WHO CARES?: Examining the biological and social impacts of leprosy on adolescents in Medieval England through clinical and isotopic models of care.

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CHAPTER 1. INTRODUCTION

1.1 BROAD RESEARCH CONTEXT

Leprosy, also known as Hansen's Disease or Mycobacterial neurodermatosis is defined a chronic infectious disease of the peripheral nervous system caused by *Mycobacterium leprae* or *Mycobacterium lepromatosis* (Butlin and Lockwood 2020). Contrary to social beliefs, the rates of morbidity are low today, affecting only a small percentage of people exposed (~5%) (Goulart and Goulart 2008; Suzuki et al. 2012). Current notions of leprosy usually conjure up Medieval imaginings of extreme social stigma and negligent treatment, but these perceptions are not anachronistic for people with the disease today. Those infected with leprosy still carry sentences of discrimination, leading to seclusion from their communities (Heijnders 2004; Rafferty 2005; Jacob and Franco-Paredes 2008; Sermrittirong and van Brakel 2014; Mankar et al. 2011). This inappropriate treatment can be linked to racist and colonialist medical campaigns from the 18th to 20th centuries AD that promulgated the idea that the negative treatment of people with leprosy in the Medieval period was key to the disease's eradication (Ashmead 1897; Edmond 2006:65-102). However, more recent historical and archaeological evidence counter these long-accepted beliefs (Touati 2000; Roberts 2002; Rawcliffe 2006; Roffey and Tucker 2012; Brenner 2010; Roberts 2013; Roberts 2018; Roberts 2020: 280). Because historically derived stigma and discrimination are cited as a primary reason for the continued global burden of the disease (World Health Organization 2015; 2019), it is worth considering the early contexts in which leprosy established its footing in England.

This thesis combines contextual data, palaeopathological analyses, amelogenin peptide and multi-isotope methods, and clinical models to view the biological and social impacts of leprosy in the past through a lens of "caring" for those affected. Analyses were conducted on individuals who were affected by lepromatous leprosy with a particular focus on adolescent populations from two Early to Late Medieval (c. 9th – 12th centuries AD) archaeological sites in England: the parish cemetery of St. John at the Castle Gate/Timberhill (Norwich, in modern day Norfolk) and the North Cemetery of the St. Mary Magdalen Leprosy Hospital (Winchester, modern day Hampshire). Adolescents

were specifically selected for analyses due to their uniquely liminal biological and social status (Gowland and Penny-Mason 2018; Lewis 2016), making them well suited to reflect the capacity of societal potential for care.

The age of adolescence in this research is broadly designated to individuals between c. 10-25 years old in line with modern biological (Sawyer et al. 2016; Patton et al. 2016) and Medieval social definitions (Gilchrist 2012:34; Cochelin 2013:3-6; Lewis 2016). Biological alterations to the body commences with adrenarche and continues through the "pubertal deceleration" period; they are marked by periods of interstitial growth, rapid metabolic turnover, significant endocrine changes, and immune precariousness (Auchus and Rainey 2004; Clark and Kumar 2009:996-997; Klein and Flanagan 2016; Lewis et al. 2016). The combination of these effects have the potential to lead to a more severe leprosy manifestation, thus making adolescents the ideal cohort to view rapid biological changes, and gauge resulting social responses and outcomes through cross-disciplinary models of care and treatment.

1.1.1 Contextualising leprosy and medical care in the early-late medieval transition (9th – 12th centuries AD)

The latter centuries of the Early Medieval period (c. 9th – 11th centuries AD) in England is populated with major socio-political, economic, and environmental changes, almost unabated by conflict (Higham and Ryan 2013:232-442). Viking invasions of England (from the 8th century AD onwards) punctuated the Early Medieval period and transformed the cultural landscape with large swathes of land, particularly in the North and the East, falling under Danelaw (Higham and Ryan 2013: 235). During this 'Viking Age' the Saxon city of Winchester, in modern day Hampshire, became the primary royal administrative centre and the seat of power facilitating the unification of England, imbuing its culturally mixed inhabitants with a unique 'Anglo-Danish' identity (Roffey and Lavelle 2015: 17-25). This period also saw widespread increases in urban settlements, food production, trade networks, social stratification, and notable increases in temperature (i.e. the Medieval Warm Period from c. 900-1200 AD) (Gardiner 2011; O'Connell and Hull 2011; Ryan and Higham 2013:232-283; Roffey and Lavelle 2015: 7-25; Hughes and Diaz 1994: 134; Mann et al. 2009). The increases in temperature in the Northern Hemisphere during the 'Medieval

Warm Period' hypothetically catalysed many of these changes by creating a more amenable environment for economic and population growth, and widespread colonisation (Lamb 1965; Hughes and Diaz 1994: 128-133; Broecker 2001). It is against this fluctuating backdrop that increases in palaeopathological evidence for leprosy, the advancement of medicine, and the transformation of the hospital (and establishment of leprosaria) occurs.

1.1.2 Leprosy in the Early Medieval period (5th – 11th centuries AD)

Some possible skeletal evidence for leprosy in England exists prior to the Early Medieval period (Roberts 2020: Appendix 3), but it is not until the 6th – 7th centuries AD onwards that we see a marked increase in prevalence rates for leprosy in the archaeological record (see Chapter 2.4.3). Presently, the earliest confirmed evidence of leprosy in England comes from the remains of an Early Medieval adult male (415-545 cal AD) from Great Chesterford (Essex) (Inskip et al. 2015). Much like other areas of Europe during this time (with the exception of Hungary; see Chapter 2) the evidence for leprosy within cemeteries is sparse and usually confined to one or two cases. By the latter centuries of the Early Medieval period (c. 9th and 10th centuries AD), cemeteries in England begin to reveal multiple individuals with skeletal lesions diagnostic of lepromatous leprosy in parish sites (e.g. St. John at the Castle Gate/Timberhill in Norwich, Norfolk) and in early leprosy hospital cemeteries (e.g. St. Mary Magdalen in Winchester, Hampshire). This foundation and proliferation of leprosy evidence makes the Early Medieval period a prime setting for understanding early social and biological impacts of the disease and its subsequent changes and continuities into the Late Medieval transition (11th -12th centuries AD). Many historians often uncritically assert that individuals who developed the disease during these formative contexts were met with ostracism and community exclusion; however, very little evidence for leprosy and its subsequent treatment (social or medical) exists prior to the 12th century AD (Rubin 1970; Roffey 2012). This research set out to address this incongruence by evaluating current historical and archaeological evidence with a critical lens and combining this evidence with cross-disciplinary methods (e.g. palaeopathological analyses, clinical models, multi-isotope analyses, amelogenin peptide analyses) to further nuance leprosy in its Medieval context. The true impacts of disease *per se* are predominantly social rather than biological phenomena (Inhorn and

Brown 2000; Cunningham 2002). Therefore, in assessing the gravity of these impacts, we must further explore social concepts of care in the past and their bearing on people with leprosy.

1.1.3 Medical care, hospitals, and the leprosarium in the early-Late Medieval transition

Both documentary and archaeological evidence for the origins and daily life within leprosaria is scarce, and therefore relies upon a synthesis of geographically and chronologically broad data sets. In the pre-Christian eras, nascent forms of hospitals appear in the historical records of China, India, Mesopotamia, and Sri Lanka in the 1st millennium BC but, in the West, the idea of hospitality embodied a different meaning (Retief and Cilliers 2006). The words hospital and hospice are Latin derivatives of *hospitium*, which is translated as the reception of a guest, hospitality/room, or friendly entertainment (Dainton 1961:17-18; Huggon 2018). Early European hospitals (4th – 10th centuries AD) served three distinct functions: places of refuge for travellers and pilgrims; shelter for the indigent; and eventually places for the care (both spiritual and medical) of the infirm (Orme and Webster 1995; Retief and Cilliers 2006). From the early Christian-era (4th century AD), hospitals/hospices gradually transitioned from places of refuge to charitable, purpose-built institutions, like leprosaria, in the 10th - 11th centuries (Retief and Cilliers 2006; Huggon 2018). These changes were incited by radical reforms implemented by St. Benedict of Nursia (6th century AD) included a mandate for all monasteries to provide a separate space dedicated to the care and treatment of the sick (de Waal 1995; Orme and Webster 1995: 17-18; Retief and Cilliers 2006). Major initiatives, known as the *Rule of St. Benedict*, were formally codified in England by Bishop Aethelwold of Winchester (c. 950), and explicitly adopted by all monasteries and nunneries by 973 AD under the *Regularis Concordia* (Orme and Webster 1995: 17-18; Kornexl 2014). It is under this monastic model that the leprosarium in England also emerged and operated (Rawcliffe 2006: 326). Under the *Rule*, sick inmates occupying these spaces were to be given the utmost importance and afforded food (e.g. good quality meat), clothing, shelter, regular baths, and care directed at their health problems (Orme and Webster 1995:17-19; de Waal 1995:64). Despite the *Rule* mandating that care

for sick take precedence in these monastic-run institutions, little evidence exists regarding what that 'care' may have looked like in practice, and the social mores surrounding it. Evidence for lived experiences within leprosy hospitals is limited, and most previous research has relied upon mixtures of broad and anecdotal data sets, leading to interpretations that these institutions were neglectful communes for expelled and unwanted individuals on the fringes of society, with negligible care after admittance (Brody 1975: 68-75; Richards 2000: 48-53; Orme and Webster 1995: 24-31).

Medical practitioners in the Early Medieval period were known as 'leeches' and although they largely interacted with the community within monastic institutional frameworks, they were not necessarily part of the clerical community (Rubin 1970). For example, Bede describes the physician Cynefrid (c. 680 AD), who attended the Abbey of Ely for 16 years as not having a bound monastic position (*ibid*). The dualistic nature of the physician as a secular and non-secular position is further evidenced by the founding of a "school" formed by Archbishop Theodore in Canterbury in the 7th century AD for the purpose of training of leeches (physicians) from wide geographical and religious spheres (*ibid*). Perhaps the best example, however, is the compilation of *Bald's Leechbooks*, a c. 10th century AD medical compendium that has direct links with the royal seat in Winchester, and possibly the St. Mary Magdalen Leprosy Hospital also in Winchester during the time of its initial foundation (Cockayne 1865; 1866; Rubin 1970; Roffey 2012). *Bald's Leechbook*s recommend a range of medical treatments for ailments and illnesses (including leprosy; see Chapter 2), and are conspicuously devoid of religious overtones (Cockayne 1865; 1866). Historians cite spiritual and moral corruption in the Medieval mindset as the primary aetiological factor in leprosy contraction and development (Brody 1974: 55-56; Watts 1997: 41-43; Richards 2000: 71-72; Covey 2001), however, the secularism evident in Early Medieval medical treatments may signify an acknowledgement of the separation of body and soul in mainstream medical and social thought in pre-Conquest England (Rubin 1970). This body/soul duality disappears in the Later Medieval period (c. 12th – 16th centuries) with a more restricted approach to the administration of hospital care and function placing spiritual and moral cleanliness above an individual's biological needs (Orme and Webster 1995: 56-64). If alternative views regarding leprosy contraction and treatment existed in the Early Medieval mindset,

people may have responded differently to the disease both within their communities and institutionally.

The role and function of the leprosy hospital during its formative period is even less well-defined. The earliest notion of a leprosarium is attributed to St. Basil of Caesarea of Cappadocia (modern-day Turkey), who founded the Basilica at Caesarea in 369 AD that featured an area dedicated to the care of individuals with leprosy (Retief and Cilliers 2006). Although firm evidence for leprosy in England is present from the 5th century AD onwards (Inskip et al. 2015), evidence detailing the formation of the leprosarium is sparse. The earliest dated leprosarium in Western Europe is the medieval hospital of St. Mary Magdalen (Winchester; 9th – 12th centuries AD)(Roffey and Marter 2012; Roffey 2012). Here, archaeological evidence for Early Medieval timber structures, a small masonry chapel, and a carefully constructed cemetery (North Cemetery) with individuals with skeletal lesions diagnostic of leprosy suggests a community of care (Roffey and Tucker 2012). At some point in the mid-12th century, a new chapel with associated masonry structures, including an infirmary, were built over the initial leprosarium and North Cemetery at St. Mary Magdalen, and a new cemetery (South Cemetery) laid south of the re-founded chapel (Roffey and Marter 2012; Roffey and Tucker 2012).

Although some documentary evidence for early hospitals exist (see Table 1.1), there is currently no archaeological evidence for leprosy hospital structures prior to the initial phase of St. Mary Magdalen in Winchester (Roffey 2012). However, the Late Saxon (9th – 10th centuries AD) cemetery at St. John at the Castle Gate/Timberhill (Norwich, Norfolk) warrants some consideration. Here, the remains of up to 35 out of 184 individuals show skeletal lesions consistent with, or diagnostic of, lepromatous leprosy. Archaeologists highlight similar genetic affinities and burial rites (e.g. earthen pillows, flints supporting the skulls, organic coffins) between individuals buried at St. John with the nearby, contemporaneous Farmer's Avenue cemetery, and suggest St. John's may have been constructed as an overflow burial ground (Shepherd Popescu 2009:268-269). However, it is worth noting that both the location of St. John's at the Castle entrance and its access to a secure water source (via the Great Cockey streams) is mimicked in 12th century AD leprosaria after the expansion of the Norwich Castle walls (Rawcliffe 2006: 312-313;

Shepherd Popescu 2009: 269). It could therefore be that St. John at the Castle Gate may present as a very early leprosarium community, and any associated early leprosarium timber buildings could have been destroyed by building works in the post-Conquest era (c. 11th century).

In the post-Conquest period (c. 1066 AD to 15th century AD), Huggon (2018) estimates approximately 1100 hospitals operated in England and Wales, with approximately one-third functioning as leprosaria (see Table 1.1 for early institutions). The increase in leprosaria in England sharply rose from the 12th century onwards (Roberts 1986). Many reason that these leprosy hospitals did not provide any care *per se* and cite their peripheral locations as evidence that they were purposely-neglected communes for people forcefully expelled from their home communities (Brody 1974: 66-67; Watts 1997:49-63; Richards 2000:134-139; Orme and Webster 1995:24-31; Covey 2001). However, Rawcliffe (2006:307-309) has highlighted that leprosaria, like other major hospitals, were conspicuously situated in locations with significant footfall, potentially enabling the collection of charitable donations from passers-by to maintain the support of these institutions from the Early Norman period (11th – 12th centuries AD) onwards. Leprosaria and people with leprosy were also favoured beneficiaries of Late Saxon and Early Norman kings, queens, noblemen, bishops, and abbots, who paid patronage to these institutions in the forms of monetary compensation, and donations of food, land, and palliative care (e.g. publicly washing feet and cleaning sores) (*ibid: 302-314*; Table 1.1). Archbishop Lanfranc (c. 1089 AD), the Archbishop of Canterbury and trusted counsellor to William the Conqueror, described people with leprosy as an earthly representation of the suffering of Christ, and deserving of charity and not ostracism (Rawcliffe 2006:122; Roffey 2012). Lanfranc endowed one of the first post-Norman leprosy hospitals at Harbledown (modern day Kent), where he ensured the preservation of Saxon medical practices and Benedictine traditions in the immediate Early-Late Medieval transition period (Rawcliffe 2006: 305; Roffey 2012). The Harbledown leprosarium shows archaeological similarities to the initial leprosarium at St. Mary Magdalen (e.g. adjacent to important roads, exclusive access to water, placed on a hill, timber buildings surrounding a chapel)(Roffey 2017).

Most archaeological comparative information comes from leprosaria that operated during the Later Medieval period (i.e. post 12th – 16th centuries AD), and although they do not offer a contemporary perspective, they provide a glimpse of daily life within the leprosarium. For example, the St James and Mary Magdalene leprosarium in Chichester (12^{th} – 15^{th} centuries AD) revealed approximately 24% of individuals with skeletal lesions consistent with lepromatous leprosy (Lee and Magilton 1989). According to documentary evidence, the St. James and Mary Magdalene leprosarium, like others in England, was segregated by sex and inmates were provided food, shelter, and clothing from the charity and gifts various benefactors (Lee and Magilton 2008:265). Other documentary evidence from the Later Medieval period indicates that inmates of leprosaria received tolls from food entering the cities including flour, grain, salt, cheeses, fish, fruit, and other foodstuffs (Rawcliffe 2006: 313-314). Many leprosaria gained tax exempt status in the 13^{th} century and issues of monetary improprieties by priors and black market sales of donations and gifts began to change civic views of these charitable institutions (Rawcliffe 2006: 299-300, 314-322; Magilton 2008:58)

Further afield, the St. Jørgen's cemetery (13^{th} – 16^{th} centuries AD) in Naevsted, Sjaelland (Denmark) was excavated by physician Møller-Christensen who first detailed the bone changes associated with leprosy (1961; 1978). The cemetery revealed 70% of individuals had bone lesions diagnostic of leprosy (Møller-Christensen 1978), and carbon and nitrogen analyses by Brozou and colleagues (2019) demonstrated a diet of terrestrial C_3 diet with a small amount of fish. Interestingly, like its English counterparts, the St. Jørgen leprosarium came under scrutiny for monetary improprieties, ultimately resulting in the Danish Crown taking over Church properties in the 16^{th} century AD (Andersen 1969:83-85). Whilst these comparative institutions are helpful in their respective contexts, they only offer a glimpse of institutional life from a later chronological perspective. Hospital administration and practice seems to have shifted from local monastic oversight to broader civil authority oversight from the 12^{th} century AD onwards (Orme and Webster 2005: 20-31; Retief and Cilliers 2006), therefore these pre-12^{th} century AD formative contexts perhaps provide a more accurate reflection of the social attitudes towards leprosy at this time.

TABLE 1.1 – Chronological list of Early-Late Medieval hospitals and leprosaria in England founded before 1150 AD (after Page 1905; Page 1906; Page 1971; Cockburn et al. 1969; Rubin 1970; Satchell 1998; Rawcliffe 2006; Atkins and Popescu 2010; Roffey 2012).

LOCATION	DATE FOUNDED	DEDICATION	PATRON	MONARCH
WINCHESTER (HAMPSHIRE)	c. 931-934	St. John?	Bishop Beornstan	King Aethelstan
YORK (YORKSHIRE)	c. 937	St. Peter's	King Aethelstan	King Aethelstan
WORCESTER (WORCESTERSHIRE)	< 992	(unconfirmed)	St. Oswald (Archbishop of York)	Æthelred the Unready
WORCESTER (WORCESTERSHIRE)	c. 1066	(unconfirmed)	St. Wulfstan (Bishop of Worcester)	Edward the Confessor
LAUNCESTON (CORNWALL)	c.1068	St. Leonard	Earl Brian	William the Conqueror
ROCHESTER	c. 1078	St. Bartholomew	Bishop Gundulf of Rochester	William the Conqueror
HARBLEDOWN (KENT)	c. 1087	St. Nicholas	Archbishop Lanfranc	William the Conqueror
CHATHAM (KENT)	c. 1092	St. Bartholomew	Bishop Gundulf of Rochester	William II
HOLBORN (LONDON)	c. 1101	St. Giles	Queen Maud	Henry I
COLCHESTER (ESSEX)	c. 1101-1103	St. Mary Magdalen	Eudo Dapifer	Henry I
WHITBY (YORKSHIRE)	c. 1109	St. Michael	Whitby Abbey	Henry I
DUDSTON (GLOUCESTERSHIRE)	< 1115	St. Mary Magdalene	Ivo of Chartres	Henry I
SPROWSTON (NORFOLK)	< 1119	St. Mary Magdalen	Bishop Herbert de Losinga	Henry I
PETERBOROUGH (CAMBRIDGESHIRE)	c. 1125	St. Leonard	Abbot John de Seez	Henry I
OXFORD (OXFORDSHIRE)	1126	St. Bartholomew	The Crown	Henry I
READING (BERKSHIRE)	<1135	St. Mary Magdalen	Abbot Ancher	Henry I
BURY ST. EDMUNDS (SUFFOLK)	<1135	St. Peter's	Abbot Anselm	Henry I
AYELSBURY (BUCKINGHAMSHIRE)	< 1135	St. Leonard & St. John the Baptist	Robert Ilhale, William	Henry I

			atte Hide, William son of Robert, John Palnok, Samson son of William, and Reginald Wauncy	
COLCHESTER (ESSEX)	< 1135	St. Mary Magdalen	St. John's Abbey	Henry I
LINCOLN (LINCOLNSHIRE)	< 1135	Holy Innocents	Burton Lazars	Henry I
NEWCASTLE (TYNE AND WEAR)	< 1135	St. Mary Magdalen	Town of Newcastle	Henry I
WARWICK (WARWICKSHIRE)	c. 1135	St. Michael	Earl of Warwick	Henry I
SHREWSBURY (SHROPSHIRE)	1136	St. Giles	The Crown & Shrewsbury Abbey	Stephen
RIPON (YORKSHIRE)	< 1139	St. Mary Magdalen	Archbishop Thurstan	Henry I/Stephen
BUCKLAND (DOVER)	1141	St. Bartholomew	Dover Priory	Stephen
YORK (YORKSHIRE)	1142	St. Nicholas	The Crown	Stephen
KING'S LYNN (NORFOLK)	< 1145	St. Mary Magdalen	Peter the Chaplain	Stephen
ST. ALBANS (HERTFORDSHIRE)	c. 1129-1146	St. Julian	Geoffrey de Gorham	Henry I/Stephen
WINCHESTER (HAMPSHIRE)	c. 1148	St. Mary Magdalen (Re-foundation)	Bishop Henry de Blois	Stephen
NORTHAMPTON (NORTHAMPTONSHIRE)	c. 1150	St. Leonard	Town of Northampton	Stephen

Although contrary archaeological and historical evidence (Touati 2000; Roberts 2002; Rawcliffe 2006; Roffey and Tucker 2012; Brenner 2010; Roberts 2013; Roberts 2018; Roberts 2020: 280) does not completely dismiss the notion that people with leprosy may have been stigmatised and neglected in the Medieval period, there is a clear disconnect between interpretations of the biological and social milieu surrounding leprosy in the past. To better assess these inconsistent understandings of leprosy in the Medieval period, cross-disciplinary analyses of skeletons excavated from both Early-Late Medieval parish and leprosarium contexts will offer more insight into their lived experiences, along

with the care and treatment of adolescents with leprosy during this formative period. The differing contexts of these broadly contemporaneous archaeological sites (i.e. parish cemetery vs. leprosy hospital) shows the potential for diverse aspects of care to be evidenced in these groups. Scrutiny of historical data for the appearance and dissemination of leprosy prior to the 12th century AD and exploration of the palaeopathological evidence for leprosy can help to create a benchmark for understanding the disease in the past. Cross-disciplinary analyses provide data to create a deeper understanding of the lived experiences of people with leprosy in the past. Models of past care based on the clinical impact of having skeletal lesions of leprosy, compiled through the Index of Care framework (Tilley and Cameron 2014), can provide some evidence-based support for the care and treatment for individuals buried within cemeteries associated with leprosy hospitals. Strontium and oxygen isotope analyses can determine whether individuals were local or non-local when compared to their burial locations, and the data can be contextualised alongside mortuary treatments. Lastly, amelogenin peptide analysis from samples of dental enamel and carbon and nitrogen isotope analyses of samples of (incremental) dentine from adolescents buried at the St. Mary Magdalen Leprosy Hospital can be used to reveal aspects of engendered social identity (e.g. biological sex) and life history to understand both early life stresses that may compromise immunity as well as end of life nutritional provision.

1.2 Aims, hypothetical models, and research questions

The overall aim of this research is to investigate the biological and social impacts of leprosy on adolescents buried within parish and leprosarium contexts in the past and evaluate whether the concept of care can be supported. This thesis hopes to achieve this aim by addressing the following research questions:

Research Questions and Hypothetical Model 1:

is there any evidence of mobility in adolescents with leprosy?

- Is there is a difference between the mobility histories of people who were buried in leprosy hospital cemeteries versus those who are found buried in parish cemeteries?

- Are there differences in mobility histories based upon sex and, if so, are they similar for both the leprosy hospital and parish church sites?
- Is there any evidence for individuals with the infection travelling long distances to the leprosy hospital?

If the historical data holds true and people with leprosy were stigmatised, we may expect to find those affected by leprosy, including adolescents, segregated in areas beyond the geographical bounds of their home communities, or buried in a non-normative manner. If a difference exists between the leprosy hospital site and the parish cemetery, then an argument may be made for the person seeking care in a hospital, rather than facing expulsion, i.e. people inhumed in general parish cemeteries prove to be of local geographical origin in contrast to those inhumed at leprosy hospitals.

Research Questions and Hypothetical Model 2:

Were individuals provided with care in the leprosarium?

- Does the Index of Care framework support the provision of care (medical intervention, nutrition, direct and community support) at the St. Mary Magdalen Leprosy Hospital?
- Do we see any dietary shifts during the life histories of people interred in the cemetery associated with the St. Mary Magdalen Leprosy Hospital site?
- Is there any evidence for individuals being fed a similar diet?
- Do we see fluctuations in $\delta^{13}C$ or $\delta^{15}N$ that may distinguish between food consumed and stress experienced?

Many historical sources (Brody 1974: 66-67; Watts 1997:49-63; Richards 2000:134-139; Orme and Webster 1995:24-31; Covey 2001) claim that leprosy hospitals were not care facilities and their patients faced neglect. However, the archaeological context of the North Cemetery associated with the St. Mary Magdalen Leprosy Hospital in Winchester suggests this may not be the case. The Index of Care framework (Tilley and Cameron 2014; Tilley and Schrenk 2017) can potentially be used to help support or refute whether a level of individual and communal care was provided to individuals based upon the interpretation of the biological impacts of observed skeletal lesions. If evidence of care can be supported, then a socioececonomic structure likely existed within the community

for this care to be provided. Additionally, if the people interred in the leprosarium cemetery of St. Mary Magdalen prove to be of a different geographic origin, we must consider whether they may have travelled there to receive care and treatment, and not exclusively assume that they were exiled to these facilities. One aspect of care and treatment that may be viewed isotopically is lifelong dietary provisions, evidenced by stable isotope analyses of carbon and nitrogen isotopes from samples of incremental dentine. If we see dentine profile patterns suggestive of long-term nutritional stress preceding death (e.g. high nitrogen and low carbon), an argument may be put forward that these people were not cared for. However, if we observe dentine profile patterns demonstrating a population convergence of $\delta^{13}C$ and a rise or fall in $\delta^{15}N$ values near death, we can use this as a potential proxy to distinguish a person who had a "leprosarium diet" from a person who had pathophysiological stress due to leprosy.

Research Questions and Hypothetical Model 3:

Can we see indications of nutritional or pathophysiological stress in the dentine collagen profiles of people with leprosy over the life course?

- If so, do they align with the onset of pathological lesions whose timing can be estimated (e.g. linear enamel hypoplasia)?
- Are there any isotopic signals that may indicate the onset of pathophysiological disruption induced by leprosy; for example, can we identify at what age the effects of leprosy began to physiologically manifest in the past?
- Can we see any isotopic shifts during the years preceding death in individuals with leprosy?

