment of mastoiditis (e.g., Hindi et al. 2014; Koç et al. 2003; Park et al. 2000; Sistani et al. 2019). CT facilities have also become more common than X-ray facilities in the UK, appearing more readily in doctors' and vets' practices, dentists' offices, and universities with medical schools. For example, a Google search of X-ray and CT facilities in Sheffield, UK reveals seven compared to 17 search results, respectively (removing erroneous search results; accessed 19th November 2020). The fixed nature of both CT and X-ray systems, however, requires paleopathologists to transport human skeletal remains to facilities with a CT or X-ray system (e.g., Collins et al. 2010). Not only is access to such systems highly competitive; but such work also requires specialized technical support and inter-departmental/-institutional collaboration. Not all archaeologists are situated to facilitate this level of access or cooperation.

Table 1.3 Previous studies that analysed the prevalence of mastoiditis and the populations they examined.

Work Cited	Time Period	Region/Country	Site(s)
Casna and Schrader 2021	Post-medieval (1500–1850CE)	Netherlands	Arnhem
Collins 2019	Medieval (872–1552CE)	Iceland	Hofstaðir, Keldudalur, Skeljastaðir, and Skiðuklaustur
Dalby et al. 1993	Early Anglo-Saxon (4th–7th centuries)	England	Baldock, Kingsholm, Barton, Raunds, Brough, and Chichester
Flohr and Schultz 2009a,b	Early medieval	Germany	Dirnstein and Rhens
Floreanova et al. 2020	Natufian (12,900–10,250BCE) and Pre-pottery Neolithic (10,000–7,900BCE)	Southern Levant	Various sites
	Chalcolithic (4,500–3,300BCE)	Southern Levant	Various sites

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Floreanova et al. 2020	Roman (63BCE–324CE), Byzantine (324–638CE), and Ottoman (1517–1917CE)	Southern Levant	Various sites
Gregg and Steele 1982	Pre-colonial	USA	William H. Over Museum collection, Sully Site, Sioux in the US National Museum, and Crow from the Crow Creek Massacre site all in South Dakota
	Post-colonial	USA	William H. Over Museum collection, Sully Site, Sioux in the US National Museum, and Crow from the Crow Creek Massacre site, all in South Dakota
Gregg et al. 1981	Mid-14th century	USA	Crow Creek Site, South Dakota
Gregg et al. 1965a	Pre-colonial	USA	Sully Burial Site, South Dakota
Gregg et al. 1965b	Pre-colonial	USA	Various Indigenous peoples in South Dakota
Holzhueter et al. 1965	Pre-colonial	USA	Arikara, Sioux Historic, and Middle Plains Woodland populations in South Dakota
Homøe et al. 1996	Pre-17th century	Greenland	Inuit from the west and southeast coast of Greenland in the Greenland collections, Laboratory of Biological Anthropology, University of Copenhagen

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Homøe et al. 1996 continued	18th–19th centuries	Greenland	Inuit from the west and southeast coast of Greenland in the Greenland collections, Laboratory of Biological Anthropology, University of Copenhagen
Krenz-Niedbała and Łukasik 2016	Medieval (10th–14th centuries)	Poland	Cedynia
	Early-modern (14th–17th centuries)	Poland	Słaboszewo
Oxenham et al. 2005	Bronze and Iron Ages (6000–1700BP)	Northern Vietnam	Con Co Ngua, Quy Chu, Nui Nap, Thieu Duong, Vinh Quang, Min Duc, Duong Co, Dinh Chang, Doi Son, Chau Son, Dong Xa, Quy Chu, Nui Nap, and Dong Mom
Primeau et al. 2018	Medieval (c.1050–c.1536CE)	Denmark	Randers
	Medieval (1150–1550CE)	Denmark	Tjærby
Purchase et al. 2019	Early Neolithic (8000–6800BP)	Russia	Shamanka II, Siberia (sample)
Qvist and Grøntved 2001	Medieval (1050–1200CE)	Denmark	Nordby
	Medieval (1150–1350CE)	Denmark	Tirup
Rathbun and Mallin 1977	Pre-historic (1300–300CE)	Iran	Dinkha Tepe
Schuler-ellis 1979	1920–1970CE	USA	Terry Collection Black sample
	1920–1970CE	USA	Terry Collection white sample

(Table continued on next page)

Work Cited	Time Period	Region/Country	Site(s)
Schulter-ellis 1979 continued	c.1900CE	USA	Inuit from Riley D. Moore Collection
	400–1400CE	USA	Illinois Mound, Titterington Collection
Titche et al. 1981	Pre-colonial	USA	Grasshopper, Kinishba, Turkey Creek, and Point of Pines, all in Arizona

Paleopathologists also diagnose mastoiditis differently to clinicians, as the soft tissue indicators largely observed by clinicians (Bluestone 1998; Rae and Ronan 2018; Qvarnberg 1982) are unavailable to most paleopathologists. Thus, many of those paleopathologists who have studied mastoiditis have largely done so to develop alternative methods that can be used to diagnose and interpret mastoiditis in human skeletal remains. Those studies have focused on classifying the appearance of mastoiditis from large skeletal populations/samples (e.g., Collins and Jónsson 2010; Flohr et al. 2009; Flohr and Schultz 2009a,b; Gregg et al. 1965a,b, 1981; Purchase et al. 2019; Rathbun and Mallin 1997; Schultz et al. 2007); or been case studies, describing the appearance of the infection in one or two individuals (e.g., Zhang et al. 2020). Less often is mastoiditis study incorporated into larger projects or site reports involving the examination of multiple bony lesions to assess health experience within and between skeletal populations/samples (e.g., Casna and Schrader 2021; Collins 2019; Krenz-Niedbała and Łukasik 2016b; Primeau et al. 2018, 2019; Purchase 2016; Qvist and Grøntved 2001); as there exists no accessible, non-destructive method for the visualisation and subsequent diagnosis of mastoiditis in human skeletal remains.

1.4 Aim and Objectives

The palaeopathological study of mastoiditis holds significant potential to enhance the understanding of childhood and adult health experiences, physiological stress, frailty, and morbidity. Yet the limitations inherent in the methods used to study the disease have created a major barrier to its wider adoption as part of a standard suite of pathological lesions utilised to explore health experiences in skeletal remains. Previous studies of mastoiditis have used traditional X-ray imagery, CT scanning, or sectioning (further employing endoscopy, scanning electron microscopy, among other techniques, to examine the sectioned samples) (Aufderheide and Rodríguez-Martín 1998; Collins and Jónsson 2010; Flohr and Schultz 2009a,b; Homøe et al. 1992, 1996; Homøe and Lynnerup 1991; Lynnerup et al. 2000; Primeau

et al. 2018, 2019; Zhang et al. 2020). So, the study of mastoiditis is limited to projects either with access to such imaging facilities or willing to perform destructive testing. Consequently, mastoiditis is rarely studied. Despite the benefits of mastoiditis research being known, no project has been devoted to making the palaeopathological study of mastoiditis more accessible. Thus, there exists a niche for the development of an accessible, non-destructive method of imaging the mastoid air cells that can be performed by every paleopathologist in the average human osteology laboratory; and that produces images as diagnostic as traditional wall-mounted X-ray and CT imaging.

The development and popularisation of hand-held X-ray technology has created an opportunity to address this need. Such technology is designed to be user-friendly and caters to various skill-levels. Thus, one does not need to be a radiologist to use a hand-held system safely and effectively (KAVO 2020). When compared, portable X-ray systems have been found to be as precise as traditional wall-mounted systems (Coy et al. 1997; Hoogeveen et al. 2019; Pittayapat et al. 2010a,b). Crucially, the portability of such systems eliminates the need to transport skeletal individuals, as imaging can be performed in the osteology laboratory. Indeed, hand-held X-ray systems have already proven ideal for dental osteoarchaeology research (e.g., Tomczyk et al. 2017a,b). This is a logical use of such systems, as portable X-rays were originally designed for dental imaging in the context of forensic investigation, and veterinary and military medicine (Coy et al. 1997; Tyson et al. 2011; Pittayapat et al. 2010a,b). Further, the versatility of portable systems lends itself to other forms of osteoarchaeological investigation. However, the user is limited by the size of the sensor and where it can be placed. This precludes the study of larger bones and bony structure.

This project comes on the heels of my master's research, in which I, among other things, utilised a hand-held X-ray system to diagnose mastoiditis in human skeletal remains (Purchase 2016). This was the first time a system such as this was used in this way. The method proved to be effective, producing high quality images (Purchase et al. 2019); but further study devoted solely to the development of this method was needed before the potential of this method could be understood and the area developed.

The aim of this PhD project was to develop and test a new method of diagnosing mastoiditis in human skeletal remains that was grounded in modern clinical practices, non-destructive, and accessible; and, in doing so, to expand our understanding of the epidemiology[9] and etiology[10] of mastoiditis with reference to the frailty impact of mastoiditis and its relationships

[9] Epidemiology is the incidence and distribution of disease.
[10] Etiology is the cause(s) of disease.

with environmental risk factors. This aim was grounded in practical objectives that serve as the framework for this study:

1. To develop a non-destructive method of imaging the mastoid processes of skeletal individuals that was more accessible than wall-mounted X-ray and CT.
2. To characterize the epidemiology of mastoiditis in two temporally distinct samples of human skeletal remains to better understand who the disease affected, and the frailty impact that residual CM had on adults.
3. To characterize the etiology of mastoiditis in the populations in relation to two respiratory infections (evidenced by lesions indicative of MS and LRI), to better understand its co-occurrence with other forms of respiratory/-related disease and identify the environmental factors that may have placed individuals at risk of these infections.
4. To better understand the lifeways of those living with respiratory infections in the broader context of public health.

The project's methodology addressed each objective in turn. First, a preliminary study was carried out to develop a method of X-raying the mastoid processes with a hand-held X-ray system. This was done in consultation with clinicians, Dr. Jaydip Ray (Professor of Otology Neurotology and Consultant Otologist Neurotologist at the University of Sheffield and consultant ENT Surgeon at Sheffield Teaching Hospital) and Dr. Charles Romanowski (consultant neuroradiologists at Sheffield Teaching Hospital). This method was developed using adult individuals (16+ years), to rule-out changes in appearance related to the growth and development of the MAC as a source of diagnostic error. The radiographs were assessed in light of both the clinical and palaeopathological literature concerning mastoiditis, resulting in criteria for diagnosing adult and childhood mastoid infection.

Second, the distribution of mastoiditis in the study sample was assessed. Binomial tests for the equality of proportions and Chi-square tests for co-occurrence were performed to test for significant associations in the distribution of mastoiditis amongst groups (the grave types, biological sexes, and age at death groups) and between the populations. Additionally, the frailty effects of residual CM were assessed by comparing the proportion of individuals who were diagnosed with residual CM and adult mastoiditis.

Third, because otitis media and mastoiditis are often the result of environmental risk factors similar to those that cause or exacerbate respiratory infections; and because the middle ear cleft is anatomically attached to the nasopharynx *via* the Eustachian tube, mastoiditis was assessed alongside two respiratory infections to help to better understand which risk factors were present in the environment and assess the health of the populations.

MS and LRI were examined, as they are widely studied by paleopathologists and have standardised methods of recording that were employed here (Boocock et al. 1995; Davies-Barrett et al. 2019). The frequency of MS, LRI, and mastoiditis was compared using binomial tests for the equality of proportions; and the distributions of each infection between/among the groups and populations was tested and compared to that of mastoiditis using binomial tests for the equality of proportions and Chi-square tests for co-occurrence. In doing so, the epidemiology and etiology of mastoiditis and respiratory/-related disease were characterized and explored in the two temporally distinct populations in light of the differing risk factors present in the environments.

1.5 Conclusions

In sum, this project aimed to create and test an accessible and non-destructive method for the imaging of skeletal mastoid processes that allows for the accurate diagnosis of residual CM and adult mastoiditis. It also aimed to use the data obtained to provide novel information about the health and lived experiences of two past populations, revealing the benefits and future potential of integrating assessment of mastoiditis into the standard suite of skeletal stress markers used by paleopathologists. In the next chapter the current clinical understanding of mastoiditis is explored. This is followed by a discussion of the current archaeological understanding of mastoiditis; the methods developed during, and employed by, this project; a presentation of the results of the project; a discussion of the results in the context of the two time periods studied; and, finally, concluding remarks on the outcomes of this project.

Chapter 2
Clinical Background

2.1 Introduction

This chapter presents the current clinical understanding of mastoiditis (mastoid infection), otitis media (OM) (middle ear infection), sinusitis (sinus infection), and lower respiratory infection (LRI). Mastoiditis is the focus of this chapter, as it is for this project. Because mastoiditis is highly related to OM and OM is often discussed clinically alongside mastoiditis, OM is also a focus. Sinusitis and LRI are discussed in relation to mastoiditis and OM to highlight how studying these infections in concert can inform our understanding of risk factors present in the environment.

This discussion of clinical data is intended to inform the interpretation of the lesions observed in the human skeletal remains assessed in this thesis and facilitate informed discussion of the lifeways of the individuals being studied. This is not to say that the modern clinical understanding of a disease can be applied directly to past populations. In fact, we are warned this cannot be done (Mays 2012; Ortner 2011, 2012; Ragsdale 1997; Ragsdale and Lehmer 2012; Roberts and Manchester 2005:5–6; Schultz 2001), because the diseases we experience today may have different virulence or pathophysiologies to those in the past and involve bone in different ways (Roberts and Manchester 2005:5–6). Rather, the clinical literature is best used to inform discussions around individuals' interactions with and adaptations to the environment and the risk factors therein (Roberts and Manchester 2005:5–6); and to inform our understanding of the biological processes involved in infection and their impact on the appearance of bony lesions (Mays 2012; Ortner 2011, 2012; Ragsdale 1997;

Ragsdale and Lehmer 2012; Schultz 2001). Yet, archaeologists should not be afraid to consult the clinical literature, as it provides the most thorough explanations of, and rigorous research available into, disease. As long as archaeologists heed these warnings and use the clinical literature to contextualize their findings and not expect the clinical literature to mirror them, palaeopathological research will be the richer for it.

2.2 Mastoiditis and Otitis Media

Mastoiditis is the infection of the mastoid air cells (MAC), and OM is the infection of the middle ear (NHS 2019a). Mastoiditis is typically secondary to OM (NHS 2018b). Both are common, sometimes fatal, infections (NHS 2019a). This was the case in the past (Paul and Wilkinson 2012) and remains so today (Wilson et al. 2017). To contextualise the palaeopathological study of mastoiditis and OM, this section reviews and summarises the anatomy and development of the middle ear cleft (the middle ear, MAC, and Eustachian tube); the function of the temporal bone air cells; the clinical definition of mastoiditis and OM, including the differences between acute and chronic forms; and the debate over the pathogenesis of mastoiditis and OM.

Anatomy of the Middle Ear Cleft

The following section discusses the anatomy of the temporal bone, with particular attention paid to the air cells of the mastoid portion of the temporal bone. The relevant clinical terminology is introduced, and the anatomy of the structures are described, beginning with the temporal bone in general and then focusing on the middle ear cleft.

There are various ways to divide the temporal bone (see Allam 1969; Jadhav et al. 2014; Schuknecht 1974; Tremble 1934; Vasiliu 1968). It can be structurally divided into four portions: the squamous, petromastoid, tympanic, and styloid process (Jadhav et al. 2014). Or it can be divided into five regions based on how it pneumatizes (or how the bone develops air-filled cavities) (Mariam Webster Dictionary 2020): the middle ear, squamomastoid/mastoid, perilabyrinthine, petrous apex, and accessory (Allam 1969). The latter terms will be used here as consistently as possible to complement the discussion of the pneumatization of the temporal bone; however, there is little consensus in the literature as to the terms used (Virapongse et al. 1985), so other terms may appear as necessary (see Figures 2.1 and 2.2).

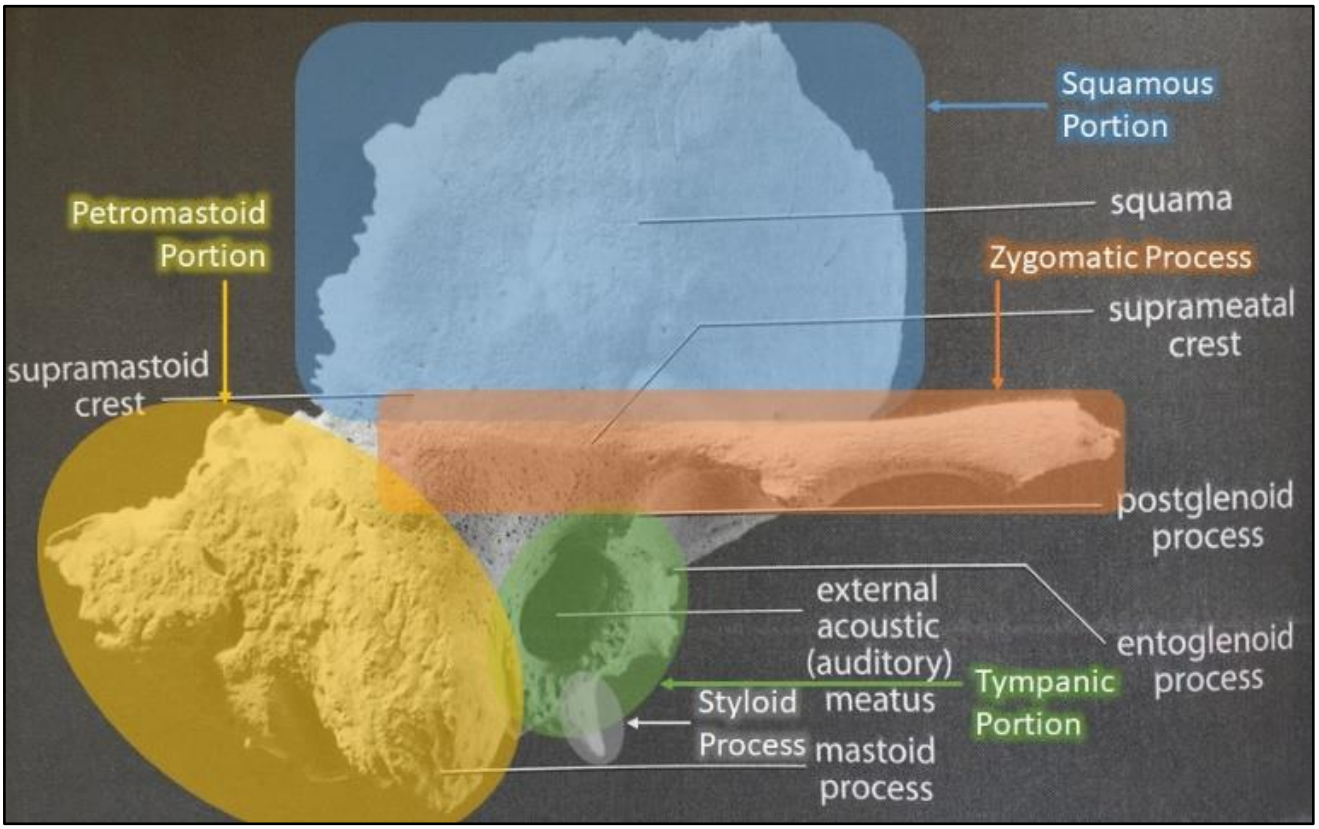


Figure 2.1 Lateral view, showing the gross anatomical features and four portions of the temporal bone (adapted from White and Folkens 2005:96).

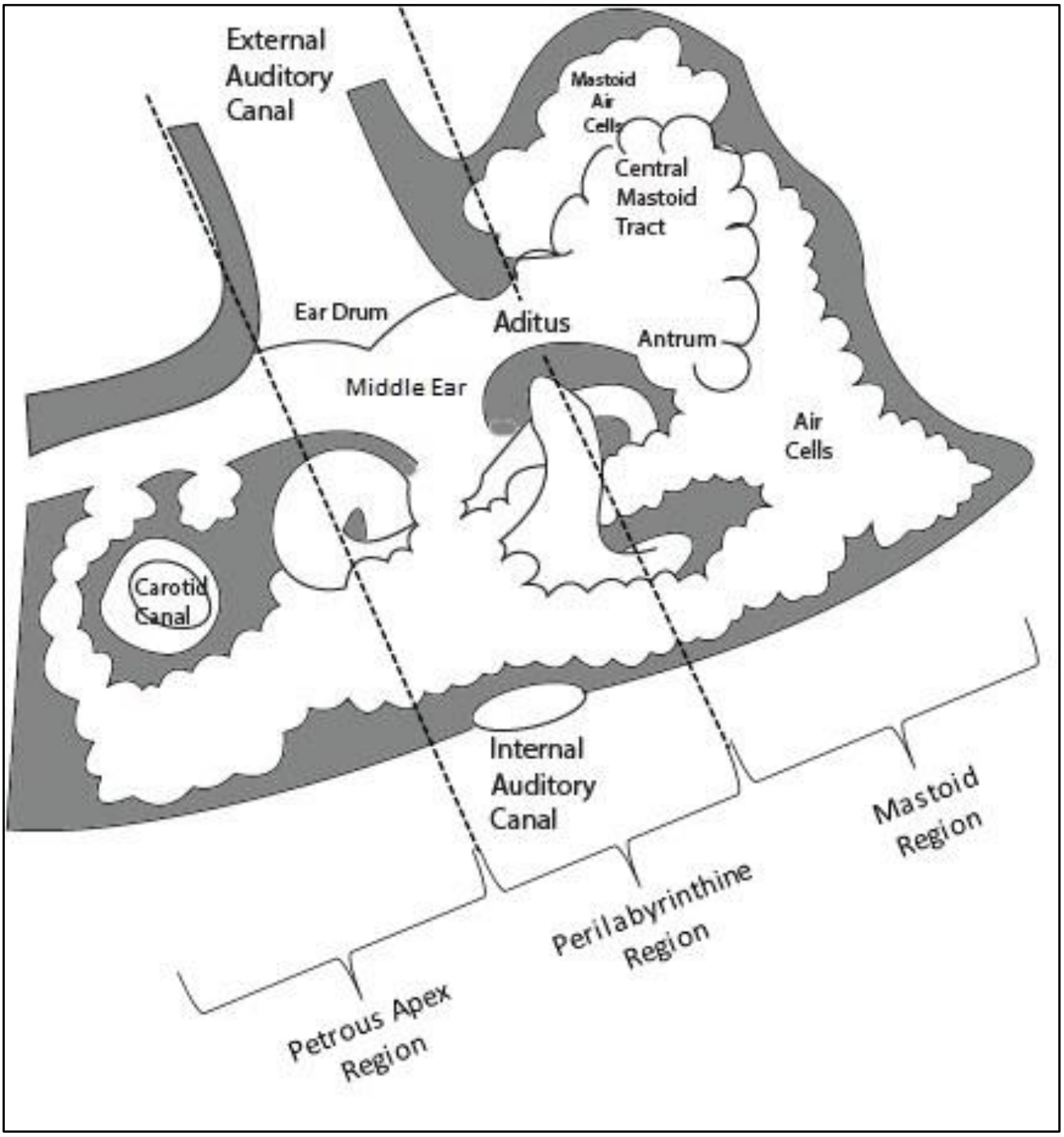


Figure 2.2 Axial view, showing anatomical features, regions in the temporal bone, and pneumatization tracts (adapted from Schuknecht 1974 in Virapongse et al. 1985:474).

The middle ear refers to the portion of the ear that contains the auditory ossicles (the malleus, incus, and stapes) and is medial to the annulus and the *pars flaccida* and *pars tensa* (or tympanic membrane or eardrum) and lateral to the cochlea and semicircular canals of the inner ear. It is composed of the epitympanum (or roof, recess, or attic) superior to the oval window and annulus, the mesotympanum (or medial wall) medially, the retrotympanum (or mastoid wall or posterior tympanum) posteriorly, the hypotympanum (or floor) inferiorly, the protympanum (or anterior wall) anteriorly, and the lateral wall laterally (see Figure 2.3) (Bitar

et al. 1996; Isaacson 2014). The terminology used to label the middle ear is problematic, as there are different terms used throughout the literature.

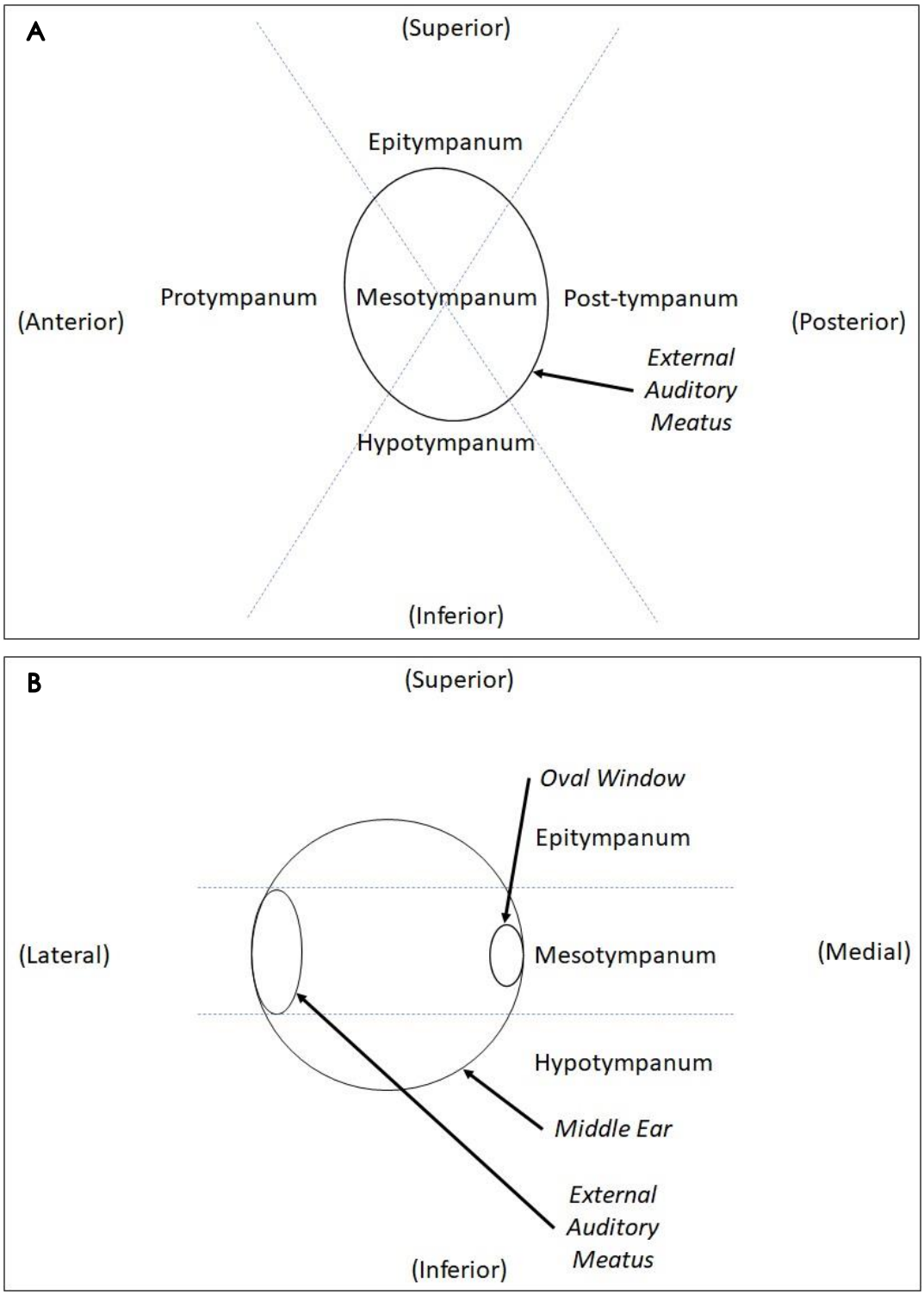


Figure 2.3 A) Sagittal view and **B)** Coronal view of an abstracted middle ear. The dashed lines indicate the theoretical boundaries between each anatomical portion. Anatomical landmarks are indicated by arrows and named by italicized labels.

The middle ear is a highly connected structure. It communicates with the inner ear indirectly *via* the oval window and with the nasopharynx directly *via* the Eustachian tube; both structures are in/open into the mesotympanum (Isaacson 2014). The facial nerve (or seventh cranial nerve) passes through the middle ear along the epitympanum, through the bone posterior to the retrotympanum, and out of the stylomastoid foramen. The tympanic nerve enters the middle ear through various openings in the mesotympanum, passes over the promontory (the convex surface in the middle of the mesotympanum), and exits the middle ear in the hypotympanum. The auditory ossicles are suspended from the walls of the middle ear by tendons; the *stapedius* muscle attaches to the stapes and the *tensor tympani* muscle attaches to the malleus. The *tensor tympani* travels through the temporal bone, superior and parallel to the Eustachian tube. All but the styloid process are normally pneumatized and these air cells are continuous (Allam 1969; Bast and Forester 1939).

The MAC communicate with the middle ear via an air cell called the *aditus* in the epitympanic recess in the superior portion of the epitympanum (Bitar et al. 1996; Donaldson et al. 1992 Isaacson 2014). The MAC are composed of the mastoid *antrum*, which communicates directly with the *aditus* and the central tract; the central tract, which is the large air cell that extends inferiorly through the mastoid; and the peripheral area, which includes the smaller air cells that communicate with and surround the mastoid *antrum* (Virapongse et al. 1985) (see Figure 2.4).

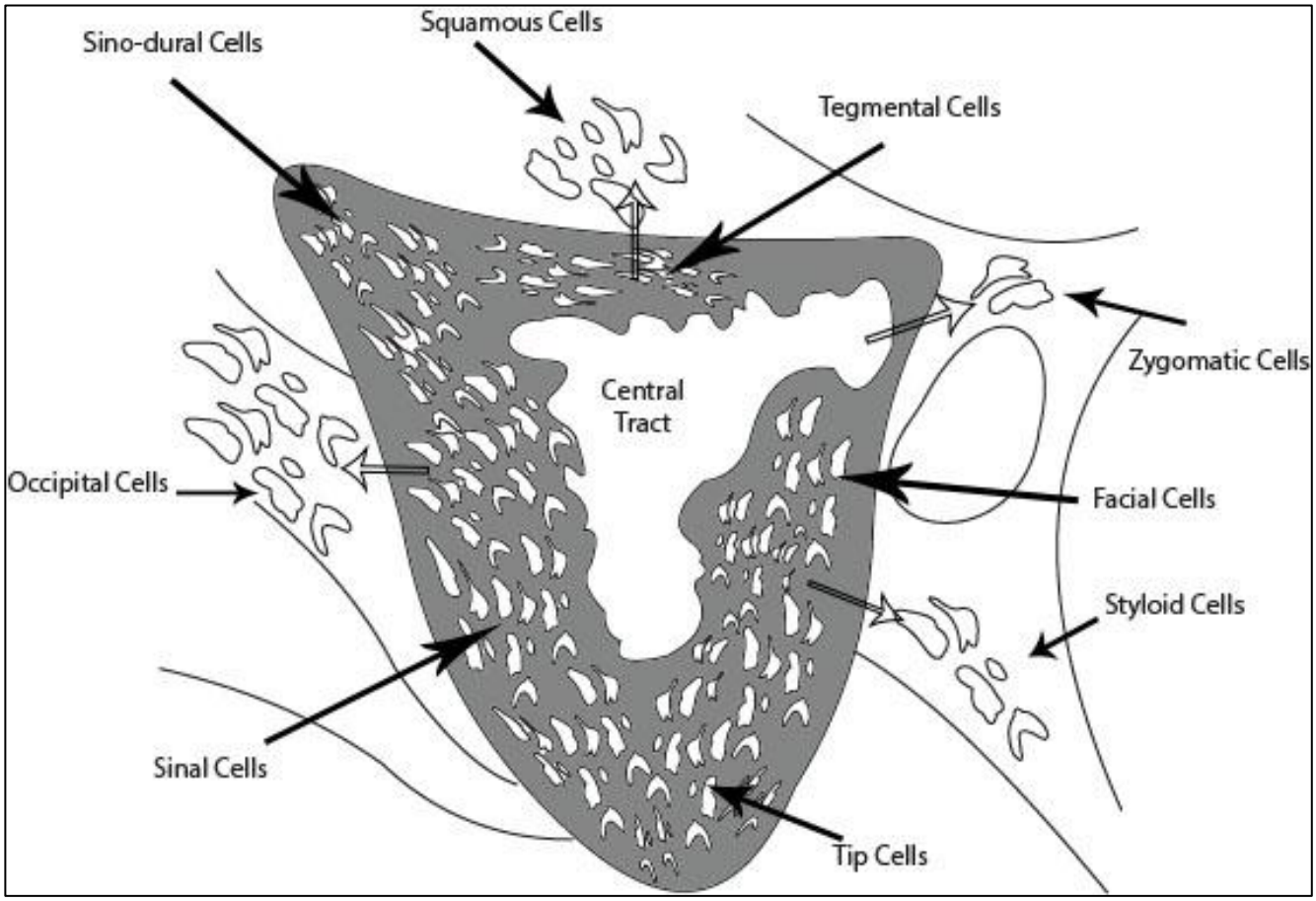


Figure 2.4 Sagittal view, showing the anatomy of the mastoid air cells. Clear arrows show communication between cells (adapted from Shuknecht 1974 in Virapongse et al. 1985:474).

The middle ear cleft is a highly connected series of structures in the temporal bone, which makes it susceptible to both secondary and systemic infections (Bluestone 1998; Rae and Ronan 2018). Infections can progress rapidly and be complicated by numerous factors. As a result, mastoiditis and OM are common (NHS 2018b) and can be severe if left untreated (NHS 2018b and 2019). The structure of the temporal bone air cells is the subject of frequent clinical studies, as the degree to which a patient's temporal bone is pneumatized effects how well they will recover from OM and mastoiditis (Koç et al. 2003); and the size and pattern of a patient's temporal bone air cells reveals how exudate and other fluids will move through the temporal bone following surgery and/or abscess (Jadhav et al. 2014).

Development of the Middle Ear Cleft

Infection during the development of the middle ear and MAC can affect the formation of the structures themselves. It is therefore important for paleopathologists to have a solid understanding of the development of the temporal bone, to be able to identify both normal and abnormal structures by age. To begin, the temporal bone undergoes both intramembranous and endochondral ossification (White and Folkens 2005: 98). At birth, the

temporal bone is in three parts (the squama, petrous part, and tympanic ring) that grow together to form one bone (White and Folkens 2005: 98). The mesotympanum grows only a little after birth, while the hypotympanum and epitympanum continue to grow throughout childhood (Isaacson 2014).

There are three stages to mastoid pneumatization[11]: rapid pneumatization between birth and one-year, linear pneumatization between one and six years, and slow pneumatization until puberty (Cinamon 2009; Gencer et al. 2013; O'Tuama and Swanson 1986). During the early fetal period, the middle ear cleft is filled with loose mesenchyme. This is reabsorbed between the fetal period and the first year. During the fetal period, in the 22nd to 24th weeks of gestation, four endothelial extensions of the lining of the Eustachian tube invade the middle ear, pneumatizing it. One of these, the *saccus medius*, extends to pneumatize the mastoid antrum and petrous portion of the temporal bone while another, the *saccus superior*, pneumatizes the MAC (Isaacson 2014). By birth, both the middle ear and mastoid antrum are pneumatized (Mansour et al. 2013; Petrus and Lo 1997). The rest of the temporal bone pneumatizes throughout childhood (Mansour et al. 2013; Petrus and Lo 1997). A fully-pneumatized temporal bone is defined as *hyper*cellular, while a partially pneumatized, or un-pneumatized, temporal bone is *hypo*cellular (Koç et al. 2003). The age at which pneumatization ceases is cited differently throughout the literature. Some quote the earlier age range of between eight and nine years (Bayramoğlu et al. 1997); others cite the later ages of 10 years in females and 15 years in males (Diamant 1940b; Rubensohn 1965); and yet others use the generic cut off point of "puberty" (e.g., Cinamon 2009; Sadé et al. 2006; Tos and Stangerup 1985). To rule out diagnostic error relating to juvenile bone appearance, 16 years was used as the cut off point for this project, as it is the latest threshold noted in the literature. All those 16 years and older should have pneumatized MAC regardless of which cut off point is correct.

Numerous methods have been used to measure the mean volume of the air cells: water weight (Silberger 1950), pressure transducer (Anderasson 1976), planimetric (Andreasson 1976; Frisberg and Zsigmond 1965), X-ray (Andreasson 1976; Frisberg and Zsigmond 1965; Sadé et al. 1989), and CT (Han et al. 2007; Isono et al. 1999; Jadhiv et al. 2014; Koç et al. 2003; Park et al. 2000; Todd et al. 1987). These methods measure the size of the space in the air cells either in cm^3 or mL. However, the complexity of these structures combined with the high levels of interpopulation variation has resulted in a variety of different mean volumes being quoted in the literature (see Table 2.1). In their seventy-year

[11] Pneumatization is the aeration of the bone *via* the growth of interconnected, epithelium-lined air-filled cells (Mansour et al. 2013).

retrospective assessment, Cinamon (2009) found the mean size for the average adult MAC to be 12cm^3 or 8mL.

Table 2.1 The mean volumes of temporal bone air cell system and mastoid air cells reported in the clinical literature. TACS refers to the entire temporal bone air cell system.

Study	Method	Area Included	Mean Volume (in cm^3)
Frisberg and Zsigmond 1965	Volumetric	TACS	12.22
Han et al. 2007	CT	TACS	15.28 ± 5.34
Koç et al. 2003	CT	MAC	7.9
Park et al. 2000	CT	MAC	10.43
Sadé et al. 1989	X-ray	MAC	12.9 ± 4
Silbinger 1950	Water weight	TACS	9
Anderasson 1976	Pressure transducer	TACS	5.83
Isono et al. 1999	CT	TACS	6
Todd et al. 1987	CT	MAC	7.59 ± 3.9

In general, until puberty, females have larger MAC than males; after puberty, both biological sexes have similarly sized air cells (Cinamon 2009). MAC size can vary between ethnic groups and individuals of different body sizes. While the pattern of the air cells in the temporal bone varies greatly between individuals (Bitar et al. 1996; Jadhav et al. 2014), pairs of temporal bones from the same individual generally display bilateral symmetry (Diamant 1940b; Myerson et al. 1934). Even when the volume of the air cells differs between sides, the structure and shape of the air cells is usually symmetrical (Isono et al. 1999; Virapongse et al. 1985). Thus, Virapongse *et al.* (1985) state that asymmetric pneumatization is usually indicative of unilateral infection. Identifying healthy adult MAC, infected adult MAC, and MAC stunted by childhood mastoiditis is critical to this project. To expand this discussion, the remainder of this section considers the function of the MAC and the etiology and pathophysiology of mastoiditis and OM.

Function of the Middle Ear Cleft

There are many possible reasons why the temporal bone is pneumatized. These include for the reception, resonance, and dissipation of sound; the insulation and protection of the skull; reducing the mass of the skull; or the maintenance of an air reservoir to sustain atmospheric air pressure in the middle ear (Tumarkin 1957). Of key importance to this study is the latter, as

maintaining atmospheric air pressure in the middle ear is important to the health of the entire middle ear cleft.

The MAC may have a role in gas exchange as a passive way of maintaining middle ear pressure (Sadé 1992). Unlike the other structures of the middle ear, the MAC are covered in highly vascularised cuboidal epithelium that have a close relationship with the basement membrane. Histologically, this is the same respiratory epithelium as that of the lungs and nose and is capable of gas effusion and diffusion. All these tissues exist at a lower pressure than the middle ear to facilitate gas diffusion. Thus, the total area of mucosa membrane (based on the pneumatization of the MAC) may relate to the rate at which the structure can exchange gas (Ars et al. 1997; Okubo and Watanabe 1988; Sadé 1992). Inflammation caused by infection in the MAC can over vascularise the tissues of the MAC and slow or stop gas diffusion (Sadé 1992).

When the pressure of the middle ear drops, the soft tissues that line the MAC (specifically the *pars flaccida* and *pars tensa*) retract from the boney walls. This decreases the volume in the MAC and raises the air pressure back to a healthy level (760mmHg) (Koç et al. 2003; Park et al. 2000; Sadé 1997). It should be noted that since air pressure is variable by latitude, the size of the MAC is influenced by the latitude of the region in which the individual matured (Collins and Jónsson 2018). Mucosa oedema and exudate buildup in the MAC can buffer the middle ear in the same way (Koç et al. 2003; Sadé 1997). If the retraction of the tissues is insufficient to buffer the ear, permanent soft tissue lesions (Görür et al. 2006) or a cholesteatoma[12] may form in the air cells, increasing the risk of further infection (Koç et al. 2003; Sadé 1997), multi-directional bone erosion, and intracranial complications (Mathews et al. 1988). These complications will be discussed with chronic middle ear infections below (see section 2.2.4.).

Keeping structures connected to the middle ear, such as the Eustachian tube and, to a lesser extent, the nasopharynx, clear and unblocked by mucus and inflammation is critical to the health of the middle ear (Sadé 1992; Todd 1994 in Sistani et al. 2019); as is the normal function and pneumatization of the middle ear cleft. A disfunction, such as that caused by infection, in one structure can affect the other structures and result in a host of complications.

[12] A cholesteatoma is "a mass formed by keratinizing squamous epithelium in the middle ear and/or mastoid, subepithelial connective tissue and by the progressive accumulation of keratin debris with/without surrounding inflammatory reaction" (Olszewska et al. 2015:83).

Acute and Chronic Infections in the Middle Ear Cleft

Infections are either acute or chronic based on how long they last. The cut-off point between acute and chronic for mastoiditis and OM varies in the clinical literature based on the guidelines being used. In general, acute OM (AOM) and acute mastoiditis are said to last between 10 and 60 days (Bluestone 1998; Groth et al. 2012). Typically, archaeologists do not expect to see bony involvement in acute infections, but mastoiditis is an exception (Flohr and Schultz 2009a,b). This disparity results from an overgeneralization on the part of archaeologists, who limit the definition of "acute" infection to those that "killed people very quickly" and therefore do not affect the bone (Roberts and Manchester 2007:12). This does not align with the clinical reality of disease, which is more complex. Thus, for the present study, it is important to understand the acute manifestations of mastoiditis and OM, as well as the chronic. This subsection describes the different manifestations of both acute and chronic mastoiditis and OM—their pathogenesis, clinical presentations, symptoms, complications, and risk factors.

Infection of the middle ear and/or MACs usually occurs *via* the Eustachian tube (Bluestone 1998; Rae and Ronan 2018) or haematopoietically from a systemic infection (Rae and Ronan 2018) caused by viruses or bacteria such as *Haemophilus influenzae* (Bluestone 1998). AOM can also occur secondary to a viral upper respiratory infection. Thus, AOM can be an isolated infection or secondary to another infection. AOM is defined as an infection that results in the inflammation of the middle ear and presents with effusion therein (Rae and Ronan 2018). Mastoiditis is a serious and frequent complication of AOM (Bluestone 1998; Groth et al. 2012; Wilson et al. 2017). In one study, acute mastoiditis and AOM co-occurred in 97% of children examined (Groth et al. 2012). Thus, both acute mastoiditis and AOM are referred to here collectively as AOM.

AOM is a highly misunderstood disease. It is difficult to diagnose, and the clinical literature is full of inaccurate and unsupported diagnoses. Thus, caution must be taken when interpreting the clinical literature, especially with regard to the pathogens responsible for individual cases of AOM (Rae and Ronan 2018). Bacteria and viruses are most frequently identified in cases of AOM, appearing alone or in combination: namely, *Streptococcus pneumoniae* (Bluestone 1998; Groth et al. 2012; Rae and Ronan 2018), *Moraxella catarrhalis* (Rae and Ronan 2018), *Streptococcus pyogenes* (Bluestone 1998; Groth et al. 2012; Rae and Ronan 2018), and *Haemophilus influenzae* (Bluestone 1998; Rae and Ronan 2018). *H. influenza* was the pathogen most frequently responsible for many types of AOM in the 1980s. However, today, drug-resistant *Strep. pneumoniae* and *pyogenes* are the most common (Rae and Ronan 2018). This change in virulence highlights the direct impact modern health

care has had on the pathogenesis of AOM. While it is advised that recent changes in pathogen type and virulence must be accounted for when studying archaeological populations (Roberts and Manchester 2005), this particular case emphasises how important this consideration is for this, and any other, study of mastoiditis and OM.

There are multiple ways to divide and define acute infections (see Table 2.2). Rae and Ronan (2018:137-8) and Bluestone (1998) describe four different types of AOM based on the duration of each type of infection: 1) that without bony involvement; 2) that with bony involvement and no air cell coalescence; 3) that with bony involvement and air cell coalescence; and 4) that with a lingering infection and intra-temporal and/or intracranial complications. The first type of acute mastoiditis does not involve the bone. It resolves itself when the exudate in the middle ear cleft drains *via* the Eustachian tube (Bluestone 1998). Rae and Ronan (2018) refer to this type simply as AOM and do not consider mastoiditis to be a complication. However, when the *aditus ad antrum* becomes blocked by an edema or a buildup of granulated tissue, as is the case for the three other types of acute infections, the infection can spread to the periosteum *via* the emissary veins and illicit a host of bony changes (Bluestone 1998; Rae and Ronan 2018).

Table 2.2 Types of acute and chronic mastoiditis and otitis media. AOM occurs via the Eustachian tube, often from a viral upper respiratory infection, or haematopoietically from another site of infection (Bluestone 1998; Rae and Ronan 2018). Childhood mastoiditis can stunt the pneumatization of the temporal bone air cell and result in primary hypocellularity, or permanently small temporal bone air cells (Sadé et al. 2006; Tos and Stangerup 1985).

Acute or Chronic	Age	Type	Bony Involvement and Differentiation	Pathogens	Pathogenesis	Works Cited
Acute	Child and Adult	Type 1 (or AOM)	Neither bony involvement nor mastoiditis.	*Streptococcus pneumoniae, Moraxella catarrhalis, Streptococcus pyogenes,* and *Haemophilius influenzae*.	AOM	Bluestone 1998; Robb and Williamson 2018; Rosenfeld and Kay 2003
		Type 2 (or AM with sub-periosteal new bone)	With bony involvement and no air cell coalescence.		AOM followed by the blockage of the *aditus ad antrum*.	
		Type 3 (or AM osteitis or acute coalescent mastoiditis)	With bony involvement and air cell coalescence.			
		Type 4 (or sub-acute or masked mastoiditis)	With chronic infection and intra-temporal and/or intracranial complications.			
		AOME (or serous, secretory, and non-suppurative AOM)	AOM with effusion through an intact membrane.			

(Table continued on next page)

Acute or Chronic	Age	Type	Bony Involvement and Differentiation	Pathogens	Pathogenesis	Works Cited
Chronic	Child and Adult	COME (or residual CM with effusion)	Thickening of the bony air cell walls and effusion through an intact tympanic membrane.	*P. aeruginosa* and *Streptococcus aureus*	Lingering AOM or a secondary infection *via* the Eustachian tube or a perforation in the tympanic membrane.	Bluestone 1998; Fliss et al. 1992; Kenna and Bluestone 1986; Papastavros et al. 1989; Vartiainen and Vertiainen 1996; Yuen et al. 1995; Qvarnberg 1982
		CSOM (or chronic otomastoiditis)	Thickening of the bony air cell walls and discharge through a perforated tympanic membrane.			

The second type of acute infection is named acute mastoiditis with sub-periosteal reaction (Rae and Ronan 2018). It presents clinically as fever; pain, redness, mild tenderness, and/or edema posterior to the ear; and/or displacement of the pinna (Bluestone 1998). Welin (1941) notes that after two weeks of acute mastoiditis, the infection can be diagnosed based on the thinning and decalcifying of the air cell walls. This should be visible in this type of AOM both radiographically and histologically.

The third type of acute infection is marked by the spread of exudate through the temporal bone. Rae and Ronan (2018) call this acute mastoid osteitis. Bluestone (1998:15) explains that exudate follows six known pathways through the temporal bone air cells. As it travels, the exudate destroys the bony walls of the air cells and can damage the delicate structures within the temporal bone, such as the facial nerve. This type of acute infection presents similarly to the first two, but can also include temperature spikes, swelling and/or displacement of the posterosuperior external auditory canal wall, abscess (postauricular sub-periosteal, zygomatic, Bezold, or retro- or para-pharyngeal) (Rae and Ronan 2018), and/or perforation of the tympanic membrane (Bluestone 1998). Bezold abscesses are of interest here as they perforate through the middle mastoid cortex. Exudate drains from the middle ear cleft into the parapharyngeal space and mediastinum following the sternocleidomastoid muscle and posterior triangle (Castillo et al. 1998; Dobben et al. 2000; Maroldi et al. 2001; Rae and Ronan 2018; Tremble 1934).

Subacute or masked mastoiditis (Bluestone 1998; Rae and Ronan 2018), the fourth and final type of AOM, presents with few of the normal symptoms of AOM. It lasts longer than other AOM—10–14 days (Rae and Ronan 2018)—and is marked by the spread of the infection to other parts of the temporal bone and/or intercranial space (Bluestone 1998). This form of acute infection is rare (Mawson and Ludman 1979) and its complications can be deadly (Bluestone 1998).

The symptoms and presentation of AOM differ depending on the type of acute infection, the involvement of the MAC, the immune response of the individual (Rae and Ronan 2018), and the presence of a cholesteatoma (Go et al. 2000). It is the presence of the latter that results in the majority of intracranial complications (Go et al. 2000). In general, AOM presents as the "rapid onset of otalgia (or earache), hearing loss, otorrhoea, fever…irritability, coryzal symptoms, vomiting…and clumsiness" (Rae and Ronan 2018:138). In children, it can also present as crying and the refusal to breast or bottle feed. Rarely does the tympanic membrane perforate. When it does, it may heal spontaneously or persist and be a risk factor for further middle ear infections (Rae and Ronan 2018). Groth *et al.* (2012) note that sub-periosteal abscess is the most common complication of acute mastoiditis regardless of age.

AOM with effusion (AOME; or serous, secretory, and non-suppurative AOM) is separate from AOM and receives a lot of clinical attention (see Table 2.2). OME can be acute or chronic and is "the accumulation of mucus within the middle ear and sometimes the (MAC)" and the effusion of fluid through an intact tympanic membrane (Robb and Williamson 2018:115). Cases of OME that last shorter than three months are considered to be acute (Bluestone 1984). OME is most common in children under the age of three (Teele et al. 1989). Children with OME present with hearing loss, redness and tenderness in the area of the external auditory canal, and/or a bulging tympanic membrane (Robb and Williamson 2018; Rosenfeld and Kay 2003). OME can cause a delay in speech and language acquisition, poor behaviour and academic performance, and problems with balance (Robb and Williamson 2018; Rosenfeld and Kay 2003). AOM neither present in a single way, nor follow a linear progression. Rather, "AOM" encompass a host of disease pathways, complications, and symptoms; and encompasses a host of bony changes.

Chronic OM (COM) and chronic mastoiditis are synonymous, as COM involves the inflammation of the middle ear cleft, which by definition, involves both the middle ear and MAC (Hellier 2018). Thus, both are referred to here as COM (see Table 2.2). COM is the result of a lingering acute infection or a secondary infection *via* the Eustachian tube or a perforation in the tympanic membrane (Bluestone 1998). The pathogens most often involved in chronic infections are *P. aeruginosa* and *S. aureus* (Fliss et al. 1992; Kenna and Bluestone 1986; Papastavros et al. 1989; Vartiainen and Vertiainen 1996; Yuen et al. 1995). COM is diagnosed clinically by the presence of exudate in the middle ear and/or the temporal bone air cells, and the thickening of the bony air cell walls (Qvarnberg 1982).

Chronic infections are classified as either COM with effusion (COME; referred to as "chronic mastoiditis with effusion" in Bluestone; but referred to here as "otitis media" rather than "mastoiditis" for consistency with our other terms as it involves the entire middle ear cleft) or chronic suppurative OM (CSOM; or chronic otomastoiditis). Both refer to the chronic infection of the middle ear cleft, but COME includes effusion through an intact tympanic membrane, while CSOM includes discharge out of a perforated tympanic membrane. CSOM can often result in hearing loss or—less frequently—intracranial complications, facial paralysis, or labyrinthitis (Bluestone 1998). Globally, CSOM is one of the most common childhood diseases and is often associated with poverty (Bluestone 1998).

Either form of COM can be further complicated by the presence of a cholesteatoma (Bluestone 1998; Hellier 2018). These can be congenital or acquired (Hellier 2018) and are common in hypocellular ears (Sadé 1992, 1993; Sato et al. 1990 both; Sato et al. 1997). The

clinical literature divides the placement and symptomology of cholesteatoma based on their origin and the age of the patient.

Congenital cholesteatoma form within the middle ear, next to the tympanic membrane, while acquired cholesteatoma form within the middle ear or the MAC (Hellier 2018). In adults, cholesteatoma occur more often in the middle ear attic or MAC and are usually accompanied by a retraction pocket or a perforated eardrum. In children, they can be congenital, can occur more often in hypocellular MAC or the tympanic cavity, and are usually not accompanied by a perforated eardrum (Sadé and Fuchs 1994). There is no description of what to expect when these two groups overlap.

In general, the symptoms and complications of COM vary depending on the presence of a cholesteatoma (Hellier 2018). COM both with and without a cholesteatoma can result in the erosion and/or fixation of the auditory ossicles; and COM with a cholesteatoma (COMC) can result in the extension of the cholesteatoma into and the erosion of the MAC (Hellier 2018). Identifying the presence of a cholesteatoma is critical to treating COM and understanding the infection's further complications.

The effects of COM can persist once the infection is healed, thus Hellier (2018) encourages clinicians to distinguish between active COM and inactive/healed COM. The later encompasses the physiological changes wrought by active COM and the risk factors they represent moving forward: "submucosal scarring and fibrosis, increased mucous-producing cells, bony erosion or new bone growth, perforation or thinning of the tympanic membrane and changes in the labyrinth" (Hellier 2018:155). Thus, COM can be itself a risk factor for having future infections in the middle ear cleft. As presently discussed, the effect these changes have on individual susceptibility to further OM and mastoid infections is the subject of much clinical debate.

The bony effects of the later stages of AOM and COM are similar. Both can result in the destruction of the MAC and new bone growth therein (Bluestone 1998; Hellier 2018; Rae and Ronan 2018); however, AOM "takes the plane of least resistance" (Mathews et al. 1988) and COM, by definition, results in the thickening of the bony air cells walls (Qvarnberg 1982). Thus, the ability to differentiate AOM from COM archaeologically is difficult, but theoretically possible.

Today, most patients with mastoiditis and OM are children (NHS 2018b). Rosenfeld and Kay (2003) found that approximately 30%–40% of the people included in their retrospective study had recurrent OM with effusion (OME) as children, while Teele *et al.* (1989) found that 80% of all children they studied had at least one episode of OME in their lifetime. Similarly, in a retrospective study of 678 cases of acute mastoiditis in Swedish children, Groth *et al.* (2012)

found that acute mastoiditis occurred predominantly in children under the age of seven years, the majority of whom were younger than two years (55%) and male (61%). While mastoiditis and OM are predominantly diseases of childhood, they can also occur, albeit less frequently, in adults (Bluestone 1998; Robb and Williamson 2018).

Childhood and adult infection are different in their frequency, complications, and implications. First, there are more risk factors for developing mastoiditis and OM among children. Most importantly, children have an immature immune system (Roberts and Manchester 2007), making them more susceptible to all forms of infection. Non-adults also have a shorter and more horizontal Eustachian tube than in adults, which makes it easier for pathogens to enter the middle ear and harder for the middle ear to drain (Mansour et al. 2013:141–153). During a viral upper respiratory infection, non-adults' Eustachian tubes function less efficiently than those of adults (Doyle et al. 1999, 2000). Additionally, mastoiditis and OM are linked to community acquisition most critically, to living in a household with older children or other children with a history of OM, and to attending a daycare with four or more other children under the age of three-and-a-half years old (Chonmaitree et al. 2008; Uhari et al. 1996). Lastly, childhood mastoiditis can stunt the pneumatization of the temporal bone air cells and result in primary hypocellularity, or permanently small temporal bone air cells (Sadé et al. 2006; Tos and Stangerup 1985). This leaves the individual more at risk of suffering from future episodes of mastoiditis or OM, as the air cells are too small to buffer the middle ear cleft from changes in air pressure.

Insufficient breastfeeding can also leave infants and children more at risk of OM and mastoiditis than those exclusively breastfed for at least the first two to three months of their life (Bowatte et al. 2015; Lodge et al. 2016; Uhari et al. 1996). Breastfeeding primes infant immunomodulation and gut microbiota, which allows them to better combat infection (Cerini and Aldrovandi 2013; Raheem et al. 2017; Riskin et al. 1996; Victoria et al. 2016; WHO 2018b). Exclusive breastfeeding for the first two to three months has been observed to reduce the risk of OM in infants and children by approximately 40–50% (Bowatte et al. 2015; Lodge et al. 2016; Uhari et al. 1996).

Further to those specific to children, there are a host of risk factors for developing acute or chronic mastoiditis and OM that apply to both children and adults. These include being male (Jaillon et al. 2019; Trigunaite et al. 2015) and exposed to smoky air (Uhari et al. 1996; Zhang et al. 2014). Sexual dimorphism is linked to decreased innate and adaptive immunity in males compared to females (Jaillon et al. 2019; Trigunaite et al. 2015). Differences in genetics and hormones are known to account for some of these differences, including (but not limited to) the regulation and expression of pattern recognition molecules, the structure and function of neutrophils, and the number and function of monocytes,

macrophages, natural killer cells, and plasmacytoid dendritic cells. Air pollution, especially cigarette smoke, triggers an allergic reaction and irritates the respiratory system, affecting the middle ear *via* the Eustachian tube (Csákányi et al. 2012; Uhari et al. 1996). Mastoiditis and OM are also more common in those with a clinical history of infection, such as having upper respiratory tract infection(s), allergies, Eustachian tube disfunction, a compromised immune system, or a genetic predisposition to mastoiditis and OM (see the sub-section below for further discussion of this risk factor) (Chonmaitree et al. 2008; Csákányi et al. 2012; Mathews et al. 1988; Rye et al. 2011; Uhari et al. 1996). Robb and Williamson (2018) encourage clinicians to consider the multifactorial and situational nature of risk factors, suggesting that clinical studies should prioritise controlling for what variables they can to identify the causative factor involved in individual cases. While they found that multiple risk factors may be present in individual cases, only one may be the root of an individual infection.

AOM and COM can both affect the bone and are, therefore, both of interest to archaeologists. A detailed insight into the different ways in which the bone can be affected (stunting the pneumatization of the temporal bone, thinning air cells walls, sub-periosteal reaction, abscessing, and more) is important for interpreting the possible disease processes involved in the formation of archaeological bony lesions. The next section explores the impact and/or cause of middle ear infection and the debate between the environmental and genetic theories.

The Environmental and Genetic Theories Debate

The understanding of the pathogenesis of mastoiditis and OM is entrenched in the debate between the so-called genetic and environmental theories (Koç et al. 2003). Clinicians know that there is a relationship between hypocellularity, and COM (Bayramoğlu et al. 1997; Sato et al. 1997) and OME (Görür et al. 2006; Maxwell et al. 1994); but as of yet, they have been unable to prove how that hypocellularity occurs (Koç et al. 2003). This section explores the arguments made on both sides of the debate and their relevance to palaeopathology.

In 1918, Wittmaack proposed that inflammation in the middle ear cleft, caused by infection and/or a blocked Eustachian tube, affects the health of the mucosal epithelia that line the middle ear, and that this inflammation alters the pneumatization of the middle ear and its associated air cells. In sum, that outside factors which effect the health of the middle ear cleft can alter the pneumatization therein. This became known as the environmental theory. The environmental theory has since been supported by many other researchers (Aoki et al. 1986, 1989; Bayramoğlu 1997; Ikarashi et al. 1994; Koç et al. 2003; Robinson et al. 1993; Sadé et al. 1997; Shim et al. 2012; Tos and Stangerup 1984).

Two landmark studies which support the environmental theory require discussion. The first is the study by Aoki *et al.* (1986). The authors conducted an experiment using pig models to study the relationship between mastoid pneumatization and the health of the middle ear cleft. Pigs have a similar middle ear structure to humans and, so, are a good proxy for the function and health of human ears. The pigs were divided into two groups. In one, the authors injected silicone into the area around the pigs' Eustachian tube to simulate OME. In the other, the authors injected paraffin into the pigs' middle ear to simulate the swelling and exudate buildup seen in cases of COM. Injections were given only to one ear, so the other could act as a control. The animals were killed, sectioned, and examined histologically in sub-groups 13 days, 21 days, and 164–180 days after the injections. Those in the second group showed more severe and numerous problems in the affected ear than those in the first group, including decreased MAC pneumatization. The authors concluded that the number of symptoms and the degree to which their mastoids are pneumatized was dictated by both the severity and length of infection and the individual's age—or mastoid developmental stage—when infected (Aoki et al. 1986). This study showed direct correlation between middle ear cleft inflammation and the development of hypocellularity.

In the second study, the same researchers treated two groups of children, both with OME, based on the degree to which their mastoids where pneumatized. Those whose mastoids were hypercellular were treated conservatively, while those with hypocellular mastoids had a ventilation tube inserted. Both groups healed to the same degree, despite the differences in their treatments. This, the authors argued, demonstrated that individuals with fully pneumatized mastoids (hypercellular) could heal completely from OM with little medical intervention and, thus, that the MAC play a large role in maintaining the health of the middle ear system (Aoki et al. 1989). While this study did not explore the environmental theory directly, it supported it indirectly by demonstrating the connection between the size of the MAC and the health of the middle ear cleft. Many studies have since found that the degree to which a patient's MAC are pneumatized is positively correlated to the likelihood of them recovering from a middle ear or mastoid infection (e.g., Görür et al. 2006; Özkul et al. 1999; Sadé 1992; Shambaugh and Glasscock 1990).

On the other side of the argument is the genetic (or normal variant or hereditary) theory. It was first proposed by Cheatle (1910). He hypothesized that hypocellularity was a risk factor for developing OM (1910). Thirty years later, the theory was popularized by Diamant (1940a,b), who expanded it to also state that air cell size is genetically predetermined (Diamant 1940a,b). Both facets of the theory have been well researched and supported by other clinicians (e.g., Cinamon 2006; Sadé et al. 2006). A retrospective study by Sadé *et al.* (2006) is often cited. Here four groups of children (2–11 years), from

before and after the common clinical use of antibiotics, with or without a history of recurrent AOM, were studied. It was found that hypocellularity was not associated with AOM and, therefore, that one was not a risk factor for the other (2006). This study does not support the environmental theory and, thus, suggests that something other than inflammation determines the size of the MAC; in the authors' opinion, this factor is genetics.

It is difficult to conclude whether inflammation or genetics are behind hypocellularity, especially since correlation does not prove causation. Kim *et al.* (2010) argue for example, that the environmental theory, rather than the genetic theory, is more likely to account for individual variation. But, more conservative thinkers, such as Song *et al.* (2017), argue that both theories have merit and should both be considered when discussing the pathophysiology of mastoiditis and OM. In general, the latter appears to be the trend in the clinical literature in lieu of a definitive explanation (Song et al. 2017; Virapongse 1985). Until the role and function of the air cells are fully understood, it will remain critical to be transparent about the debate to avoid unconscious bias.

In this section the anatomy of the middle ear cleft and the development and function of the temporal bone air cells have been reviewed; the risk factors, pathogenesis, clinical presentation, symptoms, and complications of acute and chronic mastoiditis and OM have been outlined; and the environmental *versus* genetic theories debate has been discussed. This research serves as a modern benchmark to which the archaeological research can be compared. Research into the development and function of the air cells is at the cutting edge of mastoiditis and OM research. As such, not only can palaeopathological studies such as this benefit from a sound understanding of clinical work, but they can also provide an example of the bony effects of untreated middle ear infections, which can offer new insights into the clinical understanding of disease progression and interactions. The following two sections discuss sinusitis and LRI and explore how these infections, and the structures and risk factors involved, relate to mastoiditis and OM.

2.3 Sinusitis

Sinusitis is the inflammation of at least one paranasal sinus (PNS) (Chadha and Chadha 2007). It is diagnosed clinically when at least two of the following symptoms persist for a minimum of 7–10 days (Bocian 1993): "blockage or congestion; discharge or postnasal drip; facial pain or pressure; (and) reduction or loss of smell" (Chadha and Chadha 2007:1165). Fetor oris[13],

[13] Fetor oris is bad breath (Kaimenyi 1985).

periorbital swelling, headache, odontalgia[14], hyposmia[15] (Bocian 1993), cough, sore throat, fatigue, and fever (Torpy 2009) may also be present. Discharge becomes increasingly purulent and thick as the infection persists (DeMuri and Wald 2012).

This section examines the anatomy and function of the PNS; and explores the relationships between the health of the PNS and the health of the middle ear cleft. Lastly, the pathophysiology and risk factors responsible for the development of sinusitis are discussed. The rate of infection within the maxillary sinuses is highlighted, as that was the specific sinus infection examined by this project.

Anatomy and Function of the Paranasal Sinuses

There are four pairs of PNS in the human head: the frontal, ethmoid, sphenoid, and maxillary (Martini et al. 2012). These are air-filled cavities in the bone that are lined with mucosal epithelium and cilia (or hairs) that rhythmically beat to move mucus out of the sinus, *via* an ostium (or opening), and into the nasal cavity (Evans 1994; Gwaltney 1996; Melén 1994). They serve to filter air inhaled through the nose before it is inhaled deeper into the lungs (Evans 1994; Gwaltney 1996). When functioning properly, air-born pathogens and pollutants should become trapped in the mucosal lining of the sinuses and be expelled, *via* the ostia, into the nasal cavity to be swallowed or expelled out of the nose (Evans 1994).

The sinuses connect to the nasal cavity in two structural regions: the osteomeatal complex/middle meatus and the superior meatus (see Figure 2.5). Both are defined by the conchae. The osteomeatal complex and middle meatus are located superior to the inferior nasal concha and inferior to the middle nasal concha; and the superior meatus is located superior to the middle nasal concha and inferior to the superior nasal concha. The frontal, anterior and middle ethmoid, and maxillary sinuses open into the osteomeatal complex, which, in turn, opens into the middle meatus. The posterior ethmoid and sphenoid sinuses open into the superior meatus. Posterior-inferior to the nasal cavity is the nasopharynx, where the Eustachian tubes open (Brook 2009; Gray 1918; Martini et al. 2012).

[14] Odontalgia is toothache (Clark 2006).
[15] Hyposmia is the loss of the sense of smell (Gaines 2010).

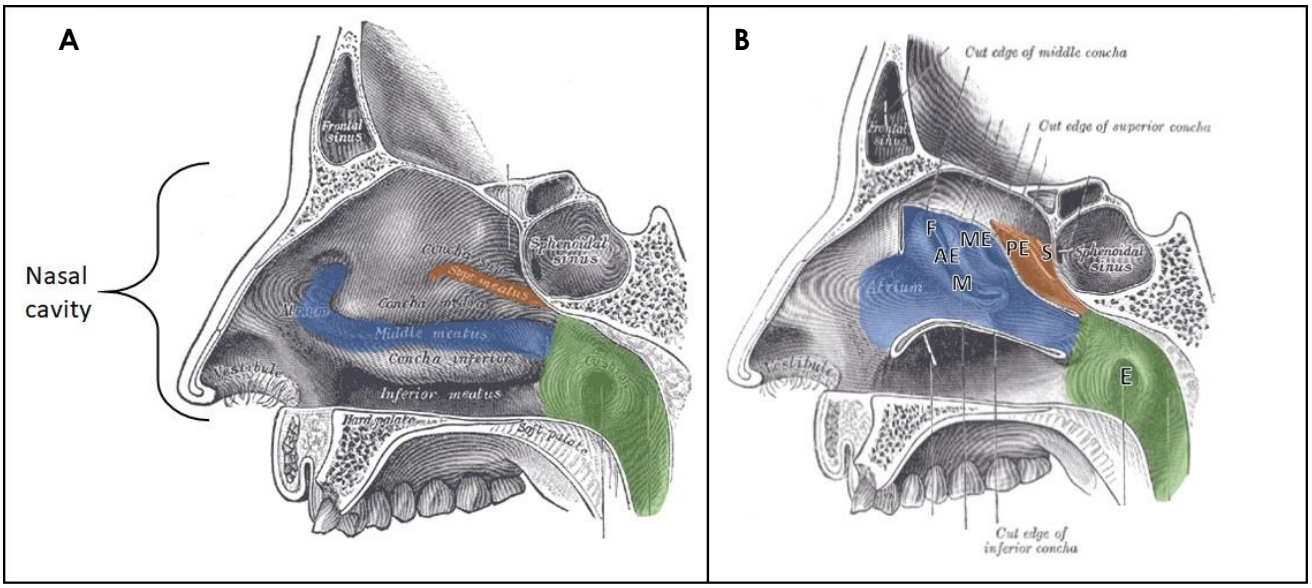


Figure 2.5 Right section of the nasal cavity in the sagittal plane showing the middle meatus in blue, the superior meatus in orange, and the nasopharynx in green with **A)** the nasal conchae and **B)** the inferior and medial conchae cut away to reveal the ostia for the Eustachian tube (E) and sinuses: the frontal (F), maxillary (M), sphenoid (S), the anterior ethmoid (AE), middle ethmoid (ME), and the posterior ethmoid (PE) (adapted from Gray 1918:994–5).

Much research has been carried out to investigate if there is a relationship between the pneumatization of the MAC and PNS. The results, however, have been mixed. While many studies have found a positive correlation between the pneumatization of the MAC and the sphenoid sinuses (Kim et al. 2010; Hindi et al. 2014; Song et al. 2017), others found no positive correlation between the pneumatization of the MAC and the PNS (e.g., Lee et al. 2012). As the debate between the environmental and genetic theories shows, pneumatization is complex and not yet fully understood. The same debate extends to the pneumatization of the sinuses. There are, however, two theories to explain the correlation between the pneumatization of the MAC and sphenoid sinuses that lend some weight to the environmental theory.

Firstly, the correlation is largely attributed to the structural relationship between the sphenoid sinus ostia and the Eustachian tubes (Kim et al. 2010; Song et al. 2017). Their proximity may suggest that inflammation in the shared area—specifically in the adenoid tissue (situated along the superior and superior-posterior walls of the nasopharynx)—affects how patent the ostia and Eustachian tubes are. If they swell shut, this can lead to an infection in the structures. In a child, such infection can stunt pneumatization (Kim et al. 2010; Song et al. 2017; Marchinio et al. 2007; Pagella et al. 2010). It should be noted, however, that one study failed to show a developmental relationship between the size of adenoid tissue

and the health of MAC and, instead, attributed the health of the middle ears strictly to the health of the Eustachian tubes (Apuhan et al. 2011).

Secondly, the correlation is also attributed to the increased exposure of the MAC and sphenoid sinuses to infection during development. The ethmoid and maxillary sinuses are present at birth (Bocian 1993; Brook 2009; Wald 2011), while the other sinuses develop postnatally; all the sinuses grow and pneumatize through until adolescence (Brook 2009) and are fully grown by approximately 12 years (Cinamon 2009). The sphenoid sinuses, however, are the last PNS to be fully developed—similar to the development of the MAC (Hindi et al. 2014; Kim et al. 2010; Tan et al. 2003). Since both structures take longer to develop than the other PNS, they are at a greater risk of being developmentally delayed by infection (Tan et al. 2003). Again, the results of some studies conflict with this theory. One study found that environmental factors appeared to affect the development of the MAC more than the PNS (Lee et al. 2012), while another found that the PNS undergo a developmental process different to that of the MAC (Thomas and Raman 1989).

Both these theories—and their conflicting studies—show that the final size of the MAC and PNS are not solely dictated by an individual's genetics. External factors, such as infection and inflammation, can affect their size. Regardless of the processes involved or the relationships between the structures, external environmental factors likely have a role in the pneumatization and health of the MAC and PNS. To better understand what these environmental factors are, the remainder of this section discusses the pathophysiology of sinusitis and its risk factors.

Pathophysiology of Sinusitis

The inflammation of paranasal sinus mucosa can be brought on by infection or other conditions such as allergies, congenital disease, or obstruction (e.g., tumour, deviated septum, foreign body) (Bocian 1993; Torpy 2009). Infections can be viral (Chadha and Chadha 2007; Bocian 1993; Torpy 2009) and/or bacterial (Chadha and Chadha 2007; Brook 2009; Torpy 2009), or, rarely, fungal in origin (Bocian 1993). Primary infections are usually caused by a community-acquired viral upper respiratory infection (URI) (Chadha and Chadha 2007; Bocian 1993; Torpy 2009). Thus, rhinovirus, adenovirus, influenza, and parainfluenza viruses—common community-acquired viral URI—are most often responsible for sinusitis (Brook 2009; Torpy 2009; Wald 2011).

Primary sinusitis causes a cascade of a/effects within the sinuses: inflammation; the production of excess mucus; the paralysis of the cilia; the buildup of mucus; the blockage of the ostium; and, ultimately, the de-oxygenation and buildup of negative pressure (Chadha and Chadha 2007; Wald 2011). The latter creates the right environment within the sinus for a

secondary bacterial infection to take hold (Brook 2012; Wald 2011). *Streptococcus pneumoniae, Haemophilus influenzae,* or *Moraxella catarrhalis* are the bacteria the most often responsible for such secondary infections (Chadha and Chadha 2007; Brook 2009; Torpy 2009).

Primary PNS infection often occurs *via* the inhalation of pathogens, but the PNS can also become infected if their ostia become mechanically blocked by debris or inflammation. This creates the same de-oxygenated and low-pressure environment in the sinus as previously described; triggers the same inflammatory pathways; and creates the same environment favourable to secondary infection (Bocian 1993; Torpy 2009). Alternatively, MS can be odontogenic (or result from maxillary dental abscess or inflammation from dental disease) (Hajiioannou et al. 2010; Brook 2006). Such instances account for approximately 10%–12% of all MS cases (Brook 2006; Mehra and Murad 2004). Maxillary sinuses that included direct communication between the oral and sinus cavities were not included in this study in an attempt to limit cases of odontogenic sinusitis, as the pathology of odontogenic sinusitis is different (oral rather than air-born) from other sinus infections (Brook 2006; Kretzschmar and Kretzschmar 2003). As a result, its pathophysiology is not discussed in detail here.

A virus, bacteria, or fungi can be responsible for an acute, recurrent acute, subacute, chronic, or an acute exacerbation of a chronic infection (Brook 2009); but there is a disagreement in the literature regarding what constitutes an acute and chronic infection. One source states that acute infections last fewer than 10 days (Brook 2009), while another states that chronic infections last longer than 12 weeks (Chadha and Chadha 2007). How to define those infections persisting between 10 days and 12 weeks is unclear and likely varies. What is clear, is that acute infections generally resolve within a few days, while chronic infections can persist for months.

The anterior ethmoid and maxillary sinuses are the PNS most frequently infected (Bocian 1993). Similar to the relationship between the size of the MAC and sphenoid sinuses, this is due to the structures' location and development. Their ostia both open to the osteomeatal complex; therefore, an infection or mechanical blockage affecting one will likely affect the other (Bocian 1993). They are also the only PNS present since birth and can, therefore, become infected immediately (Bocian 1993; Brook 2009; Wald 2011).

While this study concerned mastoiditis, and the MAC have a relationship with the sphenoid sinuses, the reason MS was studied here, and not sphenoid sinusitis, was because the maxillary sinuses are frequently infected. Thus, there was more of an opportunity to study the presence of risk factors within the environment and, ultimately, to understand the environment in which the individuals lived, by studying MS. While the relationship between the MAC and sphenoid sinuses is fascinating, it reveals more about the structure and

development of the middle ear complex and sphenoid sinuses than the environment. Since sinusitis was studied to contextualize and inform the understanding of mastoiditis, we studied MS.

Risk Factors for Developing Sinusitis

The risk factors for developing a sinus infection are as follows: having an URI (Gwaltney 1996; Chang et al. 2018), asthma (Hoover et al. 1997; Chang et al. 2018), eczema (Chang et al. 2018), allergic rhinitis (Chang et al. 2018), atopy[16] (Chadha and Chadha 2007; Hoover et al. 1997; Chang et al. 2018), chronic inflammation (specifically IgG4 level ≥40mg/dl) (Hoover et al. 1997), a high white blood cell count (specifically peripheral eosinophil ≥200pJ) (Hoover et al. 1997), diabetes (Brook 2009), or a comorbidity such as chronic cardiac, hepatic, or renal disease (Brook 2009); being immunocompromised (Brook 2009); being a child in daycare or living with a child in daycare(Brook 2009); being under-breastfed as an infant (Chonmaitree et al. 2016; WHO 2018b); or living in an environment with air pollution (Li et al. 2018; Pieta et al. 2015), especially from cigarette or pipe smoke (Brook 2009; Richardson 1988 in Slavin 1988). The winter season can also increase the risk of developing sinusitis (Brook 2009; NHS 2000). While sinusitis is more likely to develop in children (Brook 2009), being 50 years or above can also be a risk factor for developing chronic sinusitis (HHP 2020; Hoover et al. 1997).

Sinusitis shares many of its risk factors with OM and, consequently, mastoiditis. Specifically, they are frequently instigated by a URI acquired in in the community, especially at a daycare (Chonmaitree et al. 2016; Wald 2011); common in individuals exposed to air pollution, especially smoke (Brook 2009; Richardson 1988; Chonmaitree et al. 2008; Uhari et al. 1996); and who are under-breastfed (Brook 2009; Chonmaitree et al. 2016; Tobin and Aldrovandi 2013; Victoria et al. 2016); and present in individuals with allergic and other immunologic diseases (Hoover et al. 1997; Chang et al. 2018; Chonmaitree et al. 2008; Uhari et al. 1996). The latter is an individual, often genetic, risk factor and, therefore, difficult to infer archaeologically. The first three factors, however, can relate to community health and cultural practices. The impacts of daycare, breastfeeding, and air pollution are discussed in detail below in section 2.5.

[16] Atopy is a genetic predisposition to developing an allergic disease (Chadha and Chadha 2007; Hoover et al. 1997; Chang et al. 2018).

2.4 Lower Respiratory Infection

Lower respiratory infection (LRI) refers to the infection of organs in the respiratory system inferior to the trachea (those superior to, and including, the trachea are part of the upper respiratory system). Infections are named in one of two ways. Some are named according to which organ(s) of the lower respiratory system (LRS) is infected: bronchitis (or bronchus infection), bronchiolitis (or bronchiole infection), chest infection, and pneumonia (or lung infection) (NHS 2018d). Other LRI affect multiple organs/systems and, so, are named according to the pathogen responsible for the infection, for example, tuberculosis (TB) (FIRS 2017). LRI are usually more persistent and serious than URI (NHS 2018d). They are the fourth cause of death globally, killing three million people in 2016—more than any other communicable disease, and all maternal, neonatal, and nutritional conditions combined (WHO 2018c). The "big five" LRI named by the Forum of International Respiratory Societies with the WHO are chronic obstructive pulmonary disease (COPD), asthma, acute LRI, TB, and lung cancer (FIRS 2017).

Unlike other infections, "chronic" LRI does not refer solely to infections with a long duration. Rather, in the clinical literature, *infectious* LRI are acute or chronic, such as bacterial bronchitis (Montanare et al. 2018); but chronic LRI can have another meaning. In the clinical literature, chronic LRI refers mainly to non-infectious conditions that affect the LRS, such as emphysema and COPD (Burney et al. 2017; WHO 2019b). To not conflate infectious disease with non-infectious disease, this section only discusses trends in acute LRI (ALRI).

ALRI are diagnosed clinically by the presence of an acute cough (Mok and Simpson 1984; Moore et al. 2008) and one or more of the following symptoms: sputum (Moore et al. 2008; Hay et al. 2017), chest pain (Hay et al. 2017), dyspnea[17] (Moore et al. 2008; Hay et al. 2017), and wheezing breath (Mok and Simpson 1984; Hay et al. 2017). Other symptoms include sleep disturbance, a restriction in the types/number of activities an individual can undertake, and a general sense of feeling unwell (Moore et al. 2008).

This section examines the anatomy and function of the LRS; and explores the relationships between the health of the LRS and the health of the middle ear cleft. Following a discussion of the pathophysiology and risk factors responsible for the development of ALRI are discussed, there is a discussion of the risk factors shared by mastoiditis, OM, sinusitis, and ALRI.

[17] Dyspnea refers to shortness of breath (Moore et al. 2008; Hay et al. 2017).

Anatomy and Function of the Lower Respiratory System

The lower respiratory system includes the trachea, bronchi, bronchioles, alveolar ducts, and alveoli (Patwa and Shah 2015) (see Figure 2.6). Once air has been inhaled into the nasal cavity and been conducted through the pharynx (and potentially through the sinuses), it passes though the larynx and into the LRS. The trachea subdivides into the bronchi; and the bronchi into the bronchioles; and the bronchioles into the alveolar ducts; and so on for 23 generations until the air reaches the alveoli (Weibel 1963). All the branches after the left and right bronchi form the left and right lungs.

The trachea and bronchi are hollow pseudostratified columnar and mucosal epithelial tubes, supported by cartilaginous rings, and lined with smooth muscle and cilia. The trachea is also supported posteriorly by the *trachealis* muscle (Ugalde et al. 2007). The bronchioles that branch off of the bronchi are hollow columnar and cuboidal epithelial tubes, unsupported by cartilaginous rings, and lined with thicker smooth muscle than that in the trachea and bronchi (Patwa and Shah 2015).

To protect the lungs, they are encased within both the pleural cavity and the pleura. The plural cavity is the space formed by the ribs, sternum, and intercostal muscles laterally and anteriorly, the vertebrae posteriorly, the diaphragm inferiorly, and the cervical pleura superiorly. The inner walls of the cavity and the lungs are both covered with a thin layer of tissue called the pleura. And the narrow space separating the pleura (or the pleural space) is filled with fluid (or pleural fluid) for cushioning and ease of movement (Mayo Clinic 2020; NHS 2018b) (see Figure 2.6).

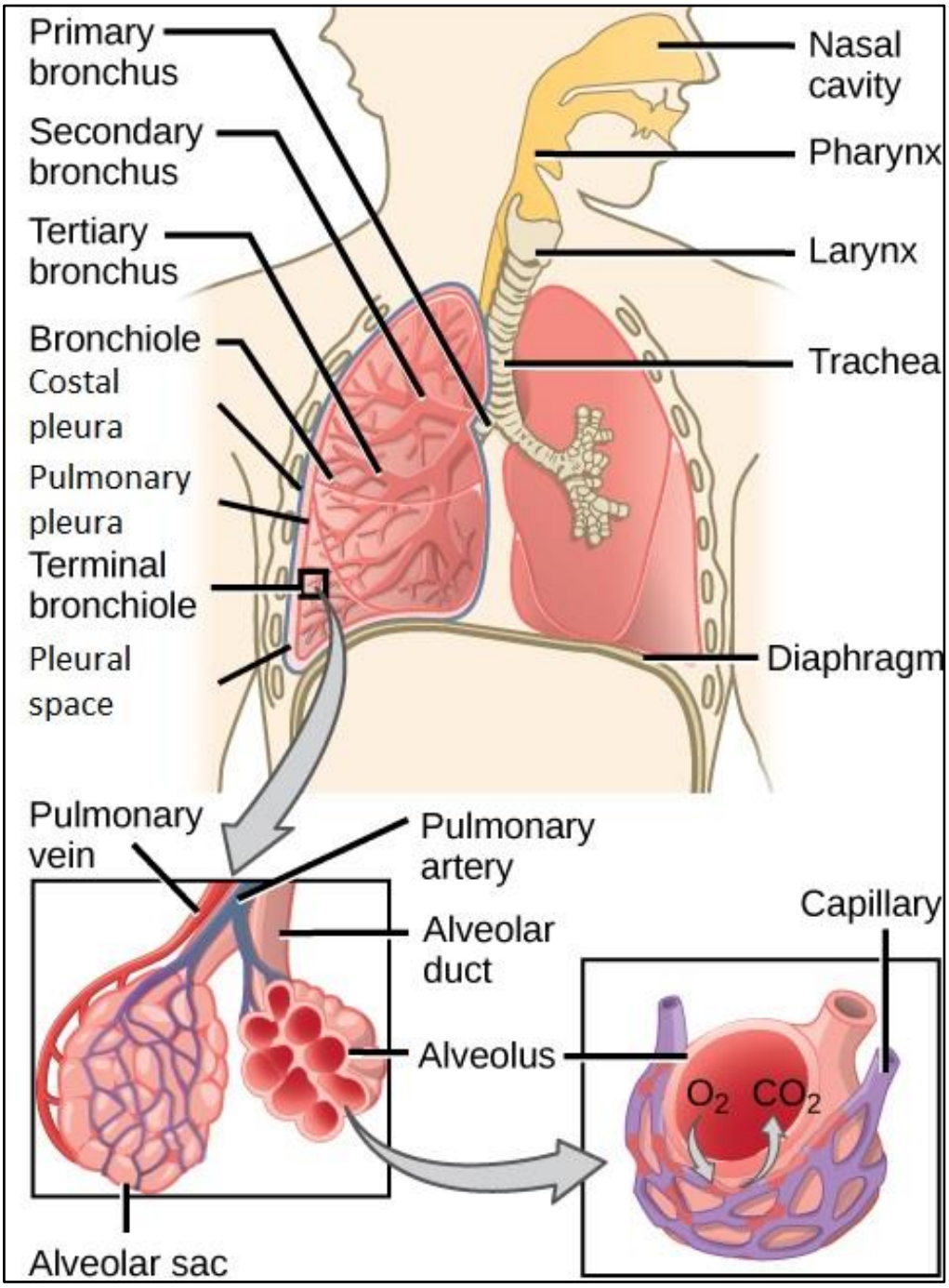


Figure 2.6 Anatomy of the lower respiratory system and pleural cavity (Molnar and Gair 2015).