Individual frailty and early life nutritional stress can contribute to disease development in life and earlier mortality (Ulijaszek 1990; 1996; Barker 2004; Langley-Evans 2015; Gowland 2015). Thus, it can be proposed that if individuals with signs of non-specific childhood stress (e.g. linear enamel hypoplasia) display a disruption to their dentine isotopic profiles, they may have been more susceptible to lepromatous leprosy due to an underdeveloped or compromised immune system as a result of this disruption. Further, if a pattern of disruption in the dentine profiles that corresponds with the timing of

pathological lesions can be observed, we may be able to use this as a proxy to understand the effects of leprosy on the metabolism of carbon and nitrogen compounds.

1.3 Structure of the Thesis

The results and their discussion in this thesis are included in four manuscripts addressing the research questions and hypothetical models. These are "bookended" by background chapters, a detailed materials and methods chapter, and a wider discussion and conclusion chapter. Each chapter and manuscript retains its own references section for easier bibliographic access. By structuring the thesis with the results presented within fully prepared manuscripts, ready for submission for publication, the author acknowledges there will be an element of repetition (i.e. re-summarising elements of the background and materials and methods chapters).

Chapter Two provides an in-depth look at leprosy through present and past perspectives of the disease. This chapter discusses the clinical features of the disease, its current epidemiological trends, the immune spectrum, and the long-term impacts of those who live with the disease. It continues with a detailed review of the historical evidence for leprosy with an examination of its documentary origins through classical medical texts, a critical evaluation of its longstanding association with Biblical scripture, and changes in understandings through time and space. Archaeological data for the disease further helps to verify its presence geographically and chronologically to better evidence transmission, disease burden, and mortuary treatment from prehistory to the 12th century AD.

Chapter Three centres on the synergistic relationships between isotopes and palaeopathology. This chapter first offers a basic review of multiple isotope systems, including how they are measured, how they are subsumed within the human body, and how they have been used to produce nuanced data in archaeological studies. Of particular note in this chapter is their potential in palaeopathology, including a review of literature that has successfully linked both disease and stress, and their influence on isotope systems.

Chapter Four provides detailed information for the skeletons selected for this study and the methods used to analyse them. This includes the context of the cemeteries studied,

mortuary treatments of the individuals buried there, and the prevalence of individuals with leprosy within their respective cemeteries. The methods section first provides the osteological and palaeopathological methods used to analyse the skeletons, as well as the methods for evidencing care through the Index of Care framework. The chapter continues with the criteria for isotope sample selection, sampling protocols, detailed isotope analysis methods, and isotope quality parameters, and amelogenin peptide methods.

Chapter Five provides the resulting data from the above analyses through the production of four manuscripts. Manuscript 1 offers evidence for the care of adolescents in the Medieval period, using the Index of Care framework focused on an individual buried at St. Mary Magdalen leprosarium as a case study. Manuscript 2 provides strontium and oxygen isotope results for adolescents with leprosy buried in the parish cemetery of St. John at the Castle Gate/Timberhill in Norwich and interprets these data within their 'Anglo-Scandinavian' contexts. Manuscript 3 examines social identity and mobility of indviduals buried in the cemetery of St. Mary Magdalen Leprosy Hospital through amelogenin peptide and strontium and oxygen isotope analysis. Lastly, Manuscript 4 examines carbon and nitrogen incremental dentine profiles from 10 adolescents from St. Mary Magdalen Leprosy Hospital to examine aspects of dietary intake within the leprosarium through their lives, and whether patterns of stress can be identified within the dentine profiles. All manuscripts are written solely by the first author with editorial input and revisions recommended by the supervision team.

Lastly, Chapter Six synthesizes the results from each manuscript to discuss the major trends and conclusions from the study, and the big picture impacts that can be drawn from this research. It also returns to the original research aim, hypothetical models, and questions, and provides limitations of the research, further offering suggestions for future research directions.

1.4 Research Impact

Modern perceptions of leprosy tend to invoke images of Medieval stigma, isolation, and both communal and medical care neglect. The negative treatment of people with leprosy

in the past is often cited as a justification for the continuing stigma surrounding leprosy in some parts of the world today (Heijnders 2004; Rafferty 2005; Jacob and Franco-Paredes 2008; Sermrittirong and van Brakel 2014; World Health Organization 2015). Therefore, it is worth examining the biological and social milieu of this disease in which young people with leprosy in the past lived, and the models of care and treatment that may be interpreted from this study in order to challenge these longstanding beliefs.

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CHAPTER 2. PRESENT, PAST, AND ARCHAEOLOGICAL PERSPECTIVES OF LEPROSY

2.1 INTRODUCTION

Infectious diseases can be primary agents in cultural transformations, as a result of social, economic, and political disruption generated by epidemics and pandemics, and they can have chronic physical and psychologically debilitating effects on people (Inhorn and Brown 1990). In order to understand how leprosy affected sociocultural systems in the past and the biological consequences of the disease on people and their communities, a review of the clinical, historical, and bioarchaeological evidence is warranted. This chapter begins by reviewing the clinical features of leprosy, including: the current epidemiology of the disease; the causes of leprosy and potential modes of transmission; the pathogenesis of the bacterium; and the documented biological and social consequences of people with leprosy today. The chapter continues by contextualizing these clinical understandings with a view to the past, considering historical understandings that existed previous to, and during, the Early Medieval period, which likely affected the individuals analysed for this study. The chapter culminates with an overview of the bioarchaeology of leprosy, offering a more coherent understanding of leprosy in the past by combining previous archaeological and palaeopathological studies that help to underscore the theoretical framework of this study.

2.2 BIOLOGICAL AND CLINICAL FEATURES OF LEPROSY

Leprosy, also known as Hansen's Disease or Mycobacterial neurodermatosis, is a chronic infectious disease mainly affecting the skin and the peripheral nervous system (Butlin and Lockwood 2020). The disease is caused by a bacterium, which may be *Mycobacterium leprae* or *Mycobacterium lepromatosis* (rare). It is granulomatous in nature and forms masses of macrophages (an immune system cell responsible for detecting pathogens) in the skin (Scollard et al. 2006; Walker and Lockwood 2006). The bacterium is an intracellular, obligate bacillus, meaning that it is typically dependent on a host environment for survival (Walker and Lockwood 2006; Scollard et al. 2006; Lydyard et al. 2010). Leprosy was the first pathogen to be linked to a bacterial origin by Norwegian

physician Gerhard Armauer Hansen in 1873, when he discovered multiple bacilli from a biopsy taken from a patient's nasal region under a microscope (Bennett et al. 2008; Suzuki et al. 2012). Although the last major European endemic (c. 3000 individuals) of leprosy peaked in the mid-18th century, leprosy remains a significant problem in developing parts of the world due to poor diagnostic tools, poverty, limited access to a healthy diet, lack of education, lack of access to medical treatments, vast ranges in immunity, social stigma, and a lack of effective access to preventive and curative strategies (Pardillo et. al 2007; Jacob and Paredes 2008; Bennett et al. 2008; Goulart and Goulart 2008, World Health Organization 2013).

2.2.1 EPIDEMIOLOGY

The World Health Organization (WHO) set out to eradicate leprosy by the year 2000 and, as such, continually tracks worldwide leprosy incidence and prevalence. Although eradication has not yet been achieved despite the WHO's implementation of multidrug therapy (MDT) in 1981, over 16 million individuals from 122 countries have been cured of the disease (Nordeen 1995; World Health Organization 2012, 2013). It is important to note, however, that though patients may be cured, some still face permanent deformity and/or disability with lifelong stigma. MDT is freely available for leprosy patients, but leprosy persists globally with over 200,000 new indivduals diagnosed every year (World Health Organization 2013; 2018; 2020). It is important to note that these persistent infections are not only reflective of individual health, but are suggestive of community health with continuing transmission.

Currently, leprosy is overwhelmingly found in regions of Southeast Asia, which accounts for the largest proportion of all new cases detected worldwide (~ 71 per million; World Health Organization 2018; 2020). Other regions currently reporting new cases include the Americas (30 per million), Africa (18 per million), the Eastern Mediterranean (6 per million), and the Western Pacific (2 per million) (*ibid*; see Figure 2.1). Furthermore, 95% of all new cases were reported from just 16 countries, whereas the remaining 5% came from the rest of the world. This uneven distribution amongst and within countries is indicative of a high endemic burden in these regions. Whilst leprosy new case data

remain insignificant in Europe (<0.1 per million), the WHO believes the disease still persists at low levels (*ibid*).

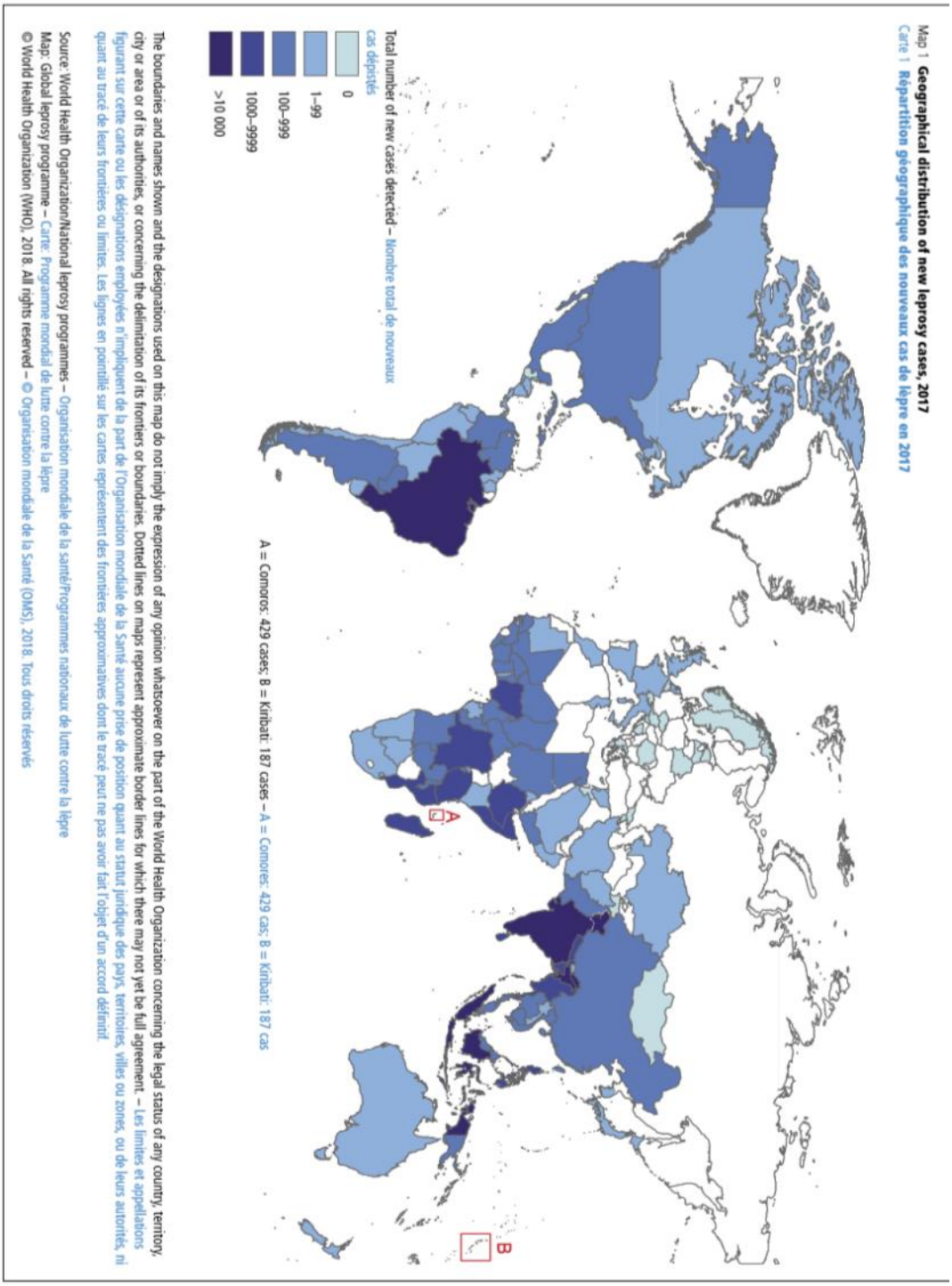


Figure 2.1. Worldwide prevalence of leprosy based on 2017 data (World Health Organization 2018: 451; Open Access).

2.2.2. WHO IT AFFECTS TODAY

Monitoring demographic trends in new case detection, disease relapse, and disability is invaluable in viewing leprosy in evolutionary, biological, and socio-political perspectives.

However, careful interpretation of these demographic trends needs to be utilized. For example, according to the WHO (2012; 2020), males have a greater tendency to develop the most severe form of leprosy, but this may be due to sex and gender inequalities in particular regions, which could impose limitations on access to healthcare; i.e. women may not be able to travel to clinics or it may be considered a cultural/religious taboo for women/girls to be examined by male doctors (Fine 1982). Notwithstanding, the male-to-female ratio for countries reporting >100 new cases of leprosy worldwide is always more than half (World Health Organization 2018). The detection of new cases of leprosy in children has a significantly wide range. Of the countries reporting >100 new cases, Argentina shows the lowest incidence of childhood leprosy with a rate of 0.6% of the affected population, whereas 41.3% of new cases in Micronesia are found in children and adolescents <15 years (*ibid*). These example figures were anticipated to decline in 2009 with increasing implementation of therapeutic strategies, early detection, and falling transmission rates, but sadly they increased globally in 2012 (World Health Organization 2012; 2013).

Since 2005, the worldwide prevalence for leprosy in individuals <15 years hovers at approximately 9% of affected populations, with up to 35.5% of children affected in highly endemic areas (Butlin and Withington 2018). In comparison to adults, children are at increased risk of developing the more severe lepromatous form, with some communities showing 80.5% of children out of the total affected population displaying multibacillary signs and symptoms (*ibid*). Although the incidence rates of leprosy diagnoses in children halved from 2005-15 (407,791 to 210,740), issues with delayed diagnosis, inadequate nutrition, immunodeficiencies, and endocrine disruption as a consequence of puberty continue to complicate elimination efforts (Davey and Schenck 1964; John et al. 2005; Butlin and Withington 2018). For these endemic communities where leprosy notably affects children, treatment, monitoring, and destigmatisation are of utmost importance to familial, community, and hospital care networks (Butlin and Withington 2018). Unsurprisingly, leprosy rates in children tend to be higher in those who have family members with leprosy, and community stigma remains a significant barrier for early detection and treatment in these younger cohorts (Fine 1982; Jopling and McDougall 1988; Jain et al. 2002; Goulart and Goulart 2008; Lydyard et al. 2010). Although childhood

leprosy and recommended courses of care and treatment are documented by epidemiologists and leprologists today, historical and archaeological evidence of the disease and its treatment in children is much scarcer.

Leprosy is the leading cause of infective, neurological disability and, if untreated, has the potential to incite permanently debilitating changes to the human body (Lockwood 2002). This means that although a person may be completely cured of leprosy, they may still endure the biological effects and debilitating disfigurements. In addition to leprosy incidence and prevalence, the WHO tracks the distribution of visible deformities, known as grade 2 disabilities (G2D), as an indication of the disease's morbidity rates and the effectiveness of their monitoring programmes, reflecting prevention, education, and treatment (World Health Organization 2012, 2013; Suzuki et al. 2012). The most recent statistics suggests that G2D reported in 2012 were at their highest in over a decade, representing approximately 0.25 per 100,000 of the total world population (World Health Organization 2013; 2018). Unfortunately, the WHO does not separate disability figures for younger cohorts, however some regional studies indicate the rate of G2D of children can be up to 21.3% within affected communities (Butlin and Withington 2018).

2.2.3 BACTERIOLOGY

M. leprae (and *M. lepromatosis*) is parasitic in nature, relying on and residing intracellularly within the body's macrophages and Schwann cells (the cells which makeup the myelin sheaths of peripheral nerve fibres) (Scollard et al. 2006; Lydyard et al. 2010; Suzuki et al. 2012; Han and Silva 2014). This predilection in the nerve cells may help to conceal the bacteria from the host's immune defences. Biomolecular evidence shows that leprosy bacteria actually have the ability to genomically reprogramme Schwann cells and turn them into stem cells, a factor that likely eases their concealment and dissemination (Fine 1982: 163; Wegner 2013). *M. leprae* can also persist outside the human body in varying conditions for several days, although some reports have suggested longer (Fine 1982; Desikan 1995; Walker and Lockwood 2006). Potential longer term environmental reservoirs certainly warrant further study.

Since its discovery in the 19th century, attempts to culture *M. leprae in vitro*, or outside of a human or animal, have failed. This remains a major disadvantage to leprosy research prohibiting our understanding of transmission and pathogenesis, and vaccine development. Nevertheless, some bacteriological studies were possible after it was discovered in the 1960s that *M. leprae* could be grown *in vivo* using a mouse's footpad, which remains the primary method for cultivating *M. leprae* (Fine 1982; Scollard 2006). Further bacteriological studies discovered that the nine-banded armadillo was a 'natural' reservoir of leprosy in North America (Fine 1982; Bennett et al. 2008; Walker and Lockwood 2006). Other animal reservoirs added to the list now include red squirrels, chimpanzees, and mangabey monkeys, although to a much lesser extent (Avanzi et al. 2016; Lydyard et al 2010; Han and Silva 2014).

M. leprae microscopically presents as a rod-shaped bacillus, either straight or slightly curved, and measures approximately 0.3 μm in diameter. Like other pathogens in the genus *Mycobacterium* (e.g. tuberculosis), *M. leprae* is an acid-fast organism, meaning that it resists decolourisation by acids during the staining process. This is due to the robusticity of their cell walls owing to their mycolic acid content, thereby concealing its presence under microscopic view (Fine 1982; Jopling and McDougall 1988; Lydyard et al. 2010; Suzuki et al. 2012). Again, like other bacteria in the *Mycobacterium* genus, *M. leprae* can only be viewed through the Ziehl-Neelsen method, which uses a special mixture of acid alcohol and carbon fuschin (a reddish-pink dye) to stain the bacterium (Jopling and McDougall 1988). This procedure is not only important in determining the presence of the bacteria, but also crucial for recording the pathogen load, known as the Bacterial Index (BI). The BI aids in determining the 'type' of leprosy one has (see 2.2.5 Ranges in Immunity) as well as tracks progression in treatment (Ridley 1974; Jopling and McDougall 1988).

Major confounding factors of *M. Leprae* are its slow growth and long incubation period. *M. leprae* optimally thrives at approximately 27-30° C/80.6-86° F, which may explain its predilection for cooler parts of the body (e.g. skin, superficial nerves, upper respiratory tract, testes) (Fine 1982; Jopling and McDougall 1988; Lydyard et al. 2010). Because the bacteria cannot be cultured *in vitro*, growth rates must be measured in animal reservoirs,

e.g. the mouse footpad (Fine 1982; Scollard et al. 2006; Suzuki et al. 2012). Studies have revealed an extremely low growth rate for *M.leprae*, completing one division at a rate of every 10-14 days (Fine 1982; Jopling and McDougall 1988; Lydyard et al. 2010). This incredibly slow growth, the slowest of any known pathogen, is ascribed as the main reason for the extreme variability in incubation period. The incubation period (i.e. the time it takes from initial infection to symptoms to appear) for leprosy ranges from weeks to over 30 years, with an average of four years for paucibacillary leprosy to 10 years for multibacillary leprosy (Ridley and Jopling 1988; Lydyard et al. 2010; Bhat and Prakash 2012). Inconsistent incubation periods were echoed in animal models demonstrated by a chimpanzee infected with *M. leprae* in infancy, but not revealing any pathogenic signs of the disease for over 30 years (Suzuki et al. 2012). These incredibly varied incubation periods obviously pose problems for adequate reporting and detection in regions where the disease is endemic or in carriers unknowingly moving around with the disease locally, nationally, or internationally (Bennett et al. 2008; World Health Organization 2012).

2.2.4 Modes of Transmission

Despite knowing the causative bacterium (*M. leprae/M. lepromatosis*), methods of leprosy transmission are not fully understood due to its inability to be cultured and studied *in vitro*. Even though some animals possess the ability to be infected with *M. leprae* (e.g. mice, armadillos, primates, red squirrels, etc.), human-to-human transmission is the normal mode of contracting leprosy 'in nature'. However, wild armadillos infecting humans in the southern USA merits more attention (Suzuki et al. 2012; World Health Organization 2012; 2013). Because the incubation period for leprosy is so variable, many people may harbour and spread the disease unknowingly. As a result, familial and frequent community contacts pose the greatest risk for contracting leprosy in endemic regions (Jain et al. 2002; World Health Organization 2012). It is of note that once MDT treatment commences, the infectiousness of a person with leprosy generally ceases (*ibid*).

It is presumed that *M. leprae* is transmitted in humans primarily through droplet inhalation via the respiratory system after periods of long-term exposure, but some evidence exists that the bacterium can enter the body through exposed surfaces of

broken skin (McDougall et al. 1975; Job et al. 2008). It is unclear how the pathogen fully spreads and metabolises throughout the body, but is thought to be taken up by alveolar macrophages where they live and multiply in the nasal mucosa and opportunistically metabolise during anabolic phases (Scollard et al. 2006; Lydyard et al. 2010). Subsequent to initial exposure and colonisation the bacteria are then likely spread through the circulatory system and go on to infect the Schwann cells in the neural tissues (McDougall et al. 1975; Scollard et al. 2006; Lydyard et al. 2010).

2.2.5 Ranges in Immunity

The WHO classifies leprosy as a 'spectral disease' due to its ability to manifest in a variety of different clinical forms (World Health Organization 2012: 12). In the 1960s, Ridley and Jopling proposed a five-group classification scheme for leprosy based on clinical and histological manifestations, which correlated with host immunity (Ridley and Jopling 1966; Ridley 1974). In general, this system is still in practice today, albeit with slight modifications (Figure 2.2). These classifications range in severity from early indeterminate leprosy (I), and then continue on to: polar tuberculoid (TT); borderline tuberculoid (BT); mid-borderline leprosy (BB); borderline lepromatous (BL); and polar lepromatous (LL) (Ridley and Jopling 1966; Ridley 1974; Jopling and McDougall 1988:17-21). Indeterminate leprosy is recorded when the person's immunological response cannot be determined. This type is most common in children where there may not be any histopathologic or clinical signs of leprosy, except for a hypopigmented skin lesion with potential sensory loss (Fine 1982; Bloom and Godal 1983; Jopling and McDougall 1988:21). At this stage, the lesions may spontaneously resolve, or progress to one of the classification stages (*ibid*). In 1981, the WHO created an additional 'two type' system dependent on bacillary load as evidenced through skin-smears (World Health Organization 2012). These types are paucibacillary (PB), which include the I, TT, and BT forms of leprosy, and multibacillary (MB), which include the BB, BL, and LL types of leprosy (*ibid*). In the event that a skin smear is not available, cases may be classified as PB if less than five skin lesions appear, or MB if six or more lesions are present (*ibid*).

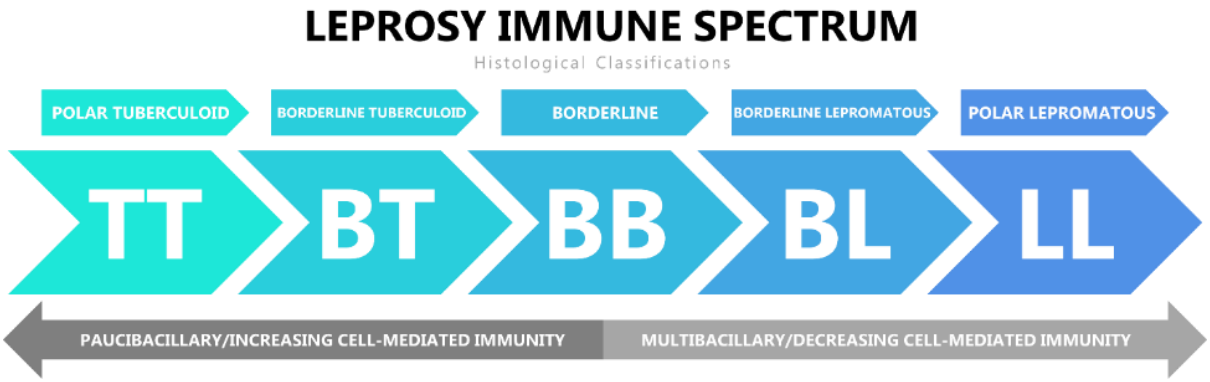


Figure 2.2 Immune spectrum of leprosy based on Ridley and Jopling (1966) and Jopling and McDougall (1988) classifications. Author's own.

After five decades of intensive study, the underlying mechanisms that elicit the range of cellular immune responses remain unexplained (Scollard et al. 2006; Goulart and Goulart 2008). As such, the immune system over the life course remains worthy of study. Increasing studies are exploring links between leprosy and genetic profiles of patients, finding in some instances that certain genes may make people more susceptible or resistant (Abel et al. 1998; Kang and Chae 2001; Mira et al. 2004; Alcaïs et al. 2005; Alter et al. 2008; Krause-Kyora et al. 2018). Although the exact gene mechanisms in leprosy remain unknown, the genes of the human leukocyte antigen system (HLA) and non-HLA will differentially influence leprosy susceptibility (*ibid*). For example, variations in multiple genes (CCDC122, CD13orf31, NOD2, TNFSF15, HLADR, RIPK2, LRRK2, MRC1, PARK2, PARCRG, and LTA) show some influence over leprosy susceptibility and development (Mira et al. 2004; Roy et al. 1997; Sarno et al. 2000; Cooke and Hill 2001; Alcaïs et al. 2007; Zhang et al. 2009; Franceschi et al. 2009) as well as different alleles of the vitamin D receptor (VDR) gene (Roy et al. 1999). Whilst these studies aid in understanding innate immunological predispositions, multiple socioeconomic factors also factor into leprosy susceptibility and transmission (World Health Organization 2012; 2018).

There is some support for cross-immunity between leprosy and tuberculosis, i.e. those who are exposed to tuberculosis or other *Mycobacteria* are more likely to develop a natural defence against leprosy (Chaussinand 1948; Manchester 1984; Leitman et al. 1997; Crespo et al. 2019). However, this is extremely dependent on the reproductive rate

of the leprosy bacilli *in vivo* (*ibid*). The cross-immunity hypothesis has been echoed in studies that have shown the BCG vaccine for tuberculosis may provide some protection against leprosy to varying degrees depending on the population (Merle et al. 2010; Kar and Gupta 2015).

2.2.6 Clinical Features and Treatment

The clinical manifestations of leprosy are highly dependent on an individual's cellular immune response (Figure 2.2). Initially, individuals with leprosy generally present with skin lesions and peripheral numbness or weakness (Walker and Lockwood 2006). In people with tuberculoid leprosy, skin lesions (plaques) are limited in number, asymmetric, and have well-defined margins. Anaesthesia of the lesions and their surrounding area may cause hair loss (alopecia), an inability to sweat normally (anhidrosis), thickening of the skin (hyperkeratosis), and limited damage to the peripheral nerves (Walker and Lockwood 2006; Scollard et al. 2006; Lastória and Abreu 2014). Immune responses in tuberculoid leprosy are usually self-limiting.

In contrast to this, patients with lepromatous leprosy tend to present widely distributed, symmetrically located skin lesions (nodules) with peripheral oedema (*ibid*). In individuals with a poor cellular immune response, *M. leprae* continue to multiply and colonise the body haematogenously, with a preference for the cooler areas of the body. As the disease progresses, flesh-coloured nodules (i.e. lepromas) may present, and dermal infiltration and thickening may occur, particularly of the face. In addition, the eyebrows and eyelashes are lost from affected skin on the face (madarosis), inflammation of the nasal mucosa occurs resulting in stuffiness and nosebleeds (epistaxis), infiltration and destruction of the palate, anterior nasal spine, and septum occcurs, and the nasal aperture widens. Laryngeal involvement can lead to an inability to speak and/or airway obstruction, and testicular atrophy and hypogonadism can occur in men (Fleury and Duerksen 2006; Scollard et al. 2006; Walker and Lockwood 2006; Lastória and Abreu 2014). Further neuropathic involvement complicates the clinical manifestations of lepromatous leprosy and affects the sensory, motor, and autonomic functions of the nervous systems. This causes destruction of the dermal nerves which can lead to deterioration in function, and soft and hard tissue damage, including atrophy and

resorption. Finally, facial paralysis due to damage to the 7th cranial nerve and blindness due to damage to the 5th cranial nerve occur in 3.2% of modern patients (Walker and Lockwood 2006). Lepromatous leprosy shows the capacity to insidiously damage all bodily systems, particularly the skeleton, the endocrine glands, and kidney function (Lastória and Abreu 2014).

Multidrug therapy (MDT) was recommended by the World Health Organization in 1981 with the aim of eradicating the disease globally. Current treatment protocols, free for all from 1995, call for a combination of the antibiotics dapsone, clofazimine, and rifampicin for six months for paucibacillary and 12 months for multi-bacillary leprosy (Kar and Gupta 2015). Relapse rates following MDT remain relatively low (<7%), with the likelihood of relapse increasing based on bacterial load; i.e. multibacillary patients with a high BI are more likely to relapse than paucibacillary patients, and be non-compliant with treatment due to various social factors; i.e. they may face stigma for attending leprosy clinics (Walker and Lockwood 2006; World Health Organization 2013).

2.2.7 Long-term effects of leprosy

Without treatment or resolution, the clinical features of leprosy can accumulate, leaving people with permanent disfigurements and deleterious effects on their physical, psychological, social and economic well-being (Rafferty 2005; Scollard et al. 2006; Jacob and Franco-Paredes 2008; van Brakel et al. 2018). Because this damage is irreversible, early detection and treatment is key in order to avoid physical disabilities, but the modern-day intersectional stigma associated with the disease can be equally damaging (Jacob and Franco-Paredes 2008; Sermrittirong and van Brakel 2014; van Brakel et al. 2018). In several current belief systems, leprosy is regarded as an outward representation for sins or misdeeds committed in life and brought upon oneself. These ideas have traversed through colonial pathways into areas where rejection from familial roles and community creates a more injurious identity crisis and transformation than in Western civilisations (Rafferty 2005; Jacob and Franco-Paredes 2008; Sermrittirong and van Brakel 2014). Consequently, these negative beliefs lead to a level of self-stigma for people with leprosy that greatly impacts overall mental health and social interactions in similar ways to external forms of discrimination (van Brakel et al. 2018). These negative

impacts result in a loss of a normal socioeconomic livelihood and quality of life, the effect of which is captured in one patient's quote*:*

'We can endure losing fingers and toes, eyes and nose, but what we cannot endure is to be rejected by those nearest and dearest' (Rafferty 2005: 120).

Many programs have attempted to reduce the stigma attached to leprosy, including introducing alternative terms for the disease (e.g. Hansen's Disease and Mycobacterial neurodermatosis), and focussing on the biological aspects of the infections (Butlin and Lockwood 2020). However, the interpersonal, intrapersonal, communal, and political environments in which people with leprosy live are ingrained and stem from deeply embedded historical accounts of leprosy. Without alteration of these narratives, the rejection and alienation of people with leprosy are unlikely to cease (Jacob and Franco-Paredes 2008; Sermrittirong and van Brakel 2014; van Brakel et al. 2018).

2.3 Leprosy in the Past – Historical Understandings and Paradigm Shifts

In current mind sets, the idea of leprosy invokes images of Medieval stigma and isolation. However, the assumption of a widespread hostility towards people with leprosy in the past is primarily anecdotal, and largely an artefact of conquest, racism and colonialism (Rawcliffe 2006:13-29; Jacob and Franco-Paredes 2008; Edmond 2006:61-109). Fiction writers were inspired by grotesque fantasies of people with leprosy (Touati 2000; Edmond 2006:29-37) and aggressively deleterious isolation campaigns led by physicians like Dr. Albert Ashmead in the late 19th century and early 20th century (Ashmead 1895; 1897a; 1897b; 1897c; 1897d; 1899; 1901a; 1901b). This led to worldwide legislative changes and helped to solidify an ingrained prejudice against people infected with leprosy, past and present. It is therefore worth interrogating available historical evidence for leprosy to better gauge past societal reactions.

2.3.1 What's in a name? - Historical evidence for leprosy

Many documentary sources are used to trace leprosy trajectories through time, but they are riddled with descriptive vagaries or are uncritically designated as a direct correlation

with modern leprosy, without any interrogation of their original usage or meaning. It is therefore worth evaluating these documentary sources to better understand both the disease course and responses to the disease through time and space.

Medical historians have often cited the Ancient Egyptian medical treatise known as the Ebers papyrus (1550-1350 BC) as bearing the first historicomedical descriptions of leprosy (Ebbell 1935; Browne 1970). Subsequent scholarship has revised the early translations of the document and found that the Chons' swellings and uchedu once translated as leprosy, bear no resemblance to the modern-day condition (Browne 1970; 1975). Because ancient Egypt was a major medical epicentre in antiquity, the presence of leprosy would likely be noted amongst the other medical conditions affecting the New Kingdom if it were present in this area at this time period (*ibid*). Despite early mistranslations, many historians often miscite the appearance of leprosy in the Ebers Papyrus, demonstrating the need to reinvestigate the historiography of the disease. At present, the oldest literary evidence available that clinically aligns with leprosy comes from India, from a collection of Sanskrit medical texts entitled, *The Sushruta Samhita* (c. 600 BC) (Bhishagratna 1963; Manchester and Roberts 1989). In the Sanskrit texts, '*Kushtham'* is described as a disease causing contraction of the skin, deformity of the limbs, suppuration of the affected skin, loss of fingers, sinking of the nose, redness of the eyes, and ulcerations (Bhishagratna 1963: 36-40). Similar descriptions of leprosy are present in 3rd century BC documents from China (McLeod and Yates 1981). The *Feng-chen shih*, a form of law code detailed in 1155 bamboo strips buried with a judge from the Chhin period (3rd century BC) details a medico-legal case involving an individual with Lai/Li, which has been translated from the description as possible leprosy. The translation from the medico-legal text is as follows:

At the age of three, [he] became sick with sores on his head… [He] has no eyebrows. The bridge of his nose is destroyed, his nasal cavity is collapsed; if you prick his nose, he does not sneeze. His elbows and knees (missing text) down to (missing text) the soles of both feet are defective and are suppurating at one place. His hands have no hair. I ordered him to shout, and the ch`i of his voice was hoarse. It is leprosy (Lai/Li) (McLeod and Yates 1981:153).

Based upon the descriptions of Lai/Li and its comparable signs and symptoms to modern-day leprosy, Lu and Needham (1988) revisited the Confucian medical text, the *Huang Ti Nei Ching* (c. 5th – 3rd century BC), for other descriptions of Lai/Li and found further parallels with modern day presentations of leprosy, namely peripheral anaesthesia and nasal collapse (368). According to the *Huang Ti Nei Ching* (c. 2nd century BC), the medical focus surrounding Lai/Li was devoted to examining diet, lifestyle, and the environment as a means for the disease's development and subsequent treatment (Hulsewé 1985; Lu and Needham 2012).

Although these texts present fairly convincing evidence of leprosy's presence, or alternatively a disease causing similar changes to leprosy in East Asia, descriptions of the disease in the West are poorly aligned with modern-day presentations of leprosy. This is particularly interesting because etymologically, the modern-day word for leprosy stems from the ancient Greek term, lepra (λέπρα), by way of a Hebrew translation from the Old Testament. It is therefore worth exploring how the disease became transliterated into Greek and Hebrew narratives by exploring the myth of λέπρα and Biblical representations of leprosy in order to see where they converge.

2.3.2 The Myth of Leprosy

The oldest written use of the word leprosy, parsed from the Ancient Greek, λέπρην, comes from Herodotus' The Histories (Book 1 (Clio), Chapter 138; c. 440 BC) where he describes the culture of Persia (Iran). In this chapter, he states that within Persia:

ὃς ἂν δὲ τῶν ἀστῶν λέπρην ἢ λεύκην ἔχῃ, ἐς πόλιν οὗτος οὐ κατέρχεται οὐδὲ συμμίσγεται τοῖσι ἄλλοισι Πέρσῃσι: φασὶ δέ μιν ἐς τὸν ἥλιον ἁμαρτόντα τι ταῦτα ἔχειν. ξεῖνον δὲ πάντα τὸν λαμβανόμενον ὑπὸ τουτέων πολλοὶ ἐξελαύνουσι ἐκ τῆς χώρης, καὶ τὰς λευκὰς περιστεράς, τὴν αὐτὴν αἰτίην ἐπιφέροντες.

Translation: The citizen who has leprosy or the white sickness may not come into town or mingle with other Persians. They say that he is so afflicted because he has sinned in some way against the sun. Every stranger who gets such a disease, many drive out of the country; and they do the same to white doves, for the reason given (Godley 1920: 178-179).

Figure 2.3 Picture of a living person's hand showing 'λεύκη' or vitiligo. Wikicommons: distributed under a CC BY-SA 3.0 license.

The brief description of the manifestation of 'leprosy or the white sickness' (λέπρην ἢ λεύκην), may apply to someone with a skin condition such as vitiligo, psoriasis, eczema, albinism, or any other disease that can cause abnormal whiteness in the skin. In subsequent texts, the words λέπρην and λεύκην are used interchangeably in the Classical world, and in the Modern Greek language, λεύκη is the term for vitiligo (Figure 2.3).

Although the first documented reference of 'lepra' is found in the histories of Herodotus from the 5th century BC, the concept of leprosy as a skin condition can be traced further back through the oral traditions of Homeric mythology. Classical geographers Strabo (1st century BC) and Pausanius (2nd century AD) wrote that in the time of Heracles (c. 13th century BC), there was a town known as Lepreum, founded by one of the children of Pyrgaeus: Lepreus or Leprea (Jones and Ormerod 1918:3-6; Jones 1924: 490-493; Figure 2.4). The town of Lepreum was also thought to also have a population of people with a heritable skin condition, and it was made famous for its healing waters (River Alphos)

that could be used to treat people with the condition 'λεύκην' (Jones and Ormerod 1918:3-6; Jones 1924: 490-493). This further demonstrates both the association and interchangeability between λέπρην ἢ λεύκην (lepran and leukan) in Classical views, and that the dermatological condition could be alleviated with some degree of medical treatment. It is also important to note that the social reactions to people with these conditions are inconsequential throughout the Classical world, and change with Biblical narratives after Christianity spreads. For a more in-depth look at the development of social reactions towards leprosy, an evaluation of Biblical representations of the conditions translated as leprosy is necessary for comparison.

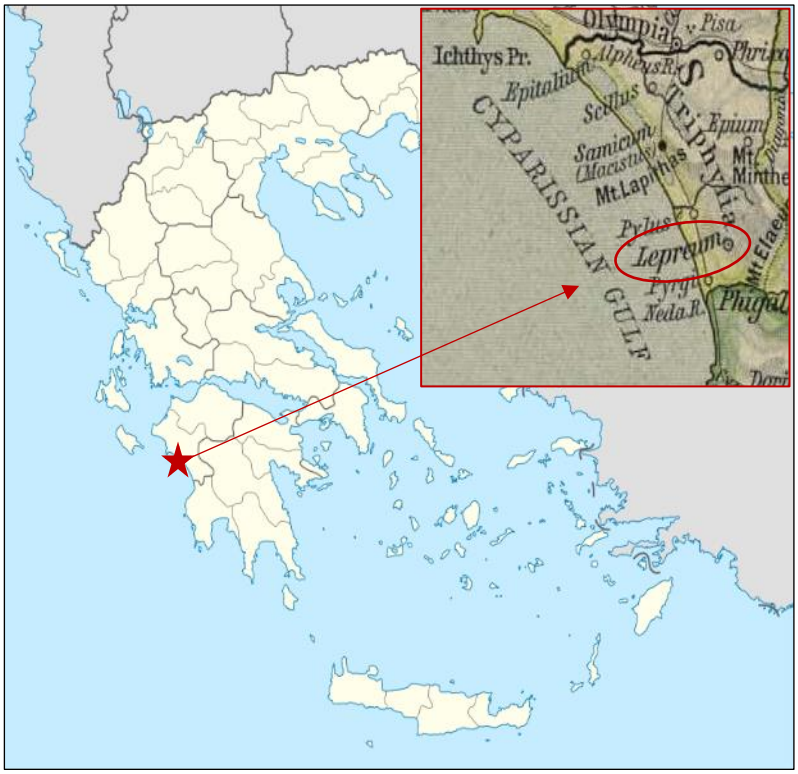


Figure 2.4 Location of the ancient city Lepreum superimposed onto modern-day Greece (Author's Own; modified from Perry-Castañeda Library Map Collection, Historical Atlas by William R. Shepherd 1926, Public Domain).

2.3.3 Leprosy in Leviticus?

It is clear from the descriptions in Herodotus and the Homeric oral traditions, that the word leprosy in the ancient mindset was not describing the condition that we know as leprosy today. However, one of the largest foundations of leprosy causation and societal responses to stigma stems from the translations of the *Pentateuch* from the Old Testament/Torah; specifically the translations of 'leprosy' from the books of Leviticus.

The *Pentateuch*, or Five Books of Moses, were composed in Hebrew during the Persian period of Judaean history (538-332 BC), and represent many people's familiarity with the disease, both past and present (Blenkinsopp 1998). There are also mentions of leprosy from the *Historical Books* of the Old Testament (Chronicles and Kings), which were completed some time later in the 4th century BC (Coggins 2003), and from several of the books in the New Testament (Matthew, Mark, Luke, Gospels), compiled in the 4th century AD (Lindberg 2006: 15; Miller and Nesbitt 2014:18-21).

Levitical texts describe a condition called tsara'ath or zara'at, and they detail the subsequent medical and social protocol for people with this condition. Saint Jerome provides the earliest translations of Leviticus (13:2) from Hebrew into Latin via *The Latin Vulgate* (c. 4th century AD) in which tsara'ath is translated as lepra (Jerome, n.d.). In the *Vulgate*, Jerome translates the descriptions of Tsara'ath as such:

locutus est Dominus ad Mosen et Aaron dicens homo in cuius carne et cute ortus fuerit diversus color sive pustula aut quasi lucens quippiam id est plaga leprae adducetur ad Aaron sacerdotem vel ad unum quemlibet filiorum eius

Translation: *God spoke to Moses and to Aaron saying: the man whose skin has a rising/swelling, or a pustule, or a bright spot; he is stricken with a leprosy plague, and he shall then be taken to Aaron the priest or to one of his sons (Jerome n.d.; translated by the author).*

The chapter continues to detail different ways of diagnosing and treating, both medical and social. This passage continues:

plaga leprae si fuerit in homine adducetur ad sacerdotem (13.10) et videbit eum; cumque color albus in cute fuerit et capillorum mutarit aspectum ipsa quoque caro viva apparuerit lepra vetustissima iudicabitur atque inolita cuti contaminabit itaque eum sacerdos et ***non recludet quia perspicue inmunditia est.***

Translation: *If the leprosy plague is in a man, then he shall be brought to the priest (13.10) and the priest shall examine him. When the colour is white in the skin, and it has turned the hair white, and there is raw flesh in the rising (13.11), it is a chronic leprosy in the skin of his body, and the priest shall pronounce him unclean.* ***There is no need to isolate him because he is unclean*** *(13.12) (Jerome n.d. - Leviticus 13.9-13.12).*

This last passage is quite revealing as it mandates that individuals with chronic skin conditions may be seen as 'unclean', but they are not isolated from society. This shows a

contrast to the way in which the Persians, who ruled Judaea during the composition of Leviticus, treated those who exhibited whiteness in their skin (see section 2.3.2). This may be due in part by a culturally adaptive mechanism of the Judaeans at this time to recognise the Persian laws, whilst imbuing a sense of their own religious purpose as a reasoning that aligns with their religious doctrine. Further, leprosy is described more as a consequence of various medical conditions including boils, burns, itching possibly due to things like lice or dandruff, or any skin condition that can cause whiteness, raw flesh, or a rash, as opposed to a consequence of offending a God in the Persian polytheistic pantheon as documented by Herodotus. Interestingly, *Leviticus* details that leprosy can also affect clothes, walls, and leather, much in the same way that mould may affect them (*ibid*). Levitical treatments for leprosy may involve temporary quarantine, bathing, and washing, cutting, or burning of clothes/leather/walls. Many scholars interpret the 'unclean' aspects of a person with a leprosy as a manifestation of inner sinfulness/ misdeeds, but in *The Latin Vulgate* the text implies that the term 'unclean' is used in its literal form, that is it was hygiene and not sin that was the primary aetiological factor for the condition (Jerome n.d.; Browne 1975). In these Biblical contexts, leprosy is a nondescript term for a dermatological condition, like psoriasis, eczema, or other skin disease, or a consequence of heresy, and is far removed from the modern-day conception of leprosy. This is especially curious as Roman medical practitioners of the 1st – 3rd centuries AD produced descriptions of diseases (e.g. *elephantiasis graecorum*) similar to the modern-day iteration of leprosy. Therefore, when Jerome was producing the *Latin Vulgate* in the 4th century AD, he would have possessed knowledge of the condition, and indeed publicly praised Fabiola of Rome for creating a facility to care for those who had it (Miller and Smith-Savage 2006). In order to investigate this further, an examination of descriptions of diseases similar to modern-day leprosy during the Classical and Roman periods may reveal how these distinctively different medical conditions converged biologically and socially in the Medieval period.

2.3.4 Entangled Terminology – Medical traditions of 'leprosy' in the Classical, Islamic, and Early Medieval Periods

Classical medicine formed the primary foundation for subsequent medical practices until the post-Medieval period, and therefore its details regarding the definition and treatment of leprosy are key to creating a diachronic understanding of the disease. Theories and remedies from the *Hippocratic corpus* and Galenic medicine dominated both Near Eastern and Western medical thought until the 17th century AD, influencing social and biological treatment of a full array of medical insults (Kealey 1981; Conrad et al. 1998:71-77; Mount 2016:35).

Classical medicine generally commences with the *Hippocratic Corpus*, a medical treatise composed between 420-350 BC and grounded in humoral theory from the teachings of Hippocrates (Conrad et al. 1998:11-35). Although Hippocrates does not detail specific signs and symptoms in his work, he does list 'lepra' as a seasonal disease common in spring, or *Aphorismi* (Adams 1868:308). He mentions the condition again in *De alimento*, describing how the treatment of a range of skin conditions, including lepra and alphos (translated as 'white leprosy') is variable based on the person's individual nature, and that nutriment (nourishment) may or may not help (*ibid*: 350). Although these mentions in the works of Hippocrates are too vague to determine what the diseases lepra and alphos are referring to, based on other Classical descriptions and Homeric oral traditions, it is likely a skin condition such as psoriasis or seasonal eczema.

Interestingly, in *De morborum differentiis* by Galen (2nd century AD), he groups elephantiasis, psorai, leprai, and alphoi together as dermatological diseases that cannot be differentiated with respect to their aetiology, and further lumps alphoi, leprai, and leukai together as an assembly of skin conditions that produce an excess of black bile (Johnston 2006: 141-148; Brock 1916:41). In *De symptomatum causis II*, Galen groups the symptoms of lepra and psoriasis:

For itching occurs in those who do not wash, or are filthy, or have indigestion, or who eat unwholesome foods, and far more certainly in those with psora or lepra, because the humour is more copious and thicker in such conditions. And because of this, they scratch greatly and more than all those who itch without such a condition (Johnston 2006:261).

Much like the Hippocratic descriptions, Galenic descriptions of leprosy (lepra) do not equate with current clinical manifestations of leprosy. However, alternative conditions

with descriptions comparable to modern-day leprosy are found in medical traditions in the Roman period (1st - 4th centuries AD). Medical historians such as Aulus Cornelius Celsus and Aretaeus of Cappadoccia detailed the presence of two conditions in the 1st century AD: elephantiasis *graecorum* and elephantiasis *arabum*, with the former as the most likely candidate for leprosy, whilst the latter likely describes filariasis (Celsus 1935: 173-174; Adams 1972:236-240). Galen also describes the signs and symptoms of elephas/elephantia in *De causis morborum* as a condition that causes the mouth to thicken, the nose to resorb, and the ears to show lesions that give the appearance of 'pointedness' (Johnston 2006: 174). This aligns more with the modern concept of leprosy, but perhaps the most convincing historical evidence for the appearance of the disease can be found in Aretaeus of Cappadocia's 1st century AD account of elephas:

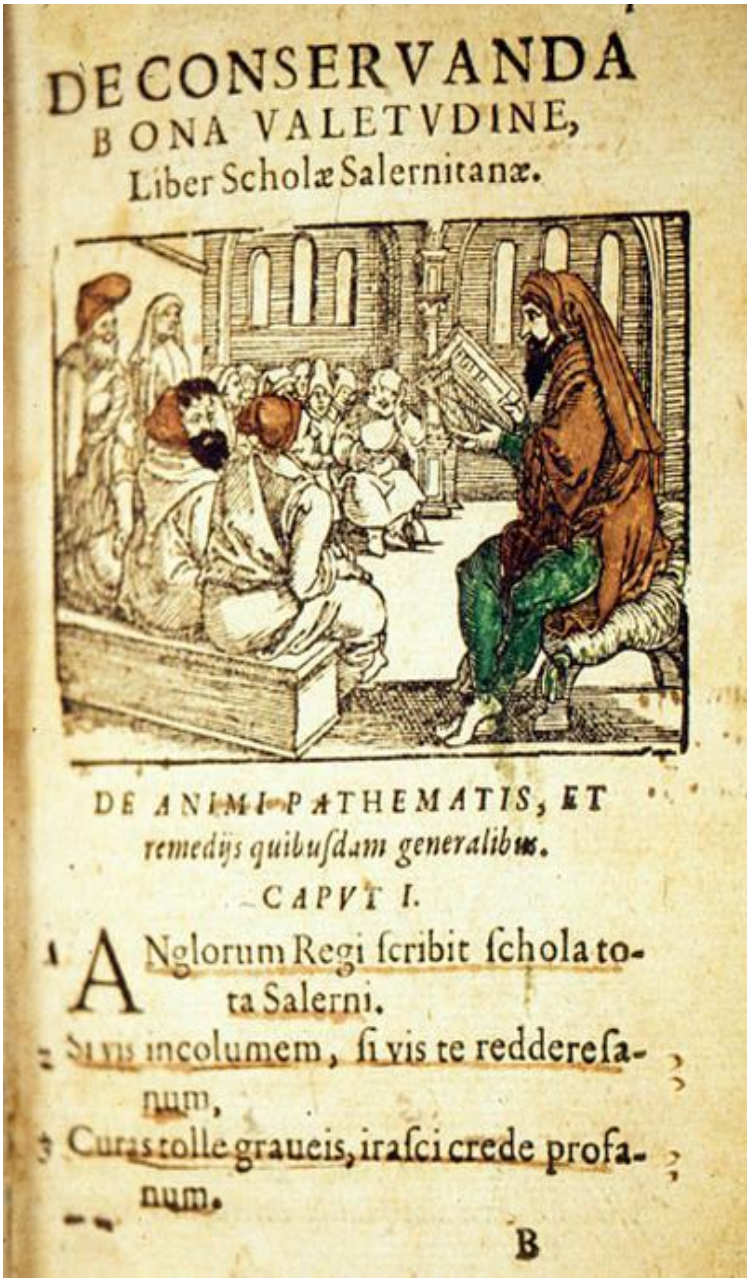
Tumours prominent, not continuous with one another anywhere, but thick and rough, and the intermediate space cracked, like the skin of the elephant. Veins enlarged, not from abundance of blood, but from thickness of the skin; and for no long time is the situation of them manifest, the whole surface being elevated equally in the swelling. The hairs on the whole body die prematurely, on the hands, the thighs, the legs, and again on the pubes; scanty on the chin, and also the hairs on the head are scarce. And still more frequently premature hoariness, and sudden baldness; in a very short time the pubes and chin naked of hair, or if a few hairs should remain, they are more unseemly than where they are gone. The skin of the head deeply cracked; wrinkles frequent, deep, rough; tumours on the face hard, sharp; sometimes white at the top, but more green at the base. Pulse small, dull, languid, as if moved with difficulty through the mud; veins on the temples elevated, and also those under the tongue; bowels bilious; tongue roughened with vari, resembling hailstones; not unusual for the whole frame to be full of such (and thus also in unsound victims, the flesh is full of these tubercles resembling hail). But if the affection be much raised up from the parts within, and appear upon the extremities, lichens occur on the extremities of the fingers; there is pruritus on the knees, and the patients rub the itchy parts with pleasure. And the lichen sometimes embraces the chin all round; it reddens the cheeks, but is attended with no great swelling; eyes misty, resembling bronze; eyebrows prominent, thick, bald, inclining downwards, tumid from contraction of the intermediate space; colour livid or black eyelid, therefore, much retracted to cover the eyes, as in enraged lions; on this account it is named leontium. Wherefore it is not like to the lions and elephants only, but also in the eyelids "resembles swift night." Nose, with black protuberances, rugged; prominence of the lips thickened, but lower part livid; nose elongated; teeth not white indeed, but appearing to be so under a dark body; ears red, black, contracted, resembling the elephant, so that they appear to have a greater size than usual; ulcers upon the base of the ears, discharge of ichor, with pruritus; shrivelled all over the body with rough wrinkles;

but likewise deep fissures, like black furrows on the skin; and for this reason the disease has got the name of elephas. Cracks on the feet and heels, as far as the middle of the toes; but if the ailment still further increase, the tumours become ulcerated, so that on the cheeks, chin, fingers, and knees, there are fetid and incurable ulcers, some of which are springing up on one part, while others are subsiding on another. Sometimes, too, certain of the members of the patient will die, so as to drop off, such as the nose, the fingers, the feet, the privy parts, and the whole hands; for the ailment does not prove fatal, so as to relieve the patient from a foul life and dreadful sufferings, until he has been divided limb from limb. For it is long-lived, like the animal, the elephant. But if there be a sudden pain of the limbs, it attacks much more grievously, spreading sometimes to this part, and sometimes to that. Appetite for food not amiss; taste indiscriminate, neither food nor drink affords pleasure; aversion to all things from a painful feeling; atrophy; libidinous desires of a rabid nature; spontaneous lassitude; the figure of each of the limbs heavy, and even the small limbs are oppressive to the patient. Moreover, the body is offended with everything, takes delight neither in baths nor abstinence from them, neither in food nor in abstinence from it, neither in motion nor in rest, for the disease has established itself in all the parts. Sleep slight, worse than insomnolency, from its fantasies; strong dyspnœa, suffocation as if from strangling. In this way certain patients have passed from life, sleeping the sleep which knows no waking, even until death (Adams 1972:236-240).