The primary function of the LRS is gas exchange and this occurs in the alveoli (Patwa and Shah 2015). The alveoli are thinner and less muscular than the other segments of the LRS (Hughes and West 2008) and highly vascularized. Gas exchange occurs as a result of pressure differentials between the alveolus, pulmonary artery, and pulmonary veins (Hughes et al. 1968). The lungs maintain inflation, gas exchange function, and blood flow *via* the constant presences of a reserve volume of air therein (Kavanach and Hedenstierna 2015).

The LRS is connected to the upper respiratory system directly; however, it is not closely related to the middle ear cleft, like the sphenoid sinuses in the URS. Rather, the relationship between the middle ear cleft, sinuses, and LRS is apparent in their infectious pathology and, in some instances, their pathophysiology. The following sections discuss the pathophysiology of ALRI and the risk factors for such infections.

Pathophysiology of Acute Lower Respiratory Infections

ALRI can be viral or bacterial in origin (Niederman and Krilov 2013; Shi et al. 2015). The pathogens most often responsible for ALRI are bacteria (NHS 2018d). *Streptococcus pneumoniae* (Shi et al. 2015; FIRS 2017) and *Haemophilus influenzae* (especially type B) (Shi et al. 2015; Montaner et al. 2018) cause the majority of the pneumonia cases globally. This is different in children, with two-thirds of childhood pneumonia being a community-acquired viral infection (Walker et al. 2013; Ruuskanen et al. 2011) and most often caused by the respiratory syncytial virus (RSV) (Shi et al. 2015; FIRS 2017). Globally, a quarter of the population is infected with latent *Mycobacterium tuberculosis* (WHO 2020d).

The pathophysiology of ALRI is difficult to discuss in detail, since ALRI encompasses a host of infections, each with their own pathophysiology. Even presenting examples referring to the most common ALRI, pneumonia (FIRS 2017), is complex, as it too encompasses a host of diseases (WHO 2019b). Simply, ALRI are respiratory in origin and arise when a pathogen, usually carried in a drop of sputum, is inhaled into the LRS (NHS 2019b). LRI can also be secondary to a URI (Barthelson, et al. 1998; Chonmaitree et al. 2016).

Complications from LRI can move beyond the LRS and involve the pleura or pleural cavity: such as pleurisy[18] (or pleuritis), pleural effusion[19], and empyema[20] (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017). While empyema is rare (NHS 2018b), pleurisy and pleural effusion are more common. Pleurisy can occur in approximately half of all TB patients (Valdés et al. 1998); and pleural effusion in 15%–44% of patients with pneumonia (Chalmers et al. 2009; Dean et al. 2016).

A single case of LRI can have a long-term effect on individual morbidity. Both pneumonia and TB, for example, can damage lung function, create pulmonary abnormalities, lead to chronic respiratory disease, and increase an individual's susceptibility to future respiratory related-diseases (Johnston et al. 1998; Ravimohan et al. 2018; FIRS 2017).

[18] Pleurisy refers to the infection of the pleural cavity (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017).
[19] Pleural effusion refers to fluid buildup in the pleural cavity (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017).
[20] Empyema refers to pockets of exudate buildup in the pleural cavity (Mayo Clinic 2020; NHS 2018b; Yang et al. 2017).

In one study, one third of the patients who developed pleurisy as a result of TB subsequently developed a more severe form of TB (Valdés et al. 1998).

Adult ALRI do not share a pathophysiology with OM and sinusitis; but ALRI, OM, and sinusitis are caused by similar—and, often, the same—pathogens (Chadha and Chadha 2007; Bocian 1993; Shi et al. 2015; Torpy 2009; FIRS 2017). Childhood ALRI, however, does share the same pathophysiology as OM and sinusitis, with both usually being viral in origin (Walker et al. 2013; Ruuskanen et al. 2011). Having an URI can also increase and individual's risk of contracting an ALRI (Barthelson et al. 1998; Chonmaitree et al. 2016), while having an LRI can increase an individual's risk from further respiratory infection in general (e.g., Ravimohan et al. 2018). ALRI also share many of the same risk factors as OM and sinusitis. The remainder of this chapter explores these relationships.

Risk Factors for Lower Respiratory Infections

Risk factors for ALRI include the following: living in an overcrowded household, especially one with multiple children (Gilani et al. 2012; Rehfuess et al. 2013; Rossi et al. 2007; Simoes 2003; FIRS 2017; Wonodi et al. 2012); attending daycare or having children who attend daycare (Gilani et al. 2012; Rehfuess et al. 2013; Rossi et al. 2007; Simoes 2003; FIRS 2017; Wonodi et al. 2012); being exposed to air containing allergens and microbes (FIRS 2017), and indoor and outdoor air pollution (FIRS 2017; Gilani et al. 2012; Rehfuess et al. 2013; Wonodi et al. 2012)—especially tobacco smoke, smoke from a cooking/heating fire, and industrial toxins (Adane et al. 2020; Bulkow et al. 2012; Dagvadorj et al. 2016; Rehfuess et al. 2013; FIRS 2017); having atopy (Mok and Simpson 1984), a chronic health condition (FIRS 2017), or an HIV infection (Gilani et al. 2012; Rehfuess et al. 2013; FIRS 2017; Wonodi et al. 2012); neither being sufficiently immunized (Gilani et al. 2012; Rehfuess et al. 2013; FIRS 2017; Wonodi et al. 2012) nor practicing frequent handwashing (Rehfuess et al. 2013); being malnourished (Gilani et al. 2012; Rehfuess et al. 2013; FIRS 2017; Wonodi et al. 2012); having a low birth weight (Dagvadorj et al. 2016; Gilani et al. 2012; Rehfuess et al. 2013; Rossi et al. 2007; Wonodi et al. 2012); not being, or being insufficiently, breastfed as an infant (Bulkow et al. 2012; Dagvadorj et al. 2016; Lanari et al. 2013; Rehfuess et al. 2013; FIRS 2017); and, finally, being an infant/child or a senior (FIRS 2017). Certain LRI are also more frequent in the winter (Burney et al. 2017).

Not all the factors involved in the genesis of an infection are causative (Robb and Williamson 2018). To identify the causative factor(s), the interactions between factors must be understood. To visualize the relationships between each factor and their impact on childhood mortality from ALRI in populations from sub-Saharan Africa, Rehfuess *et al.* (2013) created a novel causal diagram using systems model theory to visualise all the risk factors

involved in ALRI (see Figure 2.7). Using this diagram, they found that "wealth, maternal education and, to a lesser extent, paternal education are highly protective against solid fuel use" (Rehfuess et al. 2013:14), which they found to be "a major risk factor for ALRI among African children" (Rehfuess et al. 2013:14). In effect, by studying the frequency of infection and analysing the associated risk factors, this project aimed to reverse engineer the diagram by Refuess *et al.* (2013) to contextualize the populations we studied.

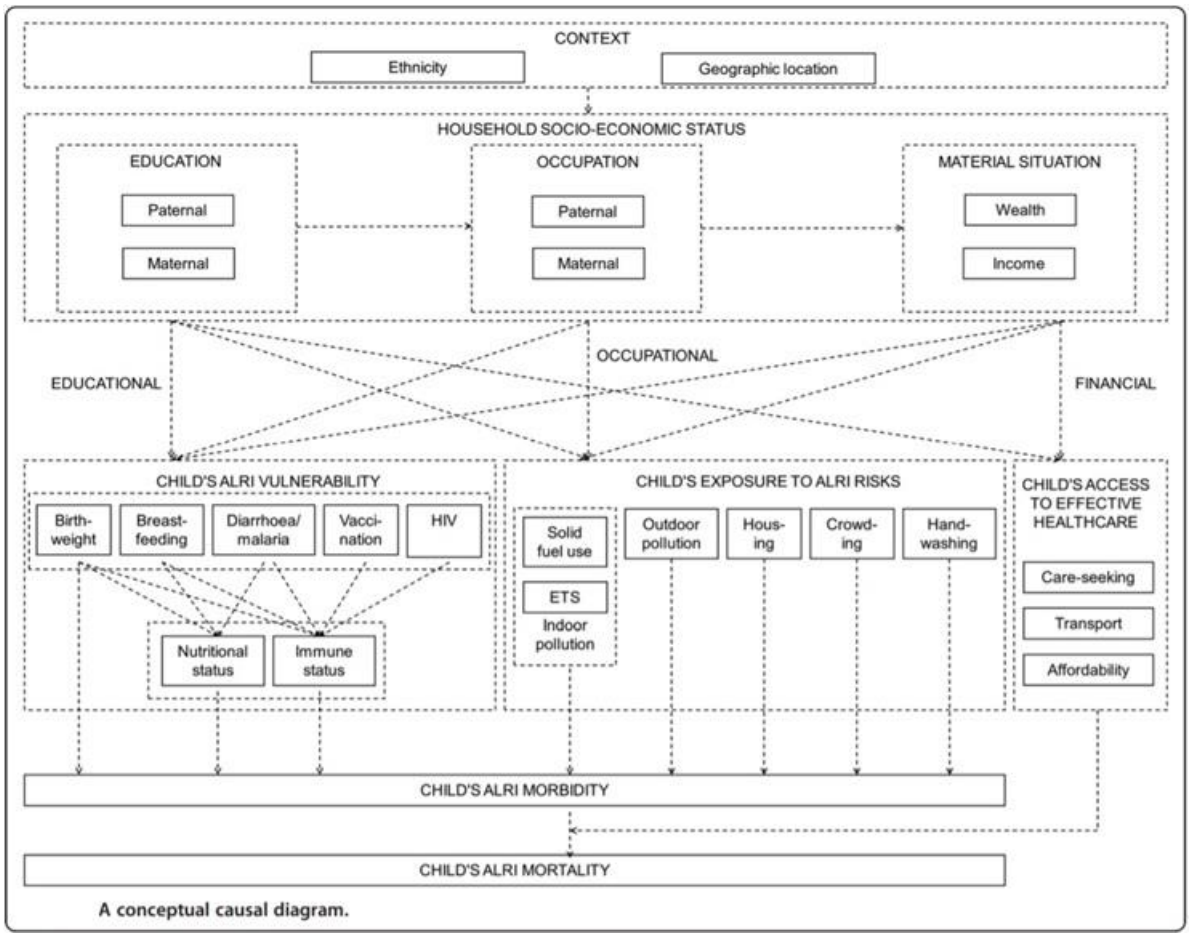


Figure 2.7 A causal diagram visualizing the factors leading to mortality from childhood ALRI (adapted from Rehfuess et al. 2013:9).

2.5 Shared Risk Factors

Mastoiditis, OM, sinusitis, and LRI share some risk factors—most notably, community-acquisition and daycare attendance, breastfeeding, and air pollution. The impact of these factors on an individual's risk of developing a respiratory-related disease is discussed from a clinical perspective in this final section. Since mastoiditis is a frequent complication of AOM and a sequela of COM, mastoiditis is included in the following discussions of OM (Bluestone 1998; Groth et al. 2012; Hellier 2018; Wilson et al. 2017).

Community-Acquisition and Daycare

OM and sinusitis have the same pathophysiology (Wald 2011). Both are usually caused by a primary community-acquired viral URI followed by a secondary bacterial infection (Wald 2011). Communicable viral URI are most common in children (especially those 3–24 months old), as are most acute sinus and middle ear infections (Chonmaitree et al. 2016; Brook 2009; Wald 2011). In one study, 61% of children with a viral URI developed AOM or OME (Chavanet 2008), while in another approximately 80% of children with a viral URI developed acute bacterial sinusitis (DeMuri and Wald 2012). While viral LRI are less common than viral URI, they are often more severe (Marom et al. 2014); and, like OM and sinusitis, they disproportionately affect children—especially those younger than five years (Niederman and Krilov 2013; Chonmaitree et al. 2016; FIRS 2017).

Being around people who have a communicable infection increases an individual's risk of contracting them. Thus, being around infants and children is one of the most critical risk factors for OM, sinusitis, and LRI (Chonmaitre et al. 2016; Uhari et al. 1996). Today, infants and children congregate in daycares, resulting in daycare attendance being one of the most critical risk factors for developing childhood OM, sinusitis, and LRI (e.g., DeMuri and Wald 2012; Holberg et al. 1993; Uhari et al. 1996).

It should also be noted that community-acquisition is a significant factor for all ALRI, since all pathogens that cause ALRI, viral and bacterial, are communicable. In particular cases, some can become epidemic or pandemic. For example, the WHO state that *H. influenzae* "leads to respiratory tract infections in 5–15% of the (global) population and severe illness in 3–5 million people" annually (FIRS 2017). The epidemic and pandemic caused by the SARS and SARS-CoV-2/COVID-19 viruses, respectively, serve as recent examples of community-acquired LRI (FIRS 2017; WHO 2020c). Thus, for viral URI and LRI, community-acquisition is a vital factor to observe, locally and globally.

Breastfeeding

Breastfeeding is key to infant health, as it provides both essential nutrients and passive immunity from the mother during a phase when the infant's own immune system is developing (Adkins et al. 2004). Breast milk contains antibodies and constituents (such as leukocytes, growth factors, hormones, and vitamins) which colonize the infant gastrointestinal tract (Cerini and Aldrovandi 2013). These function as antimicrobial, anti-inflammatory, and immunomodulatory factors (Labbok et al. 2004). The prebiotics in breastmilk, for example, promote the growth of the environment-specific intestinal microbiota necessary for optimal region-specific immunity (Palma et al. 2012; Yatsunenko et al. 2012). Breastmilk is also adaptive, as the antibodies present can change in response to

the pathogens to which an infant is exposed (Riskin et al. 2012). The efficacy of breastfeeding depends on how long an infant is breastfed—with longer, exclusive periods being the most protective (Lawrence and Pane 2007). The WHO and UNICEF advocate that infants should be breastfed exclusively for the first six months of their lives and then be breastfed in association with nutritious supplementary foods until they are two years (WHO 2018b).

Breastfeeding has a positive effect on decreasing respiratory-related infections in particular, by stimulating the lymphoid tissue that line the lungs (mucosa associated lymphoid tissue or MALT) and because it contains antibodies (such as secretory Immunoglobulin A or sIgA) specific to mediating adaptive, humoral immunity in the mucosa of the lungs (Chonmaitree et al. 2016; Cerini and Aldrovandi 2013; Victoria et al. 2016). This strengthens the mucosal immune system, which is the primary defence against inhaled pathogens (Cerini and Aldrovandi 2013). The act of breastfeeding may also help to mechanically clear airways, to some degree, *via* the acts of sucking and swallowing (Lodge et al. 2016).

Numerous studies have highlighted the protective benefit of breastfeeding on OM (Bowatte et al. 2015; Chonmaitree et al. 2016; Lodge et al. 2016; Uhari et al. 1996; Victoria et al. 2016) and, in turn, mastoiditis as a squeal of OM (Bluestone 1998; NHS 2018b; Robb and Williamson 2018). To illustrate, the longitudinal (2008–2014) study of infants and children from Texas, United States of America, by Chonmaitree *et al.* (2016) found that prolonged breastfeeding was positively correlated with significantly fewer instances of URI and AOM (Chonmaitree et al. 2016). Another study found that, if universally practiced, breastfeeding could eliminate a third of all respiratory infections (Victoria et al. 2016), with exclusive breastfeeding in the first two years of an infant's life being the most beneficial factor in lowering a child's risk of developing AOM (Victoria et al. 2016).

Air Pollution

Air pollution refers to air-suspended particulate matter (PM) 10µm in diameter or smaller (such as sulfate, nitrates, ammonia, sodium chloride, black carbon, mineral dust, and water) and/or harmful chemicals—especially ozone (O_3), nitrogen dioxide (NO_2), and sulphur dioxide (SO_2) (Beach and Hanlon 2017; McDuffie et al. 2021; WHO 2018a). PM harms and irritates the tissues in the respiratory system when inhaled, triggering and/or worsening respiratory disease (Armelagos and Barnes 1999; Jiang et al. 2016; Sunyer 2001; Trevino 1996). Fine particulate matter less than 2.5µm in diameter ($PM_{2.5}$) are exceptionally harmful, as they can not only damage and irritate the respiratory system (McDuffie et al. 2021), but they can also enter the bloodstream and lead to type 2 diabetes (Esposito et al. 2016: He et al. 2017),

cardiovascular disease (BHF 2021), cancer (CRUK 2019; Turner et al. 2020), and miscarriage in women (Xue et al. 2021). Globally, 91% of people live with air that exceeds WHO pollution guidelines (WHO 2020a) and seven million deaths are related to air pollution annually, the majority from pneumonia (WHO 2020d). In 2017, 1.05 million people died as a result of $PM_{2.5}$ generated by fossil-fuel combustion, with the most offensive fuel being coal (McDuffie et al. 2021).

Air pollutants can be divided into those found indoor and outdoor. Indoor air pollutants overwhelmingly include particulate matter and smoke from cooking and heating fires: particularly kerosene lamps; dung, wood, or coal fires; or burning in insufficiently ventilated stoves/fireplaces/rooms (WHO 2020e). Globally, women and children are disproportionately affected by indoor air pollutants, as they spend more time indoors, exposed to the pollution. Outdoor air pollutants include smoke from agriculture and waste management, and vehicle, industrial, and agricultural exhaust. Those who work outside are disproportionately affected by outdoor air pollutants. Compared to adults, infants and children are biologically more susceptible to the effects of air pollution generally, because of their naturally higher breathing rates, narrower airways, and immature lungs and immune systems (Darrow et al. 2014).

There are many ways in which air can become polluted, and all of them are harmful to human health. Three studies showcase different causative risk factors for respiratory-related disease related to air pollution in different cultural and natural environments. First, in Budapest, Hungary, living with a parent who smokes more than doubles the risk of recurrent childhood AOM (Csákányi et al. 2012). Second, in Northern Bangladesh, spending a minimum of thirty minutes by the stove a day (indoors or outdoors) is predictive of childhood RTI and doubles a child's risk of contracting an RTI (Nasanen-Gilmore et al. 2015). Third, in Northwest Ethiopia, living in a house with insufficient ventilation (specifically living in a house without a chimney, eaves space, or an improved cookstove) is most predictive of an LRI (Adane et al. 2020). Understanding the interactions between different factors helps to identify the causative factor(s) in a specific population, and by contextualising the rate of RTI in a population, the true burden of disease, and the impact of environmental risk factors on human health, can be understood.

2.6 Conclusions

This discussion of the clinical literature is intended to inform the interpretation of the lesions observed on the skeletal individuals assessed by this project and to facilitate the informed discussion of the lifeways of the individuals studied. The pathophysiology and virulence of

modern diseases was not assumed to be directly comparable to the burden of disease in past populations (Mays 2012; Ortner 2011, 2012; Ragsdale 1997; Ragsdale and Lehmer 2012; Roberts and Manchester 2005:5–6; Schultz 2001). Rather, the risk factors responsible for causing such infections were studied to inform past population's interactions and adaptations to their environments; and the clinical literature was consulted to inform our understanding of the biological processes involved in infection and their impact on the appearance of bony lesions (Mays 2012; Ortner 2011, 2012; Ragsdale 1997; Ragsdale and Lehmer 2012; Schultz 2001). While illuminating aspects of life in the past, the present study also has clinical relevance, informing the environmental and genetic theories debate by demonstrating the effect various risk factors have on diseases untreated by modern medicine.

Chapter 3
Archaeological Background

3.1 Introduction

This chapter explores the evolution of the palaeopathological understanding of the presentation and diagnosis of mastoiditis, sinusitis, and lower respiratory infection (LRI) in skeletal populations, and how the presence of these diseases in archaeological populations has been interpreted. In doing so, this chapter summarizes the context of this project, highlights themes in the literature, and discusses what this project adds to the field. Mastoiditis, sinusitis, and LRI are discussed in that order. The discussion of LRI features four non-/specific types of LRI, to illustrate that multiple diseases are considered LRI and that not all of them are currently archaeologically identified.

3.2 Mastoiditis

This section summarises the research that has been undertaken regarding the appearance and prevalence of mastoiditis in archaeological populations. It explores the limited body of archaeological research examining the methods utilised for visualising the mastoid, the interpretation of different forms of pathological change in the mastoid, and the ways in which data concerning mastoid infection have been interpreted within their archaeological and historical context. Several significant gaps are identified: the lack of an accessible, non-destructive, and consistent methodology for visualising and diagnosing mastoiditis in skeletal individuals; limited archaeological research into mastoiditis in general; and the need for

archaeologists to move on from discussing the appearance of mastoiditis, to discussing what it can tell us about the health of populations and their interactions with and adaptations to the environment.

To study mastoiditis in archaeological human remains, researchers have had to adopt methods to visualise internal bony structures. The earliest bioarchaeologists to study mastoiditis used traditional, wall-mounted X-ray systems to image temporal bones in standardised clinical planes (e.g., the Law, Stenvers, and submento-vertical planes) (Gregg et al. 1965a, b; Rathbun and Mallin 1977; Schulter-ellis 1979; Titche et al. 1981). Archaeologists have continued to follow medicine's lead and now employ CT scans and scanning-electron microscopy to image the mastoid air cells (MAC) (Collins and Jónsson 2018; Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b; Primeau et al. 2018). While employing such techniques allows archaeologists to produce high-fidelity images, it limits novel projects to those with access to such large, fixed machines. Sectioning is also a method of visualising the MAC, which provides direct access to visually observe the macro- and microscopic structure of the air cells (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b). However, sectioning is destructive and, so its application is limited to those projects with such permissions, and it is unlikely to be the preferred method if non-destructive methods are available.

Some of the earliest archaeologists to study mastoiditis, Gregg *et al.* (1965a,b) and Rathbun and Mallin (1977), used Tremble's (1934) clinical bone typology to categorise the internal structure of the temporal bone. The typology organized the MAC into four types based on the macroscopic, radiolographic, and cast appearance of the bone: pneumatized, diploic, sclerotic, and mixed. Pneumatized bone was described as a network of connected bone cells lined with a mucus membrane extending from the tympanic cavity, periosteum, epithelium, and connective tissues; diploic bone was small, tightly packed MAC lined with periosteum and filled with marrow; sclerotic bone was dense "ivory-like" bone with few to no cells; and mixed bone was a combination of pneumatic and diploic bone (Tremble 1934:173). Tremble (1934), and the archaeologists who used his typology (Gregg et al. 1965a,b; Titche et al. 1981), failed, however, to explore the biological processes which caused these formations. Similar short-falls were present in the work of Schulter-ellis (1979), who categorized six types of bone in the MAC of skeletal individuals from multiple collections (the Terry Collection of Black and White populations, the Riley D. Moore Collection of (an) Inuit population(s), and the Titterington Collection of the Illinois Mound Indigenous population) but often failed to explore how these bone types formed. Rather, many of these early studies were more concerned with identifying perceived health and anatomical differences between populations of different races than exploring either the biological process behind these changes or the impact of mastoiditis on the populations (Gregg et al.

1965a,b; Schulter-ellis 1979). As such, they failed to associate the bone categories the researchers defined with the biological processes that formed them. This limits the meaningful application of these categories and the information that can be inferred from them.

Recently, in a series of publications, a group of biologists with an interest in biological anthropology, described the various types of MAC morphology they observed in a skeletal population from early medieval Dirmstein, Germany (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b). They explored the biological processes that likely caused each bone type by sectioning and imaging the temporal bones, using macroscopic, endoscopic, and low-power, back-scattered electron, scanning-electron, and light microscopic techniques. They classified three main types of MAC: hypercellular, primary hypocellular, and secondary hypocellular. Hypercellular MAC were those which were fully pneumatized, while primary hypocellular MAC were those which did not (fully) pneumatize, because mastoiditis during childhood stunted the process. While these forms had been discussed widely in the clinical literature previously (Tremble 1934; Gregg et al. 1965a,b, 1981; Titche et al. 1981; Titche and Steele 1982; Loveland et al. 1990; Homøe and Lynnerup 1991; Homøe et al. 1994, 1995, 1996, 2001; Lynnerup et al. 2000), what had not been previously identified were three types of bone indicative of *secondary* hypocellularity, or a proliferative, rather than destructive, reaction to mastoiditis in adults. Secondary hypocellularity was represented by a combination of pathological woven bone and/or dense, sclerotic woven and/or lamellar bone occluding and remodelling the MAC (see Figures 3.1 and 3.2) (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b). Secondary hypocellularity could form in primary hypocellular temporal bones (creating a combined type of bone indicative of both childhood and adult infection) or hypercellular temporal bones (indicative of an adult infection). Sub-clinical, acute mastoiditis can also affect the bone and, as Flohr and Schultz (2009a) suggested, may have accounted for some of the cases of secondary hypocellularity they observed. Specifically, they identified various types of pathological bone proliferation (e.g., rootstock-like bone proliferation) and Howship's lacunae (a microscopic indicator of bone resorption) (e.g., Jung and Chole 2002) that, they argue, likely resulted from sub-clinical, acute mastoiditis (2009a).

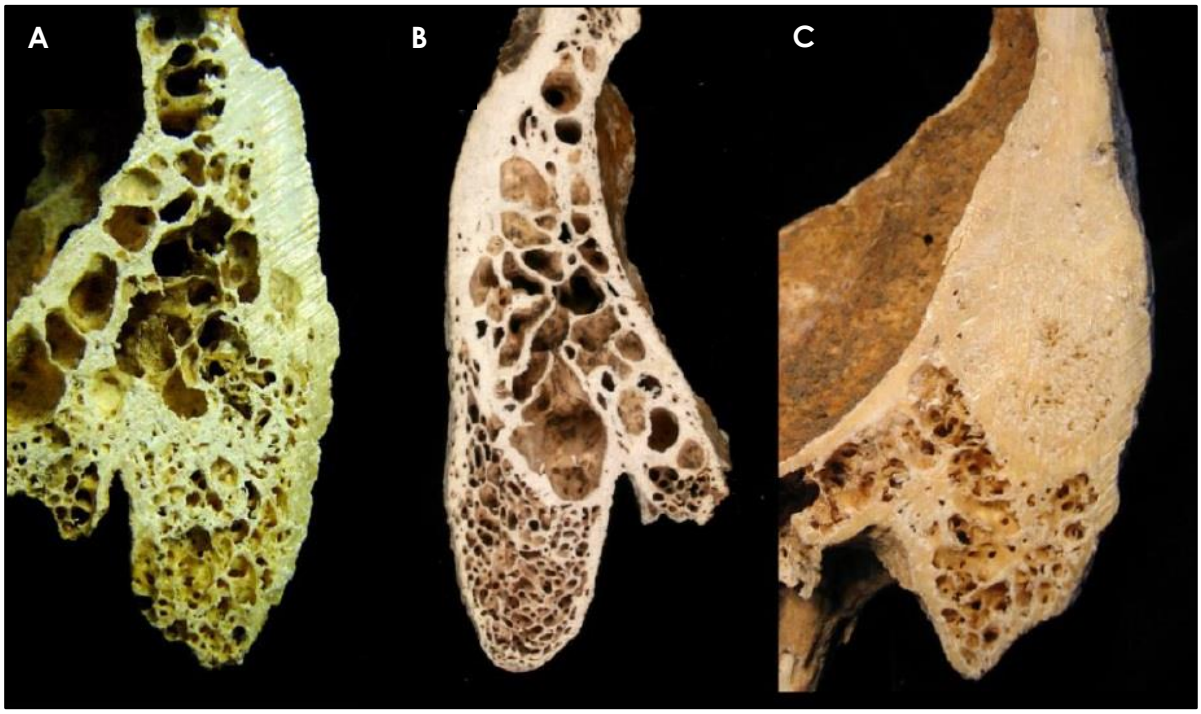


Figure 3.1 The three types of secondary hypocellularity identified by Flohr et al. (2009, 2017, 2019; Flohr and Schultz 2009a,b). **A)** Type I, in which sclerotic bone (new and remodelled woven and lamellar bone) fill and compact hypercellular (superior) and diploic (inferior) air cells; the sclerotic bone spreads to fill the MAC over time. **B)** Type II, in which a sclerotic boarder separates hypercellular air cells (superior) from diploic air cells (inferior). **C)** Type III, in which sclerotic bone, formed by a remodeled woven bone core surrounded by a border of dense trabeculae, is superior to diploic bone in the apex of the mastoid process. Image adapted from Flohr *et al.* (2017:1).

Figure 3.2 Hypercellular, or normal, mastoid air cells (Flohr et al. 2009:444).

Despite the fact that mastoiditis is a common infection in modern populations, especially amongst children (NHS 2019a; Wilson et al. 2017), and despite recent advances in the understanding of the appearance of mastoiditis in skeletal remains (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b; Homøe et al. 2001; Lynnerup et al. 2000; Qvist and Grøntved 2001), mastoiditis remains understudied archaeologically. Only a handful of projects led by archaeologists have examined mastoiditis in archaeological populations in the last two decades, all of which appear to have taken place in the last ten years (Collins 2019; Collins and Jónsson 2010; Krenz-Niedbała and Łukasik 2016b; Olivé-Busom et al. 2021; Primeau et al. 2018, 2019; Purchase 2016; Purchase et al. 2019; Zhang et al. 2020). The studies by Collins (2019), Collins and Jónsson (2010), Krenz-Niedbała and Łukasik (2016b), Olivé-Busom *et al.* (2021), Primeau *et al.* (2018, 2019), Purchase (2016), and Purchase *et al.* (2019) all examined the prevalence of mastoiditis in archaeological populations. The studies by Olivé-Busom *et al.* (2021) and Zhang *et al.* (2020) were case studies that examined one and two individuals, respectively, with fistulae in their temporal bone(s). A range of criteria was used in these studies to diagnose mastoiditis: for example, asymmetry between the morphology of the left and right MAC (Primeau et al. 2018, 2019), the degree of pneumatization/hypocellularity/sclerosis in the MAC (Collins 2019; Collins and Jónsson 2010; Primeau et al. 2018, 2019; Purchase 2016; Purchase et al. 2019), or the appearance of probable bone destruction in the MAC (Purchase 2016; Purchase et al. 2019; Zhang et al. 2020). Each of these studied used sectioning, CT, or SEM to visualise the MAC and diagnose mastoiditis, save for the studies by Krenz-Niedbała and Łukasik (2016b) and Olivé-Busom *et al.* (2021), which used X-ray imaging. Only the study conducted for my Master's research (Purchase 2016; Purchase et al. 2019) made use of portable X-ray technology.

The recent increase in archeological interest in mastoiditis is encouraging. It is our hope that mastoiditis will be explored more widely, so archaeological discussions of the disease can move from its bony appearance, to what it can tell us about the health of populations and their interactions with the environment. Indeed, mastoiditis research has a lot to offer archaeological investigation. Studying archaeological populations' interactions with the environment can provide another avenue for exploring the paleoenvironment. Most intriguingly, mastoiditis is one of only a few skeletal indicators of childhood physiological stress that remain visible on the adult skeleton: along with linear enamel hypoplasia (LEH), cribra orbitalia (CO), long bone length, and vertebral growth (Aufderheide and Rodríguez-Martín 1998; Kyle et al. 2016; Primeau et al. 2018; Swales 2012; Gowland 2015; Gowland et al. 2016; Larsen 2015; Orellana-González et al. 2020; Primeau et al. 2018, 2019; Redfern and DeWitte 2011; Roberts and Manchester 2005; Yaussy et al. 2016). As a result, a life history approach could be taken to the study of residual CM, in which the impact of childhood physiological

stress can be observed on the health of adult individuals. When paired with a study of AM, the correlation between the two infections in the same individuals could illustrate patterns in individual and population level health over lifetimes and could shed light on the effect of primary hypocellularity on individual risk of mastoiditis. To date, no archaeological project has explicitly explored mastoiditis in this way.

Two of the most recent publications by Primeau, Homøe, and Lynnerup (2018, 2019) did, however, compare the rate of hypocellularity indicative of mastoiditis (what they call infectious middle ear disease) to that of LEH and Harris lines in an urban and a rural population from medieval Denmark (1050–1536CE). The authors noted a statistically insignificantly higher rate of mastoiditis, LEH, and Harris lines in the urban, rather than the rural, population; and a significant correlation between mastoiditis and Harris lines (Primeau et al. 2018). In the same populations, they also noted an increase in mastoiditis and LEH and a decrease in Harris lines over time, corresponding to the transition from the Medieval Warm Period to the Little Ice Age (Primeau et al. 2019). They explained that all three lesions were caused by different biological processes that typically occur during different stages of skeletal development (Primeau et al. 2018). As a result, the presence of each reflected a different type of physiological stress during a different period of childhood. They concluded that the urban population was either more physiologically stressed than the rural population and lived in a harsher environment, or, paradoxically, that the urban population was more nutritionally secure than the rural population; and that both populations were affected negatively by climate change (Primeau et al. 2019). The research of Primeau *et al.* (2018, 2019) demonstrates that residual CM can be used as a marker of childhood physiological stress alongside established markers of childhood health, such as LEH and Harris lines; and that its presence reveals different information to that reveled by LEH and Harris lines, making it a valuable additional marker to study.

The current project develops on this small body of archaeological research in two substantive ways. With the development of portable imaging hardware, and a detailed bone typology indicative of distinct periods of infection, this project aimed to create an accessible, non-destructive method of imaging the MAC, and to adapt the typology of Flohr *et al.* (2009, 2017, 2019; Flohr and Schultz 2009a,b) to diagnose individuals with residual CM, AM, or no evidence of mastoiditis. An objective of this project was to analyse the presence of residual CM and AM using a life history approach, and to treat residual CM as an indicator of childhood physiological stress, to characterize respiratory-related health over lifetimes. The approach to imaging, diagnosing, and interpreting mastoiditis is unique to this project.

3.3 Sinusitis

This section discusses the bony appearance of sinusitis and summarises what has been learned by studying the presence of sinusitis in archaeological populations. This discussion contextualizes our study of maxillary sinusitis (MS) within the archaeological literature and highlights that there remain gaps in the archaeological understanding of sinusitis, such as the appearance and prevalence of sinusitis amongst infants and children, and the identification of, and the understanding of interactions amongst, key environmental risk factors for sinusitis in populations from disparate environments.

Clinicians are not concerned with the appearance or involvement of bone in diagnosing sinusitis (Boocock et al. 1995; Lewis et al. 1995). Rather, sinusitis is diagnosed clinically from soft tissue changes. Thus, paleopathologists have created their own methods and standards for diagnosing sinusitis in skeletal populations. Zuckerkandl (1882) was the first to have studied the appearance of sinusitis in dry bone, but their work is cited little in the English literature, as it is in German. Instead, Wells (1964, 1977), who published in English, is often credited in the English literature as the first (Boocock et al. 1995; Digangi and Sirianni 2017; Lewis et al. 1995; Panhuysen et al. 1997; Roberts 2007). Following the publications of Zuckerkandl (1882) and Wells (1964, 1977), other works commented on the presence of sinusitis in archaeological populations (Gregg and Gregg 1987; Lilley et al. 1994; Willey and Emerson 1993), but they neither classified the appearance of the lesions they observed, nor explored what sinusitis may have meant for the individuals' or populations' interactions with the environment.

Boocock *et al.* (1995) were the first to test a palaeopathological hypothesis concerning sinusitis at the population level and one of the first to have explored the appearance, etiology, and pathogenesis of MS in an archaeological population. They were also the first to categorise the appearance of MS in dry bone (Boocock et al. 1995; Lewis et al. 1995). Boocock *et al.* (1995) defined four types of lesions within the maxillary sinuses that reflected different stages of chronic sinusitis (used by this project and, so, discussed in detail in Chapter 4). This typology has since been used to classify chronic sinusitis lesions within all the sinuses (Krenz-Niedbała and Łukasik 2016a; Liebe-Harkort 2010; Lewis et al. 1995), though the maxillary sinuses remain those the most studied. This is primarily because the maxillary sinuses are the largest paranasal sinuses and the easiest to access visually through fractured maxillae or with the aid of an endoscope (Roberts 2007).

A close relationship has been found to exist between a population's environment and their risk level for developing sinusitis. Many archaeologists study the natural, built, and social environments of a population alongside the frequency of sinusitis to better understand the

lived experience of the population and the etiology of sinusitis (see Table 1.1). Lewis (2016:139) goes so far as to say that she considers Roberts' 2009 analysis of sinusitis (and rib lesions) in medieval England to be a contextual study of the living environment. Chronic exposure to domestic and/or industrial air pollution, over crowding, and living in under-ventilated houses and/or a cold, dry, or windy climate have been identified as risk factors for sinusitis in archaeological populations which irritate the upper respiratory system (URI) and increase an individual's chance of contracting community-acquired infections (see Table 1.1). Varying exposure to environmental risk factors has been cited as a contributing factor behind the differences in demographic disease levels within and between archaeological populations.

To illustrate, Davies-Barrett *et al.* (2021) found 93.8% of individuals from Late Intermediate Period (1000–1476CE) Pachacamac, Peru had skeletal evidence of MS (n=39). They hypothesised that air pollution, overcrowding, and high levels of social mixing at a holy site of pilgrimage put the population at a high risk of sinusitis. Alternatively, Purchase (2016) found 40.4% of individuals from Late Neolithic (6000/5800–5200BP) to Early Bronze Age (5200/5000–3400BP) Lokomotiv and Ust'-Ida I, Siberia, Russia had skeletal evidence of MS. They hypothesised that the cold climate and exposure to wood smoke in poorly ventilated dwellings placed individuals at risk of sinusitis. These individuals were hunter-fisher-gatherers, and, so, experienced less social mixing than those from the religious site of Pachacamac. Their genetics, and interactions between other biological and environmental factors, appear to have placed those from Pachacamac at greater risk of experiencing sinusitis than those from Lokomotiv and Ust'-Ida I. By studying the prevalence of sinusitis in individuals from disparate environments, archaeologists have better understood the impact that environmental factors have had on human health and have identified factors that appear to have placed individuals at the greatest risk of sinusitis. For example, in her multi-site, multi-period analysis of MS, Roberts (2007:804) concluded that "people who hunted and gathered, or who lived in a rural environment and practiced agriculture, or who had high status, were less susceptible to sinusitis in the past" compared to those from other built, social, or natural environments.

The study of sinusitis highlights the benefits of holistic, contextual respiratory-related disease research. It has contributed greatly to the understanding of community-level experiences of health within the built, social, and natural environments, while also augmenting the clinical understanding of the pathophysiology of sinusitis in bone. In many ways, the study of maxillary sinusitis sets the bar for other palaeopathological respiratory-related disease research, including the study of mastoiditis.

3.4 Lower Respiratory Infection

This section discusses the appearance of LRI on the ribs with reference to four non-specific and specific infections. As with mastoiditis and sinusitis, the etiologies of LRI are clinically well understood, but how the bone is involved is not. Thus, the archaeological understanding of LRI has been evolving independently from the clinical understanding, with the latter largely focused on the appearance of tuberculosis (TB), as it is the LRI that leaves the most distinctive bony lesions (Ortner 2003; Ortner and Putschar 1985). As such, the following discussion is dominated by research concerning TB and demonstrates how it has been studied as an indicator of broader patterns in public health. The research by Davies-Barrett *et al.* (2019) is highlighted, as she recently developed a recording method for VSRL designed to facilitate differential diagnosis. Chronic inflammation in any of the tissues underlying the ribs can trigger changes in the overlying (visceral) surface of the ribs (Aufderheide et al. 2002; Davies-Barrett et al. 2019; Holloway et al. 2011; Kelley and Micozzi 1984; Lambert 2002; Matos and Santos 2006; Molto 1992 1990; Ortner 2003; Pfeiffer 1984; Roberts 2007; Roberts et al. 1994, 1998; Santos and Roberts 2001, 2006). Many VSRL provide non-/specific evidence of LRI. However, other processes and diseases can mimic, or also form, VSRL (e.g., some taphonomic processes, cancer, hypertrophic osteoarthropathy, osteomyelitis, treponematosis infections, emphysema, and septicemia) (Anselmo et al. 2016; Aufderheide et al. 2002; Binder and Saad 2017; Lambert 2002; Roberts et al. 2016; Santos and Roberts 2001). To exclude non-infectious disease processes from diagnosis, Davies-Barrett *et al.* (2019) argued that only proliferative VSRL should be considered to be evidence of LRI, as inflammation of the pleural cavity triggers the formation of sub-periosteal bone formation on the VSRL, a feature of LRI observed both clinically and archaeologically (Cooper et al. 2016; Davies-Barrett et al. 2019; Marudanayagam and Gnanadoss 2006). Additionally, only lesions on the visceral surface of the rib shaft (and possibly extending to the rib head and angle) were considered evidence of LRI (Davies-Barrett et al. 2019). Lesions located exclusively on parts of the rib other than the visceral surface were considered likely to have originated from mechanical strain from soft tissue attachment, anatomical variation, or taphonomy; and lesions located on both the visceral and external surfaces were likely caused by trauma or a fracture callus (Davies-Barrett et al. 2019).

With appropriate differential diagnosis, some non-/specific infections can be identified from VSRL (Aufderheide et al. 2002; Davies-Barrett et al. 2019; Holloway et al. 2011; Kelley and Micozzi 1984; Molto 1990; Roberts 2007; Roberts et al. 1994; Santos and Roberts 2001). Since the early 1900s, archaeologists have been identifying rib lesions in skeletal and mummified human remains caused by TB infection (Cave 1939; Nathanson and Cohen 1941; Hrdlička

1908; Rosencrantz et al. 1941). By the second half of the century, VSRL indicative of actinomycosis and treponematosis infections were identified (e.g., Lambert 2002; Molto 1990). Other LRI, while included in differential diagnoses, were not identified in archaeological remains until the turn of the millennium (Anselmo et al. 2016; Aufderheide et al. 2002; Binder and Saad 2017; Roberts et al. 2016; Santos and Roberts 2001). To explore LRI research in more detail, this section summarises the appearance and etiology of particular LRI (TB, pneumonia, bronchitis, and actinomycosis infection causing empyema) that can be diagnosed from skeletal evidence; and discusses what the presence of these lesions in a population means about population health.

Tuberculosis

Today, TB infection is a major cause of LRI and one of the top ten causes of death globally (World Health Organization 2018). TB is caused by bacteria from the genus *Mycobacterium*, with *M. tuberculosis* and *M. bovis* most likely to infect humans (Mays et al. 2002). The former is a human infection transmitted through the inhalation of infected droplets. The latter is a zoonosis carried by cattle that can be transmitted to humans through the consumption of infected dairy products and meat, or, like *M. tuberculosis*, the inhalation of infected droplets (O'Reilly and Daborn 1995).

Ribs were studied more often than other bones in the earliest archaeological studies of TB. For example, in their study of the Hamann-Todd Osteological Collection, Kelley and Micozzi (1984) claimed that they increased their sample size by a factor of two by only studying VSRL rather than also examining vertebrae. Their argument was that Pott's disease[21] only develops in 7% of TB infections, while chronic pulmonary disease occurs in 90% of individuals with active TB. Thus, studying rib lesions alone yielded more results (Kelley and Micozzi 1984). In particular, they found the 4th–8th ribs were affected by VSRL indicative of TB more than other ribs (Kelley and Micozzi 1984). However, this study relied too heavily on the collection's medical records. It treated the recorded cause of death as fact and did not consider that TB may have been confused with other LRI, that individuals may have been suffering from multiple infections, or, to avoid stigma, that a TB infection may have been disguised by the patient, or the patient's family, as another LRI (as discussed in Santos and Roberts 2001).

Further, Roberts *et al.* (1994) also analysed VSRL in individuals with a known cause of death—this time using the Terry Collection. In comparing individuals' ages, biological sexes,

[21] Pott's disease refers to lytic lesions on the anterior portions of the thoracic and lumbar vertebrae and, ultimately, the collapse (kyphosis) and fusion of said vertebrae (Ortner 2003).

and the placements, number, and extent of rib lesions with individuals' causes of death, the researchers were able to conclude that individuals who were recorded as having died from TB were more likely to have VSRL (61.6% or 157/255) than those who reportedly died from something else (15.2% or 165/1086). They suggested that those who were recorded as having died of other causes could also have suffered chronically with TB, inflating the latter count. They found that VSRL were most frequently found on the 1st–4th ribs and on the rib head, neck, or angle, and that individuals who died of TB were found to have died younger than those who died of other causes. In support of Kelley and Micozzi's (1984) argument, they also found that few of the individuals with VSRL had Pott's disease (Roberts et al. 1994).

Over time, archaeologists began to argue that to accurately differentiate TB induced VSRL from other VSRL, other skeletal features had to be considered: specifically, Pott's disease (Ortner 2003); vertebral hypervascularization (Maczel 2004); inflammatory induced lesions on the articular surfaces, long bones, endocranial surfaces, and phalanges (Maczel 2004; Steinbock 1976); and the ossification of the pleura (Lombardi and Caceres 2000). Mays *et al.* (2002), for example, failed to link VSRL with molecular evidence of TB. They found aDNA from the *M. tuberculosis* family in one sample, but also in two of their controls. Thus, they concluded that the presence of rib lesions alone was not sufficient to diagnose TB in the population they studied. Caution should be taken when making general assumptions about the bony appearance of TB, however; as some researchers have noted changes in the pathophysiology of TB over time (Holloway et al. 2011; Pedersen et al. 2019b). After reviewing 530 archaeological cases of TB from 221 sites around the world from 151 references, Holloway *et al.* (2011) discovered that following the advent of agriculture, the location of TB induced lesions changed from the vertebrae to other bones, such as the long bones and the bones in the joints, hands, and feet (Holloway et al. 2011). Similarly, Pendersen *et al.* (2019b) noted an increase in VSRL indicative of TB from the medieval to the early modern period in populations in Ribe, Denmark. In general, TB has been found to affect the left ribs more frequently than the right at a ratio of 2:1 (Kelley and Micozzi 1984; Lambert 2002; Santos and Roberts 2006); and the upper ribs more than the lower ribs, particularly those above the 8th rib (Kelley and Micozzi 1984; Roberts et al. 1994). Ultimately, the differential diagnosis of TB in dry bone is difficult and requires the examination of many bones or the isolation of the pathogens, to be certain of a diagnosis. What is more, it is only symptomatic in 5–15% of cases (World Health Organization 2018) and only 3–8% of these involve the skeleton in some way (Ortner 2003; Ortner and Putschar 1985; Resnick and Niwayama 1995; Roberts and Buikstra 2003). Thus, the number of skeletal individuals with lesions indicative of TB underestimates the number of infected individuals within a population.

Despite the problems inherent in diagnosing TB its presence in a sample can be an effective indicator of wider issues affecting a population's morbidity, as it is an opportunistic infection (Lambert 2002; Lewis 2016; Pedersen et al. 2019b; Van de Vijver 2018). For instance, in a medieval and post-medieval Belgian cemetery, Van de Vijver (2018) found that more people from plain earth burials (interpreted as reflecting a poorer socio-economic background), and significantly more adolescents and males, presented with TB lesions than those buried in other grave types, those of other ages, or females, respectively. Thus, the author concluded that poor, adolescent, dependant males—likely suggestive of apprentices, servants, and immigrants—were more physiologically stressed and, therefore, more susceptible to TB than the rest of the population (Van de Vijver 2018). Recently, Pedersen *et al.* (2019b) studied the prevalence of TB amongst medieval and early modern skeletal populations from Ribe, Denmark and similarly found that those of low social status (based on the historic status of the cemeteries) were more likely to have TB than those of higher social status. Through their studies of TB, both Van de Vijver (2018) and Pedersen *et al.* (2019b) identified groups whose lived experiences were unique to their contemporaries and likely reflective of deprivation.

Similarly, Holloway *et al.* (2011) studied TB as an indicator of poor public health and were able to identify historic trends which greatly impacted public health. They reported that there had been a decrease in the frequency of TB infection over time, significantly so between the pre-urban (pre-800 C. E. in northern Europe) and early modern (post-1500 C. E. in northern Europe) time periods. The authors hypothesised that this was due to communities developing a more robust immunity to TB; a decrease in the virulence of TB due to a relaxation in natural selection processes; or a reduction in the number of people developing bone lesions. They concluded that some of these changes may have been attributable to a decrease in physiological stress and an increase in sanitation in the early modern period.

TB is the most heavily researched specific LRI in archaeological populations. Its study has revealed a lot about the diversity of health experiences in past populations and in populations over time. The following non-specific and specific LRI are less commonly researched, but their discussion demonstrates the ways in which archaeologists research the bony appearance of disease and how the understanding of disease evolves over time.

Pneumonia

Pneumonia is the infection and inflammation of the alveoli of the lungs (WHO 2019). Though it is, today, the most common infectious cause of death in children globally (WHO 2019) and is likely to have been a common infection in the past (Roberts et al. 2016), it is not known to leave prominent lesions on the ribs (Aufderheide et al. 2002; Kelley and Micozzi 1984; Roberts

et al. 2016). Thus, few archaeologists have studied its bony appearance or identified it in differential diagnosis. This subsection summarises the results of three studies which have.

First, Kelley and Micozzi (1984) found only two individual whose cause of death with pneumonia with VSRL in the Hamman-Todd Osteological Collection. Only one (1/385) presented with sub-periosteal VSRL (on the 3rd–12th left ribs) and another with minor pathological alterations on two ribs. The authors argued that the pneumonia diagnosis was unreliable, as no autopsy was performed post-mortem to confirm the individual's cause of death. Thus, the lesions on the skeleton of the first individual may more likely have been the result of TB, and not pneumonia, as pneumonia did not appear to affect the ribs of individuals included in the population.

Second, Aufderheide *et al.* (2002) X-rayed the chests of eight mummies from the AZ-75 Cemetery in the Azapa Valley, Chile and found soft tissue lesions (chronic empyema) indicative of silicate pneumoconiosis. This led the authors to hypothesise that the individuals died from complications relating to acute pneumonia. While soft tissue lesions were, they concluded, indicative of pneumonia, the bony lesions were not. They did not correspond with the overlying empyema and were, instead, the results of another LRI (Aufderheide et al. 2002).

Third, Roberts *et al.* (2016) considered chronic pneumonia in their diagnosis of VSRL in a post-medieval British individual with phossy jaw. Unfortunately, the rib lesions were not the focus of this study and so, multiple diseases were proposed for the rib lesions (e.g., chronic bronchitis and cancer), but these were not investigated further.

Bronchitis

Bronchitis, like pneumonia, is not often diagnosed archaeologically (e.g., Roberts et al. 2016; Santos and Roberts 2001) and definitive diagnoses have depended on the cause of death being known (Santos and Roberts 2001). In analysing TB in the Coimbra Identified Skeletal Sample, Santos and Roberts (2001) examined two individuals whose cause of death was recorded as chronic bronchitis. They found that the texture, porosity, and location of the VSRL was different to that in individuals who were said to have died from TB. Because this is a study of TB, however, the VSRL in these two individuals are not described beyond being more porous than the other VSRL observed. The authors warn that TB was often considered to be a taboo disease, so some individuals who are recorded as having died from other LRI, such as bronchitis, may have died from a hidden case of TB. As a result, even these cases are not certain (Santos and Roberts 2001).

Actinomycosis Infection Causing Empyema

Chronic actinomycosis infections are rare today (NHS 2017) and only one archaeological project reports to have diagnosed the infection in antiquity (Molto 1990). The bony lesions are described as having been caused by a "chronic suppurative infection" (Molto 1990:443) (empyema) in the right lung, caused by an *Actinomyces israelii* infection. The VSRL are present on multiple right ribs and are characterised by the concomitant destruction and thickening of the cortex. They appeared as sub-periosteal bone growth penetrated by vascular channels and foramina. Molto argued that the involvement of the right lung (and ultimately, the right ribs) was characteristic of *A. israelii* infection. However, the majority of the left ribs are absent; thus, it is impossible to conclude that the lesions affected the right ribs more than the left. Since these lesions remain uncorroborated, they should not yet be considered typical of Actinomycosis infection causing empyema.

When collecting data regarding VSRL, Davies-Barrett *et al.* (2019) encouraged collecting data at the level of the rib section (neck, angle, and shaft) to facilitate possible current and future differential diagnoses of LRI. Data was collected in this way to facilitate the analysis of true prevalence rates of VSRL and future differential diagnoses. Since there currently is a lack of differential diagnostic methods for assessing the etiology of certain VSRL, this project considered VSRL to be an indicator of non-specific infection and respiratory health. Being unable to diagnose specific LRI was not detrimental to the project, as the presence of any LRI communicated much regarding individual symptoms and community level risk.

3.5 Conclusions

The palaeopathological study of respiratory-related infection has been neither consistent nor exhaustive. Much attention has been paid to specific infections such as tuberculosis and infections in relatively easily viewed anatomical areas like the maxillary sinuses. Less attention has been paid, however, to non-specific infections, such as mastoiditis, that require imaging or sectioning to view. Still, such lesions offer a wealth of potential information and should be analysed more frequently to construct a clearer picture of community lifeways. Researchers should be encouraged to engage with clinical practitioners to foster collaborations; to develop and follow replicable data collection methods to enhance comparability between projects; and to study respiratory infections whenever possible to broaden the understanding of the human experience and the health implications of critical factors such as urbanization and poverty. The aim of this project is to address each of these requirements. In the next chapter the materials and methods selected to undertake the study are presented.

Chapter 4
Materials

4.1 Introduction

For this project, two skeletal populations were strategically selected for their excellent preservation, large population size, and accessibility, both being curated in the Department of Archaeology Human Osteological Collection at the University of Sheffield. These populations are from the Black Gate Cemetery, Newcastle-upon-Tyne (also known as Black Gate) and the Church of St. Hilda, Coronation Street, South Shields (also known as Coronation Street) (see Figure 4.1). The two populations also represented two periods of history in England's north-east during which exposure to risk factors associated with respiratory-related disease would have drastically varied. Information regarding the archaeological and historical context of each population is summarised in this chapter, to provide context for this project. Extra information beyond site character and demography is also presented below. Specifically, evidence for social status/class, housing, occupation, and nutrition.

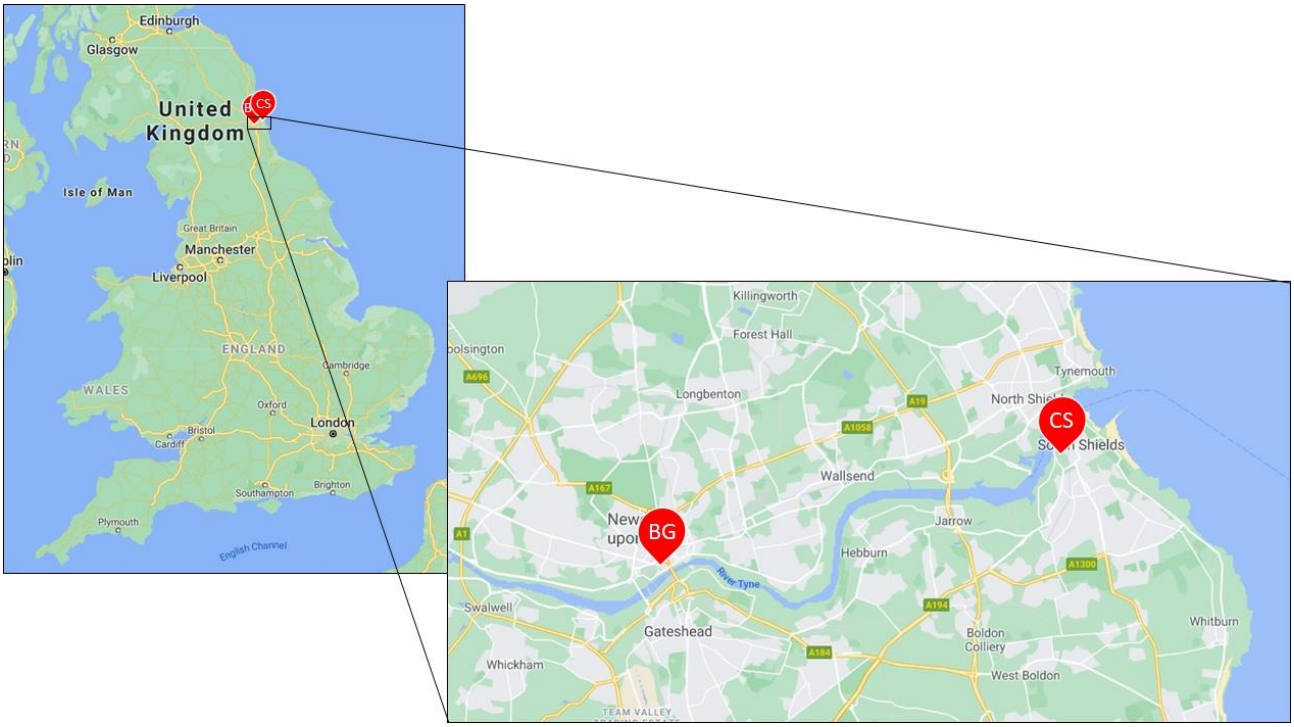


Figure 4.1 The position of the sites within England and in the county of Tyne and Wear. BG is Black Gate and CS is Coronation Street (maps generated using Google Maps).

4.2 Biological Sex and Age at Death Estimation

This project makes use of biological sex and age at death estimations established by Swales (2012) and Raynor *et al.* (2011), for the Black Gate and Coronation Street populations, respectively. The methods these researchers used to arrive at these estimations are summarised here and the number of individuals from each biological sex and age at death are presented below. As Swales (2012) emphasises, it should be noted that age at death estimations do not reflect individual's cultural identify: for example, individual status and responsibilities. Rather, age at death estimations reflect biological age and have no baring on cultural perception. Similarly, the binary biological sex estimation (male/female) is neither biologically accurate (Karkazis 2019; Griffiths 2018; Vosberg et al. 2021), nor reflective of the genders recognized by cultures throughout history (e.g., Dezhamkhooy and Yazdi 2013; Liao et al. 2012; Power 2020). In both the Anglo-Saxon/Saxo-Norman and Industrial periods, British Christian culture promoted two exclusive genders—male and female—which corresponded with binary biological sex presentation (Talvacchia and Larrimore 2014). This binary aligns with the binary estimation used to determine biological sex archaeologically. So, while we cannot be certain that the biological sex estimations reflect the gender of the individuals studied, they may reflect the socio-cultural identity they were ascribed. It should be noted, however, that medieval individuals did recognize intersex and gender-queer individuals as representing a third gender, as discussed in certain medical texts from the 14th century

(Whittington 2018); thus, the binary biological sex estimation used here fails to account for the true diversity in the populations.

Both Swales (2012) and Raynor *et al.* (2011) estimated the biological sex of Black Gate individuals by classifying sexually dimorphic features of the skull and pelvis, following the system established by Buikstra and Ubelaker (1994). Raynor *et al.* (2011) also made use of the classification systems described in Phenice (1969) and Bass (1987); and Swales (2012) also measured the heads of the femur and humerus, following the forensic guidelines in Stewart (1979:100). Black Gate individuals were classified as male, possible male, female, possible female, or unknown, while Coronation Street individuals were classified as male, probable male, possible male, female, probable female, possible female, and unknown. In this project, all probable and possible age groups were combined with the associated certain age group to create pooled male and pooled female classifications.

Age at death in adults, those studied here, was assessed differently for each population. For the Black Gate population, Swales (2012) observed bone degeneration on the pubic symphysis (Todd 1921; Brooks and Suchey 1990), sternal rib ends (Işcan et al. 1984; Yoder et al. 2001), auricular surface (Lovejoy et al. 1985), and teeth (Miles 1962) to estimate the age at death of adults. For the Coronation Street population, Raynor *et al.* (2011) observed the degree of bone degeneration on the auricular surface and pubic symphysis (Brooks and Suchey 1990; Buckberry and Chamberlain 2002), as well as epiphysial fusion (Scheuer and Black 2000).

Both Swales (2012) and Raynor *et al.* (2011) used 18 years as the cut-off point between adolescents and young adults; and both included a generic "adult" age group in their classification in which individuals were placed whose age at death could not be confidently estimated beyond 18+ years. This project considered those 16+ to be adults, as the bony structures studied in this project are fully developed by 16 years and, so, the appearance of these structures was not confounded by immature morphology. Thus, adolescents who were estimated to be 16+ years old were included in the analysis. However, no adolescent whose age at death was estimated to be 16+ years was included in this project. Thus, all the adult individuals herein were, in fact, 18+ years.

4.3 Black Gate Cemetery, Newcastle-upon-Tyne

The Black Gate cemetery was located on a promontory on the north bank of the River Tyne, in a place where the river naturally narrows. The earliest burials in the cemetery were contained within the walls of a Roman fort, the *Pons Aelius*, which was abandoned in the late 4th–early 5th centuries CE. One individual (SK99) was radiocarbon dated to the phase of

Roman occupation (211–357 CE) (Boulter and Rega 1993; Nolan 2010). However, stratigraphic and radiocarbon dating indicated that all the other burials dated to the 7th–12th centuries CE. The late Anglo-Saxon period is also referred to as the later early medieval period (650–1066 CE); and the Saxo-Norman period (1066–1150 CE) is also referred to as the high medieval period, despite including an additional 66 years (1066–1216 CE). Since the terms late Anglo-Saxon and Saxo-Norman are predominantly used in the literature regarding Black Gate (Nolan 2010), those terms are used here.

Little is known for certain about the community or communities that used the Black Gate cemetery in the late Anglo-Saxon period. The Black Gate cemetery had many hallmarks of an Anglo-Saxon church cemetery. For example, the remains of a potential Anglo-Saxon church were associated with the site (Nolan 2010:187), and the wide range of burial types present at the site suggested the cemetery served a range of social statuses. The high number of congenital malformations in the Black Gate population were also indicative of a small community with a close kinship structure (Swales 2018). As such, the cemetery at Black Gate may have served most or all members of multiple small, or one moderately sized, local agrarian lay community or communities. There is conflicting evidence to suggest the cemetery may have also served a religious community of monks (Boulter and Rega 1993; Nolan 2010). The monks may have been from Monkchester, a monastic town documented in some historic sources and potentially located in present-day Newcastle-upon-Tyne (see the History of the Church of Durham, 1104–1107 to 1115; the History of the Kings of England 1129; and the Life of St Oswin King of Northumbria, 644–651) (Raine 1838; Swales 2018; Simeon 2000). During this period, Newcastle-upon-Tyne may have also been home to a royal estate: *Ad Murum* (Bede in Nolan 2010). However, the presence of either Monkchester or *Ad Murum* is not supported archaeologically (Nolan 2010).

While the specific community served by the Black Gate cemetery remains little understood, the history of the region in a general sense is much better known. The 7th century appears to have been a golden age for the kingdom of Northumbria (Hawkes and Mills 1999; Petts and Turner 2011). The former kingdoms of Bernicia and Deira consolidated their forces to form the new kingdom of Northumbria under the leadership of the joint *Bretwalda* (or Lord of Britain); and the conversion to Christianity spurred on the founding of religious houses across the kingdom. Even after becoming part of the kingdom of England in 948, Northumbria remained a strong cultural and religious centre. This was not to say that the Northumbrian late Anglo-Saxon period was entirely peaceful. Most notably, the late 8th century saw the arrival of the Vikings on British shores. Their presence defined the late Anglo-Saxon period, as they raided and settled throughout Northumbria (Hawkes and Mills 1999; Petts and Turner 2011).

Graves from the Black Gate cemetery were characteristic of the Christian burial tradition: inhumations generally without grave goods, in an east-west alignment, and a supine-extended position (Boulter and Rega 1993; Nolan 2010). They were predominantly earth-cut; though, both elaborate (stone built or rubble-lined cists and wooden chest burials) and elaborate variations (pillow stones, earmuff stones, head boxes/head cists) were also present (Buckberry 2007; Craig-Atkins 2012; Nolan 2010; Swales 2018). Intercutting and the cramped nature of some of the burials, and the non-gendered nature of the grave types, body positions, and spatial positioning within the cemetery seem to have reflected familial burial areas and the familial-based social structure of the late Anglo-Saxon period, which valued children, women, and men as members of distinct households (Sayer 2013; Scofield 2015; Swales 2018).

In Area C (see Figure 4.2), there was a high concentration of infants, children, adult males, and stone-built cist burials (Craig-Atkins et al. 2018; Swales 2018). There were also ten adult (30+ years) male and unsexed individuals with osteological evidence of gout who were buried in Area C (Boulter and Rega 1993:33). Gout is an acquired disease caused by a diet high in fat and alcohol (NHS 2020a), typical of elite status or monastic life (Swales 2012). The high concentration of adult males and stone-built cist burials, and the presence of individuals with gout, is possibly suggestive of a segregated elite and/or monastic area of burial (Swales 2018). Additionally, the high concentration of infant and child burials next to the church is consistent with the practice of eaves-drip burials, as seen at Raunds Furnells (Northamptonshire), Cherry Hinton (Cambridgeshire), and Spofforth (North Yorkshire) (Craig-Atkins 2014; Craig-Atkins et al. 2018). In sum, Area C appears to have been a segregated high status section of the cemetery. Whether it contained monastic burials is uncertain, but the demography and high concentration of elite burials in this area is consistent with contemporary mixed cemeteries with segregated sections, such as that at Wearmouth, which appears to have contained three distinct burial areas, including a monastic area, a general lay area, and a high status lay area (Cramp 2006). That the area coincided with eaves-drip burials suggests that those infants and children interred therein were honoured in their proximity to the church and association with elite burials.

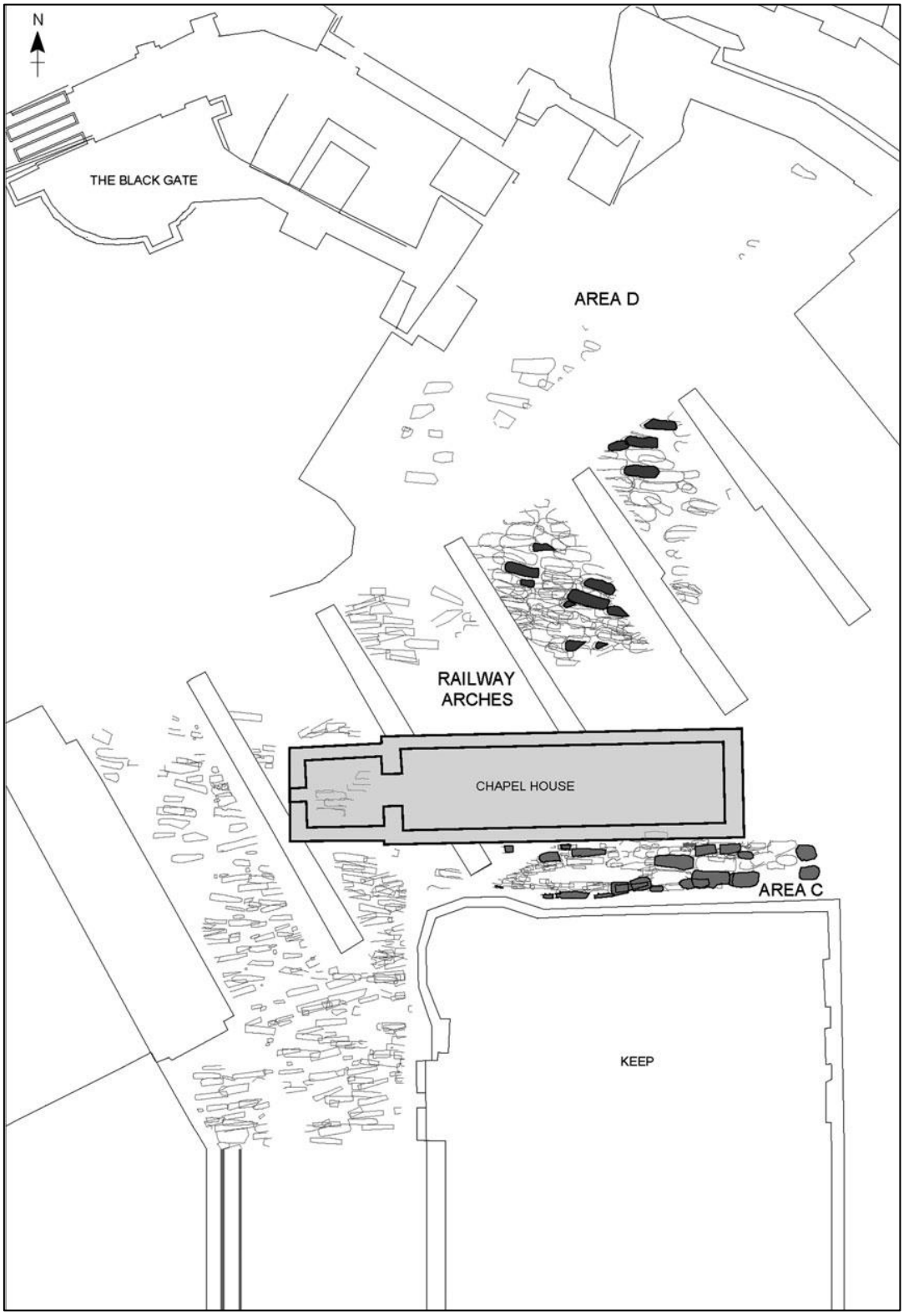


Figure 4.2 The Black Gate cemetery, with individual burials shown and elaborate burials highlighted in dark greys (image by Swales 2018 adapted from Nolan 2010).

In the autumn of 1080, following the Norman Conquest of England and the subsequent "Harrying of the North", Robert Curthose, son of William I, built an earthwork castle over the cemetery, enclosing the cemetery and its associated stone church within the castle's bailey (Nolan 2010). Although some burials were destroyed by ditches related to the castle and

ramparts, the cemetery continued to be used, likely for significant religious authorities and those who lived in the castle compound (Boulter and Rega 1993). In 1168–1178, local rebellion and a prolonged border war with the Scots appear to have led Henry II to replace the wooden keep with one made of stone. Construction destroyed much of the remaining cemetery and largely signaled the end of burial at the site (Boulter and Rega 1993; Nolan 2010).

Stone-built cists with recumbent head and foot stone markers appear to have been indicative of Saxo-Norman burial (Boulter and Rega 1993). Radiocarbon dating put burials of this style at the later end of the site's use (Nolan 2010); this burial style also first appeared at other Northumbrian sites during the Saxo-Norman period (Swales 2018). However, some graves of this style were partially destroyed by the 12th century construction of the stone keep (Nolan 2010). This implied either that stone-built cists were also used in the late Anglo-Saxon period; or that those building the keep did not consider, or did not care to preserve, earlier Saxo-Norman burials.

The cemetery was excavated over thirteen seasons (1973–1992) by Newcastle City Council as part of a larger excavation preceding masonry consolidation and landscaping (see Figure 4.3). Burials dating to the medieval period were intercut and disturbed by a variety of later activities, including, most significantly, by arch footings for a mid-19th century railway viaduct (Boulter and Rega 1993; Nolan 2010). Because of the extensive destruction, it was impossible to estimate the cemetery's original size (Nolan 2010). Regardless, Black Gate is the largest Christian Anglo-Saxon and Saxon-Norman single-site assemblage excavated in the north-east of England.

Figure 4.3 A modern photo of the castle in Newcastle-upon-Tyne (McNaughton 2007). The Black Gate is in the foreground and the castle is in the background, with the railway bridge cutting between the two.

The skeletal population is curated by the Department of Archaeology at the University of Sheffield and has been the subject of numerous archaeological studies (Boulter and Rega 1993; Nolan 2010; Swales 2012). The remainder of this section summarises the demographic profile of the Black Gate population. The following subsections discuss the demography, social status, housing, occupation, and nutrition of the Black Gate population in more detail.

Demography

There are 464 Black Gate individuals housed at the University of Sheffield and 263 were included in the present study. Only adult (16+ years) individuals were included in this project. Swales (2012) divided individuals into eleven age groups and five biological sex groups, including an unknown group for both age and biological sex. Poorly preserved individuals who lacked diagnostic skeletal traits were not assessed for their biological sex (Nolan 2010). Of the entire population, 56.7% (263/464) were adults; and 45.0% (209/464) and 6.3% (29/464) were confidently and tentatively biologically sexed, respectively (Tables 4.1 and 4.2). For this study, the confident and tentative groups pooled (pooled males 26.3%, 122/464 and pooled females 25.0%, 116/464). A large number of non-adults and biologically female individuals present in the population suggested the cemetery was predominantly, if not entirely, used by a lay community (Nolan 2010). The age at death distribution was consistent with an attritional

cemetery, with death peaking first in young childhood and again amongst senior adults, a trend typical of Anglo-Saxon populations (Swales 2012).

Table 4.1 Ages of all adult individuals in the Black Gate population (Swales 2012). The generic 'Adult' group included individuals whose age could not be determined specifically but were known to be 18 years or older.

Group Name	Age Range	Number of Adults
Young Adult	16–25 years	33
Prime Adult	26–35 years	70
Mature Adult	36–45 years	69
Senior Adult	45+ years	88
Adult	18+ years	3
Total	All	263

Table 4.2 Biological sexes of the adult individuals in the Black Gate population, including the total number of individuals included in each group (Swales 2012).

Biological Sexes	Total in Population	Pooled Sexes
Male	108	122
Probable Male	14	
Female	101	116
Probable Female	15	
Unknown	25	25
Total Adults	263	263

Social Status

Later Anglo-Saxon (8th–11th centuries) society was hierarchical, with kings concentrating power and ruling over lower classes of (in descending order) *ealdormen* (non-hereditary noblemen), *thanes* (leaders), *ceorls* (free people), to *theows* (slaves) (Ryan and Higham 2013:125–77; Harrison 2016; Wright 2015). Locally, kingdoms were broken into households (also referred to as home farms, kin, or *familia*) (Stanley 2008; Wright 2015). This extended grouping included all classes who laboured for a lord and social significance was extended to lower classes through their relationship with a household (Stanley 2008).

At a basic level, the investment of time, money, and expertise in the creation of an elaborate grave strongly suggests a reflection of the deceased's status in their burial. However, inferring social status from grave type should be done with caution, as grave type

has the potential to represent a distorted picture of the status of the individual buried therein (Craig-Atkins and Buckberry 2010:128). It has been argued, for example, that the burial of later Anglo-Saxon children in prominent locations in cemeteries does not reflect the high status of the young individual; but, rather, that of the household they were a part of (Hadley 2010:109–10).

There were five body positions and eight grave types identified in the Black Gate cemetery, with styles ranging from plain to elaborate (Swales 2018). The body position and grave type was recorded for each Black Gate individual and the distribution of adults buried in each position and grave type is summarised in Tables 4.3 and 4.4. All age groups, save adolescents, were buried in elaborate graves (Swales 2012), suggesting achieved status did not dictate an individual's grave type. However, when studied along with other evidence, such as that obtained *via* osteological investigation, grave type can be cautiously used to examine social status. For example, while Swales (2018) noted that individuals from all grave types had a similar mortality profile, there was consistently more physiological stress amongst individuals from elaborate graves than from any other grave type. She hypothesised that elaborate variation graves contained individuals of a particular form of intermediate social status. These individuals had different lifeways to the rest of the population and/or were more robust, and so, they survive acute disease and formed bony lesions indicative of chronic physiological stress.

Table 4.3 Body positions of the adult individuals in the Black Gate population.

Body Position	Total in Population
Flexed	3
Left Side	12
Right Side	56
Prone	6
Supine	164
Unrecorded	22
Total Adults	263

Table 4.43 Grave types of the adult individuals in the Black Gate population.

Grave Type	Total in Population
Chest	1
Cist	7
Coffin	32
Earmuffs	4
Probable Earmuffs	1
Head Box	3
Pillow Stone	2
Rubble Cist	2
Plain burial	186
Unrecorded	25
Total Adults	263

Housing

Later Anglo-Saxon households were focused on a hall, a rectangular timber structure with a thatched roof, a main hearth, with smaller rooms leading off one or either end (Addyman 1972). Excavated later Anglo-Saxon halls have demonstrated varying construction methods (Brennan and Hamerow 2015:345), but all share a similar layout and demonstrate a propensity for having been built of timber (Crewe 2011). Examples excavated in the kingdom of Northumbria include those at Yeavering (Addyman 1972:284; McBride 2020:1; Pearson 1998) and Thirlings (McBride 2020:1). Halls are believed to have been flexible spaces, used for ceremony, eating, sleeping, and perhaps working (Crowley 2003:8), although there are a range of other buildings that are encountered in early medieval settlements that may have also provided living and/or working accommodation (Addyman 1972; Crewe 2010:7). These include smaller timber structures which were built a few feet into the ground. The dirt floor of these sunken feature buildings may have served as a cellar below ground-level floorboards or as the floor of the building itself (Addyman 1972; Crewe 2011; Tipper 2004).

Occupation

Farming was the primary task undertaken by most late Anglo-Saxon individuals (Gilani et al. 2012; Rehfuess et al. 2013; Rossi et al. 2007; Ryan and Higham 2013:420–2; Simoes 2003; FIRS 2017; Wonodi et al. 2012). The so-called Medieval Agricultural Revolution took place during the late Anglo-Saxon period and saw the transition from grazing to open field, small-scale, intensive cereal farming (Hamerow et al. 2019). Later Anglo-Saxon industry also included ceramic production; copper, bronze, gold, silver, lead, and iron smelting, smithing and

minting; and wool and linen production, processing, and weaving (Hall 2018; Ryan and Higham 2013:145–7). Excavations at early medieval towns have uncovered evidence of craft activities localized to specific streets or neighbourhoods, such as woodworking and metalworking at Coppergate in York (Mainman and Rogers 2004:469; Ottaway 1992:151–3). Into the 11th century, Domesday Book records show that farming remained the dominant occupation of free people and slaves; and trades such as milling, baking, brewing, tailoring, washing, and shoemaking; and industries such as ceramics, textiles, bone and ivory, metal, and salt manufacturing were also common occupations (Gilani et al. 2012; Rehfuess et al. 2013; Rossi et al. 2007; Ryan and Higham 2013:420–2; Simoes 2003; FIRS 2017; Wonodi et al. 2012).

Nutrition

Swales (2012) concluded that the Black Gate population consumed a diet typical of the late Anglo-Saxon period, in which cereals—specifically, breads—were the staple. Critically, the population's diet appeared to have been varied enough to satisfy their nutritional requirements, as a comparison between long bone growth and dental eruption failed to show evidence of non-adult growth stunting (Swales 2018). Additionally, there was no rickets or osteomalacia noted in the adult population (Table 4.5).

Table 4.5 A summary of the pathological lesions in adults discussed in Nolan *et al.* (2017) and Swales (2012) by crude prevalence. AMTL is ante-mortem tooth loss; DJD is degenerative joint disease; JD is joint disease; CO is cribra orbitalia; PH is porotic hyperostosis; LEH is linear enamel hypoplasia; SPNBF is sub-periosteal new bone formation; TB is tuberculosis.

Lesion	Black Gate	Non-Adults	Adults
Traumatic injury	6.2% (40/643)	3.0% (6/202)	7.7% (34/441)
Trauma to the ribs	1.8% (8/454)	1.9% (3/158)	1.7% (5/296)
Dental calculus	90.3% (262/290)	53.7% (22/41)	96.4% (240/249)
Dental caries	38.0% (116/305)	26.0% (13/50)	40.4% (103/255)
Dental abscess	9.5% (29/305)	3.8% (2/52)	11.5% (29/253)
Periodontitis	NA	NA	NA
AMTL	26.2% (80/305)	2.0% (1/51)	31.1% (79/254)
DJD	75.5% (306/405)	NA	75.5% (306/405)
Appendicular JD	64.1% (252/393)	NA	64.1% (252/393)
Spinal JD	74.1% (244/315)	NA	74.1% (244/315)
Osteoarthritis	NA	NA	NA

(Table continued on next page)

Lesion	Black Gate	Non-Adults	Adults
Metabolic disease	NA	NA	NA
Rickets	NA	NA	NA
Congenital disease	NA	NA	NA
Neoplasia	NA	NA	NA
CO	33.6% (110/327)	52.4% (65/124)	22.2% (45/203)
PH	6.4% (24/374)	11.5% (16/139)	3.4% (8/235)
LEH	54.0% (122/226)	56.4% (22/39)	53.5% (100/187)
SPNBF	NA	NA	NA
Non-specific bone inflammation	NA	NA	NA
Chronic maxillary sinusitis	29.1% (107/368)	24.8% (28/113)	31.0% (79/255)
TB or respiratory infection	33.6% (74/220)	32.1% (18/56)	34.1% (56/164)
Specific infections	NA	NA	NA

Some non-adult individuals, however, presented with evidence of periods of mild stress: specifically, rickets, CO, porotic hyperostosis, LEH, and scurvy (Swales 2012). The most dangerous period for individuals was likely the period of weaning, due to increased exposure to foreign pathogens and parasites (Cerini and Aldrovandi 2013; Palma et al. 2012; Riskin et al. 2012; Yatsunenko et al. 2012) and an increased reliance on potentially nutritionally and immunologically inadequate foods, as compared to breastmilk. In the Black Gate population, individuals were weaned between six or nine months and one year of age, with some individuals not fully weaned until two years (MacPherson 2005). The weaning practices of the Black Gate population are similar to those of other later Anglo-Saxon populations, in which most children were fully weaned by approximately one year (Haydock et al. 2013).

4.4 The Church of Saint Hilda, Coronation Street, South Shields

The parish church of Saint Hilda (also known as Coronation Street) is located on Coronation Street in South Shields, Tyne and Wear, on the south bank of the River Tyne, near the coast of the North Sea (see Figure 4.1). A tidal mill dam (also known as Mill Dam Creek or River Branin) (Hodgson 1903:121) formed a physical barrier to the south of the church grounds until 1827. The cemetery is dated to the Industrial Period, with excavated burials dating specifically from c. 1750–1855. The Industrial Period (1760–1820/40) spanned the Georgian (1714–1837) and early Victorian (1837–1901) Periods. As the term Industrial was predominantly used in the literature regarding the Coronation Street assemblage, and since the Industrial Period more

closely mirrored the time scale of Coronation Street (*c.* 1750–1855) than the Georgian and Victorian Periods, that term was used here.

The Coronation Street cemetery had two distinct phases of use, separated by an archaeologically visible period of expansion (Raynor et al. 2011). The earlier horizon dated to the turn of the 18th century and included burials outside of the original cemetery bounds. These extra-mural burials were dominated by preterm and perinatal infants. Thus, they have been interpreted as the irregular burials of unbaptised individuals. The later horizon dated to *c.* 1818–1855 and was characterized by intense usage across a wider area and the inclusion of domestic and industrial waste.