Although the 'elephas' Aretaeus is describing may also be indicative of other conditions, it is the first Western description of a condition that also fits some of the symptomatic criteria for modern-day leprosy, potentially indicating leprosy was not known in the West until the early Roman period (1st – 2nd centuries AD). Other classical physicians mention clues as to when this particular condition first appeared in the Eastern Mediterranean. In *De Medicina*, Aulus Cornelius Celsus details the geographical boundaries of elphas/elephantiasis in the 1st century AD, stating that although the Greeks know of and named the condition (ἐλεφαντίασιν), it was not known in Italy or beyond the precincts of the Eastern Mediterranean region (Celsus 1935:174-175). Similarly, Galen wrote that the physician Philon (1st century AD) was of the opinion that the disease first appeared in the Eastern Mediterranean shortly before his own time, and Rufus of Ephesus, a notable physician in the 2nd century AD, pondered how descriptions of such a devastating disease could escape the notice of the venerated physicians previous to his time (referring to early Egyptian and Hippocratic medical practitioners), implying that the condition was relatively recent (Smith 1873:Philon-28-29; Nutton 2012:29-36).

Despite the Classical medical traditions of Hippocrates and Galen constituting a large component of scientific medical thought through time and space, numerous waves of migratory expansions into Western Europe, including Britain, in the Late Roman and Early Medieval periods brought a more religious ideology to medical practices, whilst writings from medical contemporaries of Galen such as Aretaeus, Celsus, and Rufus of Ephesus became embodied into Arabian medical practices in the Near and Middle East (Miller and Smith-Savage 2006; Nutton 2012:299-317). Near the end of the Roman period, as polities broke down in the Western spheres of the Empire, the Eastern part of the Empire continued to thrive into the Byzantine era (4th -15th centuries AD), particularly with advances in medicine (Conrad et al. 1998:193-138; Nutton 2012:299-317). The Arabic medical traditions expounded and were adapted from the Classical traditions as a result of profound changes in socio-political and economic systems, and health. Recurring cycles of disease (e.g. The Justiniac Plague in the 6th century AD), had grave consequences for people in urban environments and their hinterlands, which facilitated a replacement of the Classical Greek and Roman traditions with epistemological Arabian medical frameworks (*ibid*). During the early Byzantine period (5th – 8th centuries AD), Arabian medicine continued to develop and adopt folk practices based on engagement with the East (i.e. Persia and beyond) (*ibid*). In the 8th and 9th centuries AD, a revival of the Classical traditions (e.g. humoral theory) occurred in the Arab world with the translation of hundreds of medical texts from Greek and Latin into Arabic. This then merged with the epistemological foundations of Arabian medicine, resulting in medical practices and hybridised treatises that detailed classifications and subsequent palliative and pharmacological treatments for diseases, such as elephantiasis, or elephancia (Conrad et al. 1998: 140-141; Demaitre 2007: 86-91; Miller and Nesbitt 2014: 21-22). One of the most notable medical practitioners in the Arab world was Ibn al-Jazzar (10th century AD) of Kairouan (modern-day Tunisia), who composed the *Kitab Zad al-musafir wa-qut al-hadir* (Provision for the Traveller and the Nourishment of the Settled), in which he classified different manifestations of the disease *elephantia* (*ibid*). Ibn al-Jazzar's works were later translated into a Latin treatise called *The Viaticum* in the late 11th century AD by Constantine the African for the Salerno School in Italy. Constantine the African migrated from North Africa to become a monk at the Benedictine Abbey of

Montecassino in Southern Italy and, during the course of his translations of Ibn al-Jazzar's works, he employed the term *lepra* as a word to describe the disease, and *elephancia* as its extreme manifestation (Conrad et al. 1998:141-142; Nutton 2012:29-30; Miller and Nesbitt 2014:74-75; Demaitre 2007:87-91). From there, Constantine's translations were widely disseminated throughout Europe in monastic scriptoria and eventually used in teaching at the School of Salerno in (modern day) Italy by Constantine himself (Conrad et al. 1998; Figure 2.5). Concurrent to *The Viaticum*, Constantine the African also translated the compendium of 'Ali ibn al-'Abbas al-Majusi (a.k.a. Haly Abbas - another prominent Arabic medical practitioner of the 10th century AD) into a work known as *The Pantegni* (Conrad et al. 1998:141-143; Demaitre 2007:87-91; Miller and Nesbitt 2014:66-67). Constantine composed a stand-alone section derived from Book IV of *The Pantegni* called the *Liber de Elephancia*, in which two manuscript copies acquired the word *lepra* as a substitute for *elephancia* (*ibid*). The dissemination of Constantine's translations of major Arabic works into succinct medical textbooks for the Latin-reading practitioners at Salerno bolstered the association of the Eastern form of elephantiasis with the Western concept of Biblical lepra. Moreover, the 12th century AD Latin translations of the eminent Persian medical philosopher Ibn Sina's (a.k.a. Avicenna) *Canon of Medicine* described elephantiasis as a consequence of several conditions (one of which was translated as *lepra*) (Gruner 1973). This helped to firmly cement the entanglement between the socially-charged Biblical concept of lepra, already familiar to most in Medieval Europe via St. Jerome's *Latin Vulgate*, with the clinical signs and symptoms of modern-day leprosy in the medical West from the 12th century AD onwards (*ibid*). Therefore, the conflation of lepra with clinical leprosy is not a Biblical mistranslation, but a medical mistranslation due to an absence of knowledge for a Latin medical counterpart; this has created lasting and grave social ramifications.


DE CONSERVANDA
BONA VALETVDINE,
Liber Scholæ Salernitanæ.

DE ANIMI PATHEMATIS, ET
remedijs quibuſdam generalibus.
CAPVT I.

ANglorum Regi ſcribit ſchola tota Salerni.
Si vis incolumem, ſi vis te reddere ſanum,
Curas tolle graueis, iraſci crede profanum.

B

Figure 2.5 An early illustrated work depicting Constantine the African lecturing to the school of Salerno. Created: 1573 published by Christian Egenolf Erben in Frankfurt. Public Domain.

Because the Biblical and medical perceptions of modern-day leprosy did not coalesce until the 12th century AD, societal responses, understandings for diagnoses, and care and treatment likely differed prior to this time. In Early Medieval Europe, evidence for medical care is scant, but Early Medieval texts such as the *Ex herbis feminis/Pseudo-Dioscorides* (6th century AD), the *Herbarium/Pseudo-Apuleius* (c. 6th – 11th centuries AD), the *Lacnunga* (c. 7th - 10th century AD), and Bald's Leechbook (c. 10th century AD) help to

contextualise medical thought and response to disease prior to the widely disseminated medical paradigm shifts from Montecassino and Salerno.

Evidence from these Early Medieval medical compendia are largely prescriptive and palliative, but do associate elephantiasis as a severe form of 'hreofle', the Old English word most analogous to modern-day leprosy (Cockayne 1866; Doyle 2017). Earlier texts (*Ex herbis feminis* and the *Herbarium*) imply that hreofle/leprosy is a disease native to England, but later texts (the *Lacnunga* and *Bald's Leechbook*) treat it both as an indigenous and as a foreign disease, keeping the foreign names lepra/elephantiasis (Cockayne 1865; 1866; Doyle 2017). This is particularly strange as the authors of the *Lacnunga* and *Leechbook* would have most certainly been familiar with the *Vulgate* descriptions of lepra, but the prescriptive care and treatment is conspicuously devoid of religious undertones or associations. This is likely due to the acknowledgement of a separation of the body and soul in the Early Medieval medical mind set, a belief system that notably changes in the 12th century AD (Rubin 1970; Kealey 1981). Although hreofle/lepra are mentioned in all of these Early Medieval texts, *Bald's Leechbook* provides dedicated advice and remedies in all three volumes. Chapter 32 of Leechbook I outlines the recommended treatments for leprosy, ranging from medicinal foods, salves (ointments), bathing, massages, and drinks (Table 2.1).

TABLE 2.1 - Translations of treatments for leprosy and elephantiasis from Bald's Leechbook I (Doyle 2017: 11; 87-89; Cockayne 1865: 9; 78-79).

Old English	*Translation*
.xxxii. Læcedomas wið þam yflan blæce hu man þa sealfa 7 baþu 7 drencas wiþ ðon wyrcean scyle 7 wiþ hreofum lice 7 wið adeadedum lice bæþ 7 sealfa wiþ þon. bæþ 7 sealfa 7 drencas wiþ þam miclan lice 7 swile ealles fiftyne læcedomas.	32. Leeches/Treatments for the bad blotch, how one should make salves and baths for that, and for the leprous body and for the unfeeling(nerve-damaged) body, baths and salves for that; baths and salves and drinks for the mickle body or elephantiasis; fifteen treatments in all.
I.32.6 Læcedom wiþ hreofum lice. adelfe ompran 7 gelodwyrt gecnuwa. wyl þonne on buteran do hwon sealtes to.	A treatment for the leprous body, dig dock and silverweed, pound, then boil in butter, add enough salt to it.

*I.32.7 Wiþ deadu*m *lice stæþwyrt merce gnid on ealoð sele drincan.*	For the deadened (nerve-damaged) body, grind sea lavender and wild celery in ale, give to drink.
*I.32.8 Wið hreofle well on hlonde cwicrinde. ellenrinde niþewearde. æscrinde. 7 wad. elmrind. hemlic do þon*ne *buteran on 7 hunig.*	For leprosy, boil rowan bark, the bark of the lower part of elder-bark, ash bark, woad, elm bark, and hemlock in urine, then add butter and honey.
I.32.9 Wiþ hreofle wegbræde læcewyrt. leac. minte. magþa. eolone. swefl gecnuwa wiþ rysle do þæs s<w>efles swilcan þara wyrta twæde. |	For leprosy pound plantain, ribwort plantain, leek, mint, chamomile, elecampane and sulphur with grease, use twice as much sulphur as of the herbs.
(29v) I.32.10 Wiþ hreofle eft genim horses rysele gemeng swiþe wiþ sealte smire mid.	For leprosy again, take horse fat, mix well with salt, smear with.
*I.32.11 Bæþ wiþ hreofle. wyl on wætere æscrinde. cwicbeam rinde. holen rinde. fulan beames. anan beames. secg. þeorwyrt. hegerife. marubian. beþe mid. 7 þæ*t *lic gnid mid þære hegerifan.*	A bath for leprosy, boil ash bark, rowan bark, holly bark, alder buckthorn's (bark), spindle's (bark), sedge, *þeorwyrt*, cleavers, and white horehound in water, bathe with that and massage the body with the cleavers.
*I.32.12 Wyrc sealf of marubian on buteran. of wyrm meluwe. of haran sprecele. hegerifan. genim healfe þa sealfe gemeng wiþ gecnuwade elenan smire oþ þæ*t *batige. siþþan mid þære oþerne.*	Make a salve of white horehound in butter, and of worm-meal and viper's bugloss and cleavers, take half of the salve, mix with ground elecampane and smear until it gets better, after that with the other half.
I.32.13 Bæþ wiþ þam miclan lice eolone brom. ifig. mucwyrt ælf þone. beolone. cottuc. efelastan wyl on wætere swiþe geot on bydene 7 sitte on.	A bath for elephantiasis, elecampane, broom, ivy, mugwort, bittersweet nightshade, henbane, common mallow, dog's mercury, boil well in water, pour into a bathtub and sit in it.
*I.32.14 Drince þisne drenc wiþ þon. betonican curmille hofe. agrimonia. springwyrt. reade netle. elehtre. saluie. singrene. alexandria. sie geworht of wiliscum ealað drince o*n *þa*m *baþe 7 ne læte on þone eþm.*	Drink this drink with that, betony, knapweed, ground-ivy, agrimony, caper spurge, red nettle, lupin, common sage, lesser periwinkle and alexanders; let it be made of foreign ale; drink in the bath and do not let the steam in.

(29v 30r) I.32.15 Sealf wiþ þam miclan lice. elene þung ompre. grundeswelge. holecersan. wegbræde. efelaste. ontre. hofe. galluc. celeþonian. cottuc wel on buteran | eal togædere healf sie swines rysele oððe horses smeru. smire þonne mid.	For elephantiasis: elecampane, poison, groundsel, watercress, plantain, dog's mercury, garden radish, ground-ivy, gallnut, greater celandine, common mallow, boil all together in butter, let half be pig's fat or horse's grease, then smear with.
I.32.16 Wið swile genim wegbrædan nioþowearde gecnuwa wiþ rysele lege 7 gebind on þone swile.	For swelling take the lower part of plantain, grind with grease, place over the swelling and bind it.

In subsequent volumes of the *Leechbooks*, Doyle (2017) noted direct parallels between the Old English treatments and recommended remedies from Latin medical sources, such as Orabasius' *Euporistes* (4th century AD) and the *Medicina Plinii* (4th century AD), indicating that Classical medical traditions from the Eastern Mediterranean remained in use through the 10th century AD in Early Medieval England, either through literary tradition or contact with the Arabic medical spheres. The *Leechbooks'* remedies intimate an early conflation of leprosy and elephantiasis, which may be a byproduct of contact with the Arab medical world, but this semantic blurring is used to denote the extent of the disease, rather than using it as a substitution for it, as seen with later Arabic to Latin translations. In all likelihood, both Classical medical traditions and contact with the Arab world likely contributed to Early Medieval medical practices in England. Medical pluralism, i.e. separation of body and soul, and a focus on care and treatment embodied in Early Medieval medical practices is possibly why the archaeological evidence for the medical condition of leprosy poorly aligns with notions of Medieval leprosy stigma.

Most knowledge regarding leprosy in the past is heavily reliant on historical sources, but these should be considered as secondary forms of evidence, or at most corroboration with available physical evidence from archaeological human remains (Murphy and Manchester 2002). As new archaeological findings come to light, a re-examination of available primary evidence, i.e. skeletons revealing evidence for leprosy, is required to evaluate widely cited assumptions about the treatment of people with leprosy in the past

and in order to craft a more holistic understanding of both the social and biological impact of the disease in the past.

2.4 Bioarchaeology of Leprosy

According to Gowland and Knüsel (2006: ix), human remains are the direct evidence of our ancestors in the past and, with that, represent direct evidence of disease in a certain place and time. Whilst historical evidence does not provide sufficient verification for the origins and spread of leprosy diachronically and geographically, archaeological skeletal remains displaying skeletal evidence of leprosy may. Direct evidence of unique skeletal changes linked to the clinical signs of leprosy can help to validate the existence of the disease in specific times and places (Møller-Christensen 1961; Andersen 1969; Browne 1975). Therefore, it worth reviewing the current available bioarchaeological evidence of leprosy from prehistory through to the Early Medieval period, when the disease becomes embedded as a feature of the wider disease landscape in order to understand its development and social significance.

2.4.1 Skeletal changes in leprosy

As leprosy is a disease of the peripheral nervous system (see section 2.2), the pathogenesis of the condition may lead to desensitisation of parts of the body such as the hands and feet, subsequent damage and ulceration, and involvement of the bones. Cranial nerve involvement can also occur, and direct inhalation of the bacteria into the nose and mouth can lead to damage to the facial bones (Møller-Christensen 1961; Roberts and Manchester 2005: 198-199). Additionally, the loss of sensation can potentially cause fatal secondary conditions such as osteomyelitis and sepsis, which can also produce skeletal lesions in the long-term (*ibid*). Since leprosy has a broad immune spectrum, with up to 95% of people showing a natural immunity to the disease (Goulart and Goulart 2008; Rodrigues and Lockwood 2011), the pathogenesis and severity of clinical features within and between individuals varies greatly on the immune spectrum. Individuals may develop the paucibacillary form of the disease (tuberculoid leprosy) with the least severe signs and symptoms, for example skin lesions (granulomata) with few leprosy bacilli within them and a robust immune response in the host (Ridley and Jopling

1966; Goulart and Goulart 2008; Rodrigues and Lockwood 2011). This milder form of the leprosy is generally confined to the soft tissues, and at present is only proposed with the aid of ancient DNA analyses in bioarchaeology. However, Matos (2010) does suggests that bone changes in the hands and feet can sometimes be identified, retrospectively.

In contrast, the multibacillary form of the disease (lepromatous leprosy) incorporates the most severe signs and symptoms, and it is most likely to elicit characteristic skeletal changes that allow us to view the disease archaeologically. Lepromatous leprosy affects the extremities (hands and feet) as well as the mucosa of the upper airways, which can lead to destruction of the facial bones and nasal collapse (*facies leprosa*) (Longmore et al. 2014:490; Goulart and Goulart 2008). The specific bone changes related to clinical signs of lepromatous leprosy were first described by Vilhelm Møller-Christensen (1961; 1978; Møller-Christensen and Andersen 1953), a Danish physician who excavated and analysed archaeological human remains from the Late Medieval period Naevsted leprosarium in Denmark. Møller-Christensen and colleagues (*ibid*) noted that lepromatous leprosy will cause circumferential atrophy of the short bones of the hands and feet, and alveolar resorption of the maxilla with potential loss of the incisors, resorption of the anterior nasal spine, widening and flattening of the nasal bones, and remodelling of the nasal aperture (Longmore et al. 2014: 490; Møller-Christensen 1961). Andersen and Manchester (1992) coined these latter responses as rhinomaxillary syndrome. Rhinomaxillary syndrome, coupled with atrophy and resorption of the bones of the hands and feet, are considered pathognomonic signs of leprosy in bioarchaeology and are diagnostic components of the skeletal changes in individuals with lepromatous leprosy (Møller-Christensen 1961; Andersen and Manchester 1987; 1988; 1992; Andersen et al. 1992; Andersen et al. 1994; Table 2.2). A substantial period of time must elapse (period of chronicity) before an untreated person would manifest these osseous changes, and the longer an individual lives with the disease, the greater the potential for more severe skeletal changes (Wood et al. 1992).

TABLE 2.2 - Common skeletal expressions of leprosy (Møller-Christensen 1961; Andersen and Manchester 1987; 1988; 1992; Andersen et al. 1992; Andersen et al. 1994)

Rhinomaxillary syndrome:

• Resorption of the anterior nasal spine
• Remodelling and widening of the margins of the nasal aperture
• Pitting/porosity of nasal aspect of the maxilla
• Resportion of the maxillary alveolus, with potential loss of maxillary incisors
• Pitting/porosity and/or destruction of the nasal conchae
Neuropathic changes/atrophy of the bones of the hands and feet
Volar grooving in the distal ends of the phalanges of the hands and feet due to fixed flexion deformity/contractures of the proximal interphalangeal joints
Tarsal exostoses
Sub-periosteal new bone formation on the bones of the lower legs and feet, and possibly the forearm bones
Leprogenic odontodysplasia (arrested development in dental growth, especially affecting the maxillary incisors)

It is important to remember that individuals with leprosy can express the signs and symptoms of the disease anywhere within and between the two poles of the immune spectrum (paucibacillary – multibacillary) (Ridley and Jopling 1966). Moreover, the skeletal response to long-term infection is limited and the body is capable of producing skeletal changes that may mimic leprosy. This can make it difficult to diagnose leprosy foremost over other conditions, and create difficulties identifying the disease through time and space, or developing prevalence rates in the past (Wood et al. 1992; Filipek and Roberts 2018; Table 2.3). In sum, although leprosy has a diagnostic suite of skeletal changes, when recording human remains that represent people who may have had leprosy, it is best practice to go back to first principles in palaeopathology: record the basic bone changes representing a pathological stimulus (bone formation and destruction), then document the distribution pattern of the bone changes, and finally compile differential diagnoses. This process will help to avoid the misidentification of leprosy in the past through time and space, which could have a knock-on effect of erroneous interpretations (*ibid*; Table 2.3).

TABLE 2.3 - Differential diagnoses to consider for leprous bone changes (Ortner 2003: 263-265; Papadakis and McPhee 2007: 580-581; Filipek and Roberts 2018; Roberts 2020:131-162).

Differential Diagnoses	**Bones affected**

Treponematoses (yaws, bejel, venereal and congenital syphilis)	Facial, hand, foot, lower leg bones
Lupus vulgaris (skin TB)	Hands, foot, facial bones (rare)
Non-specific infections (osteomyelitis and periostitis due to other systemic stressor; can be concomitant)	Hand, foot, lower leg bones
Septic arthritis (can be concomitant)	Hand, foot, lower leg bones
Diffuse cutaneous leishmaniasis (Figure 2.6)	Facial bones
Rhinomaxillary changes due to other condition (e.g. the treponematoses, congenital saddle nose deformity; infective – rhinoscleroma, etc.)	Facial bones
Trauma (fracture, amputation of toes/feet[natural or intentional])	Foot, lower leg bones
Sarcoidosis	Hand, foot bones
Systemic lupus erythematosus	Hand, foot bones
Amyloidosis or other neuropathic condition (can be concomitant)	Hand, foot bones
Diabetes mellitus neuropathy (or other disease that can cause Charcot joints)	Foot bones
Maduromycosisis (Madura foot), or mycetoma (Figure 2.7)	Foot bones
Sporotrichosis	Facial bones
Psoriatic arthritis	Hand, foot bones
Orofacial gangrene (e.g. Noma in children)	Facial bones

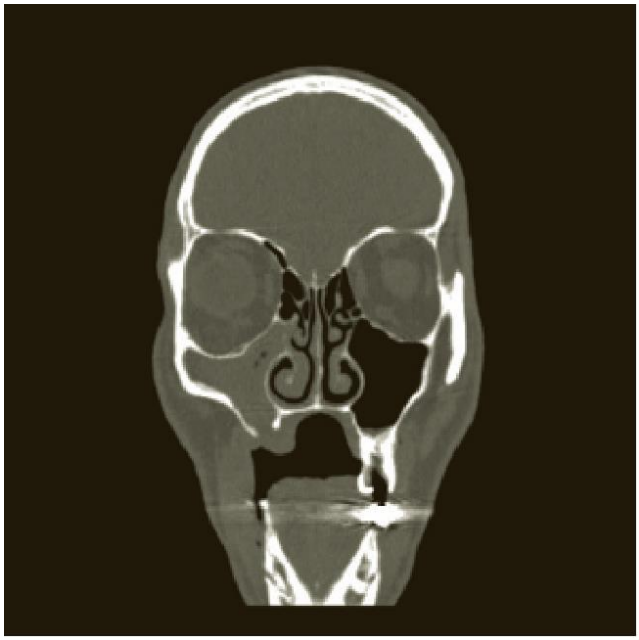


Figure 2.6 Coronal CT scan from male patient with leishmaniasis infection, showing similar bone chances to rhinomaxillary syndrome. Note: maxillary changes in leishmaniasis are often asymmetric (Jabbr et al. 2017: 252. Open Access).

Figure 2.7 A right foot showing skeletal changes consistent with Madura foot, or mycetoma, which can be confused for bone changes associated with leprosy (Ortner 2003: 323).

2.4.2 Leprosy in human remains through time and space

At present, there are three sources of information for leprosy in the archaeological record: macroscopic changes in human remains, analyses of *M. leprae* aDNA from individuals identified with skeletal lesions associated with leprosy, and contextual settings, such as leprosaria (leprosy hospitals). Most of the literature identifying leprosy in archaeological human remains from prehistory through the Early Medieval period is scant and sometimes fails to provide adequate information, including a differential diagnosis, or misidentifies the osseous changes thereby complicating the analysis of primary evidence of leprosy through time and space. According to Roberts' (2020: 191-280) global survey of skeletal evidence for leprosy, only three continents (Asia, Africa, and Europe) have revealed archaeological human remains bearing signs of the disease prior to the Late Medieval Period when leprosy as both a biological and social condition begins to become more endemic. The current bioarchaeological evidence from these areas is detailed below. Evidence from Britain is the focus in a following section.

(I) Prehistoric to Early Medieval Evidence from Asia and the Middle East

The earliest proposed evidence for leprosy in prehistory comes from excavations at Karataş-Semayük (Turkey), and dates to c. 2700-2300 BC (Mellink and Angel 1970). Excavations yielded the remains of an individual (416 KA), who had osseous changes that could have resulted from leprosy (Figure 2.8). However, the authors also proposed

that the changes may be due to a secondary infection from a crush injury, and acknowledged that there were no other skeletal changes consistent with a leprosy diagnosis (*ibid*). At present, Robbins and colleagues (2009) offer the strongest evidence of the oldest case of leprosy in Asian prehistory. Their analyses of the remains of an adult male (Individual 1997) from India (c. 2000 BC) identified pathological changes that could be associated with leprosy, including remodelling around the nasal aperture of the maxilla (Figure 2.9), maxillary antemortem tooth loss, and sub-periosteal new bone formation on the tibiae (Robbins et al. 2009). Robbins and colleagues do offer alternative diagnoses for these bone changes, including leishmaniasis (which does occur in India today), which is equally possible given the asymmetry in the maxillary lesions and the new bone formation on the glabellar region (*ibid*).

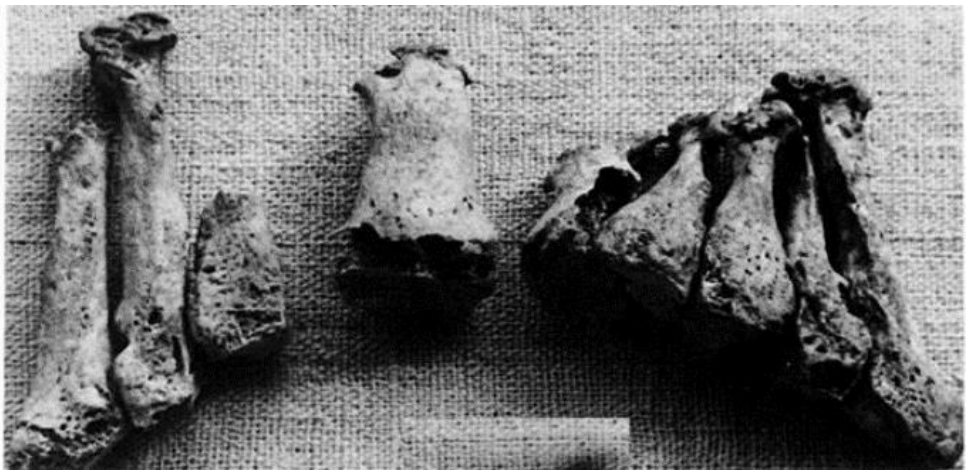


Figure 2.8 Dorsal aspects of metatarsals showing bone changes that may be caused by leprosy or a secondary infection subsequent to a crushing injury from Individual 416 KA (Mellink and Angel 1970: 82).

Evidence from other areas around Asia in the 2nd and 1st millennia is generally problematic in helping to trace leprosy through time and space. For example, Adriaty and colleagues (2012) present *M. leprae* aDNA evidence from an Indonesian skull reportedly from 1000 BC, but they offer no archaeological or scientific evidence as to how this date was established. Tayles and Buckley (2004) describe two male skeletons (one from 300-200 BC and one from 200 AD) with hand and foot joint alterations that could be leprous changes, but the 'cupping' deformities on the phalanges are more reminiscent of psoriatic arthritis, which the authors acknowledge is a possibility.

Zhenbiao (1994) suggests the skull of a female from the Han Dynasty (200 BC – 200 AD) shows evidence of leprosy, but only provide a description of a lesion on the hard palate and some damage to the nasal region, which is not conclusive enough for a diagnosis. Blau and Yagodin (2005) present a female from a Central Asian high-status Kurgan Burial (c. 1st century AD) with bone changes consistent with rhinomaxillary syndrome (Figure 2.10) and subperiosteal new bone formation on the tibiae and fibulae.

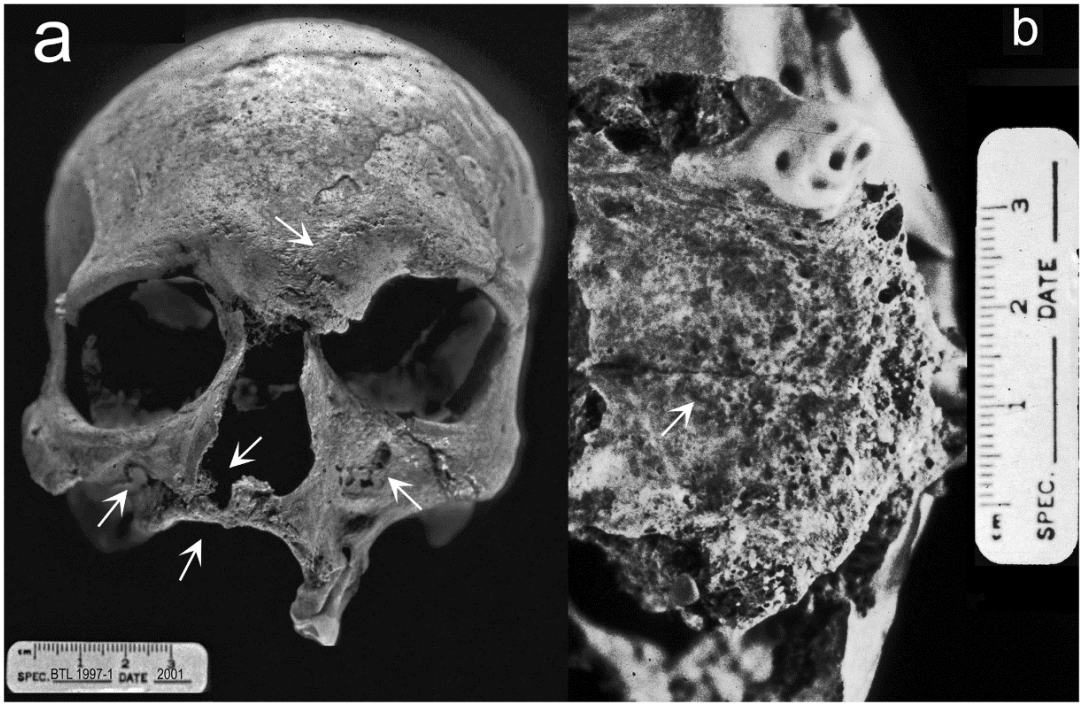


Figure 2.9 Image of Individual 1997 showing nasal and alveolar resorption and pitting on the hard palate of the maxilla (Robbins et al. 2009: https://doi.org/10.1371/journal.pone.0005669.g003. Open Access).

Unfortunately, the hand and foot bones were not available for analysis, but subsequent *M. leprae* aDNA analyses by Donoghue and colleagues (2015) revealed the presence of *Mycobacterium leprae*, making it the first biomolecularly verified evidence of the disease in time and space (Table 2.4). Skeletal evidence of leprosy within Asia is not reported again until the 4th - 8th centuries AD in children and adults in familial tombs and monastic sites in Israel and elsewhere in the Eastern Mediterranean (Manchester 1993; Rafi et al. 1994; Rubini et al. 2014a). The inclusion of these individuals within the

normal burial traditions of their collective communities signifies that, archaeologically, evidence of stigma was absent during these time periods and places.

Figure 2.10 Cranium of a middle-aged female from Uzbekistan with bone changes consistent with rhinomaxillary syndrome (Blau and Yagodin 2005: 153).

TABLE 2.4 - Current Skeletal Evidence for leprosy in Asia (Prehistory to Early Medieval periods).

SITE/LOCATION	TIME PERIOD	LIKELIHOOD	CITATION
KARATAŞ-SEMAYÜK (TURKEY)	2700 – 2300 BC	Possible	Mellink and Angel (1970)
BALATHAL (INDIA)	c. 2000 BC	Probable	Robbins et al. (2009)
NOEN U-LOKE (THAILAND)	300 BC – 200 AD	Possible	Tayles and Buckley (2004)
SHAANXI PROVINCE (CHINA)	200 BC – 200 AD	Inconclusive	Zhenbiao (1994)
USTYURT PLATEAU (UZBEKISTAN)	80 – 240 AD	Confirmed (*Mycobacterium leprae* aDNA+; 3L Type)	Blau and Yagodin (2005)
BET GUVRIN (ISRAEL)	300 – 600 AD	Confirmed (*Mycobacterium leprae* aDNA +)	Manchester (1993)
MONASTERY OF ST. JOHN THE BAPTIST (ISRAEL)	c. 600 AD	Confirmed (*Mycobacterium leprae* aDNA +)	Rafi et al. (1994)
KOVUKLUKAYA (TURKEY)	8^{th} – 10^{th} centuries AD	Confirmed (*Mycobacterium leprae* aDNA +)	Rubini et al. (2014a)

(Cases were deemed possible if showing lesions consistent with leprosy; probable if showing lesions diagnostic of leprosy; and confirmed where aDNA has verified the presence of *Mycobacterium leprae* using DNA analysis. The author acknowledges that a positive *M. leprae* result does not directly link to the bone changes, meaning there is a small possibility that the lesions were due to a prior or concomittant pathological insult)

(II) Prehistoric to Early Medieval Evidence from Africa

Almost all evidence of disease from archaeological human remains analysed in Africa come from Egypt and its surrounding areas (e.g. Sudan). Despite the wealth of human skeletal remains from this region, there are very few cases of leprosy present in the archaeological literature. Notable among these are four 'white' skulls from the Dakhleh Oasis in Egypt, dating to the Ptolemaic period (2^{nd} – 1^{st} centuries BC) (Dzierzykray-Rogalski 1980). Dzierzykray-Rogalski (1980) presented evidence of pathological changes on the bones of these individuals but he did not offer alternative diagnoses, and the evidence cited in the diagnoses was not adequately illustrated in the accompanying figures. In the absence of other indicators of leprosy, the diagnosis of lepromatous leprosy seems suspect and it would be highly beneficial for these skulls to be re-evaluated using more current diagnostic criteria. This critical reappraisal of oft-cited diagnoses is an integral aspect to understanding a disease's origins and spread through time, and underscores the need for rigorous reinterrogation of formative literature.

Conversely, Molto (2002) presents evidence of leprosy from four males dating to the Roman period (c. 300-450 AD) in the Kellis 2 cemetery of the Dakhleh Oasis in modern-day Egypt. Molto's analysis identifies two individuals with changes that are consistent with lepromatous leprosy including rhinomaxillary syndrome and atrophy of the bones of the hands and feet, and two individuals possessing probable evidence of the disease, but in a potentially less-severe form, or who had died before the bone changes had progressed (*ibid;* Figure 2.11).

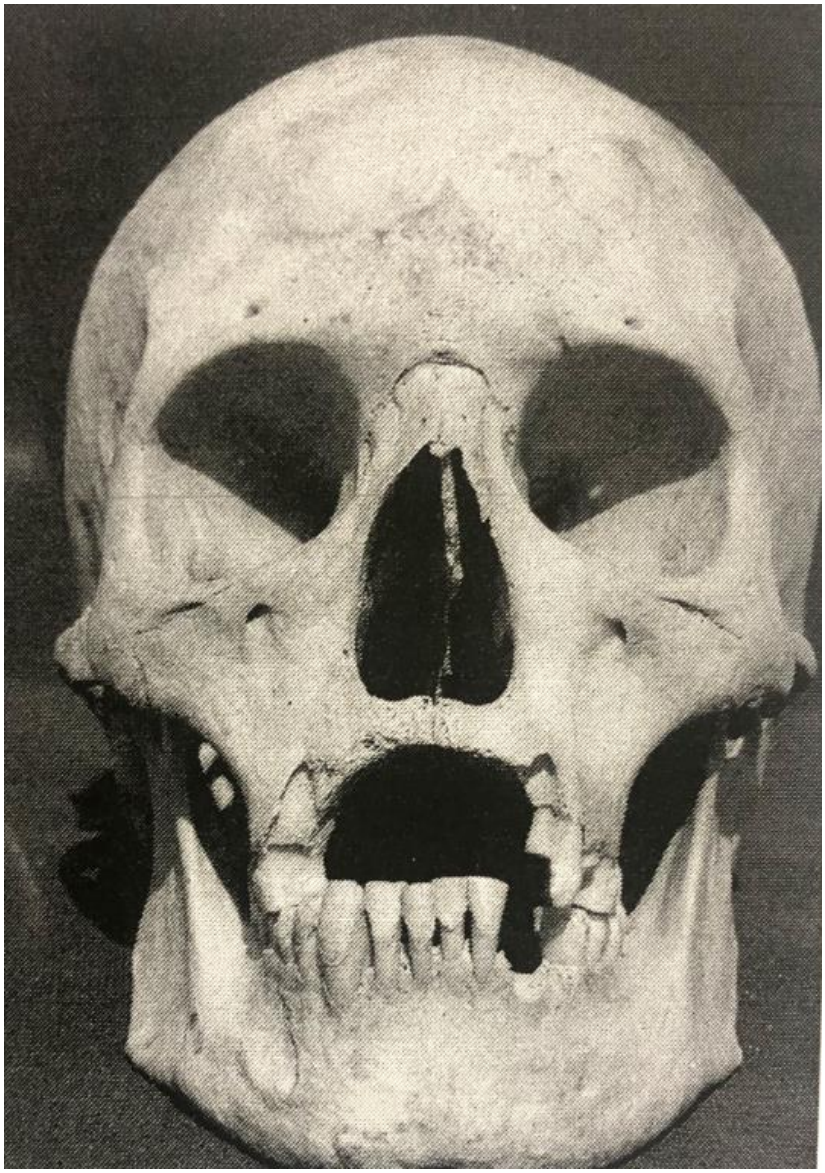


Figure 2.11 Skull of Individual B6 from the Kellis 2 cemetery in the Dakhleh Oasis (Egypt) displaying signs of rhinomaxillary syndrome (Molto 2002: 187).

With regard to the development of palaeopathology, early interest in the antiquity of diseases came from Grafton Elliot-Smith, a neuro-anatomist who allegedly dissected c. 30,000 mummies, and his associate Warren Dawson, an insurance salesman who became a self-taught anthropologist (Burne and Young 1911; Elliot-Smith and Dawson 1924). In their analyses of Egyptian and Nubian mummies, they identified one potential case of leprosy from a Coptic Christian burial located in El Biga (modern-day Sudan), which was restudied by Møller-Christensen and Hughes in the 1960s. Møller-Christensen and Hughes (1966) confirmed Elliot-Smith's earlier assessment and

confirmed that the cranium from El Biga did possess bone changes associated with rhinomaxillary syndrome, and thus was consistent with leprosy (Figure 2.12). At present, these are the only published archaeological examples of leprosy prior to the Later Medieval Period (c. 12th century AD) from the African continent. Monot and colleagues' (2005; 2009) genomic analyses of extant and ancient *M. leprae* strains indicate an East African or Near Eastern origin for leprosy, and hypothesise a wider transmission of the disease along the Silk Road from the 1st century AD. If accurate, this would mean that the rarity of evidence of leprosy in archaeological human remains in Africa (and Asia) is likely due to a combination of factors including access, training in the identification of diseases in human remains, research interests, and skeletal preservation (Baker and Judd 2012:209; Roberts and Buikstra 2012:771).

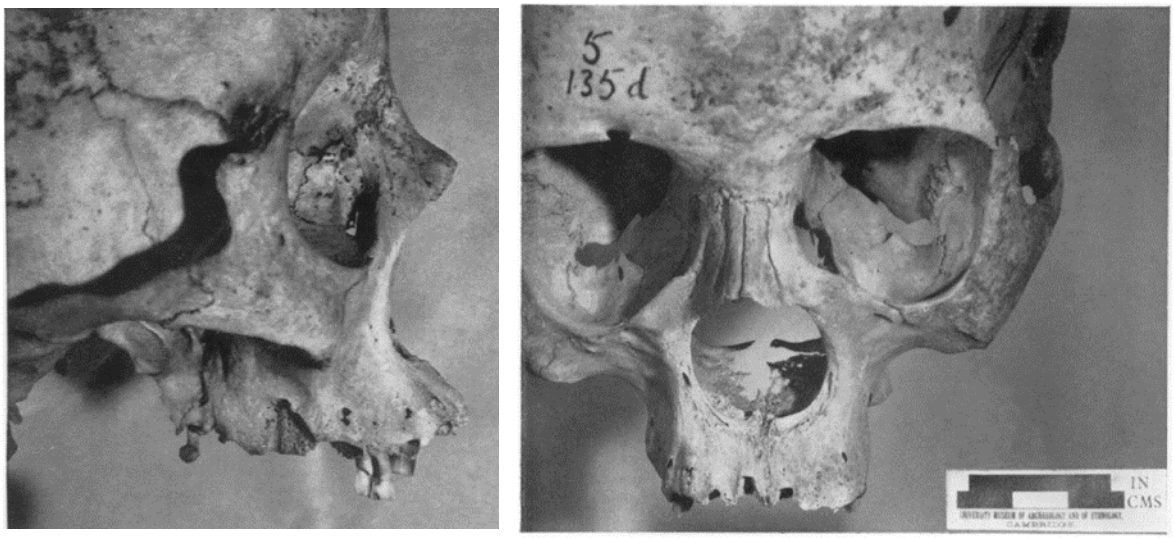


Figure 2.12 The cranium of the individual from El Biga (Sudan) demonstrating resorption of the anterior nasal spine, remodelling of the nasal aperture, and changes to the hard palate associated with the rhinomaxillary changes consistent with leprosy (Møller-Christensen and Hughes 1966: 244-45).

TABLE 2.5 - Current Skeletal Evidence for Leprosy in Africa (Prehistory to Early Medieval periods).

SITE/LOCATION	TIME PERIOD	LIKELIHOOD	CITATION
DAKHLEH OASIS (EGYPT)	c. 200 BC	Possible	Dzierzykray-Rogalski (1980).

KELLIS 2, DAKHLEH OASIS (EGYPT)	c. 300 – 450 AD	Confirmed (*Mycobacterium leprae* aDNA+)	Molto (2002)
EL BIGA (SUDAN)	4th – 7th centuries AD	Probable	Elliot-Smith and Dawson (1924); Møller-Christensen and Hughes, (1966).

(Cases were deemed possible if showing lesions consistent with leprosy; probable if showing lesions diagnostic of leprosy; and confirmed where aDNA has verified the presence of *Mycobacterium leprae* using DNA analysis. The author acknowledges that a positive *M. leprae* result does not directly link to the bone changes, meaning there is a small possibility that the lesions were due to a prior or concomittant pathological insult)

(III) Prehistoric to Early Medieval Evidence from Continental Europe

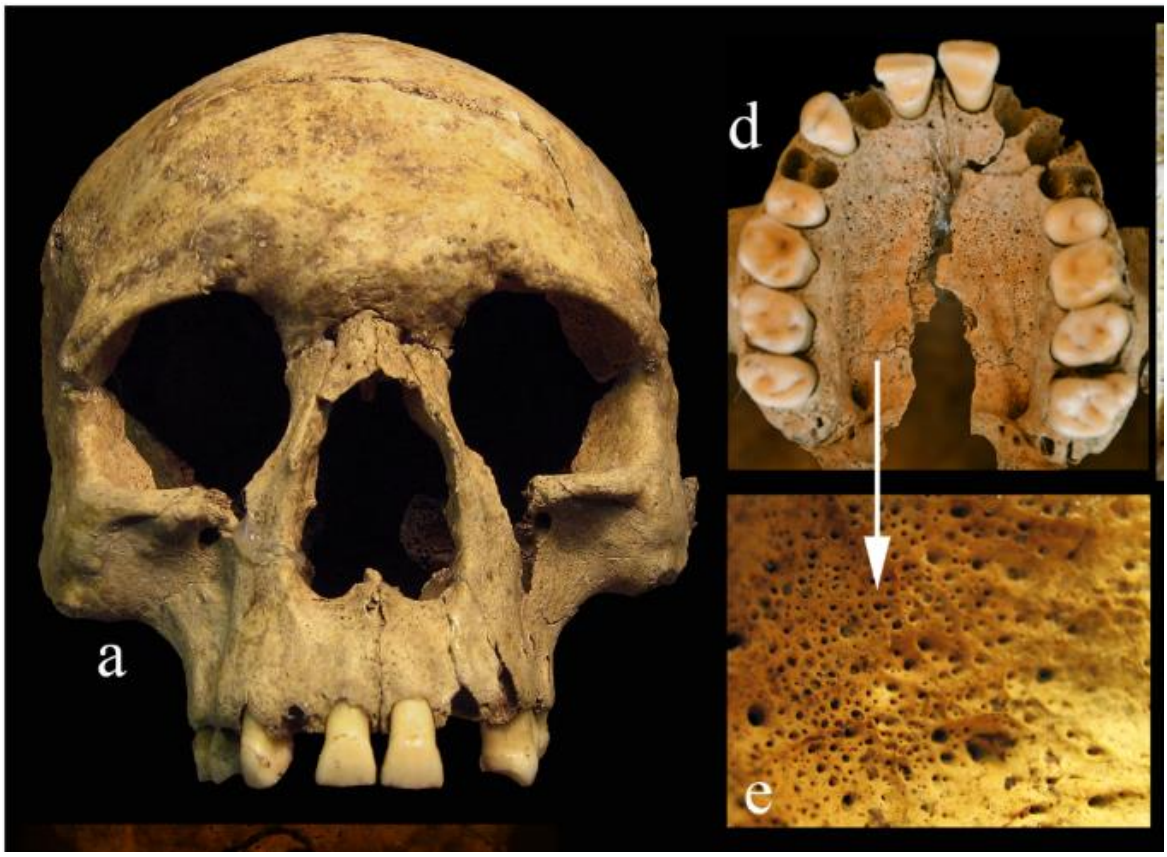


Figure 2.13 Image of a young-adult male from the Copper Age (c. 3780-3650 BC) Carpathian Basin displaying maxillary porosity possibly due to leprosy (Köhler et al. 2017: https://doi.org/10.1371/journal.pone.0185966.g004. Open Access).

In contrast to the current evidence in Asia and Africa, Prehistoric to Early Medieval Continental Europe is relatively well-populated with examples of human remains potentially and definitively exhibiting changes consistent with leprosy. This is likely an artefact of a combination of bioarchaeology training programmes, especially in the UK,

research interests, and access to thousands of human remains within European institutions (Roberts and Buikstra 2012: 770-772). Despite this presenting a somewhat subversive view regarding disease trajectories through time, an evaluation of the skeletal evidence in Europe can nonetheless provide us with a broader approach to this disease through place and time.

Within modern-day Europe, Köhler and colleagues (2017) suggest the oldest case of leprosy comes from the remains of a young-adult male (18-22 years old) from the Copper Age (3780–3650 cal BC) who was buried in the Carpathian Basin (modern-day Hungary). Although the researchers do provide a differential diagnosis, the osseous changes presented are equally as likely to be a consequence of trauma, dental disease, and non-specific infection (*ibid*; Figure 2.13). Three attempts to extract *M. leprae* and *M. tuberculosis* aDNA from this individual, as well as from four others yielded negative results, but other aDNA extraction (e.g. mtDNA) was successful, implying the lack of pathogen DNA was not due to taphonomic factors (*ibid*). Lipid extraction and mycolic acid analyses may help to determine the presence of *M. leprae* in these skeletons in future.

Figure 2.14 Right (top row) and left (bottom row) metatarsals from individual t. 74 buried at the necropolis of Casalecchio di Reno (Marriotti et al. 2005: 314).

In modern-day Italy, Mariotti et al. (2005) found evidence of leprosy in the skeletal remains of an adult ('Celtic') male from the necropolis of Casalecchio di Reno (Bologna) from the early 4th – 3rd centuries BC. The diagnosis was based on macroscopic and radiographic evidence consistent with documented leprous changes. The postcranial changes do show what appears to be an infective process in the foot bones (Figure 2.14) that may be due to leprosy, but they may also be a consequence of other disease processes including diabetes and mycetoma (Table 2.3). The authors note that although they observed some maxillary changes, the presence of the anterior nasal spine in combination with postmortem damage made the assessment of these changes indeterminable for leprosy (*ibid*).

Kjellström (2012) refers to an unpublished example of lepromatous leprosy from the Iron Age/Early Roman Period (c. 1st – 4th centuries AD) in her paper about Later Medieval leprosy in Sweden. However, without further information, it is not possible to

verify the presence of leprosy in this place and time. This emphasises the unfortunate barriers of unpublished archaeological reports and big picture approaches to disease at both the biological and social levels, especially given the significance of leprosy in Scandinavia in the Later Medieval period. Notwithstanding, verifiable archaeological evidence of leprosy begins to increase from the Roman to Early Medieval periods within Europe, likely due to extended trade networks along the Silk Road, the movement of people long and short distances, and repeated military conflicts (Mark 2002; Binder 2018; Donoghue et al. 2018; Mark 2019). Buikstra and Lagia (2009) identified possible leprosy in a Christian (3rd – 6th centuries AD) ossuary on the island of Kos, Greece, but the context was commingled and made the evaluation of the available skeletal elements difficult. They describe a number of metatarsals as 'pencil-like' and noted the presence of subperiosteal new bone formation on many of the long bones, citing the possibility that these changes may be a consequence of leprosy. However, as the researchers have not yet identified any other pathognomonic changes, confirmation of leprosy in this place and time is still pending (Buikstra and Lagia 2009: 16).

Figure 2.15 Skull of Individual GM162 (4 – 5 years old) from Martellona (c. 2nd – 3rd centuries AD) showing possible skeletal changes associated with lepromatous leprosy (Rubini et al. 2014a: 574).

Rubini and colleagues (2014a) identify both a non-adult (4 – 5 years old) from Roman period Martellona (Central Italy, c. 2nd – 3rd centuries AD) and a non-adult (4 – 5 months old) from Byzantine Period Turkey (c. 8th – 10th centuries AD) with macroscopic changes and aDNA verification of *Mycobacterium leprae*, respectively (see Table 2.6). Although the bone changes in the skull of the Roman Period child (Individual GM162) resemble lepromatous leprosy, results for *M. leprae* aDNA extraction were negative (although that does not mean they did not have it), and the cortical surface was heavily affected by taphonomic changes, which could be mimicking leprosy (Figure 2.15). Further, no postcranial bones were available for examination. Rubini and colleagues offer a differential diagnosis for the bone changes in the Roman child, but discount many of the plausible alternatives (2014a). Rubini and colleagues (2014b) also present a case study of three maxillae from Palombara (Central Italy) radiocarbon dated to 450 - 500 AD, with bone changes diagnosed as leprosy. The maxillae were found amongst a large commingled context and could not be associated with other skeletal elements (*ibid*). They describe the three maxillae of the individuals from Palombara as having

pathognomonic signs of lepromatous leprosy, however the figures presented demonstrate lesions that are consistent with, but not diagnostic of the condition (e.g. porosity on the hard palate).

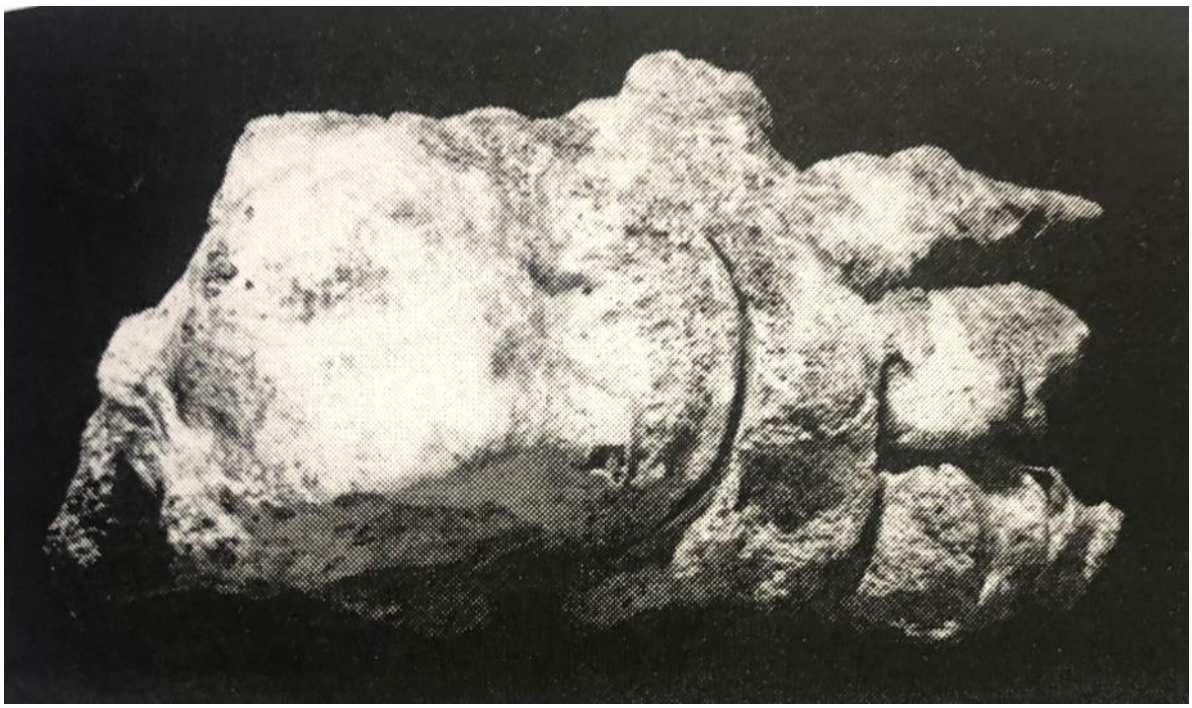


Figure 2.16 Individual 23 from Vaison La Romaine (Southern France, c. 500 AD) showing complete resorption of the phalanges and most of the metatarsals, tarsal coalition, and tarsal exostoses (Blondiaux et al. 2002: 109).