In the 18th century, at the time for the earlier burial horizon, South Shields was growing rapidly with industrialization. St. Hilda's was the parish's only church (Raynor et al. 2011). Thus, as the population grew, the church building was expanded in 1753, 1786, 1810, and 1811 (Mackenzie 1834:31), as was the cemetery sometime in the late 18th century. The church sat to the south of the market square and included a glebe[22] to the east. Graves were rarely intercut, but grave shafts were often reused (Raynor et al. 2011). Single-break coffins were common; though, simple wooden boxes were also in use, presumably for infants and the poorer residents of the town. Coffin construction varied, from one layer of wood to two layers of wood, a layer of lead (inside, outside, or between the wooden boards), and upholstery fabric.

The transition between the earlier and later burial horizons was marked by periods of cemetery levelling and expansion. In *c.* 1800 (*Anon* 1809) and 1816–1818 (Salmon 1856:17) the southern church yard and mill dam were backfilled with ballast by the town's unemployed (Raynor et al. 2011). Excavated burials from this period are few, but generally follow the previous period's patterns and styles.

The later burial horizon followed the levelling of 1818 and was marked by heavy cemetery use. Despite further enlargement of the cemetery westward in 1843 (Fordyce 1857:715), by the mid 19th century, the cemetery was overcrowded and a public health concern. It was officially closed on 1st July 1855 (Fordyce 1857:715); however, burial in family plots continued until the 1860s (Raynor et al. 2011) (see Figure 4.4).

[22] A glebe is church land invested to a vicar or their incumbent (Merriam-Webster 2021).

Figure 4.4 The cemetery and church of St. Hilda's in 1863 (used with permission from South Tyneside Council 2020 ©). Archival caption reads as follows: "Looking from Coronation Street over St Hilda's Church and the graveyard towards the Market Place. The Old Town Hall with market stalls is visible on the left. In the background there is a windmill up near the ballast hills. Cottages in foreground adjoin the Charity School."

Burial practices in this period were diverse, reflective of individual taste and affordability, but relatively orderly (Raynor et al. 2011). Intercutting of graves was common, with disturbed individuals often gathered as charnel remains and reburied, some in mixed assemblages. While coffin burial in earth-cut graves was the norm, more expensive brick shaft graves were also present. Most plots included multiple individuals buried in quick succession one on top of the other. Individuals were usually supine in a north-east or south-west alignment. Some individuals were buried with coins over their eyes and, at least one (SK353), interred in a shroud. Significantly, three individuals were identified by preserved text on their coffin plates: Jane Prince (SK381), Ann Purvis (SK472), and Isabelle (unreadable surname) (SK941).

Parts of the southern section of the cemetery were excavated in 2006–2007 by Oxford Archaeology North, for Archaeological Research and Consultancy at the University of

Sheffield, on the behalf of Henry Boot Developments as part of watching briefs and mitigation in advance of constructing a supermarket wall and maintenance to sewer pumping stations (Raynor et al. 2011). The population is one of the largest Industrial cemetery populations excavated in the north-east of England. The excavated remains included both burial horizons and the transitional horizon. The collection is curated by the Department of Archaeology at the University of Sheffield. The remainder of this section summarises the demographic profile of the Coronation Street population. The following subsections discuss the demography, class, housing, occupation, and nutrition of the Coronation Street population in more detail.

Demography

There are 221 Coronation Street individuals housed in the Department of Archaeology at the University of Sheffield. Raynor *et al.* (2011) divided the population into twelve age groups and five biological sex groups, including an unknown group for both age and biological sex. Most age groups were similar in range to those used in Black Gate (see Table 4.6). Adults (16+ years) and non-adults (<16 years) comprised 53.5% (or 123/230) and 40.0% (92/230) of the population, respectively. As with Black Gate, only the adult population was included in this project. The biological sex was confidently and tentatively determined in 30.4% (70/230) and 14.8% (34/230) of the adult population, respectively (see Table 4.7). For this study, the confident and tentative groups pooled (pooled males 21.3% or 49/230 and pooled females 23.5% or 54/230).

The age at death distribution was similar to other post-medieval sites and indicative of an attritional assemblage (Newman 2016; Raynor et al. 2011): with a slight peak in young adults followed by a strong peak in older adults (Raynor et al. 2011). This later peak occurred earlier for males (36–45 years) than it did for females (45+ years). Females outnumber males 3:1 in young adult deaths. Where this trend has also been observed in similar post-medieval sites it has been interpreted as reflective of maternal disadvantage or maternal death in childbirth (Newman 2016). In non-adults, deaths decrease after birth, with the most deaths in pre-term and perinate groups. Pre-term and perinate births may reflect a high risk of death among the poorer classes, as poor mothers were unable to provide adequate nutrition and were more physiologically stressed than higher class mothers (Newman 2016).

Table 4.6 Ages of all adult individuals in the Coronation Street population (Raynor et al. 2011). The generic 'Adult' group included individuals whose age could not be determined specifically but were known to be 18 years or older.

Group Name	Age Range	Number of Adults
Young Adult	18-25 years	10
Prime Adult	26-35 years	22
Mature Adult	36–45 years	39
Older Adult	45+ years	22
Adult	18+ years	30
Total	All	123

Table 4.7 Biological sexes of the adult individuals in Coronation Street population, including the total number of individuals included in each group (Raynor et al. 2011). Pooled sexes were Pooled Female and Pooled Male.

Biological Sexes	Total in Population	Pooled Groups
Male	32	49
Probable Male	17	
Female	37	54
Probable Female	17	
Unknown	20	20
Total Adults	123	123

Class

Information regarding burial practices and patterns is only provided for Black Gate, as the equivalent was not available for the Coronation Street population. In the latter, 177 burials included physical evidence of wooden coffins; but these were poorly preserved (Raynor et al. 2011). This led Raynor *et al.* (2011) to conclude that coffins were likely used more widely at the site, despite being archaeologically invisible. As such, the surviving evidence is not representative of individual burial style or social status, so individual comparisons cannot be made. Notwithstanding, class was a key factor in individual lived experience. Thus, the following sub-sections highlight the effect of class on housing, occupation, and nutrition. Class is also a key theme in the discussion chapter.

Housing

The population of Britain nearly doubled between 1700 and 1800, with towns and cities growing the fastest (Western and Bekvalac 2020:73–105; White 2009). Urbanization was fuelled by the immigration of people looking for work and moving from rural settings to urban centers (Encyclopaedia Britannica 2019). In 1850, the Tyne Improvement Commission ordered the dredging of the Tyne River and the construction of a series of piers, which caused a boom in shipping (Encyclopaedia Britannica 2000) and a sharp increase in production and manufacturing in South Shields (Mood 2007:21; Rayno et al. 2011:17). This economic explosion ultimately transformed South Shields from a port of Newcastle-upon-Tyne to a county borough of *c.* 75,000 in 1850 (Kelly's Directory of Durham 1890).

Construction boomed in the Industrial Period, and poor-quality mass housing that could be erected cheaply, with brick building methods, was the norm (Rudge 2012). Traditional stone and thatch building methods created warmer homes; but brick construction was faster and more economical, and, so, became the construction method of choice. The houses that were built were poorly constructed (or "jerrybuilt"), without porches, double glazing, or tightly fitted windows and doors (Muthesius 1982). Perhaps ironically, and to the advantage of the subpar builders, the fires that heated the homes required good ventilation to draw fresh air. This was achieved intentionally *via* chimneys and unintentionally through poor construction. As a result, houses were draughty, leaky, and energy inefficient (Boardman 1991; Rudge 2012). The contemporary backlash against unsanitary living conditions lead to a push for more fresh air, space, and light (Rudge 2012). As a result, the British were slow to abandon room construction without fireplaces, despite the draught, as chimneys allowed in what was seen as beneficial fresh air and combatted indoor humidity and condensation (Burberry 1978; Gaskell 1987; Muthesius 1982).

Compared to the middle and upper classes, who favoured semi-detached homes and tended to live one household to a home, the Coronation Street poor often had to live communally in higher-density, drafty housing, with inadequate sanitation, and nearer to industry (Rudge 2012; *Shields Daily Gazette* 1885; Sutcliffe 2006; Western and Bekvalec 2020:91; Wohl 2002). While fostering insufficient urban planning, rapid urbanization also decreased domestic and public sanitation (Encyclopaedia Britannica 2019). For example, insufficient urban planning caused a series of cholera outbreaks in South Shields in the mid-19th century (Historic England 2013). As a result, parliament was forced to pass an act and push for the construction of a water pumping station (built 1859–1861).

Finally, coal (or sea coal) had been growing in popularity as a domestic heating source, especially amongst the poor, since the 17th century (Brimblecombe 2011:22–36); and it skyrocketed in popularity in the 18th century (Burberry 1978), overtaking wood, peat, and

turf (Rudge 2012). Figure 4.5 illustrates the popularity of coal as a heating source and population density across England in 1851 (Beach and Hanlon 2017:2661). The area around South Shields (indicated by the arrow) is in the fourth quartile in both instances.

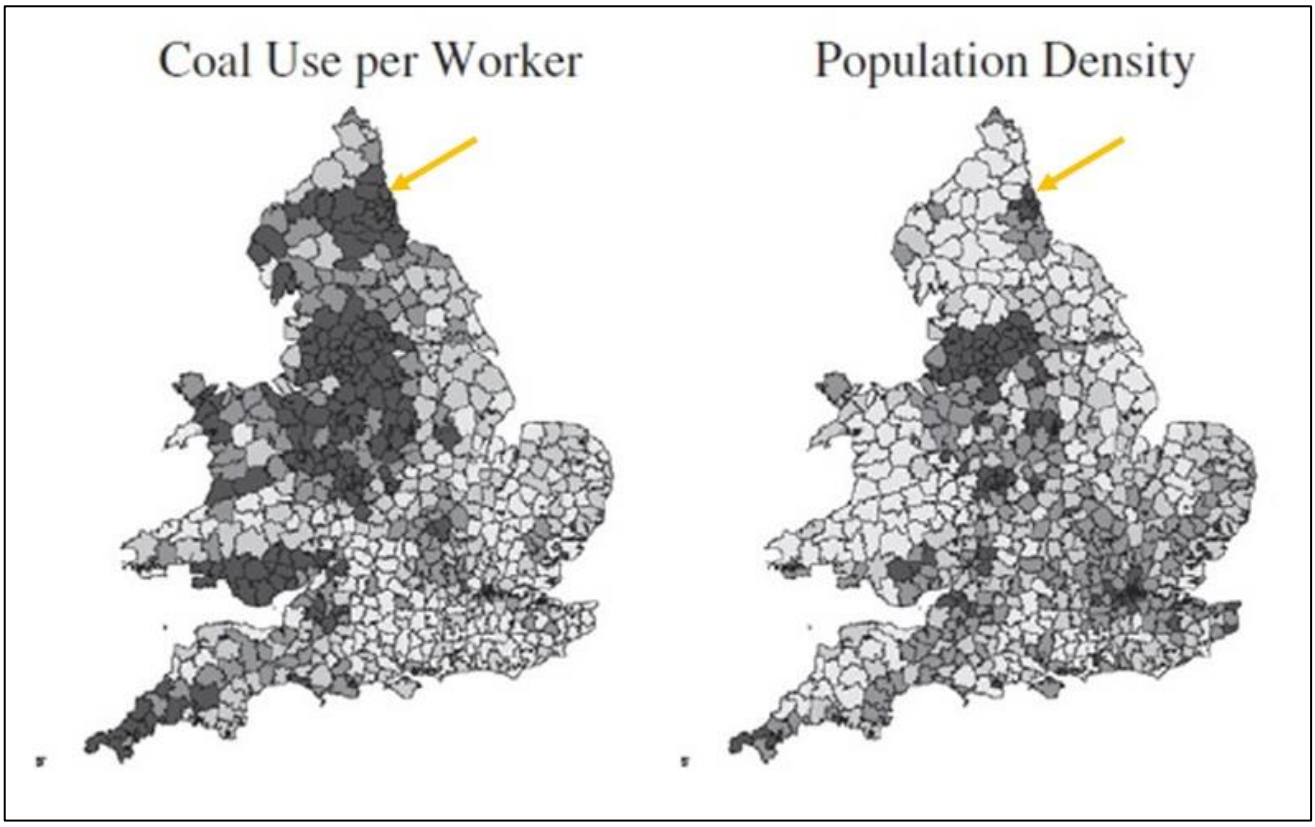


Figure 4.5 A heat map of England in 1850–1860 by 1851, illustrating the average(?) coal use per worker per annum(?) and the average(?) population density. Shading represents quartiles, with darker colours representing higher quartiles. The arrow indicates South Shields. Edited from Beach and Hanlon (2017:2661).

Occupation

The people of South Shields were heavily involved in shipping, manufacturing, and production. The most common occupations being in the railway; salt pans; lime kilns; gas-works; ship building docks; and iron, steel, glass, and tile factories (Kelly's Directory of Durham 1890; Simpson 2017). South Shields also formed part of The Great Northern Coal Field. There were four collieries in South Shields in this period, and the nearest to St. Hilda's Church, St. Hilda's Colliery, was less than a mile away (Simpson 2017). Previous research has uncovered various forms of occupation-induced physiological stress amongst Coronation Street males. An analysis of non-adult growth revealed that Coronation Street non-adults were significantly smaller than both modern and comparative archaeological data; and that vertebral stunting first occurred between five and nine years (Newman 2016). It was concluded that

this was likely indicative of the increased physical demands caused by employment, which often began around nine years of age. Traumatic injury and spinal joint disease were also common amongst adults, the latter being especially common amongst males and peaking in mature adults, consistent with other industrializing populations (Raynor et al. 2011).

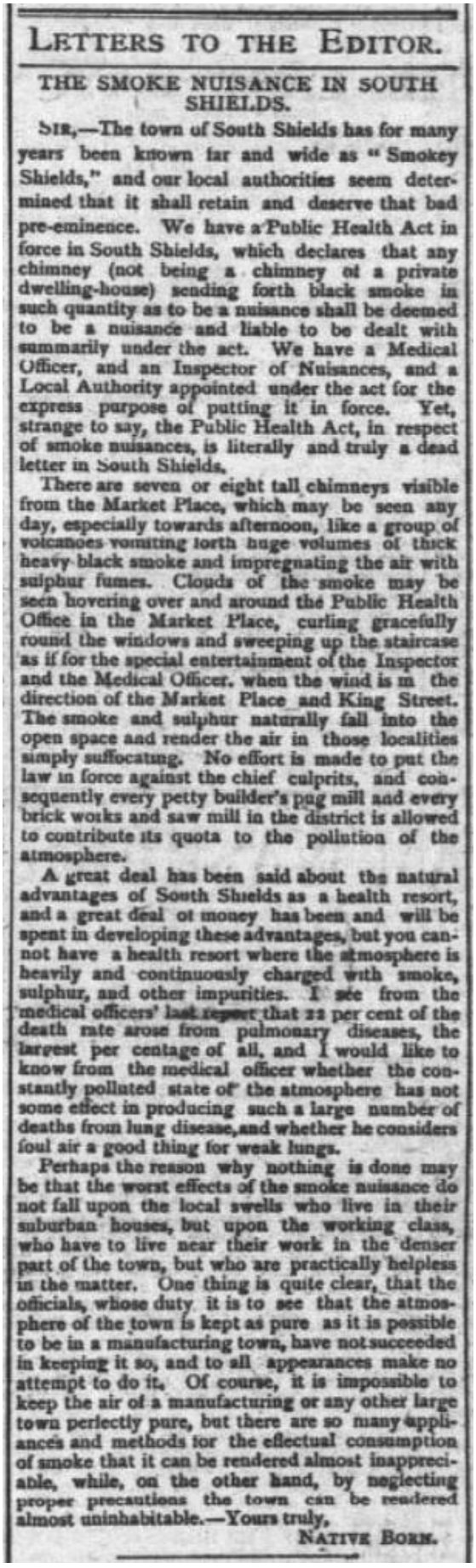
The trend of the 18th and 19th centuries was that males were the primary breadwinners (Boyd 2020:382); and between 1790 and 1914, the North-East of England had the lowest female employment rate (*c.* 18–20% employed), the lowest marriage age, and the highest marriage rates in the country (Mood 2007). The mechanization of traditionally female-dominated industries, such a weaving and spinning (Bythell 1993; Mood 2007), resulted in little secure work for women. And when women did work, they were underpaid compared to men. As a result, some women chose—or had no choice but—to engage in piece work that could be brought home (Western and Bekvalac 2020:81). This is not to say that women in South Shields did not work. In the 1881 census, 14.13% of South Shields women were recorded as employed and this rose to 21.50% in 1891 (Mood 2007:53,55). The most common female-held professions reported in the latter period were (in descending popularity): domestic service, clothing, textiles, the professions (e.g., school proprietresses or nurse) (Bennett et al. 2020; Hannam 1984), paper and printing, brick/cement/pottery/glass, agriculture, and food/drink/lodging. Censuses likely under-estimated female labour, as husbands (those who completed the census) under-valued, or were ashamed of, their wife's work; the work was casual or seasonal; or it was associated with their husband's work and was not viewed as separate (Higgs and Wilkinson 2016; Mood 2007:47–9). Parliamentary Papers from 1842 record that women and children were known to sometimes work in collieries across the country (Halsall 1998). Finally, wills from this period record female involvement in the economy: for example, Jane Blakiston from Westoe, South Shields died 1778 and had "clayworks and 'the liberty to make tiles', Westoe; tenements at Westoe" (Beaumont 2019:229); Mrs. Ann Brodrick from South Shields died in 1784 and "carried husband's (Lockwood) business on, building four ships" (Beaumont 2019:229); and Mrs. Elizabeth Cookson of South Shields died 1788 and "leased three salt-pans; rentier of properties" (Beaumont 2019:231). Despite the patriarchal, elite lens of history, these historical accounts of women's work suggest that some women were actively employed or employers. Thus, while many women lived and/or worked in their/someone's home, some women worked outside of the home.

The industrial poor were employed from a young age (Goose and Honeyman 2013; Humphries 2010), undertook the most arduous and dangerous work (Otter 2006; Ruslan et al. 2020), and often worked until they died (Western and Bekvalac 2020:171). The physiological strain such work placed on Coronation Street individuals is reported to have resulted in

vertebral stunting in children entering the workforce (Newman 2016; Sharpe 2012) and physical stress throughout their lives. For example, there was a high rate (30.8% or 36/117) of traumatic physical injury in the Coronation Street population, characteristic of an industrializing population, with injuries occurring 20.5% (24/117) more often in males than females, consistent with, among other things, work-place accidents (Raynor et al. 2011). The emphasis in the period, regardless of injury or disease, was always on a speedy recovery and a quick return to work, or, as one contemporary source put it, a return to "usefulness" (Kentish 1817). Indeed, after being crushed by a horse, one British labourer was deemed "a little distorted, but not so as to impede him from working" (Appendix to the First Report of the Commissioners 1842).

To further illustrate the level of occupational hardship habitual to some poor Coronation Street individuals, a young adult Coronation Street male presented with lesions tentatively suggestive of phossy Jaw (or malformation caused by overexposure to phosphorous) (Raynor et al. 2011). This individual may have worked in a factory where the chemical was used (e.g., matchstick manufacturing). Roberts *et al.* (2016) tentatively diagnosed a contemporaneous non-adult from North Shields with phossy jaw. The authors highlight that matchstick manufacturing attracted destitute individuals (Emsley 2000) to work up to sixteen hour days in "deplorable" conditions (Simon 1863). Additionally, an 1863 Public Health Report found that 11% of workers developed phossy jaw after an average of five years of exposure to the toxin (Simon 1863). It is likely some individuals from South Shields worked in similarly toxic conditions; they may have even worked in the same factory as this individual from North Shields.

Finally, coal was used to heat the home and also to power industry: from steam engines to gas lighting (Western and Bekvalac 2020:78). South Shields was also home to multiple heavily air polluting industries, as listed above. Public figures had been concerned with worsening air quality since coal first started being used in the 13th century (Western and Bekvalac 2020:76–7). By the Industrial era, the ubiquity of black coal dust in British towns and cities influenced a change in fashion and the adoption of off-white and dark clothing (Beach and Hanlon 2017:2656; Brinblecombe 2011:64). One 1885 letter to the editor of the *Shields Daily Gazette* complains of the air pollution in South Shields (or "Smoky Shields"; see Figure 4.6). They explain that the authorities do nothing to enforce the Public Health Act and protect the residence of South Shields from the ill health effects of smoke, caused by the town's greatest polluters. They highlight a medical officer's report that indicated that 22% of deaths were from LRI; and blame the officials for doing nothing because those suffering the most were the poor, who live closer to work and, therefore, closer to the smoke. Indeed, even today, air pollution remains a grave public health concern (DEFRA 2021).



LETTERS TO THE EDITOR.

THE SMOKE NUISANCE IN SOUTH SHIELDS.

SIR,—The town of South Shields has for many years been known far and wide as "Smokey Shields," and our local authorities seem determined that it shall retain and deserve that bad pre-eminence. We have a Public Health Act in force in South Shields, which declares that any chimney (not being a chimney of a private dwelling-house) sending forth black smoke in such quantity as to be a nuisance shall be deemed to be a nuisance and liable to be dealt with summarily under the act. We have a Medical Officer, and an Inspector of Nuisances, and a Local Authority appointed under the act for the express purpose of putting it in force. Yet, strange to say, the Public Health Act, in respect of smoke nuisances, is literally and truly a dead letter in South Shields.

There are seven or eight tall chimneys visible from the Market Place, which may be seen any day, especially towards afternoon, like a group of volcanoes vomiting forth huge volumes of thick heavy black smoke and impregnating the air with sulphur fumes. Clouds of the smoke may be seen hovering over and around the Public Health Office in the Market Place, curling gracefully round the windows and sweeping up the staircase as if for the special entertainment of the Inspector and the Medical Officer, when the wind is in the direction of the Market Place and King Street. The smoke and sulphur naturally fall into the open space and render the air in those localities simply suffocating. No effort is made to put the law in force against the chief culprits, and consequently every petty builder's pug mill and every brick works and saw mill in the district is allowed to contribute its quota to the pollution of the atmosphere.

A great deal has been said about the natural advantages of South Shields as a health resort, and a great deal of money has been and will be spent in developing these advantages, but you cannot have a health resort where the atmosphere is heavily and continuously charged with smoke, sulphur, and other impurities. I see from the medical officers' last report that 22 per cent of the death rate arose from pulmonary diseases, the largest per centage of all, and I would like to know from the medical officer whether the constantly polluted state of the atmosphere has not some effect in producing such a large number of deaths from lung disease, and whether he considers foul air a good thing for weak lungs.

Perhaps the reason why nothing is done may be that the worst effects of the smoke nuisance do not fall upon the local swells who live in their suburban houses, but upon the working class, who have to live near their work in the denser part of the town, but who are practically helpless in the matter. One thing is quite clear, that the officials, whose duty it is to see that the atmosphere of the town is kept as pure as it is possible to be in a manufacturing town, have not succeeded in keeping it so, and to all appearances make no attempt to do it. Of course, it is impossible to keep the air of a manufacturing or any other large town perfectly pure, but there are so many appliances and methods for the effectual consumption of smoke that it can be rendered almost inappreciable, while, on the other hand, by neglecting proper precautions the town can be rendered almost uninhabitable.—Yours truly,

NATIVE BORN.

Figure 4.6 Letter to the editor of the Shields Daily Gazette (Monday 20 July 1885) complaining about smoke pollution.

Nutrition

Bread composed the bulk of an Industrial era diet, followed by meat (usually bacon), and other sundries, such as potatoes, cheese, butter, milk, tea and/or coffee, and sugar (Griffin 2018). A wide range of items were available for purchase, including many imported items; and, before 1850, many industrial families cooked/baked their food at home rather than rely on expensive pre-prepared foods (Griffin 2018; Intoxicating Spaces 2021). However, vegetables (a natural source of vitamins, such as Vitamin C) were lacking in many lower and lower-middle class diets, due to the price and scarcity of vegetables in industrial towns and cities (Horrell and Oxley 2012). One 1844 source recorded that the family of a spinner in Manchester "never taste any other vegetable than potatoes" (Pike 1966:52-53). As a result, malnourishment (which can result in LEH, Harris lines, and metabolic diseases) (Roberts and Manchester 2005:221–51) and vitamin and mineral deficiencies (such as anemia, which may cause CO) were common amongst the poor, and while they rarely resulted in starvation, they impacted individual morbidity (Clayton and Rowbotham 2008).

Newman (2016:214–47) observed high levels of nutritional stress amongst Coronation Street individuals—in particular, high rates of CO (45%), LEH (60%), Harris lines (40%), and metabolic disease (30%) (Table 4.8) (Newman 2016:174–247). The poorer classes of South Shields would have had the least food security (Roberts and Cox 2003). Raynor *et al.* (2011:53) also noted more LEH, more dental caries, and less dental calculus amongst Coronation Street females than males; this suggested females may have consumed more carbohydrates than males, while males may have consumed more protein and/or had less adequate dental hygiene than females (Raynor et al. 2011:49, 51).

Table 4.8 A summary of the pathological lesions discussed in Raynor et al. (2011) by crude prevalence where possible. Those marked with a * are by true prevalence, a.k.a. by bone. Total observable numbers not reported. ANTL is ante-mortem tooth loss; DJD is degenerative joint disease; JD is joint disease; CO is cribra orbitalia; PH is porotic hyperostosis; LEH is linear enamel hypoplasia; SPNBF is sub-periosteal new bone formation; TB is tuberculosis.

Lesion	Non-Adults	Adults
Traumatic injury	30.8% (36/117)	1
Trauma to the ribs	0.8% (12/1495)*	1
Dental calculus	86.0% (86/100)	5.3% (15/284)*
Dental caries	49.0% (49/100)	7.0% (20/284)*
Dental abscess	25.3% (24/95)	0
Periodontitis	20.5% (24/117)	0
AMTL	76.9% (90/117)	0
DJD	Extensive; not reported	NA
Appendicular JD	NA	NA
Spinal JD	81.2% (95/117)	NA
Osteoarthritis	29.0% (34/117)	NA
Metabolic disease	11.96% (14/117)	0
Rickets	3.42% (4/117)	0
Congenital disease	25.6% (30/117)	3
Neoplasia	3.4% (4/117)	NA
CO	7.29% (7/96)	24.0% (6/25)
PH	NA	NA
LEH	33.3% (30/90)	3.87% (11/284)*
SPNBF	NA	NA
Non-specific bone inflammation	23.1% (27/117)	NA
Chronic maxillary sinusitis	12.0% (14/80)	NA
TB or respiratory infection	1.7% (2/117)	1
Specific infections	7.69% (9/117)	NA

There is some contemporary documentary and osteological evidence for the preferential feeding of males, the primary breadwinners, to the detriment of their wives and children—especially the youngest (Gowland et al. 2018; Griffin 2018; Horrell and Oxley 2012; Reedy 2020). The stunted growth of most Industrial era children, followed often by exclusive male catch-up growth, suggested that British Industrial societies engaged in the preferential

feeding, care, and support of non-adult males over that of non-adult females (Gowland et al. 2018; Griffin 2018; Horrell and Oxley 2012; Reedy 2020).

Weaning also occurred early in Industrial populations, often between six months and one year; though, sometimes occurring before six months and, occasionally, infants were not breastfed at all (Henderson et al. 2014; Newman and Gowland 2016; Newman 2016:195–6). Early/not weaning was practiced by women of all classes (Newman and Gowland 2016), though for different reasons (Stevens et al. 2009). Amongst the upper classes, society events usually did not include children; in the middle classes, women often had to return to managing their husband's business; and, in the lower class, women were given little time off work to give birth and recover (Newman 2016; Perkin 1993; Stevens et al. 2009). As a result, regardless of class, children had to be weaned early and left at home or in care. Some individuals employed wetnurses, rather than wean their children early. However, as it was for mothers, the quality of the milk was dependent on the nutritional health of the wetnurse (Fildes 1995); and dietary stress was observed in both mothers and children in the Coronation Street population (Newman 2016:174–247; Raynor et al. 2011:79).

Smoking tobacco was a habit introduced to England as part of the psychoactive revolution, when colonial trade resulted in the import of substances such as tobacco, coffee, and opium to Britain (Intoxicating Spaces 2021). It became fashionable for men in the 16th century but did not become fashionable for women until the late 19th century (Hilton 1995; Western and Bekvalac 2020:92, 94–5). By the 19th century, cigarette smoking had become a perceived problem by the anti-tobacco movement, especially amongst "boy labourers" (Hilton 1995). A study by Western and Bekvalac (2020:94) found that tobacco smoking in Industrial London populations differed by class, with the poor smoking slightly more than other classes. Previous studies noted five Coronation Street individuals with dental wear consistent with habitual pipe smoking: the youngest was 15 years and all those who could be biologically sexed were male (Raynor et al. 2011:55).

Amongst the upper and upper-middle classes, smoking often took place at social clubs and became a key part of the bourgeois-liberal masculine ideal (Hilton 2000). Smoking rooms within the upper and upper-middle class home were also popular in this period (Logan 2001). These were gendered spaces, which excluded women (Martine 1866). The lower classes smoked almost everywhere: at home, in communal places, such as public houses and inns, and while working (Hilton 2000; Western and Bekvalac 2020:92–3). While smoking was becoming less taboo amongst women by the latter half of the 19th century, contemporary sources suggest that smoking continued to be viewed as an improper habit for middle and upper class women and a vice of the poor. One source describes a poor

London home where there was "a sprinkling of ugly, shabbily dressed women, sprawling their elbows on porter-slopped tables…smoking rank tobacco" (Rowe 1881).

4.5 Conclusions

The two skeletal populations included in this project were both from the north-east of England. Due to their temporal separation, characterized by a change from a farming to an industrial lifestyle, they provided the project with excellent means of studying the health effects of industrialization and urbanization, while largely controlling for regionality. Because both populations have been studied by other archaeologists, the new method of visualizing and diagnosing mastoiditis was able to be grounded in the context these studies provided. This facilitated the wider conversations later in this thesis regarding mortality, morbidity, risk, and adaptation.

Chapter 5
Methodology

5.1 Introduction

This chapter details the methodologies employed throughout this research in four sections. The first section discusses the novel method of imaging and diagnosing mastoiditis. It describes the preliminary analysis, conducted to test and develop the method used in the wider study. Next, the finalized method is described, including the inclusion criteria, the imaging method, and the diagnostic and recording method. The second and third sections describe the methods used to analyze the maxillary sinuses and visceral rib surfaces for lesions indicative of maxillary sinusitis (MS) and lower respiratory infection (LRI), respectively. To assess MS and LRI, robust methodologies developed by other researchers are followed. The inclusion, diagnosis, and recording criteria are described. The fourth section discusses how this project gathered, organized, and analysed data to answer the research questions, meet the objectives, and, ultimately, achieve the aim of the project.

5.2 Mastoiditis

This section describes the method developed by this project for the imaging and diagnosis of mastoiditis in human skeletal remains. A preliminary analysis of 40 individuals was conducted in advance of the larger study to develop and refine the method. The findings of this analysis are discussed first. Next, the criteria were used to assess the inclusion of each mastoid process in the study; the procedures followed for taking diagnostic radiographs; and the

method used to diagnose individuals with no evidence of mastoiditis, and those with residual childhood mastoiditis (residual CM) and/or adult mastoiditis (AM) are presented.

When imaging the mastoid processes, both during the preliminary analysis and the wider study, health and safety protocols were followed for working with radiation, in-line with the Department of Archaeology and the University of Sheffield health and safety guidelines. All users were trained how to use the equipment and how to safely work with radiation. Access to the laboratory was restricted when the X-ray generator was in use. Personal radiation monitors, one worn on each hand and one clipped to the shirt, were consistently checked to monitor exposure. A Giger counter was available to periodically test the residual radiation in the laboratory and that being emitted by the X-ray generator. The equipment was also routinely checked by laboratory technicians to make sure it was working properly. The Aribex NOMAD Pro hand-held X-ray generator was equipped with a radiation shield, so a lead apron did not have to be worn while operating the system. No health and safety concerns were encountered during the course of this project.

Preliminary Analysis

A preliminary analysis was conducted in the autumn and winter of 2019–2020, the goal of which was to improve upon the method of visualising and diagnosing mastoiditis developed as a part of my master's project (Purchase 2016; Purchase et al. 2019) and to develop a new method to be used in the present study. In the method developed as part of my Master's project, the complete and undamaged mastoid processes of adult individuals (18+ years) were imaged medio-laterally (n=56) (see Figure 5.1). To achieve this, the Aribex NOMAD sensor was temporarily fixed to the lateral surface of the mastoid process with modelling clay and, if the temporal bone was disarticulated from the rest of the cranium, the bone was placed on a table on its lateral side so an X-ray could be taken from the medial side, through the mastoid process, to the sensor affixed to the lateral surface of the process. If the temporal bone was articulated, the cranium was inverted on its axis and the X-ray was taken in the same way.

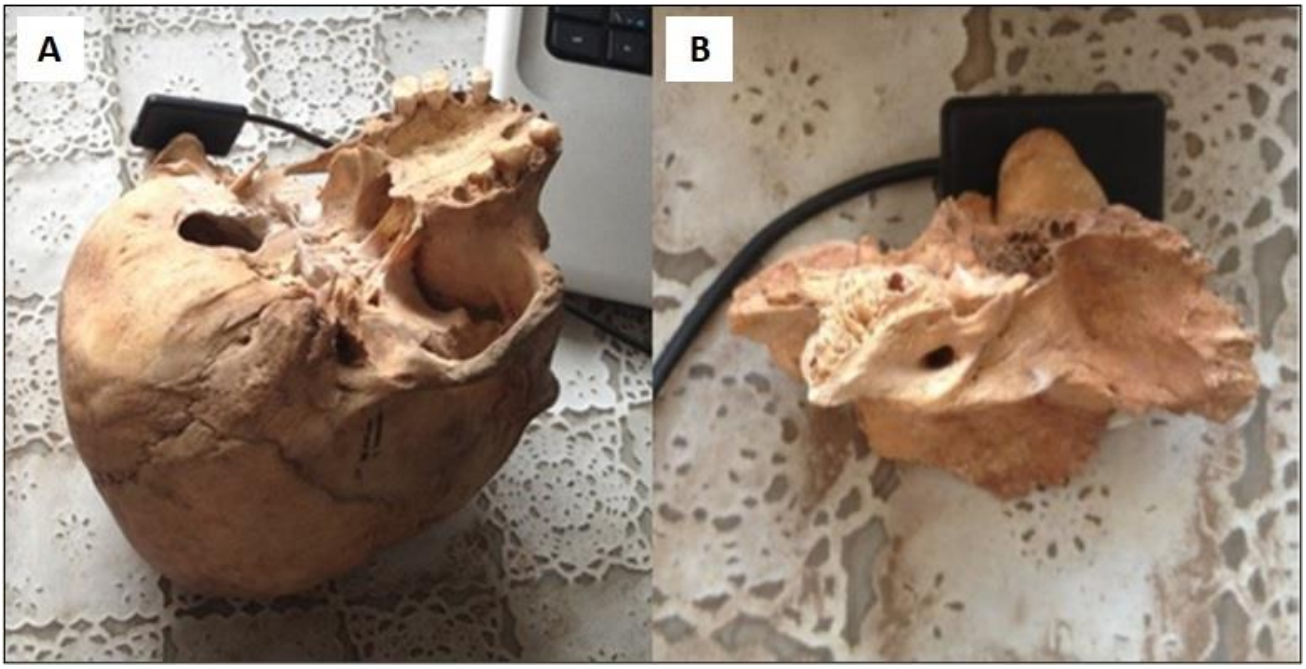


Figure 5.1 Image adapted from Purchase et al. (2019) illustrating how individuals were prepared for X-ray **A)** if the temporal bone was articulated with the rest of the cranium and **B)** if the temporal bone was disarticulated from the rest of the cranium.

This method was used to diagnosed four types of bone from the radiographs: hypercellular, fully hypocellular, partially hypocellular, and lytic and sclerotic bone (see Figure 5.2) (Purchase et al. 2019).

- Hypercellular bone appeared as large, well defined air cells, indicative of an individual who had no mastoiditis during their lifetime.
- Hypocellular bone appeared as densely packed, small air cells.
- Both fully and partially hypocellular bone were predictive of an individual who had experienced childhood mastoiditis, for whom the pneumatization process had been fully or partially stunted.
- Lytic and sclerotic bone appeared as (a) large air cell(s), fully or partially surrounded by sclerotic margin(s). The sclerotic bone was dense and was not punctuated by air cells. Lytic and sclerotic bone was indicative of an individual who had mastoiditis as an adult.

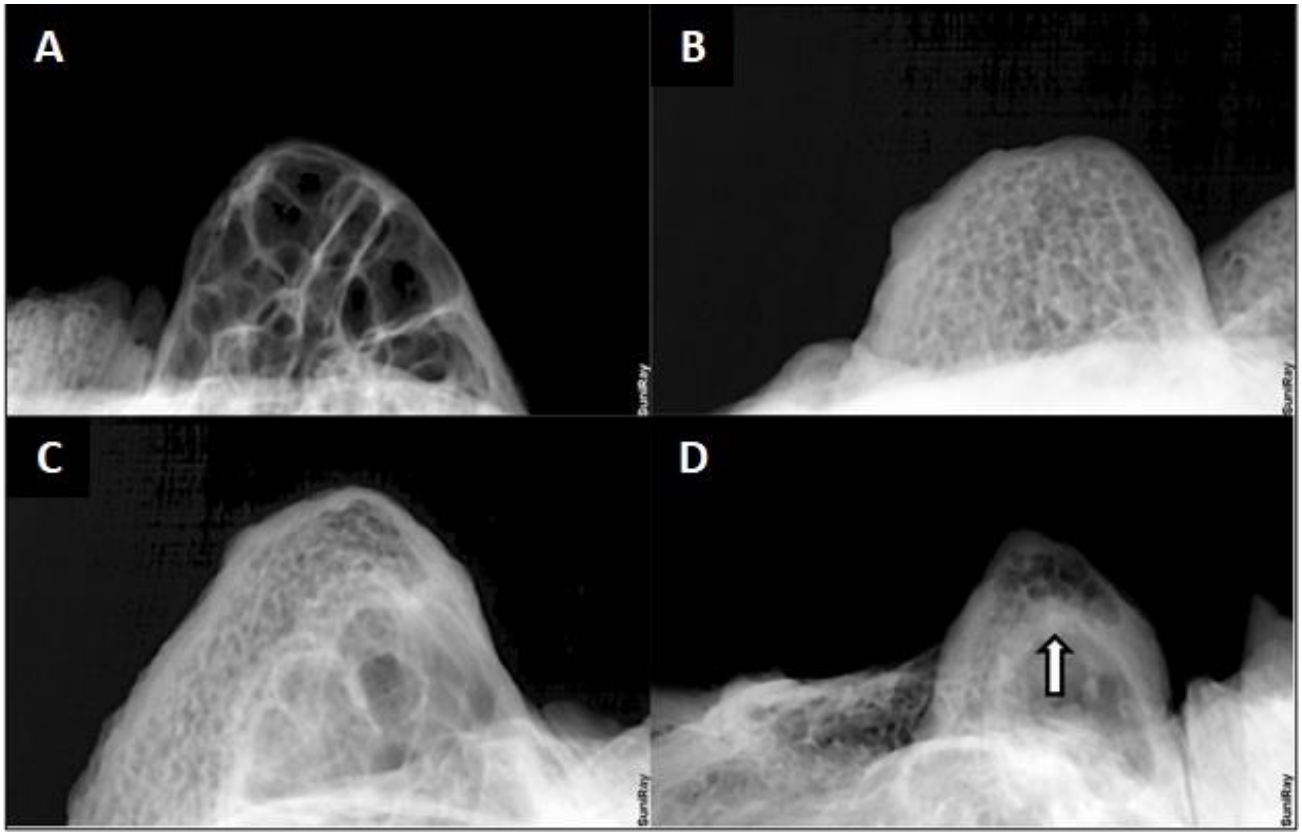


Figure 5.2 Image from Purchase et al. (2019) illustrating **A)** a hypercellular, or normal, mastoid process, **B)** a fully hypocellular mastoid process, **C)** a partially hypocellular mastoid process, and **D)** a lytic and sclerotic mastoid process. The arrow indicates a band of sclerotic bone surrounding a lytic void in the mastoid process.

To identify ways in which the method could be improved, a sample of 40 individuals (of both biological sexes) from the Black Gate population were studied (see Tables 5.1 and 5.2). Young and older children and adolescents were included in the analysis to determine if mastoiditis could be reliably diagnosed in non-adults. Both biological males and females were included so sexual dimorphism was considered when establishing the imaging and diagnostic methods: for example, biological female mastoid processes are generally larger than those of biological males until puberty, after which the opposite is generally true (Cinamon 2009). Both left and right temporal bones, if present and whole, were imaged in three planes rather than just one: the lateral-medial, anterior-posterior, and inferior-superior planes. The lateral-medial plane was similar to the medial-lateral plane described in Purchase *et al.* (2019), but the image was taken from the lateral to medial side rather than the medial to lateral side. This was done to see if it made a difference to the quality of the radiograph, as the mastoid process was closer to the X-ray generator, but further away from the sensor. The latter two planes were used to test if they were more diagnostic than the medial-lateral plane or to see if they provided additional diagnostic value to the medial-lateral plane. The set up for imaging was also improved upon from the first study reported in

Purchase *et al.* (2019). Now, the temporal bone or cranium were supported by a Styrofoam ring and/or foam. The sensor was then placed on the foam, behind the mastoid process and opposite the X-ray generator. When the temporal bone was articulated to the rest of the cranium, it was found to sometimes be necessary to slot the sensor into a custom carved block of foam, which was easier to balance than the sensor alone (see Figure 5.3). These improvements protected the bones from the hard table-top; eliminated the need to temporarily fix the sensor to the temporal bone; and standardised the distance between the sensor and the X-ray generator.

Table 5.1 The biological sexes of the Black Gate individuals included in the preliminary analysis. Pooled groups were obtained by combining probable and certain males or females.

Biological Sex	Total in Preliminary Analysis
Pooled Males	15
Pooled Females	12
Unknown	13
Total	40

Table 5.2 The age groups of the Black Gate individuals included in the preliminary analysis. The generic 'Adult' group included individuals whose age could not be determined specifically but were known to be 18 years or older.

Age Group	Total in Preliminary Analysis
Young Child	4
Older Child	4
Adolescent	5
Young Adult	6
Prime Adult	8
Mature Adult	7
Senior Adult	6
Total	40

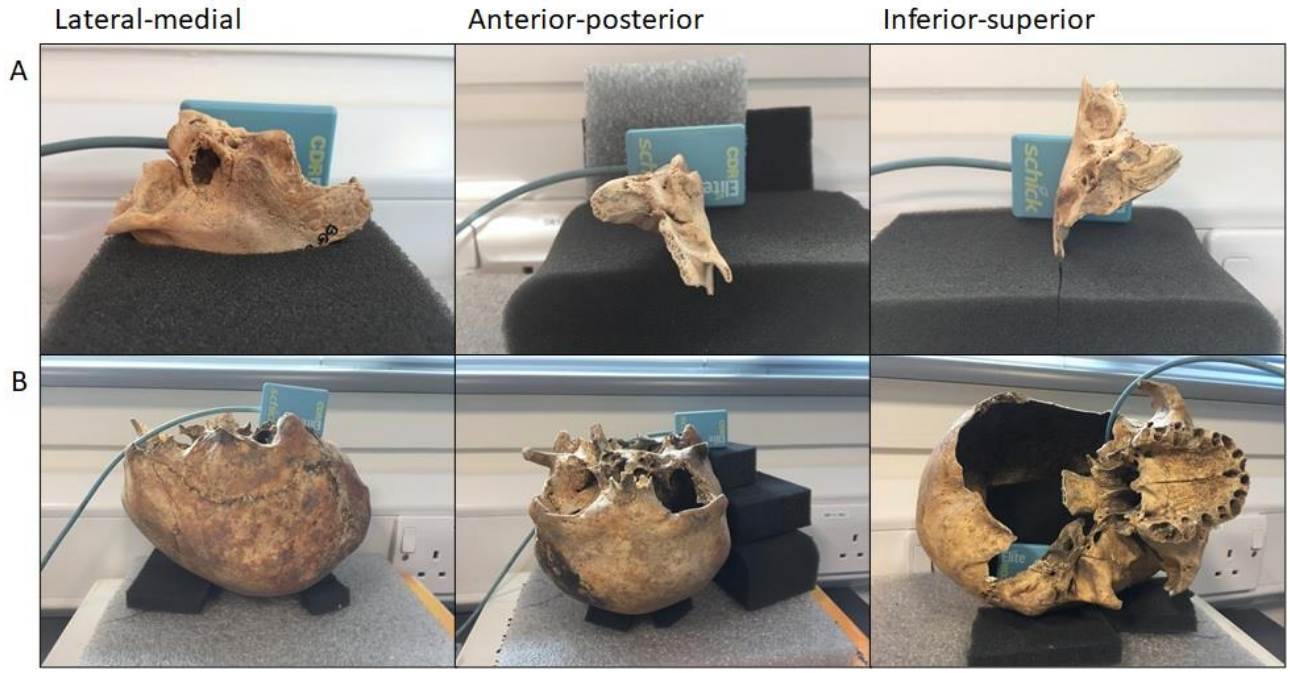


Figure 5.34 The three imaging planes tested during the preliminary analysis showing how the planes were achieved when imaging a **A)** disarticulated and **B)** articulated temporal bone.

The lateral-medial plane was achieved by inverting the disarticulated temporal bone/cranium on its axis (so the transverse plane was parallel with the tabletop), positioning the temporal bone so that the medial (internal) surface was away from the X-ray generator. The sensor was positioned so it was parallel to the lateral surface of the mastoid process. The anterior-posterior plane was also achieved by inverting the disarticulated temporal bone/cranium on its axis (so the transverse plane was parallel with the tabletop) and positioning the temporal bone so that the anterior surface of the mastoid process faced the X-ray generator. The sensor was positioned parallel to the posterior surface of the mastoid process. Lastly, the inferior-superior plane was achieved by placing the squamous portion of the disarticulated temporal bone in a cut in the foam, which gently held the bone, so the inferior portion of the mastoid process faced the X-ray generator and the sagittal plane was parallel with the tabletop, or by placing the articulated cranium on its side, with the sagittal plane parallel to the tabletop, so that the mastoid process being imaged was on the side closest to the tabletop. The sensor was then positioned inside the cranium (if post-mortem fractures allowed) so that it was anatomically superior to the mastoid process. If the cranium was not broken post-mortem in such a way that the sensor could be placed anatomically superior to the mastoid process, the X-ray was not taken.

After all the individuals were imaged, the bone morphology was assessed for evidence of hypercellularity, primary hypocellularity, and secondary hypocellularity in line with the morphology described in the publications by Flohr *et al.* (Flohr et al. 2009, 2017, 2019;

Flohr and Schultz 2009a,b) (see Figure 5.4 and Tables 5.3 and 5.4). Data were collected by mastoid process; to obtain individual diagnoses, results were pooled in the favour of infection. For example, if an individual had a hypercellular left mastoid process (indicative of a mastoid process that had never been infected) and a primary hypocellular right mastoid process (indicative of residual childhood mastoiditis), then the individual was diagnosed with residual childhood mastoiditis. This was done to facilitate individual-level discussions of health. Left and right data were retained to study the laterality of the infection.

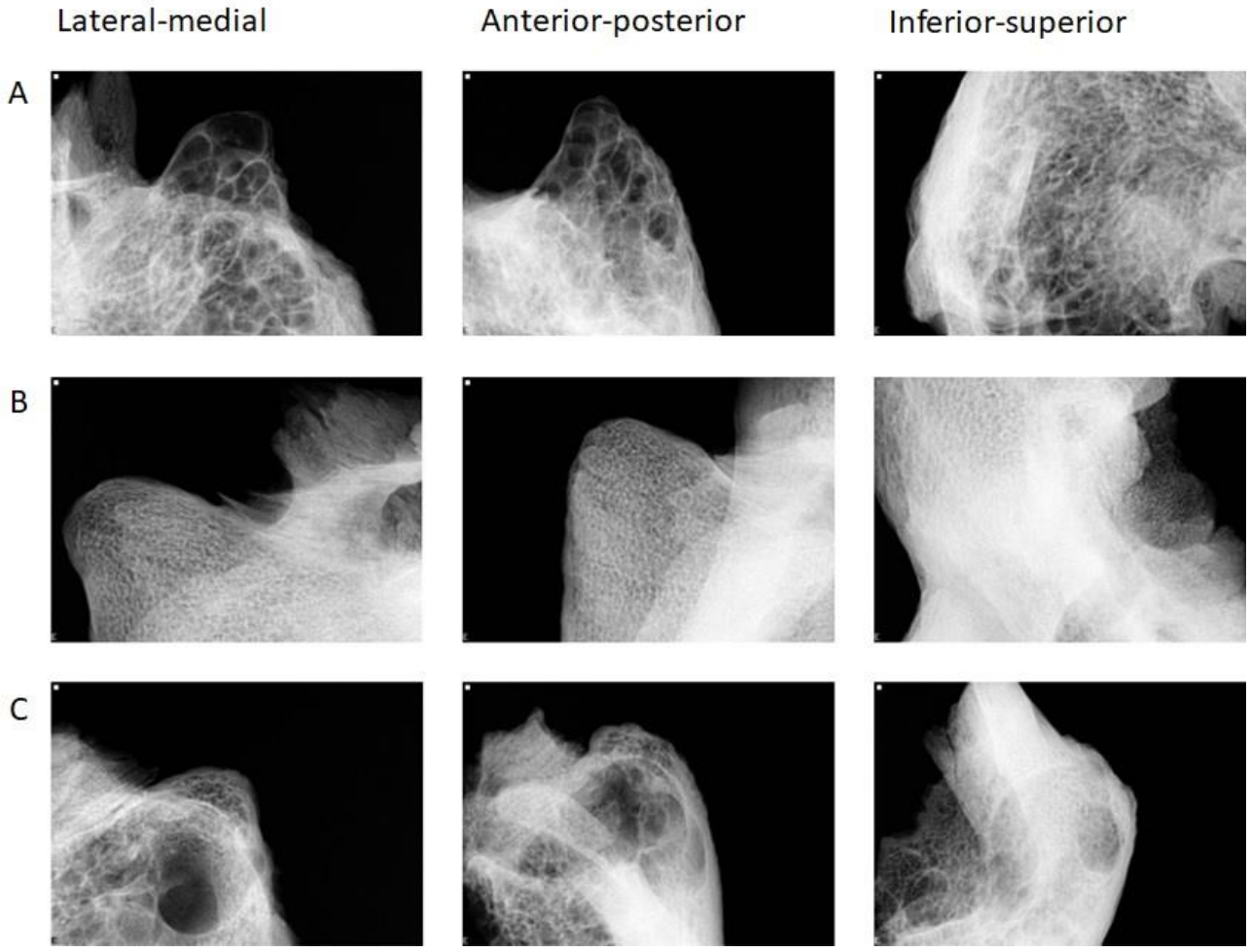


Figure 5.45 Mastoid processes from three different individuals visualised in three different planes. Each individual demonstrates a different type of bone. **A)** Hypercellularity diagnostic of an individual who has never been infected (BG547, right mastoid process). **B)** Primary hypocellularity diagnostic of residual childhood mastoiditis (BG56, left mastoid process). **C)** Secondary hypocellularity diagnostic of adult mastoiditis (BG51, right mastoid process).

Table 5.3 The true prevalence rate of left and right mastoid processes included in the preliminary analysis with each type of mastoid air cell. Hypercellular bone is normal.

Bone Type	Left Mastoid Process		Right Mastoid Process	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Hypercellular	11	45.8%	15	44.1%
Primarily Hypocellular	10	41.7%	15	44.1%
Secondarily Hypocellular	2	8.3%	4	11.8%
Total	24	100%	34	100%

Table 5.4 The crude prevalence rate of Black Gate individuals included in the preliminary analysis with childhood and/or adult mastoiditis diagnosis. Never infected refers to an individual with no evidence of mastoiditis (hypercellular mastoid air cells).

Diagnosis	Number Affected	Crude Prevalence
Never Infected	10	25.0%
Childhood Mastoiditis	13	48.1%
Adult Mastoiditis	6	15.0%
Total	40	100%

In general, the prevalence of residual childhood mastoiditis (residual CM) was high (48.1% or 13/27), while the prevalence of adult mastoiditis was relatively low (15.0% or 6/40). Since mastoiditis is, today, more common in children than adults (Bluestone1998; Groth et al. 2012; Wilson et al. 2017), this pattern suggested that Black Gate non-adults were at high risk of infection like children today. The method produced radiographs diagnostic of childhood and adult mastoiditis and individuals who had never been infected; but more research and study were needed to be able to diagnose individuals with both childhood *and* adult mastoiditis.

In non-adults, it was impossible to determine if primary hypocellularity was pathological (the result of stunting due to infection) or natural non-adult morphology and evidence that pneumatization had not yet begun in the individual. Pneumatisation occurs between the age of one year and puberty, approximately 12 years (Cinamon 2009; Gencer et al. 2013; O'Tuama and Swanson 1986). Hypercellularity also appeared differently in non-adults than it did in adults. In adults, hypercellular mastoid air cells (MAC) were relatively large and rounded. In non-adults, hypercellular MAC were narrow and elongated, with non-continuous cell walls (see Figure 5.5). MAC were also sometimes larger in the superior portion of the temporal bone, around the area of the *aditus ad antrum* and central tract (Bitar et al.

1996; Donaldson et al. 1992; Isaacson 2014). This likely reflected the progression of pneumatization from the middle ear into the MAC. Since the diagnosis of non-adult MAC was complicated by the pneumatization of the MAC, it was decided to exclude non-adults from this project. They remain a fascinating area of future research for a project devoted to tracking the pneumatization of MAC in diverse populations.

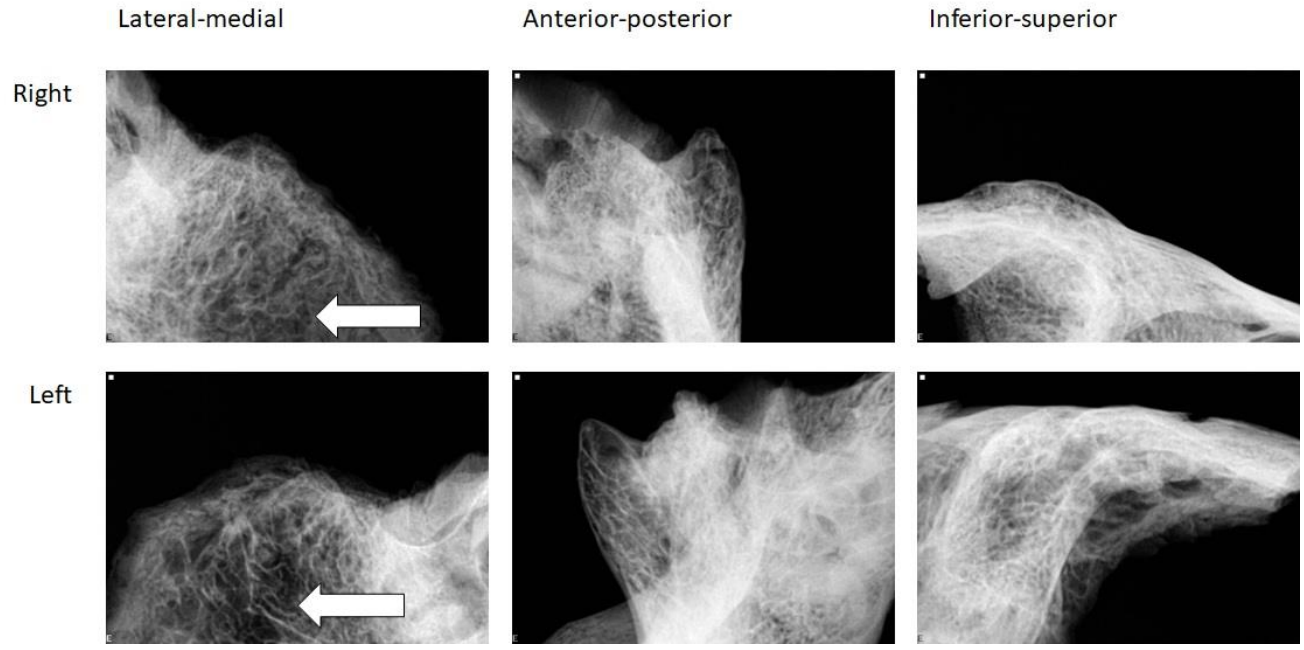


Figure 5.5 Black Gate Individual 479 (young child, 5.5–6.5 years), radiographs in three planes. Pneumatization created air cells that were elongated with non-continuous cell walls. The arrows indicate larger air cells around the area of the *aditus ad antrum* and central tract. Note how small the immature mastoid process appears. In the lateral-medial view, it barely projects above the petrous portion of the temporal bone.

To better understand the radiographic appearance of secondary hypocellularity, mastoid processes were X-rayed if they had post-mortem breaks in their cortex and there was sub-periosteal new bone visible in their MAC (see Figures 5.6 and 5.7). These radiographs were examined to see if sub-periosteal new bone was visible in the radiographs. Sub-periosteal new bone reliably appeared as clouding or as small, disjointed air cells/bone growth on the radiographs, thus this was one of the features used to assess the presence of secondary hypocellularity.

Figure 5.66 Right mastoid process of Black Gate Individual 433 with remodelled spicules of sub-periosteal bone visible in the mastoid air cells through a post-mortem break in the cortex of the superior-medial portion of the mastoid process. Arrows indicate the remodelled spicules in the enlarged photo. Spicules were visible radiographically (see Figure 5.7).

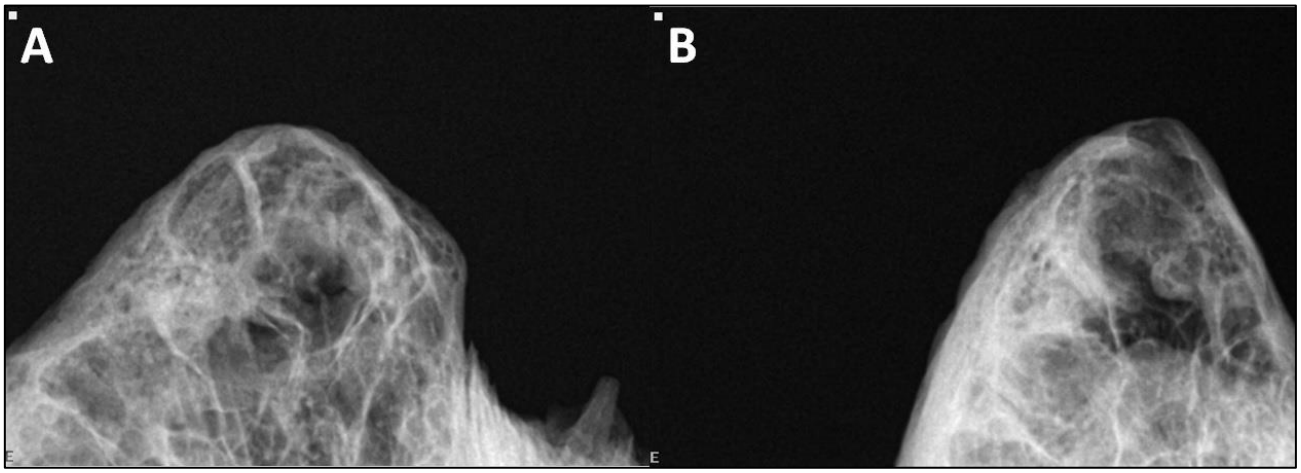


Figure 5.7 Black Gate Individual 433 with a secondary hypocellular right mastoid process imaged in the **A)** medial-lateral and **B)** posterior-anterior. The void in the center of the process, radiographically apparent by the dark–black exposure, is indicative of a post-mortem fracture in the mastoid process (see Figure 5.6).

Following further study of the radiographs and in consultation with Dr. Jaydip Ray (secondary supervisor to this project and Honorary Professor of Otology Neurotology and Consultant Otologist Neurotologist in the Department of Neuroscience at the University of Sheffield), it was determined that lytic lesions were observable in some individuals. Thus, an additional category of bone, called lytic and secondary hypocellularity (LSH), was created to reflect the presence of pathological bone destruction in some secondary hypocellular MAC (see

Figure 5.8). Lastly, some individuals presented with a combination of primary and secondary hypocellularity/LSH, which presented as small, dense air cells, some of which were secondarily filled with sub-periosteal bone growth and/or destroyed by lytic lesions (see Figure 5.8).

The diagnostic method created following the preliminary analysis, and used in this project, is described in detail in the diagnosis sub-section below.

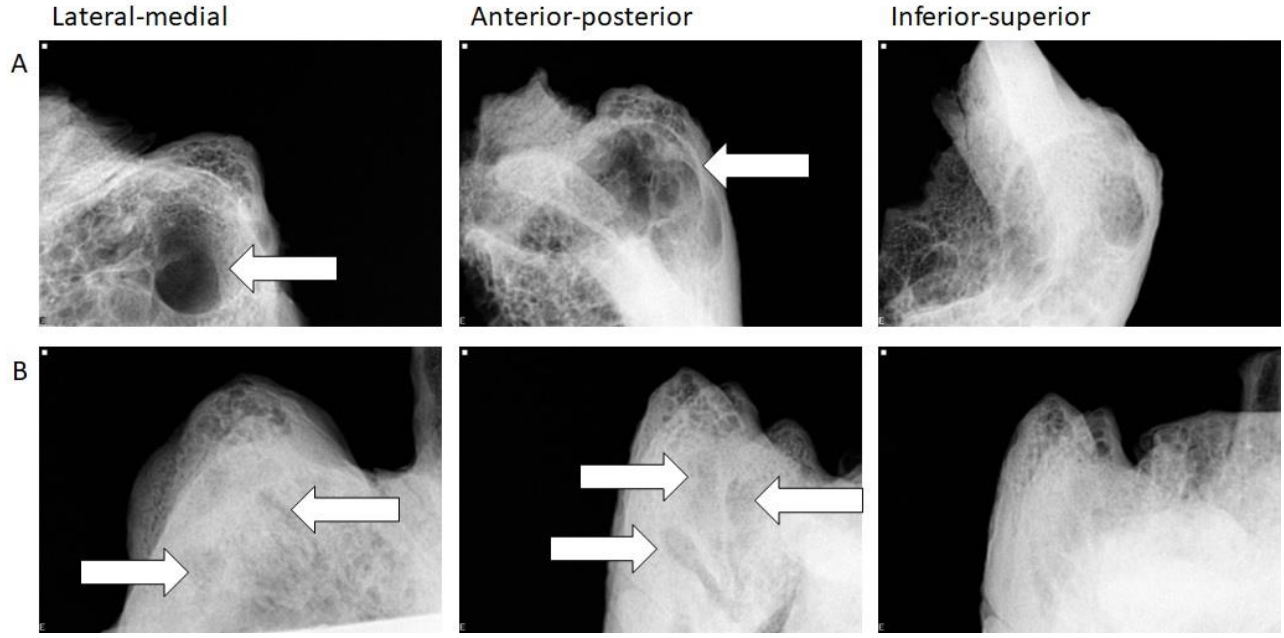


Figure 5.8 Two additional bone types identified during the preliminary analysis. **A)** Lytic and secondary hypocellularity diagnostic adult mastoiditis (BG51, right) and **B)** Both primary hypocellularity and LSH diagnostic of a mix of residual childhood mastoiditis and adult mastoiditis (BG287, left). Arrows indicate lytic lesions that destroyed the pre-existing air cell structure.

Regrettably, COVID-19 restrictions halted a CT study, in collaboration with Dr. Charles Romanowski (Consultant Neuroradiologist at Sheffield Teaching Hospital) and Sheffield Teaching Hospital, intended to compare the radiographs and diagnoses obtained using my method to those obtained by CT scanning the same skeletal individuals. CT scanning is the standard clinical tool for both the diagnosis and assessment of mastoiditis in the medical field (Hindi et al. 2014; Koç et al. 2003; Park et al. 2000; Sistani et al. 2019). In those studies that have examined mastoiditis archaeologically, CT has been used, because it produces high-fidelity radiographs and follows clinical methods (Collins 2019; Collins and Jónsson 2010; Primeau et al. 2018, 2019; Zhang et al. 2020). The aim of my study was to test my method against the clinical standard to see if the radiographs I obtained were as diagnostic. If they were, this would have demonstrated that my method was more accessible *and* as

diagnostic. Between national lockdowns, CT scans of two skeletal individuals were taken; but their analysis was put on hold and, ultimately, cancelled, due to the national implementation of further lockdown restrictions. Ultimately, the entire CT study was cancelled for the same reason.

Ray and Romanowski also consulted on my imaging method as I undertook my preliminary study. Their recommendations influenced the development of the method used in the wider study. After they observed me taking X-rays, they made a series of suggestions designed to improve the quality of radiographs and the replicability of the method. First, they recommended I place the sensor in the custom carved block of foam and position that on a tripod, so the sensor could be consistently perpendicular to the tabletop and parallel to the X-ray generator. Because the sensor never moved, transitioning between planes was faster and the distance between the sensor and the X-ray generator remained consistent. Second, they encouraged me to use anatomic landmarks to consistently align the mastoid process with the face of the sensor. Both the first and second recommendation were intended to increase the consistency between the radiographs and aid in the replicability of the method. Third, together we decided that the inferior-superior imaging plane was unnecessary. It was difficult—and sometimes impossible—to obtain in individuals with temporal bones articulated with the rest of the cranium; and it produced radiographs that were less diagnostic than those obtained using the other two planes, as the MAC were obscured by those superior. Fourth, Ray and Romanowski encouraged me to place the mastoid process as close to the face of the sensor as possible, to create clear radiographs with minimal blur. This meant rejecting the lateral-medial plane tested in the preliminary analysis and reverting to the medial-lateral plane used in Purchase *et al.* (2019). Finally, they encouraged me to tilt the temporal bone/cranium towards the X-ray generator in the medial-lateral plane, and to angle the bone/cranium in the anterior-posterior plane, to stop anatomical elements from obscuring the radiograph of the mastoid process. Their consultation helped me finalise the method presented in the imaging sub-section below.

Thus, I believe the new diagnostic method is more accurate than the former (Purchase et al. 2019). What were called "fully" or "partially" hypocellular MAC in Purchase *et al.* (2019), are now recognized as reflecting two distinct disease states. "Fully hypocellular" MAC are primarily hypocellular MAC; and "partially hypocellular" MAC are hypercellular MAC. The latter represent normal variation in which hypocellular bone is retained at the inferior mastoid process due to traction placed on the mastoid process by the sternocleidomastoid muscle (Flohr and Schultz 2009). What were called "lytic and sclerotic" MAC are now called secondarily hypocellular MAC or LSH MAC. The updated terminology

keeps the diagnosis consistent with the clinical language (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b) and accurately reflects the adult disease process.

All the adult individuals included in the preliminary study were re-imaged and re-diagnosed using the method developed from the preliminary analysis and were then included in the wider study. Since the goal of the preliminary analysis was to refine the imaging and diagnostic method, no statistical analysis was performed solely on the data generated from this analysis. The remainder of this section details the method developed from the preliminary analysis and used in this project.

Inclusion Criteria

Individuals were assessed for their inclusion in the study based on one criterion: the presence of at least one mastoid process with intact MAC. An observable mastoid process was defined as unfractured post-mortem with MAC that were unaffected by destructive taphonomic processes. This ensured the MAC included in the study reflected in *vivo* morphology not taphonomic change. To maximize the sample size, individuals with a mastoid process damaged post-excavation were included in the study only if the cortical bone was damaged but the MAC appeared undamaged. Post-excavation taphonomic damage was distinguished from pre-excavation damage by the colour of the exposed bone: clean, white broken edges. If the cortical bone appeared to be damaged post-excavation and the exposed MAC did not, then the mastoid process was considered observable. If the cortical bone appeared to have been destroyed pre-excavation, then the mastoid process was considered unobservable, as it was likely that the MAC had also been destroyed/altered.

MAC are usually bilaterally pneumatized (Flohr et al. 2009, 2019; Titche et al. 1981; Virapongse et al. 1985). Therefore, one observable mastoid process per individual was deemed sufficient for the individual to be considered observable for mastoiditis. Nevertheless, when possible, both mastoid processes were imaged and recorded, as unilateral pneumatization can be a sign of mastoiditis (Flohr et al. 2019; Virapongse et al. 1985).

All adult individuals (16+ years) were observed. Both the clinical (Groth et al. 2012; Luntz et al. 2012; Mathews et al. 1988; NHS 2017) and palaeopathological (Krenz-Niedbała and Łukasik 2016b; Schultz et al. 2007) literature note that children (up to approximately seven years of age) rather than adults are more likely to suffer from mastoiditis. Despite this, most researchers have excluded children from their studies to avoid conflating growing and pneumatizing bone with pathology (Flohr and Schultz 2009a; Schultz et al. 2007). As observed here in the preliminary analysis, primary hypocellularity and hypercellularity appeared in both

young and older children which made it impossible to determine in whom pneumatization was stunted and in whom pneumatization was progressing as normal. Thus, non-adults were excluded from the wider study to eliminate this source of error. Rather, the rate of childhood mastoiditis was studied by examining the rate of primary hypocellularity (diagnostic of residual CM) in the adult population.

Imaging

All observable mastoid processes were imaged using a NOMAD Pro hand-held X-ray system. This is a system developed for high-fidelity, flexible intraoral dental imaging performed both in and out of the dental clinic (KAVO 2020). The size of the sensor and portability of the system make the NOMAD Pro well suited for archaeological research (Purchase et al. 2019). The sensor was plugged into a laptop via a USB and the digital radiographs were received by the Super Eyes software. The software allowed the radiographs to be saved to the computer as a jpg photo. The sensor, X-ray generator (or generator), and imaging platform were set up in the following way. The sensor was held by a custom-carved block of foam (see Figure 5.9). It gripped the back and sides of the sensor and did not obscure the imaging surface. The block of foam was then fit into a smartphone mount on a tripod. The tripod was placed on the tabletop behind the imaging platform (a box lid). The tripod and imaging platform remained in position throughout the imaging process. X-ray images were taken with the generator positioned above the edge of the imaging platform, directly opposite the sensor. The distance between the sensor and the generator was 60cm. By not moving the sensor or imaging platform, and by not changing the position of the generator when imaging, the distance between the sensor and the generator remained consistent and produced consistently exposed radiographs.

Figure 5.9 A disarticulated temporal bone held in place by foam blocks. The sensor can be seen on the left, held in a custom carved block of foam in the grip of a smartphone mount on a tripod.

The temporal bone was inverted, axis down, on the imaging platform and held in place with soft foam. If disarticulated, the squamous portion was inserted into a cut in one block of foam (see Figure 5.9). If the temporal bone was articulated, then the cranium was placed, axis down, on a foam ring (see Figure 5.10). Since archaeological bone is inconsistent in its fragmentation/articulation, the method of securing the bone to achieve the planes sometimes varied (e.g., additional blocks of foam may have been necessary to safely hold and support the bone in position).

Figure 5.107 An articulated temporal bone held in place and supported by a Styrofoam ring. The cardboard box lid can be seen below the ring, elevating the mastoid process to the same height as the sensor (visible in the background).

The mastoid process was imaged in two planes: medial-lateral 30° off the sagittal plane and posterior-anterior 30° off the coronal plane. To align the temporal bone with the sensor, an imaginary line must be drawn on the bone. This line was oriented with the sensor's imaging surface to consistently achieve the planes. Three lines were used depending on the fragmentation of the temporal bone. The first line was the TMJ-MP line. It runs between the point where the articular tubercle meets the mandibular fossa in the temporomandibular joint (TMJ) and the inferior tip of the mastoid process (MP). In most skulls, this roughly follows the inferior edge of the external auditory meatus (EAM) (see Figure 5.11A). When the TMJ is not present, the second line is used: the EAM-MP line. This runs between the anterior-inferior edge of the EAM and the inferior tip of the mastoid process (see Figure 5.11B). The EAM-MP line is only used when the TMJ is not present, as the morphology of the TMJ is more consistent than that of the EAM. In exceptional circumstances, when the temporal bone was so fragmented that the mastoid process was separate from the rest of the bone and the TMJ-MP and EAM-MP lines could not be drawn, then the third line was used. This was the MP line.

Here, a theoretical line was drawn following the natural, oblong structure of the mastoid process: between the anterior and posterior extremities of the mastoid process (see Figure 5.11C). Because this is a smaller line, it was less preferable to the other two, but used when necessary to retain consistency between the planes. If the MP line was not present, then the bone was considered unobservable.

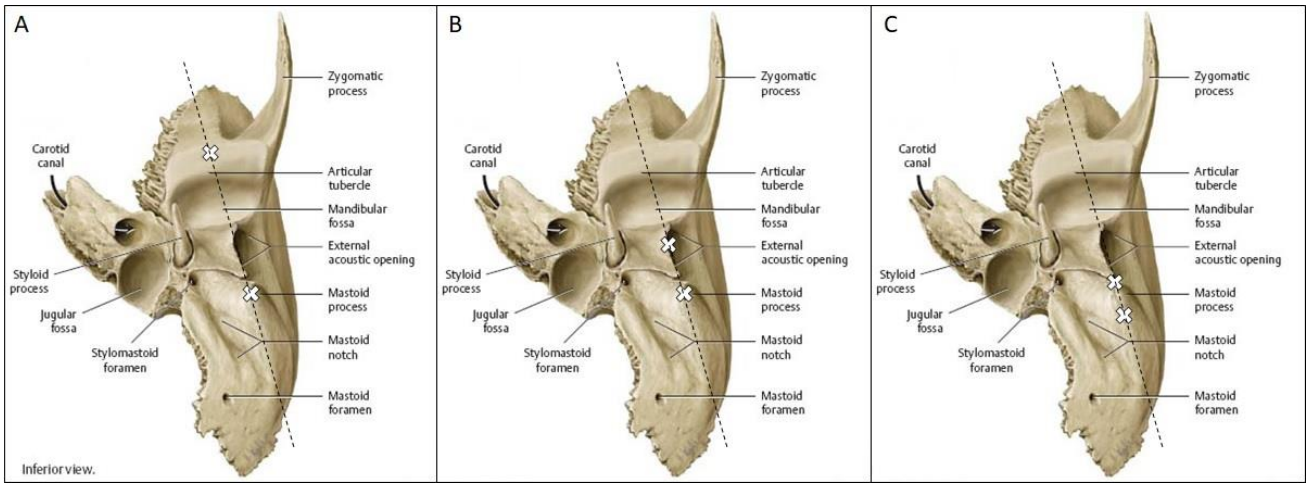


Figure 5.118 Dashed line showing **A)** the TMJ-MP line, **B)** the EAM-MP line, and **C)** the MP line. x on the bones marks the points used to create the lines (image adapted from Gilroy et al. 2008:454).

To take the radiograph medial-lateral 20° off the sagittal plane, the bone was oriented so that the theoretical line was parallel with the sensor's imaging surface (or in the sagittal plane) and the lateral surface of the mastoid process was as close to the sensor's imaging surface as possible. Then the inferior portion of the bone was angled 20° off the sagittal plane, away from the sensor's imaging surface, towards the generator. Thus, in fully articulated skulls, the image of the mastoid process was unobscured by the contralateral mastoid process. A piece of triangular foam was placed under the bone/foam/foam ring to achieve this angle (Figure 5.12).

Figure 5.12 Setup for imaging medial-laterally 20° off the sagittal plane.

To image posterior-anterior 45° off the coronal plane, the theoretical line was positioned parallel to the sensor's imaging surface (or in the sagittal plane) with the anterior of the temporal bone positioned towards the sensor and the posterior positioned towards the generator. The bone was then pivoted 45°, so that the TMP/EAM/anterior end of the theoretical line was 45° off of the sagittal plane. Thus, the zygomatic bone, if present, was next to the sensor and not obscuring the image of the mastoid process. The anterior surface of the mastoid process was as close to the sensor's imaging surface as possible (see Figure 5.13).

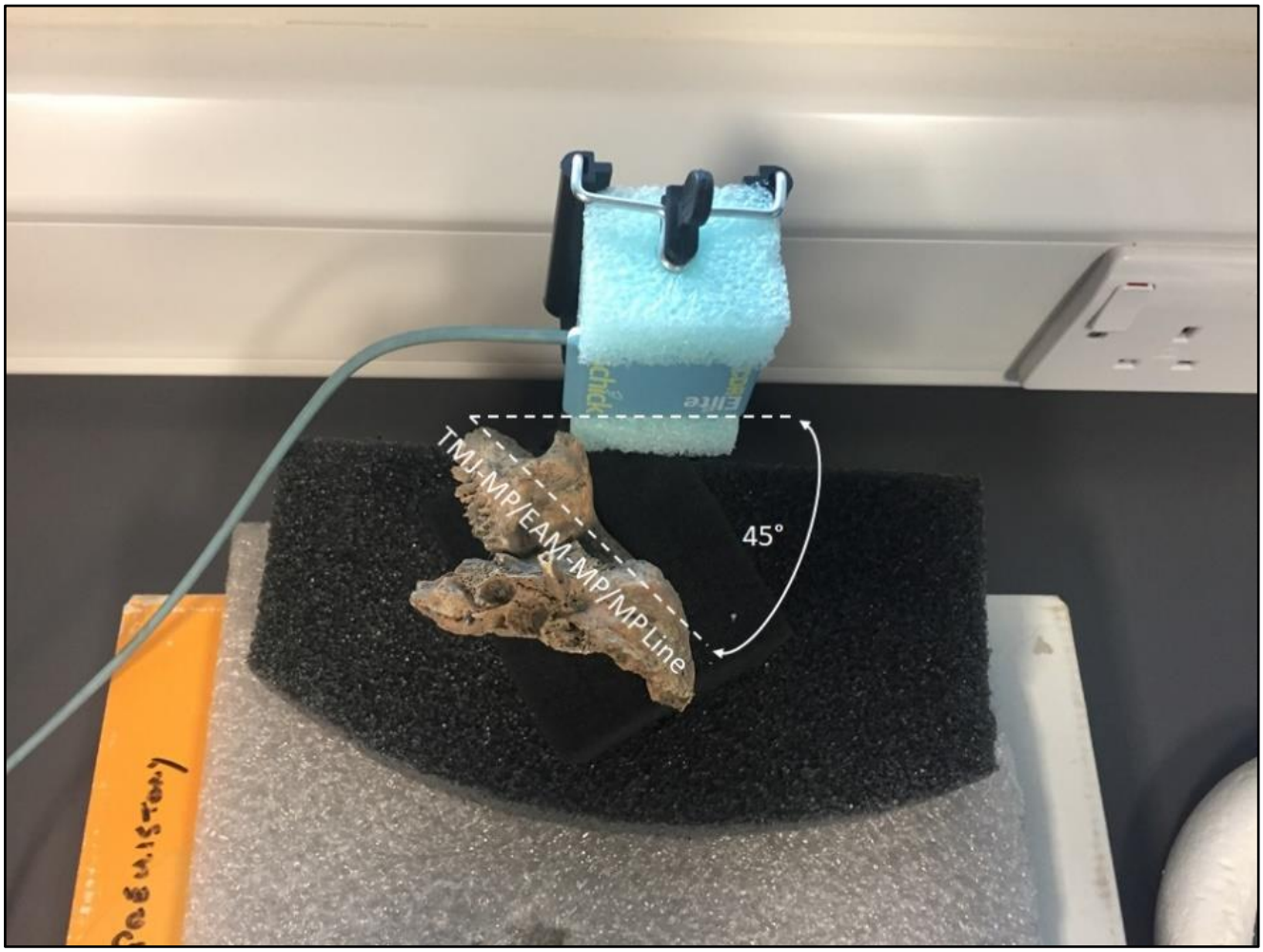


Figure 5.139 Setup for imaging posterior-anteriorly 45° off the TMJ-MP/EAM-MP/MP Line.

Diagnosis and Recording

In this section, the radiological features that characterize adult and childhood mastoiditis are described as well as the terms used to define them. This includes hypercellular, primary hypocellular, and secondary hypocellular bone. The terms "hypercellular" and "hypocellular" are used here, in keeping with the clinical (Koç et al. 2003) and recent archaeological (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b) literature. The terms "pneumatic", "diploic", "sclerotic", "mixed", and "atypical" (Gregg and Steele 1982; Tremble 1934) also appear in the archaeological literature to classify the morphology of the MAC. For consistency with the modern literature, they were only used here to describe aspects of the MAC's morphology, not to classify the morphology. At the end of the section, a flow chart summarises the diagnostic method developed and used in this project.

In general, *hyper*cellular bone is defined by the presence of large air cells and *hypo*cellular bone is defined by that of many small air cells. Specifically, hypercellular bone is fully pneumatized adult bone (see Figure 5.14). The extent to which each individual's MAC are pneumatized varies (and may be genetically determined, according to the genetic

theory) (Cheatle 1910; Cinamon 2009; Diamant 1940a, b; Sadé et al. 2006). So long as the MAC walls were the same density and uninterrupted by lytic or sclerotic lesion(s), the radiograph was diagnosed as hypercellular.

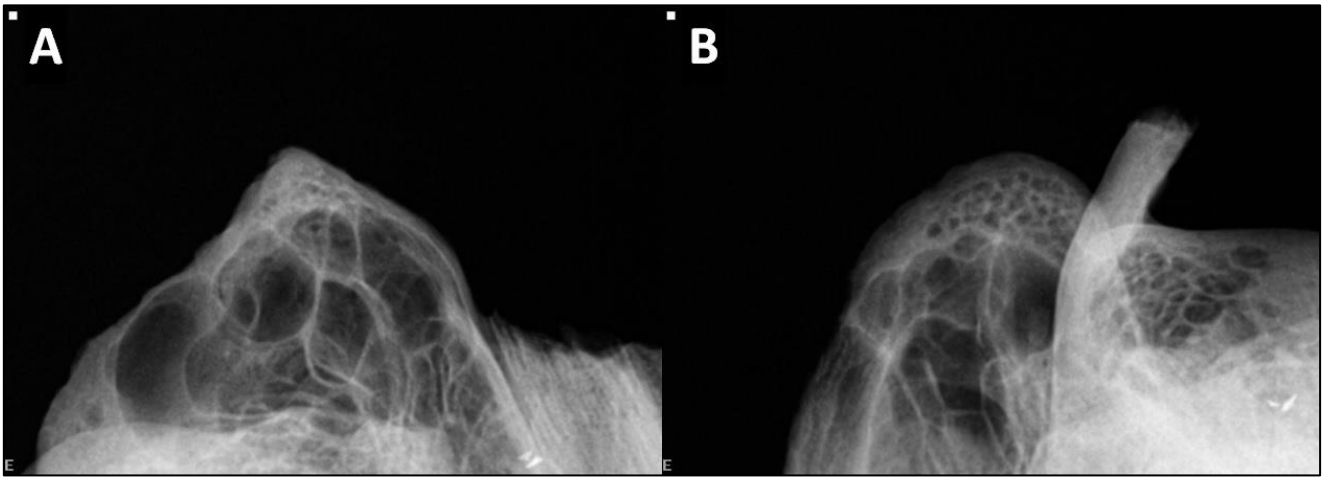


Figure 5.14 Coronation Street Individual 195 with a hypercellular right mastoid process imaged in the **A)** medial-lateral and **B)** posterior-anterior planes. The air cells are large and clear. Note the residual primary hypocellularity in the tip of the mastoid process. This is a a natural variation that may reflect a genetic predisposition towards incomplete pneumatization (Cheatle 1910; Cinamon 2009; Diamant 1940a, b; Sadé et al. 2006) or tension placed on the process by the sternocleidomastoid muscle (Flohr and Schultz 2009).

In contrast, hypocellular bone is pathological in origin. It represents either childhood or adult mastoiditis: primary or secondary hypocellularity, respectively. Primary hypocellularity is the result of the complete or partial stunting of the pneumatization process by inflammation and the retention of a juvenile bone structure in the temporal bone (Sadé et al. 2006; Tos and Stangerup 1985). Primary hypocellularity is diagnostic of childhood mastoiditis and is a risk factor for further episodes of mastoiditis (Sadé et al. 2006; Tos and Stangerup 1985). Any mastoid process that was filled consistently with small, dense cells similar to those pictured in Figure 5.15, was recorded as primary hypocellular and the individual diagnosed with at least one episode of childhood mastoiditis.

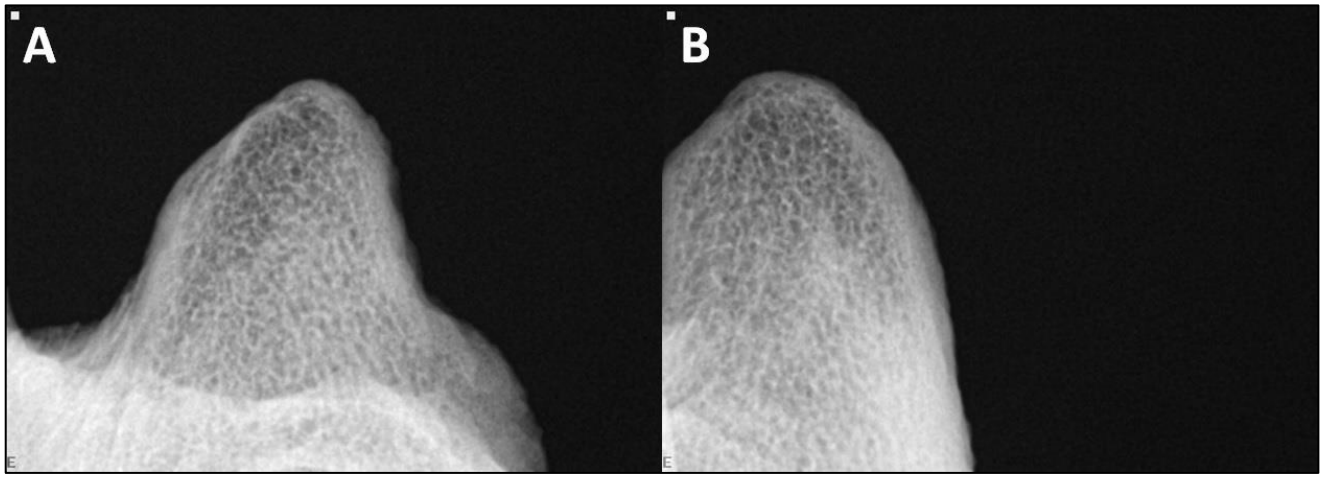


Figure 5.15 Black Gate Individual 154, primary hypocellular left mastoid process imaged in the **A)** medial-lateral and **B)** posterior-anterior. The air cells are small and clear.

Secondary hypocellularity is the result of inflammation that triggers bone destroying and/or forming processes that result in the proliferation of sub-periosteal new bone growth inside pre-existing and/or pathologically enlarged air cells (Aoki et al. 1986; Qvarnberg 1982). In some individuals, this is characterized by the proliferation of sub-periosteal new bone within the MAC (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b). This appeared in the radiographs as clouding within the MAC. Adult mastoiditis was also diagnosed based on the presence of lytic and sclerotic lesions that interrupted the otherwise regular structure of the MAC. As discussed in the clinical background chapter, both acute and chronic infections result in thinned and decalcified MAC walls with sclerotic boundaries (Hellier 2018; Hug 2000; Virapongse et al. 1985; Qvarnberg 1982). The thinning of the MAC walls is a natural progression of inflammation (Bluestone 1998; Hellier 2018; Mathews et al. 1988; Welin 1941) or the result of a cholesteatoma (Mathews et al. 1988). Two types of secondary hypocellularity were classified in the preliminary analysis. If the MAC appeared disjointed and filled with sub-periosteal new bone growth, then the MAC was classified as having secondary hypocellularity (see Figure 5.16). If there was lytic destruction in the mastoid process, which appeared to interrupt the otherwise consistent morphology of the MAC, accompanied by sclerotic or secondary hypocellular bone around the edges of the lytic lesion, then the MAC was classified as LSH (see Figure 5.17). LSH was often observed in individuals who had residual CM and subsequently had mastoiditis as adults.

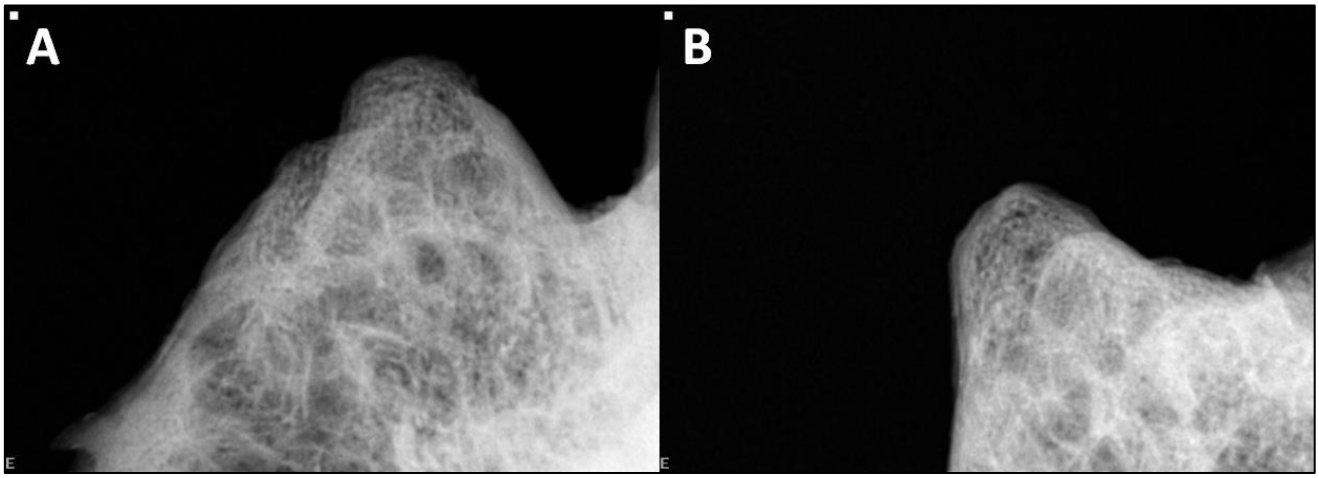


Figure 5.1610 Black Gate Individual 77, secondary hypocellular right mastoid process imaged in the **A)** medial-lateral and **B)** posterior-anterior. The air cells are large, but secondarily filled with sub-periosteal bone. Note the residual primary hypocellularity in the tip of the mastoid process. This is a natural variation that may reflect a genetic predisposition towards incomplete pneumatization (Cheatle 1910; Cinamon 2009; Diamant 1940a, b; Sadé et al. 2006) or tension placed on the process by the sternocleidomastoid muscle (Flohr and Schultz 2009).

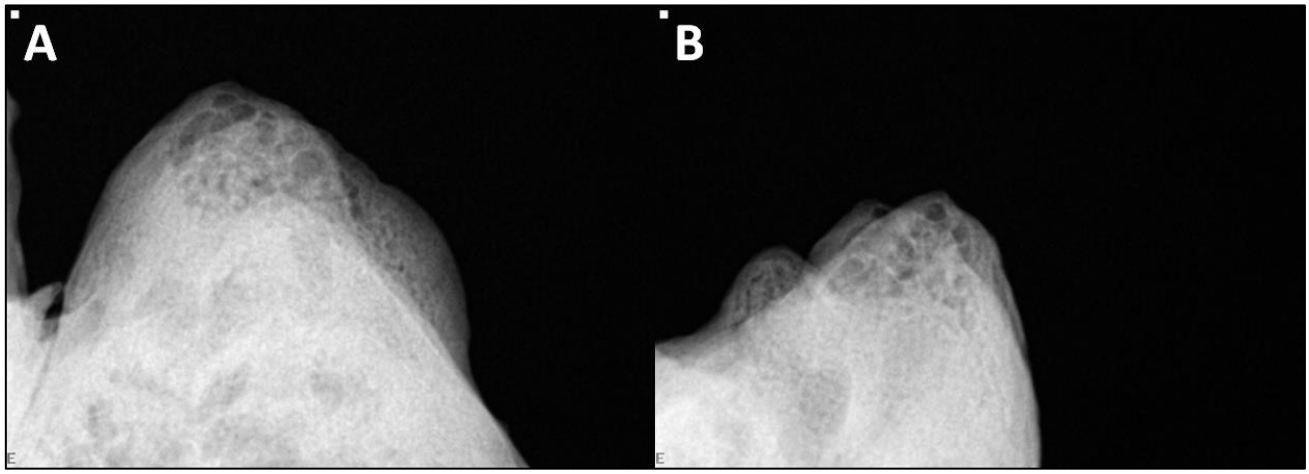


Figure 5.17 Black Gate Individual 287, lytic and secondary hypocellular (LSH) and primary hypocellularity left mastoid process imaged in the **A)** medial-lateral and **B)** posterior-anterior. The air cells are small, and secondarily filled with sub-periosteal bone and/or destroyed by lytic lesions.

Some individuals were diagnosed with both childhood and adult mastoiditis. This was defined by a primary hypocellular bone structure, that was subsequently affected by lytic and/or secondary hypocellularity. There was no evidence of pneumatized, adult hypercellular bone. Rather, primary hypocellular bone was secondarily filled by sub-periosteal bone, creating a stippled look in a radiograph (see Figure 5.17). If LSH was present, lytic lesions interrupted the

MAC and were often surrounded by sclerotic and/or secondarily hypocellular bone, creating the same stippled look in a radiograph (see Figure 5.18). Finally, Figure 5.19 summarises the diagnostic method developed and used by this project and demonstrates how bone can be classified from a radiograph and how a diagnosis can be achieved.

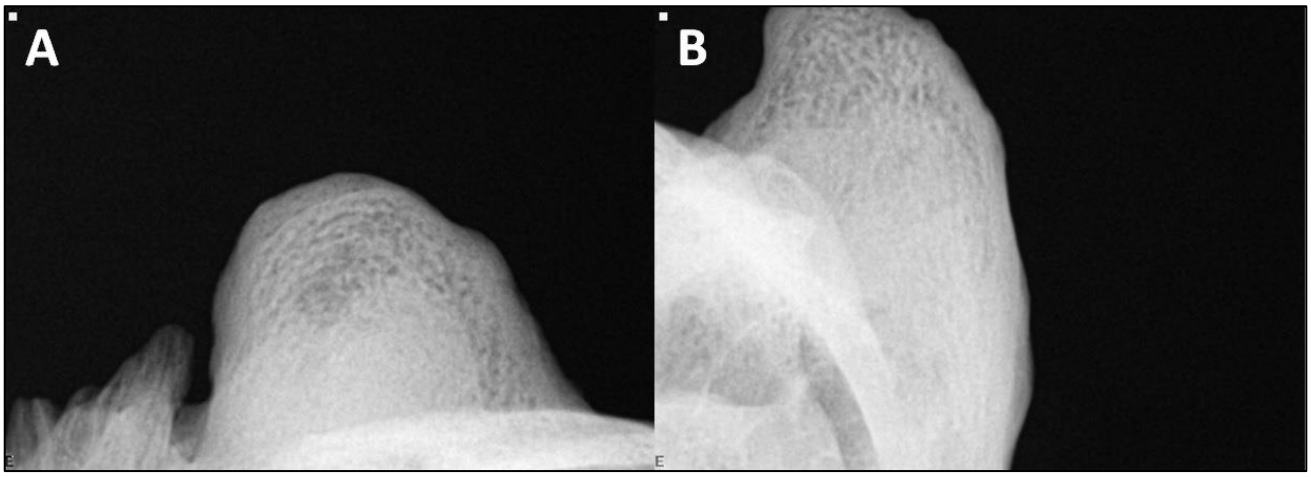


Figure 5.18 Black Gate Individual 83, primary and secondary hypocellular left mastoid process imaged in the **A)** medial-lateral and **B)** posterior-anterior. The air cells are small, and the middle MAC are secondarily filled with sub-periosteal bone.

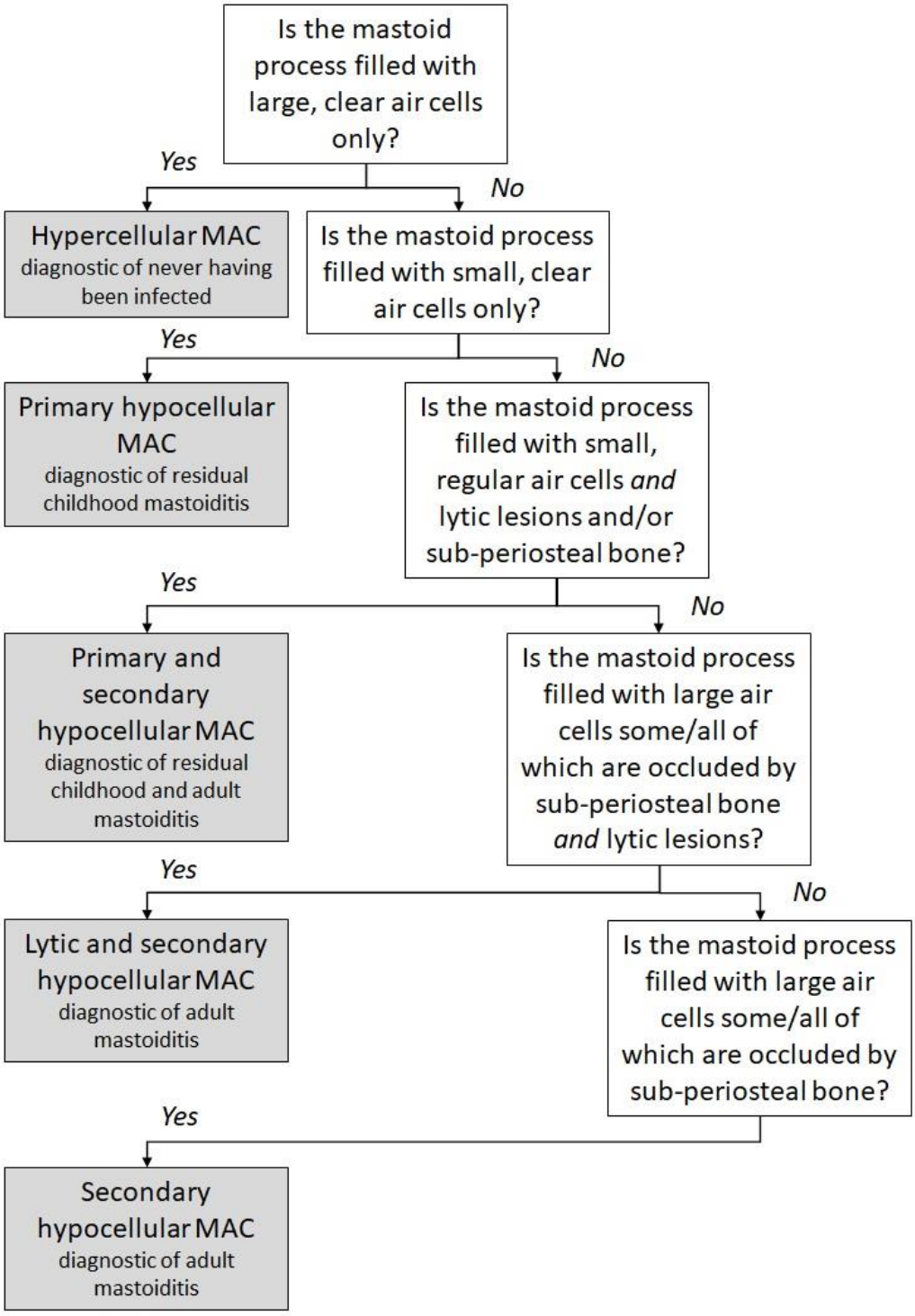


Figure 5.19 Flow chart summarising the method of diagnosing residual childhood and/or adult mastoiditis or never infected mastoid air cells based on radiographs of the mastoid process. MAC refers to mastoid air cells.

Both binary (mastoiditis present/absent) and categorical (primary hypocellular, secondary hypocellular, or hypercellular) data were recorded at the level of the anatomical structure—the left or right mastoid process. If an individual had data for both the left and right mastoid processes, then they could be analysed for unilateral and bilateral mastoiditis. If both radiographs were diagnosed the same (e.g., hypercellular), then the individual was

recorded as bilateral for that diagnosis (e.g., bilateral hypercellular and mastoiditis as absent). If one radiograph was diagnosed as hypocellular and the other as hypercellular, the individual was diagnosed with unilateral mastoiditis and mastoiditis as present overall. If only one radiograph was present for an individual, then the diagnosis of that radiograph was the diagnosis of the individual and they were considered unobservable for unilateral and bilateral mastoiditis. In this way, the behaviour of mastoiditis was studied within and between populations at both the individual and population levels.

This methodology for the X-ray imaging and visual diagnosis of mastoiditis from radiographs is innovative in its use of a portable, hand-held X-ray system and its creation of novel imaging planes. The method of diagnosing hypocellularity is clinically founded and explores the diagnosis of an overlooked age group—children. It remains a minimum estimate of infection, however, as early stages of infection may not be visible radiographically and remodeled lesions may appear hypercellular radiographically. The remainder of this chapter outlines the methodology used in the data collection, analysis, and diagnosis of maxillary sinusitis and lower respiratory infection.

5.3 Maxillary Sinusitis

The following section outlines the methodology used to diagnose and record maxillary sinusitis in the samples studied. It describes the inclusion criteria and diagnostic criteria, and the method of data recording. This project makes use of the diagnostic criteria developed by Boocock *et al.* (1995) and that is described and illustrated.

Inclusion Criteria

An individual was considered observable for maxillary sinusitis if they were older than 16 years and at least half the floor of one sinus was unobscured by dirt or other debris, undestroyed by taphonomic processes, showed no osteological evidence of apical abscessing, and was able to be examined with the naked eye. The maxillary sinuses were assessed visually and only *via* pre-existing breaks, so as to not be invasive or destructive (e.g., Liebe-Harkort 2010).

Non-adults (<16 years) were eliminated to avoid false positives, as the sinuses do not complete growth until 12 years (Cinamon 2009) and bone growth and remodelling can create pitting that can be mistaken for pathology (Lewis et al. 1995; Roberts 2007; Schultz et al. 2007). This is the same age limitation set by Lewis *et al.* (1995). Others have set the exclusion limit at five months (Schultz et al. 2007) and 20 years (Roberts 2007). Setting the exclusion limit at five months was too young to exclude all signs of growth, while a limit of 20

years was considered overly conservative. Adults (16+ years) were analysed to be consistent with the inclusion criteria for mastoiditis.

Half of the floor of one maxillary sinus was used as a minimum requirement, as the majority of lesions reported in the archaeological literature have been located on the maxillary sinus floor (Roberts and Manchester 2005:174; Lewis 2007:137; Roberts 2007). Since lesions can be unilateral (Roberts 2007) but are not always so (Boocock et al. 1995), the diagnosis represents a minimum number of affected individuals—there may be false negative diagnoses. This was accounted for in the analysis and interpretation of data. Requiring a minimum of one maxillary sinus for diagnosis was consistent with previous archaeological research (Boocock et al. 1995; Krenz-Niedbała and Łukasik 2016a; Liebe-Harkort 2010; Roberts 2007; Lewis et al. 1995) and maximized the sample size.

As explained in section 2.3.2, maxillary sinusitis can be odontogenic (Hajiioannou et al. 2010; Brook 2006). Including such cases would complicate a discussion of environmental risk factors. To exclude them, maxillary sinuses that presented with an oroantral fistula in their floor were deemed unobservable, as such lesions are evidence of a direct connection between the sinus and the mouth *via* an apical abscess (Boocock et al. 1995; Krenz-Niedbała and Łukasik 2016a; Liebe-Harkort 2010; Roberts 2007; Lewis et al. 1995). Individuals with bony evidence of periodontitis were not eliminated from the study. As a result, some of the cases of maxillary sinusitis reported here may have been odontogenic.

Diagnosis and Recording

Diagnosis followed the categories laid out by Boocock *et al.* (1995:486). In their early work concerning the etiology and pathogenesis of maxillary sinusitis, they defined four types of lesions within the maxillary sinuses that reflected the presence of chronic sinusitis. This typology has since become the standard used by other paleopathologists and has been used to classify chronic sinusitis lesions within the other facial sinuses (Casna and Schrader 2021; Collins 2019; Davies-Barrett 2018; Davies-Barrett et al. 2021a,b; Digangi and Sirianni 2017; Krenz-Niedbała and Łukasik 2016a; Lewis 2002; Lewis et al. 1995; Merrett and Pfeiffer 2000; Panhuysen et al. 1997; Purchase 2016; Roberts 2007; Shapland et al. 2015).

The types are as follows: pitting, spicules, remodelled spicules, and white pitted bone (Boocock et al. 1995). Pitting is fine and often accompanies other types of lesions. Spicules are cancellous and thin bone growths that appear surficial to the natural bone surface. Remodelled spicules, conversely, are spicules that appear to have merged with each other and the natural bone surface. And white pitted bone is often isolated and can extend to the external bone surface (Boocock et al. 1995) (see Figure 5.20).

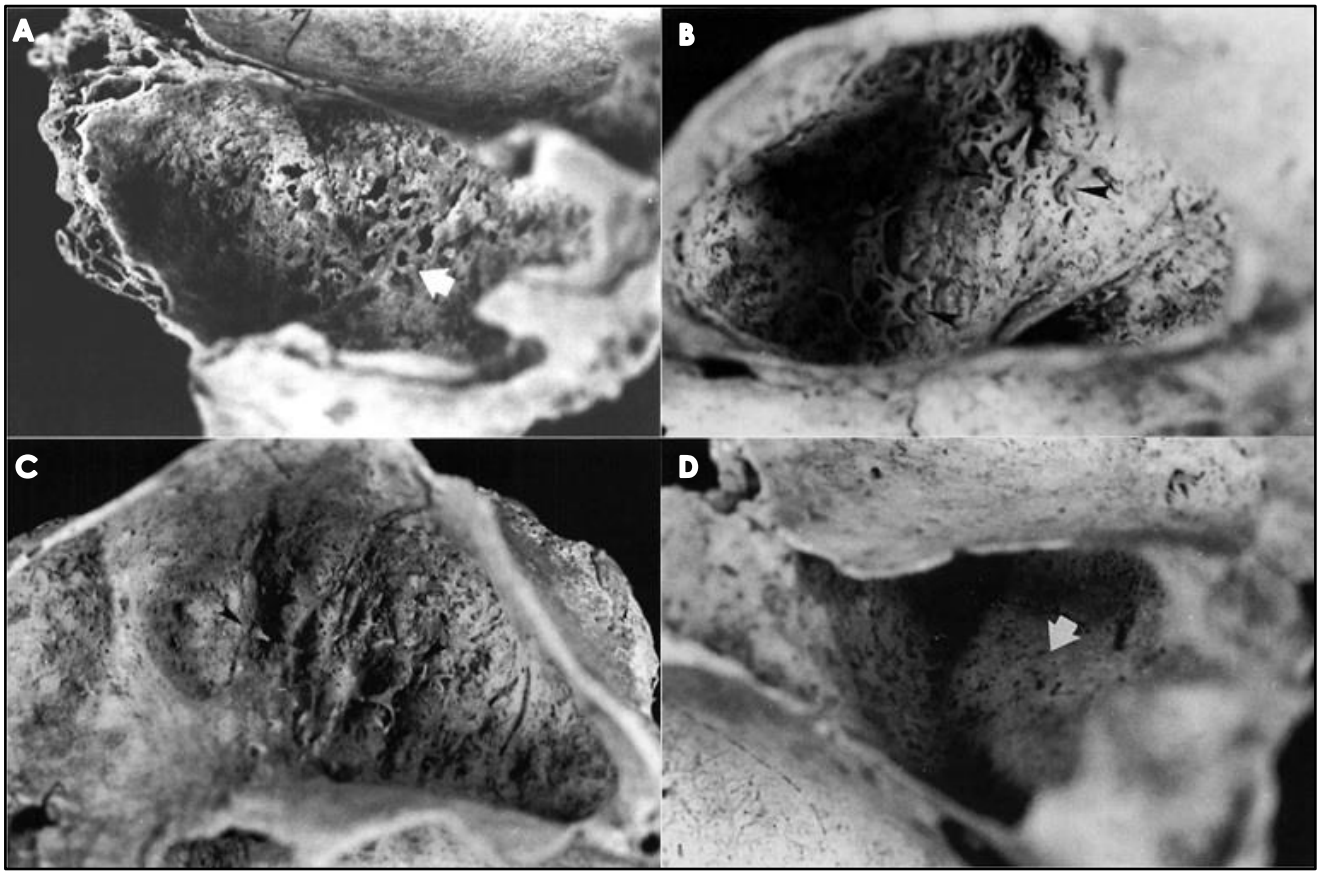


Figure 5.20 The four types of bony lesions characteristic of maxillary sinusitis (indicated by arrows): **A)** pitting, **B)** spicules, **C)** remodelled spicules, and **D)** white pitted bone (adapted from Boocock et al. 1995:486–9).

Categorical data (pitting, spicules, remodelled spicules, white pitted bone, or healthy) was recorded at the level of the anatomical structure—the left or right maxillary sinus—and this was then pooled as binary (presence/absence of maxillary sinusitis) data at the level of the individual. If both left and right maxillary sinuses were observable and both or one was diagnosed with pitting, spicules, remodelled spicules, or white pitted bone, then the individual was recorded as present for maxillary sinusitis. If both maxillary sinuses were observable and both were recorded as healthy or if only one maxillary sinus was observable and was recorded as healthy, then then individual was recorded as absent for MS. The number of sinuses which were considered observable and were included in the project was recorded to construct a crude and true prevalence of lesions. And unilateral and bilateral data was recorded to analyse the pathophysiology of the infection.

5.4 Lower Respiratory Infection

Unlike mastoiditis and sinusitis, LRI is not representative of a single disease process but, rather, encompasses a host of diseases, not all of which are infectious in origin (Burney et al. 2017;

WHO 2019a). It is not conclusively known which diseases cause sub-periosteal reactions on the visceral surfaces of the ribs, as clinicians do not regularly examine the skeleton when diagnosing LRI (Mok and Simpson 1984; Moore et al. 2008) and archaeologists are hampered by comorbidities and misdiagnoses and misreporting in the historical documentation which accompanies some skeletal collections (Kelley and Micozzi 1984; Roberts et al. 1994). Thus, the study of LRI requires an approach that facilitates differential diagnosis and eliminates lesions caused by non-infectious diseases. The methodology developed by Davies-Barrett *et al.* (2019) records the true prevalence of proliferative visceral surface rib lesions (VSRL), to facilitate such diagnoses and distinctions. Therefore, it was employed by this project. The following describes and illustrates the use and application of this methodology within this project. As with the previous section, it begins by discussing the inclusion criteria and ends with detailing the data recording method.

Inclusion Criteria

Inclusion was determined based on individual and element-specific criteria. Firstly, Davies-Barrett *et al.* (2019) set their limit at 17 years (using the fusion of the costal-vertebral facet as an age indicator); however, this project set the age limit at 16 years to remain consistent with the inclusion age set for diagnosing mastoiditis and MS. In young adults, special attention was given to the appearance of the bone to avoid misdiagnosing growth as VSRL (Davies-Barrett et al. 2019; Lewis 2017; Ortner 2012).

Secondly, using the zoning method (Knüsel and Outram 2004), Davies-Barrett *et al.* (2019) divided every rib into three anatomical zones: the head, angle, and shaft. The head included all the elements of the rib posterior to and including the tubercle. The angle included the portion between the tubercle and the oblique line. And the shaft included the remaining portion anterior to the oblique line (see Figure 5.21).

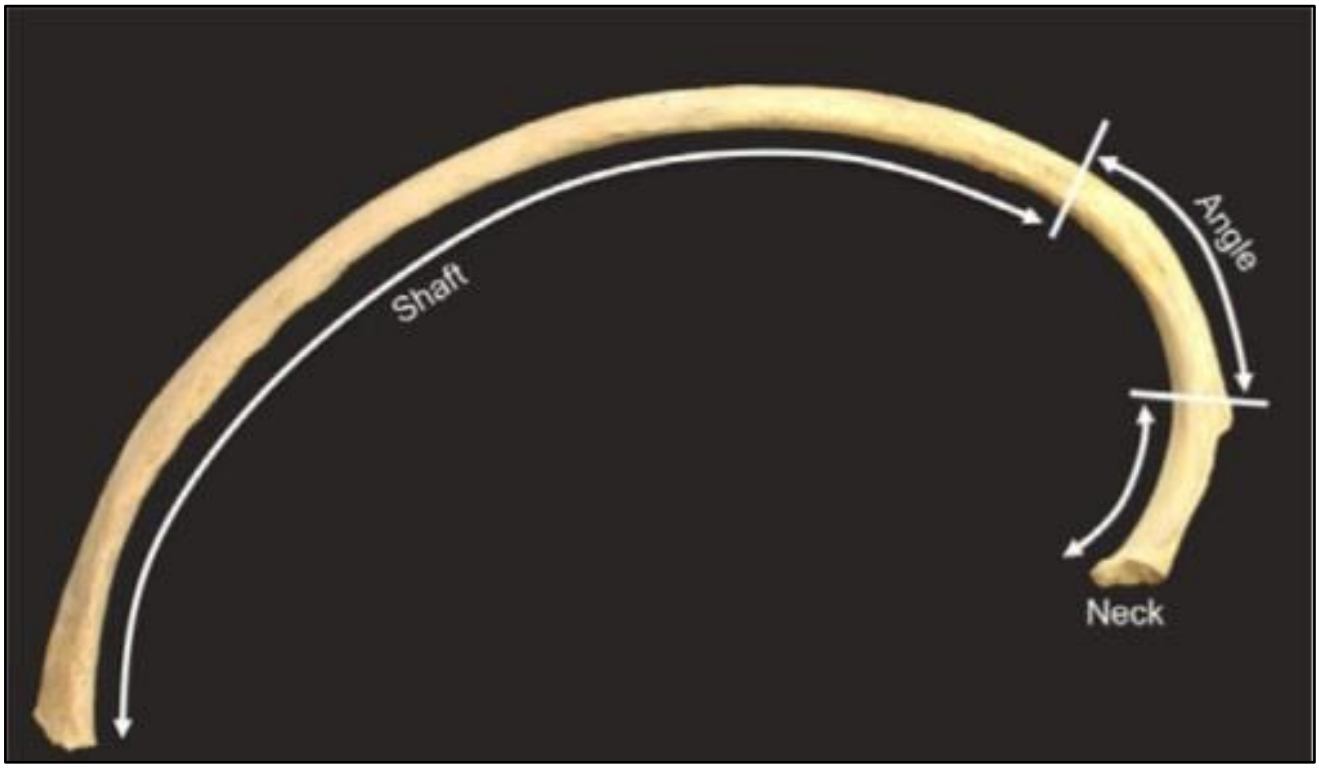


Figure 5.21 The three zones into which every rib was theoretically divided (from Davis-Barrett et al. 2019:534).

Some LRI and non-infectious stimuli, such as muscle traction (Nicklisch et al. 2012), create VSRL in specific zones (Kelley and Micozzi 1984; Matos and Santos 2006; Roberts et al. 1994). Thus, recording the zone in which a lesion presented could facilitate differential diagnosis of VSRL and help identify non-infectious lesions (Davies-Barrett et al. 2019). If at least half of a zone was present, then it was considered observable and recorded as present (Davies-Barrett et al. 2019). If a lesion was present on a fragment less than half-its natural size, it was recorded if the zone was identifiable and well preserved to represent the infection rate as accurately as possible.

Diagnosis and Recording

The methodology developed by Davies-Barrett *et al.* (2019) recorded the true prevalence of lesions. This referred to the prevalence of lesions per observable zone oriented within the rib cage within a sample; this was different from a crude prevalence that referred to the prevalence of lesions per individual within a sample (Davies-Barrett et al. 2019). Using a true prevalence rate accounted for the individual as well as the position of the lesion in the rib cage and the rib cage's fragmentation and preservation; thus, creating larger sample sizes in highly fragmented samples, facilitating differential diagnosis, and improving comparability between sites. In comparison, crude prevalence recorded the presence of a lesion within a sample with only the individual as context (Davies-Barrett et al. 2019). It should be noted that

this remained a minimum estimate, as not all LRI leave bony lesions (Ortner 2003; Ortner and Putschar 1985), lesions can remodel over time, and not all lesions or ribs preserve (Roberts and Manchester 2005:12–4; Wood et al. 1992). These factors were partially accounted for by recording the fragmentation and the cortical preservation of the ribs; and the absence of evidence was accounted for in the discussion. This section explains how VSRL were diagnosed and recorded in this project using this method.

To create a true prevalence, the following features were recorded for every zone: the fragmentation and cortical preservation scores for the entire rib cage, the side of the body (left or right), the seriation of the rib, and the zone. The fragmentation of the rib cage was scored using a scale developed by Davies-Barrett *et al.* (2019) (see Table 5.5) and the cortical surface preservation was scored using a guideline developed by McKinley (2004) for BABAO (see Figure 5.22). Individuals whose ribs scored a 5 or 5+ in cortical preservation were not included in the analysis, as they were too eroded to accurately reflect the true morphology of the rib. Finally, the seriation of the rib was recorded in groups based on the position of the rib in the rib cage: upper (ribs 1–3), upper-middle (ribs 4–6), lower-middle (ribs 7–9), and lower (ribs 10–12) (Davies-Barrett et al. 2019:533).

Table 5.5 Rib cage fragmentation scores (format adapted from Davies-Barrett et al. 2019:433).

Score	Description
None (0)	• No fragmentation except very minor "chipping" at peripheral and vulnerable areas.
Minor (1)	• Fragmentation consists of large fragments, easily pieced back together. • Less than 25% of each element is affected.
Moderate (2)	• Between 25 and 75% of each element is fragmented. • Pieces may range in size. • The morphology of the elements may still be recognized when pieces are aligned together.
Severe (3)	• Fragmentation is extreme, with small, often unidentifiable fragments consisting of more than 75% of each element.

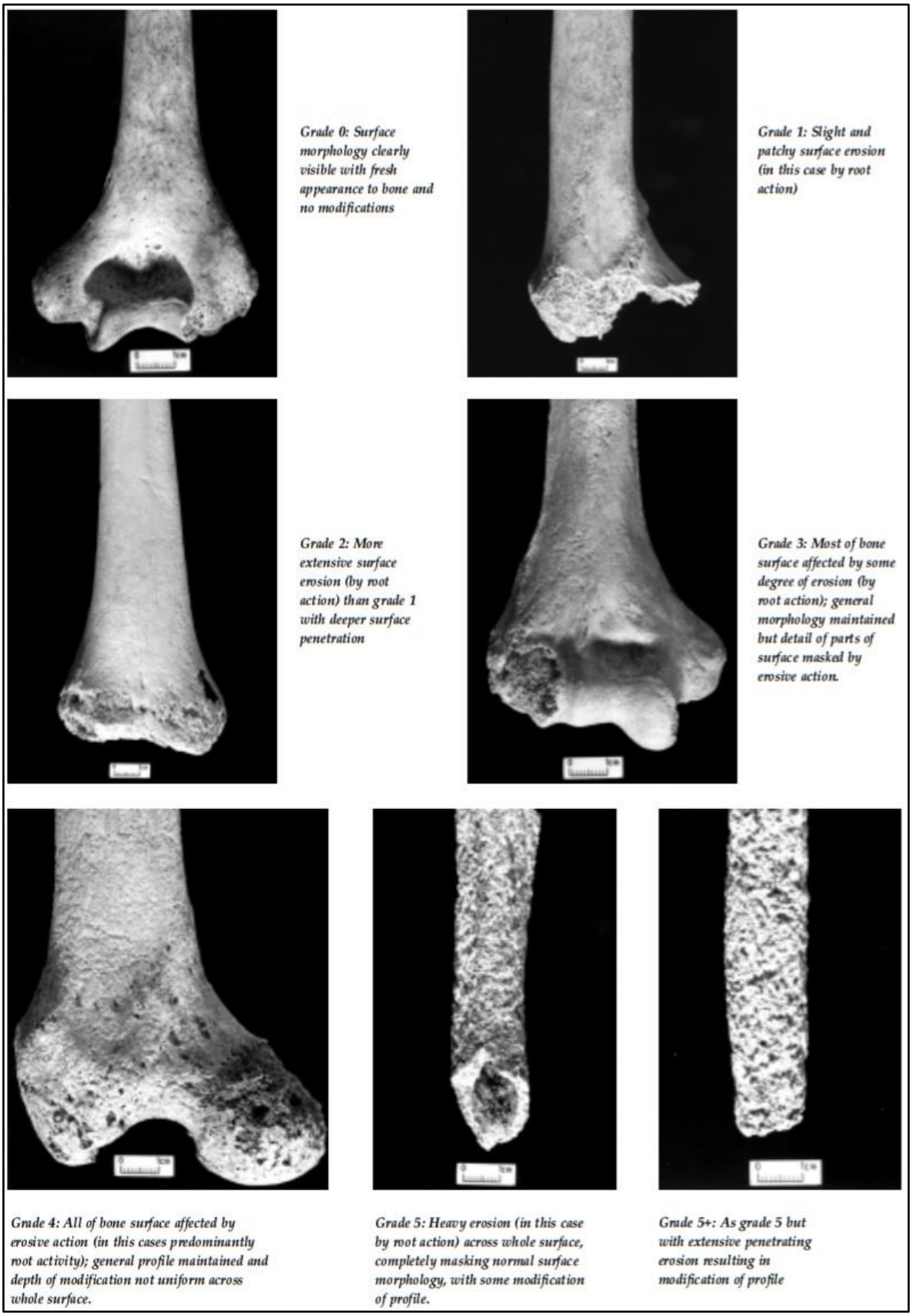


Figure 5.2211 Guidelines for recording cortical surface preservation (McKinley 2004:16).

To account for the various possible etiologies of VSRL, only diffuse woven bone and porous remodelling lamellar bone were considered to be indicative of an LRI and were recorded

here (Davies-Barrett et al. 2019). Osteoclastic reactions (while sometimes caused by tuberculosis, actinomycosis, and pneumonia) (Madeo et al. 2013) can occur as part of osteomyelitis or cancer (Ragsdale et al. 2018). Thus, to eliminate false positives, this project excluded lytic VSRL. Only remodelled lamellar lesions that were distinctly separate from the natural cortical surface were recorded, to not misdiagnose highly remodeled lamellar bone as infectious in origin (Davies-Barrett et al. 2019:534). Not all sources of error could be excluded, since diffuse woven bone and porous remodelling lamellar bone can be indicative of infection and of trauma or a tumor (Waldron 2009:117; Davies-Barrett et al. 2019:534). This was considered in the analysis. Table 5.6, from Matos and Santos (2006:192), describes the appearance of pathological woven and lamellar bone and was employed by Davies-Barrett *et al.* (2019)—and this project—to describe the appearance of lesions.

Table 5.6 The macroscopic appearance of woven and lamellar bone (format adapted from Matos and Santos 2006:192).

Characteristics	Woven Bone	Lamellar Bone
Surface	• Heterogeneous unstructured appearance. • Thin, scalloped, and irregular.	• Homogenous structured appearance. • Thick, compact, and dense.
Porosity	• Intense porosity, with pores usually small in diameter. • Many sinus vascular grooves and channels.	• Sparse or nonexistent porosity. • Pores usually large in diameter and scattered.
Limits and cortical integration	• Limits well-defined. • Irregular margins. • Cortical integration non-existent. • Detachable appearance.	• Regular limits but difficult to differentiate from original cortex of rib. • High cortical integration.
Remodelling	• Few signs of remodelling. • No particular arrangement of fibrous components.	• Remodeling evident. • Fibrous structures parallel oriented to anterior-posterior axis of rib.

Figure 5.23 The visceral surface of the shaft of an upper right rib (CS127) covered in diffuse woven bone (indicated by arrow).

Figure 5.24 The visceral surfaces of the head, angle, and shaft of three lower left ribs (CS928) covered in porous remodelled lamellar bone (indicated by arrows).

For every individual, the fragmentation score for the rib cage (0–3) and the average cortical preservation score (grade 0–5+) were recorded; as well as the number of present necks, angles, and shafts; and upper, upper-middle, lower-middle, and lower ribs by side. If there was diffuse woven bone and porous remodelling lamellar bone present, then the number of necks, angles, and shafts; and upper, upper-middle, lower-middle, and lower ribs by side with lesions were recorded along with the type of lesion(s) present (diffuse woven bone,

porous remodelling lamellar bone, or both). To pool this as binary data (LRI present or absent) at the level of the individual, LRI was considered absent if an individual had no diffuse woven bone or porous remodelling lamellar bone on any of their ribs; if an individual had one or more of these lesions, then LRI was recorded as present for that individual.

Differential diagnosis of the recorded VSRL for every individual was beyond the scope of this research. The presence of VSRL were intended to inform a discussion of the broad environmental risk factors affecting the samples to, in turn, inform the interpretation of the presence of mastoiditis within the samples. Thus, differential diagnosis was tangential to the aims of this project. Rather, this methodology was employed to improve comparability between the sites analysed here and those of other researchers.

The methodology developed by Davies-Barrett *et al.* (2019) is rigorous. It addressed the problems inherent in the palaeopathological study of VSRL and created a data set with a large sample size that reflected the true prevalence of lesions indicative of infection as accurately as possible. The data produced allowed for detailed discussion of the prevalence of LRI. The side, position, and seriation of the ribs were analysed when discussing the diagnosis and pathophysiology of LRI; and the true and crude prevalence of LRI in the population was discussed at the level of the individual.

5.5 Statistical Analysis

This final section refers to the treatment and analysis of data in this project. It outlines the ways in which data were organized; defines the statistical questions asked of the data; and presents and explains the reasoning behind the use of tests for the equality of proportions, Chi-squared tests for co-occurrence, general liner model (GLM) construction, and stepwise logistic regression analyses. Data were recorded as both categorical and continuous variables (see Table 5.7). For every test, the significance group and standard value were set at 95% and five percent (or a=0.05), respectively. A small standard value reduces the chances of committing a Type 1 error[23] and five percent is the statistically accepted value to use when assessing significance (Crawley 2013; Mendenhall et al. 2001:343–5).

To address the objectives of the project, the following statistical questions were asked of the data:

[23] A Type 1 error refers to the rejection of a true null hypothesis (Crawley 2013; Mendenhall et al. 2001:343–5).

1. What was the prevalence and distribution of mastoiditis, maxillary sinusitis, and LRI in the populations?
2. Was the prevalence of mastoiditis, maxillary sinusitis, and LRI different across populations and/or population sub-groups?
3. Did mastoiditis, maxillary sinusitis, and LRI co-occur and, if so, in what population sub-groups?
4. Which population sub-groups were predictive of mastoiditis, maxillary sinusitis, and LRI?

Table 5.7 The types and functions of the variables recorded in the dataframe.

Types of Variables	Recorded Variables
Categorical	• The age, biological sex, and cemetery association of each individual • The type of cellularity in the mastoid processes • The unilateral and bilateral nature of the cellularity in the mastoid processes • The type of bony indicator of sinusitis observed in the maxillary sinuses • The fragmentation and cortical preservation scores for the ribs • The type of VSRL • The presence or absence of mastoiditis, maxillary sinusitis, or LRI (binary)
Continuous	• The total number of observable rib necks, angles, and shafts per side • The total number of observable upper, upper-middle, lower-middle, and lower ribs per side • The number of observable rib necks, angles, and shafts per side with lesions • The number of observable upper, upper-middle, lower-middle, and lower ribs per side with lesions

To address the statistical questions, the open-source statistical software R was used (version 3.3.2: www.r-project.org). R is a software environment that enables users to program statistical tests and access pre-programmed statistical packages (The R Foundation 2021). It provided the flexibility that was needed to explore the data in various ways and the power

necessary to perform complex tests using a large dataframe. Both these features were integral to this project.

The first statistical question was addressed by observing the frequency and proportion of mastoiditis, maxillary sinusitis, and LRI in the sample and in the groups that make-up the populations (e.g., age group or burial type). The second statistical question was addressed by analysing trends in the prevalence of each infection; and by performing a series of tests for the equality of proportions to test if there was any similarity between the proportion of individuals with mastoiditis, maxillary sinusitis, and LRI and if this similarity could be explained by chance alone. These tests required binary data, like the presence/absence data recorded here.

Addressing the third statistical question further defined the relationships amongst the infections, the populations, and the groups to characterize the etiology of mastoiditis. Here, Chi-squared tests for co-occurrence were undertaken to test if mastoiditis, maxillary sinusitis, and LRI co-occurred any more than was expected through chance alone. These tests allowed the frequency of two infections to be tested within the population or within an individual group while controlling for the other groups (Crawley 2013; Mendenhall et al. 2001:627–61).

To address the fourth statistical question, the relationship between the sub-groups and the presence of mastoiditis, maxillary sinusitis, and LRI was modeled using general liner models (GLM). Since the response variables (presence/absence of infection) used were binary and the explanatory variables (e.g., age group) were categorical, the classic tests for outliers (boxplots and Cleveland dotplots), normalcy (QQ plots and histograms), and homogeneity (conditional boxplots and multi-panel boxplots) could not be performed (Zuur et al. 2010). GLMs are robust for categorical explanatory variable, non-homogeneity, and non-normality; thus, GLMs were used to model these relationships (Crawley 2013; Mendenhall et al. 2001:581–24).

The relationships between the sub-groups and mastoiditis, maxillary sinusitis, and LRI were further defined by stepwise logistic regression analysis to test if every level (e.g., dying at a young age) of every group (e.g., age) was predictive of infection or if specific levels and/or specific groups were most predictive of infection. The pre-programed function step was used in R to automatically perform the series of tests necessary for a stepwise regression, removing human error and accounting for all the variance in the data.

The individuals included in this project are dead and, therefore, experimental controls could not be placed on them. The only tool available to control for/eliminate background variance in the data was statistics. Thus, statistical power was increased by collecting and testing a large sample, and background variance was controlled/eliminated by performing

different types of tests using the entire population and groups to account and control for all the ways in which the explanatory variables effected the response variables.

5.6 Conclusions

The methods described in this chapter were designed to address the objectives of this project and achieve its aim. The methods for the imaging and diagnosis of mastoiditis were grounded in modern clinical practices, non-destructive, and more accessible than CT scanning. The diagnosis and analysis of maxillary sinusitis and LRI, along side mastoiditis, were selected to expand our understanding of the epidemiology and etiology of mastoiditis and its impact on the lifeways of those living with respiratory-related disease in the broader context of public health over time in England's north-east, in line with the aim of the project. The data obtained from the application of the methods are explored in the next chapter.

Chapter 6
Results

6.1 Introduction

This chapter presents the prevalence of residual childhood and adult mastoiditis (residual CM and AM), maxillary sinusitis (MS), and lower respiratory infection (LRI) in Black Gate and Coronation Street adults (18+ years). Prevalences are calculated in two ways, as true prevalence rates (TPR) and crude prevalence rates (CPR) (Table 6.1), and presented, first, by population, and second, by a series of demographic and osteological sub-groups (e.g., within age groups). When discussing prevalence by population, true prevalence refers to number of lesions as a proportion of observable anatomical structures and crude prevalence refers to individuals presenting pathologies as a proportion of total individuals. Crude prevalence allows for general discussions of trends amongst individuals in the populations. True prevalence compensates for there being unequal numbers of structures or individuals observable for each lesion type or pathology, respectively, and facilitates comparisons within and between populations by controlling for these inequalities.

Table 6.110 Summary of the terms used in this project to describe prevalence rates, with definitions and examples.

Prevalence	Definitions	Examples
Crude	The proportion of individuals in a population with a pathology by the number of individuals observable for that pathology in that population.	The number of individuals in Black Gate with childhood mastoiditis / the total number of Black Gate individuals observable for childhood mastoiditis.
True	The proportion of structures with a bony lesion by the number of observable structures in that population.	The number of left mastoid processes from Black Gate with a primary hypocellular bone structure / the total number of observable left mastoid processes from Black Gate.

This chapter also presents the results of the statistical analyses. Where appropriate, the results of the tests for the equality of proportions are discussed together with the prevalence of mastoiditis, maxillary sinusitis, and LRI by population. The co-occurrence of the pathologies within the populations and the comparative prevalence of the pathologies between the populations are then discussed alongside the results of the Chi-squared tests and the remaining tests for the equality of proportions, respectively. Finally, the multivariate models are presented.

6.2 Mastoiditis Prevalence

The results from the X-ray and subsequent diagnosis of individual mastoid process health are outlined here. The total prevalence of residual CM or AM amongst those observable for each were used when referring to the prevalence of residual CM and AM separately. When discussing the prevalence of residual CM and AM within a population, residual CM, AM, and 'mix of residual CM and AM' were used to not count individuals with both types of mastoiditis twice.

The null hypothesis being tested by the tests for the equality of proportions was that there was no similarity among the proportions of individuals with mastoiditis, MS, or LRI and that any similarity between these proportions could be explained by chance alone. The results of these tests are discussed throughout the following sections.

Black Gate

In Black Gate, there were 174 individuals (or 66.2% of the population) that fit the criteria for observation and were X-rayed. There was a higher prevalence of AM (43.1%) than residual CM (28.2%); however, both residual CM and AM co-occurred in 16.1% of the population (28 individuals). Only 12.6% of the population had neither residual CM nor AM (Table 6.3). Overall, 77 (44.3%) and 103 (59.2%) individuals presented some evidence of residual CM and AM, respectively (Table 6.2).

Table 6.2 The crude prevalence rate of Black Gate individuals with residual childhood and adult mastoiditis. Never infected refers to an individual with no evidence of mastoiditis (hypercellular, or normal, mastoid air cells).

Diagnosis	Residual Childhood Mastoiditis		Adult Mastoiditis	
	Number Affected	Crude Prevalence	Number Affected	Crude Prevalence
Never Infected	97	55.70%	71	40.80%
Mastoiditis	77	44.3%	103	59.2%
Total	174	100.0%	174	100.0%

Table 6.3 The crude prevalence rate of Black Gate individuals with residual childhood and/or adult mastoiditis diagnosis. Never infected refers to an individual with no evidence of mastoiditis (hypercellular, or normal, mastoid air cells).

Diagnosis	Number Affected	Crude Prevalence
Never Infected	22	12.6%
Only Residual Childhood Mastoiditis	49	28.2%
Only Adult Mastoiditis	75	43.1%
Mix of Residual Childhood and Adult Mastoiditis	28	16.1%
Total	174	100.0%

The proportion of observable mastoids from the left (149, 56.7%) and right (154, 58.6%) sides were close to equal. Tables 6.4–6.6 present the true prevalence of each air cell type and diagnosis by observable left and right side within the population, respectively. The distribution of air cell types and diagnoses between the left and the right mastoid processes revealed no pattern (Table 6.6). There was a slightly greater proportion of residual CM and AM on the left

(38.9% and 51.1%, respectively) compared to the right (32.5% and 45.0%, respectively); however, this difference was not significant (p=0.241 and 0.224, respectively).

Table 6.4 The true prevalence rate mastoid processes with each air cell type in the Black Gate population. LSH refers to Lytic and Secondary Hypocellularity.

Air Cell Type	Left Mastoid Process		Right Mastoid Process	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Hypercellular	24	16.1%	24	15.6%
Primary Hypocellularity	58	38.9%	50	32.5%
Secondary Hypocellularity	42	28.2%	52	33.8%
LSH	25	16.8%	28	18.2%
Total	149	100.0%	154	100.0%

Table 6.5 The true prevalence rate of mastoid processes with residual childhood or adult mastoiditis diagnosis in the Black Gate population. Never infected refers to an individual with no evidence of mastoiditis (hypercellular, or normal, mastoid air cells).

Diagnosis	Left Mastoid		Right Mastoid	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Never Infected	24	16.1%	24	15.6%
Residual Childhood Mastoiditis	58	38.9%	50	32.5%
Adult Mastoiditis	67	45.0%	80	51.9%
Total	149	100.0%	154	100.0%

Table 6.6 The results of the binomial tests for the equality of proportions comparing the true prevalence of left/right residual childhood and adult mastoiditis in Black Gate mastoid processes. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis	Adult Mastoiditis
	Left/Right	Left/Right
p-value	0.241	0.224
df	1	1
X^2	1.3771	1.4778

Table 6.7 shows the proportion of individuals with unilateral or bilateral lesions. Table 6.8 shows the results of the binomial test for the equality of proportions comparing the prevalence of unilateral and bilateral mastoiditis. There were nearly equal proportions of individuals with residual CM (45, 17.1%) and AM (55, 20.9%) who were observable for uni-/bilaterality (Table 6.7). The sample of individuals who could be studied for laterality was quite small, so overarching hypotheses could not be made. However, trends were noted. Both residual CM and AM were significantly more likely to be bilateral than unilateral (Table 6.8). While two thirds of childhood lesions were bilateral, adult lesions were even more likely to occur in both mastoids (81.8%).

Table 6.7 The crude prevalence rate of Black Gate individuals with uni-/bilateral residual childhood/adult mastoiditis.

Diagnosis	Residual Childhood Mastoiditis		Adult Mastoiditis	
	Number Affected	Crude Prevalence	Number Affected	Crude Prevalence
Unilateral	15	33.3%	10	18.2%
Bilateral	30	66.7%	45	81.8%
Total	45	100.0%	55	100.0%

Table 6.8 The results of the binomial tests for the equality of proportions comparing the crude prevalence of uni-/bilateral residual childhood and adult mastoiditis in Black Gate individuals. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis	Adult Mastoiditis
	Uni-/Bilateral	Uni-/Bilateral
p-value	0.002	2.49^{-11}
df	1	1
X^2	10	44.545

Tables 6.9–6.16 present the true prevalence of individuals diagnosed with residual childhood (77 individuals, 29.3% of the population) and adult (103 individuals, 39.2% of the population) mastoiditis divided by biological sex, age group, grave type, and body position; and the results of the associated tests for equality of proportions. Adult mastoiditis was more prevalent than residual childhood mastoiditis in adults of both biological sexes (Figure 6.1, Table 6.9). Neither biological sex was more likely than the other to be infected with either condition (Tables 6.9–6.10). In general, the true prevalence of AM increased with age at death (Figure

6.2, Table 6.11). While no significant linear relationship existed between the proportion of mastoids infected and age at death, the relationship between AM and age at death approached significance (p=0.064; Tables 6.12–6.13). There were more individuals who had experienced residual CM in the young and prime age groups, suggesting a possible association between residual CM and premature mortality. Amongst the grave types and body positions, which can be indicative of an individual's projected social status, no statistical patterns were observable for those with mastoiditis (Tables 6.14–6.16).

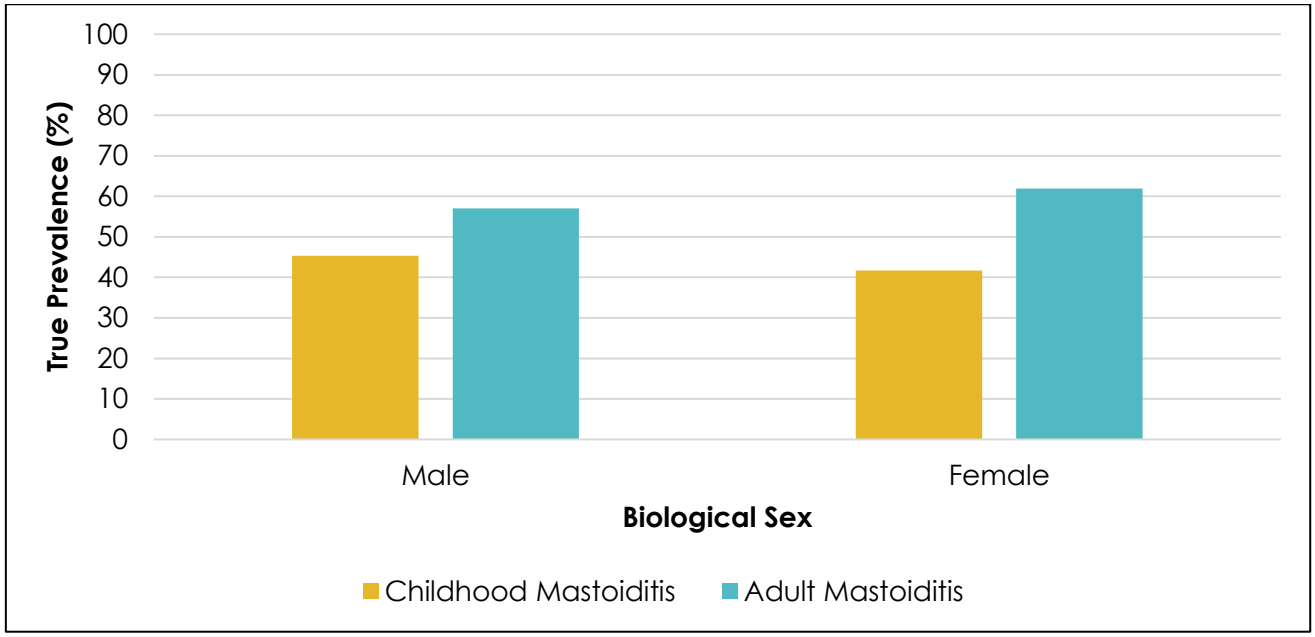


Figure 6.1 The true prevalence rate of Black Gate individuals with childhood and adult mastoiditis by biological sex.

Table 6.9 The true prevalence rate of Black Gate individuals with residual childhood and adult mastoiditis by biological sex. NoA is Number Affected; Prop. Is Proportion; and True Prev. is True Prevalence.

Biological Sex	Residual Childhood Mastoiditis			Adult Mastoiditis		
	NoA	Observable	True Prev.	NoA	Observable	True Prev.
Male	39	86	45.3%	49	86	57.0%
Female	35	84	41.7%	52	84	61.9%
Indeterminate	3	4	75.0%	2	4	50.0%
Total	77	174	44.3%	103	174	59.2%

Table 6.10 The results of the binomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Black Gate males and females. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis	Adult Mastoiditis
	Male/Female	Male/Female
p-value	0.628	0.513
df	1	1
X^2	0.23439	0.42796

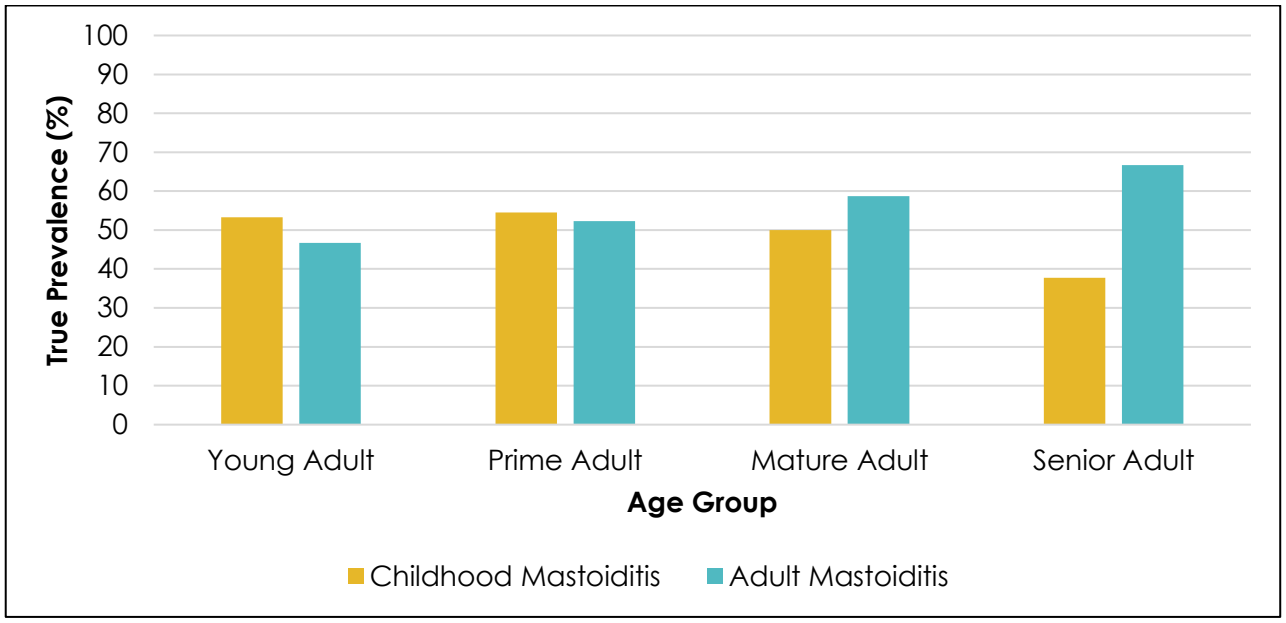


Figure 6.212 The true prevalence rate of Black Gate individuals with childhood and adult mastoiditis by age group.

Table 6.11 The true prevalence rate of Black Gate individuals with residual childhood and adult mastoiditis by age group. NoA is Number Affected; Prop. Is Proportion; and True Prev. is True Prevalence.

Age	Residual Childhood Mastoiditis			Adult Mastoiditis		
	NoA	Observable	True Prev.	NoA	Observable	True Prev.
Adult	0	0	0.0%	0	0	0.0%
Young Adult	8	15	53.3%	7	15	46.7%
Prime Adult	20	44	45.5%	23	44	52.3%
Mature Adult	23	46	50.0%	27	46	58.7%
Senior Adult	26	69	37.7%	46	69	66.7%
Total	77	174	44.3%	103	174	59.2%

Table 6.12 The results of the multinomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Black Gate individuals from each age at death. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis		Adult Mastoiditis	
	Equality of Prop.	Linear Trend	Equality of Prop.	Linear Trend
p-value	0.503	0.233	0.328	0.064
df	3	1	3	1
X-squared	2.3509	1.4221	3.4471	3.4258

Table 6.13 The results of the binomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Black Gate individuals belonging to each age group. Significant p-values are marked in green. df is degrees of freedom.

Data	Output	Y/P	P/M	M/S	Y/M	Y/S	M/S
Residual Childhood Mastoiditis							
	p-value	0.598	0.666	0.191	1	0.407	0.533
	df	1	1	1	1	1	1
	X^2	0.27848	0.18623	1.7128	5.27E-32	0.68747	0.38911
Adult Mastoiditis							
	p-value	0.708	0.540	0.385	0.606	0.246	0.183
	df	1	1	1	1	1	1
	X^2	0.14067	0.37574	0.75641	0.26543	1.3448	1.7749

Table 6.14 The true prevalence rate of Black Gate individuals with residual childhood and adult mastoiditis by grave type. NoA is Number Affected; Prop. Is Proportion; Ob. Is Observable; and True Prev. is True Prevalence. One individual who was recorded as being from a probable earmuff grave (recorded as "Earmuffs?") was recorded in this project as being from an earmuff grave.

Grave Type	Residual Childhood Mastoiditis			Adult Mastoiditis		
	NoA	Observable	True Prev.	NoA	Observable	True Prev.
Plain	58	125	46.4%	70	125	56.0%
Earmuffs	2	4	50.0%	2	4	50.0%
Pillow Stone	0	2	0.0%	1	2	50.0%
Head Box	0	3	0.0%	3	3	100.0%
Coffin	9	23	39.1%	16	23	69.6%
Chest	0	0	0.0%	0	0	0.0%
Rubble Cist	1	2	50.0%	2	2	100.0%
Cist	2	5	40.0%	2	5	40.0%
Unidentified	5	10	50.0%	7	10	70.0%
Total	77	174	44.3%	103	174	59.2%

Table 6.15 The results of the binomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Black Gate individuals belonging to each grave type. Significant p-values are marked in green. df is degrees of freedom. C is coffin. P is plain. E is elaborate (chest, rubble cist, and cist). EV is elaborate variation (earmuffs, pillow stone, and head box). E&EV is elaborate and elaborate variation.

Data	Output	C/P	C/E&EV	P/E&EV	C/E	C/EV	P/E	P/EV	E/EV
Residual Childhood Mastoiditis									
	p-value	0.520	0.614	0.251	0.860	0.365	0.855	0.159	0.377
	df	1	1	1	1	1	1	1	1
	X^2	0.41434	0.25465	1.3172	0.031056	0.81979	0.033473	1.9848	0.78038
Adult Mastoiditis									
	p-value	0.226	0.645	0.621	0.542	0.874	0.953	0.533	0.696
	df	1	1	1	1	1	1	1	1
	X^2	1.4685	0.21196	0.24415	0.37267	0.025296	0.003515	0.38911	0.15238

Table 6.16 The true prevalence rate of Black Gate individuals with childhood and adult mastoiditis by body position. NoA is Number Affected; Prop. Is Proportion; and True Prev. is True Prevalence.

Body Position	Residual Childhood Mastoiditis			Adult Mastoiditis		
	NoA	Observable	True Prev.	NoA	Observable	True Prev.
Supine	50	112	44.6%	65	112	58.0%
Flexed	2	3	66.7%	1	3	33.3%
Left Side	7	10	70.0%	5	10	50.0%
Right Side	13	38	34.2%	27	38	71.1%
Prone	2	6	33.3%	3	6	50.0%
Unidentified	3	5	60.0%	2	5	40.0%
Total	77	174	44.3%	103	174	59.2%

Coronation Street

There were 78 individuals (or 63.4% of the population) that fit the criteria for observation and were X-rayed (Tables 6.17–6.18). Of those observable for each infection type, 39 (50.0%) and 37 (47.4%) individuals presented with some evidence of residual CM and AM, respectively (Tables 6.17). Both residual CM and AM co-occurred in 19.2% of the population (15 individuals). Only 21.8% of the population had neither residual CM nor AM (Table 6.18).

Table 6.17 The crude prevalence rate of Coronation Street individuals with residual childhood and adult mastoiditis. Never infected refers to an individual with no evidence of mastoiditis (hypercellular, or normal, mastoid air cells).

Diagnosis	Residual Childhood Mastoiditis		Adult Mastoiditis	
	Number Affected	Crude Prevalence	Number Affected	Crude Prevalence
Never Infected	39	50.0%	41	52.6%
Mastoiditis	39	50.0%	37	47.4%
Total	78	100.0%	78	100.0%

Table 6.18 The crude prevalence rate of Coronation Street individuals with residual childhood and/or adult mastoiditis diagnosis. Never infected refers to an individual with no evidence of mastoiditis (hypercellular, or normal, mastoid air cells).

Diagnosis	Number Affected	Crude Prevalence
Never Infected	17	21.8%
Only Residual Childhood Mastoiditis	24	30.8%
Only Adult Mastoiditis	22	28.2%
Mix of Residual Childhood and Adult Mastoiditis	15	19.2%
Observable	78	63.4%

The proportion of observable mastoids from the left (74, 60.1%) and right (78, 63.4%) sides was close to equal. Tables 6.19–6.20 present the true prevalence of each air cell type and diagnosis by observable left and right side within the population, respectively. The distribution of air cell types and diagnoses between the left and right mastoid processes were nearly even (see Table 6.21). As in the Black Gate population, the distribution of lesions indicative of AM showed that secondary hypocellularity was slightly more common than lytic and secondary hypocellular (LSH) MAC on the left and right; and that no significant difference existed between the proportion of either pathology on the left or right (p=0.825 for residual CM and p=0.930 for AM).

Table 6.19 The true prevalence rate of mastoid processes with each air cell type in the Coronation Street population. LSH refers to Lytic and Secondary Hypocellularity.

Air Cell Type	Left Mastoid Process		Right Mastoid Process	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Hypercellular	17	23.0%	16	20.5%
Primary Hypocellularity	30	40.5%	33	42.3%
Secondary Hypocellularity	15	20.3%	16	20.5%
LSH	12	16.2%	13	16.7%
Total	74	100.0%	78	100.0%

Table 6.20 The true prevalence rate of mastoid processes with residual childhood or adult mastoiditis diagnosis in the Coronation Street population.

Diagnosis	Left Mastoid Process		Right Mastoid Process	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Healthy	17	22.9%	16	20.5%
Residual Childhood Mastoiditis	30	40.5%	33	42.3%
Adult Mastoiditis	27	36.5%	29	37.2%
Total	74	100.0%	78	100.0%

Table 6.21 The results of the binomial tests for the equality of proportions comparing the true prevalence of left/right residual childhood and adult mastoiditis in Coronation Street mastoid processes. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis	Adult Mastoiditis
	Left/Right	Left/Right
p-value	0.825	0.930
df	1	1
X^2	0.048864	0.007838

Table 6.22 shows the proportion of individuals with unilateral or bilateral lesions. Table 6.23 shows the results of the binomial tests for the equality of proportions comparing the prevalence of unilateral and bilateral mastoiditis. There were nearly equal proportions of individuals with residual CM (28, 22.8%) and AM (26, 21.1%) who were observable for uni-/bilaterality (Table 6.22). As with the Black Gate population, mastoiditis was more often bilateral than unilateral (Table 6.23). This difference was highly significant for both residual CM and AM (1.90^{-5} and 1.03^{-4}, respectively).

Table 6.22 The crude prevalence rate of Coronation Street individuals with uni-/bilateral residual childhood/adult mastoiditis.

Diagnosis	Residual Childhood Mastoiditis		Adult Mastoiditis	
	Number Affected	Crude Prevalence	Number Affected	Crude Prevalence
Unilateral	6	21.4%	6	23.1%
Bilateral	22	78.6%	20	76.9%
Total	28	100.0%	26	100.0%

Table 6.23 The results of the binomial tests for the equality of proportions comparing the crude prevalence of uni-/bilateral residual childhood and adult mastoiditis in Coronation Street individuals. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis	Adult Mastoiditis
	Uni-/Bilateral	Uni-/Bilateral
p-value	1.90^{-5}	1.03^{-4}
df	1	1
X^2	18.286	15.077

Tables 6.24–6.28 present the true prevalence of residual childhood (39 individuals, 31.7% of the population) and adult mastoiditis (37 individuals, 30.1% of the population) by biological sex and age group, and the results of the associated tests for equality of proportions. This study had no access to data regarding body position or grave type for the Coronation Street population.

In this population, there was insignificantly more residual CM in males than females (p=0.345) and more AM in females than males, but this was not statistically significant (p=0.672) (Figure 6.3; Tables 6.24–6.25). While AM increased with age at death (though not significantly), as in the Black Gate population, residual CM peaked in individuals who died in the mature age group (Figure 6.4; Tables 6.26–6.28). However, the proportion of residual CM in mature individuals was not significantly different to that in individuals from other age groups.

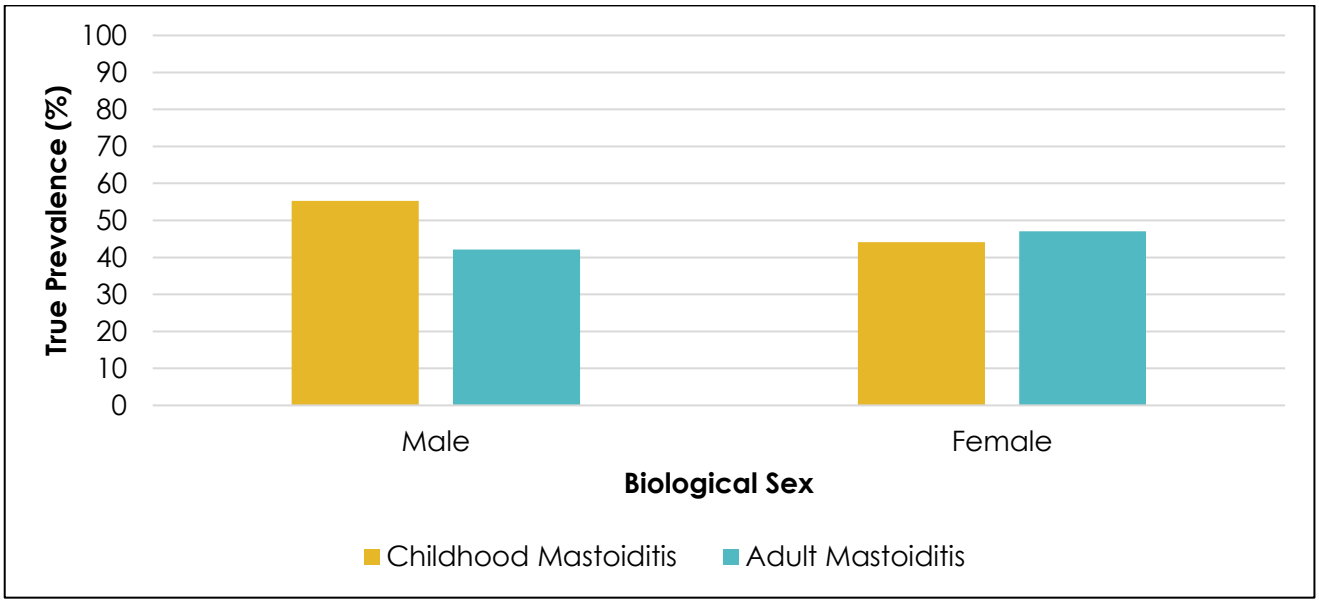


Figure 6.313 The true prevalence rate of Coronation Street individuals with childhood and adult mastoiditis by biological sex.

Table 6.24 The true prevalence rate of Coronation Street individuals with residual childhood and adult mastoiditis by biological sex. NoA is Number Affected; Prop. Is Proportion; and True Prev. is True Prevalence.

Biological Sex	Residual Childhood Mastoiditis			Adult Mastoiditis		
	NoA	Observable	True Prev.	NoA	Observable	True Prev.
Male	21	38	55.3%	16	38	42.1%
Female	15	34	44.1%	16	34	47.1%
Total	36	72	50.0%	32	72	44.4%

Table 6.25 The results of the binomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Coronation Street males and females. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis	Adult Mastoiditis
	Male/Female	Male/Female
p-value	0.345	0.673
df	1	1
X^2	0.89164	0.17833

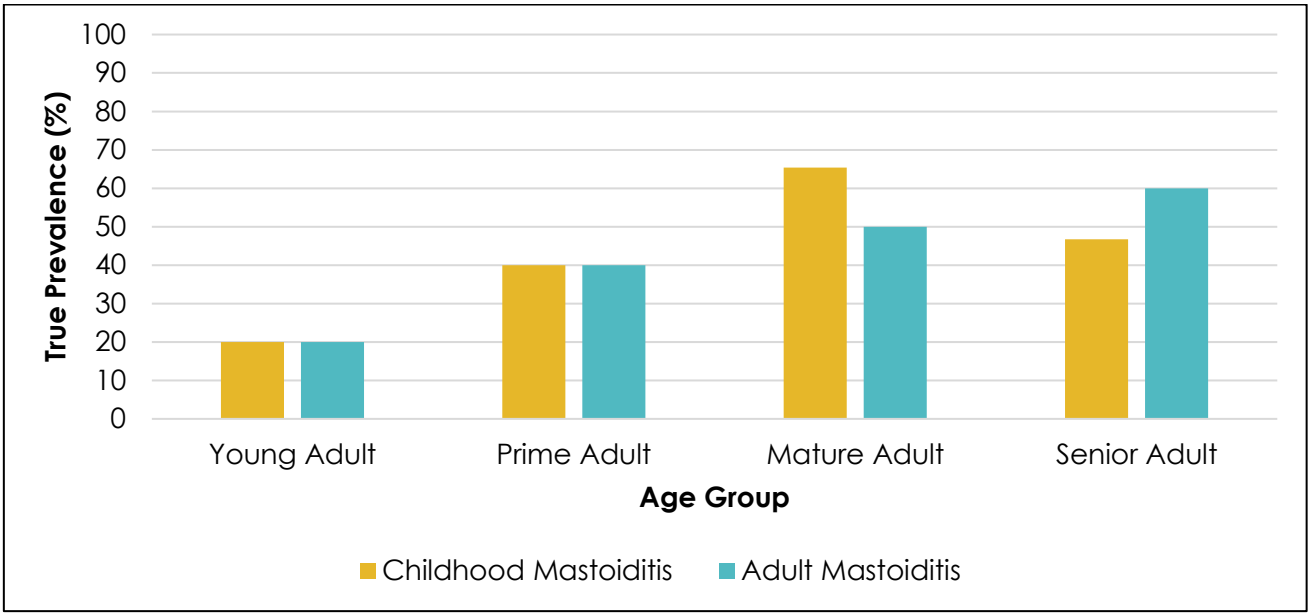


Figure 6.4 The true prevalence rate of Coronation Street individuals with childhood and adult mastoiditis by biological sex.

Table 6.26 The true prevalence rate of Coronation Street individuals with residual childhood and adult mastoiditis by age group. NoA is Number Affected; Prop. Is Proportion; and True Prev. is True Prevalence.

Age	Residual Childhood Mastoiditis			Adult Mastoiditis		
	NoA	Observable	True Prev.	NoA	Observable	True Prev.
Adult	8	17	47.1%	8	17	47.1%
Young Adult	1	5	20.0%	1	5	20.0%
Prime Adult	6	15	40.0%	6	15	40.0%
Mature Adult	17	26	65.4%	13	26	50.0%
Senior Adult	7	15	46.7%	9	15	60.0%
Total	39	78	50.0%	37	78	47.4%

Table 6.27 The results of the binomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Coronation Street individuals from each age group. Significant p-values are marked in green. df is degrees of freedom.

Data	Output	Y/P	P/M	M/S	Y/M	Y/S	M/S
Residual Childhood Mastoiditis							
	p-value	0.417	0.115	0.241	0.165	0.598	1
	df	1	1	1	1	1	1
	X^2	0.65934	2.4888	1.3731	1.9283	0.27778	6.69^{-33}
Adult Mastoiditis							
	p-value	0.417	0.536	0.536	0.457	0.302	0.465
	df	1	1	1	1	1	1
	X^2	0.65934	0.38254	0.38254	0.55332	1.0667	0.53333

Table 6.28 The results of the multinomial tests for the equality of proportions comparing the true prevalence of residual childhood and adult mastoiditis in Coronation Street individuals from each age at death. Significant p-values are marked in green. df is degrees of freedom.

Output	Residual Childhood Mastoiditis		Adult Mastoiditis	
	Equality of Prop.	**Linear Trend**	**Equality of Prop.**	**Linear Trend**
p-value	0.178	0.240	0.414	0.098
df	3	1	3	1
X-squared	4.9131	1.3786	2.8594	2.7452

6.3 Maxillary Sinusitis Prevalence

The results from the visual, macroscopic analysis of maxillary sinus health are outlined here. True prevalence is calculated as the number of affected sinuses as a proportion of observable sinuses and presented by the type of new bone formation according to the classification method described by Boocock *et al.* (1995). Each bone type, save for never infected, was diagnosed as evidence of chronic sinusitis.

Black Gate

In the Black Gate population, there were 195 maxillary sinuses from 126 individuals (or 47.9% of the population) that fit the criteria for observation. Under half of the population were diagnosed with MS (51, 40.5%; Table 6.29). The proportion of observable maxillary sinuses from the left (96, 76.2%) and right (99, 78.6%) sides was nearly equal.

Table 6.29 The crude prevalence rate of Black Gate individuals with maxillary sinusitis. Never infected refers to an individual with no evidence of maxillary sinusitis.

Diagnosis	Number Affected	Crude Prevalence
Never Infected	75	59.5%
Sinusitis	51	40.5%
Total	126	100.0%

Tables 6.30–6.31 present the true prevalence of each bone type and diagnosis by observable left and right side within the population, respectively. Table 6.30 shows that spicules (22.9% and 24.2%) were markedly the most common form of new bone, followed by remodelled spicules (7.3% and 10.1%), pitting (2.1% and 1.0%), and white pitted bone (1.0% and 1.0%). None of the bone types co-occurred. Lesions were equally common in the left and right sinuses (p=0.657; Table 6.32); and MS was marginally more likely to be bilateral (58.6%) than unilateral (41.4%), though this difference was insignificant (p=0.294; Table 6.33–6.34).

Table 6.30 The true prevalence rate of each bone type in the Black Gate population. Never infected refers to an individual with no evidence of maxillary sinusitis.

Bone Type	Left Maxillary Sinus		Right Maxillary Sinus	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Never Infected	64	66.7%	63	63.6%
Pitting	2	2.1%	1	1.0%
Spicules	22	22.9%	24	24.2%
Remodeled Spicules	7	7.3%	10	10.1%
White Pitted Bone	1	1.0%	1	1.0%
Total	96	100.0%	99	100.0%

Table 6.31 The true prevalence rate of Black Gate individuals with maxillary sinusitis diagnosis. Never infected refers to an individual with no evidence of maxillary sinusitis.

Diagnosis	Left Maxillary Sinus		Right Maxillary Sinus	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Never Infected	64	66.7%	63	63.6%
Sinusitis	32	33.3%	36	36.4%
Total	96	100.0%	99	100.0%

Table 6.32 The results of the binomial tests for the equality of proportions comparing the true prevalence of left/right maxillary sinusitis in Black Gate maxillary sinuses. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis
	Left/Right
p-value	0.657
df	1
X^2	0.19706

Table 6.33 The crude prevalence rate of Black Gate individuals with uni-/bilateral maxillary sinusitis.

Sinusitis	Number Affected	Crude Prevalence
Unilateral	12	41.4%
Bilateral	17	58.6%
Total	29	100.0%

Table 6.34 The results of the binomial tests for the equality of proportions comparing the crude prevalence of uni-/bilateral maxillary sinusitis in Black Gate individuals. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis
	Uni-/Bilateral
p-value	0.294
df	1
X^2	1.1034

Tables 6.35–6.42 present the true prevalence of individuals with maxillary sinusitis by biological sex, age group, grave type, and body position, and the results of the associated tests for the equality of proportions. Slightly more females (45.6%) than males (38.5%) were diagnosed with MS (Figure 6.5, Table 6.35), although this difference was not statistically significant (p=0.424) (Table 6.36). MS decreased linearly with age at death (significantly from 61.5% in Young Adults to 22.9% in Senior Adults; Table 6.37–6.39), with the most significant change occurring between mature and senior age (p=0.027) (Figure 6.6, Table 6.39). No distinction or statistical pattern were present in the distribution of individuals with MS by grave type or body position (Tables 6.40–6.42).

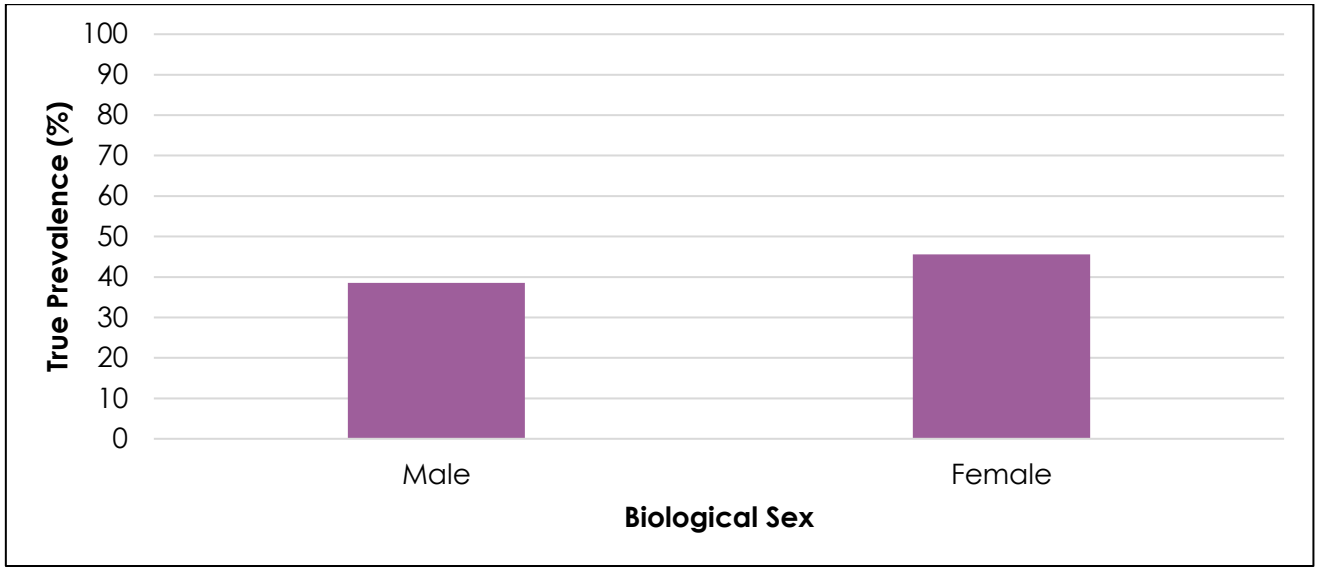


Figure 6.5 The true prevalence rate of Black Gate individuals with maxillary sinusitis by biological sex.