Blondiaux and colleagues (1994; 2002) present evidence of leprous bone changes to the bones of the feet and lower limbs in two individuals from c. 500 AD France. Individual 23 from Vaison La Romaine (Southern France) was a mature adult male showing evidence of both rhinomaxillary syndrome and atrophy of the bones of the hands, lower limbs, and feet (Figure 2.16), and individual 67 from Neuville Sur Escaut (Northern France) showed bone changes only to the lower limbs and feet (*ibid*). Blondiaux and colleagues (2002) also highlighted through crystallographic, radiographic, and microradiographic methods that the histological bone changes in leprosy mimicked treponemal disease, but could be discriminated from tuberculosis. Given that treponemal diseases and leprosy can be similar in macroscopic and histological osseous appearance (Schultz and Roberts 2002), it is crucial to include treponematoses in any differential diagnosis for leprosy in bioarchaeology.

An Early Medieval cemetery in Lauchheim in modern-day Germany (5th – 7th centuries AD) also reveals skeletal evidence of leprosy in this time period and place (Boldsen 2008). Boldsen (2008) evaluated and scored skeletal lesions associated with leprosy from the remains of 110 individuals and estimated a prevalence amongst the Early Medieval Lauchheim population to be approximately 16%. Rubini and Zaio (2009) and Belcastro and colleagues (2005) present more evidence from Central Italy of possible and likely bone changes associated with leprosy from Avar contexts dating from the 6th – 8th centuries AD. The Avars were a semi-nomadic barbarian group associated with modern-day Hungary that ultimately derived from Mongolia, Siberia, and Transcaucasia (Georgia, Armenia, Azerbaijan) (*ibid*). Belcastro and colleagues (2005) present convincing evidence of leprosy from a male aged 20-25 excavated from tomb 144 in the cemetery of Vicenne-Campochiaro. Individual t. 144 showed skeletal indicators consistent with lepromatous leprosy including acro-osteolysis of the hands and feet, and rhinomaxillary changes including hard palate perforations, pitting and remodelling of the nasal aperture, and subperiosteal new bone formation on the bones of the lower limbs (Belcastro et al. 2005). Similarly, Rubini and Zaio (2009) present evidence of two individuals from a similar Avar-associated burial context in the early-Medieval (6th – 8th centuries AD) necropolis of Morrione (Central Italy); an adult female (aged 40-46) bearing possible skeletal signs of lepromatous leprosy, as well as an adult male (aged 50-55 years) with more convincing evidence of lepromatous leprosy, including rhinomaxillary syndrome and acro-osteolysis of the hand and foot bones.

Figure 2.17 The maxilla of an Avar-period (6th – 8th centuries AD) adult male from Morrione (Central Italy) showing bone changes diagnostic of lepromatous leprosy (Rubini and Zaio 2009: 2775).

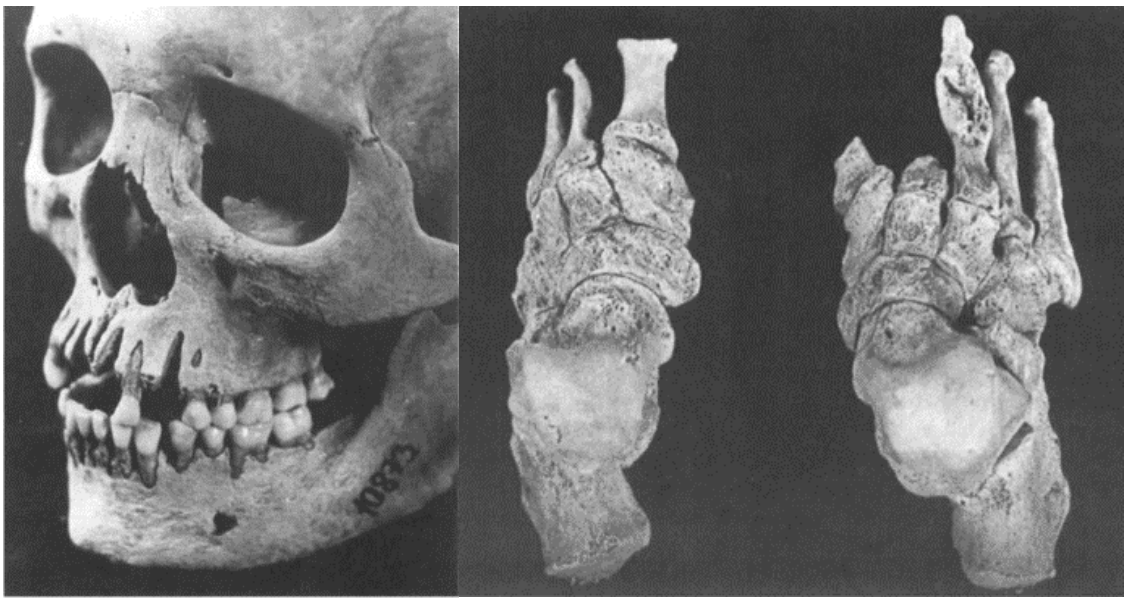


Figure 2.18 Facial and foot skeletal changes diagnostic of lepromatous leprosy from an older adult female (aged 50-60) excavated from Sárrétudvari-Hízóföld (10th century AD)(Pálfi 1991: 100).

Congruent with the Italian burial contexts, much of the archaeological evidence for

leprosy in Continental Europe comes from similar time periods in modern-day Hungary and its surroundings. Pálfi (1991) presented the first archaeological evidence of lepromatous leprosy in a 50-60 year-old female excavated from Sárrétudvari-Hízóföld, a 10th century AD cemetery site. Individual 202 displayed skeletal lesions consistent with lepromatous leprosy, including rhinomaxillary syndrome, acro-osteolysis of the feet, and tarsal coalition (Pálfi 1991; Figure 2.18).

Since Pálfi's (1991) formative work, numerous other cases of leprosy have been identified from modern-day Hungary and its surrounding regions, including modern-day Austria, Czechia, and Croatia (Table 2.6). The late 7th century AD Avar-period cemetery of Szeged-Kiskundorozsma-Daruhalom dűlő II currently provides the earliest verified evidence of leprosy in modern-day Southern Hungary (Paluch and Szalontai 2004; Mészaros et al. 2006; Molnár et al. 2006). Up to nine individuals within this cemetery consisting of 94 burials (9.5%) show bone changes consistent with rhinomaxillary syndrome, subperiosteal new bone formation on the bones of the lower limbs, and acro-osteolysis of the foot bones (*ibid*). The presence of the aDNA of *Mycobacterium leprae* within three of these individuals was later verified by Donoghue and colleagues in a broader study on aDNA analyses of leprosy within this time period and region (Donoghue et al. 2015). An additional eight Early Medieval (7th – 11th centuries AD) cemetery sites within modern-day Hungary have revealed individuals (n= 11) with bone changes consistent with lepromatous leprosy, possibly indicating that either leprosy was endemic within this time period and place, the type of leprosy was more virulent and left more pathognomonic skeletal changes, and/or there is an increased research interest there, which may skew its representation (Table 2.6) (Haas et al. 2000, Molnár and Marcsik 2002, Fóthi et al. 2002, Mészaros et al. 2006, Marcsik et al. 2009, Pálfi and Molnár 2009, Csóri et al. 2009, Donaghue et al. 2015). Similarly, other parts of Central Europe, including modern-day Austria, Czechia, and Croatia have revealed cemeteries with individuals showing skeletal lesions consistent with lepromatous leprosy. In modern-day Croatia, at the Early Medieval (8th – 9th centuries AD) Radašinovci cemetery site, 6/124 individuals (four males, two females) showed evidence of lepromatous leprosy within the facial, hand, foot, and lower limb bones; they were buried within the normal confines of the cemetery (Slaus 2006; Bedic et al. 2019; Adamic and Slaus 2016).

Two additional female individuals (one adult and one older adolescent) from the archaeological excavations at Bijelo Brdo (10th – 11th centuries AD) in modern-day Croatia also show evidence for rhinomaxillary syndrome but, as the excavations occurred at the turn of the 20th century, many of the skeletal remains were not recovered and the excavation records are scant (Bedic et al. 2019). Because of the sparsity of skeletal elements available for evaluation, Bedic and colleagues (2019) analysed the suspected maxilla for aDNA evidence of *Mycobacterium leprae*, which verified the disease was present. Similar cases of leprosy have been identified in 8th – 10th centuries AD archaeological contexts in both modern-day Austria and Czechia. At the Avar-period (8th – 9th century AD) site of Zwölfaxing (Austria), an older female (65-70 years) and a younger male (25-30 years) show possible changes in the facial bones, but present more firm evidence in acro-osteolysis of the foot bones (Szilvássy 1980; Donaghue et al. 2015).

Figure 2.19 Skull of Individual 188 from Prušánky 1 (Czechia) displaying rhinomaxillary changes associated with lepromatous leprosy (Donoghue et al. 2015: S1).

At the Greater Moravian Empire (9th – 10th centuries AD) site of Pružánky 1 (Czechia), the remains of individual 188 (aged 12-14) were recovered displaying skeletal signs of lepromatous leprosy, including rhinomaxillary syndrome (Figure 2.19), as well as some destructive lesions in the hand and foot bones. (Taylor and Donoghue 2011; Donoghue et al. 2015; Schuenemann et al. 2018). Subsequent biomolecular verification of the disease in this child was undertaken by Donoghue and colleagues (2015).

By the 11th century AD, skeletal evidence of lepromatous leprosy was firmly established in Scandinavia, and it remained an epicentre for the disease until the 20th century. In modern-day Sweden, at the site of Humlegården (Sigtuna), individuals 19 and 34 (900 – 1100 cal AD) and at least eight others from the 12th – 14th centuries AD revealed skeletal indicators of lepromatous leprosy, including rhinomaxillary syndrome, subperiosteal new bone formation on the bones of the lower limbs, and resorption of the hand and foot bones (Kjellström 2010; Economou et al. 2013). Leprosy has also been identified in several 12th century AD sites in modern-day Denmark, including Odense, St. Mikkel, Viborg, and Refshale, and an abundance of examples are found in the Later Medieval periods (Møller-Christensen 1961; Andersen 1969; Boldsen 2009).

TABLE 2.6 - Current Skeletal Evidence for Leprosy in Continental Europe (Prehistory to Early Medieval periods).

SITE/LOCATION	TIME PERIOD	LIKELIHOOD	CITATION
CARPATHIAN BASIN (HUNGARY)	3780–3650 cal BC	Possible	Kohler et al. (2019)
CASALECCHIO DI RENO, BOLOGNA (ITALY)	c. 4th – 3rd centuries BC	Possible	Mariotti et al. (2005)
SCANIA (DENMARK)	1st – 4th centuries AD	Possible	Kjellstrom (2012)
MARTELLONA (CENTRAL ITALY)	2nd – 3rd centuries AD	Possible	Rubini et al. (2014a)
KOS (GREECE)	3rd – 6th century AD	Possible	Buikstra and Lagia (2009)
PALOMBARA (CENTRAL ITALY)	450-500 AD	Possible	Rubini et al. (2014b)
VAISON LA ROMAINE (SOUTHERN FRANCE)	c. 500 AD	Probable	Blondiaux et al. (2002)
NEUVILLE SUR ESCAUT (NORTHERN FRANCE)	5th – 6th centuries AD	Probable	Blondiaux et al. (2002)

LAUCHHEIM (GERMANY)	5th – 7th centuries AD	Probable	Boldsen (2008)
MORRIONE (CENTRAL ITALY)	6th – 8th centuries AD	Confirmed (*Mycobacterium leprae* aDNA+)	Rubini and Zaio (2009)
VICENNE-CAMPOCHIARO (ITALY)	7th century AD	Confirmed (*Mycobacterium leprae* aDNA+)	Belcastro et al. (2005)
SZEGED-KISKUNDOROZSMA-DARUHALOM DŰLŐ II (HUNGARY)	7th century AD	Confirmed (*Mycobacterium leprae* aDNA+; 3K Type)	Paluch and Szalontai (2004); Mészaros et al. (2006); Molnár et al. (2006)
SZENTES-KISTŐKE (HUNGARY)	7th – 8th century AD	Confirmed (*Mycobacterium leprae* aDNA+)	Pálfi and Molnár (2009)
SZARVAS GREXA, TÉGLAGYÁR (HUNGARY)	7th – 9th century AD	Confirmed (*Mycobacterium leprae* aDNA+)	Molnár and Marcsik (2002); Marcsik et al. 2009
BÉLMEGYER-CSÖMÖKI DOMB: 22 (HUNGARY)	8th – 9th centuries AD	Confirmed (*Mycobacterium leprae* aDNA+)	Molnár et al. (2015)
RADAŠINOVCI (CROATIA)	8th – 9th centuries AD	Confirmed (*Mycobacterium leprae* aDNA+)	Watson and Lockwood (2009)
ZWÖLFAXING (AUSTRIA)	8th – 9th centuries AD	Confirmed (*Mycobacterium leprae* aDNA+)	Szilvássy (1980)
PRUŠÁNKY (CZECHIA)	9th – 10th centuries AD	Confirmed (Mycobacterium leprae aDNA+; 3M Type)	Donoghue et al. (2015)
SÁRRÉTUDVARI-HÍZÓFÖLD (HUNGARY)	10th century AD	Confirmed (*Mycobacterium leprae* aDNA+)	Pálfi (1991)
HAJDÚDOROG-GYÚLÁS (HUNGARY)	10th century AD	Confirmed (*Mycobacterium leprae* aDNA+)	Csóri et al. (2009)
PÜSPÖKLADÁNY-EPERJESVÖLGY (HUNGARY)	10th – 11th centuries AD	Confirmed (*Mycobacterium leprae* aDNA+; 3K and 3M Type)	Csóri et al. (2009)
HUMLEGÅRDEN, SIGTUNA (SWEDEN)	900-1100 cal AD	Confirmed (*Mycobacterium leprae* aDNA+)	Kjellström (2010); Economou et al. (2013)

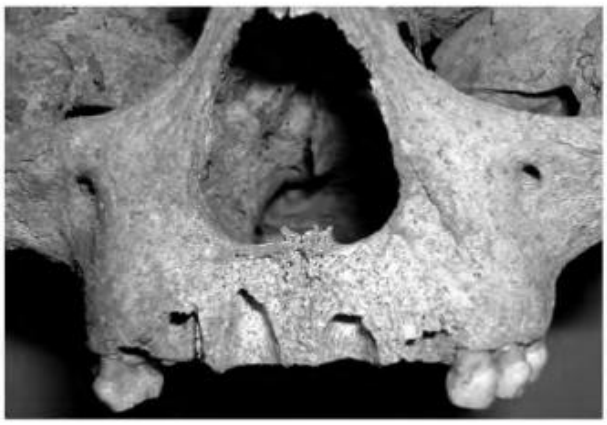
(Cases were deemed possible if showing lesions consistent with leprosy; probable if showing lesions diagnostic of leprosy; and confirmed where aDNA has verified the presence of *Mycobacterium leprae* using DNA analysis. The author acknowledges that a positive *M. leprae* result does not directly link to the bone changes, meaning there is a small possibility that the lesions were due to a prior or concomittant pathological insult)

2.4.3 Bioarchaeology of Leprosy in Prehistoric to Early Medieval Britain

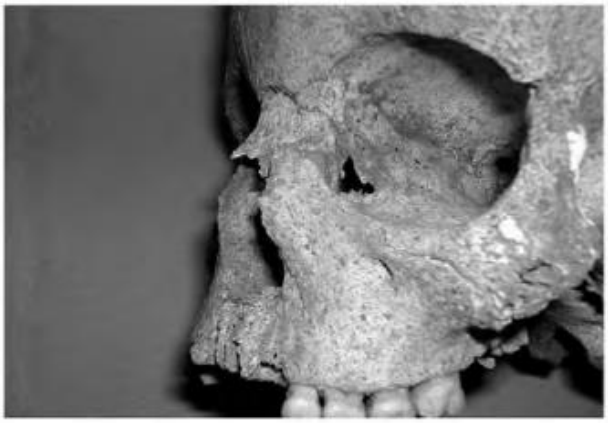
The oldest possible evidence for leprosy from the Britain comes from modern-day Scotland. In their site report, Roberts (2007) poses the possibility of rhinomaxillary syndrome based on erosions of the anterior nasal spine in the remains of a child (aged 6-8) recovered from a cist burial in East Lothian, Scotland (Figure 2.20). The author is conservative in their leprosy assessment, offering alternative diagnoses of an unidentified congenital anomaly or an infection affecting the rhinomaxillary region. This conservatism is likely due to the absence of other available elements for evaluation (e.g.

the bones of the hands, feet, and lower limbs), and the radiocarbon date of the remains (2280-1970 cal B.C.), which would not only predate other evidence of leprosy in Britain by 1500 years, but also offer one of the earliest cases of leprosy identified in the world. aDNA analyses were attempted to verify the presence of the bacterium, but *Mycobacterium leprae* was not detected due to either preservation factors or misdiagnosis (Roberts 2007). Interestingly, *Mycobacterium tuberculosis* was detected, indicating that this child may have suffered from lupus vulgaris, or simply harboured tuberculosis bacilli (*ibid*).

It is not until the Romano-British period (c. 4th century AD) when possible skeletal evidence of leprosy reappears in the archaeological record and increases into the Early Medieval period (Reader 1974; Roberts 2002; Table 2.7; see also Roberts 2020: Appendix 3). The earliest confirmed case of leprosy in Britain date to the 5th – 6th centuries AD from Great Chesterford in Essex. Inskip and colleagues (2015) analysed the remains of a 21-35 year old male with skeletal lesions consistent with lepromatous leprosy. Biomolecular analyses verified the presence of strain type 3I of *Mycobacterium leprae* (*ibid*). Researchers have identified this type in other Early Medieval skeletons displaying skeletal evidence of leprosy, and it is extant amongst red squirrel populations on Brownsea Island in Dorset (Schuenemann et al. 2013; Economou et al. 2013; Avanzi et al. 2016; Inskip et al. 2017).



Illus 15 Burial 11; widening and remodelling of nasal aperture; and resorption of the alveolar process of the maxilla



Illus 16 Burial 11; rhino-maxillary changes in profile

Figure 2.20 Cranium of a child from Scotland (c. 2000 BC) showing widening of the nasal aperture and resorption of the maxilla (Roberts 2007:23.Open Access).

Most of the Early Medieval sites revealing individuals with possible and confirmed cases of leprosy are found in England, either in the East or the South (Manchester and Roberts 1989; Roberts 2002: Table 2.7; see also Roberts 2020: Appendix 3). This may be an indication of Early Medieval trade and migratory routes creating pockets of endemicity in England, or it may be a consequence of archaeological factors such as poor preservation (not seen in North and West where acidic soils affect preservation) or increased building works in the southern and eastern regions where excavation is more common (in advance of modern development). However, in the Early Medieval period, areas such as modern day East Anglia and Hampshire have shown increasing immigration and population densities (Härke 2011; Bevan 2012) and, therefore, it would not be

unsurprising to discover a higher prevalence of leprous skeletons in these more urbanised areas (Inskip et al. 2017).

As with Asia, Africa, and the rest of Europe, the majority of archaeological sites revealing leprous skeletons are from "normal" burial contexts for the location and time period, and they usually only reveal one or a few individuals with bone changes consistent with lepromatous leprosy (Manchester and Roberts 1989; Roberts 2002; Table 2.7; see also Roberts 2020: 191-280). There may be others with leprosy but without bone changes at the time of death represented in the burial assemblages (e.g. those with tuberculoid leprosy), there is nothing to indicate they were stigmatised based on having leprosy. Roberts (2002) surveyed 41 archaeological sites from the Roman to post-Medieval periods in Britain that yielded individuals with skeletal lesions consistent with leprosy, and found that 36/41 sites had individuals buried within the normal confines of the communities. The remaining five were buried in cemeteries associated with leprosy hospital sites, which tended to produce multiple individuals with leprous bone changes (*ibid*). Although leprosy hospital sites tend to reveal higher concentrations of individuals with skeletal lesions diagnostic of leprosy, other Early Medieval contexts also reveal high percentages of people with leprosy within normal parish cemetery contexts. For example, archaeologists reported a significant number of people with leprosy (24/181), including adolescents, at the Late Saxon St. John at the Castle Gate/Timberhill site (980-1050 AD) near Norwich Castle (Anderson 1996; 1998; Shepherd Popescu 2009). The biocultural contexts in which the monastic hospitals and leprosaria developed will be discussed in the following chapter.

TABLE 2.7 - Current and Possible Skeletal Evidence for Leprosy in Britain (Prehistory to Early Medieval periods).

SITE/LOCATION	TIME PERIOD	CITATION
EAST LOTHIAN, SCOTLAND	2280 – 1970 cal B.C.	Roberts (2007)
POUNDBURY, DORSET	4th century AD	Reader (1974)
CANNINGTON, SOMERSET	3rd – 8th centuries AD	Brothwell et al. (2000)
GREAT CHESTERFORD, ESSEX	415 – 545 cal AD	Inskip et al. (2015)

BROUGHTON LODGE, WILLOUGHBY-ON-THE-WOLDS, NOTTINGHAMSHIRE	5th – 7th centuries AD	Roberts (1993)
COLLINGOURNE DUCIS, WILTSHIRE	5th – 7th centuries AD	Dinwiddy (2016)
BECKFORD, GLOUCESTERSHIRE	6th century AD	Wells (1962)
BARRINGTON EDIX HILL, CAMBRIDGESHIRE	575 – 650 cal AD	Duhig (1998)
DUNSTABLE, BEDFORDSHIRE	650 – 670 cal AD	Beavan and Mays (2013)
BURWELL, CAMBRIDGESHIRE	7th century AD	Møller-Christensen and Hughes (1962)
ECCLES, KENT	7th century AD	Manchester (1981)
TEAN, SCILLY ISLES	7th – 8th centuries AD	Brothwell (1961)
BRANDON, NORFOLK	7th – 8th centuries AD	Anderson (2014)
ST. ANDREWS, SCOTLAND	8th century AD	Lunt (2013)
HOXNE, SUFFOLK	885 – 1015 cal AD	Inskip et al. (2017)
ST. JOHN'S TIMBERHILL, NORWICH, NORFOLK	10th – 11th centuries AD	Anderson (1996; 1998); Watson and Lockwood (2009); Bayliss et al. (2009); *This study*
ST. CATHERINE'S, NORWICH, NORFOLK	10th – 11th centuries AD	Wells (1962)
SCHOOL STREET, IPSWICH, SUFFOLK	10th – 11th centuries AD	Mays (1989)
WHARRAM PERCY, YORKSHIRE	960–1100 cal AD	Taylor et al. (2006); Mays (2007)
WINCHESTER, HAMPSHIRE	10th – 12th centuries AD	Roffey and Tucker (2012); Schuenneman et al. (2013); Roffey et al. (2017)
RAUNDS FURNELL, NORTHAMPTONSHIRE	10th – 12th centuries AD	Kerudin et al. (2019)

(Cases were deemed possible if showing lesions consistent with leprosy; probable if showing lesions diagnostic of leprosy; and confirmed where aDNA has verified the presence of Mycobacterium leprae)

2.4.4 Summary of the bioarchaeology of leprosy from prehistory to the Early Medieval period

At present, the aforementioned evidence provides the current archaeological picture of leprosy in Britain and the rest of Europe, and Asia and Africa (prehistory to the 12th century AD). By viewing the historical and archaeological evidence of leprosy in tandem, it appears that concrete evidence of leprosy does not begin until the first millenium AD with the expansion of the Silk Road in the 1st century AD, which slightly changes previous narratives, particularly the Biblical connotations associated with the disease. Additionally, it is important to note that in instances with excavation records, individuals were almost always buried with normal burial rites for their respective cemetery populations, as noted by Roberts (2002; and more recently 2020: 280). This includes in monastic contexts, familial tombs, high-status cemetery contexts, and "normal" parish cemeteries. If, as the majority of historical sources cite, leprosy was a feared and stigmatised condition, then one would expect to find individuals with noticeable changes of the disease buried within "abnormal" or 'deviant' contexts. Because the postmortem treatment of the body in most contexts involved a closeness with ritual elements of bathing and shrouding (Gilchrist and Sloane 2005:78-80; Roffey and Tucker 2012), and ecclesiastical or community/familial burial, it stands to reason that individuals with leprosy were not as stigmatised as later texts suggest, but rather accepted and included as members of their community, both in life and death.

During the 12th century AD, evidence for leprosy increases substantially (Roberts 2002; Roberts 2020: see 190-280 for an overview). With an increase in leprosy prevalence, sociocultural reactions towards the disease also shift until the disease's rapid decline in the 14th – 15th centuries (Roberts 2013). Only after its resurgence with increased exploration, colonization, and migration (16th – 20th centuries AD) do attitudes towards leprosy shift again, most notably in a negative and prejudicial manner (Edmond 2006:51-60). It is imperative to evaluate these earlier contexts to understand past mindsets towards the disease and build a deeper understanding of the attitudes affecting the biological and social responses of leprosy in the past.

2.5 References

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CHAPTER 3. APPLICATIONS OF ISOTOPE ANALYSES IN PALAEOPATHOLOGY

3.1 INTRODUCTION

The isotopic ratios of tissues from human skeletal remains have generated an abundance of useful data on life course particulars over the past four decades. Using biogeochemical principles to study stable and radiogenic isotopes accumulated within these tissues, researchers have been able to reconstruct both biological and cultural specifics, including palaeodietary reconstruction and movements that underscore the individualised differences within and between people. One aspect of these investigations that is still in its nascence stage, however, is the relevance of these isotope systems in congruence with pathology, specifically the application of these methods and their interpretations in association with the development of pathological skeletal changes. This chapter will explore this potential through first discussing the basic fundamentals of isotope biochemistry; briefly reviewing the use of stable isotopes in archaeology, with particular focus on their application in palaeopathology; and finally, discussing future directions for these methods of analyses within this field.

3.2 FUNDAMENTALS OF ISOTOPE BIOGEOCHEMISTRY

An isotope can be defined as an atom of an element with the same number of protons in their nucleus, but a differing number of neutrons (Pollard et al. 2007; Pollard et al. 2017). This variation in the number of neutrons leads to differences in atomic weights, or masses, which is denoted by preceding the atomic element in superscript (e.g. ^{14}C) (*ibid*). Isotopes are either radioactive or stable in nature. Radioactive isotopes, also known as radioisotopes, are unstable isotopes that emit subatomic particles as they decay that can be measured at a specific rate known as a half-life (Brown and Brown 2011:80). The most widely used radioactive isotope application in archaeology is radiocarbon dating (^{14}C), which is used to date organic materials (Taylor 2014: 6226-7). It is important to note, however, that some isotopes may be radiogenic (e.g. ^{87}Sr) (Sealy et al. 1995; Schwarcz et al. 2010). A radiogenic isotope (daughter) is produced through the decay of a radioactive isotope (parent), and may either be radioactive in nature (radionuclides), or stable

(stable nuclides) (Sharp 2017:1-7; Pollard et al. 2007:233-235). One 'stable' radiogenic isotope commonly used in archaeology is strontium-87 (^{87}Sr), which is detailed later in this chapter (Lide 2002:1-15).