Table 6.35 The true prevalence rate of Black Gate individuals with maxillary sinusitis by biological sex.

Biological Sex	Maxillary Sinusitis		
	Number Affected	Observable	True Prevalence
Male	25	65	38.5%
Female	26	57	45.6%
Unidentified	0	4	0.0%
Total	51	126	100.0%

Table 6.36 The results of the binomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Black Gate males and females. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis
	Male/Female
p-value	0.424
df	1
X^2	0.63861

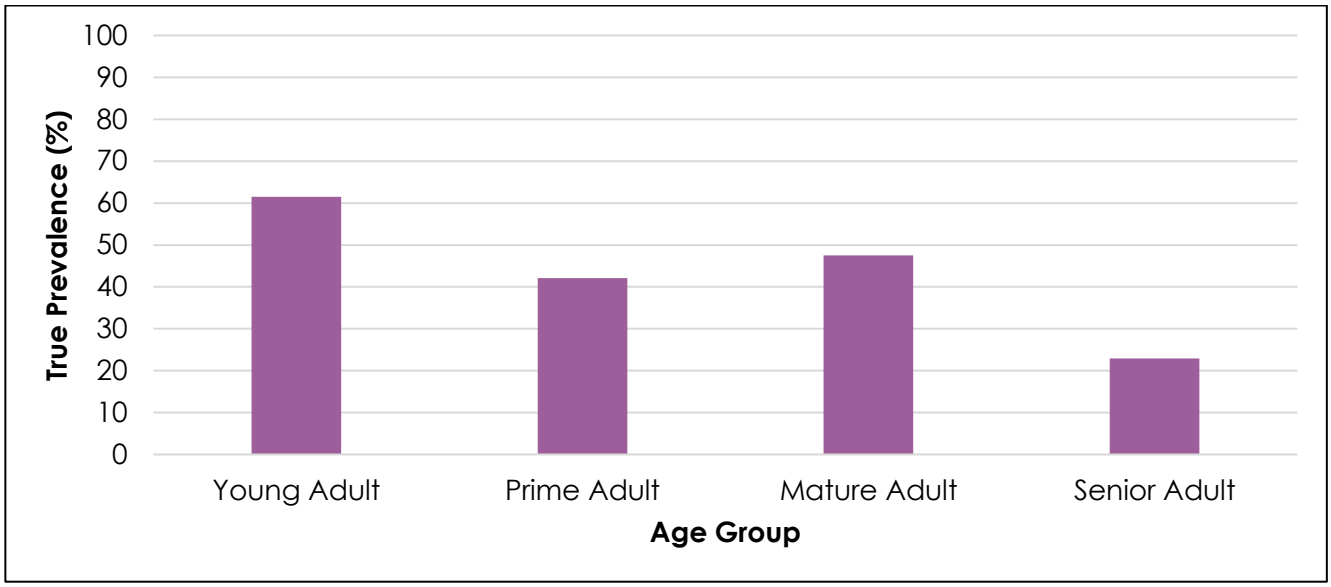


Figure 6.614 The true prevalence rate of Black Gate individuals with maxillary sinusitis by age group.

Table 6.37 The true prevalence rate of Black Gate individuals with maxillary sinusitis by age group.

Age	Maxillary Sinusitis		
	Number Affected	Observable	True Prevalence
Adult	0	0	0.0%
Young Adult	8	13	61.5%
Prime Adult	16	38	42.1%
Mature Adult	19	40	47.5%
Senior Adult	8	35	22.9%
Total	51	126	40.5%

Table 6.38 The results of the multinomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Black Gate individuals from each age group. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis	
	Equality of Proportions	Linear Trend
p-value	0.051	0.022
df	3	1
X-squared	7.7642	5.2741

Table 6.39 The results of the binomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Black Gate individuals from each age group. Significant p-values are marked in green. df is degrees of freedom. Y is Young Adult. P is Prime Adult. M is Mature Adult. S is Senior Adult.

Output	Maxillary Sinusitis					
	Y/P	P/M	M/S	Y/M	Y/S	M/S
p-value	0.226	0.632	0.027	0.575	0.029	0.134
df	1	1	1	1	1	1
X^2	1.4683	0.22927	4.92	0.31394	4.7604	2.2487

Table 6.40 The true prevalence rate of Black Gate individuals with maxillary sinusitis by grave type. One individual who was recorded as being from a probable earmuff grave (recorded as "Earmuffs?") was recorded in this project as being from an earmuff grave.

Grave Type	Maxillary Sinusitis		
	Number Affected	Observable	True Prevalence
Plain	39	95	41.1%
Earmuffs	1	1	100.0%
Pillow Stone	0	2	0.0%
Head Box	0	1	0.0%
Coffin	10	21	47.6%
Chest	0	0	0.0%
Rubble Cist	0	1	0.0%
Cist	0	2	0.0%
Unidentified	1	3	33.3%
Total	51	126	40.5%

Table 6.41 The results of the binomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Black Gate individuals belonging to each grave type. Significant p-values are marked in green. df is degrees of freedom. C is coffin. P is plain. E is elaborate (chest, rubble cist, and cist). EV is elaborate variation (earmuffs, pillow stone, and head box). E&EV is elaborate and elaborate variation.

Output	Maxillary Sinusitis							
	C/P	C/E&EV	P/E&EV	C/E	C/EV	P/E	P/EV	E/EV
p-value	0.581	0.118	0.162	0.118	0.404	0.153	0.522	0.350
df	1	1	1	1	1	1	1	1
X^2	0.30394	2.4456	1.9596	2.449	0.69767	2.0457	0.41077	0.875

Table 6.42 The true prevalence rate of Black Gate individuals with maxillary sinusitis by body position.

Body Position	Maxillary Sinusitis		
	Number Affected	Observable	True Prevalence
Supine	36	85	42.4%
Flexed	0	0	0.0%
Left Side	3	4	75.0%
Right Side	12	28	42.9%
Prone	0	4	0.0%
Unidentified	0	5	0.0%
Total	51	126	40.5%

Coronation Street

In the Coronation Street population, there were 62 maxillary sinuses from 38 individuals (or 30.9% of the population) that fit the criteria for observation (Table 6.43). Over half of the population were diagnosed with MS (24, 63.2%).

Table 6.43 The crude prevalence rate of Coronation Street individuals with maxillary sinusitis. Never infected refers to an individual with no evidence of maxillary sinusitis.

Diagnosis	Number Affected	Crude Prevalence
Never Infected	14	36.8%
Sinusitis	24	63.2%
Total	38	100.0%

The proportion of observable maxillary sinuses from the left (30, 78.9%) and right (32, 84.2%) sides was similar (Tables 6.44–6.45). Tables 6.44 and 6.45 present the true prevalence of each bone type and diagnosis by observable left and right side within the population, respectively. Table 6.44 shows that spicules (50.0% on both sides) were markedly the most common form of new bone, followed by white pitted bone (10.0% and 3.1%), and both pitting and remodeled spicules (0.0% and 3.1%). None of the bone types co-occurred.

MS was equally common in both the left and right sinuses (p=0.96; Table 6.46); and MS was three times more likely to be bilateral (75.0%) than unilateral (25.0%; Table 6.47). The latter difference was highly significant (p=0.013; Table 6.48).

Table 6.44 The true prevalence rate of each air cell type in the Coronation Street population. Never infected refers to an individual with no evidence of maxillary sinusitis.

Bone Type	Left Maxillary Sinus		Right Maxillary Sinus	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Never Infected	12	40.0%	13	26.0%
Pitting	0	0.0%	1	3.1%
Spicules	15	50.0%	16	50.0%
Remodeled Spicules	0	0.0%	1	3.1%
White Pitted Bone	3	10.0%	1	3.1%
Total	30	100.0%	32	100.0%

Table 6.45 The true prevalence rate of Coronation Street individuals with maxillary sinuses diagnosis. Never infected refers to an individual with no evidence of maxillary sinusitis.

Diagnosis	Left Maxillary Sinus		Right Maxillary Sinus	
	Number Affected	True Prevalence	Number Affected	True Prevalence
Never Infected	12	40.0%	13	26.0%
Sinusitis	18	60.0%	19	59.4%
Total	30	100.0%	32	100.0%

Table 6.46 The results of the binomial tests for the equality of proportions comparing the true prevalence of left/right maxillary sinusitis in Coronation Street maxillary sinuses. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis
	Left/Right
p-value	0.96
df	1
X^2	0.002514

Table 6.47 The crude prevalence rate of Coronation Street individuals with uni-/bilateral maxillary sinusitis.

Sinusitis	Number Affected	Crude Prevalence
Unilateral	4	25.0%
Bilateral	12	75.0%
Total	16	100.0%

Table 6.48 The results of the binomial tests for the equality of proportions comparing the crude prevalence of uni-/bilateral maxillary sinusitis in Coronation Street individuals. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis
	Uni-/Bilateral
p-value	0.013
df	1
X^2	6.125

Table 6.49–6.53 present the true prevalence of individuals with maxillary sinusitis by biological sex and age group, and the results of the associated tests for the equality of proportions. Approximately twice as many females (77.3%) as males (38.5%) were diagnosed with MS (Figure 6.7, Table 6.49). This difference was highly significant (p=0.022; Table 6.50). Unlike in the Black Gate population, no linear relationship existed between the prevalence of maxillary sinusitis and age at death (p=0.202) (Figure 6.8, Table 6.52). Maxillary sinusitis peaked in Young Adults (100.0%) and generally decreased as age at death increased, levelling out in Mature and Senior Adults (61.5% and 62.5%, respectively; Table 6.51–6.52); however, these differences in proportion were statistically insignificant (Table 6.53).

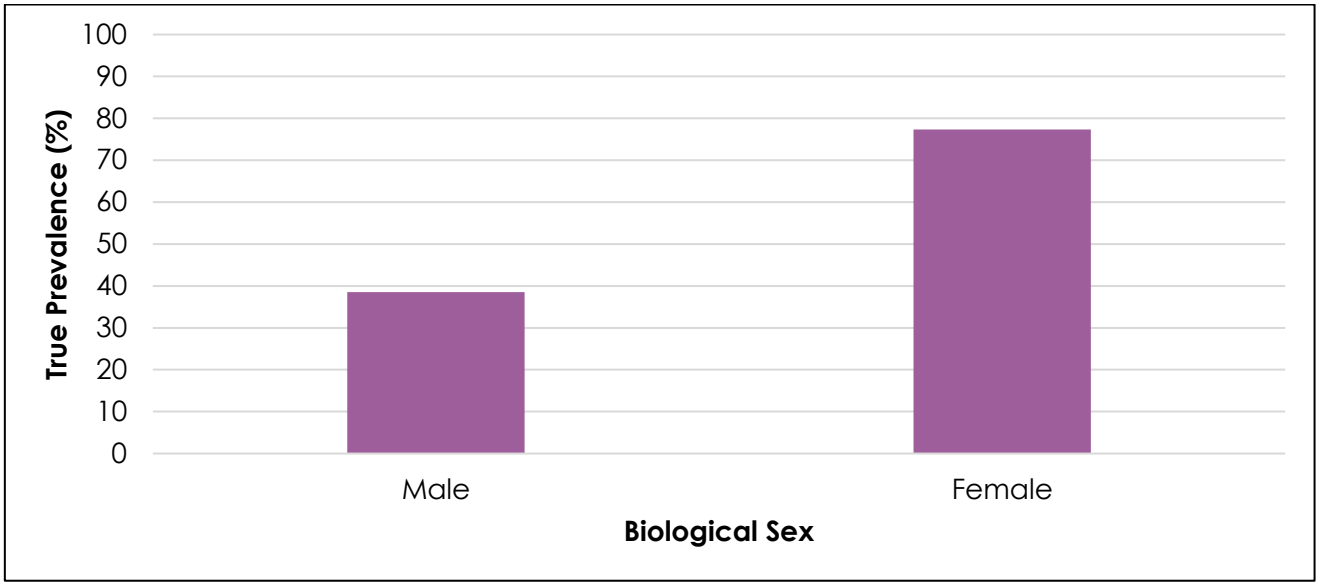


Figure 6.715 The true prevalence rate of Coronation Street individuals with maxillary sinusitis by biological sex.

Table 6.49 The true prevalence rate of Coronation Street individuals with maxillary sinusitis by biological sex.

Biological Sex	Maxillary Sinusitis		
	Number Affected	Observable	True Prevalence
Male	5	13	38.5%
Female	17	22	77.3%
Unidentified	2	3	66.7%
Total	24	38	63.2%

Table 6.50 The results of the binomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Coronation Street males and females. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis
	Male/Female
p-value	0.022
df	1
X^2	5.2721

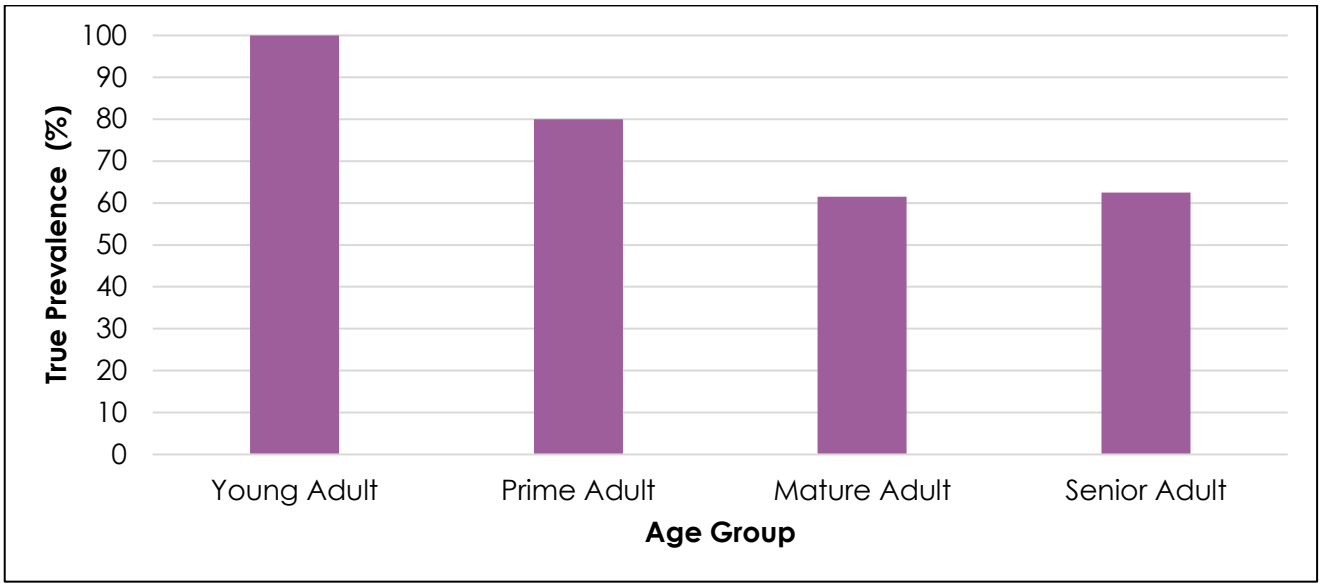


Figure 6.816 The true prevalence rate of Coronation Street individuals with maxillary sinusitis by age group.

Table 6.51 The true prevalence rate of Coronation Street individuals with maxillary sinusitis by age group.

Age	Maxillary Sinusitis		
	Number Affected	Observable	True Prevalence
Adult	4	9	44.4%
Young Adult	3	3	100.0%
Prime Adult	4	5	80.0%
Mature Adult	8	13	61.5%
Senior Adult	5	8	62.5%
Total	24	38	63.2%

Table 6.52 The results of the multinomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Coronation Street individuals from each age group. Significant p-values are marked in green. df is degrees of freedom.

Output	Maxillary Sinusitis	
	Equality of Proportions	Linear Trend
p-value	0.547	0.202
df	3	1
X-squared	2.1257	1.6257

Table 6.53 The results of the binomial tests for the equality of proportions comparing the true prevalence of maxillary sinusitis in Coronation Street individuals from each age group. Significant p-values are marked in green. df is degrees of freedom. Y is Young Adult. P is Prime Adult. M is Mature Adult. S is Senior Adult.

Output	Maxillary Sinusitis					
	Y/P	P/M	M/S	Y/M	Y/S	M/S
p-value	0.408	0.457	0.965	0.546	0.629	0.962
df	1	1	1	1	1	1
X^2	0.68571	0.55385	0.001942	0.3655	0.23394	0.002257

6.4 Lower Respiratory Infection Prevalence

The results from the visual analysis of the visceral surfaces of the ribs are outlined here. Since this project used Davies-Barrett *et al.* (2019) recording and diagnostic method, this section follows their manner of data presentation. Data was gathered to enable accurate diagnosis and represent the true prevalence of lesions in the populations. Thus, the data are presented here to facilitate interpretation of the results and enable the accurate discussion of patterns.

Black Gate

In the Black Gate population, 208 individuals (or 79.1% of the total population) fit the criteria for observation, and almost 80% of the population (208 individuals) had one or more observable rib fragment. 11.1% of the population (23 individuals) was diagnosed with LRI (Table 6.54). Porous and remodelled or lamellar (PRL) lesions were slightly more common than the other bone types (Table 6.55).

Table 6.54 The crude prevalence rate of Black Gate individuals with lower respiratory infection diagnosis. Never infected refers to an individual with no evidence of lower respiratory infection.

Diagnosis	Lower Respiratory Infection	
	Number Affected	Crude Prevalence
Never Infected	185	88.9%
LRI	23	11.1%
Total	208	100.0%

Table 6.55 The crude prevalence rate of Black Gate individuals with visceral surface rib lesions by bone type. DW refers to Diffuse Woven; PRL to Porous/Remodelled/Lamellar; Both to both Diffuse Woven and Porous/Remodelled/Lamellar. Never infected refers to an individual with no evidence of lower respiratory infection.

Bone Type	Visceral Surface Rib Lesions	
	Number Affected	Crude Prevalence
Never Infected	185	88.9%
DW	3	1.4%
PRL	12	5.8%
Both	8	3.8%
Total	208	100.0%

The minimum estimate[24] of ribs from the left (1287) and right (1297) observable for VSRL was close to equal (Table 6.56). The true prevalence of VSRL is presented in Table 6.54 (Figures 6.9 and 6.10). The proportion of VSRL increased from neck to shaft on both sides of the body and peaked in the lower-middle section of the rib cage (ribs 7-9). This pattern was observed consistently in both left and right ribs (Table 6.57). There was an insignificant discrepancy between the proportion of lesions on the left (2.9%) and right (5.5%) lower ribs (p=0.227).

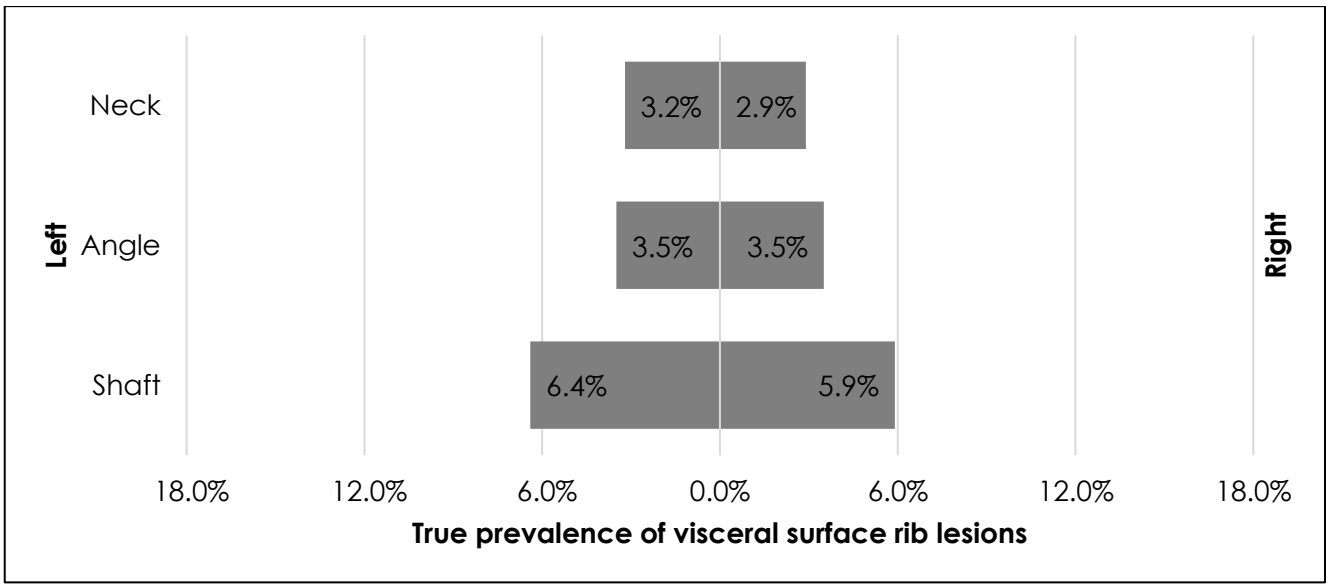


Figure 6.917 The true prevalence rate of visceral surface rib lesions on Black Gate individuals by rib section.

[24] The minimum estimate was based on the highest number of observed rib fragments of a certain type (head, neck, or shaft). The unknown true number of observed ribs may be higher than the minimum number.

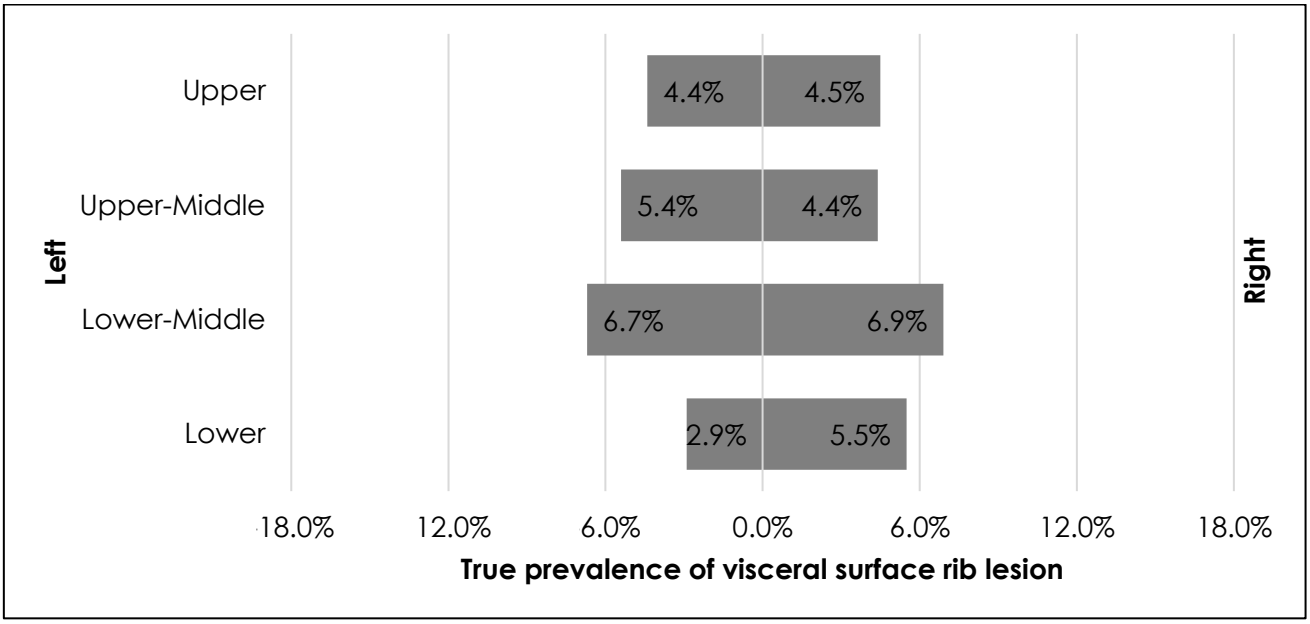


Figure 6.10 The true prevalence rate of visceral surface rib lesions on Black Gate individuals by rib cage section.

Table 6.56 The true prevalence rate of visceral surface rib lesions by section in the Black Gate population: first, by the section of the rib, then by the section of the rib cage. The proportions are scaled by the number of observable sections.

Side	Section	Visceral Surface Rib Lesions		
Left		**Number Affected**	**Observable**	**True Prevalence**
	Neck	36	1136	3.2%
	Angle	37	1070	3.5%
	Shaft	47	734	6.4%
	Upper	16	363	4.4%
	Upper-Middle	19	353	5.4%
	Lower-Middle	22	329	6.7%
	Lower	7	242	2.9%
Right				
	Neck	33	1145	2.9%
	Angle	38	1101	3.5%
	Shaft	44	741	5.9%
	Upper	16	358	4.5%
	Upper-Middle	15	344	4.4%
	Lower-Middle	25	360	6.9%
	Lower	13	235	5.5%

Table 6.57 The results of the binomial tests for the equality of proportions comparing the true prevalence of left/right visceral surface rib lesions in Black Gate ribs. Significant p-values are marked in green. df is degrees of freedom.

Output	Visceral Surface Rib Lesions						
	Neck	Angle	Shaft	Upper	U-M	L-M	Lower
p-value	0.781	1	0.792	1	0.652	1	0.227
df	1	1	1	1	1	1	1
X-squared	0.077163	3.47^{-31}	0.069263	4.99^{-30}	0.20283	6.41^{-30}	1.4627

Tables 6.58–6.65 present the true prevalence of individuals diagnosed with LRI divided by biological sex, age group, grave type, and body position; and the results of the associated tests for equality of proportions. LRI was more prevalent in males (14.4%) than females (7.6%) (Figure 6.11, Table 6.58) and generally increased with age at death (from 7.4% in Young Adults to 15.7% in Senior Adults; Figure 6.12, Table 6.60); however, these trends were statistically insignificant (Tables 6.59 and 6.61–6.62). As with the other infections, LRI was relatively evenly distributed amongst the grave types and body positions (Tables 6.63–6.65). However, Table 6.64 highlights a statistically significant difference between the prevalence of individuals diagnosed with LRI in coffin and plain grave types (p=0.007).

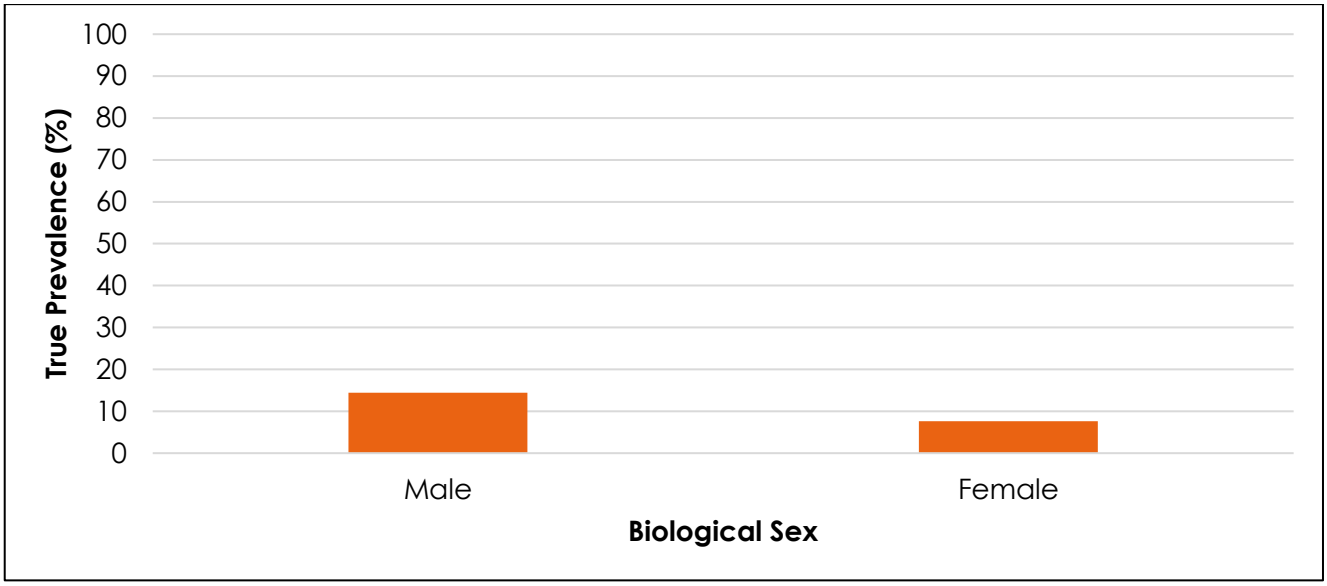


Figure 6.11 The true prevalence rate of Black Gate individuals with lower respiratory infection by biological sex.

Table 6.58 The true prevalence rate of Black Gate individuals with lower respiratory infection by biological sex.

Biological Sex	Lower Respiratory Infection		
	Number Affected	Observable	True Prevalence
Male	15	104	14.4%
Female	7	92	7.6%
Unidentified	1	12	8.3%
Total	23	208	11.1%

Table 6.59 The results of the binomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Black Gate males and females. Significant p-values are marked in green. df is degrees of freedom.

Output	Lower Respiratory Infection
	Male/Female
p-value	0.132
df	1
X^2	2.2749

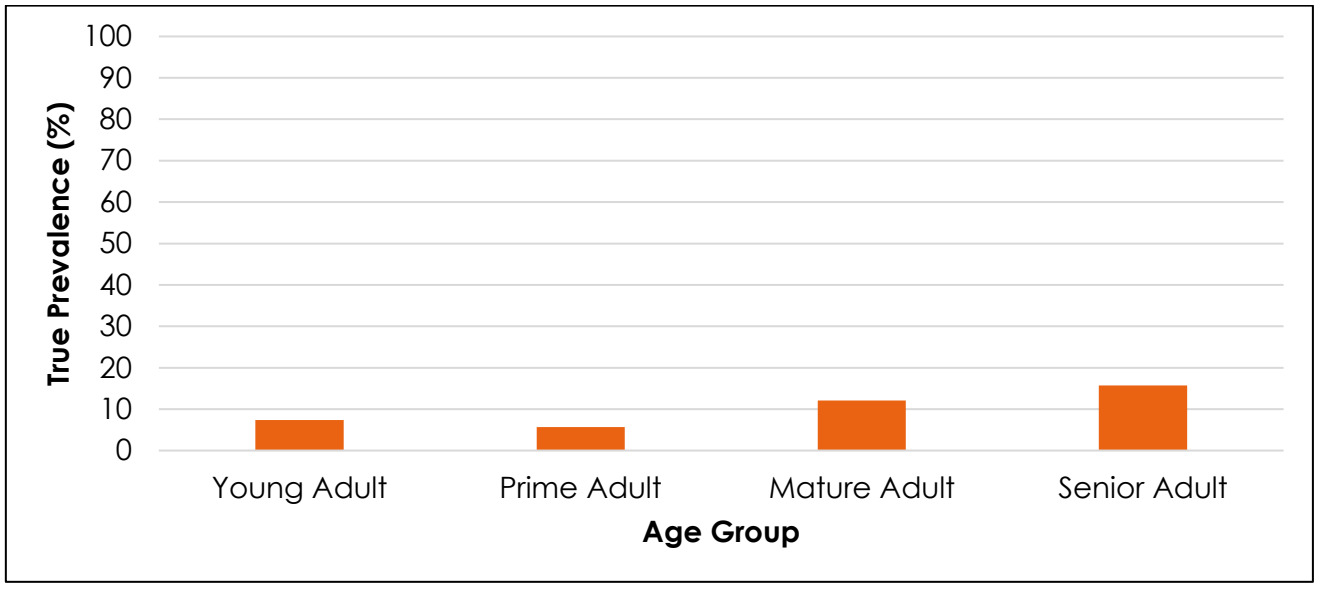


Figure 6.12 The true prevalence rate of Black Gate individuals with lower respiratory infection by age group.

Table 6.60 The true prevalence rate of Black Gate individuals with lower respiratory infection by age group.

Age	Lower Respiratory Infection		
	Number Affected	Observable	True Prevalence
Adult	0	0	0.0%
Young Adult	2	27	7.4%
Prime Adult	3	53	5.7%
Mature Adult	7	58	12.1%
Senior Adult	11	70	15.7%
Total	23	208	11.1%

Table 6.61 The results of the multinomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Black Gate individuals from each age group. Significant p-values are marked in green. df is degrees of freedom.

Output	Lower Respiratory Infection	
	Equality of Prop.	Linear Trend
p-value	0.316	0.085
df	3	1
X-squared	3.5393	2.9663

Table 6.62 The results of the binomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Black Gate individuals from each age group. Significant p-values are marked in green. df is degrees of freedom. Y is Young Adult. P is Prime Adult. M is Mature Adult. S is Senior Adult.

Output	Lower Respiratory Infection					
	Y/P	P/M	M/S	Y/M	Y/S	M/S
p-value	0.760	0.239	0.555	0.786	0.457	0.147
df	1	1	1	1	1	1
X^2	0.093175	1.3875	0.34877	0.073819	0.55328	2.1081

Table 6.63 The true prevalence rate of Black Gate individuals with lower respiratory infection by grave type. One individual who was recorded as being from a probable earmuff grave (recorded as "Earmuffs?") was recorded in this project as being from an earmuff grave.

Grave Type	Lower Respiratory Infection		
	Number Affected	Observable	True Prevalence
Plain	12	152	7.9%
Earmuffs	0	5	0.0%
Pillow Stone	0	2	0.0%
Head Box	1	3	33.3%
Coffin	7	28	25.0%
Chest	0	1	0.0%
Rubble Cist	0	2	0.0%
Cist	1	4	25.0%
Unidentified	1	11	9.1%
Total	23	208	11.1%

Table 6.64 The results of the binomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Black Gate individuals belonging to each grave type. Significant p-values are marked in green. df is degrees of freedom. C is coffin. P is plain. E is elaborate (chest, rubble cist, and cist). EV is elaborate variation (earmuffs, pillow stone, and head box). E&EV is elaborate and elaborate variation.

Output	Lower Respiratory Infection							
	C/P	C/ E&EV	P/ E&EV	C/E	C/EV	P/E	P/EV	E/EV
p-value	0.007	0.282	0.583	0.546	0.318	0.546	0.812	0.787
df	1	1	1	1	1	1	1	1
X^2	7.3275	1.1581	0.30139	0.36458	0.9975	0.36406	0.056343	0.072857

Table 6.65 The true prevalence rate of Black Gate individuals with lower respiratory infection by body position.

Body Position	Lower Respiratory Infection		
	Number Affected	Observable	True Prevalence
Supine	14	136	10.3%
Flexed	0	3	0.0%
Left Side	1	7	14.3%
Right Side	6	47	12.8%
Prone	1	6	16.7%
Unidentified	1	9	11.1%
Total	23	208	11.1%

Coronation Street

In the Coronation Street population, 88 individuals (or 71.5% of the total population) fit the criteria for observation and were observable for VSRL. Approximately a third of the population (28 individuals) was diagnosed with LRI (Table 6.66). As in the Black Gate population, PRL was the most common form of VSRL (Table 6.67).

Table 6.66 The crude prevalence rate of Coronation Street individuals with each lower respiratory infection diagnosis. LRI refers to lower respiratory infection. Never infected refers to an individual with no evidence of lower respiratory infection.

Diagnosis	Lower Respiratory Infection	
	Number Affected	Crude Prevalence
Never Infected	60	68.2%
LRI	28	31.8%
Total	88	100.0%

Table 6.67 The crude prevalence rate Coronation Street individuals with visceral surface rib lesions by bone type. DW refers to Diffuse Woven; PRL to Porous/Remodeled/Lamellar; and Both to both Diffuse Woven and Porous/Remodeled/Lamellar. Never infected refers to an individual with no evidence of lower respiratory infection.

Bone Type	Visceral Surface Rib Lesions	
	Number Affected	Crude Prevalence
Never Infected	60	68.2%
DW	4	4.5%
PRL	16	18.2%
Both	8	9.1%
Total	88	100.0%

The minimum estimate of ribs from the left (738) and right (732) observable for VSRL was close to equal (Table 6.68). The true prevalence of VSRL is presented in Table 6.68 (Figure 6.13 and 6.14). The proportion of VSRL remained relatively consistent from neck to shaft on both sides of the body and peaked in the lower-middle section of the rib cage on the right side (ribs 7-9). The difference between the number of VSRL on the left and right was insignificant throughout the rib cage (Table 6.69). These patterns were generally inconsistent with Black Gate, though both populations demonstrated a peak in lesions in the lower-middle ribs.

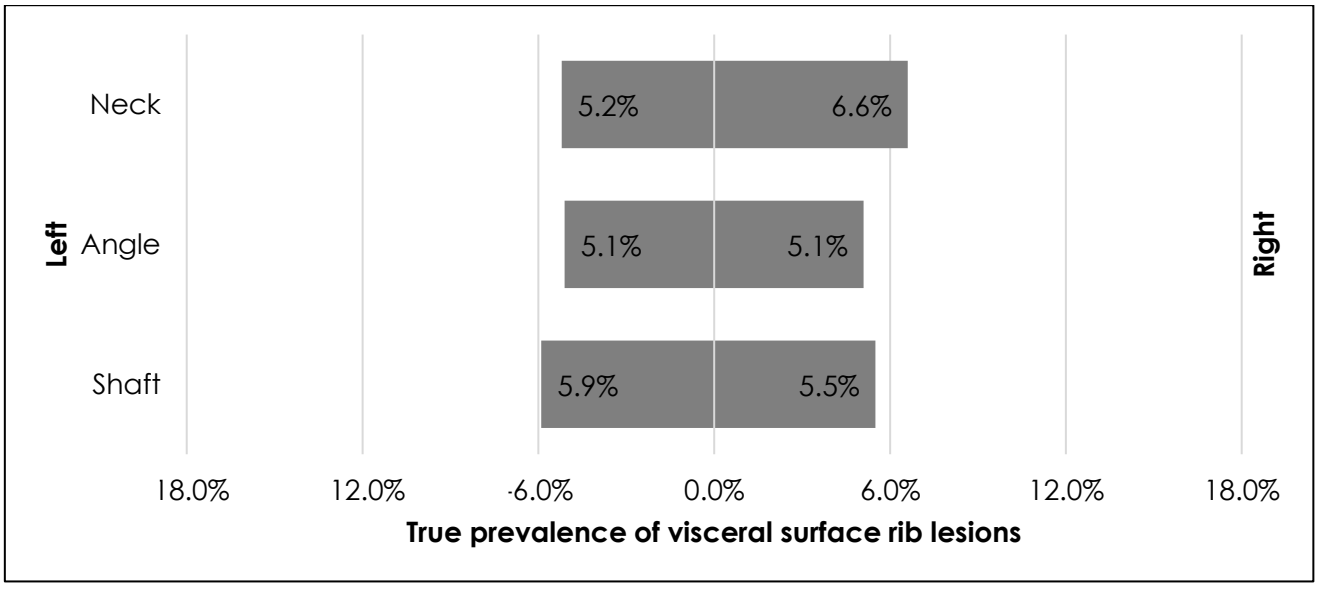


Figure 6.13 The true prevalence rate of visceral surface rib lesions on Coronation Street individuals by rib section.

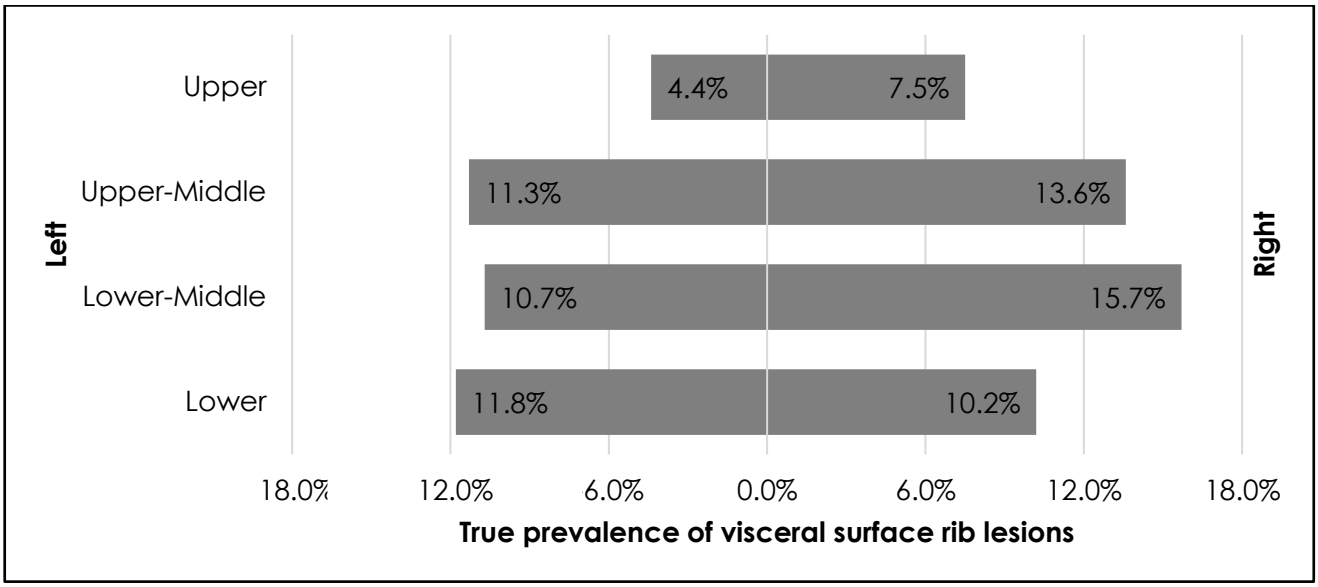


Figure 6.14 The true prevalence rate of visceral surface rib lesions on Coronation Street individuals by rib cage section.

Table 6.68 The true prevalence rate of visceral surface rib lesions by section in the Coronation Street population: first, by the section of the rib, then by the section of the rib cage. The proportions are scaled by the number of observable sections.

Side	Section	Visceral Surface Rib Lesions		
Left		**Number Affected**	**Observable**	**True Prevalence**
	Neck	36	693	5.2%
	Angle	34	667	5.1%
	Shaft	34	574	5.9%
	Upper	9	205	4.4%
	Upper-Middle	23	203	11.3%
	Lower-Middle	19	178	10.7%
	Lower	18	152	11.8%
Right				
	Neck	44	666	6.6%
	Angle	33	642	5.1%
	Shaft	29	526	5.5%
	Upper	15	201	7.5%
	Upper-Middle	27	199	13.6%
	Lower-Middle	29	185	15.7%
	Lower	15	147	10.2%

Table 6.69 The results of the binomial tests for the equality of proportions comparing the true prevalence of left/right lower respiratory infection in Coronation Street ribs. Significant p-values are marked in green. df is degrees of freedom.

Output	Visceral Surface Rib Lesions						
	Neck	Angle	Shaft	Upper	U-M	L-M	Lower
p-value	0.322	1	0.871	0.271	0.597	0.211	0.789
df	1	1	1	1	1	1	1
X^2	0.98029	1.08^{-29}	0.026397	1.2144	0.27943	1.5658	0.071455

Tables 6.70–6.74 present the true prevalence of individuals diagnosed with LRI divided by biological sex and age group, and the results of the associated tests for equality of proportions. As in the Black Gate population, there were insignificantly more males (35.7%) than females (31.0%) with LRI (Figure 6.15, Table 6.70–6.71). Unlike in the Black Gate population, the proportion of individuals diagnosed with LRI remained relatively consistent across the ages (from 37.5% in Young Adults to 33.3% in Senior Adults), with a slight peak in those who died as young adults (Figure 6.16, Table 6.72–6.74). There was no linear relationship between LRI and age at death (p=0.962; Table 6.73).

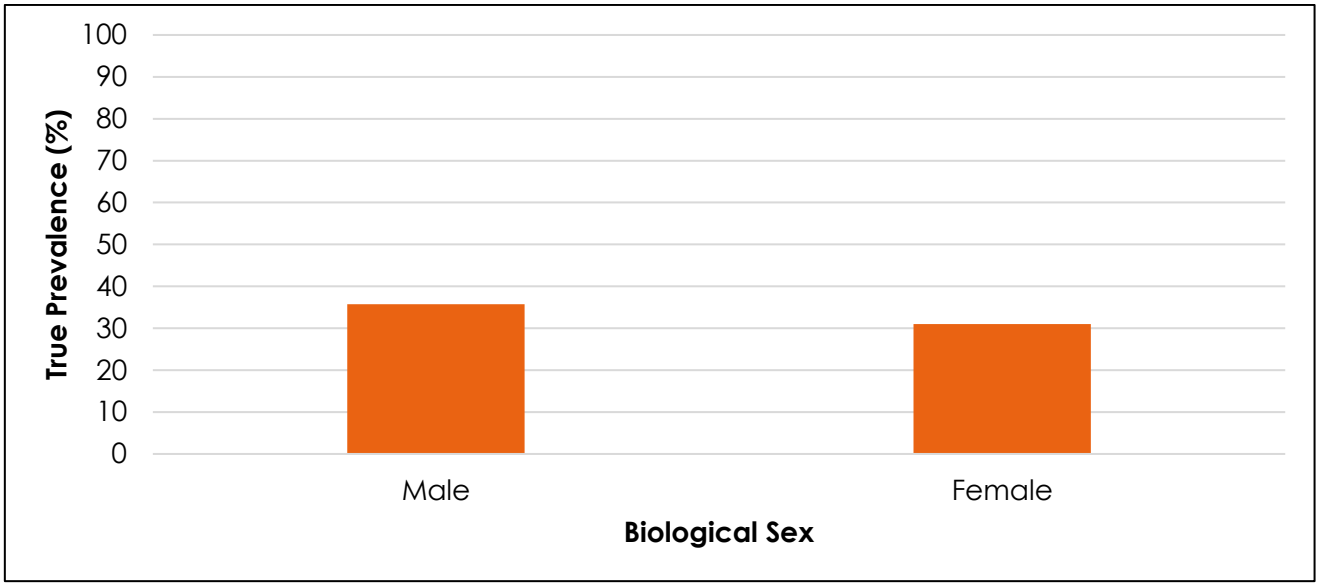


Figure 6.15 The true prevalence rate of Coronation Street individuals with lower respiratory infection by biological sex.

Table 6.70 The true prevalence rate of Coronation Street individuals with lower respiratory infection by biological sex.

Biological Sex	Lower Respiratory Infection		
	Number Affected	Observable	True Prevalence
Male	15	42	35.7%
Female	13	42	31.0%
Unidentified	0	4	0.0%
Total	28	88	31.8%

Table 6.71 The results of the binomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Coronation Street males and females. Significant p-values are marked in green. df is degrees of freedom.

Output	Lower Respiratory Infection
	Male/Female
p-value	0.643
df	1
X^2	0.21429

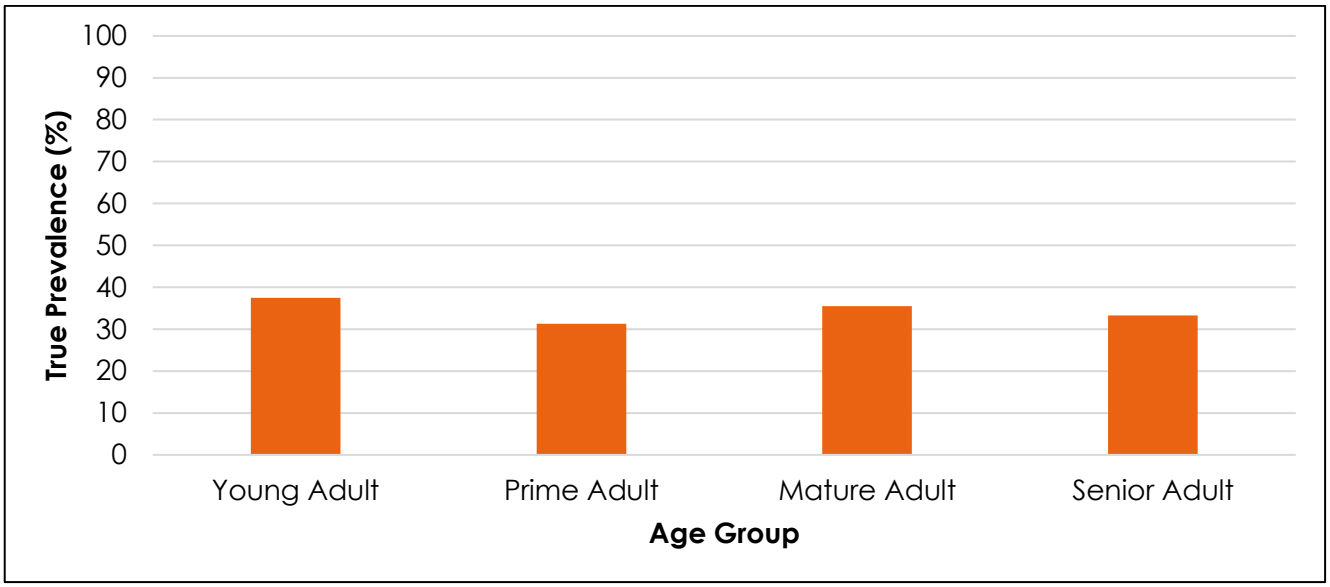


Figure 6.16 The true prevalence rate of Coronation Street individuals with lower respiratory infection by age group.

Table 6.72 The true prevalence rate of Coronation Street individuals with lower respiratory infection by age group.

Age	Lower Respiratory Infection		
	Number Affected	Observable	True Prevalence
Adult	2	12	16.7%
Young Adult	3	8	37.5%
Prime Adult	6	19	31.6%
Mature Adult	11	31	35.5%
Senior Adult	6	18	33.3%
Total	28	88	31.8%

Table 6.73 The results of the multinomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Coronation Street individuals from each age group. Significant p-values are marked in green. df is degrees of freedom.

Output	Lower Respiratory Infection	
	Equality of Prop.	Linear Trend
p-value	0.989	0.962
df	3	1
X-squared	0.12541	0.002313

Table 6.74 The results of the binomial tests for the equality of proportions comparing the true prevalence of lower respiratory infection in Coronation Street individuals from each age group. Significant p-values are marked in green. df is degrees of freedom. Y is Young Adult. P is Prime Adult. M is Mature Adult. S is Senior Adult.

Output	Lower Respiratory Infection					
	Y/P	P/M	M/S	Y/M	Y/S	M/S
p-value	0.766	0.777	0.879	1	1	1
df	1	1	1	1	1	1
X^2	0.088816	0.080047	0.023245	5.58^{-31}	7.18^{-32}	2.33^{-31}

6.5 Co-occurrence of the Pathologies

There were 12 chi-squared tests conducted to test the null hypothesis: that mastoiditis, maxillary sinusitis, and LRI did not co-occur in individuals any more than would be expected through chance alone. These tests examined the co-occurrence of residual childhood and

adult mastoiditis; residual childhood mastoiditis and maxillary sinusitis; adult mastoiditis and maxillary sinusitis; residual childhood mastoiditis and LRI; adult mastoiditis and LRI; and maxillary sinusitis and LRI. Each of these tests was run twice, first using the Black Gate data and then the Coronation Street data (Table 6.75). Because the population was tested six times, a Bonferroni correction was applied to the p-value and the threshold for significance was re-assessed to be p=0.0083. Only one test was statistically significant and caused the rejection of the null hypothesis: the test of the co-occurrence of childhood and adult mastoiditis in the Black Gate population ($p=1.13^{-7}$).

Table 6.75 The results of the Chi-square tests for the co-occurrence of infections in both populations. Significant tests are coloured green. RCM/AM is residual childhood mastoiditis and adult mastoiditis; RCM/MS is residual childhood mastoiditis and maxillary sinusitis; AM/MS is adult mastoiditis and maxillary sinusitis; RCM/LRI is residual childhood mastoiditis and lower respiratory infection; AM/LRI is adult mastoiditis and lower respiratory infection; MS/LRI is maxillary sinusitis and lower respiratory infection; and df is degrees of freedom.

Data	Output	RCM/AM	RCM/MS	AM/MS	RCM/LRI	AM/LRI	MS/LRI
Black Gate							
	p-value	1.13^{-7}	0.713	0.430	0.736	0.864	1
	df	1	1	1	1	1	1
	X^2	28.138	0.13551	0.62225	0.11329	0.029202	5.62^{-30}
Coronation Street							
	p-value	0.174	1	1	1	0.247	0.566
	df	1	1	1	1	1	1
	X^2	1.851	0	3.83^{-31}	0	1.3384	0.32897

6.6 Comparisons Between the Populations

Table 6.76 (Figure 6.17) presents the results of the remaining tests for the equality of proportions, which compared the proportion of individuals from Black Gate and Coronation Street diagnosed with each type of infection. Both the tests involving maxillary sinusitis (p=0.014) and LRI ($p=1.539^{-5}$) caused the null hypothesis to be rejected. In both instances, there was significantly more infection in the Coronation Street population than the Black Gate population. The test for adult mastoiditis was almost significant (p=0.082) and, unlike the other infection types, there were more individuals in Black Gate (59.2%) with AM than in Coronation Street (47.4%).

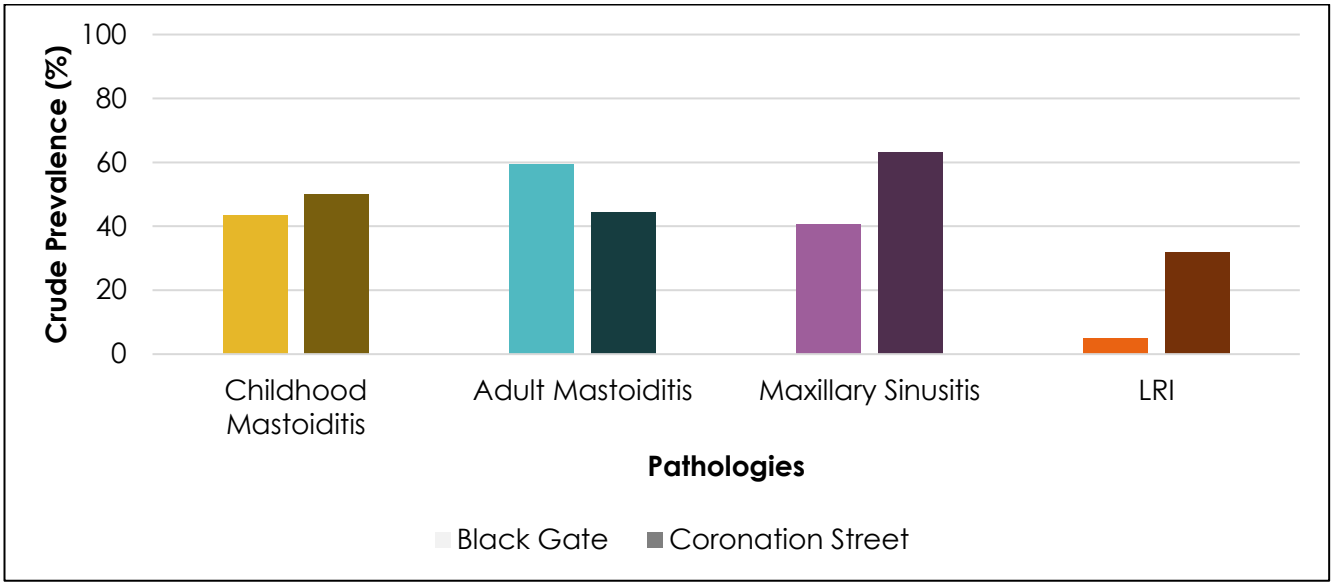


Figure 6.17 Comparing the crude prevalence rate of individuals with each pathology in each population. LRI is lower respiratory infection.

Table 6.76 The results of the binomial tests for the equality of proportion of individuals with infection from the Black Gate or Coronation Street populations. Significant tests are coloured green. Black Gate/Coronation Street is Black Gate versus Coronation Street; LRI is lower respiratory infection; and df is degrees of freedom.

Data	Output	Black Gate/Coronation Street
Residual Childhood Mastoiditis		
	p-value	0.397
	df	1
	X^2	0.71606
Adult Mastoiditis		
	p-value	0.082
	df	1
	X^2	3.0163
Maxillary Sinusitis		
	p-value	0.014
	df	1
	X^2	6.052
Individual-LRI		
	p-value	1.539^{-5}
	df	1
	X^2	18.689

These trends generally remained true by biological sex and age group (Figures 6.18–6.20). By biological sex, AM was more prevalent in Black Gate than Coronation Street. However, by biological sex, the proportion of females with residual CM (41.7% in Black Gate and 44.1% in Coronation Street) and males with MS (38.5% in Black Gate and 38.5% in Coronation Street) were similar in both populations.

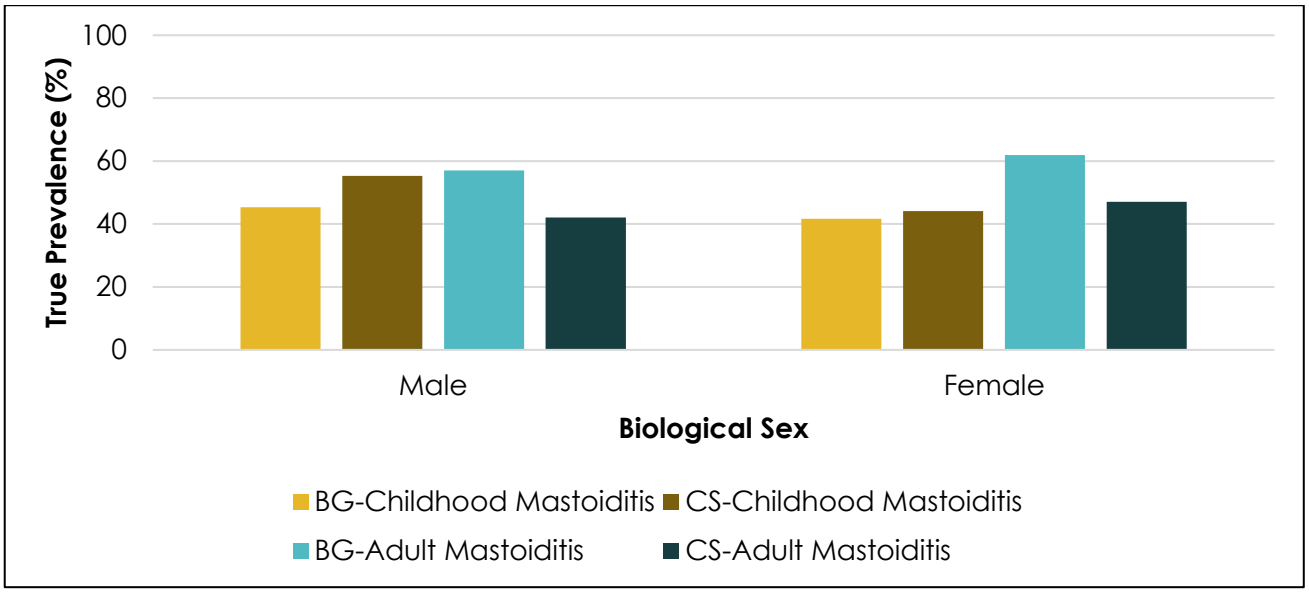


Figure 6.18 Comparing the true prevalence rate of individuals from each population with mastoiditis by biological sex.

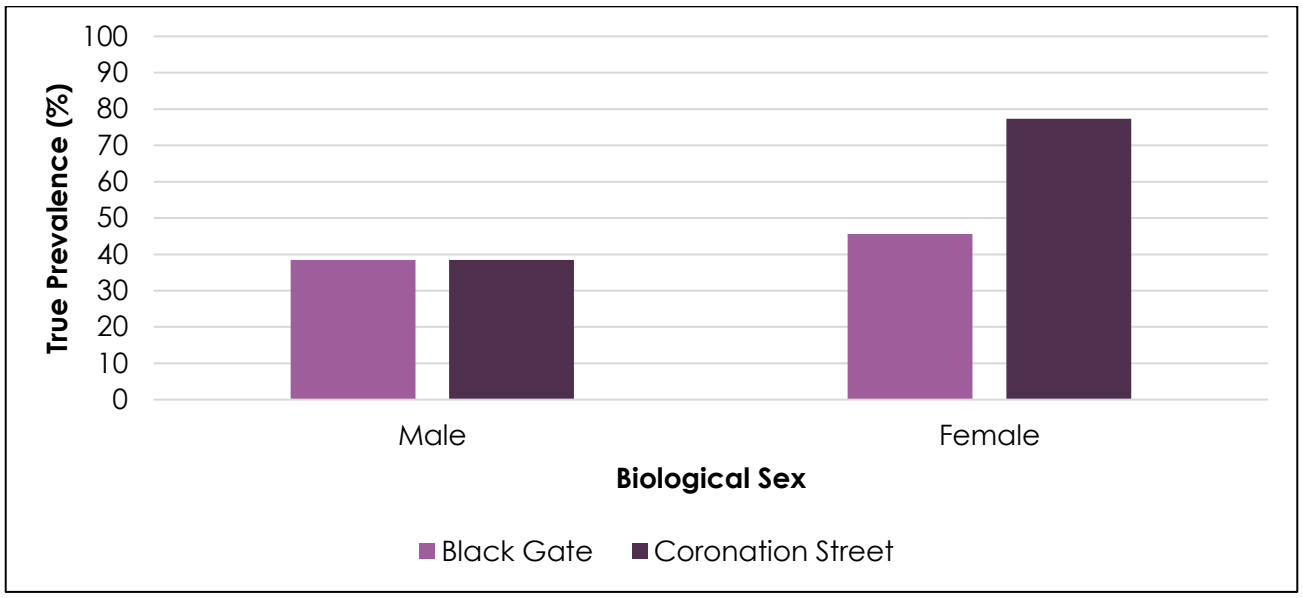


Figure 6.19 Comparing the true prevalence rate of individuals from each population with maxillary sinusitis by biological sex.

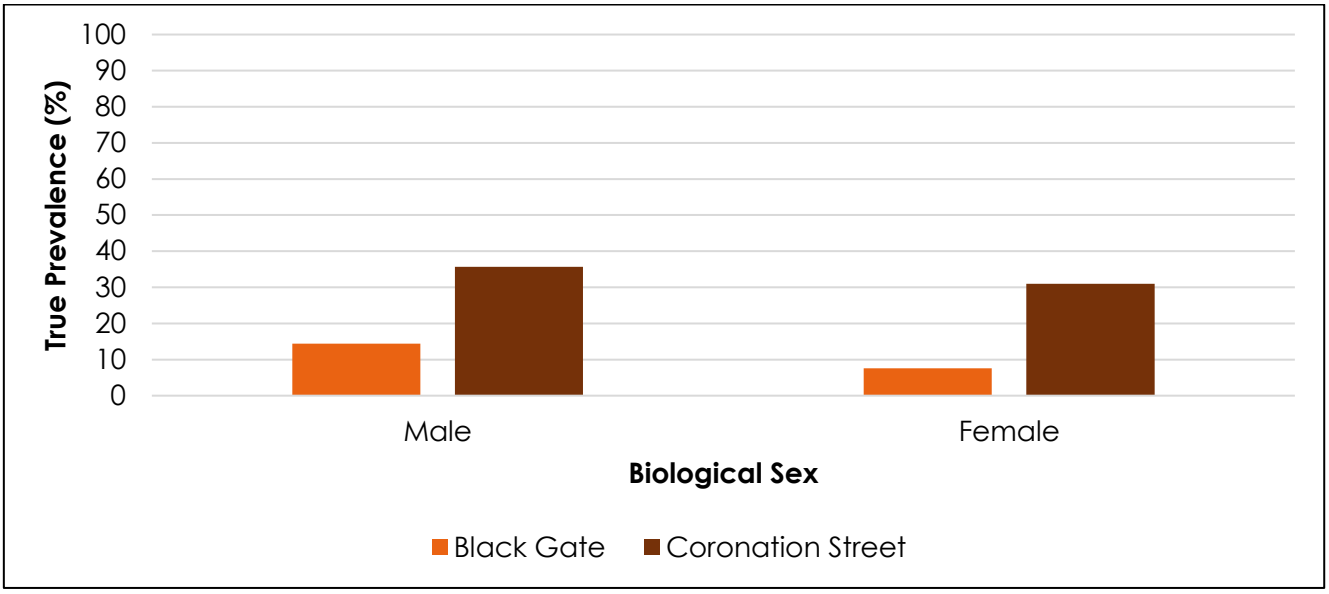


Figure 6.20 Comparing the true prevalence rate of individuals from each population with lower respiratory infection by biological sex.

By age group, there were consistently more individuals in Coronation Street with MS and LRI, and more Black Gate individuals with AM (Figures 6.21–6.23). However, this pattern varied for individuals diagnosed with residual CM. More Black Gate than Coronation Street individuals diagnosed with residual CM died at young and prime ages; while more Coronation Street than Black Gate individuals with residual CM survived until mature and senior age. In general, by age at death, there were more similarities between Black Gate and Coronation Street in the number of individuals diagnosed with residual CM and AM than MS and LRI.

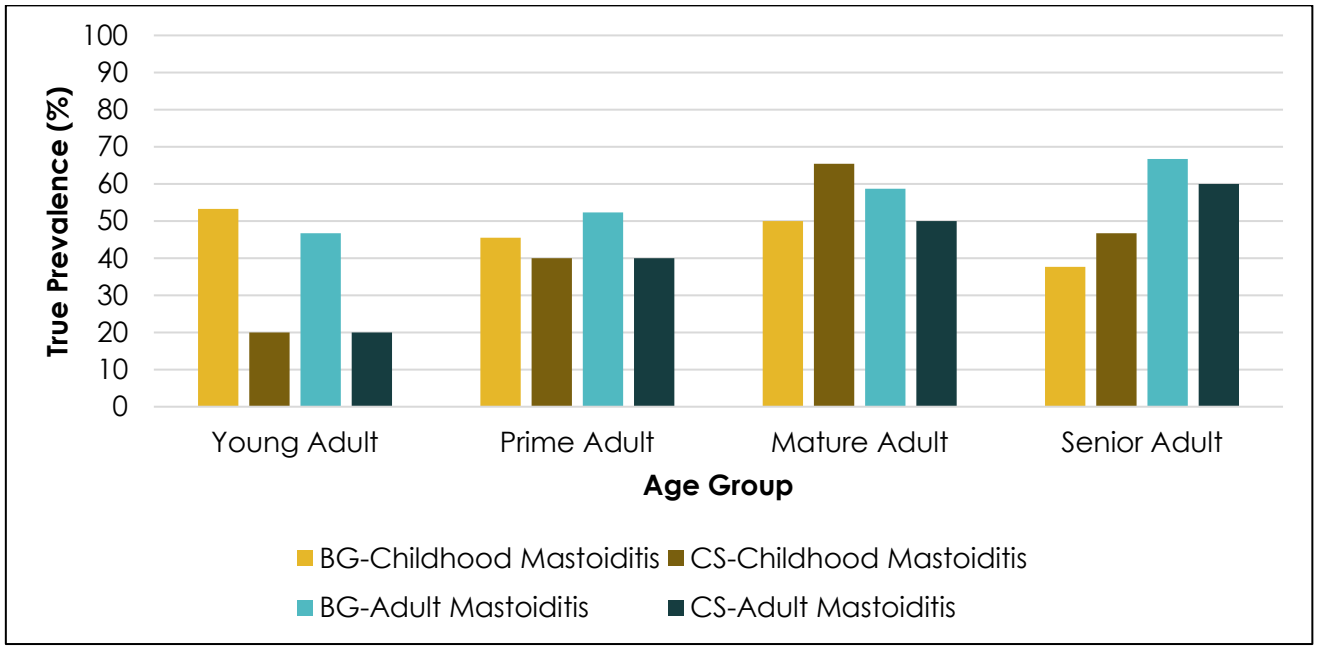


Figure 6.21 Comparing the true prevalence rate of individuals from each population with mastoiditis by age groups.

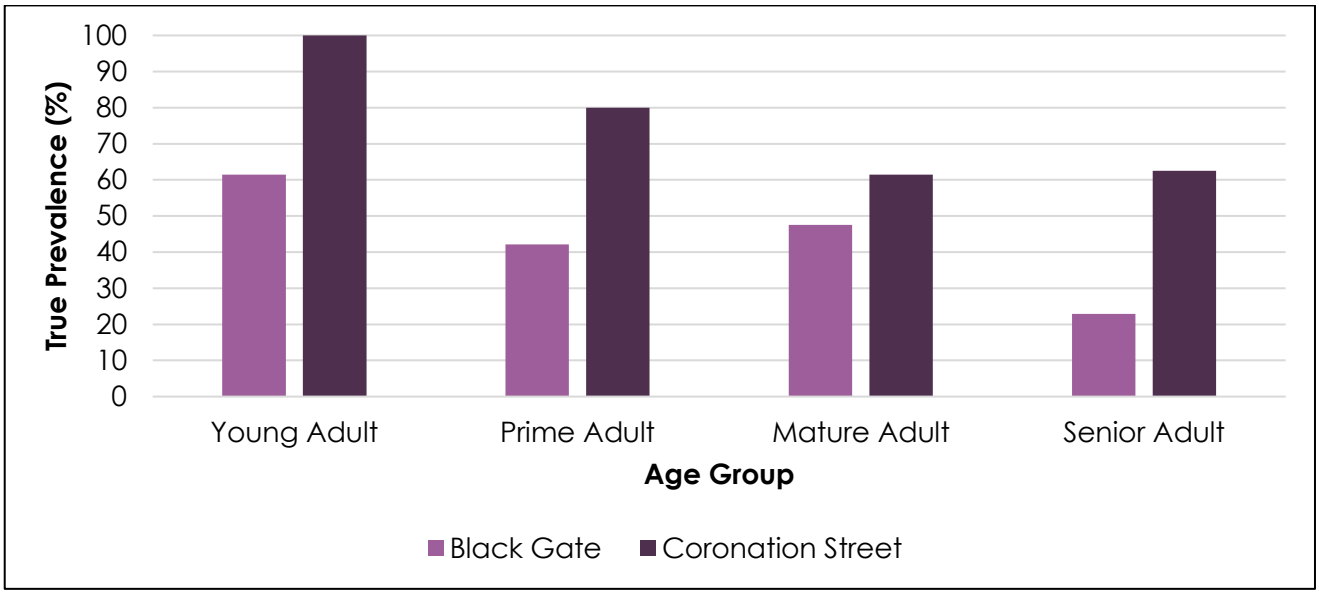


Figure 6.22 Comparing the true prevalence rate of individuals from each population with maxillary sinusitis by age groups.

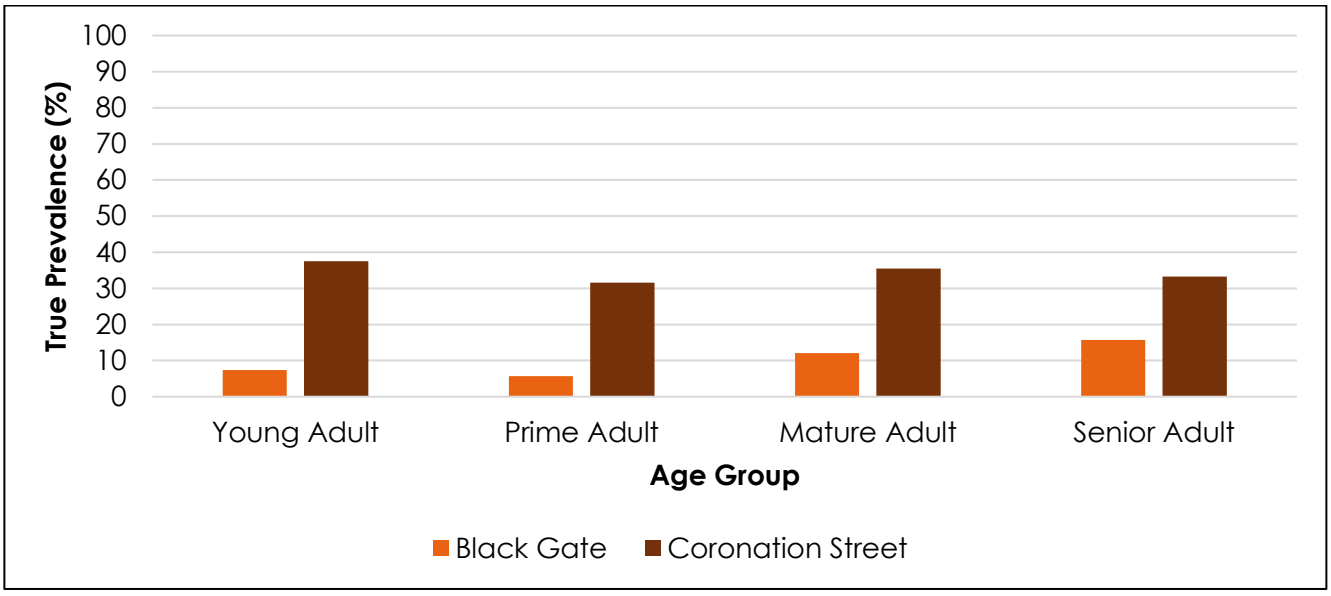


Figure 6.23 Comparing the true prevalence rate of individuals from each population with lower respiratory infection by age groups.

6.7 Multivariate Models

The null hypothesis tested by the construction of the general linear models (GLM) was that age, biological sex, and population (explanatory variables) were not predictive of infection and that any association between infection and the explanatory variables could be explained by chance alone. There were 13 GLMs constructed for Black Gate and 9 GLMs constructed for Coronation Street, each modelling the effect one explanatory variable had on another (Tables 6.77—6.78). There were four models constructed for the Black Gate

population using data regarding grave type and burial position that were not constructed for the Coronation Street population, because this data was not available.

Table 6.77 The results of general linear model construction for the Black Gate population. Significant p-values are coloured green. Param. is Parameters; P-val. is p-value; P. is Present; ND is Null Deviance; RD is Residual Deviance; AIC is Akaike information criterion; df is degrees of freedom; Pillow S. is Pillow Stone; and Rubble C. is Rubble Cist. Max. is Maxillary.

Residual Childhood Mastoiditis and											
Max. Sinusitis		LRI		Biological Sex		Age Groups		Grave Type		Body Position	
Param.	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**
Intercept	0.191	**Intercept**	0.096	**Intercept**	0.128	**Intercept**	1	**Intercept**	0.657	**Intercept**	0.571
MS-P.	0.563	**LRI-P.**	0.556	**Male**	0.658	**Prime Adult**	0.666	**Coffin**	0.971	**Left Side**	0.913
				Unsexed	0.222	**Senior Adult**	0.192	**Earmuffs**	0.765	**Prone**	0.355
						Young Adult	0.823	**Head Box**	0.991	**Right Side**	0.289
								Pillow S.	0.992	**Supine**	0.464
								Plain	0.779		
								Rubble C.	0.81		
Output											
ND	131.58		201.32		238.91		238.91		224.91		231.67
ND-df	95		146		173		173		163		168
RD	131.25		200.98		237.08		236.55		218.48		226.48
RD-df	94		145		171		170		157		164
AIC	135.25		204.98		243.08		244.55		232.48		236.48

(Table continued on next page)

Adult Mastoiditis and												
Max. Sinusitis		LRI		Biological Sex		Age Groups		Grave Type		Body Position		
Param.	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	**Param.**	**P-val.**	
Intercept	0.118	**Intercept**	0.188	**Intercept**	0.031	**Intercept**	0.241	**Intercept**	0.657	**Intercept**	0.571	
MS-P.	0.319	**LRI-P.**	0.672	**Male**	0.513	**Prime Adult**	0.54	**Coffin**	0.227	**Left Side**	0.615	
				Unsexed	0.636	**Senior Adult**	0.385	**Earmuffs**	0.765	**Prone**	0.638	
						Young Adult	0.417	**Head Box**	0.99	**Right Side**	0.224	
								Pillow S.	0.81	**Supine**	0.409	
								Plain	0.494			
								Rubble C.	0.992			
Output												
ND	131.58	201.32	235.3	235.3	218.64	223.92						
ND-df	95	146	173	173	160	165						
RD	130.58	201.14	234.73	231.84	210.83	220.57						
RD-df	94	145	171	170	154	161						
AIC	134.58	205.14	240.73	239.84	224.83	230.57						
Residual Childhood and Adult Mastoiditis												
Param.	**P-val.**											
Interc.	4.23^{-7}											
CM-P.	1.37^{-7}											

(Table continued on next page.)

Output	
ND	235.3
ND-df	173
RD	204.81
RD-df	172
AIC	208.81

Table 6.78 The results of general linear model construction for the Coronation Street population. Significant p-values are coloured green and insignificant p-values are coloured red. P-val. is p-value; ND is Null Deviance; RD is Residual Deviance; AIC is Akaike information criterion; and df is degrees of freedom.

Residual Childhood Mastoiditis and							
Maxillary Sinusitis		**LRI**		**Biological Sex**		**Age Groups**	
Parameters	**P-val.**	**Parameters**	**P-val.**	**Parameters**	**P-val.**	**Parameters**	**P-val.**
Intercept	1	**Intercept**	1	**Intercept**	0.494	**Intercept**	0.808
MS-Present	0.765	**LRI-Present**	0.86	**Male**	0.346	**Mature Adult**	0.237
				Unsexed	0.79	**Older Adult**	0.982
						Prime Adult	0.688
						Young Adult	0.298

(Table continued on next page)

Output							
ND	41.455		84.548		108.13		108.13
ND-df	29		60		77		77
RD	41.366		84.516		107.24		102.97
RD-df	28		59		75		73
AIC	45.366		88.516		113.24		112.97
Adult Mastoiditis and							
Maxillary Sinusitis		**LRI**		**Biological Sex**		**Age Groups**	
Parameters	**P-val.**	**Parameters**	**P-val.**	**Parameters**	**P-val.**	**Parameters**	**P-val.**
Intercept	0.566	**Intercept**	0.752	**Intercept**	0.732	**Intercept**	0.808
MS-Present	0.879	**LRI-Present**	0.157	**Male**	0.673	**Prime Adult**	0.85
				Unsexed	0.132	**Senior Adult**	0.465
						Young Adult	0.688
							0.298
Output							
ND	40.381		84.154		107.93		107.93
ND-df	29		60		77		77
RD	40.385		82.085		104.15		104.94
RD-df	28		59		75		73
AIC	44.385		86.085		110.15		114.94

(Table continued on next page)

Residual Childhood and Adult Mastoiditis	
Parameters	**P-val.**
Intercept	0.425
CM-Present	0.114
Output	
ND	107.93
ND-df	77
RD	105.39
RD-df	76
AIC	109.39

There were two models that were statistically significant and rejected the null hypothesis, both from Black Gate (Table 6.77). The first modeled AM and pooled biological sex (p=0.031). However, only the intercept was significant. This showed that biological sex was somewhat predictive of AM, though male biological sex itself was not. Likely, this meant that female biological sex (taken as the intercept) was predictive of AM and accounted for the model's significance.

The second significant model involved residual CM and AM (Table 6.77). Here, both the intercept ($p=4.23^{-7}$) and the remaining parameter (present childhood mastoiditis) ($p=1.37^{-7}$) were significant. This indicated that the presence and the absence of residual CM were predictive of the presence or absence of AM, respectively.

However, both of these models suffered from overdispersion[25] (Crawley 2013; Mendenhall et al. 2001:581–24), as evident by the mean deviance[26] of the models, 1.37 and 1.19. Thus, while these models accounted for some of the variance in the data, they did not account for all of it. There were innumerable unknown sources for variation in archaeological data: for example, variation amongst individuals in a population (e.g., differences in temporal periods or differences in social class unaccounted for by burial type or body position); correlations amongst individuals in a population (e.g., genetic relationships or shared occupations); or exclusion of individuals from a population (e.g., individuals unexcavated or destroyed historically) (Camacho et al. 2018; Olaniyi 2016). Aggregate data could have also caused overdispersion. This could have occurred if data clustered in a relatively small number of individuals in a population (e.g., Camacho et al. 2018; Shillito et al. 2020). In this project, some infection types occurred only in a small number of individuals, especially once those individuals were divided into variables (e.g., age at death).

Overdispersion often cannot be helped or accounted for archaeologically. Archaeological data are confined to those individuals recovered during excavation and limited to what can be inferred from the remains, grave types, body positions, and physical and stratigraphic locations. Additionally, statistical, functions that compensate for overdispersion require parameters that cannot be placed on archaeological material (e.g., collecting data in meaningful clusters to facilitate generalized estimating equations, which are robust against overdispersion) (Halekoh et al. 2006).

[25] Overdispersion means that there was more variance in the data than was accounted for by the model. This is true when the residual deviance is larger than the degrees of freedom (Crawley 2013; Mendenhall et al. 2001:581–24).

[26] The residual deviance divided by the degrees of freedom gives the mean deviance. The mean deviance is close to 1 in a properly dispersed model (Crawley 2013; Mendenhall et al. 2001:581–24).

This is not to say that the results of the GLMs were unhelpful. They were helpful in identifying which explanatory variables included in this project had the largest effect on the presence of adult mastoiditis. Constructing a maximum model and performing a stepwise multiple regression to arrive at a minimally adequate model defined which explanatory variables accounted for the majority of the variance in the data.

Regression Analyses

The null hypothesis tested by the GLM regression analyses was that no group (e.g., dying at a young age) or variable (e.g., age) was predictive of infection and that any association between the explanatory variables could be explained by chance alone. For every population, a maximum model was constructed, which modeled AM against every explanatory variable. For Black Gate, this included biological sex, age, grave type, body position, MS presence/absence, and LRI presence/absence. For Coronation Street, this included all but grave type and body position.

A step function was applied to both of the maximum models to produce minimally adequate models for each population. Table 6.79 presents the results of the maximum and minimally adequate models for Black Gate and Coronation Street. By definition, the minimally adequate models contained only those explanatory variables which were the most predictive of adult mastoiditis and accounted for the majority of the variance in the data. For Black Gate, this included MS and residual CM. Previously, the GLM that included AM and MS from Black Gate was not significant. This suggests that MS on its own was not significantly predictive of AM. This was reiterated in the maximum model, where MS appeared as one of the most significant explanatory variables; but was not significant itself. However, when MS was removed from the model, the model accounted for less variance in the data than when MS was included. Thus, MS was somewhat predictive of AM and was included in the minimally adequate model. For Coronation Street, only residual CM was included in the minimally adequate model along with AM.

Table 6.79 The results of the general linear model stepwise logistic regressions for both populations. Significant p-values from are coloured green. RCM is Residual Childhood Mastoiditis; MS is Maxillary Sinusitis; Exp. is Explanatory; ND is Null Deviance; RD is Residual Deviance; AIC is Akaike information criterion; and df is degrees of freedom.

Black Gate		Coronation Street	
Maximum Model			
Most Significant Exp. Variables	**p-value**	**Most Significant Exp. Variables**	**p-value**
RCM	0.002	RCM-Present	0.044
MS-Present	0.152		
Output			
Null Deviance	113.237		30.553
ND-df	81		23
Residual Deviance	86.845		17.372
RD-df	64		14
AIC	122.85		37.372
Minimally Adequate Model			
Output			
Included Exp. Variables	RCM and MS		RCM
Null Deviance	113.2		30.55
ND-df	81		23
Residual Deviance	96.17		26.32
RD-df	79		22
AIC	102.2		30.32

While the mean deviances of the minimally adequate models indicated that the data continued to suffer from overdispersion (1.22 and 1.20 for the Black Gate and Coronation Street models, respectively), the Akaike information criterion[27] (AIC) for both minimally adequate models are lower than those of their corresponding maximal models. This indicated that the minimally adequate models were more predictive of the data than the maximum models. Therefore, of the explanatory variables included in this project, those included in the minimally adequate models have the largest effect on the presence of AM.

[27] AIC demonstrates a model's predictive ability, where a lower AIC is more predictive than a larger AIC (Akaike 1974).