3.2.1 Isotopic Fractionation

Stable isotopes do not undergo a process of decay, and their ratios to one another within human tissues can provide a direct biogeochemical link to biocultural particulars of the past. Living organisms tend to acquire their isotopic compositions from their biogeochemical environment, including their foodwebs and water sources. Therefore, isotope ratios of human tissues can be compared to the isotopic compositions of known standard material to reveal dietary and geographic differences (Schwarcz et al. 2010: 337). However, these isotopic compositions are usually not a result of a direct transfer. Once consumed, stable isotopes can undergo a process called fractionation, which alters the proportions of isotopes within the consumer's tissues relative to the initial substrate, or origin (Brown and Brown 2011:81; Sharp 2017: 1-10-11). It is worth noting that heavier elements, such as strontium and lead, are an exception and are not usually subject to fractionation in biochemical processes due to their large masses (Pollard et al. 2007:233).

Isotope fractionation is a term widely used in stable isotope studies, but complex to comprehend without a background in biogeochemistry (Schoeller 1999). There are two primary types of isotope fractionation. The first type of fractionation involves the differential exchange of isotopes between physical phases whilst maintaining equilibrium. This is known as equilibrium fractionation and is mainly used to understand variations in palaeoclimates (Brown and Brown 2011:81). The other primary type of isotope fractionation is known as kinetic fractionation, and is the basis for understanding stable isotope variation within biological systems in the present and past (Brown and Brown 2011: 81; Katzenberg 2008:416).

Stable isotopes of elements exist in nature in unequal amounts (Table 3.1), and their masses can be classified as either 'light' or 'heavy' (Sharp 2017:1-9-1-10; Brown and Brown 2011: 80-81). The variance in an isotopes' mass helps to dictate their subsequent behaviour during chemical reactions; i.e. their different mass give them different kinetic

and bond properties, which then affects the way they behave (Schoeller 1999; Pollard et al. 2007: 232-233). The term for the physical phenomena that occurs during a chemical reaction resulting from the differences in isotope mass is known as 'isotope effects', and these effects are often greatest in lighter elements, such as carbon, nitrogen, and oxygen, due to the marked differences in the masses of their isotopes (see Table 3.1) (Katzenberg 2008: 415-416; Schoeller 1999). During a chemical reaction, these isotope effects can lead to kinetic fractionation of isotopes, which is a term used to describe the discrimination, or preference, for one isotope over the other during a reaction, thereby altering the isotopic proportions from the substrate, or origin, to the product (e.g. consumer) (Schoeller 1999; Sharp 2007:1-13; Pollard et al. 2007:233). The isotope discrimination that occurs during kinetic fractionation is unidirectional and usually favours the lighter isotopes, as the additional neutrons can create stronger bonds in heavier isotopes (*ibid).* The composite effects of kinetic fractionation on humans in the past, as well as present, is crucial to palaeodietary reconstruction. Since stable isotopes are not subject to decay, archaeologists can view isotopic variation in human tissues to better understand interactions with inherent and exogenous biological systems in the past (Katzenberg 2008: 416).

3.2.2 Assumptions of equifinality and 'stress'

Within a biological system, the number of physiological reactions via metabolic pathways is too great, and the data for the way that isotopes are distributed within living tissues is too limited to permit a fully comprehensive model for isotope fractionation (Schoeller 1999). However, an assumption exists that biological systems, like the human body, maintain a steady-state within an 'open system,' meaning that there is a general input and output that relates to the systems of their external environment (*ibid*). For example, models of kinetic fractionation would assume that humans maintain a regular diet-tissue space based on their habitual dietary intake; i.e. a person is a composite of what they eat (DeNiro and Epstein 1976, 1978; 1981; Schoeninger and Moore 1992; Reitsema and Holder 2018). This, unfortunately, introduces a problem of equifinality.

The principle of equifinality in stable isotope analyses highlights a person's isotope composition can be a result of a variety factors, especially as isotope compositions in

archaeological tissues can represent averages of long lengths of time and consumption (Schoeller 1999; Montgomery et al. 2007; Lee-Thorp 2008). This is a contentious issue that is vigorously debated, especially with regard to marine vs. terrestrial dietary intake (see Richards et al. 2003; Milner et al. 2004; Hedges 2004; Milner et al. 2006 for a great debate concerning potentials and limitations of interpreting dietary particulars). Another major issue with equifinality presents with nutritional and pathophysiological stress.

Isotope fractionation patterns can change when a biological system becomes unsteady, such as during periods of rapid growth (e.g. first year of life, growth spurts, etc.), or in the face of any extreme physiological 'stress', such as pregnancy, starvation, or illness (Schoeller 1999; Fuller et al. 2004; Waters-Rist and Katzenberg 2010). The body's response to nutritional and pathophysiological stress mediates the body's ability to fractionate and distribute certain stable isotopes, such as nitrogen, thus disturbing the assumed 'normal' diet-tissue space patterns (Reitsema 2013; Reitsema and Holder 2018). This effect will be detailed further in the nitrogen isotope section, but is one way in which we can view the impacts of acute and chronic pathologies through isotope systems.

3.2.3 Isotope Notation

As previously mentioned, stable isotopes of elements exist in nature in unequal amounts, with their masses classified as either 'light' or 'heavy' (Sharp 2017:1-9-1-10; Brown and Brown 2011: 80-81). In normal isotope nomenclature, isotope ratios are calculated as 'heavy/light' for comparative purposes (*ibid*). Lighter isotopes are generally more abundant than heavier isotopes (Table 3.1), which are usually present in trace amounts (*ibid*). Although the heavier isotopes usually exist in very small amounts, the ratio between the heavy-to-light isotopes is extremely small. Therefore, rather than be presented as minute, fractional abundances with long multidigit numbers, stable isotope ratios are expressed using the delta notation in units of per mil (parts per thousand) (Schoeller 1999; Pollard et al. 2017:352-353; Sharp 2017:2-2-3). The delta per mil notation calculates the relative deviation (δ) from a heavy-to-light isotope ratio to a reference standard (Table 3.1) of known isotopic composition in parts per thousand (‰),

effectively multiplying the difference into a workable value that does not provide a decimal place into the thousandth (or beyond) (*ibid*).

δ (‰) = ($R_{(sample)}$ /$R_{(standard)}$ - 1) x 1000; R = ratio of the heavy-to-light isotope

(Sharp 2017:2-3)

Strontium does not significantly modify or fractionate through the food chain, therefore strontium isotope ratios ($^{87}Sr/^{86}Sr$) are directly compared with the strontium isotope ratios of underlying geologies. For this reason, the delta per mil notation system is not necessary (Pollard et al. 2017: 370).

TABLE 3.1 - Stable isotopes of elements used in this thesis, their abundance in nature, and the reference standards of known isotopic composition they are measured relatively to (adapted from Sharp 2007:2-12 - 2-17; Katzenberg 2008:416)

ELEMENT	STABLE ISOTOPE MASS	ABUNDANCE IN NATURE	REFERENCE STANDARD
CARBON	12	99.985%	Vienna Peedee Belmnite (VPDB)
	13	1.11%	
NITROGEN	14	99.63%	Atmospheric Nitrogen (AIR)
	15	0.37%	
OXYGEN	16	99.759%	Standard Mean Ocean Water (SMOW)
	18	0.204%	
STRONTIUM	86	9.86%	Underlying geology
	87	7.02%	

*Strontium isotopes are compared as a ratio directly between tissue ratio and underlying geology, and not customarily used in δ notation.

3.2.4 TISSUES USED IN ARCHAEOLOGICAL ANALYSES OF STABLE ISOTOPES

A variety of archaeological human tissues can be used in stable isotope analyses. Early stable isotope analyses by DeNiro and Epstein (1976; 1978; 1981) first provided a firm link between various human tissues and dietary intake. Common human tissues that have been utilised since have included bone, teeth, hair, and fingernails (Vogel and van der Merwe 1977; Schoeninger and Moore 1992; Sealy et al. 1995; O'Connell et al. 2001). Although useful in modern studies, non-mineralised tissues such as hair and fingernails

are rarely recovered from archaeological human remains. Therefore, this section will focus on the human tissues that are used most often in stable isotope analyses: bone and teeth.

(I) Bone Apatite and Collagen

Bone is a highly specialised functional tissue. It provides support and protection to the soft tissues, helps facilitate movement, serves as a production centre for blood cells, provides storage for fats, and acts as a reservoir for essential elements such as calcium (White and Folkens 2005: 31). Bone is a composite of inorganic calcium phosphate crystals (70%) in crystalline form (hydroxyapatite), and organic, structural proteins (30%), namely, collagen (Katzenberg 2008:416). Earlier studies focussing on the isotopic composition of the inorganic structure of bone were hindered by the discovery that hydroxyapatite was subject to diagenetic alteration from the burial environment, and that carbonate values from bone were too wide in scope as they reflected an amalgamation of whole dietary intake (including carbohydrates, lipids, and protein) (Schoeninger and DeNiro 1982; Krueger and Sullivan 1984; Lee-Thorp et al. 1989). Bone collagen, however, was found to be more resistant to diagenetic change and allowed for more discriminatory analyses of dietary specifics including plant types and trophic levels (Lee-Thorp et al. 1989; Lee-Thorp 2008).

Bone collagen makes up approximately 85-90% of the organic portion of bone, and is largely composed of essential and non-essential amino acids (mainly glycine, alanine, proline, hydroxyproline) (Krueger and Sullivan 1984; Ambrose and Norr 1993: 30-32). Bone collagen is subject to degradation, but its unique, protective relationship with hydroxyapatite helps to preserve it over extremely long periods of time (Lee-Thorp 2008; Katzenberg 2008: 416). The timespan reflective in bone collagen varies due to normal metabolic turnover. Like other organic tissues, collagen is subject to lifelong remodelling (Libby et al. 1964; Hedges et al. 2007). This means that the isotopic composition reflected in the bone collagen represents long-term averages based on the element the collagen was extracted from and the age of the individual (Sealy et al. 1995; Hedges et al. 2007). For example, dense compact bone found in the appendicular skeleton has a slow metabolic turnover, and may represent a cumulative isotopic composition of up to 25

years in an adult (*ibid*). This is in not the case for highly vascularised bone with a thinner cortex and more trabecular bone, such as in parts of the axial skeleton, which has a much faster metabolic turnover rate that will reflect the dietary isotopic composition of more recent years (*ibid*). Not only does the isotopic composition of bone collagen vary with regard to its location in the body, but it will also differ based upon the age of the individual, with metabolic turnover occurring much more rapidly in younger individuals and then dramatically slowing after growth and development is fully achieved (Hedges et al. 2007). These factors must be taken into consideration when interpreting diet from the isotopic composition of bone collagen.

(II) DENTAL TISSUES

Isotopic compositions of different components in teeth are also useful in stable isotope analyses. Like bone, teeth are made up of inorganic and organic tissues, but unlike bone, teeth grow incrementally, which allows researchers to hone in on more definitive timelines with regard to isotope ratios of their constituent elements (Dean 2000: 119; Beaumont and Montgomery 2015).

Enamel constitutes the inorganic portion of a tooth and like bone is primarily comprised of calcium and phosphate in the form of a carbonated bioapatite and other substituting ions (~96%) (Fincham et al. 1999; Hillson 2005:155). This is of particular importance, because one of these substituting ions for calcium within enamel is strontium (Sr^{+}) (Hillson 2005:147). The predilection for strontium to substitute in the place of calcium during tooth development is what allows us to compare strontium isotope ratios within enamel to geological strontium isotope ratios to assess mobility histories (Ericson 1985; Bentley 2006). After crown growth and development ceases, mineralised tooth enamel undergoes relatively little change and is highly resistant to diagenetic alteration, making it the ideal tissue to view the isotope ratios of oxygen, strontium, and lead from an individual's childhood to adolescence (Hillson 2005: 158-159; Bentley 2006; Montgomery 2002; Montgomery 2010). Although enamel is highly resistant to most degradation, for analyses of carbon, issues similar to analysing carbonate from bone exist regarding burial alteration and dietary ambiguity; therefore dentine collagen is the ideal dental tissue for

stable isotope analyses of dietary carbon and nitrogen (Sealy et al. 1995; Hillson 2005:152).

Dentine is a composite dental tissue consisting of mineralised (72%) and organic matrices (20%) (Hillson 2005: 184-185). Unlike enamel, dentine is a living tissue with the majority of its organic component consisting of collagen, however it is not subject to the same lifelong metabolic turnover processes as bone collagen (*ibid*). This aids in eliminating an element of mortality bias inherent in bone collagen (Wood et al. 1992; King et al. 2018). Mortality bias, noted in a seminal paper by Wood and colleagues (1992), highlights that bioarchaeological analyses often view non-survivors and make assumptions about their health from their mortal remains, despite most fatal conditions not affecting the skeleton. This mortality bias becomes even more difficult to mitigate in bulk bone collagen sampling due to the long lengths of time reflected in the isotopic values of bone collagen. For example, if an individual dies in childhood, bulk bone sampling methods would account for an average of months or years of their life, incorporating any elevated nitrogen values due to metabolic or nutritional stress into their overall isotopic composition. This can give a subversive view of diet and facilitate false assumptions.

In contrast to bone, dentine is laid down incrementally at known rates from an early age until the apex of the root is completed (Hillson 2005:187-188; Dean and Scandrett 1995). In the permanent dentition, this normally first completes at around the age of 8.5 years for the first mandibular incisors, and continues non-sequentially through the third molars, which complete around the age of 23.5 years (AlQahtani et al. 2010). After completion of the primary dentine formation, a darker secondary dentine begins to slowly form within the walls and roof of the pulp chamber (Dean and Scandrett 1995; Hillson 2005: 187-188). Furthermore, tertiary dentine, also called irregular secondary dentine, may form in response to attrition, injury, or tooth decay (Hillson 2005:188). Since primary dentine is laid down incrementally at known rates and is not subject to metabolic turnover, the collagen extracted from increments can be sampled for stable isotope analysis of carbon and nitrogen and linked to an approximate age in an individual's life thereby reflecting the diet at the time of formation (Beaumont et al. 2013; Beaumont and Montgomery 2015). When viewing isotopic compositions from

increments of dentine collagen, it is important to avoid using teeth affected by dental pathology and notable attrition to avoid the influence of secondary dentine (*ibid*).

Recently, a discussion in the literature has emerged over which collagen (bone vs. dentine) is more reliable for reconstructing past diets (King et al. 2018; Beaumont et al. 2018; Craig-Atkins et al. 2018). In the past, the two collagen types have been used interchangeably, however Beaumont and colleagues (2013; 2018) highlight that they may not be compatible in the same way. King and colleagues (2018) note that although feeding trajectories in infants during periods of weaning may have idiosyncratic specifics, that the overall bulk bone collagen demonstrates the same weaning patterns and avoids exaggerating differences between the two. Beaumont and colleagues (2018), argue that although bulk bone collagen and bulk dentine collagen is interchangeable when looking at carbon isotope ratios, the minutiae that is present in nitrogen isotope ratios from dentine collagen allows for discrimination between diet and stress, and is therefore the better tissue when viewing isotopic compositions of non-adults.

3.3 Measurement by mass spectrometry

The principle of mass spectrometry is based on the idea that charged atoms of elements can be separated and measured based upon their mass. Mass spectrometers are analytical instruments that involve the counting of individual ions within a given material, which can then be calculated to view individual isotope ratios (Sharp 2017:2-21). Because mass spectrometry measures components at the atomic level, it is the most precise and sensitive measurement system possible, and therefore, the optimal method by which to measure the stable isotope ratios in human tissues (Pollard et al. 2007: 160-161). All mass spectrometers have three primary components: 1) a source: where the sample is ionised, accelerated, and then focussed into an ion beam; 2) the analyser: deflects the energy-focused ion beam through a magnetic or electrostatic field in turn creating several beams separated by mass, i.e. a mass spectrum; 3) the detector: measures the intensities of the ion beams as ratios to each other; e.g. heavy/light isotope ratios, which is then reported relative to the international reference standard being used (see Table 3.1 for reference standards) (Sharp 2017:2-21; Pollard et al. 2007:160-169; Figure 3.1).

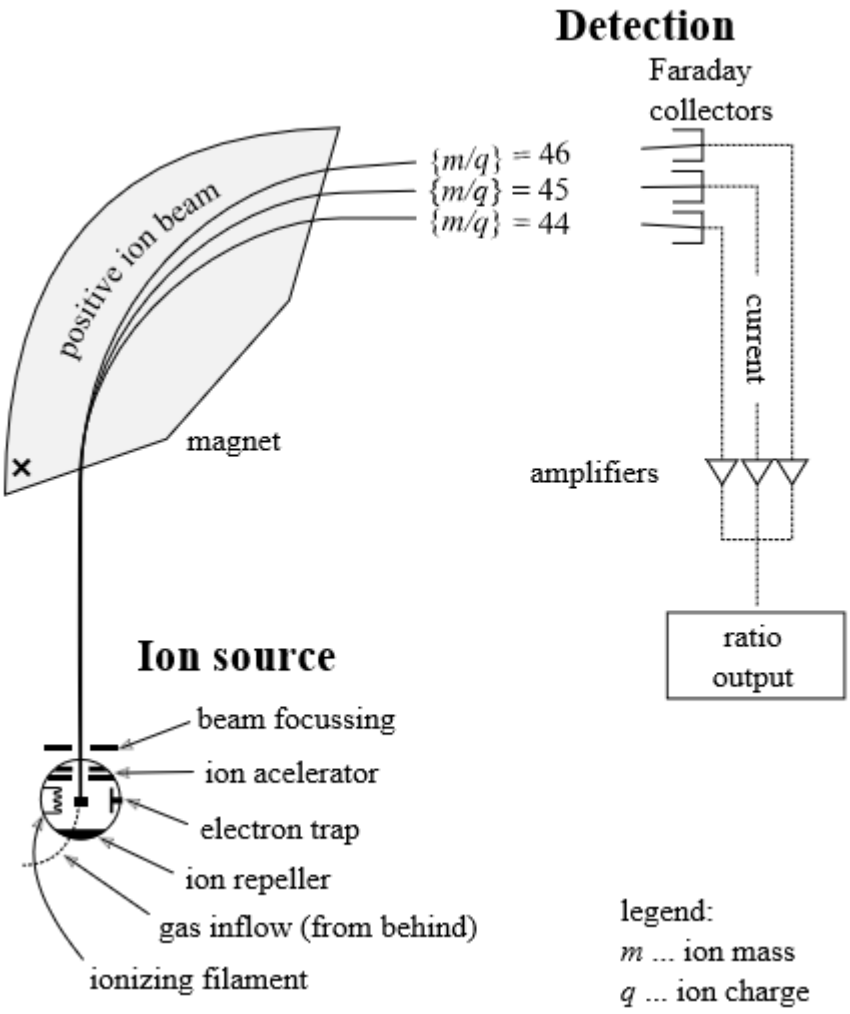

Figure 3.1 Schematic drawing of a typical isotope ratio mass spectrometer. Modified from United States Geological Survey (https://pubs.usgs.gov/of/2001/ofr01-257/images/figure1.gif; Public Domain).

Additionally, mass spectrometers for lighter isotopes generally have an inlet system for introducing gas into the source component without fractionation occurring (Sharp 2017:2-21). These mass spectrometers operate by first combusting the sample within the inlet system to produce N_2, CO_2, and H_2O gasses, and then separates the gasses before being transferred into the source component of the mass spectrometer (Katzenberg 2008:420).

Depending on the element, different types of mass spectrometers will be warranted. For light stable isotope analyses of organic materials, isotope ratio mass spectrometry (IRMS) with the continuous flow method of gas introduction is the most common (Pollard et al.

2007:169-170; Sharp 2017:2-26). For inorganic human tissues such as enamel, different instruments will be required depending on the element's mass. For structural carbonate carbon and oxygen isotope ratios in enamel, a duel inlet isotope ratio mass spectrometer dynamic inlet system is preferred (Chenery et al. 2012; Sharp 2017:2-25). This system helps to preclude non-ionised material into the mass spectrometer's source component, whilst providing a fresh, steady stream of sample and reference ionised gas (Sharp 2017:2-25 – 2-26). For heavier elements that cannot be readily converted into gas, such as strontium or lead, thermal ionisation mass spectrometry (TIMS) will be required (Pollard et al. 2007:173). This method involves placing a small amount of the sample onto a filament, loading it into the mass spectrometer via a sample turret and then electronically heating the sample (*ibid*). The heating of the sample allows the ions to be released and accelerated through the instrument (*ibid*). The types of mass spectrometers used in this study are detailed in Table 3.2.

TABLE 3.2 - Types of mass spectrometers used to analyse isotope ratios in this study

ISOTOPE	MASS SPECTROMETER TYPE
STRONTIUM	Thermal Ionisation Mass Spectrometer
OXYGEN	Dual Inlet Isotope Ratio Mass Spectrometer
CARBON	Continuous Flow Combustion Isotope Ratio Mass Spectrometer
NITROGEN	Continuous Flow Combustion Isotope Ratio Mass Spectrometer

3.4 ISOTOPE APPLICATIONS IN BIOARCHAEOLOGY AND PALAEOPATHOLOGY

The analysis of isotope ratios in human tissues is regularly utilised in archaeology to reveal cultural particulars of the past (Schwarcz and Schoeninger 1991; Schoeninger and Moore 1992; Sealy et al. 1995; Bentley 2006). This is based on the principle that stable isotope ratios in humans, plants, animals, and water differ in specific ways that can be accounted for and measured within biological systems. In order to make meaningful interpretations from the isotopic compositions of human tissues, a contextual baseline based on the values of local isotopic ecology must first be considered. This includes data from potential foodstuffs, local geology, water systems, and local foodwebs. For the past 40 years, archaeologists have primarily focused on interpreting the isotopic composition from bone and tooth collagen and bioapatite as a tracer of diet and mobility, however

advances in theory and methodology are highlighting the potential of stable isotopes in other aspects of archaeology and bioarchaeology (Lee-Thorp 2008). Studies demonstrating the application of isotope data in combination with individual pathophysiologies is now being incorporated to better understand how these systems are co-dependent, and can help elucidate past health (Richards and Montgomery 2012; Reitsema and Holder 2018). In order to understand how isotopic compositions can be meaningfully linked to pathology, a brief review of the theory and development of these analytical techniques in archaeology is warranted. The following sections are broken up by isotope systems and how they may be used in conjunction with each other to understand the nuances of 'stress' and disease in the past.

3.5 Carbon stable isotopes

This section details the role of carbon within stable isotope analyses, including its origins, subsequent metabolic pathways and processes, variations, and ways in which carbon can be interpreted with regard to past diet.

3.5.1 C_3 and C_4 biomes

The use of stable isotope analysis within an archaeological context initially focussed on studies concerned with the domestication of plants and dietary reconstruction (Vogel and van der Merwe 1977; van der Merwe and Vogel 1978; see Schwarcz and Schoeninger 1991 for reviews of early studies). This was initially based upon differences seen in the isotopic compositions of C_3 and C_4 plants, which fractionate carbon differently due to the photosynthetic pathway they employ (*ibid*). These photosynthetic pathways are based on how light is converted to chemical energy within the plants (Smith and Epstein 1971; O'Leary 1981; 1988). Most terrestrial plants in temperate areas employ a C_3, or Calvin-Benson, photosynthetic pathway that discriminates against the heavier isotope (^{13}C) (*ibid*). The resultant $\delta^{13}C$ values range from approximately -29‰ to -19‰, with an average of – 26.5‰ (Pollard et al. 2017: 354). It is important to note that there is a discrepancy in the reported ranges of $\delta^{13}C$ values for C_3 plants, with some sources giving much lower ranges, though there does appear to be some consistency with regard to a C_3 global average $\delta^{13}C$ value of approximately -26‰ (Pollard et al. 2017:354; Smith and

Epstein 1971; O'Leary 1988; Lee-Thorp 2008; Table 3.3). Important C_3 plants include cereals, wheat, barley, oats, rice, potatoes, yams, and most other vegetables (Sealy et al. 1995; Lee-Thorp 2008).

TABLE 3.3 - Ranges in $\delta^{13}C$ values for C_3 plants reported in literature

$\delta^{13}C$ RANGE FOR C_3 PLANTS	**REPORTED $\delta^{13}C$ GLOBAL AVERAGE**	**REFERENCE**
-29‰ TO -19‰	-26‰	Pollard et al. 2017: 354
-29‰ TO -25‰	-27‰	O'Leary 1988
-34‰ TO -24‰	-26‰	Smith and Epstein 1971
-36‰ TO -24‰	- 26.5‰	Lee-Thorp 2008

In contrast, a small amount of plants employ a C_4, or Hatch-Slack, photosynthetic pathway that leads to enrichment of the heavier isotope (^{13}C) (Smith and Epstein 1971; O'Leary 1981; 1988). C_4 plants can only thrive in a limited array of environments, including tropical or arid climates, therefore the range of $\delta^{13}C$ values for C_4 plants is much more restricted (Farquhar et al. 1989; Lee-Thorp 2008). Important C_4 plants include maize, millets, sorghum, sugar cane; and result in $\delta^{13}C$ values ranging from -16‰ to -12‰ (Sealy et al. 1995; Pollard et al. 2017:354). Because the fractionated ranges between $\delta^{13}C$ values of C_3 and C_4 plants do not generally overlap, discernment between the two is possible (Farquhar et al. 1989; Katzenberg 2008:423; Figure 3.2). These dissimilar poles of the $\delta^{13}C$ spectrum have formed the basis of stable isotope analyses of dietary carbon in archaeology.

(I) EUROPEAN PLANTS

European domesticates follow a C_3 photosynthetic pathway (Zohary and Hopf 2000: 1-9). Murray and Schoeninger (1988) presented the first archaeological evidence from archaeological human remains to suggest that C_4 exploitation and consumption was occurring in the Slovenian Iron Age (450-300 BC). Other studies noted similar phenomena both during a concurrent time period in Bohemia, as well as previously in Bronze Age in Italy (16^{th} – 12^{th} centuries BC) (Le Huray and Schutkowski 2005; Tafuri et al. 2009). Additional archaeological evidence of carbonised plant material indicates that millet from Asia was present in the Czech Republic and Caspian Basin from the 4^{th} millennium BC (Zohary and Hopf 2000: 7). Strangely, other C_4 plants, such as sorghum from Africa, were

exported to Asia in the Bronze Age (2nd millennium BC) and cultivated in Nubia from the 1st century AD onwards, but seemingly was not consumed in Europe (Zohary and Hopf 2000: 8). Despite the presence of millet in Europe from an early time period, through a comprehensive study, Lightfoot and colleagues (2013) demonstrated that millet was not significantly exploited after the 1st millenium BC. They also concluded that even for millet-consumers, it was not considered a 'staple' and therefore below the level of substantial isotopic detectability, except in rare circumstances (i.e. high socioeconomic status) or in Asian immigrants (Lightfoot et al. 2013). This interpretation may explain the scarcity of evidence with regard to the influence of C_4 plants in the archaeological record of Europe until the Medieval Period (O'Connell and Hull 2011; Lightfoot et al. 2012; Inskip et al. 2019).