6.8 Conclusions

In this chapter, the true and crude prevalences of mastoiditis, maxillary sinusitis and lower respiratory infection in Black Gate and Coronation Street were presented and described, as were the results of the statistical tests. It was found that similar percentages of each population had residual CM (44.3% in Black Gate and 50.0% in Coronation Street) and AM (59.2% in Black Gate and 47.4% in Coronation Street) and that both residual CM and AM were bilateral significantly more than unilateral. In both populations, MS was most common in females and young adults; however, in Black Gate, there was a significant difference in the prevalence of individuals diagnosed with MS between the mature and senior age groups. Further, in both populations, LRI was most common in males and there was a significant difference in the prevalence of individuals with LRI from plain and coffin graves.

In general, there was a higher rate of MS and LRI in Coronation Street than Black Gate. However, individuals from Black Gate presented with more AM across the biological sexes and age groups, and more residual CM in the young to prime age groups, than individuals from Coronation Street. The Chi-squared tests for co-occurrence revealed that there was significant co-occurrence between residual CM and AM in Black Gate. This held true in a GLM. Pooled sex, specifically the female sex, was also significant in a model created with AM. Finally, regression analysis showed that MS and residual CM were the only explanatory variables predictive of AM in Black Gate; and residual CM was the only explanatory variable predictive of AM in Coronation Street. The impact of these results on our understanding of mastoiditis and the health of the populations is discussed in the following chapter.

Chapter 7
Discussion

7.1 Introduction

The objectives of this chapter are to characterize the epidemiology and etiology of mastoiditis and respiratory infection within the Black Gate and Coronation Street populations and, in the context of public health, inform the understanding of the lifeways of those living therein. First, the prevalence of each infection within each population is explored. Second, the results are discussed further in the context of a range of contemporary environmental and lifestyle factors that characterise the later Anglo-Saxon/Saxo-Norman and Industrial periods to examine what the prevalence of respiratory and respiratory-related diseases reveal about exposure to health risks. Third, the prevalence of disease is compared more broadly between the two populations. This discussion explores the critical differences between the populations: the risk factors in the natural, artificial, and social environments and, ultimately, the effect these had on population mortality and morbidity.

The following sections often refer to the individuals who died before senior age (45+ years) as non-survivors of adulthood. Those who die as non-adults are traditionally referred to as non-survivors of childhood (e.g., Gowland 2015; Gowland et al. 2018; Temple 2018; Wood et al. 1992); however, this project does not include non-adults. Rather, the term is used in line with the morbidity-mortality paradox, in which individuals who survive a physiologically stressful episode are concomitantly the most resilient and also newly frail, as a result of experiencing that physiological stress (Marklein et al. 2016). Individuals who died before 45 years of age were either genetically or physiologically unprepared for the environment they

faced, or they failed to adequately adapt to their environment and, therefore, died before reaching old age. Thus, their deaths can communicate much in terms of their individual frailty, their lifeways, and the risk factors that caused their physiological stress (Marklein et al. 2016; Wood et al. 1992).

7.2 Mastoiditis

This section discusses the prevalence of each pathological bone type and the implications for the pathophysiology of mastoiditis in both populations, possible sources of error in diagnosis, and the laterality of the infections. Four bone types were recorded from the X-rays of the mastoid processes, three of which were diagnostic of mastoiditis. One bone type was indicative of childhood mastoiditis (primary hypocellularity) and two bone types were indicative of adult mastoiditis (secondary hypocellularity and lytic lesions with secondary hypocellularity (LSH)). The rate of mastoiditis was high in both populations, with more residual childhood mastoiditis (residual CM) in Coronation Street (39 individuals or 50.0%) than Black Gate (77 individuals or 44.3%) and more adult mastoiditis (AM) in Black Gate (103 individuals or 59.2%) than Coronation Street (37 individuals or 47.4%). In both populations, secondary hypocellularity was more common than LSH. All proliferative bone types are indicative of pathological changes caused by chronic adult mastoiditis (Flohr and Schultz 2009a; Gregg and Steele 1982; Titch et al. 1981; Tremble 1934). But, in these populations, mastoiditis appeared to have manifested most commonly as new bone formation rather than bone destruction with sclerotic bone formation (Flohr and Schultz 2009a,b; Flohr et al. 2019). This may suggest that mastoiditis was often accompanied by an inflammatory reaction, triggering new bone formation (Roberts and Manchester 2005:5-6); and was less often accompanied by the pooling of exudate within the mastoid air cells (MAC), triggering bone destruction (Roberts and Manchester 2005:5-6). This suggested that inflammation from otitis media was continuous within the mastoid process air cells; but that exudate rarely extended beyond the middle ear cleft, into the *aditus ad antrum*, and down into the mastoid air cells. This type of reaction is seen in acute otitis media involving the temporal bone air cells and is supported by the absence of mastoid abscesses in both populations (Bluestone 1998; Groth et al. 2012; Wilson et al. 2017). Thus, while the prevalence of mastoiditis was high in both populations, the infection was either resolved—or individuals died—before it could progress to the extreme conclusion of abscess.

Proportionally, there was more secondary hypocellularity in Black Gate than Coronation Street and similar prevalences of LSH. By ratio (obtained by simplifying the average prevalence from the left and right sides), the differences between bone types in

each population are not large: in Black Gate, 9:5, and in Coronation Street, 8:6.3 (secondary hypocellularity:LSH, respectively). The variation suggests mastoiditis had a similar pathophysiology in each population; the responsible pathogen(s) had similar virulence and/or the triggered immunological responses were similar.

All attempts were made to limit misdiagnosis. It must be acknowledged, though, that some bone types may not have resulted from mastoiditis. Three sources of error were considered. First, taphonomy has been seen to mimic the appearance of lytic lesions (Flohr and Schultz 2009a). To counter this, only observed complete mastoid processes were included in the study. However, taphonomic interference cannot be completely ruled out, as the air cells are connected to the external environment *via* the external auditory meatus, middle ear, and *aditus ad antrum*. Second, type 1 hypocellularity is a type of bone morphology caused by adult infection (Flohr et al. 2019). The lytic lesions that form in the diploic, non-pneumatized bone, however, can appear like childhood mastoiditis radiographically. Some individuals with this bone type may have been diagnosed with both adult and childhood mastoiditis. Type 1 hypocellularity is caused by a chronic form of adult mastoiditis (Flohr et al. 2019) and since most individuals presented with what appears to have been acute infections, it is unlikely many individuals had this bone type. These individuals would have also lacked the distinctive radiographic clouding, caused by pathological new bone formation, within the diploic bone diagnostic of adult mastoiditis. However, some individuals with this form of bone may have been misdiagnosed with adult, as well as childhood, mastoiditis. Thirdly, and lastly, some forms of pathological bone formation within the MAC have been associated with asymptomatic, subclinical mastoiditis (Flohr and Schultz 2009a,b). It is, therefore, possible that some diagnoses of adult mastoiditis reflect subclinical forms of the infection. While these individuals would not have known they had mastoiditis, their immune responses were already reacting to the infections and, thus, affecting their morbidity. As such, this does not change how the impact of mastoiditis on individual health is discussed; but it is considered when exploring individual lived experience.

In both populations, childhood and adult mastoiditis were bilateral significantly more often than they were unilateral. The clinical literature rarely discusses the laterality of mastoiditis in terms of the disease's pathophysiology. Rather, it is discussed with regard to the diagnosis of the disease: unilaterality is often a sign that an individual has had mastoiditis, as uninfected MAC usually develop symmetrically (Flohr et al. 2019; Homøe and Lynnerup 1991; Homøe et al. 1996; Isono et al. 1999; Primeau et al. 2018, 2019; Schulter-ellis 1979; Titche 1981; Virapongse et al. 1985; Zhang et al. 2020). This speaks more to the development of the MAC

than to the progression of mastoiditis. As such, little can be said about the progression of mastoiditis in these two populations save that the disease appears to have had a similar pathophysiology in both.

Black Gate

Three distinctive patterns in mastoiditis prevalence within the Black Gate population emerged from the data in the present study: a relatively even distribution of lesions by grave type and body position; and no significant difference between the rate of residual CM or AM amongst either the biological sexes or age groups. When considering these results, it is worth remembering that the rate of residual CM was observed in surviving adults. Thus, the rate of infection reflects individual survivorship of childhood physiological stress and increased morbidity. This impacted the understanding of the prevalence of infection between the sexes and amongst the age groups.

First, taking into consideration the low number of individuals included in some groups, the distribution of mastoiditis (both childhood and adult) amongst the burial types and body positions was mostly representative of the disease prevalence within the population as a whole. The trend for those buried in coffin graves to have more respiratory disease, as seen with LRI, was not paralleled in the mastoiditis data; and none of the binomial tests for the equality of proportions comparing the prevalence of residual CM or AM by grave type were significant.

Second, there were slightly more females with AM than males. This was contrary to the higher rate of mastoiditis observed amongst males in both modern and archaeological populations (e.g., Chonmaitree et al. 2008; Csákányi et al. 2012; Daniel et al. 1988; Flohr and Schultz 2009a; Mathews et al. 1988; Rye et al. 2011; Uhari et al. 1996); but was similar to the patterns observed in MS within the adult Black Gate population. This preponderance of AM in females likely reflects increased female frailty and/or more female exposure to risk factors such as air pollution and working with children. Since AM increases with age at death and was accumulated over a lifetime, AM likely reflected morbidity and not an increased risk of mortality. If some of the diagnoses reflect sub-clinical cases of acute infection, this would have been especially true, as these cases of mastoiditis would have been mild and not lasted long enough to greatly impact the health of the individual.

In contrast, the slightly higher rate of residual CM amongst males suggested either that males were experiencing residual CM more than females, or that more males were surviving childhood with residual CM than females. That more male non-adults experienced residual CM is consistent with the clinical literature, which highlights children and males as the most susceptible members of a population to infection (Chonmaitree et al. 2008; Csákányi et al.

2012; Mathews et al. 1988; Rye et al. 2011; Uhari et al. 1996). Indeed, differences in health between females and males begin before birth, as neonatal males are more likely to be born prematurely (Cooperstock et al. 1998; James 2000) and are more vulnerable to illness than neonatal females (Bartels et al. 2005; Friedrich et al. 2006). These differences continue throughout childhood and into adulthood and have been observed to negatively affect male vulnerability to respiratory-related disease (Chonmaitree et al. 2008; Csákányi et al. 2012; Bartels et al. 2005; Friedrich et al. 2006; Mathews et al. 1988; Rye et al. 2011; Uhari et al. 1996). Thus, it is likely that the difference in the rate of AM between the sexes is due to male vulnerability to disease rather than decreased survivorship amongst Black Gate females.

Third, the rate of residual CM remained consistent, although there was a slight drop in residual CM prevalence in those who died as senior adults. The drop highlights that most individuals who experienced residual CM died before reaching senior age. So, residual CM somewhat increased morbidity and mortality by contributing to individual heterogeneity in risk.

This was also evident when the Chi-squared tests for co-occurrence, GLM models, and regression analyses were considered. residual CM and AM co-occurred significantly, and residual CM was included (along with MS) in the minimally adequate model of AM prevalence. A significant proportion of individuals with AM also had residual CM. Thus, residual CM was a significant risk factor for developing mastoiditis later in life and, in this way, further contributed to individual morbidity. AM increased morbidity but did not appear to have drastically increase mortality, as the rate of infection increased with age at death. So, neither residual CM nor AM contributed enough systemic stress to have noticeably affected the population's morbidity profile.

The average prevalence of mastoiditis and/or OM reported in archaeological populations is 35.7%, but the reported values vary widely across sites and studies: from 1%–83.4% (see Table 7.1). The diagnostic methods and criteria to diagnose these infections differs between studies, so comparing the rates of infection was problematic. As far as I am aware, this was the first systematic study of mastoiditis in a British population, regardless of period, so no analogous comparison for our results was possible. One other British study has occurred. This was a case study of an individual from Black Gate (SK3110) with a mastoid process abscess (consultation by Dr. Charles Romanowski, who also consulted on this project) (Boulter and Rega 1993:74–5). However, no publication resulted from this investigation. Six publications regarding mastoiditis and/or OM involving medieval European archaeological populations exist (Collins 2019; Flohr and Schults 2009a,b; Primeau et al. 2018, 2019; Qvist and Grøntved 2001), two of which also examined early medieval populations (Flohr and Schultz 2009a,b).

Of these studies, the populations considered were Icelandic, Polish, Danish, and German. The latter, involving the early medieval population (Flohr and Schultz 2009a,b). Interestingly, these studies include the two most extreme prevalence rates of infection: 1%–7% (Qvist and Grøntved 2001) and 83.4% (Flohr and Schultz 2009a,b). It should be noted that the studies by Flohr and Schultz (2009a,b) included non-adults (n=64/223), unlike this study. However, Flohr and Schultz (2009a,b) found that the rate of mastoiditis increased with age, from approximately 50%–60% amongst non-adult infants to approximately 90% amongst senior adults. Thus, if anything, the inclusion of non-adults decreased the rate of infection observed in the populations. The other studies recorded more moderate rates: 29.9% (Collins 2019), 31%–34% (Primeau et al. 2019), 39.0% (Krenz-Niedbała and Łukasik 2020:245–72), and 29%–41% (Primeau et al. 2018). The rates of mastoiditis recorded in Black Gate (residual CM at 44.3% and AM at 59.2%) were higher than the moderate rates recorded in these studies, but lower than the highest rate (Flohr and Schults 2009a,b).

Table 7.1 Prevalence of mastoiditis and otitis media in various adult populations as reported in previous studies. Highlighted time periods are comparative to Black Gate or Coronation Street. * refers to results that include non-adults. ** refers to results that only include non-adults. C and T are crude and true prevalence rate, respectively. n is sample size when this differs from the number of individuals in the population. F is females. M is males. Tmp is temporal bones.

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Casna and Schrader 2021	Post-medieval (1500–1850CE)	Netherlands	Arnhem	73	NA	24.4% (C)
Collins 2019	Medieval (872–1552CE)	Iceland	Hofstaðir, Keldudalur, Skeljastaðir, and Skiðuklaustur	197	38.2% (T) n=68 **F** 16.7% (T) n=30 **M** 41.7% (T) n=34	74.5% (T) n=192 **F** 76.7% (T) n=99 **M** 72% (T) n=93
Dalby et al. 1993	Early Anglo-Saxon (4th–7th centuries)	England	Baldock, Kingsholm, Barton, Raunds, Brough, and Chichester	469	NA	0.6% (C) otosclerosis **F** 2/? **M** 0/?

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Flohr and Schultz 2009a,b	Early medieval	Germany	Dirnstein and Rhens	223* (n=159 adults)	83.40%* (C) **F** 83.7%* and 73.7%* (C) **M** 91.3%* and 88.9%* (C)	NA
Floreanova et al. 2020	Natufian (12,900–10,250BCE) and Pre-pottery Neolithic (10,000–7,900BCE)	Southern Levant	Various sites	159	NA	c. 60.6% (C)
	Chalcolithic (4,500–3,300BCE)	Southern Levant	Various sites	70	NA	80% (C)
Floreanova et al. 2020	Roman (63BCE–324CE), Byzantine (324–638CE), and Ottoman (1517–1917CE)	Southern Levant	Various sites	107	NA	c. 50.4% (C)

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Gregg and Steele 1982	Pre-colonial	USA	William H. Over Museum collection, Sully Site, Sioux in the US National Museum, and Crow from the Crow Creek Massacre site all in South Dakota	536*	39% (C)	NA
	Post-colonial	USA	William H. Over Museum collection, Sully Site, Sioux in the US National Museum, and Crow from the Crow Creek Massacre site, all in South Dakota	679*	51%* (C)	NA
Gregg et al. 1981	mid-14th century	USA	Crow Creek Site, South Dakota	486*	38.4%* (T) n=837* Tmp	NA
Gregg et al. 1965a	Pre-colonial	USA	Sully Burial Site, South Dakota	358* (n=174 adults)	55%* (C) **F** 44/?* (T) **M** 58/?* (T)	0%* (C) otosclerosis

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Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Gregg et al. 1965b	Pre-colonial	USA	Various Indigenous peoples in South Dakota	221*	78.3%* (C) **F** 53/?* (T) **M** 118/?* (T)	NA
Holzhueter et al. 1965	Pre-colonial	USA	Arikara, Sioux Historic, and Middle Plains Woodland populations in South Dakota	180	NA	0.0% (C) otosclerosis
Homøe et al. 1996	Pre-17th century	Greenland	Inuit from the west and southeast coast of Greenland in the Greenland collections, Laboratory of Biological Anthropology, University of Copenhagen	127	4.7% (C)	NA

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Homøe et al. 1996 continued	18th–19th centuries	Greenland	Inuit from the west and southeast coast of Greenland in the Greenland collections, Laboratory of Biological Anthropology, Copenhagen University	56	17.9% (C)	NA
Krenz-Niedbała and Łukasik 2016	Medieval (10th–14th centuries)	Poland	Cedynia	257**	NA	53.4%** (C)
	Early-modern (14th–17th centuries)	Poland	Słaboszewo	178**	NA	39.0%** (C)

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Oxenham et al. 2005	Bronze and Iron Ages (6000–1700BP)	Northern Vietnam	Con Co Ngua, Quy Chu, Nui Nap, Thieu Duong, Vinh Quang, Min Duc, Duong Co, Dinh Chang, Doi Son, Chau Son, Dong Xa, Quy Chu, Nui Nap, and Dong Mom	192	1.0% (C) **F** 1/? (T) **M** 1/? (T)	NA
Primeau et al. 2018	Medieval (c.1050–c.1536CE)	Denmark	Randers	148	41% (C)	NA
	Medieval (1150–1550CE)	Denmark	Tjærby	93	29% (C)	NA
Purchase et al. 2019	Early Neolithic (8000–6800BP)	Russia	Shamanka II, Siberia (sample)	56	69.6% (C)	NA
Qvist and Grøntved 2001	Medieval (1050–1200CE)	Denmark	Nordby	169*	NA	1.2%–1.5%* (C)
	Medieval (1150–1350CE)	Denmark	Tirup	490*	NA	3.6%–6.9%* (C)

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
Rathbun and Mallin 1977	Pre-historic (1300–300CE)	Iran	Dinkha Tepe	15	40% (C) **F** 1/6 (T) **M** 5/6 (T)	NA
Schuler-ellis 1979	1920–1970CE	USA	Terry Collection Black sample	114	40.4% (C) **F** 38.9% (T) n=54 **M** 41.7% (T) n=60	NA
	1920–1970CE	USA	Terry Collection white sample	202	34.2% (C) **F** 27.5% (T) n=102 **M** 41.0% (T) n=100	
	c.1900CE	USA	Inuit from Riley D. Moore Collection	186	12.4% (C) **F** 2.3% (T) n=86 **M** 21.0% (T) n=21	NA

(Continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	Number of Individuals	Mastoiditis	Otitis Media
	400–1400CE	USA	Illinois Mound, Titterington Collection	200	31.0% (C) **F** 26.5% (T) n=98 **M** 35.3% (T) n=102	NA
Titche et al. 1981	Pre-colonial	USA	Grasshopper, Kinishba, Turkey Creek, and Point of Pines, all in Arizona	422	7.6% (T) n=763 Tmp	NA

The high rates of mastoiditis in both the adult early medieval German population (Flohr and Schults 2009a,b) and Black Gate may reflect similarities in the risk factors to which the populations were exposed. The diagnostic methods used in this project closely followed the bone types outlined in the articles by Flohr and colleagues (Flohr and Schultz 2009a,b; Flohr et al. 2019,2017,2009; Schultz et al. 2007); thus, it may be that their method (reflected in ours) was more sensitive than methods used in other studies. Since the methods used in their study and ours are comparable, the results are highly comparable. Thus, the contemporaneous nature of the early medieval German population and Black Gate may have placed both populations at a similarly high risk of mastoiditis. However, similarly high rates of mastoiditis and OM were observed in some South Dakota Indigenous and terminal Pleistocene–Holocene Levant populations (14,900 BCE–1,917 CE) populations (Floreanova et al. 2020; Gregg et al. 1965a,b). While the methods used by Floreanova *et al.* (2020) and Gregg *et al.* (1965a,b) are not as comparable to ours as those of Flohr and Schults (2009a,b), Coronation Street also presented with a similarly high rate of MS. Thus, such disease levels (and, consequently, such exposure to risk factors for mastoiditis) may not be unique to the early medieval period.

Flohr and Schultz (2009a,b) also noted that the rate of mastoiditis was higher in males than females (though, insignificantly so), and increased with age at death. This study noted no significant difference in the rate of mastoiditis between the biological sexes, although there was marginally more residual CM amongst males and AM amongst females. Biological sex has little impact on mastoiditis prevalence (Flohr and Schultz 2009a); so, it is likely that the slightly higher rate of AM we noted amongst females is truly reflective of exposure to environmental risk factors, such as time spent indoors with children and around cooking and heating fires (Roberts and Manchester 2007; Mansour et al. 2013:141–153; Doyle et al. 1999, 2000; Uhari et al. 1996; Zhang et al. 2014), rather than biological susceptibility. That AM increased with age at death was also consistent with the findings of Flohr and Schults (2009a). They concluded that this was likely due to an accumulation of lesions over time; or, as we discussed, that AM increased morbidity, not mortality, with those living the longest having had longer to experience one or multiple MAC infections.

Coronation Street

Two distinctive patterns in mastoiditis prevalence within the Coronation Street population emerged from the data in the present study: no significant difference between either the rate of residual CM or AM between the biological sexes or age groups. However, some trends were present. First, residual CM was slightly more common amongst males while AM was marginally more common amongst females. That more males than females survived

residual CM and survived into adulthood, suggests that male non-adults were more robust or more buffered from risk factors for residual CM and mortality than female non-adults. In the same way, the lower rate of AM to residual CM amongst males suggested that males were either more robust, or more buffered from risk factors relating to AM, than females. The differences in the prevalences that underpin these observations are insignificant, but they highlight a preferential trend amongst males across individual life histories.

Second, residual CM was slightly more common amongst mature adults and the prevalence of AM rose steadily with age at death. residual CM peaked in mature adults. Since adults with residual CM represent those who survived childhood infection, the low rate of residual CM amongst those who died young or in their prime may not indicate adult health, but, rather, non-adult frailty. Mastoiditis is common in non-adults (Krenz-Niedbała and Łukasik 2016b; Schultz et al. 2007). More Coronation Street non-adults who experienced residual CM may have died before adulthood than in Black Gate, eliminating from the adult population those who would have died as young or prime adults under more ideal circumstances. In this way, the pattern of residual CM reflects the attritional profile of the population. The peak in mature adults dying with residual CM may have reflected the earlier peak in male deaths and an accumulation of physiological stress resulting from labour, as also seen mildly with the rate of LRI.

The rate of AM rose with age at death and was significantly correlated with residual CM. As in Black Gate, AM appeared to reflect the risk of primary hypocellularity on recurrent mastoiditis and that AM did not increase mortality. Rather, it was a common infection that over half of individuals had before their deaths, but one that increased in prevalence with age at death. Its lower prevalence amongst those who died young and in their prime, compared to individuals of the same ages in Black Gate, is likely reflective of the low prevalence of residual CM in these age groups.

No publication has concerned mastoiditis in an industrial population (Table 7.1). As such, this project is the first. Regardless, the rate of residual CM (50.0%) and AM (47.4%) reported in the Coronation Street population fall within the wide range of rates reported in previous studies, as does the overall rate of mastoiditis observed in the population (78.2%) (see Table 7.1). The diagnostic methods used here are similar to those of Florh and Schultz (2009a,b) and follow-on from those reported in Purchase *et al.* (2019). As such, their results are the most comparable to ours. In each study, a high prevalence of mastoiditis was diagnosed. This was also true for Black Gate. Thus, I suggest that pre- and post-industrial populations lived in environments that exposed them to similar levels of risk. While the types of risk factors may have varied either side of industrialization, the level of risk appears to have remained high.

7.3 Maxillary Sinusitis

This section discusses the prevalence of each pathological bone type and the implications for the pathophysiology of MS in both populations, possible sources of error within our diagnosis, and the laterality of the infection. Less than half (40.5%) of the observable individuals from Black Gate had lesions indicative of MS whereas 63.2% of those observed from Coronation Street presented lesions. In both the Black Gate and Coronation Street populations, the majority of individuals observable for MS presented with what Boocock *et al.* (1995) classed as spicules. This is consistent with the findings of other researchers (Boocock et al. 1995; Krenz-Niedbała and Łukasik 2016a; Lewis et al. 1995). Boocock *et al.* (1995:491) also reported a high proportion of white pitted bone (WPB) in the sample they studied (18.2% in the non-leprous population). This project, however, excluded any individual with obvious interaction between the mouth and the maxillary sinus, to avoid conflating odontogenic sinusitis with respiratory sinusitis and to focus this study on external risk factors which placed pressure on the population, which likely explains the low prevalence of WPB. Moreover, sinuses with pitting in the floor which appeared to extend through the alveolar bone were also excluded from this analysis, which likely accounts for the low prevalence of pitting reported by this project compared to others.

In both Black Gate and Coronation Street, spicules were more common than remodeled spicules. This was especially true in Coronation Street, where remodeled spicules were less common than WPB and just as common as pitting. In Black Gate, remodelled spicules were the second most frequent new bone type, with a ratio of spicules to remodeled spicules of 2.7:1 compared to 31:1 in Coronation Street. Spicules likely reflect the presence of inflammation within the maxillary sinus at the time of death, while remodeled spicules may reflect an early state of healing following an inflammatory event or during a chronic infection, similar to sub-periosteal reaction on long bones (DeWitte 2014a; Mays et al. 2012; Novak and Šlaus 2010; Wood et al. 1992). Thus, it may be possible to use spicules as a proxy for active MS and remodeled spicules as a proxy for healing/chronic MS.

While MS itself is unlikely to significantly contribute to an individual's mortality (NHS 2021), that so many more Coronation Street individuals may have died with active rather than healing/chronic MS suggested that the lifeways that placed Coronation Street individuals at risk of death at each age may also have been, or been associated with, the factors that placed them at a higher risk of developing sinusitis, such as overcrowding and domestic and industrial air pollution(e.g., DeWitte 2014a,b; DeWitte and Wood 2008; Mays et al. 2002; Usher 2000; Wood et al. 1992). Similarly, amongst individuals from multiple medieval London populations, DeWitte (2014a) found that active woven sub-periosteal reaction was

more indicative of frailty than healing lamellar bone, when considering the state of sub-periosteal lesions and individuals' age at death. In a similar way, this study infers that the Coronation Street population may have been frailer than the Black Gate population due to its exposure to more, and more varied, environmental risk factors.

Misdiagnosis by accidentally including odontogenic MS in this study cannot be ruled out despite excluding individuals with obvious communication between the alveolar bone and maxillary sinuses. In her study, Roberts (2007) implied that odontogenic MS was more likely to be unilateral rather than bilateral, with MS developing on the same side as the dental disease. If this is true, it could be possible that Black Gate individuals experienced more MS of dental origin than Coronation Street individuals, as unilateral MS was more common in Black Gate than Coronation Street. However, it cannot be argued that one population was more at risk of odontogenic MS than the other, as oral hygiene was poor in both populations, though consistent with contemporaneous populations (Swales 2021). While odontogenic MS cannot be ruled out as the cause of some of the cases of MS recorded here, the efforts made to eliminate it from the diagnosis increase the rigour to the interpretations made here.

Bilateral MS was more common in both Black Gate and Coronation Street than unilateral MS. At Coronation Street, this difference was significant, with 75.0% of observable individuals presenting with bilateral MS. While in Black Gate, the prevalence of unilateral and bilateral MS were closer to 2:3, respectively. This likely reflected a difference between the etiology or pathophysiology of sinusitis in the two populations. The laterality of MS is rarely reported archaeologically. Roberts (2007) simply stated that, in her study, both definitive odontogenic MS and bilateral MS were rare. However, clinically, unilateral–unlike bilateral—sinusitis is cause for medical examination (NHS 2021). Today, the most common etiologies for unilateral MS are chronic sinusitis, a dense area of fungal growth, a polyp, or a benign or malignant tumour in, or involving, the maxillary sinus (El-Feky 2021). It is possible that individuals from Black Gate, compared to those from Coronation Street, were more prone to, or exposed to more risk factors for, such pathologies.

Black Gate

Three distinctive patterns in MS prevalence within the Black Gate population emerged from the data in the present study: a relatively even distribution of lesions by grave type and body position; marginally more females (45.6%, 26/57) than males (38.5%, 25/65) were diagnosed with MS; and MS prevalence generally decreased with increasing age at death. First, all the binomial tests for the equality of proportions comparing the presence of MS by grave type

were insignificant. Groups with larger numbers of observable individuals broadly reflected the prevalence of infection within the wider population (e.g., 41.1% or 39/95 of individuals from plain graves with MS). There appeared to be no difference in exposure to risk factors for MS between differing social statuses, or between the later Anglo-Saxon and Saxo-Norman populations.

Second, there were more females than males with MS (although this pattern was not significant, p=0.424). As with AM, the preponderance of MS in females may have reflected increased female frailty and/or more female exposure to risk factors for URI. That most cases of MS may have been active at the time of death further supported the theory that these deaths were amongst frailer individuals, or individuals for whom MS, or one or more associated diseases, were the cause of death. The slightly higher rate of infection amongst biological females compared to biological males may help to explain the increase in MS amongst mature adults, as a higher prevalence of females died in the prime and mature adult groups (Swales 2012:147–9). Likewise, more females who died at a mature age also had MS, compared to those who died at a prime age. This would suggest that MS contributed to female morbidity but did not contribute strongly to their mortality rate.

Third, there was a linear trend in the prevalence of MS by age at death. From young adult to senior adult, there was a significant (p=0.022) drop in infection rate (61.5%–22.9%); and the rate of infection dropped most significantly (p=0.027) between mature and senior adults (47.5%–22.9%). The linear decline in MS prevalence with age at death is contrary to the clinical expectation, as senior age is a risk factor for developing chronic sinusitis (Harvard Health Publishing 2020; Hoover et al. 1997). That more individuals who died young experienced MS than those who died when older suggested either that younger individuals were exposed to more risk factors for MS that outweighed their biological advantage (such as engaging in childcare or agriculture), while those who survived to senior age were those who did not engage in/with such risky activities; or that those who survived to senior age were the most robust, with stronger immune responses, and more capable of surviving health insults. That being said, it was impossible to say if those who lived the longest were of a higher social status (and, therefore, robust by way of being buffered from certain health insults) or if they were more naturally robust than those who died before senior age. Unlike AM, which increased with age at death, MS likely reflected both morbidity and mortality, as it was related to younger ages.

The population prevalence of MS recorded here (40.5%) is within the range reported by the previous studies that also used the Boocock *et al.* (1995) criteria for classifying new bone formation in the maxillary sinuses (Table 7.2). Some studies found higher rates of MS amongst

biological females than biological males (Panhuysen et al. 1997; Shapland et al. 2015; Roberts 2007), though others found similar rates between the biological sexes (Boocock et al. 1995; Davies-Barrett et al. 2021b; Lewis et al. 1995; Merrett and Pfeiffer 2000) and one found more maxillary sinusitis amongst males compared to females (Davies-Barrett et al. 2021a). Thus, while this study found a slight difference in the rate of MS between the biological sexes, that this difference was insignificant is consistent with other studies of medieval populations (Boocock et al. 1995; Lewis et al. 1995). Caution should be taken when comparing rates too directly, though, due to differences in selective mortality caused by hidden heterogeneity of risk (Wood et al. 1992). Broadly comparing results, however, suggested that the Black Gate population followed expected public health trends for the later Anglo-Saxon/Saxo-Norman periods.

Table 7.11 Prevalence of maxillary sinusitis in various populations as reported in previous studies. Highlighted works used the Boocock et al. (1995) methodology. Highlighted time periods are comparative to Black Gate or Coronation Street. One study presenting prevalence by left and right maxillary sinus rather than by total. MS is Maxillary Sinusitis. * refers to results that include non-adults. ** refers to results that only include non-adults. C and T are crude and true prevalence rate, respectively. N is the number of individual in the population. n is number of individual in the sample. Results from Davies-Barrett (2018) are presented for individuals with two sinuses present and those with one sinus present. The discrepancy in the rate of MS reported by biological sex in Digangi and Sirianni (2017:159–60) is present in the literature. The data from Merrett and Pfeiffer (2000) is presented by biological sex as individuals with remodelled spicules, abscessing, and remodelled spicules and no abscessing. Inconsistency in the female and male rate of MS in Digangi and Sirianni (2017) originally appears in that text.

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Boocock et al. 1995	Late medieval (12th–17th centuries)	England	Chichester	54.9% (C) N=133	31/? (T)	47/? (T)
Casna and Schrader 2021	Post-medieval (1500–1850CE)	Netherlands	Arnhem	50.9% (C) N=73	NA	NA
Collins 2019	Medieval (872–1552CE)	Iceland	Hofstaðir, Keldudalur, Skeljastaðir, and Skiðuklaustur	56.3% (T) N=305 n=257	63.3% (T) n=90	80.7% (T) n=83

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Davies-Barrett 2018	Kerma Classique period (1750–1500BCE)	Sudan	4-L-2	64.0%/ 60.7% (T) N=31 n=25/28	66.7%/ 66.7% (T) n=6/6	61.5%/ 61.5% (T) n=13/13
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-88	66.7%/ 66.7% (T) N=8 n=3/6	0%/0% (T) n=1/1	100%/ 100% (T) n=1/2
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-100	33.3%/ 20.0% (T) N=5 n=3/5	0%/0% (T) n=0/0	0%/0% (T) n=0/1
	Meroitic and Post-Meroitic (c.300BCE–600CE)	Sudan	3-Q-33	64.3%/ 61.1% (T) N=20 n=14/18	100%/ 83.3% (T) n=4/6	55.6%/ 54.5% (T) n=9/11
	Post-Meroitic	Sudan	3-O-1	61.5%/ 57.1% (T) N=17 n=13/14	50.0%/ 50.0% (T) n=2/2	62.5%/ 44.4% (T) n=18/9

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Davies-Barrett 2018 continued	Post-Meroitic	Sudan	4-M-53	16.7%/ 28.6% (T) N=7 n=6/7	50.0%/ 50.0% (T) n=2/2	0%/0% (T) n=4/4
	Medieval	Sudan	3-J-23	55.6%/ 50.0% (T) N=67 n=45/58	42.1%/ 32.1% (T) n=19/28	70.8%/ 74.1% (T) n=24/27
	Post-Meroitic or Early Christian periods	Sudan	3-J-18	66.7%/ 56.5% (T) N=118 n=51/85	73.7%/ 62.2% (T) n=19/37	61.3%/ 54.5% (T) n=31/44
	Neolithic	Sudan	R12	35.7%/ 32.5% (T) N=43 n=14/40	NA/ 25.0% (T) n=0/4	40.0%/ 18.2% (T) n=5/11
	Kerma (2500–2050BCE)	Sudan	P37	80.0%/ 76.7% (T) N=34 n=20/23	100%/ 100% (T) n=6/7	78.6%/ 75.0% (T) n=14/16

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Davies-Barrett 2018 continued	Meroitic (c.200BCE–200CE), Post-Meroitis (c.400–700CE), and Medieval (c.800–1200BCE)	Sudan	Gabati	59.0%/ 54.5% (T) N=108 n=78/101	71.4%/ 65.8% (T) n=28/38	41.9%/ 39.0% (T) n=31/40
	Medieval	Sudan	Soba East	84.4%/ 82.4% (T) N=35 n=32/34	66.7%/ 66.7% (T) n=6/6	81.3%/ 81.3% (T) n=16/16
Davies-Barrett et al. 2021a	Late Intermediate Period (1000–1476CE)	Peru	Pachacamac	93.8% (T) N=39 n=16	100% (T) n=5	100% (T) n=7
Davies-Barrett et al. 2021b	Neolithic (6000–3100BCE)	Sudan	R12	32.5% (C) N=40	25.0% (T) n=4	18.2% (T) n=11
	Kerma (2500–1500BCE)	Sudan	P37	76.7% (C) N=30	100% (T) n=7	75.0% (T) n=16
	Kerma (2500–1500BCE)	Sudan	Fourth Cataract and Kerma Classic sites	56.4% (C) N=39	57.1% (T) n=7	62.5% (T) n=16

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Davies-Barrett et al. 2021b continued	Meroitic (300BCE–1500CE) to Post-Meroitic (350–550CE)	Sudan	Fourth Cataract and Meroitic/Post-Meroitic sites	53.8% (C) N=39	70.0% (T) n=10	45.8% (T) n=24
	Meroitic (300B–1500CE) to Post-Meroitic (350–550CE)	Sudan	Kawa	74.5% (C) N=55	77.3% (T) n=22	72.7% (T) n=22
	Meroitic (300B–1500CE) to Medieval (550–1500CE)	Sudan	Gabati	54.5% (C) N=101	65.8% (T) n=38	39.0% (T) n=40
	Medieval (550–1500CE)	Sudan	3-J-23	50.0% (C) N=58	32.1% (T) n=28	74.1% (T) n=27
	Medieval (550–1500CE)	Sudan	3-J-18	56.5% (C) N=85	62.2% (T) n=37	54.5% (T) n=44
	Medieval (550–1500CE)	Sudan	Soba East	82.4% (C) N=34	66.7% (T) n=6	81.3% (T) n=16
Digangi and Sirianni 2017	19th century	USA	Monroe County Almshouse, New York	54.5%* (T) N=301 n=99	55/36*	63/56*
Geber 2016	1845–1852CE	Ireland	Kilkenny Union Workhouse	2.0%** (C) N=345	NA	NA

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Gowland et al. 2018	c.1711–1857CE	England	Coach Lane, North Shields	15%** (T) N=81 n=56	NA	NA
	Predominantly 19th century	England	Fewstone, North Yorkshire	25%** (T) N=50 n=47	NA	NA
Gregg et al. 1981	Mid-14th century	USA	Crow Creek Site, South Dakota	0.8%* (T) N=486 n=129	NA	NA
Krenz-Niedbała & Łukasik 2016a	Medieval (10th–14th centuries)	Poland	Cedynia	18.0%** (T) N=257 n=100	NA	NA
	Early-modern (14th–17th centuries)	Poland	Słaboszewo	7.1%** (T) N=178 n=28	NA	NA
Lewis 2002	950–1500CE	England	Wharram Percy	9%** (T) N=303 n=88	NA	NA

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Lewis 2002 continued	950–1550CE	England	St. Helen-on-the-Walls	17%** (T) N=200 n=35	NA	NA
	850–1100CE	England	Raunds Furrells	10%** (T) N=142 n=20	NA	NA
	1729–1859CE	England	Christ Church, Spitalfields	3%** (T) N=186 n=33	NA	NA
Lewis et al. 1995	Late medieval (until 14th–19th centuries)	England	Wharram Percy	39% (T) N=663 n=268	42% (T) n=113	43% (T) n=169
	Late medieval (1100–1600CE)	England	St. Helen-on-the-Walls	55% (T) N=1042 n=245	45% (T) n=169	37% (T) n=144
Merrett and Pfeiffer 2000	c.1440CE	Canada	Uxbridge Ossuary, Ontario	49.8%* (T) N=457 n=207 (n=114 adults)	(T) **Rem** 84.6% **Ab** 69.2% **RemNoAb** 30.8% n=13	(T) **Rem** 76.4% **Ab** 58.5% **RemNoAb** 17.7% n=17

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Panhuysen et al. 1997	Medieval (600–800CE)	Netherlands	Boschstraat, Maastricht	25.0% (T) N=54 n=28	45.2% (T) n=62	38.5% (T) n=39
	Medieval (450–950CE)	Netherlands	Servaas, Maastricht	43.3% (T) N=282 n=67		
	Medieval (1250–1600CE)	Netherlands	Nunnery, Maastricht	41.9% (T) N=99 n=31		
Purchase 2016	Early Neolithic (8000–6800BP)	Russia	Shamanka II, Siberia	70.7% (T) N=180 n=77	87.5% (T) n=24	77.1% (T) n=48
	Late Neolithic (6000/5800–5200BP) to Early Bronze Age (5200/5000–3400BP)	Russia	Lokomotiv and Ust'-Ida I, Siberia	40.4% (T) N=67 n=67	71.4% (T) n=7	66.7% (T) n=3
Roberts 2007	1550–1675CE	USA	Hardin Village, Kentucky	51.5% (T) N=? n=33	76.5% (T) n=17	25.0% (T) n=16

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Roberts 2007 continued	1500–1600CE	USA	Aleutian Islanders	42.9% (T) N=? n=35	50.0% (T) n=22	30.8% (T) n=13
	"Late Woodland" (800–1100CE)	USA	Bluff Mounds, Illinois	38.6% (T) N=? n=70	53.6% (T) n=28	28.6% (T) n=42
	"Archaic" (4570+/-75 years–350+/-60 years)	USA	Indian Knoll, Kentucky	38.5% (T) N=? n=96	40.4% (T) n=47	36.7% (T) n=49
	500–750CE	Sudan	S and R cemetery, Kulubnarti	21.8% (T) N=? n=101	25.0% (T) n=56	17.7% (T) n=45
	18th–19th centuries	England	Christchurch, Spitalfields	18.0% (T) N=? n=394	18.3% (T) n=202	17.7% (T) n=192
	Late 1500s–early 1800s CE	USA	Site in South Dakota	17.2% (T) N=? n=87	18.0% (T) n=39	16.7% (T) n=48

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Schultz et al. 2007	14th century	USA	Grasshopper Pueblo, Arizona	50.4%** (T) N=450 n=369	NA	NA
Shapland et al. 2015	Late medieval	England	Barton-upon-Humber	N=?* All female	26.4%* (T) n=34	NA
	Late medieval	England	St. Mary Spital, London	N=?* All female	20.2%* (T) n=84	NA
	Late medieval	England	St. Oswald's Priory, Gloucester	N=?* All female	30.0%* (T) n=10	NA
	Late medieval	England	St. Helen-on-the-Walls and Fishergate House, York	N=?* All female	46.6%* (T) n=15	NA
Van de Vijver et al. 2018	Late medieval (12th–14th centuries CE)	Belgium	St. Rombout's cemetery	20.4%* (T) N=106 n=49	23.5%* (T) n=34	13.2%* (T) n=38

(Table continued on next page)

Work Cited	Time Period	Region/ Country	Site(s)	MS Total	MS Female	MS Male
Van de Vijver et al. 2018 continued	Post-medieval (15th–16th centuries CE)	Belgium	St. Rombout's cemetery	5.4%* (T) N=77 n=37	23.5%* (T) n=34	13.2%* (T) n=38
	Post-medieval (17th–18th centuries CE)	Belgium	St. Rombout's cemetery	19.2%* (T) N=65 n=26		

Coronation Street

Two distinctive patterns in MS prevalence within the Coronation Street population emerged from the data in the present study: the rate of MS was significantly higher ($p=0.022$ in biological females (77.3%, 17/22) than males (38.5%, 5/13); and the rate remained high in all age groups (c. 76.0%). First, that there was significantly more MS amongst females than males was consistent with the rate of AM, and supported the observed trend that males, regardless of age, were more buffered from risk factors for URI than females.

Second, the MS infection rate at Coronation Street remained high in all age groups: 100.0% in young adults and decreasing to approximately 60.0% in mature and senior adults. This suggests either that MS was ubiquitous in this population, with most individuals suffering from an upper respiratory infection at some point in their lives, or that MS was predictive of non-survivorship. Due to selective mortality, the rate of MS likely over-estimates the prevalence of the disease amongst the living (Wood et al. 1992). So, MS may have been common, but not to the same extent as the prevalence rates suggest at face-value. Rather, since most of the new bone formation was active at the time of death, death was likely associated with those lifeways that placed an individual at risk of MS (Wood et al. 1992).

The rate of MS observed in Coronation Street was compared to the rate recorded by other studies which used the Boocock *et al.* (1995) criteria for classifying new bone formation. However, a flaw in this comparison was that the previous post-medieval studies only considered non-adults (Geber 2016; Gowland et al. 2018; Lewis 2002), while this study examined adults. Non-adults have under-developed immune systems and, so, are more susceptible to infection than adults (Simon et al. 2015). Thus, many likely died of acute infections that did not create pathological new bone. So, the prevalences reported in these studies likely under-represented the prevalence of MS amongst adults in the same populations. Since these studies involved non-adults, they also did not examine differences in biological sex. However, that more Coronation Street females than males had MS was consistent with studies from other periods that also used the same MS diagnostic criteria (Panhuysen et al. 1997; Roberts 2007). The dearth of information regarding the burden of MS amongst post-medieval adults made comparisons difficult and highlighted the need for more work into the effects of urbanization on upper respiratory health.

The population prevalence of MS recorded here (63.2%) is high compared to the rates reported by the previous studies (see Table 7.2). Seven studies recorded rates of MS over 50% and these were from a mix of geographic and temporal periods: late medieval Britain (Boocock et al. 1995), Neolithic to medieval Sudan (Davies Barrett 2018; Davies-Barrett et al.

2021b), Late Intermediate period Peru (Davies-Barrett et al. 2021a), *c.* 1440 Ontario, Canada (Merrett and Pfeiffer 2000), Middle Holocene Siberia, Russia (Purchase 2016), and 1550–1675 Kentucky, USA (Roberts 2007). Of those studies that examined post-medieval populations, regardless of diagnostic criteria, the average rate of MS varied from 2.0% (Geber 2016) to 15.0% (Gowland et al. 2018). While mindful of complications caused by the inclusion of non-adults in some of these studies, hidden heterogeneity of risk, and comparing sites too closely (Wood et al. 1992), it was noted that the environmental risk factors faced by the Coronation Street population did not appear to have been unique to the Industrial Period; however, the level of risk faced by the Coronation Street population appeared to have been extreme for the period.

7.4 Lower Respiratory Infection

This section discusses the prevalence of each bone type and the implications for the pathophysiology of LRI in both populations, possible sources of error within our diagnosis, and the laterality of the infections. In Black Gate, 11.1% (23/208) of individuals were diagnosed with LRI and, in Coronation Street, it was 31.8% (28/88) of individuals. In both populations, the most common form of visceral surface rib lesion (VSRL) observed were what Davies-Barrett *et al.* (2019) classed as porous remodeling lamellar bone (PRL), followed by a combination of *both* PRL and diffuse woven (DW) bone (classified has as 'Both'). DW alone was the least frequently observed form of VSRL in both populations.

This was the first project to follow the Davies-Barrett *et al.* (2019) method of classifying and recording VSRL, and Davies-Barrett *et al.* (2019) do not present the prevalence of each type of bone they observed; so, there is no literature to compare our prevalence of VSRL to. We can observe, however, that both Black Gate and Coronation Street had a similar crude prevalence of the three forms of (13.0%, 52.2%, and 34.8% in Black Gate and 14.3%, 57.1%, and 28.6% in Coronation Street for DW, PRL, and Both, respectively), despite a substantially higher prevalence rate of VSRL overall at Coronation Street—three times as many individuals died with each VSRL type in Coronation Street compared to Black Gate. This suggests that although LRI was more prevalent in Coronation Street than Black Gate, individuals from both populations experienced similar physiological changes as a result of LRI and were dying at similar points in the diseases' progression (e.g., DeWitte 2014a; DeWitte and Stojanowski 2015; Mays 2012, 2018; Snoddy et al. 2020; van Schaik et al. 2014; Wood et al. 1992).

LRI are often more persistent[28] and serious than URI (NHS 2018c), as they increase individual morbidity and mortality (Hrdlička 1908). So, unlike MS, LRI may be indicative of a cause, or an associated cause, of death. That most individuals with VSRL had remodelled lesions suggested that those with bony involvement had persistent LRI and were robust enough to survive the disease in its early forms. Those who died from early-stage LRI are likely invisible paleopathologically. So, while MS was often active at the time of death and indicative of frailty and morbidity, VSRL are likely representative of the slow worsening of health and increasing morbidity, ultimately leading to mortality.

Trauma or a chest wall tumor cannot be ruled out as the source of some of these VSRL (Davies-Barrett et al. 2019:534). Thus, the classification system of Davies-Barrett *et al*. (2019) made every effort to differentiate lesions indicative of LRI from those that were not (e.g., some taphonomy, hypertrophic osteoarthropathy, osteomyelitis, treponematosis, emphysema, and septicemia) (Anselmo et al. 2016; Aufderheide et al. 2002; Binder and Saad 2017; Lambert 2002; Roberts et al. 1994, 2016; Santos and Roberts 2001). So, we are confident that few false positives were recorded here. More likely, the prevalences recorded here were an underestimation of the true prevalence of LRI in the populations, as not all LRI affect the skeleton (Roberts et al. 1994). This is a limitation of palaeopathological research and one that cannot be overcome by current methods.

There was no significant difference in the prevalence of LRI on the left or right by rib section or rib cage section in either population. Laterality of VSRL has been observed to correlate with certain diseases. Specifically, tuberculosis (TB) occurs on left ribs twice as often as it appears on right ribs (Kelley and Micozzi 1984; Lambert 2002; Santos and Roberts 2006); actinomycosis infection has been documented (arguably, not rigorously) to affect the right ribs more frequently (Molto 1990); and lobar pneumonia stimulates unilateral VSRL on the side of the infection (Waldron 2009:117). Only five Black Gate individuals had VSRL only on their right ribs and six individuals had VSRL only on their left ribs. In Coronation Street, there were nine individuals who had VSRL on the right only and ten individuals with VSRL on the left only. These individuals may have had TB, actinomycosis, or lobar pneumonia. Previous osteological examinations have diagnosed a handful of TB cases in both populations: one Black Gate case in a probable male (Nolan 2010) and four probable Coronation Street cases in a female and three unsexed individuals (Raynor et al. 2011). There are likely more

[28] The term chronic is avoided when discussing LRI, since chronic LRI can refer clinically to non-infectious conditions such as emphysema and chronic obstructive pulmonary disease (Burney et al. 2017; WHO 2019).

cases of TB in both populations, as only 3–8% of cases of TB involve the skeleton (Ortner 2003; Ortner and Putschar 1985; Resnick and Niwayama 1995; Roberts and Buikstra 2003). However, since there was no significant difference in the affected side in either population, it appeared that the majority of the LRI could not be diagnosed specifically as TB, actinomycosis, or lobar pneumonia.

In general, the pattern of lesion distribution was different between populations. The proportion of ribs with VSRL increased from neck to shaft and remained relatively consistent throughout the rib cage in Black Gate individuals; while in Coronation Street individuals, the proportion of ribs with VSRL was relatively consistent from neck to shaft, and the upper ribs had VSRL half as often as the rest of the rib cage. TB is more likely to affect the rib head and neck and the middle ribs (Kelley and Micozzi 1984; Matos and Santos 2006; Roberts et al. 1994; Santos and Roberts 2006), while non-TB LRI often affect the sternal end of the rib shaft (Matos and Santos 2006). Finally, peritonitis is more likely to affect the lower ribs (Santos and Roberts 2006). The findings of the present study suggest that the populations were experiencing LRI of different etiologies. Taken with the rates of new bone formation, we suggested that the visceral rib surfaces were responding to inflammation in similar ways, despite each population facing different types of LRI that manifested in different parts of the lungs and/or involved different respiratory-related tissues; and that mortality was associated with LRI, especially that which resulted in PRL, particularly in Coronation Street.

The extensive variation in sided or sectioned LRI likely masked specific etiologies at the population level, making it difficult to diagnose specific infections without conducting individual examinations. However, specific diagnosis was not an objective of this project. Instead, this study focused on interpreting the prevalence of infection to inform the discussion surrounding the lifeways and health of the populations and the prevalence of mastoiditis.

Black Gate

Three distinctive patterns in LRI prevalence within the Black Gate population emerged from the data in the present study: more individuals from coffin graves (25.0%, 7/28) had LRI than individuals from elaborate/elaborate variation graves (11.8%, 2/17), or plain graves (7.9%, 12/152); there were marginally more males (14.4%, 15/104) than females (7.6%, 7/92) with LRI; and the prevalence in LRI increased slightly with age at death. First, few individuals were observable for VSRL from each grave type and body position in Black Gate. Despite this, an interesting pattern emerged when grave types were grouped together by style: plain, coffin, and elaborate/elaborate variations (including earmuffs, pillow stone, head box, chest, rubble cist, and cist graves). When this was done, it was observed that more individuals from

coffin graves (25.0%, 7/28) had LRI than individuals from elaborate/elaborate variation graves (11.8%, 2/17), or plain graves (7.9%, 12/152). The difference between the number of individuals with LRI in coffin and plain graves was significant (p=0.007).

Second, there was no significant difference in LRI prevalence at Black Gate between the biological sexes. In every group, the prevalence of infection was lower than 16%. Notably, all the active cases of VSRL (or diffuse woven) (100%, 3/3) and most of the mixed active and healing (or both) (87.5%, 7/8) were present in males. The slight difference between the sexes, and the difference in lesion type, may have indicated that some males, compared to females, were more exposed to risk factors for LRI and/or were more likely to survive acute LRI and have them become persistent. These risk factors were likely different to those which females were exposed (as reflected in the higher rate of AM and MS amongst females than males); or females and males were exposed to similar risk factors, but to different degrees.

Third, the slight but non-significant increase in LRI with age at death was consistent with the mortality-morbidity paradox, in which those who survived adulthood were the frailest, as their frailty and morbidity increased with every mortality-event they survived (Marklein et al. 2016; Wood et al. 1992). Thus, those who died as seniors were more robust throughout their lives than those who died before 45 years. This complemented the MS disease profile, which indicated that the frailest, especially stressed mothers, did not survive adulthood. The number of individuals with active and healing (or porous/remodelled/lamellar) VSRL generally followed this trend, with no one lesion type increasing with age at death.

Because few studies examine VSRL exclusively—especially in the early medieval period—there is little information to compare our LRI rate to directly (Table 7.3). The two other studies of Anglo-Saxon populations that recorded LRI by VSRL, recorded low, but comparable, rates of 8.3% and 1.4% (Boylston and Addingham 1991; Wiggins et al. 1993). More information was obtained by examining studies which diagnosed TB from VSRL and other skeletal indicators. A study by Pedersen *et al.* (2019) found 4.8% of individuals from a Danish medieval population had VSRL diagnostic of TB. When the rest of the skeleton was considered for diagnosis, the prevalence of TB increased to 17%. The prevalence was higher amongst the lower (29%) than higher classes (10%). Cooper *et al.* (2016) found 9.1% of (3 individuals from) an early medieval Swiss population had VSRL diagnostic of TB. When the entire skeleton was examined, 13 individuals were diagnosed with TB. Finally, in two early medieval populations from Austria, Teschler-Nicola *et al.* (2015) found 15.9% and 14.1% of individuals with VSRL diagnostic of TB. VSRL were noted in all ages and were more common in males than females

(21.3% and 11.4%, respectively). This suggested that LRI were common diseases in the Anglo-Saxon and later medieval periods; but not ones that affected every member of a population equally.

Table 7.3 Prevalence of lower respiratory infection in various populations as reported in previous studies. Highlighted works used the Davies-Barrett et al. (2019) methodology. Highlighted time periods are comparative to Black Gate or Coronation Street. LRI is lower respiratory infection. * refers to results that include non-adults. ** refers to results that only include non-adults. Works marked with an *** are cited in Roberts *et al.* 1998 and unavailable otherwise. C and T are crude and true prevalence rate, respectively. N is number of individual in the population. n is number of individual in the sample. RS is rib section. The results for Mariotti *et al.* (2015) are presented as Group 1 (G1), Group 2 (G2), and Group 3 (G3) or those with TB, pulmonary non-TB infection, and other diseases. The results for Nicklisch *et al.* (2012) are presented as Grade 1–4 (Gr1–4) and Grade 2–4 (Gr2–4), referring to the grade at which lesions were scored.

Work Cited	Time Period	Region/ Country	Site(s)	LRI Total	LRI Female	LRI Male
Aufderheid et al. 2002	350BCE–500CE	Chile	AZ-75 cemetery, Azapa Valley (sample)	100.00% (C) N=8	NA	NA
Boulter et al.* **	Post-medieval (18th–19th centuries)	England	Newcastle Infirmary at the Fourth, Newcastle-upon-Tyne	4.30% (?) N=?	NA	NA
Boylston and Addingham 1991*; Wiggins et al. 1993***	Anglo-Saxon (8th–10th centuries)	England	ACH90 and Castledyke, Barton-upon-Humber	8.3% (?) N=? 1.4% (?) N=?	NA	NA

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Boylston and Roberts 1996*; **Roberts 1989***	Roman (4th century CE)	England	Kempston, Bedfordshire and 76 Kingsholm, Gloucester	4.6% (?) N=? 19.4% (?) N=? n=	NA	NA
Chundun 1991*, **1992***	Late medieval (12th–16th centuries)	England	Chichester Medieval Hospital, Chichester and St. Giles Hospital, Brough	17.7% (?) N=? 22.9% (?) N=?	NA	NA
Cooper et al. 2016	Early medieval (610–670CE)	Switzerland	Courroux	9.1% (C) N=39	0% (T) n=17	9.1% (T) n=22
Davies-Barrett 2018	Kerma Classique period (1750–1500BCE)	Sudan	4-L-2	23.1% (C) N=26 4.50% (T) N=883 RS	42.9% (T) n=7	25.0% (T) n=12
	Kerma Classique period (1750–1500BCE)	Sudan	4-L-88	16.7% (C) N=6 5.9% (T) N= 237 RS	0% (T) n=1	0% (T) n=4

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Davies-Barrett 2018 continued	Kerma Classique period (1750–1500BCE)	Sudan	4-L-100	0% (C) N=2 0% (T) N= 63 RS	0% (T) n=0	0% (T) n=0
	Meroitic and Post-Meroitic (c.300BCE–600CE)	Sudan	3-Q-33	45.0% (C) N=20 4.4% (T) N= 1274 RS	50.0% (T) n=8	455% (T) n=11
	Post-Meroitic	Sudan	3-O-1	52.9% (C) N=17 6.9% (T) N= 859 RS	50.0% (T) n=2	60.0% (T) n=10
	Post-Meroitic	Sudan	4-M-53	42.9% (C) N=7 0.9% (T) N= 422 RS	100% (T) n=2	0% (T) n=4

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Davies-Barrett 2018 continued	Medieval	Sudan	3-J-23	43.9% (C) N=66 8.5% (T) N= 4568 RS	42.4% (T) n=33	50.0% (T) n=30
	Post-Meroitic or Early Christian periods	Sudan	3-J-18	24.8% (C) N=113 3.3% (T) N= 7670 RS	25.5% (T) n=51	22.4% (T) n=58
	Neolithic	Sudan	R12	0% (C) N=13 0% (T) N= 411 RS	0% (T) n=3	0% (T) n=8
	Kerma (2500–2050BCE)	Sudan	P37	22.6% (C) N=31 3.8% (T) N= 1774 RS	25.0% (T) n=6	29.4% (T) n=17

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Davies-Barrett 2018 continued	Meroitis (c.200BCE–200CE), Post-Meroitis (c.400–700CE), and Medieval (c.800–1200BCE)	Sudan	Gabati	27.3% (C) N=77 2.7% (T) N= 4439 RS	27.3% (T) n=33	27.0% (T) n=37
	Medieval	Sudan	Soba East	42.9% (C) N=28 7.6% (T) N= 1373 RS	80.0% (T) n=5	31.3% (T) n=16
Davies-Barrett et al. 2021	Late Intermediate Period (1000–1476CE)	Peru	Pachacamac	33.3% (C) N=39	21.4% (T) n=14	45.5% (T) n=22
Davies-Barrett et al. 2019	Medieval (c.500–1500CE)	Sudan	3-J-23	8.50% (T) N=66 n= 4568 RS	NA	NA
	Meroitic and Post-Meroitic (c.300BCE–600CE)	Sudan	3-Q-33	4.40% (T) N=20 n= 1274 RS	NA	NA

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Davies-Barrett et al. 2019 continued	Kerma Classique period (1750–1500BCE)	Sudan	4-L-2	4.50% (T) N=26 n= 883 RS	NA	NA
Kelley and Micozzi 1984	20th century	USA	Hammann-Todd Osteological Collection	16% (C) N=445	7.7% (T) n=65	8.9% (T) n=380
Lambert 2002	Prehistoric Puebloan (1075–1280CE)	USA	Ute Mountain (5MT8651, 5MT9541, 5MT9924, 5MT9942, 5MT9943, and 5MT10010), Colorado	32%* (T) N=32 (N=19 adults) n=25	41.7%* (T) n=12	12.5%* (T) n=8
Lewis 2016	Medieval (900–1550BCE)	England	104 urban sites (primary data, Archaeological Data Service, and (un)published reports)	4.8%* (T) N=4060 n=3983	7.7%* (T) n=728	5.3%* (T) n=1125
Lewis 2016	Medieval (900–1550BCE)	England	47 rural sites (primary data, Archaeological Data Service, and (un)published reports)	4.3%* (T) N=880 n=629	3.9%* (T) n=96	6.9%* (T) n=145

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Mariotti et al. 2015	Late 19th–early 20th century	Italy	Certosa cemetery of Bologna	57.6% (C) N=244 (T) **G1** 54.2%/1.7% n=59 **G2** 7.4%/0% n=27 **G3** 8.2%/1.5% n=134	(T) **G1** 76.7%/3.3% n=30 **G2** 0%/0% n=11 **G3** 8.2%/1.5% n=55	(T) **G1** 31.0%/0% n=29 **G2** 12.5%/0% n=16 **G3** 7.6%/2.5% n=79
Mays et al. 2002	Medieval (10th–16th centuries)	England	Wharram Percy	0.8% (T) N=360 n=3	NA	NA
Nicklisch et al. 2012	Linear Pottery Culture (5400–4800BCE)	Germany	Halberstadt, Derenburg, and Karsdorf in Saxony-Anhalt	35.1% (T) N=57	**Gr1–4** 35.5% (T) n=11 **Gr2–4** 16.1% (T) n=5	**Gr1–4** 37.5% (T) n=9 **Gr2–4** 29.2% (T) n=7

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Pedersen et al. 2019	Medieval	Denmark	Ribe	4.8% (C) N=497	NA	NA
Pedersen et al. 2019 continued	Early-modern	Denmark	Ribe	13.7% (C) N=216	NA	NA
Raff et al. 2006	Pre-colonial	USA	Schild population, Illinois	2.3% (C) N=217	NA	NA
Roberts et al. 1994	1910–1940	USA	Terry Collection	N=1718 **TB** 61.6% (T) n=255 **No TB** 15.2% (T) n=1086	NA	NA
Santos and Roberts 2001	1904–1936	Portugal	Coimbra Identified Skeletal Collection (sample)	N=66 **TB** 72.2%** (T) n=18 **No TB** 4.2%** (T) n=48	NA	NA

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Santos and Roberts 2006	1904–1936	Portugal	Coimbra Identified Skeletal Collection (sample)	N=263 **TB** 85.7% (T) n=81 **No TB** 17.8% (T) n=90	NA	NA
Teschler-Nicola et al. 2015	Early-modern	Austria	Gars/Thunau	15.9% (T) N=373* n=88	NA	NA
Van de Vijver et al. 2018	Late medieval (12th–14th centuries)	Belgium	St. Rombout's cemetery	13.30%* (T) N=212 n=98	10.2%* (T) n=59	25.0%* (T) n=88
	Late medieval (15th–16th centuries)	Belgium	St. Rombout's cemetery	38.50%* (T) N=212 n=65		
	Industrial (17th–18th centuries)	Belgium	St. Rombout's cemetery	26.50%* (T) N=212 n=49		
Western and Bekvalac 2020	Pre-Industrial	England	Various site in London	2.9%* (T) N=456	c.0–3%* (T) n=?	c.0–4%* (T) n=?

(Table continued on next page)

Work Cited	Time Period	Region/	Site(s)	LRI	LRI Female	LRI Male

		Country		Total		
Western and Bekvalac 2020 continued	Pre-Industrial	England	Various site outside London	1.6%* (T) N=572	c.2–3%* (T) n=?	c.0–2%* (T) n=?
	Industrial	England	Various sites in London	19.5%* (T) N=799	c.14%–21%* (T) n=?	c.18–24%* (T) n=?
	Industrial	England	Various sites outside London	19.2%* (T) N=526	c.0–4%* (T) n=?	c.0–3%* (T) n=?

Coronation Street

Two distinctive patterns in LRI prevalence within the Coronation Street population emerged from the data in the present study: there was slightly more LRI amongst males (35.7%, 15/42) than females (31.0%, 13/42); and the prevalence of the diseases remained similar amongst the age groups (average of 34.5%). First, marginally more males than females were diagnosed with LRI. Of those with LRI, more males (62.5%, 10/16) had remodelled lesions than females (37.5%, 6/16). That the differences in disease rate and lesion type were small between the biological sexes likely suggested that both were exposed to relatively similar risk factors for LRI, and/or exposed to risk factors for LRI to similar degrees. Since more females than males were diagnosed with AM and significantly more females than males were diagnosed with MS, the risk factors males and females were exposed to, or the length of time they were exposed, were likely different to some degree.

Second, contrary to the expectations of the mortality-morbidity paradox, the rate of infection did not decrease with age at death. Rather, the disease rate remained high across all ages at death and suggested that Coronation Street individuals were frail and dying from, or with, LRI at every age. A marginal increase in individuals diagnosed with LRI in the mature age group was reflective of the mortality profile of the population, as there was a peak in male deaths at this age and slightly more males also died with LRI. This is mirrored in the distribution of active and healing lesions by age at death, with most lesion types present across all ages at death and a slight peak at the mature age at death.

Three studies examined the rate of VSRL amongst post-medieval/Industrial populations (Table 7.3) (Boulter et al. 1998; Pendersen et al. 2019; Western and Bekvalac 2020:85–92). Boulter *et al.* (1998) noted 4.3% of post-medieval British individuals with VSRL diagnostic of LRI. The rate was similar amongst Industrial British populations from outside of London (average of 4.2%), and much higher amongst those who lived in London (average of 19.2% and peak of 25.5%) (Western and Bekvalac 2020:85–92). Finally, Pendersen *et al.* (2019) diagnosed 13.7% of individuals in an early-modern Danish population with VSRL indicative of TB. Our study reported a higher rate of LRI than previous studies. This may suggest that Coronation Street individuals were more exposed to risk factors than individuals from previously studied populations. These risk factors are discussed more in the rest of the chapter.

7.5 The Populations in Context

In the following two sections (7.5.1 and 7.5.2), the data for Black Gate and Coronation Street are discussed separately. This is to reflect the difference in temporal and cultural context

between the sites, which fundamentally impacted the physical and social environments experienced by the two populations. Key themes are explored in each section: housing, occupation, nutrition, and social status/class. In the section concerning Black Gate, the discussion begins with the theme of social status, as a lack of archaeological evidence means that a reconstruction of the living environment cannot be approached adequately from settlement evidence. Thus, the social context offers the most valuable means of establishing the context of the late Anglo-Saxon period. In contrast, in the section concerning Coronation Street, social class is discussed last, as the impact of class on other aspects of Industrial life is well documented, as a result, discussing the impact of class on the other themes creates a natural summary of the section. In the final sub-section (7.5.3), the chapter concludes by drawing together contextual observations by means of explaining differences in the health of the two populations.

7.5.1 Discussing Black Gate in Context

In this section, the results concerning the Black Gate population are contextualised within the environmental and sociocultural landscape of the later Anglo-Saxon/Saxo-Norman periods in England's north-east. Four key themes are discussed: social status, housing, occupation, and nutrition. Public health is explored by discussing exposure to respiratory-related disease risk factors in the physical and social environment.

Social Status

The present study found two patterns relating to the rate of mastoiditis and LRI that can be explored in the context of social status. First, individuals buried in elaborate and elaborate variation graves had a low prevalence of residual CM compared to individuals buried in other grave types. However, they had an average rate of AM. As a result, there was a 31.2% increase in infection between childhood and adulthood among individuals from elaborate/elaborate variation graves. In the wider population, there was only an average 14.9% rise between residual CM and AM. residual CM is a risk factor for AM (Sadé et al. 2006; Tos and Stangerup 1985) and the two co-occurred significantly in both Black Gate and Coronation Street. Either individuals buried in elaborate/elaborate variation graves were buffered from risk factors for mastoiditis as children; or perhaps this cohort were frailer than their contemporaries and fewer children from this sector of society who had childhood mastoiditis survived to be buried as adults in elaborate/elaborate variation graves, consequently lowering the number of adults with evidence of residual CM. As adults, those who survived childhood to be buried in elaborate/elaborate variation graves had similar rates of AM to individuals buried in other grave types, despite fewer adults of this cohort

having the primary risk factors for AM: primary hypocellularity. Thus, to die as adults with the same prevalence of AM as that seen in their contemporaries, who had higher rates of primary hypocellularity putting them at greater risk of AM, it is likely this cohort was exposed to more risk factors for mastoiditis as adults than their contemporaries.

If elaborate/elaborate variation graves can be taken as a proxy for the elite status of the individuals buried therein, the data obtained from the present study suggests that these elite individuals likely engaged in different activities to their contemporaries. These individuals may have been *ealdormen*, *thanes*, or high status *ceorls*. They may have engaged in the pursuits of those managing, rather than working, the land, such as travelling between jurisdictions, conducting meetings, and keeping records. Such activities may have placed them into contact with many people, while administrative activities would have been carried out indoors (Gameson 2011). If they were monks, communal living and working indoors, in rooms such as the *scriptoria*, could have placed them at similar risk (Gameson 2011). In both instances, there would have been a higher chance of community acquired transmission of AM causing pathogens, and individuals would have been at risk of irritating their respiratory systems with the smoke and particulate matter from fires and candles (Adane et al. 2020; Brownlee 2018; Bulkow et al. 2012; Dagvadorj et al. 2016; FIRS 2017; Gilani et al. 2012; London et al. 2018; Pieta et al. 2015; Rehfuess et al. 2013; Trevino 1996; Wonodi et al. 2012). In sum, elite children may have been less exposed to risk factors for mastoiditis than other children; or, they may have been frailer than other children, and those who experienced mastoiditis may have died before reaching adulthood. Additionally, the adoption of professional tasks may have placed elite adult individuals at greater risk of mastoiditis than their contemporaries and caused the sharp increase in the rate of residual CM to AM that was visible in individuals buried in elaborate graves.

Second, significantly more individuals diagnosed with LRI were buried in coffins than in plain graves. It should be noted that some plain graves may have included wooden coffins that rotted and became archaeologically invisible (Green 2018; Sullivan 2004; Swales 2018). Thus, "plain" may not accurately represent the true grave type of some individuals. As such, some of the individuals with LRI from plain graves may have, in fact, been from coffin graves, albeit ones where no archaeologically-durable metalwork was used in their construction. Assuming the distinction between the coffined and plain graves is meaningful, the disparity in disease rate was significant, and potentially important for a population in which only 11.1% of the total population had LRI. That a quarter of those buried in coffin graves may have died as a result of, or due to a complication relating to an LRI. This suggested that those buried in coffin graves were distinctive from the rest of the population in some way—especially from those buried in plain graves. Perhaps coffin graves represented individuals of an

intermediate social status, as Swales (2018) argued for individuals buried in elaborate variation graves. There is precedent for this distinction, as Sullivan (2004) determined that coffin graves represented individuals of an intermediate social status at the later Anglo-Saxon site of Gilbertine Priory of St. Andrew, Fishergate, York, due to the higher material demands of burying individuals in unfurnished graves *versus* graves that included a coffin. Thus, it could be that those Black Gate individuals buried in coffin graves were from a distinct intermediate status and they carried out tasks that were significantly different to those who were buried in plain graves.

Housing

Various allergens and other respiratory irritants would have been present in halls and sunken feature buildings. Smoke from heating fires likely contributed the most to indoor air pollution. While this collected in the eaves and, when the wind permitted, was able to escape through the thatch or specially-placed holes in the roof, it is probable that the levels of airborne pollution were still extremely high. A study of contemporary Danish Viking houses of comparable construction showed that, with a fire burning, internal air pollution levels were high. The estimated daily individual $PM_{2.5}$ exposure was calculated at 410 $\mu g/m^3$ (Christensen and Ryhl-Svendsen 2015). For comparison, the WHO sets their long-term pollution target at an individual daily exposure of 35 $\mu g/m^3$ (WHO 2020a) and women living in houses with open fires in modern South Asia are exposed to 53.2 $\mu g/m^3$ on average a day (Xue et al. 2021). This would suggest that Danish Viking-age houses—and, likely, their equivalents in the UK—were highly polluted environments which exceeded the WHO air pollution target by a factor of 8.5.

Exposure to air pollution is a risk factor for mastoiditis, MS, and LRI (Brook 2009; Brownlee 2018; Chonmaitree et al. 2008; Evans 1994; Gilani et al. 2012; London et al. 2018; Li et al. 2018; Pieta et al. 2015; Rehfuess et al. 2013; Trevino 1996; Uhari et al. 1996; Wonodi et al. 2012). The relative similarity in the prevalence of AM and MS in the biological sexes suggested that both were undertaking daily tasks that exposed them to a similar level of risk. Today, globally, women and children are disproportionately affected by indoor air pollution, as they generally spend more time indoors than men (WHO 2020e). This may also have been the case for the population buried at Black Gate and was consistent with the slightly higher rate of AM and MS in females compared to males from alternating age groups; however, that the difference between the biological sexes was insignificant suggested either that tasks were not heavily gendered or that the gendered tasks of adults used indoor and outdoor spaces similarly. Alternatively, indoor chores may have fallen to children (not studied here) and younger adults of either sex, due to the latter's high rate of MS.

In studies of MS that compared sinusitis rates amongst rural and urban sites, urban sites had consistently higher rates of sinusitis than rural sites (Boocock et al. 1995a,b; Lewis et al. 1995; Panhuysen et al. 1997; Roberts 2007). This was largely attributed to lower air quality from cooking, heating, and industrial fires, as well as to higher population densities more capable of transmitting community-acquired infections. The population rate of MS reported here was consistent with the rates from most rural or rural-urban sites (Boocock et al. 1995a,b; Panhuysen et al. 1997; Roberts 2007), which may suggest that the community Black Gate served was relatively small and non/moderately urbanized.

Occupation

Each of the agricultural and industrial occupations of the late Anglo-Saxon and Saxo-Norman periods carried with it a different risk of respiratory and respiratory-related disease. The generation of, and exposure to, allergens such as dust, chaff, pollen, mites, and animal dander would have irritated the respiratory systems of those undertaking agricultural tasks or processing cereal and cereal by-products (Armelagos and Barnes 1999; Boocock et al. 1995; Merrett and Pfeiffer 2000; Roberts 2007; WHO 2020e). Similarly, individuals working in smoky conditions, indoors or outdoors, while carrying out work such as forging and smithing (Adane et al. 2020; Bulkow et al. 2012; Dagvadorj et al. 2016; Rehfuess et al. 2013; FIRS 2017); or those exposed to other air pollutants, such as fine particulate matter from agriculture or ceramic production (Aufderheide et al. 2002; Davies-Barrett 2018), would have been at risk of URI and LRI, as air pollutants irritate and harm the tissues of the respiratory system (Brook 2009; Brownlee 2018; Chonmaitree et al. 2008; Evans 1994; Gilani et al. 2012; London et al. 2018; Li et al. 2018; Pieta et al. 2015; Rehfuess et al. 2013; Trevino 1996; Uhari et al. 1996; Wonodi et al. 2012).

The shift in agricultural focus towards cereal production also fuelled rapid population growth and urbanization, particularly around markets (Hamerow et al. 2019). Other studies have noted a health disparity between later Anglo-Saxon rural and urban populations (Addyman 1972; Hall and Kenward 2004) and a decrease in health into the later medieval period as urbanization increased (Lewis 2002a,b; Swales 2012:333–9). Heightened contact with people from a wider area would have increased the risk of contracting a community-acquired viral respiratory infection, which is, today, the primary cause of URI (Chonmaitree et al. 2016; NHS 2021; Ruuskanen et al. 2011; Wald 2011; Walker et al. 2013) and LRI amongst non-adults (Walker et al. 2013; Ruuskanen et al. 2011). Person-to-person transfer also helps spread bacterial infections, which are, today, the primary cause of LRI in adults (Shi et al. 2015; Montaner et al. 2018; NHS 2018c), especially when preventative hygiene practices, such as handwashing, are uncommon (Rehfuess et al. 2013). Community acquisition is

especially common today amongst children, and carers of children, in nurseries (Chonmaitree et al. 2016; Brook 2009; Wald 2011). This may account for the slight increase in female cases of AM and MS and for the decrease in MS with age at death; as females and younger adults with young families would have been more likely to have been around children who played together and, consequently, transferred pathogens.

Living in a cold climate is also a risk factor for developing rhinitis and sinusitis (Brook 2009; Cruz and Togias 2008; NHS 2020b) and while the Medieval Warm Period (c. 950–1250) spanned the last two centuries of burial at Black Gate (McKerracher 2018), the majority of the population were alive during a period of cool, wet climate (McKerracher 2018:5–6). With the fire burning, temperatures inside later Anglo-Saxon halls would have been warmer than atmospheric temperatures (Christensen and Ryhl-Svendsen 2015). However, most agricultural activities would have occurred outside, and many crafts would have required lighting that, before the invention of the electric light, was best obtained outside or by an open door or window. Thus, time spent indoors likely did not compensate for time spent fully or partially outside; and likely exacerbated other risk factors experienced by those in the population. Brimblecombe (2011:4) noted an increase in MS from the Bronze Age, Iron Age, Romano-British period, to the Anglo-Saxon period, which he partially attributed to the cold climate and, as a result, increased time spent indoors in smoky, less-well ventilated structures than those constructed during previous periods. A combination of the cold, domestic and industrial smoke exposure, agriculture, and urbanization may have contributed to the high level of AM and MS within the population as a whole.

Nutrition

Nutritional stress was unlikely to have been a strong contributor to hidden heterogeneity in risk within the Black Gate population, as a comparison between long bone growth and dental eruption failed to show evidence of non-adult growth stunting (Swales 2018). Additionally, there was no rickets or osteomalacia noted in the adult population (Table 4.5). Swales (2012) concluded that while the population consumed a diet typical of the late Anglo-Saxon period, in which cereals—specifically, breads—were the staple, it was varied enough to satisfy the population's nutritional requirements.

While weaning was dangerous for Black Gate infants, Black Gate weaning practices were roughly in-line with modern health guidelines set by UNICEF and the WHO (2018b), which specify that infants should begin receiving supplementary foods around six months and should be partially or fully weaned by two years. Some Black Gate individuals began weaning later than recommended and most were fully weaned earlier than ideal (Victoria et al. 2016; WHO 2018b). That most individuals were not breastfeed for as long as is

recommended meant that many missed out on a year of immunological support and adequate nutrition. This likely contributed to the incidences of mild physiological stress in some non-adults (Swales 2012) and may have left some individuals more at risk of respiratory-related diseases later in life (Armelagos and Barnes 1999; Cerini and Aldrovandi 2013; Chonmaitree et al. 2016; Dagvadorj et al. 2016; FIRS 2017; Gilani et al. 2012; Rehfuess et al. 2013; Rossi et al. 2007; Victoria et al. 2013; Wonodi et al. 2012).

7.5.2 Discussing Coronation Street in Context

In this section, the results concerning the Coronation Street population are contextualised within the environmental and sociocultural landscape of the Industrial Period in England's north-east. Four key themes are discussed: housing, occupation, nutrition, and social class. As with the previous section, the objective of this section is to characterise public health by exploring exposure to respiratory-related disease risk factors in the physical and social environment.

Housing

Industrial housing was typically cold, draughty, and smoky, and this likely contributed to the high rate of respiratory-related disease present in the Coronation Street population. Today, Britain has a high number of temperature-related deaths amongst people who cannot afford to heat their industrial era homes in winter (Aylin et al. 2001; Boardman 1991; Wilkinson et al. 2001). Indeed, in such homes, the average indoor temperature with heating is only 12°C when the atmospheric temperature is 0°C (DETR 1991). This would have suited the tastes of Victorian homeowners, for whom, one source records, temperatures as low as 12°C in sitting rooms and 4°C in bedrooms was "healthy" (Cowan 1974). To combat the cold in domestic environments, industrial era people dressed warmly both indoors and outdoors alike (Rudge 2012). While good for keeping core temperatures up, dressing warmly would not have buffered the upper respiratory system from the effects of the cold and damp. Rather, chronic cold and damp exposure would irritate the sinus lining and precipitate the onset of rhinitis (Brook 2009; Cruz and Togias 2008; NHS 2020b).

Domestic exposure to coal dust and toxins would have been ubiquitous amongst individuals living in a 18th and 19th century town or city (Boyd 2020:382; Brimblecombe 2011). Coal is a dirty fuel that, when burnt, releases particulate matter (including fine particulate matter), chemicals (e.g., sulphur dioxide, carbon dioxide, nitrogen oxide), and metallics (e.g., mercury, lead, and cadmium) (Beach and Hanlon 2017; McDuffie et al. 2021). These air pollutants are extremely hazardous to health (Beach and Hanlon 2017; McDuffie et al. 2021; Western and Bekvalac 2020:74; WHO 2018a). As a result of coal burning, individual mortality

risk from respiratory infections and cancers increased nationally (Western and Bekvalac 2020:74). For example, Beach and Hanlon (2017) found that exposure to coal smoke could account for a third of all infant deaths related to urbanization in English populations between 1850 and 1860.

Other studies have linked urbanization to poor respiratory-related health. In their study of medieval Danish populations, Primeau *et al.* (2018) noted that the rate of mastoiditis increased with urbanization. Krenz-Niedbała and Łukasik (2020:245–72) noticed a similar trend in the prevalence of MS amongst medieval to early modern Polish populations. Collins (2019) also partially attributed the high rate of URI (69.3% with OM, 56.3% sinusitis, and 29.9% with mastoiditis) she observed in medieval Icelandic populations to the risks presented by high population density. Finally, Western and Bekvalac (2020:85–92) noted that the rate of LRI was higher amongst industrial era individuals who lived in London than in their contemporaries who lived outside of London. Similarly, our study found higher rates of MS and LRI amongst the Coronation Street than Black Gate population. Intriguingly, both residual CM and AM were more prevalent in the Black Gate than Coronation Street population. While counter to the expectation of finding more respiratory-related disease amongst urban/urbanizing populations, the rates of mastoiditis found in both populations were consistent with those rates of mastoiditis and OM in other pre- and post-industrial populations (Collins 2019; Flohr and Schultz 2009a,b; Floreanova et al. 2020; Gregg et al. 1965; Krenz-Niedbała and Łukasik 2020). That other populations, globally and throughout history, have had high rates of mastoiditis (Flohr and Schultz 2009a,b; Floreanova et al. 2020; Gregg et al. 1965a,b) like those observed here, cautions that while urbanization does have an impact on respiratory-related health, respiratory infection is not unique to more urbanized or industrialized periods. Rather, individual factors, such as genetics (Song et al. 2017; Virapongse 1985), likely influence frailty and also increase individual risk independent of the environment.

Furthermore, the mid-18th century saw the increasing popularity of childcare in which children from multiple households were cared for by one woman and fashionably kept indoors (Newman 2016). Pathogens are more transmissible indoors compared to outdoors (e.g., the SARS-CoV-2/COVID-19 virus can remain aerosolised and airborne for hours in an unventilated room and can be inhaled by people over two meters from the source) (Tang et al. 2021). More critically, habitually mixing households would have increased the possibility of transferring community acquired diseases between households and increased the chances of diseases becoming epidemic, as children brought pathogens home to their parents who could then spread the pathogens further (Chonmaitree et al. 2016; Brook 2009; Wald 2011).

Finally, females usually have a stronger immune response than men, but this is decreased during pregnancy and menopause due to changes in hormone levels (Barth and Lange 2020; Taneja 2018). Thus, when females are physiologically stressed, as discussed above, pregnant, or menopausal, they are less able to fight infections, frailer, and at a greater risk of contracting infections. As a result, the aforementioned environmental and lifestyle risk factors would have been of greater risk to females at different points in their lives. Both AM and MS were more prevalent amongst Coronation Street females than males, and this is consistent with their time spent inside and in the company of children, especially if their children were in nursery or if they themselves ran a nursery. While females were likely more exposed to indoor, domestic risk factors than men, men would have also been exposed to them—only to a lesser extent. The environment outside of the home would also have carried risk and have affected everyone. In the Industrial Period, urban density and air pollution were particular problems

Occupation

In the Coronation Street population, the higher rate of LRI amongst males compared to females is likely related to differences in occupation-related exposure to risk factors, as this represents a stark difference in lifeways, and exposure to risk, between the biological sexes of the period. In South Shields, the majority of the occupations were in industries that produced air pollutants (Kelly's Directory of Durham 1890; Simpson 2017), such shipping and mining. In describing St. Hilda's Colliery, one newspaper article from 1839 states, "the mine was worked by candles and blasting was used. Davy lamps were used only where the coal was softer. There were about one hundred men and boys in the mine (that day)" (NMRS 2021). The dust from various industries were known to coat every surface in a workplace—including the people—and created "dust coloured workers" (Western and Bekvalac 2020:81). Such working conditions would have exposed colliers to community-acquired respiratory diseases causing URI (Chadha and Chadha 2007; Bocian 1993; Torpy 2009) and LRI (FIRS 2017); and damp, fine particulate matter (such as coal dust), and toxins from mining (such as firedamp), exploding dynamite, and burning candles and lamps (NMRS 2021) would have irritated the lining of the sinuses and lungs and increased the risk of MS (Brownlee 2018; London et al. 2018; Pieta et al. 2015; Trevino 1996) and LRI (Adane et al. 2020; Bulkow et al. 2012; Dagvadorj et al. 2016; Gilani et al. 2012; Rehfuess et al. 2013; FIRS 2017; Wonodi et al. 2012). Firedamp refers to a mixture of flammable chemicals that can leach out of stone during mining. These chemicals can be ignited and, consequently, ignite the small coal and cause an explosion. After a firedamp explosion at St. Hilda's Colliery in 1839, 51 men and boys reportedly died from "after damp" or poisoning from toxins such as carbon monoxide (NMRS

2021; Wikipedia 2021). These individuals were buried at St. Hilda's Church and may have been studied as a part of this project (Co-Curate 2021).

The high rate of respiratory-related disease, especially active MS, in both biological sexes and across all ages at death in the Coronation Street population underscores the severe problem poor outdoor air quality likely was for the residents of industrial South Shields and suggests, as today, that poor air quality was significantly associated with mortality (Sunyer 2001; Western and Bekvalac 2020:73–4; WHO 2021). For the sinuses, breathing fine particulate matter, toxins, and metallics results in inflammation, edema, swelling, the blockage of the ostium, and, ultimately, acute or chronic sinusitis (Brownlee 2018; London et al. 2018; Pieta et al. 2015; Trevino 1996). Since MS is a risk factor for mastoiditis, the inflammation of the sinuses can result in inflammation of the Eustachian tube, the middle ear, and MAC (Sadé 1992; Todd 1994 in Sistani et al. 2019). Finally, for the LRI, the inhalation of particulate matter—especially fine particulate matter—toxins, and other allergens can cause, or aggravate, respiratory diseases (Adane et al. 2020; Bulkow et al. 2012; Dagvadorj et al. 2016; Gilani et al. 2012; Rehfuess et al. 2013; FIRS 2017; Wonodi et al. 2012) and today, often results in pneumonia (WHO 2020c). Air pollution can be particularly dangerous for individuals with cardiovascular disease (BHF 2021) or respiratory-related diseases, such as allergies (Armelagos and Barnes 1999; Trevino 1996) or chronic obstructive pulmonary disease (Jiang et al. 2016; Sunyer 2001), as it exacerbates those pre-existing conditions. The inhalation of fine particulate matter can also lead to type 2 diabetes (Esposito et al. 2016: He et al. 2017), cardiovascular disease (BHF 2021), cancer (CRUK 2019; Turner et al. 2020), and miscarriage (Xue et al. 2021). Finally, while females were exposed to more risk factors for MS, both males and females appeared to have been similarly exposed to risk factors for mastoiditis and LRI that resulted in similar rates of infection between the sexes. While factors such as over crowding and domestic and outdoor air pollution would have affected everyone to differing degrees (Gilani et al. 2012; Rehfuess et al. 2013; FIRS 2017; Wonodi et al. 2012), males would have been more exposed to work-place related toxins and other pollutants (Boyd 2020:382) and many women would have been more chronically exposed to domestic risk factors (Boyd 2020:382) such as cold and damp, air pollution, and over crowding compared to men.

Nutrition

Nutrition may have played a large role in the frailty and morbidity of Coronation Street individuals, especially as it related to access to vegetables, preferential feeding, and the duration of breast feeding. Differences in nutrition may have contributed to the slightly higher rate of AM and significantly higher rate of MS amongst females and may reflect one or more

gendered risk factors which were severe enough to have overwhelmed the strong, compared to male, female immune system (Klein 2000), and contributed to female frailty and morbidity.

Dietary stress, for example, was especially high in non-adults between the ages of one and five years, when height deviated from that seen in non-adults from contemporary sites (Newman 2016:194). UNICEF and the WHO now recommend exclusive breastfeeding for the first six months of a child's life (WHO 2018b). As this was often not practiced in the Industrial era, mothers/wetnurses were unknowingly unable to protect children from infection *via* secondary immunity (Armelagos and Barnes 1999; Cunningham 1995; Katzenberg et al. 1996) or to prime children's immune systems for the environment in which they lived (Riskin et al. 2012). Thus, children weaned early would have been immunologically unprepared to fight infection.

Additionally, Vitamins C and D in particular are known to play a role in, among other things, proper immune function (Brickley and Ives 2008; Popovitch et al. 2009), and these vitamins were noted as insufficient amongst Coronation Street mothers (Newman 2016:176–247). Weaning formula and weaning foods (such as water or cow's milk with flour or bread) lacked sufficient vitamins and minerals (Drummond and Wilbraham 1994) or contained them in forms that could not be easily absorbed by an infant (Lewis 2002). So, those infants weaned early using formula or other weaning foods were often malnourished and under protected. Furthermore, it was believed in this period that vegetables were dangerous to children's digestive systems, so few children ate them even if the family could afford them (Drummond and Wilbraham 1994). Similarly, approximately 90% of individual's Vitamin D requirements come from solar radiation (Hosseinpanah et al. 2010). However, air pollution has been found to block ultraviolet B radiation, which promotes synthesis of Vitamin D—significantly and independently contributing to Vitamin D deficiency (Hosseinpanah et al. 2010; Western and Bekvalac 2020:73). The thick smoke noted in many industrial towns and cities often allowed only a dim sunlight to permeate through the pollution (Western and Bekvalac 2020:81). As the author of the above letter to the editor complained, late 19th century South Shields was highly smoky, especially in the neighbourhoods inhabited by the poor (Shields Daily Gazette 1885). Furthermore, children were often kept indoors, as the sun was believed to be dangerous to their eyes (Drummond and Wilbraham 1994). Thus, nutritional deficiencies may have been compounded by artificial and social environmental factors which increased the disease risk of the Coronation Street population.

Class

Class played a meaningful role in the respiratory health of Industrial era individuals (Western and Bekvalac 2020:86–8). Though, there was no archaeologically distinctive class hierarchy observable amongst the Coronation Street graves, so no meaningful analysis could be made comparing individual disease rates to grave type. However, a discussion can be had regarding class exposure to risk factors for respiratory-related disease. This final section discusses housing, occupation, nutrition, and tobacco smoking, as they relate to class. Emphasis is placed on the experiences of the South Shields poor, as their life histories were the most negatively impacted by class distinctions.

First, communal and high-density living disproportionately affected the poor and increased the risk of transmitting community acquired respiratory infections (Davenport 2020). A study by Western and Bekvalac (2020:85–92) of 1028 pre-Industrial and 1325 Industrial British individuals highlighted that the prevalence of individuals with VSRL increased after industrialization (Western and Bekvalec 2020:85–92). Individuals from both outside and inside London were examined and the prevalence of infection did not vary overly between inner- and outer-city individuals pre-industrialization (2.9% and 1.6%, respectively); but it varied greatly post-industrialization (19.2% and 4.2%, respectively). Most notably, the rate of VSRL varied by status (regardless of sex), with individuals from lower status, high density neighbourhoods three times more likely to have LRI than individuals from higher status neighbourhoods (25.5% and 9.0%, respectively).

The prevalence of individuals with LRI found in this study (31.8%) is slightly higher than the prevalence of individuals with LRI in the most affected, lower status population examined from London (Bethnal Greet at 27.4%) (Western and Bekvalec 2020:86). Bethnal Green was one of the highest-density, slum neighbourhoods in London, with most of its residents engaged in market gardening, shop keeping, brick manufacturing, or silk weaving (BHO 1998). In contrast, the population of South Shields was engaged in heavy industry (Kelly's Directory of Durham 1890; Simpson 2017). Thus, chronic smoke exposure amongst the Coronation Street population in addition to high-density living, as experienced by the population of Bethnal Green and the poor of South Shields, may account for the difference in LRI prevalence between the two populations.

Second, in addition to the physical demands of Industrial era labour, exposure to crowded working environments, as well as industrial particulate matter, toxins, and other allergens would have increased a poor labourers' risk of developing a respiratory-related disease (Brook 2009; Brownlee 2018; Chonmaitree et al. 2008; Evans 1994; Gilani et al. 2012; London et al. 2018; Li et al. 2018; Pieta et al. 2015; Rehfuess et al. 2013; Trevino 1996; Uhari et al. 1996; Wonodi et al. 2012). Sharpe (2012) argues that the poor labourer's inability to

financially afford to take adequate time off of work to recover from a respiratory disease would have not only resulted in a high level of physiological stress and, possibly, growth stunting (if the labourer was a non-adult), but also, further damage to an individual's respiratory systems, leaving them more susceptible to future episodes of respiratory-related disease.

Third, class dictated food security (Clayton and Rowbotham 2008; Horrell and Oxley 2012; Roberts and Cox 2003) and influenced breastfeeding practices (Henderson et al. 2014; Newman and Gowland 2016; Newman 2016:195–6). The level of access each class had to adequate nutrition contributed to their morbidity and development. Compounding this, fashionable social practices regarding breastfeeding and childcare (Henderson et al. 2014; Newman and Gowland 2016; Newman 2016; Perkin 1993; Stevens et al. 2009), and physical obstacles such as air pollution levels (Hosseinpanah et al. 2010; Shields Daily Gazette 1885; Western and Bekvalac 2020:73), placed barriers in the way of proper development, including the development of the immune system.

Fourth, increasingly popular habits, such as tobacco smoking, placed individuals, especially men, and often those from the working class, at great risk of MS and LRI. Being exposed to cigarette and pipe smoke is a substantial risk factor for both sinusitis (Brook 2009; Slavin 1988) and LRI (Adane et al. 2020; Bulkow et al. 2012; Dagvadorj et al. 2016; Rehfuess et al. 2013; FIRS 2017), and second-hand smoke carries a similar risk for MS and LRI as first-hand smoke (NHS 2018). While smoking, and breathing tobacco smoke, has not been found to increase mortality, it does increase morbidity by exacerbating pre-existing respiratory conditions and placing individuals at a greater risk of contracting respiratory-related diseases (Walker and Henderson 2010; Western and Bekvalec 2020:92–6). The degree to which non-smoking women and children would have been exposed to, and affected by, tobacco smoking depends on the habits of those smoking. Until the 1850s, tobacco smoking remained a heavily gendered habit, though more women from the poorer than upper classes likely smoked and/or were exposed to second-hand smoke in the home.

Differences in occupation and tobacco smoking may have accounted for the slight increase in Coronation Street males compared to females with LRI. This also would have accounted for the slight increase amongst mature adults, as more males than females died at mature age (Newman 2016; Raynor et al. 2011). The slight increase in young adults with LRI may represent the frailest adult members of the population dying young after (one of) their first bout(s) of LRI. Interactions between all the risk factors individuals were exposed to likely contributed to the rate of MS and LRI visible on their skeletons today. Perhaps the effects of second-hand smoke in the home increased female morbidity and susceptibility to domestic risk factors, significantly increasing their risk of MS compared to males. Alternatively,

increased exposure to industrial particulate matter, toxins, and other allergens amongst working males may have compounded with tobacco smoking to have placed males at a slightly higher risk of LRI compared to females.

It is probable that many—if not most—of the Coronation Street individuals with respiratory-related disease were from the lower classes. For example, the decrease in residual CM amongst those who died as seniors may have reflected the positive survivorship effect of not suffering from mastoiditis as a child and, concomitantly, those least exposed to risk factors for residual CM—likely, those from the upper and upper-middle classes. Indeed, the poor appear to have been exposed to a higher risk level for respiratory-related disease than those from any other class. Thus, the rate of residual CM may reflect morbidity and mortality and may be highly reflective of the frailty of the most vulnerable members of the population. The rate of AM, MS, and LRI have also illustrated differences in individual exposure to domestic, occupational, and nutritional risk factors amongst those from different biological sexes, ages at death, and classes.

7.5.3 Comparing Population Health and Contextual Observations

The following section builds on the previous sections to explicitly discuss differences between the prevalence of mastoiditis, MS, and LRI in the Black Gate and Coronation Street populations to highlight critical differences between the frailty and morbidity, potential exposure to risk factors, and lifeways of individuals from each population.

There was no significant difference in the rate of residual CM between the Black Gate and Coronation Street populations. Despite this, residual CM was slightly more common amongst Coronation Street than Black Gate individuals, corresponding to, but less pronounced than, the trend noted in the rates of MS and LRI. However, an opposite trend was observed in the rate of AM between the sites: more Black Gate than Coronation Street individuals had AM. This may have been due to the fact that more Black Gate than Coronation Street individuals with primary hypocellularity survived until young or prime age. As a result, more Black Gate adults were at risk of AM than Coronation Street adults, as residual CM is a risk factor for AM. The correlation between residual CM and AM was corroborated in both populations by residual CM and AM significantly co-occurring, and by residual CM being included in both minimally adequate models for AM. While this could not have accounted for all the instances of AM in Black Gate, it may partly explain the discrepancy between population prevalences.

From a life history perspective, the rate of mastoiditis amongst survivors of childhood increased from residual CM to AM for all biological sexes from both populations, save for

Coronation Street males. Here, rather, the rate dropped slightly from residual CM to AM. In the Coronation Street population, MS was highly gendered, with significantly more females than males with MS. MS is a risk factor for AM and they share a similar epidemiology and etiology (Wald 2011). Thus, the difference in exposure to risk factors evidenced by the rate of MS may be observable in the rate of AM. Coronation Street males may have either been exposed to fewer risk factors for AM and MS than females, or have been more likely to die from AM, MS, or a related disease, before the disease could affect the bone.

Black Gate individuals continued to present with more AM than Coronation Street individuals across the life course; and Coronation Street individuals continued to present with more residual CM than Black Gate individuals, save for in the young and prime age groups. The latter phenomenon may be due to more Coronation Street individuals with primary hypocellularity dying before adulthood. This also resulted in lower rates of AM amongst Coronation Street young and prime adults compared to Black Gate adults at the same ages at death. The high prevalence of Coronation Street individuals who died as mature adults with evidence of residual CM was likely due to the peak in male deaths at this age, in-line with the higher rate of residual CM than AM amongst Coronation Street males. Male deaths at mature adult age may reflect deaths of lower class labourers. Thus, those who worked the most physically demanding jobs appeared to have also been those who were the most exposed to risk factors of residual CM.

Further, individuals from both populations who died as seniors had low rates of residual CM and the highest rates of AM. This trend likely indicated that individuals who survived adulthood were less likely to have had residual CM and been exposed to risk factors for residual CM; and the most likely to have accumulated bony evidence of AM over their lifetimes. Despite the fact that individuals from Coronation Street, rather than Black Gate, were more physiologically stressed and, likely, more exposed to risk factors for respiratory-related diseases, individuals from both populations showed this pattern. We can conclude that residual CM increased individual morbidity (especially by increasing the chance of contracting AM) and that AM, like LRI in the Black Gate population, accumulated with age.

There was a significant difference in the rate of MS between the Black Gate and Coronation Street populations, with significantly more individuals from Coronation Street affected than from Black Gate. In both populations, females were affected more than males; Black Gate and Coronation Street males had similar rates of MS, while more Coronation Street than Black Gate females had MS. That MS prevalence was more gendered in Coronation Street than Black Gate suggested that individual exposure to risk in the former was also more strongly gendered. The various biological, cultural, and environmental factors that affected

Coronation Street females have already been explored above: namely, biological factors such as pregnancy, breastfeeding, and menopause (Barth and Lange 2020; Fildes 1995; Riskin et al. 2012; Taneja 2018; WHO 2018b); cultural factors such as spending more time with children (Chonmaitree et al. 2016; Brook 2009; Niederman and Krilov 2013; FIRS 2017; Wald 2011) and the preferential feeding of males (Gowland et al. 2018; Griffin 2018; Horrell and Oxley 2012; Reedy 2020); and environmental factors such as spending more time indoors and exposed to cold, drafts, and coal smoke, and (for the lower classes) in cramped tobacco-smoky (Hilton 2000; Western and Bekvalac 2020:92–3) housing with other people (Davenport 2020). Perhaps the daily lives and tasks/occupations of Black Gate individual were less gendered than those from Coronation Street; or, despite being gendered, Black Gate male and female lifestyles resulted in relatively similar risk for MS.

A higher prevalence of MS at Coronation Street compared to Black Gate was present at every stage of the life course; however, more individuals from both populations died at a young age with MS than at any other age. In both populations, this was believed to have been caused by the deaths of the frailest individuals in the population; the deaths of physiologically stressed young mothers; and/or younger individuals being exposed to more risk factors for MS. For the youngest individuals in this group, these risk factors may have been similar to those which placed females at greater risk of MS, as these individuals may have continued to spend more time with their mothers. However, those entering the work force or marrying likely were exposed to certain risk factors from MS for the first time; and those who died young reflect those who were frail and/or unable to adapt to their new environment.

There was also significant difference in the prevalence of LRI between the Black Gate and Coronation Street populations, with three times as many Coronation Street as Black Gate individuals presenting LRI; however, the relationship with sex and age was not significantly different between the populations. In both populations, males had marginally more LRI than females. This may reflect, among other things, the effects of testosterone on the immune system (Chonmaitree et al. 2008; Csákányi et al. 2012; Mathews 1988; Rye et al. 2011; Uhari et al. 1996) and/or gendered tasks/occupations which increased exposure to risk factors for LRI. However, that the difference between the sexes is marginal suggested either that LRI was not similarly influenced by gendered patterns in health as MS, or that LRI was influenced by different, less gendered, risk factors than MS.

The rate of LRI infection varied by age at death: the rate increased with age in the Black Gate population and remained relatively similar in the Coronation Street population. This difference may have reflected an accumulation of infection over individual's lifetimes in the Black Gate population, and a sustained high-level of LRI infection in the Coronation

Street population. Both MS and LRI, indicated a significant difference in health between the populations, with respiratory infection being more common amongst Coronation Street than Black Gate individuals.

7.6 Conclusions

By comparing the prevalence rates of mastoiditis, MS, and LRI between the Black Gate and Coronation Street populations, this chapter has characterized the epidemiology and etiology of mastoiditis in both samples. Mastoiditis appeared to have been a common infection in the past, as it is now (NHS 2019a). Its prevalence varied widely by population; however, some trends appear to have been consistent, such as the risks factors for infection and its impact on morbidity. Others, such as the infection's relationship with biological sex, appeared to have been more culturally and/or temporally dependant. In the Black Gate population, the presence of residual CM and AM appeared to reflect the robusticity of the non-adult and adult population, respectively, and concomitantly, their morbidity. In the Coronation Street population, the rate of residual CM appeared to reflect the frailty of the non-adult population. Similarly, the rate of AM reflected the morbidity of the adult population and the presence of risk factors other than residual CM within the social and physical environment.

Further, the rate of mastoiditis recorded here was likely a more accurate reflection of individual exposure to environmental risk factors than MS and LRI, as the former generally affects the bone only during a chronic infection (Boocock 1995) and the latter only affects the bones of 3–8% of infected individuals (Ortner 2003; Ortner and Putschar 1985; Resnick and Niwayama 1995; Roberts and Buikstra 2003). Contrarily, subclinical mastoiditis can create pathological changes to the MAC (Flohr and Schultz 2009a,b). This may be one of the reasons why the rate of AM is almost 20% higher than that of MS, and approximately 33% higher than that of LRI. These differences may reflect acute infection and daily exposure to risk factors not recorded by MS or LRI. As such, true individual exposure to environmental risk factors may be higher that previously thought. Since the rate of MS and LRI in Black Gate and Coronation Street were consistent with those observed in other later Anglo-Saxon/Saxo-Norman and Industrial populations, respectively, this underestimation may be true in all populations.

More individuals from the Coronation Street than Black Gate population had lesions indicative of MS and LRI; and residual CM appeared to affect individual mortality and morbidity, while AM affected individual morbidity but did not particularly affect individual mortality. In this way, residual CM was similar to MS, which was often active at the time of

death and, therefore, likely related to cause of death. In the Black Gate population, AM was similar to LRI, as the number of individuals with evidence of each increased with age at death.

By contextualising the results within what is known, historically and archaeologically, about these two sites and periods, we have furthered the understanding of public health in the Black Gate and Coronation Street populations, and in the north-east of England over the last millennium. Black Gate appeared to have been a church cemetery that included all members of at least one local household. Individuals of a potential intermediate social status appeared to have been exposed to more risk factors for respiratory-related disease than those from other social status groups. The risks factors affecting Black Gate individuals seemed to have stemmed primarily from the environment (artificial and natural): the cool and wet climate, air pollution from agriculture and craft production, indoor smoke from cooking and heating fires, and increasing urbanization from the founding of towns. Individuals from Coronation Street were frailer and experienced higher morbidity than those from Black Gate. Their respiratory-related health was affected by similar risk factors to those which affected the Black Gate population—but at a larger scale. Industrialization, coal burning, and tobacco smoking meant that the air was polluted both within and outside the home; and rapid urbanization worsened urban planning, the quality of house construction, sanitation, and overcrowding. In addition, pronounced differences in lifestyle between social-economic groups and genders influenced every aspect of individuals' lives: from their place of residence, occupation, access to nutrition, and weaning practices. These further complications caused Coronation Street individuals to face risk factors which Black Gate individuals had not, such as insufficient breastfeeding, malnutrition, and vitamin deficiency.

Chapter 8
Conclusion

8.1 Introduction

This chapter reflects upon the success of this project. First, how the project's aim and objectives were accomplished is discussed along side a summary of the results. Next, the project's limitations are described. The discussion highlights how the national response to the SARS-CoV-2/COVID-19 pandemic created unexpected challenges for this project; and, ultimately, how these limited the scope and breadth of the project in unforeseen ways. In the subsequent chapter, two recommendations for future research are outlined. Last, the project's contributions to the field of archaeology are discussed and the thesis is concluded.

8.2 How the Aim and Objectives Were Achieved

The aim of this project was to develop and test a new method of diagnosing mastoiditis in human skeletal remains that was grounded in modern clinical practices, non-destructive, and accessible; and, in doing so, to expand our understanding of the epidemiology and etiology of mastoiditis with reference to the relationships between mastoiditis and both frailty and environmental risk factors. The aim was grounded in four practical objectives that served as the framework for this study. This section describes how each of the objectives was reached, to illustrate how the aim was successfully achieved.

1. To develop a non-destructive method of imaging the mastoid processes of skeletal individuals that was more accessible than wall-mounted X-ray and CT.

The first objective was achieved by conducting a preliminary analysis during which the imaging and diagnosing method developed during my Master's research was refined. Three imaging planes were tested for the clarity and diagnostic capacity of the radiographs they produced. Following consultation with neuroradiologist Dr. Charles Romanowski and otologist neurotologist Dr. Jaydip Ray, and an extensive review of the literature, it was decided that only two of the imaging planes were clear and diagnostic: medial-lateral and anterior-posterior. Romanowski also made further recommendations for the positioning of the sensor, X-ray generator, and temporal bone, to increase the replicability of the method and the clarity of the radiographs (21 November 2019 pers. comm.). These were to keep the sensor in a fixed position on the table throughout imaging; to place the mastoid process as close to the face of the sensor as possible; to create a theoretical line on the temporal bone to aid in the consistent alignment of the bone with the face of the sensor; to angle the temporal bone in each plane, such that the subsequent radiograph of the mastoid process is unobscured by other anatomical features; and to take X-rays a consistent distance from the sensor.

The work of Flohr *et al.* (Flohr et al. 2009, 2017, 2019; Flohr and Schultz 2009a,b) informed the classification of the mastoid air cell (MAC) morphology and the diagnosis of the disease state. Hypercellular, primary hypocellular, secondary hypocellular, and lytic and secondarily hypocellular MAC were defined. As a result, residual childhood mastoiditis (residual CM) and adult mastoiditis (AM) can be diagnosed using this method. Currently, no other method of diagnosing mastoiditis in archaeological human remains is non-destructive and as accessible as this method.

2. To characterize the epidemiology of mastoiditis in two temporally distinct samples of human skeletal remains to better understand who the disease affected, and the frailty impact that residual CM had on adults.

Both the prevalence and distribution of residual CM and AM were defined within the Black Gate and Coronation Street populations by imaging the temporal bone(s) of every adult (16+ years) individual and diagnosing the radiographs this produced. The prevalence of disease was explored across biological sexes and age at death, and in the Black Gate population, by perceived social status as reflected in grave type. The rate of mastoiditis within the populations was also compared to the rate of maxillary sinusitis (MS) and lower respiratory infection (LRI) therein. In the Black Gate population, the presence of residual CM

and AM appeared to reflect the robusticity of the non-adult and adult population, respectively, and concomitantly, their morbidity, as residual CM remained relatively consistent by age at death and AM increased with age at death. Therefore, adult individuals with residual CM did not appear to be at a higher risk of death as younger adults than as older adults, and AM did not appear to have increased individual risk of mortality. In the Coronation Street population, the rate of residual CM appeared to reflect the frailty of the non-adult population, as there were fewer individuals with residual CM in the younger age groups, which suggested that many of the non-adults who had mastoiditis had died before reaching adulthood. As a result, the rate of AM reflected the presence of risk factors other than residual CM within the social and physical environment as well as the morbidity of the adult population. In both populations, residual CM and AM co-occurred significantly, which suggested that residual CM was a risk factor for AM in the past as it is now.

3. To characterize the etiology of mastoiditis in the populations in relation to two respiratory infections (evidenced by lesions indicative of MS and LRI), to better understand its coincidence with other forms of respiratory-related disease and identify the environmental factors that may have placed individuals at risk of these infections.

The cause of the mastoiditis diagnosed in the populations was characterized by analysing the prevalence and co-occurrence of residual CM, AM, MS, and LRI within the populations and the groups in the populations. In the Black Gate population, a slight increase in AM and MS amongst females and young adults compared to males and those of other ages, may suggest that gendered activities placed females (particularly women of childbearing age) at greater risk of respiratory-related disease than males and older females. Gendered activities, such as cooking and spinning, may have taken place indoors, where there was poor ventilation and smoke from cooking and heating fires. This may suggest that some of the mastoiditis and MS experienced by women of childbearing age were community-acquired and exacerbated by smoke inhalation. Additionally, LRI—and active VSRL—were more common in males. Males were likely involved in industry and craft production more than females and may have carried out the majority of the daily agricultural tasks. As such, some of the respiratory-related diseases experienced by males may have been associated with community-acquisition (amongst those who worked communally) or may have been triggered by the inhalation of air pollutants associated with these occupations, such as dust and animal dander. Because much of the work requiring decent lighting would have been undertaken outdoors, the cool, damp climate may have also been responsible for many of

the respiratory-related disease recorded in the population. Finally, because MS and residual CM were predictive of AM in a regression analysis, this suggested that some of the cases of AM were secondary to upper respiratory infections or complicated by residual CM.

In the Coronation Street population, the significant difference in MS prevalence between females and males suggested that exposure to risk factors for MS was heavily gendered. Since the north-east had the lowest female employment rate in the country in this period, this suggested that many of these risk factors confronted by females were domestic. Factors such as exposure to coal smoke, drafty houses, and children (who may have been exposed to other children) would have affected all females, while other factors such as exposure to tobacco smoke, over-crowding, and poor sanitation would have especially affected the poorer residents. As a result, upper respiratory infection amongst females was likely to have been community-acquired and/or exacerbated by air pollution and the cold. The difference in LRI prevalence between males and females was not significant, but also suggested that some males were exposed to different risk factors than some females. These factors were likely related to occupation (which, in South Shields, was predominantly heavy industry and manufacturing) or gendered habits (such as tobacco smoking). As with MS, the cause of many of these LRI was likely community-acquisition or may have been caused/exacerbated by exposure to industrial pollutants. residual CM was also predictive of AM in the Coronation Street population. Thus, some of the adult infections diagnosed in the population may have been associated with individuals' inability to buffer against further infection.

In both populations, the etiology of mastoiditis was likely tied to community-acquisition and/or exposure to other natural, artificial, or social factors which either aggravated the respiratory system or made the individual frailer. For example, in the Coronation Street population, food insecurity and inadequate breastfeeding may have made the poor of South Shields frailer than their contemporaries, and frailer than those from Black Gate. Thus, it is likely they would have been the least capable of fighting and surviving respiratory-related diseases. While the specific cause of an infection cannot be determined in archaeological populations, risk *can* be characterized. This project has characterized risk in late Anglo-Saxon/Saxo-Norman Newcastle-upon-Tyne and in Industrial South Shields, and has hypothesized the etiologies of the mastoiditis, MS, and LRI experienced in both populations.

4. To better understand the lifeways of those living with respiratory infections in the broader context of public health.

While largely controlling for regionality, the health effects of the transition from farming to industrialization were explored within the late Anglo-Saxon/Saxo-Norman Black Gate population and the Coronation Street Industrial population by evaluating the presence of residual CM, AM, MS, and LRI. By contextualising the results within what was known archaeologically and historically about these populations, and other contemporaneous populations, environmental factors were identified which likely influenced the population's level of risk for respiratory-related disease; and the ways of life of those who inhabited these sites were better understood.

It was concluded that the Black Gate and Coronation Street populations experienced similar levels of risk, despite facing different types of environmental factors. The Black Gate population faced respiratory health challenges caused by domestic, agricultural, and industrial air pollution, and increasing urbanization from the founding of towns. In general, there were few indicators that risk was gendered or associated with certain age groups; however, differences in the prevalence of residual CM and LRI amongst those buried in elaborate/elaborate variation graves and coffin graves, respectively, may have indicated that social status played a role in dictating individual lifeways and, therefore, influenced individual exposure to risk factors for respiratory-related disease in late Anglo-Saxon/Saxo-Norman Newcastle-upon-Tyne. However, the relative consistency of residual CM, AM, MS, and LRI amongst the ages at death and biological sexes suggested that there was little difference in the frailty of individuals. The only significant differences were between potential groups of different social statuses. As such, public health appeared to have been relatively consistent, in which members of individual households had similar access to resources and occupied relatively similar environments.

In Industrial South Shields, however, public health appeared to have been poor. Industrialization and rapid urbanization increased the severity and ubiquity of risk compared to the Black Gate population, especially for the poorest residents, and resulted in increased frailty and morbidity. In particular, domestic and industrial coal burning; pollution from heavy industry and manufacturing; and cold, draughty, overcrowded, and high-density housing placed individuals at a higher risk of developing respiratory-related disease. Individual risk was further complicated by gendered and classed differences in housing, occupation, nutrition access, and tobacco smoking habits. Significant/large differences in respiratory-related disease between the biological sexes suggested access to some risky environments were limited by gender. Similarly, that it was common for individuals to die with active MS suggested that MS was related to mortality and that the population was frail. Classism and sexism are likely to have contributed greatly to individual frailty and population morbidity; and the ubiquity and severity of some risk factors, such as air pollution, likely placed all

individuals at great risk of respiratory-related disease. In sum, the prevalence of respiratory-related disease within these two populations communicated much in terms of people's way of life, the hardships they faced, and their adaptations to their environment.

8.3 Limitations of this Study

As with any study, there were limitations to this project that must be addressed. This section describes these limitations and what was done, if possible, to reduce their impact on the success of the project. These limitations are addressed from the most general to the most specific.

The problems of demographic nonstationarity, selective mortality, and hidden heterogeneity of risk, as outlined in the Osteological Paradox (Wood et al. 1992), were dealt with throughout this project to varying degrees. The problem of demographic nonstationarity was less applicable to this project than the other two problems, as this project did not deal explicitly with demography. Selective mortality and hidden heterogeneity of risk were dealt with by limiting how often the rate of infection within the Black Gate and Coronation Street populations were compared to the rate of infection within other populations. When these discussions inevitably occurred, the attempt was to compare the levels of risk and less to directly correlate the true prevalence of disease within each population. These problems were also dealt with by qualifying discussions concerning the rate of disease at each age at death so as not to assume that the rate of infection within each age group reflected the disease load of individuals at each age in life. Rather, individuals at each age at death were determined to reflect an aggregate of those who were the most physiologically stressed at each age over time. Thus, the rate of infection within age groups was seen to reflect the factors that stood in the way of health for individuals of each age over a large span of time. Notwithstanding, these issues likely permeated throughout this work due to the nature of the material being studied and, therefore, they must be noted here.

The accuracy of the diagnosis was limited by the mode of visualising the MAC—namely, using X-ray imaging. As a result, some of the details available through other forms of imaging were not available using this method. Of particular interest is the ability to visualise Howship's lacunae using SEM or thin-section microscopy (Flohr and Schultz 2009b). Identifying the presence of such features allows researchers to diagnose MAC enlargement as pathological rather than natural variation or taphonomy. Without the ability to corroborate a diagnosis using the macroscopic morphology of the MAC with microscopic indicators of pathological bone remodelling, there will remain a source of error in diagnosis. By heavily basing the diagnostic method created by this project on the bone types identified by Flohr

et al. (2009, 2017, 2019; Flohr and Schultz 2009a,b), who studied bone morphology macro-/microscopically, I believe the bone types defined here are reflective of specific disease states. I also believe that the accessibility and non-destructive nature of the method gives this method of imaging more mass appeal than SEM and thin-section microscopy.

The majority of the limitations faced by this project arose as a result of the UK government's response to the SARS-CoV-2/COVID-19 pandemic. The transition to virtual working at the University of Sheffield and the temporary closure of the Department of Archaeology facilities occurred in my second year, when I was collecting data. As a result, my project was limited to examining two populations. Originally, I had hoped to examine more, to widen the scope and power of my research. However, the Black Gate and Coronation Street populations were excellent collections to prioritise upon my return to the laboratory, as they are large, well-preserved populations and their analysis allowed me to examine differences in respiratory-related health over time, while controlling for regionality. Their excellent preservation also meant that most of the individuals were preserved adequately to be included in my mastoiditis study. It is regrettable that my sample size is not larger, although, I believe it was adequate to test my method and demonstrate its usefulness.

The temporary closure of university facilities and implementation of social distancing also meant that I had to cancel a replication study I had planned to conduct during 2020. It had been my intention to recruit a cohort of graduate archaeology students to volunteer to take part in a replication study in which they would replicate my method of imaging the mastoid process and diagnosing the subsequent radiographs. The goal was to examine if my method was replicable and to identify areas of weakness within my method and/or the description of my method that caused it to fail. The cancellation of the study is a limitation to the project overall, as the only person to have tested this method is me. It is my hope that the rigorous assessment of the method which followed the preliminary analysis identified and addressed many of the problems in the method, so that there were few problems remaining. I acknowledge that a replication study should be completed prior to the publication of the method.

Finally, and perhaps the most disappointing, was the cancellation of a CT study in partnership with Dr. Charles Romanowski and a local teaching hospital. It was my aim to create a method that was as diagnostic as CT and, to test this, I hoped to compare the radiographs obtained following my method to those obtained imaging the same individuals using CT as though they were patients today. CT is the standard clinical tool to image the temporal bone and diagnose mastoiditis and, as a result, is favoured by many archaeologists studying mastoiditis. The identification of hypercellular, primary hypocellular, secondary

hypocellular, and lytic lesions in the radiographs means that the method was able to produce radiographs in which the bone types diagnostic of residual CM, AM, and un-infected bone were identifiable. Thus, I believe the project was successful. However, the degree to which the project was successful could not be tested. I remain in contact with Dr Romanowski and he is still interested in collaborating with me on this study. It is my hope that this study can be carried out as a visiting researcher study once the pandemic-induced strain on the National Health Service has subsided.

8.4 Recommendations for Future Research

Some of the areas for future research relate to the parts of the project that were cancelled as a result of the SARS-CoV-2/COVID-19 pandemic: a replication study and CT study. These are not repeated here, as they are discussed in section 8.4. Two other areas for future research are outlined here. These were research questions that arose over the course of this project but were outside of the project's scope and, so, could not be investigated further.

The retention of primary hypocellularity throughout the life course has wider implications for the study of individual life history and the impact of early life stress on frailty. Thus, there is potential for a study that analyses the impact of childhood physiological stress on individual success, as measured by the rate of residual CM and other frailty markers, such as active sub-periosteal bone growth, cribra orbitalia, porotic hyperostosis, linear enamel hypoplasia, height, and bone density. A study such as this could illustrate the relationships between mastoiditis and early life physiological stress, and the impact of such stress later in life.

The impact of breastfeeding on respiratory health is discussed often in the clinical literature but is understudied archaeologically. A study concerning the relationship between insufficient breastfeeding, frailty markers, nutritional stress markers, and respiratory-related disease could shed light on the wider implications of this practice on morbidity and mortality, and cultural perceptions of health and the value of mothers' milk. This project has gone some way to exploring these relationships, but an independent study devoted to these questions could delve deeper.

8.5 Contributions to the Field

Despite how common mastoiditis is today, how severely the infection can impact individual quality of life, and the infection's relationship with respiratory health, mastoiditis is understudied archaeologically. The archaeological studies that have involved mastoiditis

have used destructive methods to visualise the MAC or have required the use of large, fixed imaging systems such as X-ray or CT. I identified this niche and, in this project, developed a method of imaging the MAC that is non-destructive and accessible, and which produces radiographs diagnostic of residual CM, AM, and MAC that have not been infected. As such, mastoiditis can now be studied widely, and those studies which use this method will be comparable to one another, as they will use the same imaging and diagnostic methodology. This has large implications for the field of archaeology, as the application of this method, and the wider study of mastoiditis in the past, will yield a wealth of information about community lifeways, morbidity, and mortality, and individual experiences with frailty and disability.

In conducting this research, this project has revealed three key points which require highlighting. First, mastoiditis likely provided a more accurate reflection of individual exposure to environmental risk factors than MS or LRI. MS and LRI only affect bone when chronic or in 3–8% of individuals, respectively (Boocock 1995; Ortner 2003; Ortner and Putschar 1985; Resnick and Niwayama 1995; Roberts and Buikstra 2003). Alternatively, mastoiditis affects bone in its subclinical, acute form (Flohr and Schultz 2009a,b). Thus, the presence of mastoiditis in a population is likely to provide a more accurate reflection of morbidity, mortality, exposure to risk factors, and adaptation to the environment than other forms of respiratory-related disease.

Second, this project has shown that a life history approach can be applied to the study of mastoiditis to reveal life-long trends in individual frailty and adaptation. Residual CM is a unique lesion; it is a permanent indicator of childhood physiological stress and itself a risk factor for AM. Thus, its study can provide a unique perspective on health in the past in a way that the study of few other lesions can.

Third, this project has clinical relevance. It has illustrated how mastoiditis affected the MAC in pre-antibiotic populations and has provided novel information about the impact of the environment on individuals' risk of developing mastoiditis in two population that were regionally similar, yet temporally distinct. In this way, this project has provided clinicians with a novel study of the pathophysiology of mastoiditis and has informed the environmental and genetic theories debate. That both populations experienced similar levels of risk, despite their different environments, may suggest that a certain amount of their risk was genetic. However, the numerous patterns in the results, which were illustrative of exposure to environmental risk, strongly suggests that much, if not most, of the factors associated with an individuals' chance of developing mastoiditis were environmental. The interaction between environmental stress and frailty appears to have contributed to individual risk of developing mastoiditis and other respiratory-related disease.

Finally, archaeological research into respiratory-related disease has been non-exhaustive. There remain gaps in the research: such as the rate of MS in Anglo-Saxon population and the bony appearance of certain LRI. This project has contributed new information on the rate and appearance of MS and LRI in late Anglo-Saxon/Saxo-Norman and Industrial era populations. In this way, this project is novel. However, data is most informative when part of a collection. It is our hope that the recent formation of research groups, such as the Bioarchaeology Respiratory Network, and the wider use of diagnostic and recording methods such as that for VSRL used here (Davies-Barrett et al. 2019), will encourage other researchers to continue this work.

8.6 Conclusions

This project has shed a unique light on past human health by creating an accessible, non-destructive method of imaging the MAC and diagnosing mastoiditis in archaeologically derived populations from England's north-east. It has also put this method to use by exploring the epidemiology and etiology of respiratory-related disease over time—from the founding of towns and the intensification of farming to the explosion of high-density living and industrialization. Issues such as equality, food security, safe housing, public sanitation, air pollution, urbanization, and workplace safety are as pressing today as they were in the past. To study the relationships between natural, artificial, and social environmental factors and human health is to explore the impact of these issues and human's interactions with them. This project has devised a new lens through which these issues can be viewed. All you have to do, is point and shoot.

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Appendix A

This appendix includes all the data collected for this project, as well as the demographic data from Swales (2012) (Black Gate) and Raynor *et al.* (2011) (Coronation Street). The demographic date is listed first, followed by the data regarding mastoiditis, maxillary sinusitis, and visceral surface rib lesions and lower respiratory infection. The data for Black Gate comes first, followed by that for Coronation Street. To begin, the column titles are explained.

Skeleton.No/Skel.No – Skeleton number
Sex – Biological sex
Pooled.Sex – Pooled biological sex
Age – Age at death
Age.Range – Age at death range
Grave.Type – Grave type
Body.Position – Body position
No.Ob.M – Number of observable mastoid processes
M.L – Left mastoid air cells
M.R – Right mastoid air cells
M.Adult – Adult mastoiditis
M.Childhood – Residual childhood mastoiditis
A.C.B.H. – Adult mastoiditis, residual childhood mastoiditis, both adult and residual childhood mastoiditis, or healthy (also known as mastoid air cells showing no sign of infection)
U.B.Childh. – Unilateral or bilateral residual childhood mastoiditis
U.B.Adult – Unilateral or bilateral residual childhood mastoiditis
No.Ob.Max.S – Number of observable maxillary sinuses
M.S.L – Left maxillary sinusitis
M.S.R – Right maxillary sinusitis
M.S.Ind – Individual maxillary sinusitis diagnosis
M.S.Uni.Bi. – Maxillary sinusitis unilateral or bilateral
No.ob.R.N.L – Number of observable left neck rib fragments
No.ob.R.A.L - Number of observable left angle rib fragments
No.ob.R.S.L - Number of observable left shaft rib fragments
No.ob.R.N.R - Number of observable right neck rib fragments
No.ob.R.A.R. - Number of observable right angle rib fragments

No.ob.R.S.R. - Number of observable right shaft rib fragments

LU1–3 – Number of observable left upper ribs 1–3

LMU4–6 - Number of observable left middle-upper ribs 4–6

LML7–9 - Number of observable left middle-lower ribs 7–9

LL10–12 - Number of observable left lower ribs 10–12

RU1–3 - Number of observable right upper ribs 1–3

RMU4–6 - Number of observable right middle-upper ribs 4–6

RML7–9 - Number of observable right middle-lower ribs 7–9

RL10–12 - Number of observable right lower ribs 11–12

Av.frag.score – Average fragmentation score

Av.cort.pres – Average cortical preservation

#LL – Total number of left visceral surface rib lesions

#RL – Total number of right visceral surface rib lesions

Uni.Side – Side of unilateral visceral surface rib lesions

Les.Type - Visceral surface rib lesions type

No.L.R.N.L – Number of left neck ribs fragments with visceral surface rib lesions

No.L.R.A.L - Number of left angle ribs fragments with visceral surface rib lesions

No.L.R.S.L - Number of left shaft ribs fragments with visceral surface rib lesions

No.L.R.N.R - Number of right neck ribs fragments with visceral surface rib lesions

No.L.R.A.R. - Number of right angle ribs fragments with visceral surface rib lesions

No.L.R.S.R. - Number of right shaft ribs fragments with visceral surface rib lesions

L.LU1–3 – Number of left upper ribs with lesions

L.LMU4–6 – Number of left middle-upper ribs with visceral surface rib lesions

L.LML7–9 – Number of left middle-lower ribs with visceral surface rib lesions

L.LL10–12 – Number of left lower ribs with visceral surface rib lesions

L.RU1–3 – Number of right upper ribs with visceral surface rib lesions

L.RMU4–6 – Number of right middle-upper ribs with visceral surface rib lesions

L.RML7–9 – Number of right middle-lower ribs with visceral surface rib lesions

L.RL10–12 – Number of right lower ribs with visceral surface rib lesions

Rib.Ind – Individual lower respiratory infection diagnosis

Black Gate-Demography

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
8	Male	Male	45-55 years	Senior.Adult	Plain	Right Side
18	Male	Male	30-40 years	Prime.Adult	NA	NA
22	Male	Male	40-50 years	Senior.Adult	Plain	Supine
23	Prob.Female	Female	20-60 years	Prime.Adult	Plain	Supine
27	Male	Male	25-30 years	Prime.Adult	Plain	Right Side
31	Unsexed	Unsexed	40-50 years	Senior.Adult	Plain	Right Side
33	Female	Female	45-55 years	Senior.Adult	Plain	Supine
40	Male	Male	25-30 years	Prime.Adult	Coffin	Right Side
42	Female	Female	17-19 years	Young.Adult	Plain	Supine
51	Female	Female	35-60 years	Senior.Adult	Coffin	Supine
52	Male	Male	50-60 years	Senior.Adult	Coffin	Right Side
53	Female	Female	25-35 years	Prime.Adult	Coffin	Supine
54	Prob.Male	Male	25-35 years	Prime.Adult	Plain	Supine
56	Male	Male	35-45 years	Mature.Adult	Plain	Supine
58	Male	Male	35-55 years	Mature.Adult	Plain	Supine
63	Prob.Female	Female	28-35 years	Prime.Adult	NA	NA

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
66	Male	Male	50-60 years	Senior.Adult	Plain	Right Side
69	Female	Female	35-45 years	Mature.Adult	Coffin	Right Side
70	Unsexed	Unsexed	25-35 years	Prime.Adult	Plain	Supine
71	Unsexed	Unsexed	25-35 years	Prime.Adult	Plain	Supine
74	Male	Male	40-50 years	Senior.Adult	Plain	Right Side
75	Unsexed	Unsexed	35-45 years	Mature.Adult	Plain	Right Side
77	Female	Female	25-30 years	Prime.Adult	Plain	Right Side
78	Female	Female	30-40 years	Mature.Adult	Plain	Supine
83	Male	Male	45-65 years	Senior.Adult	Plain	Right Side
89	Male	Male	23-26 years	Young.Adult	Plain	Supine
92	Female	Female	26-32 years	Prime.Adult	Plain	Supine
95	Female	Female	30-40 years	Mature.Adult	Coffin	Supine
97	Male	Male	45-55 years	Senior.Adult	Plain	Supine
99	Male	Male	50-60 years	Senior.Adult	Coffin	Supine
102	Female	Female	45-55 years	Senior.Adult	Plain	Right Side
111	Male	Male	25-35 years	Prime.Adult	Plain	Right Side

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
115	Female	Female	40-50 years	Senior.Adult	Plain	Supine
116	Prob.Male	Male	25-35 years	Prime.Adult	Plain	Supine
120	Female	Female	25-35 years	Prime.Adult	Coffin	Right Side
121	Male	Male	40-60 years	Senior.Adult	Plain	Right Side
124	Female	Female	35-45 years	Mature.Adult	Plain	Right Side
127	Female	Female	40-60 years	Senior.Adult	Plain	Right Side
128	Male	Male	40-65 years	Senior.Adult	Plain	Supine
130	Prob.Female	Female	45-55 years	Senior.Adult	Plain	Supine
133	Male	Male	17-20 years	Young.Adult	Plain	Right Side
134	Male	Male	35-45 years	Mature.Adult	Plain	Supine
137	Female	Female	25-35 years	Prime.Adult	Plain	Supine
139	Female	Female	40-50 years	Senior.Adult	Plain	Supine
147	Prob.Male	Male	45-55 years	Senior.Adult	Plain	Supine
148	Prob.Male	Male	35-45 years	Mature.Adult	NA	Supine
150	Unsexed	Unsexed	19-24 years	Young.Adult	NA	NA
151	Male	Male	40-50 years	Senior.Adult	Plain	Right Side

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
153	Male	Male	20-25 years	Young.Adult	Coffin	Supine
154	Male	Male	32-38 years	Prime.Adult	Plain	Supine
155	Female	Female	17-19 years	Young.Adult	Coffin	Right Side
159	Female	Female	45-60 years	Senior.Adult	Coffin	Supine
161	Male	Male	30-45 years	Mature.Adult	Plain	Supine
163	Female	Female	40-50 years	Senior.Adult	Plain	Right Side
164	Female	Female	35-45 years	Mature.Adult	Plain	Supine
167	Female	Female	25-30 years	Prime.Adult	Plain	Right Side
170	Female	Female	35-45 years	Mature.Adult	Coffin	Left Side
175	Male	Male	35-45 years	Mature.Adult	Plain	Prone
176	Female	Female	55-65 years	Senior.Adult	Plain	Flexed
177	Female	Female	45-55 years	Senior.Adult	Coffin	Prone
178	Female	Female	50-60 years	Senior.Adult	Coffin	Left Side
179	Female	Female	25-30 years	Prime.Adult	Plain	Right Side
181	Male	Male	40-50 years	Senior.Adult	Plain	Right Side
183	Female	Female	40-50 years	Senior.Adult	Coffin	Right Side

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
185	Male	Male	50-60 years	Senior.Adult	Plain	Right Side
186	Female	Female	50-60 years	Senior.Adult	Plain	Right Side
188	Male	Male	28-36 years	Prime.Adult	Plain	Right Side
190	Unsexed	Unsexed	17-19 years	Young.Adult	NA	NA
198	Male	Male	30-38 years	Prime.Adult	Plain	Supine
199	Unsexed	Unsexed	17-19 years	Young.Adult	NA	NA
200	Female	Female	25-35 years	Prime.Adult	Plain	Right Side
205	Female	Female	40-55 years	Senior.Adult	Plain	Right Side
206	Male	Male	35-45 years	Mature.Adult	Plain	Right Side
207	Female	Female	25-35 years	Prime.Adult	Plain	Right Side
211	Male	Male	35-45 years	Mature.Adult	Plain	Supine
212	Female	Female	25-35 years	Prime.Adult	Plain	Right Side
216	Female	Female	25-35 years	Prime.Adult	Plain	Right Side
218	Unsexed	Unsexed	18-20 years	Young.Adult	NA	NA
219	Female	Female	35-45 years	Mature.Adult	Plain	Supine
220	Female	Female	22-28 years	Prime.Adult	Plain	Left Side

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
224	Male	Male	50-60 years	Senior.Adult	Plain	Supine
229	Male	Male	25-30 years	Prime.Adult	Plain	Supine
236	Male	Male	30-40 years	Mature.Adult	Coffin	Right Side
240	Male	Male	17-19 years	Young.Adult	Plain	Supine
242	Male	Male	45-50 years	Senior.Adult	Coffin	Right Side
243	Female	Female	45-60 years	Senior.Adult	Coffin	Supine
245	Female	Female	50-60 years	Senior.Adult	Plain	Right Side
246	Female	Female	40-50 years	Senior.Adult	NA	Supine
249	Female	Female	45-55 years	Senior.Adult	Coffin	Supine
250	Prob.Male	Male	30-50 years	Mature.Adult	Plain	Supine
251	Male	Male	40-50 years	Senior.Adult	Plain	Supine
252	Unsexed	Unsexed	19-21 years	Young.Adult	NA	NA
256	Female	Female	40-50 years	Senior.Adult	Coffin	Supine
268	Male	Male	19-20 years	Young.Adult	Plain	Right Side
269	Female	Female	50-60 years	Senior.Adult	NA	NA
271	Female	Female	40-50 years	Senior.Adult	Plain	Flexed

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
275	Male	Male	35-40 years	Mature.Adult	Plain	Supine
277	Male	Male	35-45 years	Mature.Adult	Coffin	Prone
280	Female	Female	35-45 years	Mature.Adult	Coffin	Supine
282	Male	Male	35-45 years	Mature.Adult	Coffin	Right Side
283	Male	Male	34-40 years	Mature.Adult	Plain	Supine
284	Male	Male	45-55 years	Senior.Adult	Coffin	Right Side
286	Unsexed	Unsexed	33-39 years	Prime.Adult	Plain	Right Side
287	Male	Male	50-60 years	Senior.Adult	Plain	Right Side
288	Female	Female	26-32 years	Prime.Adult	Coffin	Supine
289	Female	Female	45-55 years	Senior.Adult	Coffin	Left Side
290	Male	Male	55-60 years	Senior.Adult	Plain	Right Side
293	Male	Male	30-40 years	Prime.Adult	Coffin	Supine
297	Male	Male	35-45 years	Mature.Adult	Plain	Right Side
299	Female	Female	28-34 years	Prime.Adult	Coffin	Right Side
300	Female	Female	25-35 years	Prime.Adult	Plain	Right Side
301	Unsexed	Unsexed	18-25 years	Young.Adult	NA	NA

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
302	Female	Female	50-60 years	Senior.Adult	Coffin	Right Side
304	Male	Male	25-35 years	Prime.Adult	Plain	Supine
305	Male	Male	32-44 years	Mature.Adult	Plain	Right Side
306	Female	Female	45-55 years	Senior.Adult	Plain	Left Side
308	Female	Female	40-55 years	Senior.Adult	Coffin	Right Side
315	Prob.Female	Female	45-60 years	Senior.Adult	Plain	Right Side
318	Male	Male	20-30 years	Prime.Adult	Plain	Supine
330	Female	Female	25-35 years	Prime.Adult	NA	NA
331	Prob.Female	Female	25-35 years	Prime.Adult	Coffin	Supine
333	Prob.Female	Female	40-60 years	Senior.Adult	Plain	Supine
334	Male	Male	40-50 years	Senior.Adult	Plain	Supine
335	Female	Female	35-45 years	Mature.Adult	NA	Supine
336	Female	Female	32-42 years	Mature.Adult	Plain	Supine
337	Female	Female	45-55 years	Senior.Adult	Plain	Supine
338	Prob.Female	Female	25-45? years	Prime.Adult	Plain	Supine
341	Female	Female	45+	Senior.Adult	Plain	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
342	Male	Male	18-25 years	Young.Adult	Plain	Right Side
344	Female	Female	25-35 years	Prime.Adult	Plain	Supine
345	Female	Female	35-45 years	Mature.Adult	Plain	Right Side
347	Female	Female	45-55 years	Senior.Adult	Plain	Supine
349	Male	Male	45+ years	Senior.Adult	Plain	Supine
351	Male	Male	45-55 years	Senior.Adult	Plain	Supine
356	Unsexed	Unsexed	18+	Adult	NA	NA
368	Male	Male	35-45 years	Mature.Adult	Cist	Supine
375	Female	Female	25-35 years	Prime.Adult	Cist	Supine
377	Male	Male	35-45 years	Mature.Adult	Cist	Supine
381	Female	Female	30-40 years	Mature.Adult	Cist	Supine
386	Male	Male	20-25 years	Young.Adult	Plain	Left Side
390	Male	Male	45-50 years	Senior.Adult	Plain	Supine
393	Unsexed	Unsexed	18+ years	Adult	NA	NA
400	Male	Male	40-60 years	Senior.Adult	Plain	Supine
404	Male	Male	25-35 years	Prime.Adult	Plain	Left Side
408	Male	Male	40-50 years	Senior.Adult	Plain	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
409	Male	Male	25-35 years	Prime.Adult	Plain	Prone
410	Prob.Male	Male	25-35 years	Prime.Adult	Plain	Supine
411	Male	Male	50-60 years	Senior.Adult	Plain	Supine
413	Prob.Male	Male	35-45 years	Mature.Adult	Plain	Supine
415	Male	Male	48-54 years	Senior.Adult	Plain	Supine
417	Female	Female	35-45 years	Mature.Adult	Plain	Supine
420	Female	Female	35-45 years	Mature.Adult	Plain	Supine
423	Male	Male	45-55 years	Senior.Adult	Plain	Supine
425	Unsexed	Unsexed	50-52 years	Senior.Adult	Plain	Supine
427	Male	Male	35-55 years	Mature.Adult	Plain	Supine
428	Female	Female	35-40 years	Mature.Adult	Plain	Supine
432	Female	Female	25-35 years	Prime.Adult	Earmuffs	Supine
433	Male	Male	28-34 years	Prime.Adult	Plain	Supine
434	Male	Male	40-50 years	Mature.Adult	Plain	Supine
435	Male	Male	30-40 years	Prime.Adult	Rubble Cist	Supine
437	Male	Male	40-50 years	Senior.Adult	Plain	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
439	Prob.Female	Female	30-50 years	Mature.Adult	Plain	Supine
441	Unsexed	Unsexed	18+	Adult	Plain	Supine
442	Male	Male	25-35 years	Prime.Adult	Plain	Supine
446	Male	Male	35-55 years	Senior.Adult	Plain	Supine
448	Female	Female	40-49 years	Mature.Adult	Head Box	Supine
451	Male	Male	35-45 years	Mature.Adult	Plain	Supine
453	Prob.Male	Male	35-45 years	Mature.Adult	Plain	Supine
454	Female	Female	35-45 years	Mature.Adult	Plain	Left Side
455	Male	Male	19-20 years	Young.Adult	NA	Right Side
457	Male	Male	25-30 years	Prime.Adult	Earmuffs	Supine
460	Male	Male	50-60 years	Senior.Adult	Plain	Supine
462	Male	Male	35-45 years	Mature.Adult	Plain	Supine
464	Female	Female	30-40 years	Prime.Adult	Plain	Supine
465	Prob.Female	Female	25-35 years	Prime.Adult	Plain	Right Side
468	Male	Male	50-60 years	Senior.Adult	Plain	Supine
473	Prob.Male	Male	30-50 years	Mature.Adult	Plain	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
480	Female	Female	39-46 years	Mature.Adult	Plain	Supine
481	Male	Male	50-60 years	Senior.Adult	Plain	Supine
482	Female	Female	40-50 years	Senior.Adult	Cist	Supine
487	Female	Female	25-29 years	Prime.Adult	Earmuffs?	Supine
488	Prob.Male	Male	50-55 years	Senior.Adult	Plain	Prone
491	Male	Male	34-44 years	Mature.Adult	Plain	Supine
493	Prob.Male	Male	15-25 years	Young.Adult	Cist	Supine
494	Male	Male	25-45 years	Prime.Adult	Plain	Right Side
498	Female	Female	17-19 years	Young.Adult	Plain	Supine
499	Male	Male	25-35 years	Prime.Adult	Cist	Supine
513	Female	Female	20-25 years	Young.Adult	Plain	Right Side
525	Male	Male	40-50 years	Senior.Adult	Plain	Left Side
530	Female	Female	30-40 years	Mature.Adult	Plain	Supine
534	Female	Female	17-25 years	Young.Adult	Plain	Supine
547	Male	Male	20-24 years	Young.Adult	Plain	Supine
548	Female	Female	45-60 years	Senior.Adult	Plain	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
549	Female	Female	30-40 years	Prime.Adult	Plain	Supine
552	Female	Female	50-60 years	Senior.Adult	Plain	Supine
553	Female	Female	30-45 years	Mature.Adult	Plain	Supine
555	Female	Female	20-25 years	Young.Adult	Plain	Supine
556	Prob.Female	Female	35-55 years	Mature.Adult	Plain	Supine
557	Male	Male	40-50 years	Senior.Adult	Plain	Supine
559	Female	Female	45-50 years	Senior.Adult	Earmuffs	Supine
560	Female	Female	38-44 years	Mature.Adult	Plain	Supine
561	Unsexed	Unsexed	18-25 years	Young.Adult	NA	NA
562	Unsexed	Unsexed	25-35 years	Prime.Adult	Plain	Left Side
566	Female	Female	30-40 years	Prime.Adult	Plain	Supine
567	Male	Male	20-25 years	Young.Adult	Plain	Supine
568	Female	Female	35-50 years	Mature.Adult	Plain	Supine
571	Unsexed	Unsexed	18-21 years	Young.Adult	NA	NA
572	Male	Male	40-50 years	Senior.Adult	Pillow Stone	Supine
577	Prob.Female	Female	45-60 years	Senior.Adult	Pillow Stone	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
578	Male	Male	19-25 years	Young.Adult	Plain	Supine
579	Female	Female	30-40 years	Mature.Adult	Plain	Supine
580	Female	Female	40-50 years	Senior.Adult	Rubble Cist	Supine
582	Male	Male	25-35 years	Prime.Adult	Plain	Supine
585	Male	Male	25-30 years	Prime.Adult	Plain	Supine
587	Female	Female	35-45 years	Mature.Adult	Plain	Supine
588	Prob.Male	Male	45-60 years	Senior.Adult	Plain	Supine
589	Female	Female	35-45 years	Mature.Adult	Plain	Supine
591	Female	Female	30-40 years	Mature.Adult	Plain	Supine
597	Male	Male	30-50 years	Mature.Adult	Plain	Supine
599	Female	Female	25-35 years	Prime.Adult	Plain	Supine
600	Female	Female	25-35 years	Prime.Adult	Plain	Supine
602	Female	Female	35-45 years	Mature.Adult	Plain	Supine
603	Male	Male	28-32 years	Prime.Adult	Plain	Supine
604	Male	Male	25-30 years	Prime.Adult	Plain	Supine
605	Male	Male	50-60 years	Senior.Adult	Head Box	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
606	Prob.Male	Male	30-40 years	Prime.Adult	Plain	Supine
607	Male	Male	26-34 years	Prime.Adult	Plain	Supine
609	Male	Male	30-40 years	Prime.Adult	Plain	Supine
612	Prob.Male	Male	45-60 years	Senior.Adult	Coffin	Supine
619	Female	Female	45-59 years	Senior.Adult	Chest	Supine
620	Female	Female	50-60 years	Senior.Adult	Head Box	Supine
622	Male	Male	20-25 years	Young.Adult	Plain	Supine
625	Female	Female	45-55 years	Senior.Adult	Earmuffs	Supine
626	Prob.Female	Female	20-25 years	Young.Adult	Plain	Supine
628	Male	Male	35-45 years	Mature.Adult	Plain	Supine
631	Female	Female	34-48 years	Mature.Adult	NA	Left Side
634	Male	Male	30-35 years	Prime.Adult	Plain	Supine
635	Female	Female	28-34 years	Prime.Adult	Plain	Supine
637	Male	Male	40-45 years	Mature.Adult	Plain	Supine
638	Male	Male	35-45 years	Mature.Adult	Plain	Prone
640	Female	Female	30-40 years	Prime.Adult	Plain	Supine

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
641	Prob.Female	Female	35-45 years	Mature.Adult	NA	Left Side
642	Prob.Female	Female	30-45 years	Mature.Adult	Plain	Supine
644	Unsexed	Unsexed	40-50 years	Senior.Adult	NA	NA
645	Unsexed	Unsexed	30-40 years	Prime.Adult	NA	NA
651	Male	Male	35-45 years	Mature.Adult	Plain	Supine
652	Male	Male	40-50 years	Senior.Adult	Plain	Supine
654	Unsexed	Unsexed	22-26 years	Young.Adult	Plain	Supine
656	Unsexed	Unsexed	35-40 years	Mature.Adult	Plain	Supine
657	Male	Male	40-50 years	Senior.Adult	Plain	Supine
659	Female	Female	25-35 years	Prime.Adult	Plain	Supine
660	Male	Male	50-60 years	Senior.Adult	Plain	Flexed
157i	Female	Female	35-45 years	Mature.Adult	Plain	Supine
158i	Male	Male	35-45 years	Mature.Adult	Plain	Supine
158ii	Prob.Female	Female	50-60 years	Senior.Adult	Plain	NA
191i	Male	Male	18-20 years	Young.Adult	Plain	Supine
43ii	Female	Female	30-40 years	Mature.Adult	NA	NA

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age	Age.Range	Grave.Type	Body.Position
461i	Male	Male	25-35 years	Prime.Adult	Plain	Supine
461ii	Unsexed	Unsexed	18-25 years	Young.Adult	NA	NA
533i	Unsexed	Unsexed	18-25 years	Young.Adult	NA	NA
76i	Male	Male	20-25 years	Young.Adult	Plain	Supine
82i	Female	Female	35-45 years	Mature.Adult	Plain	NA
91ii	Unsexed	Unsexed	25-35 years	Prime.Adult	Plain	NA

Black Gate-Mastoiditis

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
8	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
18	1	NA	S.Hyp	Present	Present	Both	NA	NA
22	2	S.Hyp	S.Hyp	Present	Present	Both	Uni	Bi
23	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
27	0	NA	NA	NA	NA	NA	NA	NA
31	0	NA	NA	NA	NA	NA	NA	NA
33	0	NA	NA	NA	NA	NA	NA	NA
40	0	NA	NA	NA	NA	NA	NA	NA
42	0	NA	NA	NA	NA	NA	NA	NA
51	1	NA	LSH	Present	Present	Both	NA	NA
52	0	NA	NA	NA	NA	NA	NA	NA
53	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
54	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
56	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
58	0	NA	NA	NA	NA	NA	NA	NA
63	1	NA	Absent	Absent	Present	Childhood	NA	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
66	1	Absent	NA	Absent	Present	Childhood	NA	NA
69	0	NA	NA	NA	NA	NA	NA	NA
70	1	Absent	NA	Absent	Present	Childhood	NA	NA
71	1	LSH	NA	Present	Present	Both	NA	NA
74	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
75	0	NA	NA	NA	NA	NA	NA	NA
77	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
78	0	NA	NA	NA	NA	NA	NA	NA
83	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
89	0	NA	NA	NA	NA	NA	NA	NA
92	1	NA	LSH	Present	Absent	Adult	NA	NA
95	0	NA	NA	NA	NA	NA	NA	NA
97	2	S.Hyp	S.Hyp	Present	Present	Both	Uni	Bi
99	0	NA	NA	NA	NA	NA	NA	NA
102	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
111	0	NA	NA	NA	NA	NA	NA	NA
115	2	LSH	S.Hyp	Present	Absent	Adult	NA	Bi
116	1	LSH	NA	Present	Absent	Adult	NA	NA
120	2	Hyper	LSH	Present	Absent	Adult	NA	Uni
121	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
124	0	NA	NA	NA	NA	NA	NA	NA
127	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
128	1	NA	LSH	Present	Absent	Adult	NA	NA
130	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
133	1	S.Hyp	NA	Present	Present	Both	NA	NA
134	2	Hyper	Absent	Absent	Present	Childhood	Uni	NA
137	1	NA	Absent	Absent	Present	Childhood	NA	NA
139	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
147	0	NA	NA	NA	NA	NA	NA	NA
148	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
150	0	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
151	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
153	1	NA	LSH	Present	Absent	Adult	NA	NA
154	1	Absent	NA	Absent	Present	Childhood	NA	NA
155	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
159	2	Hyper	LSH	Present	Absent	Adult	NA	Uni
161	2	LSH	LSH	Present	Absent	Adult	NA	Bi
163	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
164	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
167	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
170	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
175	1	NA	LSH	Present	Absent	Adult	NA	NA
176	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
177	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
178	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
179	0	NA	NA	NA	NA	NA	NA	NA
181	1	NA	LSH	Present	Absent	Adult	NA	NA
183	1	S.Hyp	NA	Present	Present	Both	NA	NA
185	2	Hyper	S.Hyp	Present	Absent	Adult	NA	Uni
186	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
188	0	NA	NA	NA	NA	NA	NA	NA
190	0	NA	NA	NA	NA	NA	NA	NA
198	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
199	0	NA	NA	NA	NA	NA	NA	NA
200	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
205	2	LSH	LSH	Present	Present	Both	Bi	Bi
206	2	Hyper	Hyper	Absent	Absent	Healthy	NA	Bi
207	0	NA	NA	NA	NA	NA	NA	NA
211	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
212	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
216	1	NA	LSH	Present	Present	Both	NA	NA
218	0	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
219	0	NA	NA	NA	NA	NA	NA	NA
220	2	Hyper	Absent	Absent	Present	Childhood	Uni	NA
224	0	NA	NA	NA	NA	NA	NA	NA
229	0	NA	NA	NA	NA	NA	NA	NA
236	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
240	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
242	2	Hyper	Hyper	Absent	Absent	Healthy	NA	Bi
243	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
245	0	NA	NA	NA	NA	NA	NA	NA
246	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
249	2	S.Hyp	LSH	Present	Present	Both	Uni	Bi
250	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
251	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
252	2	Absent	Hyper	Absent	Present	Childhood	Uni	NA
256	2	LSH	Hyper	Present	Absent	Adult	NA	Uni
268	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
269	2	LSH	LSH	Present	Absent	Adult	NA	Bi
271	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
275	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
277	2	Absent	NA	Absent	Present	Childhood	NA	NA
280	0	NA	NA	NA	NA	NA	NA	NA
282	0	NA	NA	NA	NA	NA	NA	NA
283	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
284	2	S.Hyp	S.Hyp	Present	Present	Both	Uni	Bi
286	1	LSH	NA	Present	Absent	Adult	NA	NA
287	2	LSH	LSH	Present	Present	Both	Bi	Bi
288	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
289	2	LSH	LSH	Present	Present	Both	Bi	Bi
290	1	Absent	NA	Absent	Present	Childhood	NA	NA
293	0	NA	NA	NA	NA	NA	NA	NA
297	1	Absent	NA	Absent	Present	Childhood	NA	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
299	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
300	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
301	0	NA	NA	NA	NA	NA	NA	NA
302	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
304	2	LSH	LSH	Present	Absent	Adult	NA	Bi
305	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
306	1	NA	Absent	Absent	Present	Childhood	NA	NA
308	0	NA	NA	NA	NA	NA	NA	NA
315	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
318	0	NA	NA	NA	NA	NA	NA	NA
330	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
331	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
333	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
334	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
335	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
336	2	Absent	Absent	Absent	Present	Childhood	Uni	NA
337	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
338	1	Absent	NA	Absent	Present	Childhood	NA	NA
341	1	NA	LSH	Present	Absent	Adult	NA	NA
342	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
344	0	NA	NA	NA	NA	NA	NA	NA
345	0	NA	NA	NA	NA	NA	NA	NA
347	1	LSH	NA	Present	Absent	Adult	NA	NA
349	0	NA	NA	NA	NA	NA	NA	NA
351	0	NA	NA	NA	NA	NA	NA	NA
356	0	NA	NA	NA	NA	NA	NA	NA
368	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
375	2	LSH	Hyper	Present	Absent	Adult	NA	Uni
377	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
381	0	NA	NA	NA	NA	NA	NA	NA
386	2	Absent	Absent	Absent	Present	Childhood	Bi	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
390	0	NA	NA	NA	NA	NA	NA	NA
393	0	NA	NA	NA	NA	NA	NA	NA
400	0	NA	NA	NA	NA	NA	NA	NA
404	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
408	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
409	1	Absent	NA	Absent	Present	Childhood	NA	NA
410	0	NA	NA	NA	NA	NA	NA	NA
411	1	LSH	NA	Present	Absent	Adult	NA	NA
413	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
415	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
417	2	Absent	S.Hyp	Present	Present	Both	Bi	Uni
420	1	Absent	NA	Absent	Present	Childhood	NA	NA
423	0	NA	NA	NA	NA	NA	NA	NA
425	0	NA	NA	NA	NA	NA	NA	NA
427	1	LSH	NA	Present	Present	Both	NA	NA
428	1	NA	LSH	Present	Absent	Adult	NA	NA
432	2	Hyper	S.Hyp	Present	Absent	Adult	NA	Uni
433	2	LSH	LSH	Present	Absent	Adult	NA	Bi
434	2	Hyper	Absent	Absent	Present	Childhood	Bi	NA
435	2	LSH	LSH	Present	Present	Both	Uni	Bi
437	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
439	0	NA	NA	NA	NA	NA	NA	NA
441	0	NA	NA	NA	NA	NA	NA	NA
442	0	NA	NA	NA	NA	NA	NA	NA
446	0	NA	NA	NA	NA	NA	NA	NA
448	2	LSH	LSH	Present	Absent	Adult	NA	Bi
451	0	NA	NA	NA	NA	NA	NA	NA
453	0	NA	NA	NA	NA	NA	NA	NA
454	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
455	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
457	2	Hyper	Hyper	Absent	Present	Childhood	Bi	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
460	1	NA	S.Hyp	Present	Present	Both	NA	NA
462	0	NA	NA	NA	NA	NA	NA	NA
464	1	NA	Absent	Absent	Present	Childhood	NA	NA
465	0	NA	NA	NA	NA	NA	NA	NA
468	1	Absent	NA	Absent	Present	Childhood	NA	NA
473	1	LSH	NA	Present	Absent	Adult	NA	NA
480	2	Absent	Hyper	Absent	Present	Childhood	Uni	NA
481	1	LSH	NA	Present	Absent	Adult	NA	NA
482	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
487	1	Absent	NA	Absent	Present	Childhood	NA	NA
488	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
491	1	NA	LSH	Present	Absent	Adult	NA	NA
493	1	NA	Absent	Absent	Present	Childhood	NA	NA
494	0	NA	NA	NA	NA	NA	NA	NA
498	0	NA	NA	NA	NA	NA	NA	NA
499	0	NA	NA	NA	NA	NA	NA	NA
513	0	NA	NA	NA	NA	NA	NA	NA
525	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
530	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
534	0	NA	NA	NA	NA	NA	NA	NA
547	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
548	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
549	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
552	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
553	1	Absent	NA	Absent	Present	Childhood	NA	NA
555	1	Absent	NA	Absent	Present	Childhood	NA	NA
556	1	NA	S.Hyp	Present	Present	Both	NA	NA
557	2	LSH	LSH	Present	Absent	Adult	NA	Bi
559	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
560	2	LSH	S.Hyp	Present	Present	Both	Bi	Bi
561	0	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
562	0	NA	NA	NA	NA	NA	NA	NA
566	0	NA	NA	NA	NA	NA	NA	NA
567	0	NA	NA	NA	NA	NA	NA	NA
568	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
571	0	NA	NA	NA	NA	NA	NA	NA
572	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
577	1	NA	LSH	Present	Absent	Adult	NA	NA
578	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
579	2	Hyper	Absent	Absent	Present	Childhood	Uni	NA
580	2	S.Hyp	LSH	Present	Absent	Adult	NA	Bi
582	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
585	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
587	2	Absent	Hyper	Absent	Present	Childhood	Uni	NA
588	2	Hyper	Absent	Absent	Present	Childhood	Uni	NA
589	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
591	2	LSH	Hyper	Present	Absent	Adult	NA	Uni
597	0	NA	NA	NA	NA	NA	NA	NA
599	0	NA	NA	NA	NA	NA	NA	NA
600	0	NA	NA	NA	NA	NA	NA	NA
602	0	NA	NA	NA	NA	NA	NA	NA
603	2	Absent	S.Hyp	Present	Present	Both	Uni	Uni
604	0	NA	NA	NA	NA	NA	NA	NA
605	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
606	0	NA	NA	NA	NA	NA	NA	NA
607	0	NA	NA	NA	NA	NA	NA	NA
609	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
612	1	Absent	NA	Absent	Present	Childhood	NA	NA
619	0	NA	NA	NA	NA	NA	NA	NA
620	2	Hyper	S.Hyp	Present	Absent	Adult	NA	Uni
622	0	NA	NA	NA	NA	NA	NA	NA
625	0	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skeleton. No	No.Ob.M	M.L	M.R	M. Adult	M. Childhood	A.C.B.H.	U.B. Childh.	U.B. Adult
626	1	LSH	NA	Present	Present	Both	NA	NA
628	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
631	1	NA	LSH	Present	Present	Both	NA	NA
634	0	NA	NA	NA	NA	NA	NA	NA
635	0	NA	NA	NA	NA	NA	NA	NA
637	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
638	2	LSH	LSH	Present	Absent	Adult	Bi	NA
640	1	Absent	NA	Absent	Present	Childhood	NA	NA
641	0	NA	NA	NA	NA	NA	NA	NA
642	0	NA	NA	NA	NA	NA	NA	NA
644	0	NA	NA	NA	NA	NA	NA	NA
645	0	NA	NA	NA	NA	NA	NA	NA
651	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
652	1	Absent	NA	Absent	Present	Childhood	NA	NA
654	0	NA	NA	NA	NA	NA	NA	NA
656	0	NA	NA	NA	NA	NA	NA	NA
657	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
659	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
660	2	S.Hyp	LSH	Present	Present	Both	Uni	Bi
157i	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
158i	0	NA	NA	NA	NA	NA	NA	NA
158ii	0	NA	NA	NA	NA	NA	NA	NA
191i	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
43ii	0	NA	NA	NA	NA	NA	NA	NA
461i	1	NA	S.Hyp	Present	Present	Both	NA	NA
461ii	0	NA	NA	NA	NA	NA	NA	NA
533i	0	NA	NA	NA	NA	NA	NA	NA
76i	0	NA	NA	NA	NA	NA	NA	NA
82i	0	NA	NA	NA	NA	NA	NA	NA
91ii	0	NA	NA	NA	NA	NA	NA	NA

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
8	0	NA	NA	NA	NA
18	0	NA	NA	NA	NA
22	2	Absent	Absent	Absent	NA
23	0	NA	NA	NA	NA
27	0	NA	NA	NA	NA
31	0	NA	NA	NA	NA
33	0	NA	NA	NA	NA
40	2	Absent	Absent	Absent	NA
42	0	NA	NA	NA	NA
51	0	NA	NA	NA	NA
52	0	NA	NA	NA	NA
53	1	Spicules	NA	Present	NA
54	0	NA	NA	NA	NA
56	0	NA	NA	NA	NA
58	2	Pitting	Spicules	Present	Bi
63	0	NA	NA	NA	NA
66	0	NA	NA	NA	NA
69	0	NA	NA	NA	NA
70	0	NA	NA	NA	NA
71	0	NA	NA	NA	NA
74	0	NA	NA	NA	NA
75	0	NA	NA	NA	NA
77	0	NA	NA	NA	NA
78	0	NA	NA	NA	NA
83	0	NA	NA	NA	NA
89	2	Pitting	Absent	Present	Uni
92	2	Spicules	Spicules	Present	Bi
95	2	Spicules	Spicules	Present	Bi
97	0	NA	NA	NA	NA
99	0	NA	NA	NA	NA
102	1	Spicules	NA	Present	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
111	0	NA	NA	NA	NA
115	1	Absent	NA	Absent	NA
116	0	NA	NA	NA	NA
120	1	Absent	NA	Absent	NA
121	0	NA	NA	NA	NA
124	1	Spicules	NA	Present	NA
127	0	NA	NA	NA	NA
128	1	Absent	NA	Absent	NA
130	0	NA	NA	NA	NA
133	2	Spicules	Absent	Present	Uni
134	1	NA	Absent	Absent	NA
137	2	Absent	Absent	Absent	NA
139	1	Absent	NA	Absent	NA
147	2	Absent	Absent	Absent	NA
148	0	NA	NA	NA	NA
150	0	NA	NA	NA	NA
151	1	NA	Absent	Absent	NA
153	2	Absent	Absent	Absent	NA
154	1	Spicules	NA	Present	NA
155	1	NA	Pitting	Present	NA
159	2	Absent	Absent	Absent	NA
161	0	NA	NA	NA	NA
163	1	NA	Absent	Absent	NA
164	2	Rem.Spicules	Rem.Spicules	Present	Bi
167	1	NA	Spicules	Present	NA
170	0	NA	NA	NA	NA
175	1	Absent	NA	Absent	NA
176	0	NA	NA	NA	NA
177	1	NA	Absent	Absent	NA
178	2	Spicules	Spicules	Present	Bi
179	2	Spicules	Spicules	Present	Bi
181	0	NA	NA	NA	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
183	0	NA	NA	NA	NA
185	0	NA	NA	NA	NA
186	2	Absent	Absent	Absent	NA
188	2	Absent	Absent	Absent	NA
190	0	NA	NA	NA	NA
198	0	NA	NA	NA	NA
199	0	NA	NA	NA	NA
200	0	NA	NA	NA	NA
205	1	Absent	NA	Absent	NA
206	1	Rem.Spicules	NA	Present	NA
207	1	NA	Spicules	Present	NA
211	2	W.Pitted	W.Pitted	Present	Bi
212	2	Absent	Absent	Absent	NA
216	0	NA	NA	NA	NA
218	0	NA	NA	NA	NA
219	1	Absent	NA	Absent	NA
220	1	Absent	NA	Absent	NA
224	0	NA	NA	NA	NA
229	0	NA	NA	NA	NA
236	2	Spicules	Spicules	Present	Bi
240	0	NA	NA	NA	NA
242	2	Absent	Rem.Spicules	Present	Uni
243	2	Absent	Absent	Absent	NA
245	0	NA	NA	NA	NA
246	2	Absent	Rem.Spicules	Present	Uni
249	0	NA	NA	NA	NA
250	1	Absent	NA	Absent	NA
251	0	NA	NA	NA	NA
252	2	Absent	Absent	Absent	NA
256	0	NA	NA	NA	NA
268	0	NA	NA	NA	NA
269	0	NA	NA	NA	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
271	0	NA	NA	NA	NA
275	0	NA	NA	NA	NA
277	0	NA	NA	NA	NA
280	1	Spicules	NA	Present	NA
282	1	NA	Absent	Absent	NA
283	1	Rem.Spicules	NA	Present	NA
284	1	Absent	NA	Absent	NA
286	2	Absent	Absent	Absent	NA
287	1	Absent	NA	Absent	NA
288	2	Absent	Absent	Absent	NA
289	2	Spicules	Spicules	Present	Bi
290	2	Rem.Spicules	Absent	Present	Uni
293	1	Absent	NA	Absent	NA
297	1	NA	Absent	Absent	NA
299	0	NA	NA	NA	NA
300	2	Absent	Absent	Absent	NA
301	0	NA	NA	NA	NA
302	0	NA	NA	NA	NA
304	2	Spicules	Spicules	Present	Bi
305	1	NA	Absent	Absent	NA
306	0	NA	NA	NA	NA
308	2	Spicules	Absent	Present	Uni
315	1	NA	Absent	Absent	NA
318	0	NA	NA	NA	NA
330	2	Absent	Absent	Absent	NA
331	1	NA	Rem.Spicules	Present	NA
333	0	NA	NA	NA	NA
334	0	NA	NA	NA	NA
335	0	NA	NA	NA	NA
336	0	NA	NA	NA	NA
337	1	NA	Absent	Absent	NA
338	1	Absent	NA	Absent	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
341	0	NA	NA	NA	NA
342	0	NA	NA	NA	NA
344	0	NA	NA	NA	NA
345	0	NA	NA	NA	NA
347	2	Absent	Absent	Absent	NA
349	0	NA	NA	NA	NA
351	0	NA	NA	NA	NA
356	0	NA	NA	NA	NA
368	0	NA	NA	NA	NA
375	0	NA	NA	NA	NA
377	2	Absent	Absent	Absent	NA
381	0	NA	NA	NA	NA
386	2	Absent	Spicules	Present	Uni
390	2	Absent	Absent	Absent	NA
393	0	NA	NA	NA	NA
400	0	NA	NA	NA	NA
404	0	NA	NA	NA	NA
408	2	Absent	Absent	Absent	NA
409	2	Absent	Absent	Absent	NA
410	0	NA	NA	NA	NA
411	0	NA	NA	NA	NA
413	2	Absent	Absent	Absent	NA
415	1	NA	Spicules	Present	NA
417	2	Absent	Absent	Absent	NA
420	1	Absent	NA	Absent	NA
423	0	NA	NA	NA	NA
425	0	NA	NA	NA	NA
427	0	NA	NA	NA	NA
428	0	NA	NA	NA	NA
432	2	Absent	Spicules	Present	Uni
433	2	Absent	Absent	Absent	NA
434	2	Absent	Absent	Absent	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
435	1	NA	Absent	Absent	NA
437	0	NA	NA	NA	NA
439	0	NA	NA	NA	NA
441	0	NA	NA	NA	NA
442	0	NA	NA	NA	NA
446	0	NA	NA	NA	NA
448	2	Absent	Absent	Absent	NA
451	0	NA	NA	NA	NA
453	0	NA	NA	NA	NA
454	0	NA	NA	NA	NA
455	0	NA	NA	NA	NA
457	0	NA	NA	NA	NA
460	2	Absent	Absent	Absent	NA
462	0	NA	NA	NA	NA
464	1	NA	Spicules	Present	NA
465	0	NA	NA	NA	NA
468	0	NA	NA	NA	NA
473	1	NA	Rem.Spicules	Present	NA
480	1	NA	Spicules	Present	NA
481	0	NA	NA	NA	NA
482	0	NA	NA	NA	NA
487	0	NA	NA	NA	NA
488	2	Absent	Absent	Absent	NA
491	1	NA	Rem.Spicules	Present	NA
493	0	NA	NA	NA	NA
494	0	NA	NA	NA	NA
498	2	Absent	Absent	Absent	NA
499	1	NA	Absent	Absent	NA
513	0	NA	NA	NA	NA
525	0	NA	NA	NA	NA
530	0	NA	NA	NA	NA
534	0	NA	NA	NA	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
547	2	Absent	Absent	Absent	NA
548	0	NA	NA	NA	NA
549	2	Absent	Absent	Absent	NA
552	0	NA	NA	NA	NA
553	1	NA	Spicules	Present	NA
555	0	NA	NA	NA	NA
556	2	Absent	Absent	Absent	NA
557	1	NA	Absent	Absent	NA
559	0	NA	NA	NA	NA
560	2	Rem.Spicules	Absent	Present	Uni
561	0	NA	NA	NA	NA
562	0	NA	NA	NA	NA
566	0	NA	NA	NA	NA
567	2	Absent	Absent	Absent	NA
568	1	Absent	NA	Absent	NA
571	0	NA	NA	NA	NA
572	2	Absent	Absent	Absent	NA
577	2	Absent	Absent	Absent	NA
578	1	NA	Rem.Spicules	Present	NA
579	2	Absent	Spicules	Present	Uni
580	0	NA	NA	NA	NA
582	0	NA	NA	NA	NA
585	1	Absent	NA	Absent	NA
587	1	NA	Spicules	Present	NA
588	1	NA	Absent	Absent	NA
589	0	NA	NA	NA	NA
591	2	Spicules	Spicules	Present	Bi
597	1	Absent	NA	Absent	NA
599	0	NA	NA	NA	NA
600	0	NA	NA	NA	NA
602	0	NA	NA	NA	NA
603	2	Absent	Absent	Absent	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
604	1	NA	Spicules	Present	NA
605	0	NA	NA	NA	NA
606	2	Spicules	Rem.Spicules	Present	Bi
607	1	NA	Absent	Absent	NA
609	1	NA	Rem.Spicules	Present	NA
612	2	Absent	Absent	Absent	NA
619	0	NA	NA	NA	NA
620	0	NA	NA	NA	NA
622	2	Spicules	Spicules	Present	Bi
625	0	NA	NA	NA	NA
626	0	NA	NA	NA	NA
628	2	Rem.Spicules	Absent	Present	Uni
631	0	NA	NA	NA	NA
634	2	Spicules	Absent	Present	Uni
635	0	NA	NA	NA	NA
637	2	Absent	Absent	Absent	NA
638	0	NA	NA	NA	NA
640	2	Spicules	Spicules	Present	Bi
641	0	NA	NA	NA	NA
642	0	NA	NA	NA	NA
644	0	NA	NA	NA	NA
645	0	NA	NA	NA	NA
651	1	NA	Absent	Absent	NA
652	0	NA	NA	NA	NA
654	0	NA	NA	NA	NA
656	1	Absent	NA	Absent	NA
657	0	NA	NA	NA	NA
659	1	NA	Absent	Absent	NA
660	0	NA	NA	NA	NA
157i	1	Spicules	NA	Present	NA
158i	2	Absent	Absent	Absent	NA
158ii	2	Absent	Absent	Absent	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
191i	2	Spicules	Spicules	Present	Bi
43ii	0	NA	NA	NA	NA
461i	2	Spicules	Spicules	Present	Bi
461ii	0	NA	NA	NA	NA
533i	0	NA	NA	NA	NA
76i	2	Rem.Spicules	Rem.Spicules	Present	Bi
82i	2	Absent	Absent	Absent	NA
91ii	1	Absent	NA	Absent	NA

Black Gate-Rib Presence

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
8	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
18	5	5	0	5	4	0	1	1	3	0	2	1	2	0	Moderate	1
22	9	8	6	8	7	2	3	3	3	2	3	1	3	1	Moderate	2
23	7	9	1	6	8	0	3	1	2	1	2	2	0	2	Severe	1
27	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
31	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
33	2	3	1	2	0	2	1	NA	NA	1	1	NA	NA	NA	Moderate	3
40	7	7	1	5	8	0	3	3	2	0	3	1	2	0	Severe	2
42	9	10	12	11	10	7	3	3	3	3	3	3	2	3	Minor	3
51	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
52	4	4	1	12	9	1	2	1	1	0	3	3	3	0	Moderate	3
53	10	10	9	12	11	11	2	2	3	3	3	3	3	3	Minor	3
54	2	3	1	0	0	0	0	3	0	0	0	0	0	0	Severe	3
56	6	7	0	0	5	0	2	1	1	2	3	0	0	0	Sever	4
58	0	2	1	1	1	3	1	0	0	0	0	0	1	0	Severe	4
63	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
66	8	9	4	3	4	2	3	3	3	0	2	0	0	3	Moderate	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
69	8	6	7	2	9	0	3	3	2	2	3	3	3	NA	Moderate	2
70	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
71	0	0	0	0	3	0	0	0	0	0	0	0	0	0	Severe	2
74	3	6	3	2	5	1	3	0	2	1	3	0	2	0	Severe	4
75	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
77	5	8	1	1	2	4	3	3	2	0	2	2	0	1	Moderate	4
78	2	0	0	3	2	1	NA	1	3	NA	2	NA	3	1	Moderate	4
83	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
89	11	11	6	8	8	8	2	3	3	3	NA	3	3	2	Minor	2
92	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
95	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
97	10	8	3	12	11	6	2	2	3	NA	3	3	3	3	Minor	1
99	10	10	9	12	7	5	3	3	3	1	3	3	3	3	Moderate	3
102	0	5	1	0	0	0	0	3	2	1	0	0	0	0	Sever	2
111	0	0	0	4	4	4	0	0	0	0	2	1	3	1	Severe	2
115	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
116	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
120	5	11	8	6	12	4	3	3	3	3	3	3	3	3	Minor	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
121	6	6	2	8	10	9	3	3	2	0	2	3	3	2	Moderate	3
124	3	5	6	6	8	2	3	0	2	2	2	1	3	0	Moderate	3
127	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
128	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
130	0	0	3	0	1	4	0	NA	NA	0	0	NA	NA	0	Minor	3
133	10	5	3	6	10	3	3	3	3	1	2	1	3	0	Severe	4
134	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
137	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
139	4	3	3	1	1	1	3	2	0	0	0	0	0	0	Severe	2
147	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
148	2	1	3	5	0	1	1	3	3	NA	NA	NA	NA	NA	Minor	1
150	5	6	4	8	7	8	1	2	2	2	2	3	3	0	Moderate	2
151	10	9	5	10	10	6	3	3	3	2	2	3	3	2	Moderate	2
153	9	11	8	8	10	7	3	3	3	2	3	2	3	2	Minor	3
154	7	2	1	9	8	3	2	2	1	3	3	3	3	3	Severe	1
155	11	9	10	11	9	9	3	3	3	3	3	2	3	3	Moderate	3
159	10	9	3	6	5	4	3	3	3	3	2	3	2	0	Moderate	2
161	10	11	8	8	8	8	3	3	3	2	3	3	2	0	Moderate	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
163	8	10	5	2	9	3	2	3	3	3	2	1	0	1	Moderate	3
164	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
167	5	7	2	5	5	1	2	3	2	0	1	2	2	0	Moderate	3
170	10	11	7	11	10	7	3	3	3	1	3	3	3	2	Moderate	2
175	0	0	0	1	0	0	0	0	0	0	0	0	0	1	Severe	4
176	9	4	1	6	1	3	2	3	3	1	2	2	2	0	Severe	3
177	2	4	2	10	10	4	1	1	1	0	1	3	3	3	Severe	3
178	5	9	4	7	11	3	2	2	3	1	3	2	1	3	Moderate	2
179	10	9	8	9	9	11	3	3	2	2	3	2	2	2	Moderate	3
181	9	8	5	10	10	8	2	3	3	2	3	3	2	2	Severe	2
183	0	0	0	9	7	5	0	0	0	0	3	1	3	2	Moderate	2
185	3	2	0	2	4	1	0	1	2	0	1	0	2	0	Severe	1
186	7	7	6	10	7	6	2	2	3	2	2	3	3	2	Minor	2
188	0	1	3	3	6	5	1	0	0	0	2	1	1	2	Moderate	2
190	6	7	9	6	4	10	3	3	1	0	3	3	1	0	Moderate	4
198	7	11	7	8	11	5	3	2	2	2	3	1	3	2	Moderate	3
199	0	NA	NA	NA	NA	NA	0	NA	NA	NA	0	0	0	0	NA	NA
200	9	10	6	11	11	9	3	3	2	2	3	3	2	3	Moderate	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
205	3	4	2	6	7	7	0	3	1	0	3	1	2	1	Moderate	2
206	9	9	9	8	8	10	2	3	3	2	3	2	3	1	Minor	2
207	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
211	10	7	12	6	6	2	2	3	3	3	3	0	3	0	Minor	2
212	7	8	5	1	10	10	1	3	3	1	3	3	3	1	Moderate	4
216	3	5	5	11	11	11	1	1	1	2	3	3	2	3	Minor	4
218	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
219	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
220	12	11	4	12	5	6	3	3	3	3	3	3	3	3	Moderate	3
224	2	6	2	0	0	1	1	2	2	1	1	0	0	0	Severe	2
229	10	9	9	9	10	9	3	2	3	3	3	3	3	2	Moderate	2
236	4	4	3	5	6	4	3	2	0	0	3	3	0	0	Minor	1
240	8	10	6	8	10	1	3	2	2	2	3	3	3	1	Moderate	2
242	10	11	9	11	11	9	3	3	2	3	3	3	3	3	Moderate	1
243	12	12	7	10	10	10	3	3	3	3	3	3	3	1	Minor	3
245	4	4	4	11	11	6	0	1	1	2	3	3	3	2	Moderate	4
246	11	11	7	11	12	8	3	3	3	3	3	3	3	3	Minor	2
249	12	11	8	10	10	5	3	3	3	3	3	3	3	1	Minor	3

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
250	5	6	5	3	4	2	3	3	0	0	1	0	3	0	Moderate	3
251	4	4	5	4	4	7	2	1	1	0	2	0	2	0	Moderate	2
252	10	11	6	7	9	8	3	3	3	2	3	3	2	2	Moderate	3
256	5	11	8	5	8	6	3	3	3	2	3	3	0	2	Severe	2
268	12	12	5	11	7	5	3	3	3	3	3	3	3	3	Minor	2
269	0	0	0	0	0	0	0	0	0	0	0	0		0	NA	NA
271	8	7	4	1	4	5	3	3	2	1	3	1	2	1	Severe	3
275	12	12	8	10	10	10	3	3	3	3	3	3	3	1	Minor	1
277	11	11	7	9	11	7	2	3	3	3	3	3	3	2	Moderate	2
280	11	11	10	11	11	9	3	3	3	2	3	3	3	3	Minor	2
282	8	8	7	12	9	5	2	3	3	NA	3	3	3	3	Minor	1
283	11	5	5	8	5	3	3	3	3	3	3	1	3	1	Moderate	3
284	6	6	5	8	12	5	3	2	1	0	3	3	3	3	Moderate	3
286	1	1	1	7	5	3	1	0	0	0	2	3	2	1	Severe	2
287	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
288	10	10	5	12	12	12	3	3	3	1	3	3	3	3	Minor	1
289	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
290	0	0	0	0	7	7	0	0	0	0	0	3	3	1	Severe	4

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
293	1	1	1	7	9	3	0	0	0	1	2	3	3	1	Moderate	3
297	8	4	0	6	3	0	3	3	2	0	3	3	0	0	Severe	2
299	5	7	4	5	3	0	3	2	2	0	2	2	1	0	Moderate	2
300	12	10	8	12	11	7	3	3	3	3	3	3	3	3	Moderate	1
301	5	8	6	7	7	3	2	2	3	1	1	1	3	2	Minor	4
302	11	10	8	9	9	8	3	3	3	2	3	3	2	2	Moderate	4
304	10	11	12	12	12	11	3	3	3	3	3	3	3	3	Minor	3
305	11	9	5	7	9	4	3	3	3	2	3	3	3	2	Moderate	3
306	10	10	6	11	11	10	3	3	3	1	3	2	3	3	Minor	4
308	2	4	1	3	5	3	3	1	1	0	3	3	0	0	Moderate	2
315	11	10	5	11	7	6	3	3	3	3	2	3	3	3	Minor	0
318	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
330	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
331	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
333	3	4	2	7	7	2	2	1	2	1	3	2	2	2	Moderate	2
334	10	9	11	10	10	11	2	3	3	3	2	3	3	3	Minor	2
335	11	11	12	11	11	11	3	3	3	3	3	3	3	2	Minor	2
336	9	10	10	12	12	8	3	3	3	1	3	3	3	3	Minor	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
337	0	5	2	1	5	2	3	1	2	0	1	2	2	1	Moderate	2
338	9	9	7	0	1	4	3	3	3	1	NA	NA	NA	NA	Minor	1
341	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
342	10	12	12	7	6	3	3	3	3	2	3	2	3	0	Severe	3
344	7	4	0	5	5	3	3	2	2	1	3	3	2	1	Moderate	3
345	3	2	0	1	0	0	NA	NA	NA	NA	NA	NA	NA	NA	Moderate	3
347	4	6	8	7	6	6	3	3	2	3	3	3	3	NA	Moderate	4
349	5	5	2	7	8	10	1	1	3	1	3	2	3	1	Minor	1
351	8	10	2	10	9	2	3	3	2	1	3	3	3	2	Severe	2
356	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
368	12	12	11	12	12	12	3	3	3	3	3	3	3	3	Moderate	1
375	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
377	11	10	5	0	0	0	3	3	3	3	0	0	0	0	Minor	1
381	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
386	9	7	7	6	4	5	3	3	2	1	3	0	3	1	Moderate	3
390	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
393	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
400	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
404	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
408	1	5	5	7	5	0	2	2	3	0	0	3	3	1	Minor	3
409	8	10	4	3	6	2	2	3	3	3	2	2	2	1	Moderate	3
410	3	6	3	5	9	4	1	2	2	1	3	3	3	1	Minor	2
411	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
413	12	11	11	11	11	7	3	3	3	3	3	3	3	2	Minor	2
415	9	4	2	7	4	3	1	3	3	2	2	3	1	2	Moderate	3
417	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
420	7	3	7	0	1	1	2	3	2	0	1	0	0	0	Moderate	2
423	5	4	3	2	5	5	3	1	0	1	1	0	0	2	Moderate	2
425	1	1	1	1	0	0	2	0	0	0	1	0	0	0	Severe	3
427	0	0	3	0	0	1	0	1	2	0	1	0	0	0	Severe	3
428	6	4	0	5	3	0	0	3	3	2	2	3	2	0	Severe	1
432	12	12	9	11	9	9	3	3	3	3	2	2	3	3	Minor	1
433	6	6	2	8	8	3	2	1	3	1	3	2	3	NA	Minor	2
434	10	10	9	5	5	4	3	3	3	2	3	1	1	NA	Minor	3
435	9	6	3	8	10	9	3	2	2	3	2	3	3	1	Severe	3
437	11	9	6	12	12	4	3	3	3	2	3	3	3	2	Minor	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
439	5	3	3	6	8	3	NA	NA	NA	NA	1	3	2	1	Moderate	3
441	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
442	5	10	8	10	11	8	3	3	2	1	3	3	3	3	Minor	2
446	6	8	3	10	8	2	1	3	3	1	3	2	3	3	Minor	2
448	5	3	1	4	3	3	NA	2	2	1	1	3	NA	NA	Moderate	3
451	11	11	3	11	9	5	3	3	3	3	2	3	3	3	Minor	2
453	0	0	0	1	0	0	NA	NA	NA	NA	1	NA	NA	NA	Moderate	3
454	9	10	10	10	10	8	3	3	3	1	2	3	3	3	Minor	3
455	4	2	0	7	2	2	3	2	0	0	3	2	3	0	Minor	2
457	10	1	4	0	0	5	3	3	NA	3	1	NA	NA	1	Minor	2
460	1	3	2	5	3	3	3	3	NA	NA	3	NA	3	1	Severe	3
462	3	2	1	0	0	0	NA	NA	2	NA	NA	NA	NA	NA	Moderate	4
464	2	2	4	6	6	5	0	3	0	0	3	1	3	1	Severe	2
465	1	1	0	0	0	0	NA	NA	NA	1	NA	NA	NA	NA	Severe	2
468	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
473	0	3	2	1	2	1	3	0	0	0	2	0	0	0	Severe	3
480	8	8	5	8	10	7	2	3	3	1	3	3	3	1	Severe	3
481	10	8	3	11	10	2	2	3	3	3	3	3	3	2	Minor	1

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
482	3	1	0	1	1	0	NA	2	1	NA	NA	NA	1	NA	Severe	2
487	12	12	5	10	10	6	3	3	3	2	3	3	3	1	Minor	3
488	7	8	8	11	12	6	2	3	2	1	3	3	3	3	Moderate	3
491	0	0	0	0	1	4	0	0	0	0	1	3	0	1	Severe	2
493	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
494	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
498	6	10	1	10	10	4	3	1	3	1	2	3	3	2	Severe	1
499	12	11	9	12	12	12	3	3	3	2	3	3	3	3	Minor	1
513	10	12	6	9	11	8	3	3	3	3	3	3	3	1	Minor	1
525	11	6	6	8	6	1	3	3	3	2	2	2	3	NA	Severe	2
530	2	1	0	4	2	1	2	0	0	0	1	0	1	2	Severe	3
534	2	3	5	7	7	5	1	2	2	2	2	3	3	0	Minor	2
547	11	5	6	6	9	4	3	6	2	3	3	3	3	1	Minor	1
548	12	12	6	8	6	6	3	3	3	3	2	3	3	3	Moderate	2
549	4	1	1	7	3	0	3	0	0	1	2	3	1	1	Severe	4
552	12	8	6	10	11	5	3	3	3	3	3	3	3	3	Moderate	2
553	12	12	8	12	12	12	3	3	3	3	3	3	3	3	Minor	1
555	9	11	11	10	9	8	3	3	3	2	2	2	3	3	Minor	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
556	10	4	3	8	7	5	2	2	3	3	1	1	3	3	Minor	1
557	12	9	3	12	6	4	3	3	3	3	3	3	3	3	Minor	2
559	10	4	7	7	10	8	2	3	3	2	2	2	3	1	Moderate	1
560	7	2	2	7	4	5	3	2	1	1	2	2	3	NA	Minor	4
561	10	9	11	8	8	7	3	3	3	3	2	3	3	1	Minor	1
562	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
566	7	3	2	7	5	3	2	3	3	2	NA	2	2	3	Minor	1
567	7	8	9	2	2	0	2	2	3	2	1	1	1	0	Minor	1
568	4	8	3	9	8	3	0	3	3	2	2	3	3	1	Moderate	3
571	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
572	10	8	7	10	5	11	3	3	3	2	3	3	1	3	Moderate	3
577	0	2	2	0	0	0	2	0	0	0	0	0	0	0	Moderate	2
578	10	11	6	12	12	4	3	3	3	3	3	3	3	3	Minor	1
579	12	10	11	12	10	11	3	3	3	3	3	3	3	3	Minor	2
580	10	9	5	12	11	8	3	2	3	2	3	3	3	3	Minor	3
582	11	11	7	9	11	5	3	1	3	1	3	3	3	2	Minor	2
585	8	9	1	3	5	3	3	3	1	NA	2	2	2	NA	Moderate	3
587	11	10	9	10	10	3	3	3	2	3	3	3	3	1	Moderate	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
588	9	6	1	9	6	1	3	2	3	1	3	3	3	1	Moderate	2
589	6	3	1	12	9	3	3	2	NA	1	3	3	3	3	Moderate	2
591	12	12	10	12	12	11	3	3	3	3	3	3	3	3	Minor	1
597	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
599	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
600	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
602	3	0	2	0	0	0	3	0	0	0	0	0	0	0	Severe	2
603	12	11	9	12	12	9	3	3	3	2	3	3	3	3	Minor	2
604	12	8	6	11	11	10	3	3	3	3	3	3	3	2	Minor	2
605	10	6	2	8	4	5	3	2	3	3	2	3	3	NA	Moderate	2
606	8	5	3	10	8	4	3	1	3	1	3	3	3	2	Severe	1
607	11	9	4	9	8	5	3	3	3	1	3	2	3	3	Minor	2
609	11	12	3	11	11	5	3	3	3	3	2	3	3	3	Minor	2
612	10	4	4	10	8	6	3	2	3	3	3	3	3	2	Moderate	3
619	4	5	4	10	10	4	3	3	1	0	3	2	3	2	Moderate	2
620	7	5	4	8	6	5	3	3	1	2	3	0	3	2	Severe	3
622	0	0	0	5	3	2	0	0	0	0	2	1	2	1	Severe	1
625	8	0	1	6	2	1	3	2	0	3	2	2	0	3	Severe	4

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
626	7	4	1	11	8	2	2	2	2	1	2	3	3	3	Severe	4
628	9	7	4	10	8	9	3	3	3	1	3	3	3	2	Minor	2
631	7	5	1	7	5	1	1	2	3	1	3	2	2	1	Severe	4
634	7	9	4	7	8	0	3	3	2	0	1	1	2	0	Moderate	3
635	9	11	6	8	9	7	3	3	3	2	3	1	3	2	Moderate	3
637	3	8	3	6	8	1	3	NA	2	2	2	3	3	1	Severe	4
638	3	3	0	4	3	0	2	1	0	0	0	2	3	0	Moderate	1
640	3	5	6	1	2	2	3	2	1	0	2	0	0	0	Severe	4
641	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
642	3	5	3	5	5	2	1	2	1	NA	NA	2	3	1	Minor	3
644	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
645	8	9	4	10	9	4	3	3	3	0	3	3	3	2	Moderate	3
651	6	7	2	11	10	3	3	3	0	2	3	3	3	2	Severe	1
652	0	3	0	3	2	0	0	0	0	0	0	3	0	0	Severe	1
654	1	3	1	0	0	0	1	0	2	0	0	0	0	0	Moderate	2
656	8	5	2	10	10	4	3	2	2	1	3	2	3	2	Moderate	3
657	12	12	6	10	11	6	3	3	3	3	2	3	3	3	Minor	3
659	2	0	0	10	6	2	0	0	2	0	3	1	3	3	Severe	1

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	LML 7–9	LL 10–12	RU 1–3	RMU 4–60	RML 7–9	RL 10–12	Av. frag. score	Av.cort. pres
660	5	5	4	6	5	4	3	2	0	1	3	2	2	0	Severe	2
157i	9	9	9	8	5	5	3	3	3	3	3	2	3	0	Moderate	2
158i	12	9	1	10	9	2	3	3	3	3	3	3	2	2	Minor	1
158ii	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
191i	11	11	9	9	9	8	3	3	3	3	3	3	3	2	Minor	1
43ii	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
461i	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
461ii	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
533i	0	0	0	6	3	2	0	0	0	0	1	1	3	1	Moderate	3
76i	6	6	4	8	9	2	2	0	2	2	3	3	3	1	Moderate	1
82i	0	4	1	0	0	0	1	2	1	0	NA	NA	NA	NA	Minor	1
91ii	1	1	0	0	3	0	0	1	1	0	1	1	1	0	Severe	2

Black Gate-Visceral Surface Rib Lesions and Lower Respiratory Infection

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
8	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
18	0	0	NA	NA	0	0	NA	0	0	NA	0	0	0	NA	0	0	0	NA	Absent
22	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
23	0	0	NA	NA	0	0	0	0	0	NA	0	0	0	0	0	0	NA	0	Absent
27	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
31	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
33	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
40	0	0	NA	NA	0	0	0	0	0	NA	0	0	0	NA	0	0	0	NA	Absent
42	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
51	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
52	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	0	NA	NA	NA	0	Absent
53	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
54	0	0	NA	NA	0	0	0	NA	NA	NA	NA	0	NA	NA	NA	NA	NA	NA	Absent
56	NA	0	NA	NA	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
58	0	0	NA	NA	NA	0	0	0	0	0	0	NA	NA	NA	NA	NA	0	NA	Absent
63	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
66	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	NA	NA	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
69	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
70	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
71	0	0	NA	NA	NA	NA	NA	NA	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	Absent
74	0	0	NA	NA	0	0	0	0	0	0	0	NA	0	0	0	0	0	NA	Absent
75	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
77	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	0	NA	0	Absent
78	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
83	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
89	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
92	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
95	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
97	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
99	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0		Absent
102	0	0	NA	NA	NA	0	0	NA	NA	NA	NA	0	0	0	NA	NA	NA	NA	Absent
111	0	0	NA	NA	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
115	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
116	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
120	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
121	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	0	0	0	Absent
124	0	0	NA	NA	0	0	0	0	0	0	0	NA	0	0	0	0	0	NA	Absent
127	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
128	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
130	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
133	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
134	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
137	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
139	0	0	NA	NA	0	0	0	0	0	0	0	0	NA	NA	NA	NA	NA	NA	Absent
147	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
148	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
150	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	Absent
151	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
153	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
154	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
155	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
159	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
161	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
163	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	NA	0	Absent
164	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
167	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	0	0	NA	Absent
170	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
175	0	0	NA	NA	NA	NA	NA	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	0	Absent
176	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	Absent
177	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	0	0	0	Absent
178	7	8	NA	PRL	1	6	4	2	7	3	3	2	2	0	2	2	0	2	Present
179	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
181	0	2	R	PRL	0	0	0	1	1	0	0	0	0	0	0	1	1	0	Present
183	NA	4	NA	PRL	NA	NA	NA	0	0	4	NA	NA	NA	NA	2	2	0	0	Present
185	0	0	NA	NA	0	0	NA	0	0	0	NA	0	0	NA	0	NA	0	NA	Absent
186	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
188	0	0	NA	NA	NA	0	0	0	0	0	0	NA	NA	NA	0	0	0	0	Absent
190	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	0	0	NA	Absent
198	2	2	NA	PRL	1	2	0	1	2	0	2	0	0	0	2	0	0	0	Present
199	0	0	NA	NA	0	0	0	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
200	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
205	0	0	NA	NA	0	0	0	0	0	0	NA	0	0	NA	0	0	0	0	Absent
206	0	2	R	Both	0	0	0	0	0	1	0	0	0	0	1	0	0	0	Present
207	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
211	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	NA	0	NA	Absent
212	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
216	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
218	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
219	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
220	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
224	0	0	NA	NA	0	0	0	NA	NA	0	0	0	0	0	0	NA	NA	NA	Absent
229	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
236	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
240	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
242	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
243	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
245	0	0	NA	NA	0	0	0	0	0	0	NA	0	0	0	0	0	0	0	Absent
246	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
249	0	2	R	PRL	0	0	0	0	0	2	0	0	0	0	0	0	2	0	Present

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
250	0	0	NA	NA	0	0	0	0	0	0	0	0	NA	NA	0	NA	0	0	Absent
251	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	NA	0	NA	Absent
252	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
256	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
268	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
269	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
271	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
275	2	7	NA	Both	0	0	2	0	4	6	0	0	0	2	0	2	3	1	Present
277	3	0	L	DW	0	0	0	0	0	3	0	0	0	0	0	0	0	0	Present
280	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
282	8	12	NA	Both	8	8	2	10	11	0	0	4	4	0	2	4	4	2	Present
283	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
284	0	10	R	Both	0	0	0	9	3	0	0	0	0	0	1	3	3	3	Present
286	0	0	NA	NA	0	0	0	0	0	0	0	NA	NA	NA	0	0	0	0	Absent
287	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
288	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
289	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
290	0	0	NA	NA	NA	NA	NA	NA	0	0	NA	NA	NA	NA	NA	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
293	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
297	0	0	NA	NA	0	0	NA	0	0	NA	0	0	0	NA	0	0	NA	NA	Absent
299	0	0	NA	NA	0	0	0	0	0	NA	0	0	0	NA	0	0	0	NA	Absent
300	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
301	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
302	8	9	NA	Both	0	2	8	0	5	8	1	0	3	0	2	2	1	0	Present
304	2	0	L	PRL	0	0	2	0	0	0	0	0	2	0	0	0	0	0	Present
305	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
306	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
308	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	0	NA	NA	Absent
315	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
318	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
330	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
331	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
333	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
334	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
335	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
336	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
337	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
338	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
341	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
342	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	Absent
344	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
345	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
347	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
349	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
351	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
356	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
368	6	10	NA	Both	0	0	6	0	0	10	2	1	2	0	2	0	2	2	Present
375	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
377	0	0	NA	NA	0	0	0	NA	NA	NA	0	0	0	0	NA	NA	NA	NA	Absent
381	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
386	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	NA	0	0	Absent
390	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
393	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
400	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
404	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
408	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
409	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
410	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
411	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
413	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
415	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
417	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
420	0	0	NA	NA	0	0	0	NA	0	0	0	0	0	NA	0	NA	NA	NA	Absent
423	4	0	L	Both	4	2	0	0	0	0	2	1	NA	1	0	0	0	0	Present
425	0	0	NA	NA	0	0	0	0	NA	NA	0	NA	NA	NA	0	NA	NA	NA	Absent
427	1	0	L	PRL	NA	NA	1	NA	NA	0	NA	0	1	NA	0	NA	NA	NA	Present
428	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
432	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
433	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
434	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
435	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
437	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
439	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
441	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
442	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
446	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
448	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
451	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
453	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
454	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
455	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
457	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
460	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
462	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
464	0	0	NA	NA	0	0	0	0	0	0	NA	0	NA	NA	0	0	0	0	Absent
465	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
468	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
473	0	0	NA	NA	NA	0	0	0	0	0	0	NA	NA	NA	0	NA	NA	NA	Absent
480	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
481	9	11	NA	DW	11	9	6	11	9	2	3	3	3	2	2	3	3	3	Present

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
482	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
487	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
488	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
491	0	0	NA	NA	NA	NA	NA	NA	0	0	0	0	0	0	NA	NA	0	NA	Absent
493	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
494	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
498	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
499	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
513	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
525	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
530	0	0	NA	NA	0	0	NA	0	0	0	0	NA	NA	NA	0	NA	0	0	Absent
534	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	Absent
547	5	1	NA	PRL	5	2	1	0	0	1	3	2	0	0	0	0	0	0	Present
548	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
549	0	0	NA	NA	0	0	0	0	0	NA	0	NA	NA	0	0	0	0	0	Absent
552	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
553	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
555	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
556	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
557	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
559	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
560	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
561	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
562	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
566	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
567	10	1	NA	Both	8	8	9	2	2	0	2	3	3	2	1	1	2	0	Present
568	5	3	NA	PRL	0	1	5	0	3	3	NA	2	3	0	1	0	2	0	Present
571	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
572	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
577	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
578	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
579	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
580	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
582	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
585	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
587	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
588	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
589	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
591	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
597	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
599	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
600	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
602	0	NA	NA	NA	0	NA	0	NA	NA	NA	0	NA	NA	NA	NA	NA	NA	NA	Absent
603	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
604	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
605	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
606	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
607	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
609	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
612	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
619	2	3	NA	PRL	0	0	2	0	0	3	2	0	0	0	1	0	0	2	Present
620	3	6	NA	PRL	0	3	3	1	1	5	0	3	0	0	3	0	3	0	Present
622	NA	0	NA	NA	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
625	0	NA	NA	NA	0	NA	0	0	0	0	0	0	NA	0	0	0	NA	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
626	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
628	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
631	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA
634	0	0	NA	NA	0	0	0	0	0	NA	0	0	0	NA	0	0	0	NA	Absent
635	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
637	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
638	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
640	0	0	NA	NA	0	0	0	0	0	0	0	0	0	NA	0	NA	NA	NA	Absent
641	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
642	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
644	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
645	1	0	L	PRL	0	1	0	0	0	0	0	0	1	NA	0	0	0	0	Present
651	0	0	NA	NA	0	0	0	0	0	0	0	0	NA	0	0	0	0	0	Absent
652	0	NA	NA	NA	NA	0	NA	0	0	NA	NA	NA	NA	NA	NA	0	NA	NA	Absent
654	0	NA	NA	NA	0	0	0	NA	NA	NA	0	NA	0	NA	NA	NA	NA	NA	Absent
656	0	NA	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
657	1	0	L	DW	0	1	0	0	0	0	1	0	0	0	0	0	0	0	Present
659	0	0	NA	NA	0	NA	NA	0	0	0	NA	NA	0	NA	0	0	0	0	Absent

(Table continued on next page)

Skel. No	#LL	#RL	Uni. Side	Les. Type	No.L. R.N.L.	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LL MU 4–6	LL ML 7–9	LLL 10–12	LRU 1–3	LR MU 4–60	LR ML 7–9	LRL 10–12	Rib. Ind
660	0	0	NA	NA	0	0	0	0	0	0	0	0	NA	0	0	0	0	NA	Absent
157i	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	Absent
158i	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
158ii	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
191i	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
43ii	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
461i	0	0	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
461ii	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
533i	0	0	NA	NA	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
76i	0	0	NA	NA	0	0	0	0	0	0	0	0	0	0	0	0		0	Absent
82i	0	NA	NA	NA	NA	0	0	NA	NA	NA	0	0	0	NA	NA	NA	NA	NA	Absent
91ii	0	0	NA	NA	0	0	NA	NA	0	NA	NA	0	0	NA	0	0	0	NA	Absent

Coronation Street-Demography

Skeleton.No	Sex	Pooled.Sex	Age.Range
062	Male	Male	Mature.Adult
70	Male	Male	Mature.Adult
75	Female	Female	Adult
80.1	Female	Female	Young.Adult
100	Male	Male	Mature.Adult
107	Female	Female	Older.Adult
111	Unsexed	Unsexed	Adult
122	Male	Male	Mature.Adult
125	Male	Male	Mature.Adult
127	Female	Female	Mature.Adult
135	Male	Male	Older.Adult
159	Female	Female	Mature.Adult
179	Female	Female	Prime.Adult
184	Prob.Female	Female	Adult
189	Male	Male	Mature.Adult
195	Female	Female	Young.Adult
200	Female	Female	Mature.Adult
203	Female	Female	Prime.Adult
208	Male	Male	Older.Adult
217	Unsexed	Unsexed	Adult
222	Prob.Female	Female	Young.Adult
235	Male	Male	Older.Adult
238.1	Prob.Female	Female	Mature.Adult
241	Female	Female	Adult
243	Prob.Male	Male	Mature.Adult
249	Female	Female	Adult
256	Prob.Male	Male	Prime.Adult
264	Prob.Female	Female	Older.Adult
268	Prob.Male	Male	Mature.Adult
274	Female	Female	Older.Adult
284	Male	Male	Mature.Adult

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age.Range
289	Prob.Male	Male	Older.Adult
294	Female	Female	Adult
300	Female	Female	Prime.Adult
301	Unsexed	Unsexed	Prime.Adult
305	Prob.Male	Male	Adult
315	Prob.Female	Female	Adult
323	Male	Male	Prime.Adult
332	Male	Male	Mature.Adult
344	Unsexed	Unsexed	Mature.Adult
346	Male	Male	Mature.Adult
348	Male	Male	Adult
353	Male	Male	Prime.Adult
358	Prob.Male	Male	Mature.Adult
371	Male	Male	Prime.Adult
371	Male	Male	Prime.Adult
375	Prob.Female	Female	Mature.Adult
381	Unsexed	Unsexed	Adult
390	Male	Male	Older.Adult
404	Female	Female	Older.Adult
410	Male	Male	Young.Adult
419	Female	Female	Older.Adult
430	Prob.Male	Male	Young.Adult
435	Prob.Male	Male	Mature.Adult
442	Male	Male	Older.Adult
447	Prob.Female	Female	Older.Adult
472	Prob.Female	Female	Adult
479	Female	Female	Prime.Adult
492	Prob.Male	Male	Prime.Adult
497	Prob.Male	Male	Mature.Adult
502	Female	Female	Older.Adult
507	Female	Female	Older.Adult
512	Male	Male	Mature.Adult

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age.Range
517	Female	Female	Prime.Adult
523	Male	Male	Mature.Adult
532	Female	Female	Mature.Adult
547	Male	Male	Mature.Adult
555	Male	Male	Mature.Adult
559	Prob.Male	Male	Prime.Adult
564	Female	Female	Older.Adult
578	Female	Female	Adult
579	Prob.Male	Male	Prime.Adult
596	Female	Female	Prime.Adult
601	Male	Male	Mature.Adult
606	Male	Male	Mature.Adult
613	Prob.Female	Female	Prime.Adult
623	Prob.Female	Female	Prime.Adult
613ii	Female	Female	Young.Adult
623	Female	Female	Young.Adult
627	Prob.Male	Male	Mature.Adult
639	Female	Female	Older.Adult
644	Prob.Female	Female	Mature.Adult
649	Female	Female	Young.Adult
814	Prob.Male	Male	Prime.Adult
888	Unsexed	Unsexed	Mature.Adult
889	Unsexed	Unsexed	Adult
898	Male	Male	Adult
902	Prob.Female	Female	Older.Adult
906	Prob.Male	Male	Prime.Adult
909	Female	Female	Prime.Adult
913	Unsexed	Unsexed	Adult
917	Female	Female	Adult
920	Prob.Male	Male	Adult
925	Female	Female	Older.Adult
926	Unsexed	Unsexed	Mature.Adult

(Table continued on next page)

Skeleton.No	Sex	Pooled.Sex	Age.Range
928	Male	Male	Prime.Adult
933	Unsexed	Unsexed	Adult
935	Male	Male	Mature.Adult
941	Female	Female	Young.Adult
945	Prob.Female	Female	Young.Adult
947	Female	Female	Older.Adult
952	Female	Female	Older.Adult
954	Unsexed	Unsexed	Adult
956	Female	Female	Mature.Adult
963	Unsexed	Unsexed	Adult
965	Prob.Male	Male	Mature.Adult
969	Unsexed	Unsexed	Adult
970	Unsexed	Unsexed	Adult
974	Male	Male	Older.Adult
980	Unsexed	Unsexed	Adult
1000	Unsexed	Unsexed	Adult
987	Prob.Female	Female	Mature.Adult
991	Female	Female	Mature.Adult
995	Male	Male	Prime.Adult
998	Female	Female	Mature.Adult
1005	Unsexed	Unsexed	Adult
1007i	Unsexed	Unsexed	Adult
1010	Unsexed	Unsexed	Adult
1011	Unsexed	Unsexed	Mature.Adult
1015	Prob.Female	Female	Adult
11002	Prob.Female	Female	Mature.Adult
11007	Male	Male	Older.Adult
11008	Prob.Female	Female	Adult

Coronation Street-Mastoiditis

Skeleton. No	No. Ob.M.	M.L	M.R	M. Adult	M. Childhood	A.C. B.H.	U.B. Childh.	U.B. Adult
62	1	NA	LSH	Present	Present	Both	NA	NA
70	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
75	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
80.1	0	NA	NA	NA	NA	NA	NA	NA
100	2	Absent	S.Hyp	Present	Present	Both	Bi	Uni
107	2	LSH	LSH	Present	Absent	Adult	NA	Bi
111	1	NA	LSH	Present	Present	Both	NA	NA
122	0	NA	NA	NA	NA	NA	NA	NA
125	0	NA	NA	NA	NA	NA	NA	NA
127	0	NA	NA	NA	NA	NA	NA	NA
135	2	LSH	LSH	Present	Present	Both	Bi	Bi
159	0	NA	NA	NA	NA	NA	NA	NA
179	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
184	1	LSH	NA	Present	Present	Both	NA	NA
189	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
195	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
200	0	NA	NA	NA	NA	NA	NA	NA
203	0	NA	NA	NA	NA	NA	NA	NA
208	2	Absent	Hyper	Absent	Present	Childhood	Uni	NA
217	0	NA	NA	NA	NA	NA	NA	NA
222	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
235	0	NA	NA	NA	NA	NA	NA	NA
238.1	0	NA	NA	NA	NA	NA	NA	NA
241	2	Absent	LSH	Present	Present	Both	Uni	Uni
243	1	NA	Absent	Absent	Present	Childhood	NA	NA
249	0	NA	NA	NA	NA	NA	NA	NA
256	2	LSH	S.Hyp	Present	Present	Both	Uni	Bi
264	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
268	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
274	2	LSH	LSH	Present	Present	Both	Uni	Bi

(Table continued on next page)

Skeleton. No	No. Ob.M.	M.L	M.R	M. Adult	M. Childhood	A.C. B.H.	U.B. Childh.	U.B. Adult
284	2	LSH	Absent	Present	Present	Both	Bi	Uni
289	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
294	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
300	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
301	0	NA	NA	NA	NA	NA	NA	NA
305	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
315	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
323	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
332	2	LSH	LSH	Present	Absent	Adult	NA	Bi
344	2	S.Hyp	LSH	Present	Absent	Adult	NA	Bi
346	0	NA	NA	NA	NA	NA	NA	NA
348	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
353	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
358	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
371	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
371	0	NA	NA	NA	NA	NA	NA	NA
375	0	NA	NA	NA	NA	NA	NA	NA
381	0	NA	NA	NA	NA	NA	NA	NA
390	0	NA	NA	NA	NA	NA	NA	NA
404	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
410	0	NA	NA	NA	NA	NA	NA	NA
419	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
430	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
435	1	Absent	NA	Absent	Present	Childhood	NA	NA
442	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
447	0	NA	NA	NA	NA	NA	NA	NA
472	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
479	2	Hyper	Hyper	Absent	Absent	Healthy	NA	NA
492	0	NA	NA	NA	NA	NA	NA	NA
497	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
502	0	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skeleton. No	No. Ob.M.	M.L	M.R	M. Adult	M. Childhood	A.C. B.H.	U.B. Childh.	U.B. Adult
507	2	S.Hyp	S.Hyp	Present	Present	Both	Uni	Bi
512	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
517	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
523	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
532	2	LSH	LSH	Present	Absent	Adult	NA	Bi
547	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
555	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
559	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
564	0	NA	NA	NA	NA	NA	NA	NA
578	1	NA	Absent	Absent	Present	Childhood	NA	NA
579	2	Hyper	S.Hyp	Present	Absent	Adult	NA	Uni
596	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
601	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
606	2	LSH	LSH	Present	Absent	Adult	NA	Bi
613	1	Hyper	NA	Present	Absent	Adult	NA	NA
623	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
613ii	0	NA	NA	NA	NA	NA	NA	NA
623	0	NA	NA	NA	NA	NA	NA	NA
627	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
639	2	S.Hyp	S.Hyp	Present	Present	Both	Bi	Bi
644	0	NA	NA	NA	NA	NA	NA	NA
649	2	LSH	LSH	Present	Absent	Adult	NA	Bi
814	0	NA	NA	NA	NA	NA	NA	NA
888	0	NA	NA	NA	NA	NA	NA	NA
889	1	NA	S.Hyp	Present	Absent	Adult	NA	NA
898	1	Hyper	NA	Absent	Absent	Healthy	NA	NA
902	1	NA	Absent	Absent	Present	Childhood	NA	NA
906	0	NA	NA	NA	NA	NA	NA	NA
909	0	NA	NA	NA	NA	NA	NA	NA
913	0	NA	NA	NA	NA	NA	NA	NA
917	1	NA	Absent	Absent	Present	Childhood	NA	NA

(Table continued on next page)

Skeleton. No	No. Ob.M.	M.L	M.R	M. Adult	M. Childhood	A.C. B.H.	U.B. Childh.	U.B. Adult
920	0	NA	NA	NA	NA	NA	NA	NA
925	0	NA	NA	NA	NA	NA	NA	NA
926	1	NA	Absent	Absent	Present	Childhood	NA	NA
928	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
933	0	NA	NA	NA	NA	NA	NA	NA
935	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
941	0	NA	NA	NA	NA	NA	NA	NA
945	1	NA	Absent	Absent	Present	Childhood	NA	NA
947	0	NA	NA	NA	NA	NA	NA	NA
952	2	S.Hyp	S.Hyp	Present	Absent	Adult	NA	Bi
954	0	NA	NA	NA	NA	NA	NA	NA
956	2	Hyper	Absent	Absent	Present	Childhood	Bi	NA
963	0	NA	NA	NA	NA	NA	NA	NA
965	1	S.Hyp	NA	Present	Absent	Adult	NA	NA
969	0	NA	NA	NA	NA	NA	NA	NA
970	0	NA	NA	NA	NA	NA	NA	NA
974	1	NA	Hyper	Absent	Absent	Healthy	NA	NA
980	0	NA	NA	NA	NA	NA	NA	NA
1000	1	LSH	NA	Present	Absent	Adult	NA	NA
987	1	Absent	NA	Absent	Present	Childhood	NA	NA
991	0	NA	NA	NA	NA	NA	NA	NA
995	2	Absent	Hyper	Absent	Present	Childhood	Bi	NA
998	2	S.Hyp	Hyper	Present	Absent	Adult	NA	Uni
1005	2	LSH	LSH	Present	Present	Both	Bi	Bi
1007i	0	NA	NA	NA	NA	NA	NA	NA
1010	0	NA	NA	NA	NA	NA	NA	NA
1011	0	NA	NA	NA	NA	NA	NA	NA
1015	2	Absent	Absent	Absent	Present	Childhood	Bi	NA
11002	0	NA	NA	NA	NA	NA	NA	NA
11007	2	Absent	LSH	Present	Present	Both	Uni	Uni
11008	1	NA	Hyper	Absent	Absent	Healthy	NA	NA

Coronation Street-Maxillary Sinusitis

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
62	0	NA	NA	NA	NA
70	0	NA	NA	NA	NA
75	1	Absent	NA	Absent	NA
80.1	0	NA	NA	NA	NA
100	0	NA	NA	NA	NA
107	2	Spicules	Spicules	Present	Bi
111	0	NA	NA	NA	NA
122	0	NA	NA	NA	NA
125	0	NA	NA	NA	NA
127	0	NA	NA	NA	NA
135	0	NA	NA	NA	NA
159	0	NA	NA	NA	NA
179	0	NA	NA	NA	NA
184	2	Spicules	Absent	Present	Uni
189	0	NA	NA	NA	NA
195	2	Absent	Spicules	Present	Uni
200	0	NA	NA	NA	NA
203	0	NA	NA	NA	NA
208	0	NA	NA	NA	NA
217	0	NA	NA	NA	NA
222	1	NA	Spicules	Present	NA
235	0	NA	NA	NA	NA
238.1	0	NA	NA	NA	NA
241	0	NA	NA	NA	NA
243	1	NA	Absent	Absent	NA
249	0	NA	NA	NA	NA
256	2	Spicules	Spicules	Present	Bi
264	1	Absent	NA	NA	NA
268	0	NA	NA	NA	NA
274	0	NA	NA	NA	NA
284	0	NA	NA	NA	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
289	0	NA	NA	NA	NA
294	1	Spicules	NA	Present	NA
300	0	NA	NA	NA	NA
301	0	NA	NA	NA	NA
305	0	NA	NA	NA	NA
315	0	NA	NA	NA	NA
323	0	NA	NA	NA	NA
332	2	Absent	Absent	Absent	NA
344	2	Spicules	Spicules	Present	Bi
346	0	NA	NA	NA	NA
348	0	NA	NA	NA	NA
353	0	NA	NA	NA	NA
358	0	NA	NA	NA	NA
371	0	NA	NA	NA	NA
371	0	NA	NA	NA	NA
375	0	NA	NA	NA	NA
381	0	NA	NA	NA	NA
390	2	Absent	Absent	Absent	NA
404	0	NA	NA	NA	NA
410	0	NA	NA	NA	NA
419	1	NA	Spicules	Present	NA
430	0	NA	NA	NA	NA
435	0	NA	NA	NA	NA
442	0	NA	NA	NA	NA
447	0	NA	NA	NA	NA
472	0	NA	NA	NA	NA
479	0	NA	NA	NA	NA
492	0	NA	NA	NA	NA
497	0	NA	NA	NA	NA
502	0	NA	NA	NA	NA
507	1	NA	Absent	Absent	NA
512	2	W.Pitted	W.Pitted	Present	Bi

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
517	0	NA	NA	NA	NA
523	2	Absent	Absent	Absent	NA
532	0	NA	NA	NA	NA
547	0	NA	NA	NA	NA
555	0	NA	NA	NA	NA
559	0	NA	NA	NA	NA
564	2	Spicules	Spicules	Present	Bi
578	2	Spicules	Spicules	Present	Bi
579	1	Spicules	NA	Present	NA
596	2	Spicules	Pitting	Present	Bi
601	0	NA	NA	NA	NA
606	1	NA	Absent	Absent	NA
613	0	NA	NA	Absent	NA
623	2	Spicules	Spicules	Present	Bi
613ii	0	NA	NA	NA	NA
623	2	Spicules	Spicules	Present	Bi
627	1	W.Pitted	NA	Absent	NA
639	0	NA	NA	NA	NA
644	1	NA	Spicules	Present	NA
649	0	NA	NA	NA	NA
814	0	NA	NA	NA	NA
888	0	NA	NA	NA	NA
889	0	NA	NA	NA	NA
898	2	Absent	Absent	Absent	NA
902	2	Spicules	Spicules	Present	Bi
906	0	NA	NA	NA	NA
909	0	NA	NA	NA	NA
913	0	NA	NA	NA	NA
917	0	NA	NA	NA	NA
920	0	NA	NA	NA	NA
925	0	NA	NA	NA	NA
926	0	NA	NA	NA	NA

(Table continued on next page)

Skeleton.No	No.Ob.Max.S	M.S.L	M.S.R	M.S.Ind	M.S.Uni.Bi.
928	0	NA	NA	NA	NA
933	0	NA	NA	NA	NA
935	2	W.Pitted	Absent	Present	Uni
941	0	NA	NA	NA	NA
945	0	NA	NA	NA	NA
947	1	NA	Rem.Spicules	Present	NA
952	0	NA	NA	NA	NA
954	0	NA	NA	NA	NA
956	0	NA	NA	NA	NA
963	0	NA	NA	NA	NA
965	2	Spicules	Spicules	Present	Bi
969	0	NA	NA	NA	NA
970	0	NA	NA	NA	NA
974	0	NA	NA	NA	NA
980	0	NA	NA	NA	NA
1000	0	NA	NA	NA	NA
987	0	NA	NA	NA	NA
991	2	Spicules	Spicules	Present	Bi
995	0	NA	NA	NA	NA
998	2	Absent	Spicules	Present	Uni
1005	1	NA	Spicules	Present	NA
1007i	0	NA	NA	NA	NA
1010	2	Absent	Absent	Absent	NA
1011	0	NA	NA	NA	NA
1015	2	Absent	Absent	Absent	NA
11002	1	Spicules	NA	Present	NA
11007	2	Absent	Absent	Absent	NA
11008	2	Absent	Absent	Absent	NA

Coronation Street-Rib Presence

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
62	10	12	8	11	11	10	3	3	2	3	3	3	3	2	Minor	2
70	9	9	6	9	8	6	2	2	3	2	3	3	3	0	Moderate	2
75	4	4	1	3	3	2	3	2	0	0	2	2	1	0	Severe	2
80.1	5	1	0	2	0	0	3	2	0	0	1	1	0	0	Severe	1
100	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
107	12	10	11	11	10	11	3	3	3	3	3	3	2	3	1	Minor
111	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
122	12	12	12	1	1	4	3	3	3	3	0	0	0	1	Minor	2
125	7	6	11	4	7	6	3	3	2	2	1	3	3	1	Severe	3
127	12	12	7	10	7	9	3	3	3	3	3	3	3	1	Minor	2
135	11	11	10	12	10	10	2	3	3	3	3	3	3	3	Minor	3
159	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
179	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
184	2	2	2	2	3	1	2	0	1	0	0	2	0	0	Minor	2
189	9	11	9	11	8	9	2	3	3	3	3	3	3	3	Moderate	3
195	8	8	6	7	7	6	3	3	3	0	3	3	3	0	Minor	2
200	3	3	4	6	5	5	0	0	0	3	1	0	3	3	Moderate	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
203	11	11	9	11	10	4	3	3	3	2	3	3	3	2	Minor	2
208	3	2	2	0	2	4	0	0	3	0	0	0	2	0	Severe	3
217	9	11	3	0	0	0	3	3	2	2	0	0	0	0	Moderate	3
222	7	9	2	3	7	3	3	3	3	0	3	3	2	0	Moderate	4
235	3	2	0	3	3	4	1	2	0	0	2	1	0	0	Moderate	2
238.1	9	9	5	11	11	10	3	2	3	2	3	2	3	3	Moderate	2
241	1	1	3	1	0	0	1	0	0	0	0	1	0	0	Severe	1
243	11	11	12	9	7	1	3	3	3	3	2	3	3	2	Severe	2
249	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
256	7	9	5	4	5	4	1	2	3	3	3	3	0	0	Severe	2
264	8	4	11	12	10	11	2	3	3	3	3	3	3	3	Severe	2
268	0	0	0	0	0	4	0	0	0	0	2	1	2	0	Severe	2
274	12	12	6	9	9	2	3	3	3	3	3	3	0	3	Moderate	2
284	10	11	8	11	11	8	3	3	3	3	3	3	3	3	Moderate	2
289	9	10	10	9	11	11	3	3	3	2	3	3	3	3	Moderate	2
294	8	7	4	9	7	4	2	3	1	2	2	1	3	3	Severe	2
300	9	9	5	9	10	5	3	2	1	3	3	3	2	2	Severe	3
301	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
305	12	11	7	9	6	8	3	3	3	3	3	3	1	3	Moderate	2
315	9	11	11	5	4	2	3	3	1	2	0	3	2	1	Severe	2
323	9	10	8	11	11	9	3	3	3	2	3	3	3	3	Severe	3
332	11	9	11	11	11	11	2	3	3	3	3	3	3	3	Moderate	3
344	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
346	5	5	6	6	6	4	2	3	0	0	2	1	3	1	Severe	2
348	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
353	11	8	8	9	10	10	2	3	3	3	3	3	2	2	Severe	2
358	9	8	10	6	6	5	3	3	2	1	3	3	0	0	Minor	2
371	10	10	7	8	5	12	2	3	3	2	3	3	2	3	Severe	2
371	10	10	7	8	5	12	2	3	3	2	3	3	2	3	Severe	2
375	0	0	0	8	7	4	0	0	0	0	1	2	3	2	Severe	2
381	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
390	5	6	2	11	5	3	3	0	2	0	3	3	3	2	Severe	2
404	11	11	11	10	11	5	3	3	2	3	3	2	3	3	Moderate	2
410	10	10	8	9	9	6	3	3	3	2	2	3	2	2	Moderate	2
419	8	7	12	12	12	12	3	3	1	1	3	3	3	3	Minor	1
430	12	11	11	12	12	12	3	3	3	3	3	3	3	3	Minor	1
435	9	8	5	11	12	7	3	2	2	3	3	3	3	3	Severe	3

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
442	8	7	9	9	6	9	3	2	3	1	2	3	3	2	Severe	2
447	1	1	0	4	4	1	2	0	0	0	3	1	0	0	Severe	3
472	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
479	12	12	12	12	12	9	3	3	3	3	3	3	3	3	Minor	3
492	12	12	7	11	11	6	3	3	3	3	3	3	3	2	Moderate	2
497	5	5	2	1	2	2	3	2	0	0	2	0	0	0	Severe	3
502	6	1	0	9	7	3	2	2	2	0	3	2	3	2	Severe	2
507	11	11	12	10	7	3	3	3	3	3	3	3	3	1	Severe	2
512	12	11	10	10	12	7	3	3	3	3	3	3	3	3	Moderate	2
517	11	5	10	12	9	8	3	3	3	3	3	3	3	3	Severe	2
523	1	1	0	11	11	3	0	1	0	0	3	3	3	2	Moderate	3
532	11	10	8	9	8	7	3	3	3	3	2	3	3	1	Severe	2
547	12	11	10	9	10	8	3	3	3	3	3	2	3	1	Moderate	2
555	11	11	9	12	12	9	3	3	3	3	3	3	3	3	Moderate	2
559	8	5	8	12	12	6	3	3	2	0	3	3	3	3	Moderate	2
564	12	12	12	0	0	0	3	3	3	3	0	0	0	0	Moderate	2
578	9	9	7	0	0	1	3	3	3	0	0	0	0	0	Severe	2
579	0	0	0	12	12	12	0	0	0	0	3	3	3	3	Moderate	3

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
596	11	11	11	12	9	7	3	3	3	3	3	3	3	3	Major	2
601	12	12	6	12	12	10	3	3	3	3	3	3	3	3	Major	2
606	10	10	12	10	11	7	3	3	3	3	3	3	3	2	Major	1
613	12	12	12	0	0	0	3	3	3	3	0	0	0	0	Minor	2
623	0	0	0	12	12	6	0	0	0	0	3	3	3	3	Severe	2
613ii	12	12	12	0	0	0	3	3	3	3	0	0	0	0	Moderate	2
623	0	0	0	11	12	8	0	0	0	0	3	3	3	3	Severe	1
627	10	9	8	11	11	8	3	3	2	2	3	3	2	3	Severe	3
639	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
644	9	8	7	11	11	10	3	3	2	1	2	3	3	3	Severe	3
649	7	9	9	8	9	9	3	3	3	0	3	3	3	0	Minor	1
814	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
888	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
889	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
898	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
902	0	0	0	7	7	1	0	0	0	0	3	3	1	0	Severe	1
906	3	3	5	0	0	0	0	0	0	3	0	0	0	0	Severe	1
909	0	0	0	0	0	3	0	0	0	0	0	0	0	0	Severe	2

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
913	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
917	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
920	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
925	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
926	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
928	12	12	12	12	12	9	3	3	3	3	3	3	3	3	Minor	2
933	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
935	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
941	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
945	4	4	3	1	1	0	1	3	0	0	0	0	1	0	Moderate	2
947	10	10	9	5	5	6	3	3	3	1	2	3	0	1	Moderate	2
952	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
954	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
956	10	10	9	10	10	9	3	3	3	2	3	3	2	2	Moderate	3
963	3	3	1	1	1	0	0	1	2	0	0	0	0	1	Severe	2
965	9	10	11	9	6	12	3	3	1	3	3	3	3	3	Moderate	2
969	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
970	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA

(Table continued on next page)

Skel. No	No.ob. R.N.L	No.ob. R.A.L.	No.ob. R.S.L.	No.ob. R.N.R.	No.ob. R.A.R.	No.ob. R.S.R.	LU 1–3	LMU 4–6	MLM 7–9	LL 10–12	RU 1–3	RMU 4–6	RML 7–9	RL 10–12	Av. frag. score	Av. cort. pres.
974	10	10	5	9	7	7	3	3	3	1	3	1	3	3	Severe	2
980	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
1000	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
987	10	7	3	7	10	10	3	3	1	3	3	3	3	1	Severe	2
991	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
995	2	2	2	1	1	0	1	0	0	1	0	0	0	1	Severe	1
998	8	11	5	8	10	3	3	3	3	3	2	3	3	2	Severe	2
1005	2	2	4	7	8	4	1	2	0	0	3	1	3	0	Severe	2
1007i	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
1010	12	12	10	12	12	12	3	3	3	3	3	3	3	3	Minor	3
1011	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
1015	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
11002	9	8	3	4	7	6	3	3	3	0	3	3	1	0	Severe	2
11007	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	NA
11008	2	2	3	7	8	9	2	0	0	0	3	2	3	0	Severe	2

Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
62	0	0	0	0	4	0	0	0	0	0	1	3	0	0	Present
70	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
75	0	0	0	0	0	0	0	0	NA	NA	0	0	0	NA	Absent
80.1	0	0	NA	0	NA	0	0	0	NA	NA	0	0	NA	NA	Absent
100	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
107	0	1	0	3	0	0	0	1	0	0	1	2	0	0	Present
111	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
122	0	2	0	0	0	0	0	0	2	0	0	0	0	0	Present
125	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
127	0	0	0	3	6	2	0	0	0	0	1	3	3	0	Present
135	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
159	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
179	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
184	0	0	0	0	0	0	0	NA	0	NA	NA	0	NA	NA	Absent
189	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
195	0	0	0	0	0	0	0	0	0	0	0	0	0	0	NA
200	0	2	3	0	0	2	NA	NA	NA	3	0	NA	2	0	Present
203	0	1	0	0	0	0	0	0	1	0	0	0	0	0	Present

(Table continued on next page)

Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
208	0	0	1	0	0	1	NA	NA	1	NA	NA	NA	1	NA	Present
217	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	Absent
222	1	0	0	0	0	0	0	1	0	NA	0	0	0	NA	Present
235	3	2	0	0	0	0	1	2	NA	NA	0	0	0	0	Present
238.1	1	3	1	2	6	2	2	1	0	1	1	1	3	1	Present
241	0	0	0	0	NA	NA	0	NA	NA	NA	NA	0	NA	NA	Absent
243	2	2	6	2	2	0	0	3	1	2	0	0	2	0	Present
249	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
256	3	0	0	0	0	0	0	1	2	0	0	0	0	0	Present
264	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
268	0	0	0	0	0	1	0	0	0	0	1	0	0	0	Present
274	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
284	0	0	0	0	2	3	0	0	0	0	0	0	3	0	Present
289	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
294	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
300	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
301	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
305	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
315	2	0	0	0	0	0	0	2	0	0	NA	0	0	0	Present

(Table continued on next page)

Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
323	0	2	5	2	4	8	0	0	3	2	0	3	2	3	Present
332	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
344	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
346	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
348	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
353	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
358	0	0	0	0	0	0	0	0	0	0	NA	NA	NA	NA	Absent
371	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
371	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
375	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
381	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
390	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
404	2	2	5	0	0	0	0	2	0	3	0	0	0	0	Present
410	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
419	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
430	0	0	1	9	0	0	0	0	0	1	0	3	3	3	Present
435	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
442	0	0	0	0	0	2	0	0	0	0	0	0	2	0	Present
447	0	0	NA	0	0	0	0	NA	NA	NA	0	0	NA	NA	Absent

(Table continued on next page)

Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
472	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
479	0	5	0	1	9	2	1	0	2	2	3	3	3	3	Present
492	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
497	0	0	0	0	0	0	0	0	NA	NA	0	NA	NA	NA	Absent
502	0	0	NA	1	0	0	0	0	0	NA	0	0	0	1	Present
507	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
512	0	0	Both	0	0	4	2	0	1	1	2	0	2	0	Present
517	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
523	0	0	NA	0	0	0	NA	0	NA	NA	0	0	0	0	Absent
532	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
547	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
555	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
559	0	0	0	0	0	0	0	0	0	NA	0	0	0	0	Absent
564	0	0	0	NA	NA	NA	0	0	0	0	NA	NA	NA	NA	Absent
578	6	0	0	NA	NA	0	0	3	3	NA	NA	NA	NA	NA	Present
579	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
596	0	0	0	4	0	0	0	0	0	0	0	3	0	1	Present
601	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
606	0	0	0	0	0	0	0	0	0	0	0	0	0	NA	Absent

(Table continued on next page)

Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
613	0	0	0	NA	NA	NA	0	0	0	0	NA	NA	NA	NA	Absent
623	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
613ii	0	0	0	NA	NA	NA	0	0	0	0	NA	NA	NA	NA	Absent
623	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	0	Absent
627	2	0	0	0	0	0	0	2	0	0	0	0	0	0	Present
639	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
644	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
649	0	0	0	0	0	0	0	0	0	NA	0	0	0	NA	Absent
814	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
888	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
889	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
898	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
902	NA	NA	NA	0	0	0	NA	NA	NA	NA	0	0	0	NA	Absent
906	0	0	0	NA	NA	NA	NA	NA	NA	0	NA	NA	NA	NA	Absent
909	NA	NA	NA	NA	NA	0	NA	NA	NA	NA	NA	NA	NA	NA	Absent
913	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
917	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
920	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
925	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
926	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
928	12	12	12	12	0	2	3	3	3	3	3	3	3	3	Present
933	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
935	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
941	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
945	2	0	0	0	0	NA	0	2	NA	NA	NA	NA	NA	0	Present
947	0	0	0	0	0	0	0	0	0	0	0	0	NA	0	Absent
952	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
954	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
956	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
963	0	0	0	0	0	NA	NA	0	0	NA	NA	NA	NA	0	Absent
965	0	0	0	5	0	0	0	0	0	0	2	3	0	0	Present
969	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
970	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
974	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
980	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1000	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
987	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
991	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA

(Table continued on next page)

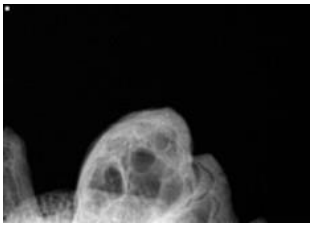
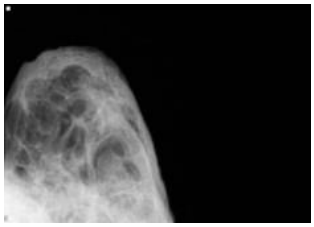
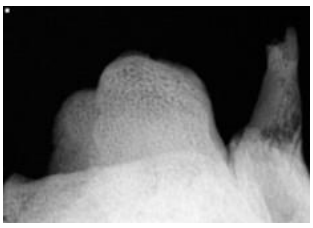
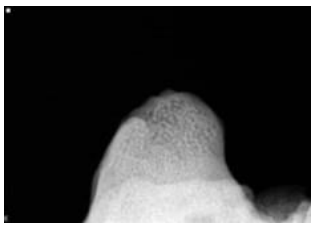
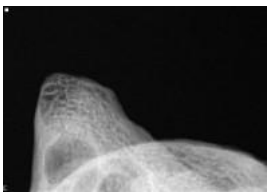
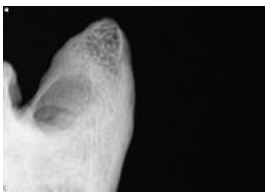
Skel. No	No.L. R.N.L	No.L. R.A.L.	No.L. R.S.L.	No.L. R.N.R.	No.L. R.A.R.	No.L. R.S.R.	LLU 1–3	LLMU 4–60	LLML 7–9	LLL 10–12	LRU 1–3	LRMU 4–60	LRML 7–9	LRL 10–12	Rib.Ind
995	0	0	0	0	0	NA	0	NA	NA	0	NA	NA	NA	0	Absent
998	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
1005	0	0	0	0	0	0	0	0	NA	NA	0	0	0	NA	Absent
1007i	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1010	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent
1011	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
1015	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
11002	0	0	0	0	0	0	0	0	0	NA	0	0	0	NA	Absent
11007	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA	NA
11008	0	0	0	0	0	0	0	0	0	0	0	0	0	0	Absent

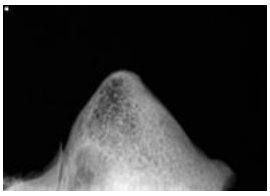
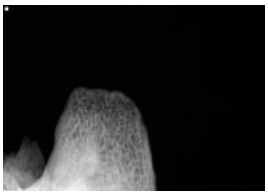
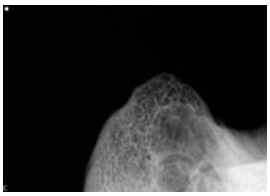
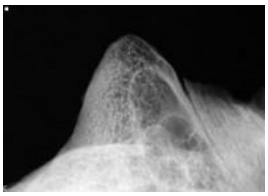
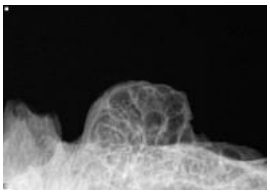
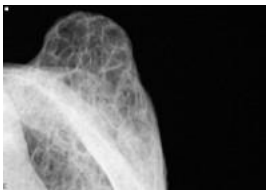
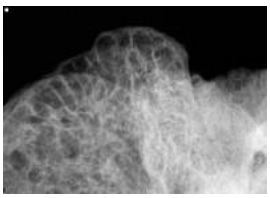
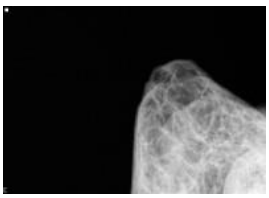
Appendix B

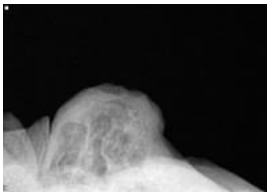
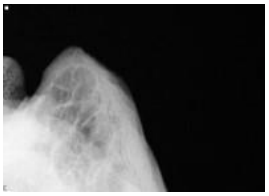
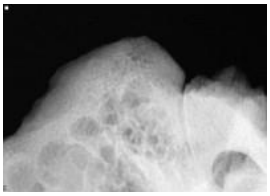
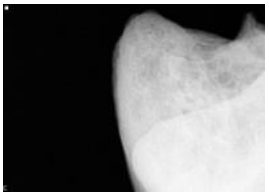
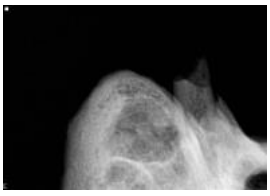
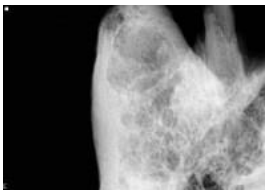
This appendix includes radiographs from a selection of individuals from both populations. A selection of radiographs is included, because including all the radiographs created such a large file that it was unusable. If you would like to see all the radiographs taken during this project, please do not hesitate to contact me, as I believe strongly in data sharing.

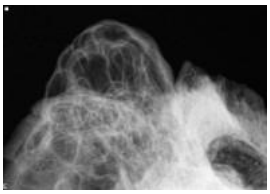
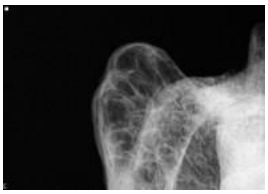
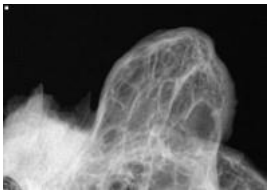
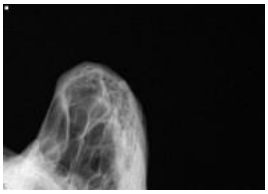
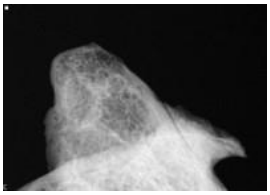
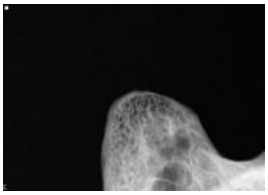
Radiographs are presented per individual and by plane. The right hand corner summarises the diagnosis of the mastoid air cells and for the individual. When X-rays were taken in the inferior-superior plane or photos were taken of the temporal bone, those appear here, too. The acronyms that appear in the figures are defined below.

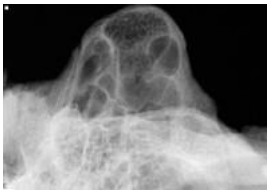
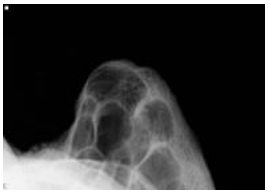
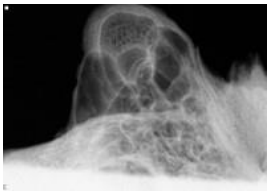
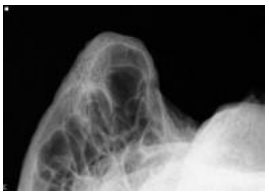
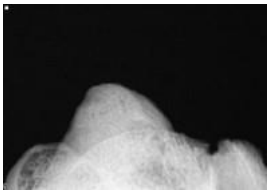
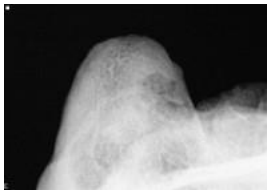
BG/CS – The Black Gate or Coronation Street individual number
L/R – Left or right mastoid process
ML – Medial-lateral plane
AP – Anterior-posterior plane
IS – Inferior-superior plane
NA – Not applicable
L – The type of left mastoid air cell structure
R – The type of right mastoid air cell structure
Ind. – Adult mastoiditis present or absent
Childhood – Residual childhood mastoiditis present or absent
A./C./Both – Adult, residual childhood, or both types of mastoiditis
Uni-/Bilateral Ch. – Unilateral or bilateral residual childhood mastoiditis
Uni-/Bilateral A. – Unilateral or bilateral adult mastoiditis

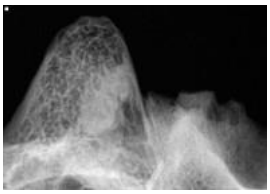
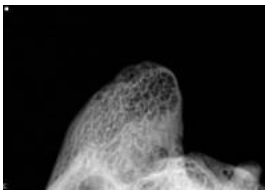
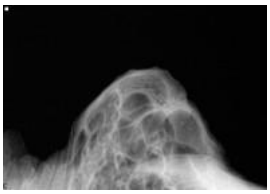
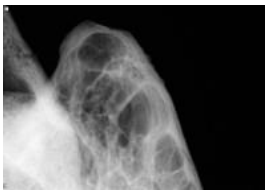
	ML	AP	IS	
BG22 L			NA	L: S. Hyp. R: S. Hyp. Ind.: Present Childhood: Present A./C./Both: Both Uni-/Bilateral Ch.: Uni. Uni-/Bilateral A.: Bi.
BG22 R			NA	
	ML	AP	IS	L: L.S.H. R: NA Ind.: Present Childhood: Present A./C./Both: Both Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
BG71 L			NA	
BG71 R	NA	NA	NA	

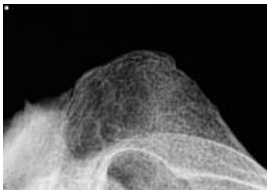
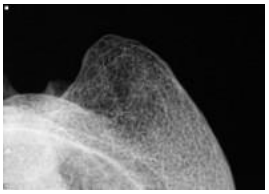
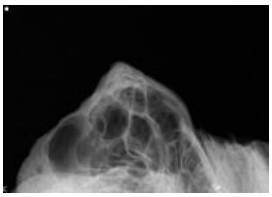
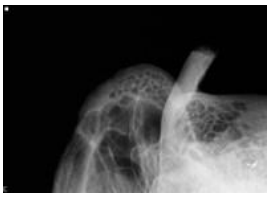
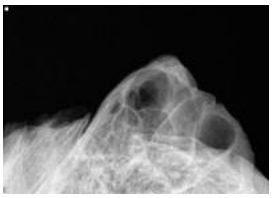
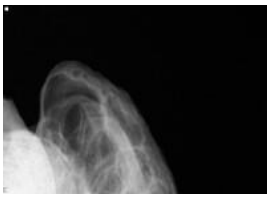
	ML	AP	IS	
BG151 L			NA	L: S. Hyp. R: S. Hyp. Ind.: Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: Bi.
BG151 R			NA	
	ML	AP	IS	
BG157i L			NA	L: S. Hyp. R: S. Hyp. Ind.: Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: Bi.
BG157i R			NA	

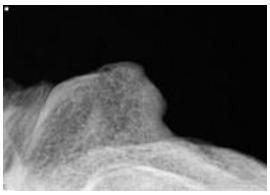
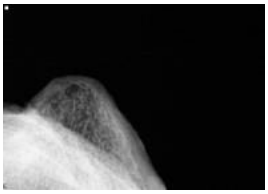
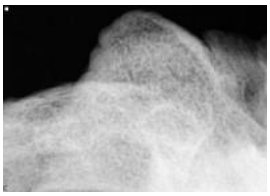
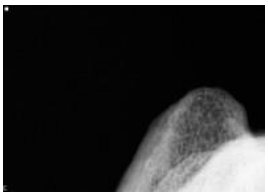
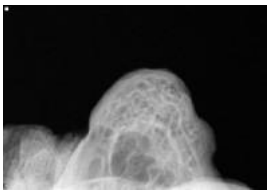
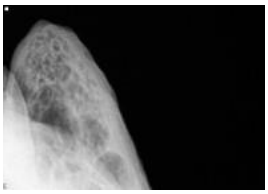
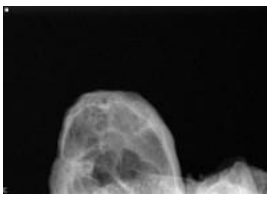
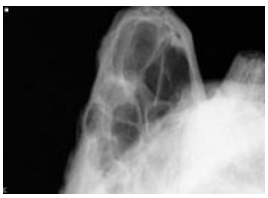
	ML	AP	IS	
BG164 L			NA	L: S. Hyp. R: S. Hyp. Ind.: Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: Bi.
BG164 R			NA	
	ML	AP	IS	L: NA R: L.S.H. Ind.: Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
BG175 L	NA	NA	NA	
BG175 R			NA	

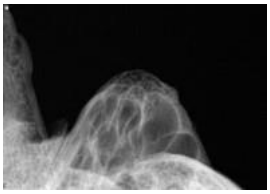
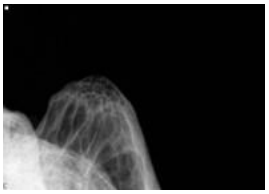
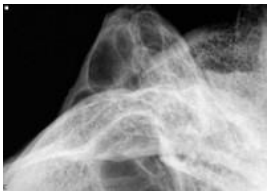
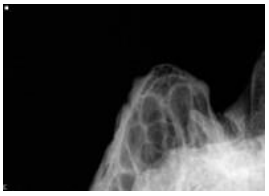
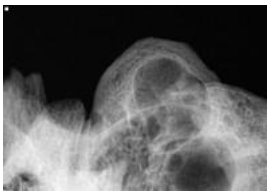
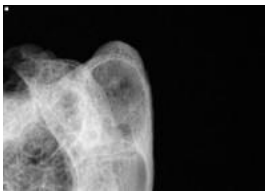
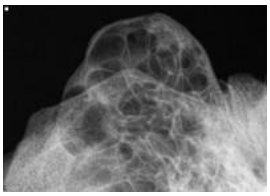
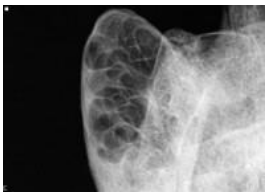
	ML	AP	IS	L: NA R: Hyper. Ind.: Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
BG176 L	NA	NA	NA	
BG176 R			NA	
	ML	AP	IS	L: Hyper. R: S. hyp. Ind.: Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: Uni.
BG185 L			NA	
BG185 R			NA	

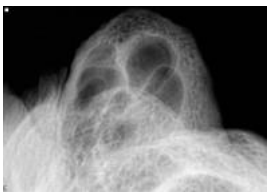
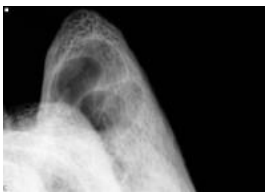
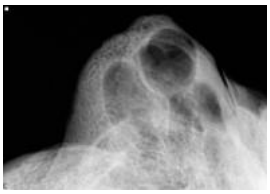
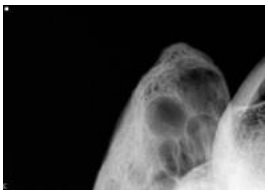
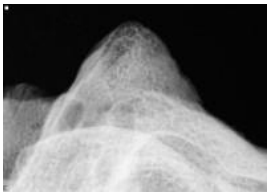
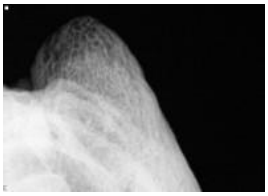
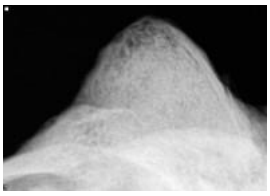
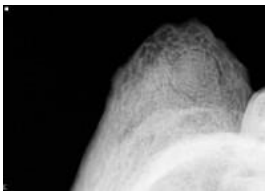
	ML	AP	IS	
BG206 L			NA	L: Hyper. R: Hyper. Ind.: Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: Bi.
BG206 R			NA	
	ML	AP	IS	
BG216 L	NA	NA	NA	L: NA R: L.S.H. Ind.: Present Childhood: Present A./C./Both: Both Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
BG216 R			NA	

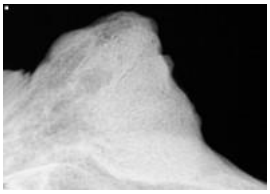
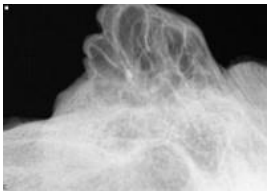
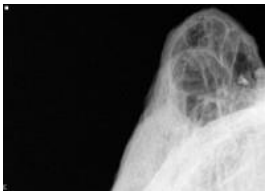
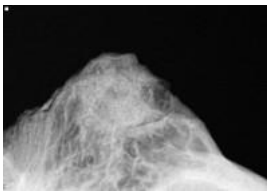
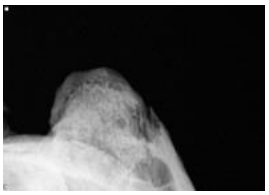
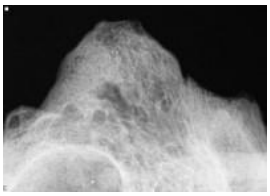
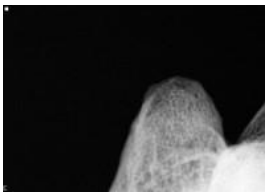
	ML	AP	IS	L: NA R: LSH Ind.: Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
BG491 L	NA	NA	NA	
BG491 R			NA	
	ML	AP	IS	**L**: Hyper **R**: NA **Ind.**: Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS179 L			NA	
CS179 R	NA	NA	NA	

	ML	AP	IS	
CS184 L			NA	**L:** LSH **R:** NA **Ind.:** Present Childhood: Present A./C./Both: Both Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS184 R	NA	NA	NA	
	ML	AP	IS	
CS195 L			NA	**L:** Hyper **R:** Hyper **Ind.:** Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS195 R			NA	

	ML	AP	IS	
CS294 L			NA	L: Absent R: Absent Ind.: Absent Childhood: Present A./C./Both: Childhood Uni-/Bilateral Ch.: Bi Uni-/Bilateral A.: NA
CS294 R			NA	
	ML	AP	IS	
CS315 L			NA	L: Hyper R: Hyper Ind.: Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS315 R			NA	

	ML	AP	IS	
CS375 L			NA	**L:** Hyper **R:** Hyper **Ind.:** Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS375 R			NA	
	ML	AP	IS	
CS404 L			NA	**L:** Hyper **R:** Hyper **Ind.:** Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS404 R			NA	

	ML	AP	IS	
CS442 L			NA	L: Hyper R: Hyper Ind.: Absent Childhood: Absent A./C./Both: Healthy Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: NA
CS442 R			NA	
	ML	AP	IS	
CS497 L			NA	L: S.Hyp R: S.Hyp Ind.: Present Childhood: Present A./C./Both: Both Uni-/Bilateral Ch.: Bi Uni-/Bilateral A.: Bi
CS497 R			NA	

	ML	AP	IS	
CS507 L			NA	**L:** S.Hyp **R:** S.Hyp **Ind.:** Present Childhood: Present A./C./Both: Both Uni-/Bilateral Ch.: Uni Uni-/Bilateral A.: Bi
CS507 R			NA	
	ML	AP	IS	
CS532 L			NA	**L:** LSH **R:** LSH **Ind.:** Present Childhood: Absent A./C./Both: Adult Uni-/Bilateral Ch.: NA Uni-/Bilateral A.: Bi
CS532 R			NA	