Some caution is necessary in the interpretation dietary intake based on $\delta^{13}C$ values due to past environments and other anthropogenic factors. In heavily forested areas, plants can display a 'canopy effect', where decreased light availability and atmospheric mixing of CO_2 from decomposition of ground waste and soil results in plants having lower $\delta^{13}C$ values; up to 5‰ (van der Merwe and Medina 1991; Tieszen 1991; Ambrose 1993; van Klinken et al. 2000). Other spatial and climatic shifts due to latitude, temperature, humidity, light intensity, water availability, and soil nutrition will all affect the $\delta^{13}C$ values of plant material (Farquhar et al. 1989; Tieszen 1991; van Klinken et al. 2000). This highlights the importance of using comparative data and correcting for these changes when making interpretations regarding dietary subsistence in the past. Another consideration in the interpretation of dietary carbon is the possibility of succulent consumption. This is due to succulents utilising a different photosynthetic pathway that yields $\delta^{13}C$ values similar to C_4 plants, however this is primarily an interpretive consideration in the New World (Farquhar et al. 1989; van Klinken et al. 2000). The 'fossil fuel effect' is a term coined to describe the depletion of atmospheric CO_2 caused by the burning of fossil fuels from the 18th century AD onwards (van Klinken et al. 2000). This anthropogenic change has resulted in a depletion of up to 1.5‰ in atmospheric CO_2, which becomes an important factor to account for if using the $\delta^{13}C$ values of modern plants and animals to interpret pre-industrial human values (*ibid*). To understand how

these different processes reflect in human tissues, it is necessary to understand how dietary carbon is incorporated into collagen.

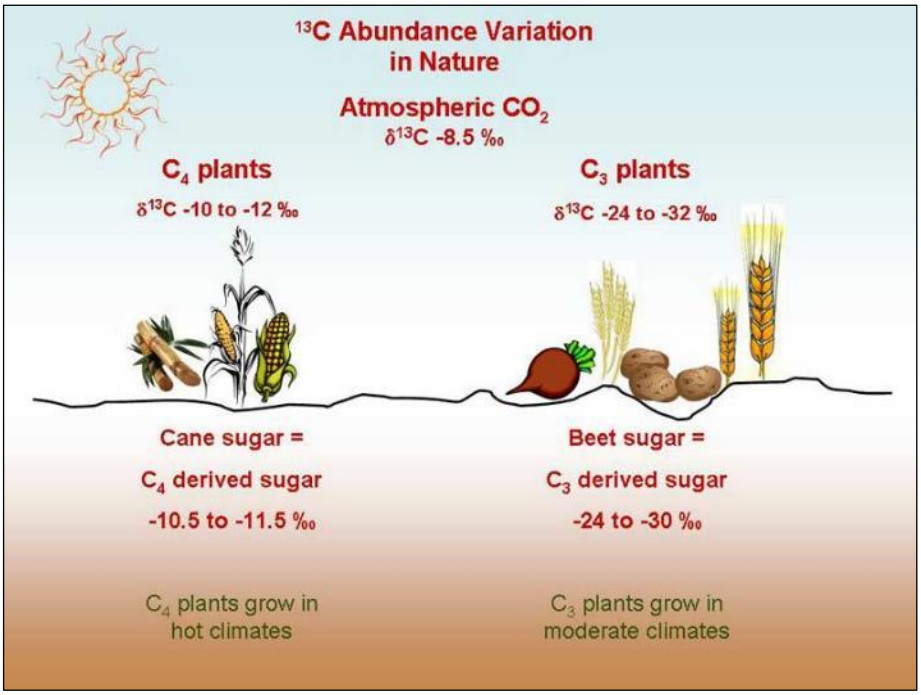

Figure 3.2 Average $\delta^{13}C$ values of modern C_3 and C_4 plants (https://www.hutton.ac.uk/research/groups/environmental-and-biochemical-sciences/isotope-applications). Open Access.

3.5.2 Dietary carbon and collagen

DeNiro and Epstein (1976; 1981) first established the link between dietary intake and the isotopic composition of human bone collagen, but noted an offset, or a diet-tissue space partition. Diet-tissue spaces can be described as the difference between the isotopic composition of the source material and the biological product via a unilineal isotopic enrichment of tissues (Tieszen 1991; Lee-Thorp 2008). These isotopic compositions differ according to their chemical properties and how they are synthesised biologically within the body (Lee-Thorp 2008). Several studies measured the isotopic differences (expressed as Δ) between diet and various human tissues and found that the $\delta^{13}C$ value of bone collagen is usually +5‰ greater than the $\delta^{13}C$ value of the total diet (van der Merwe and Vogel 1978; Krueger and Sullivan 1984; Ambrose and Norr 1993). Unfortunately, the method by which carbon is synthesised into consumer tissues is still not fully understood. At present, the debate lies between whether carbon atoms from the consumption of

different dietary macronutrients (proteins, carbohydrates, and lipids) are proportionally 'scrambled' throughout the tissues, or directly routed to corresponding consumer tissues (e.g. dietary protein > collagen) (DeNiro and Epstein 1978; Krueger and Sullivan 1984; Chisholm 1989; Schwarcz 1991; Ambrose 1993). If carbon atoms from dietary macronutrients are directly routed, then it is likely that the isotopic composition of collagen will mainly reflect the just protein intake. Ambrose and Norr (1993) and Tieszen and Fagre (1993) confirmed with animal modelling studies that dietary protein was preferentially routed towards collagen, however variations and caveats exist. Dietary intake and metabolic processes can mitigate both the carbon offset and the carbon source of collagen (Ambrose 1993; Ambrose and Norr 1993; Ambrose et al. 1997; Schwarcz 2002). Approximately 80% of the amino acids in collagen are non-essential, meaning they can be derived from sources other than diet (Ambrose et al. 1997; Schwarcz 2002). If dietary protein is scarce, or a consumer's dietary protein intake is low, the non-essential amino acids could contribute to the overall carbon isotope composition of collagen from a broader carbon pool: e.g. other macronutrients or metabolic byproducts (Schwarcz 2002). Despite the complex dynamics by which carbon is metabolically synthesised, the 5‰ offset can still be used as a general guide when interpreting $\delta^{13}C$ values of collagen.

3.5.3 Carbon in aquatic and terrestrial food webs

Perhaps the greatest consideration for the interpretations of $\delta^{13}C$ values to reconstruct past food webs lies within the differences between marine and terrestrial diets. The application of dietary carbon to determine the exploitation of aquatic resources was first explored by Tauber (1981), who demonstrated a temporal shift in Danish skeletons from high $\delta^{13}C$ values in the Mesolithic, to lower $\delta^{13}C$ values in the Neolithic signifying a change in marine exploitation to agricultural subsistence.

Chilsholm and colleagues (1982) further detailed the discrepancy in $\delta^{13}C$ values seen in terrestrial vs. marine diets, which remains of significant interest in palaeodietary reconstructions. This is due to the high variability of carbon isotope ratios seen in organisms that dwell in marine, brackish, and freshwater ecosystems, which average as an intermediate with potential overlap between the C_3 and C_4 $\delta^{13}C$ value ranges (Tauber

1981; Chisholm et al. 1982; Schoeninger and DeNiro 1984; Fuller et al. 2012). Aquatic plants, with few exceptions, follow a C_3 photosynthetic pathway, however, their carbon source is derived from dissolved bicarbonate rather than atmospheric CO_2, which shows a higher $\delta^{13}C$ value of approximately 0‰ in comparison to the atmospheric $\delta^{13}C$ value (-8‰) (Chisholm et al. 1982; Farquhar et al. 1989). Because of this ^{13}C enrichment, marine plants tend to yield higher $\delta^{13}C$ values. The $\delta^{13}C$ values measured from aquatic plants spans from approximately -39‰ to -11‰, with a global marine average of approximately -19‰ (Farquhar et al. 1989:516; Ambrose 1993:94). In the reconstruction of human diet, animal consumers who subsist on marine plants are of more interest, as humans are more likely to consume aquatic animals, rather than solely plants (Chisholm et al. 1982; Schoeninger and DeNiro 1984; Ambrose 1993). The average $\delta^{13}C$ value of marine animals is -15.6 +/- 1.6‰ and can be distinguished from C_4-based foods in areas devoid of tropical plants (*ibid*). Consumers of freshwater or brackish species can make discrimination between terrestrial and aquatic food webs far more difficult. Animals that exploit freshwater environments possess $\delta^{13}C$ values that overlap extensively with C_3-based foods, rendering them indistinguishable when looking at dietary carbon alone (Schoeninger and DeNiro 1984; Dufour et al. 1999; Fuller et al. 2012; Robson et al. 2016). As an added complication, marine and migratory birds, catadromous species (freshwater dwelling species that migrate to the sea for reproduction), and anadromous species (marine dwelling that migrate to freshwater for reproduction) largely present intermediate $\delta^{13}C$ values between C_3 and C_4 ranges (*ibid*). If aquatic foods are suspected to be a contributor of diet, measurements of complementary stable isotope analyses (e.g. nitrogen and sulphur) are warranted in order to help distinguish between the consumption of terrestrial and aquatic foods. Although worthy of merit, isotope analyses of sulphur are outside the context of this research, however nitrogen will now be discussed in detail.

3.6 NITROGEN STABLE ISOTOPES

This section discusses the stable isotopes of nitrogen used for palaeodietary reconstruction including its source, trophic-level implications, how nitrogen is biologically synthesised, and other interpretive considerations beyond diet.

3.6.1 NITROGEN UPTAKE AND VARIABILITY

Like carbon, nitrogen has only two stable isotopes (^{15}N & ^{14}N) and stable nitrogen isotope ratios ($^{15}N/^{14}N$) are expressed as a value ($\delta^{15}N$) in comparison to an international standard, i.e. Ambient Inhalable Reservoir (AIR) (Schwarcz and Schoeninger 1991; Sharp 2017: 2-16). The $\delta^{15}N$ value of atmospheric nitrogen is 0‰, and does not differ globally (Mariotti 1983). The nitrogen isotope ratios for plants are solely dependent on how they acquire nitrogen (Schwarcz and Schoeninger 1991; van Klinken et al. 2000). Plants obtain their nitrogen composition either through atmospheric N_2 from symbiotic soil bacterial (terrestrial) or cyanobacterial (aquatic) nitrogen fixation (fixers), or directly through nitrates produced from decomposition of organic matter in terrestrial or aquatic reservoirs by bacteria and fungi (non-fixers) (*ibid*) (Figure 3.3). Nitrates (NO_3) are forms of nitrogen created through bacteria that release ammonium (NH_4) as they breakdown organic matter. The ammonium is then further transformed via nitrification into soil nitrate; an essential for the subsistence and growth of plants (van Klinken et al. 2000; Gruber and Galloway 2008; Canfield et al. 2010; Figure 3.3). These variations in the acquisition of nitrogen are important to distinguish between plant type (leguminous vs. non-leguminous plants), and tropical reef or terrestrial food webs.

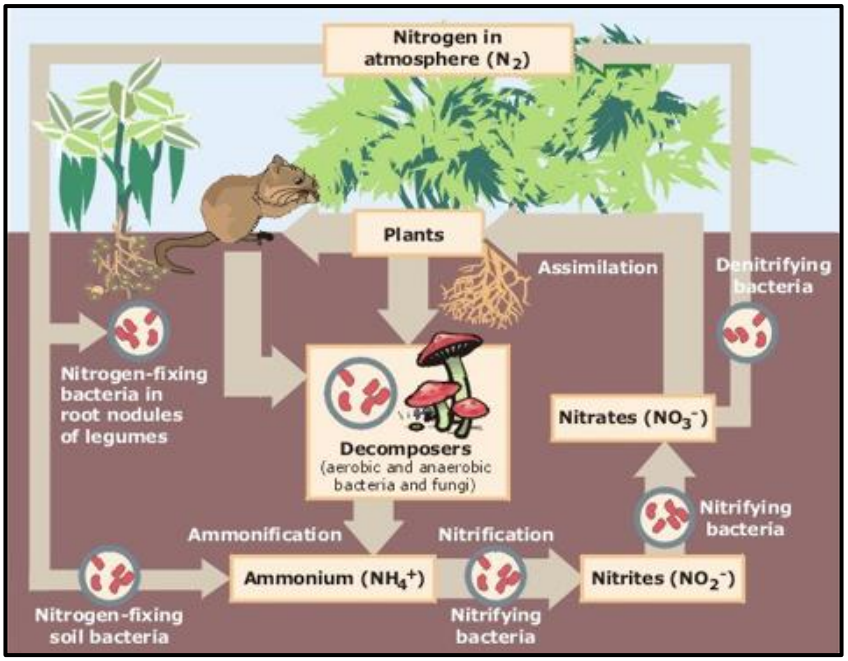

Figure 3.3 Diagram of the terrestrial nitrogen cycle (U.S. Environmental Protection Agency. https://commons.wikimedia.org/wiki/File:Nitrogen_Cycle.jpg. Public Domain.)

Most plants consumed by humans acquire nitrogen through the soil nitrification and denitrification processes (van Klinken et al. 2000). The fractionation processes that occur during this method of nitrogen acquisition are highly variable depending on soil content and climate, but leads to increases in their $\delta^{15}N$ values by up to 10‰ relative to the atmospheric $\delta^{15}N$ values (0‰) (van Klinken et al. 2000; Lee-Thorp 2008). Leguminous plants, in contrast, are considered 'nitrogen fixers', which allows them to acquire atmospheric N_2 directly from soil bacteria (van Klinken et al. 2000: 43-45). Because the means of nitrogen acquisition are more direct, the fractionation processes are minimal, leading to $\delta^{15}N$ values similar to those of atmospheric $\delta^{15}N$ values (0‰) (*ibid*). Leguminous plants include pulses, peas, beans, chickpeas, clover, alfalfa, lentils, etc, and constituted a significant portion of diet in the past (DeNiro 1987).

Like carbon isotope compositions, environmental and anthropogenic factors may have a significant effect on the $\delta^{15}N$ values of soil, plants, and primary consumers. Variations in climate including aridity, high precipitation, latitude, and environmental salinity will affect the enrichment of ^{15}N (Ambrose 1993; van Klinken et al. 2000; Lee-Thorp 2008).

For instance, forested areas and soils can show low $\delta^{15}N$ values of -6‰, whereas the salinity of coastal areas can increase $\delta^{15}N$ values to 10‰ (van Klinken et al. 2000:45-46; Ambrose 1993). Climate-wise, cooler, temperate climates generally yield lower $\delta^{15}N$ values, whereas arid environments yield much higher $\delta^{15}N$ values, potentially due to water stress; i.e. animals that live in drier areas ingest less water and therefore excrete more ^{14}N in their urea (Schoeninger and DeNiro 1984; Heaton 1987; Ambrose 1993). Further, many wild and domesticated herbivores show a wide range of $\delta^{15}N$ values, partially due to the seasonal availability of primary producers, which can be highly dependent on climate changes (Darimont and Reimchen 2002; Makarewicz 2014). The extent to which the anthropogenic domestication and exploitation of plants and animals has on the variability of $\delta^{15}N$ within a food web is still not fully explored, and may produce even wider variations amongst domesticate taxa (Bogaard et al. 2007; Hedges and Reynard 2007; Fraser et al. 2011; Makarewicz 2014). For example, anthropogenic factors on food production, such as manuring shows an increase in $\delta^{15}N$ values through the food chain. Because manure is teeming with bacteria and other organic matter, its use in crop cultivation will cause a significant enrichment of ^{15}N in the soil (Bogaard et al. 2007; Hedges and Reynard 2007; Fraser et al. 2011). Depending on the intensity of fertilisation, the use of animal manure in agricultural soil can cause elevations in the $\delta^{15}N$ values of grains and cereal stalks of between +3 to +8‰, and this increase will persists through trophic levels (*ibid*). The effects of manuring may be dependent on both climate and anthropogenic factors as well. DeNiro and Hastorf found the highest $\delta^{15}N$ values (~ 35‰) in archaeological plants from the arid, coastal environments of Peru, where a mixture between high soil salinity and human/animal faeces runoff created an unusually high ^{15}N enrichment (DeNiro and Hastorf 1985). Similar ecological niches have revealed significant diachronic shifts in $\delta^{15}N$ values from the Neolithic to the Bronze Age, implicating a deliberate change in herding practices from a terrestrial ecology to salt-marshes (Britton et al. 2008). These environmental and anthropogenic factors must be considered when attempting to distinguish a culture's subsistence patterns; particularly their reliance on terrestrial vs. aquatic food webs.

3.6.2 The consideration of nitrogen in aquatic and terrestrial food webs

Marine plants can be distinguished from terrestrial plants using nitrogen isotopes due to a greater amount of nitrification and denitrification that occurs in aquatic environments (Schoeninger and DeNiro 1984; Ambrose 1993). The increased fractionation processes result in the $\delta^{15}N$ values of primary producers approximately +4‰ greater than their terrestrial counterparts (Schoeninger and DeNiro 1984; Schwarcz and Schoeninger 1991; van Klinken et al. 2000). This enrichment in 15N continues along each trophic level in the marine foodweb, which is more extensive than a terrestrial environment (Schoeninger and DeNiro 1984; Schwarcz and Schoeninger 1991). Because of the complex range of trophic levels in marine ecosystems, cultures who rely heavily on marine diets for subsistence will yield much higher $\delta^{15}N$ values than those who rely on terrestrial fauna (*ibid*). This tends to hold true except in environments where nitrogen fixers predominate. In tropical reef ecosystems, for example, blue-green algae acquire nitrogen through bacterial and algal fixation, which will yield $\delta^{15}N$ values similar to primary producers in terrestrial environments and therefore may obscure some marine influence (Schoeninger and DeNiro 1984; Ambrose 1993; Ambrose et al. 1997).

Nitrogen isotopes can also help to distinguish freshwater fauna. Like in marine environments, $\delta^{15}N$ values of freshwater fish are higher than in terrestrial animals, however the carbon pool available to freshwater fauna is usually depleted in ^{13}C, therefore the $\delta^{13}C$ values will usually be lower than in marine fauna (Katzenberg and Weber 1999; Dufour et al. 1999; van Klinken et al. 2000; Fuller et al. 2012). Although these variations in nitrogen and carbon stable isotope ratios tend to hold true in most circumstances, Katzenberg and Weber (1999) discovered that for some freshwater fish that resided in the littoral zones (shallow waters) of Lake Baikal, the $\delta^{13}C$ values were markedly higher than anticipated. Furthermore, very few studies measuring independent freshwater fish consumption exist highlighting the need for complementary analysis of possible dietary components when trying to reconstruct human diets of the past (Ambrose 1993; Hedges and Reynard 2007; Robson et al. 2016). Lastly, although elevated $\delta^{15}N$ values and reduced $\delta^{13}C$ values may be interpreted as freshwater fish consumption, the dynamics of nitrogen fractionation and metabolism, especially when undergoing certain types of physiological stresses, may yield similar results (see 3.6.4; Schoeller 1999;

Hedges and Reynard 2007; Lee-Thorp 2008). For these reasons, the aggregate pathological-status of the skeletal remains must also be considered and addressed into archaeological interpretations.

3.6.3 Trophic-level effects in dietary carbon and nitrogen

Trophic level is a biological term used to refer to an organism's position an ecological pyramid, or a proverbial "food chain" (Lindeman 1942; Boecklen et al. 2011). A species' trophic level varies based upon their food acquisition. The first trophic level consists of primary producers which constitute almost all terrestrial plants and aquatic phototrophic plants, as they produce their own food (*ibid*). The following trophic levels in a food chain will consist of (level 2) primary consumers, (level 3) secondary consumers, (level 4) tertiary consumers, and (level 5) quaternary consumers or apex predators (Lindeman 1942; Pimm and Lawton 1978). In most ecosystems, the majority of organisms will rely on multiple food types and chains for sustenance. The intersections of these food chain networks are called foodwebs, and are specific to an organism's ecological niche (Pimm and Lawton 1978). Reconstructing trophic levels and foodwebs in humans can be extremely complex and requires contextual ancillary data. For example, Bonhommeau and colleagues (2013) estimate the average trophic level for modern humans to be similar to animals with a varied scavenging diet such as pigs (trophic level 2.21), however studies of Inuit populations that subsist on tertiary consumers like seals, are considered to be apex predators (Hobson and Welch 1992; Schwarcz and Schoeninger 1991; Hedges and Reynard 2007). It should be noted that decomposers and other saprophytic organisms are considered the end level of the food chain as they break down all organic material (Lindeman 1942). The transfer of biomass step-wise from lower trophic levels through to higher ones is known as the 'trophic level effect' and is observable in carbon and nitrogen isotope ratios (Schwarcz and Schoeninger 1991; Boecklen et al. 2011).

(I) Trophic level effects of dietary carbon

Trophic level effects are mainly a consideration of nitrogen isotope fractionation, however carbon must also be addressed. Despite not knowing the exact mechanism by which dietary carbon is synthesised into collagen, an observable shift in $\delta^{13}C$ values exists

between herbivores/omnivores and carnivores (van Klinken et al. 2000; Lee-Thorp 2008). Small trophic-level effects from 0.5 to +2‰ have been noted, though the increase in $\delta^{13}C$ from primary producer to apex consumer is not uniformly distributed between tissues, and is of little value in determining precise trophic position (Schoeninger and DeNiro 1984; van Klinken et al. 2000; Lee-Thorp 2008). For these reasons, van Klinken and colleagues (2000: 47) advise against using the term 'trophic level effects' when referencing $\delta^{13}C$ values of plant consumers.

(II) TROPHIC LEVEL EFFECTS OF NITROGEN

Unlike carbon, nitrogen isotope ratios in biological tissues are dependent on an organism's nitrogen source and subsequent metabolic effects (Ambrose 1993; Lee-Thorp 2008). A unilineal, stepwise enrichment of approximately 3‰ has been widely observed at each level of the food chain commencing with primary producers (plants and phytoplankton), however modern studies estimate that depending on the tissue type (e.g. collagen, fingernails, hair), the trophic level shifts can range from +2 to 6‰ (DeNiro and Epstein 1981; Minagawa and Wada 1984; Schoeninger and DeNiro 1984; van Klinken et al. 2000; O'Connell et al. 2012). Minagawa and Wada (1984) first demonstrated trophic-level enrichment of ^{15}N in marine environments, which have typically high $\delta^{15}N$ values due to protracted marine food chains. Schoeninger and DeNiro (1984) extended this evidence by comparing trophic-level shifts in a variety of terrestrial and marine foods, and how they relate to human diet. It is important to note that although nitrogen isotope ratios can give insight to protein consumption, the type of protein (e.g. meat vs. dairy) cannot be readily discriminated (Schoeller 1999; van Klinken et al. 2000). In general, however, humans that consume omnivorous terrestrial diets, will yield a $\delta^{15}N$ value range of between 6 to 10‰, whereas humans that subsist on primarily marine diets will yield a higher $\delta^{15}N$ value dependent on the source protein (Schoeninger and DeNiro 1984; Minagawa and Wada 1984; Schoeninger and Moore 1992). The change in nitrogen isotope ratios from dietary source to tissue is measured as a calculation of diet-tissue space (expressed as $\Delta_{Diet\text{-}Tissue}$), and whilst the principles of trophic-level enrichment seem relatively straightforward, other biochemical kinetic fractionation effects that mitigate nitrogen metabolism must be considered (van Klinken et al. 2000).

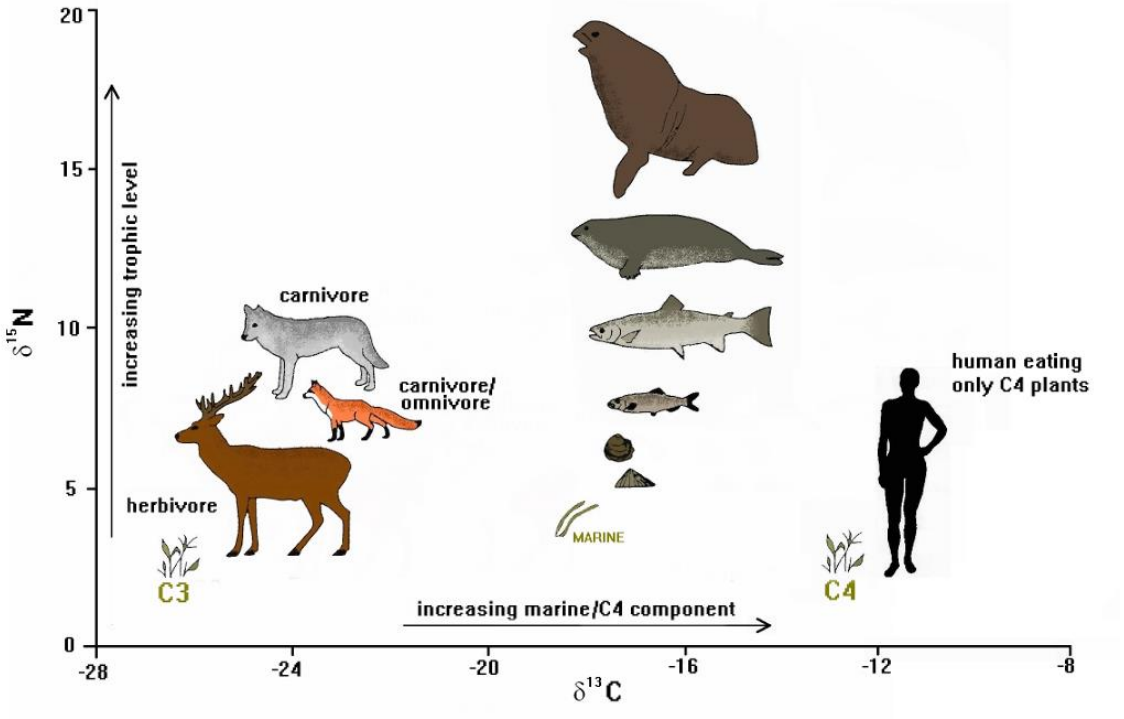

Figure 3.4 – Approximations of $\delta^{15}N$ and $\delta^{13}C$ values of various taxa based on enrichment and trophic level effects. Adapted from Schulting (1998) (http://chrono.qub.ac.uk/Research/IRMS/). Open Access.

3.6.4 NITROGEN METABOLISM

Macronutrients such as carbohydrates and lipids do not contain nitrogen, therefore the nitrogen isotope ratios of consumer tissues are mainly a reflection of dietary protein and the amino acids from which the protein is synthesized, with the addition of the trophic level effects described above (Allison 1957; Ambrose 1993; Ambrose et al. 1997; Schoeller 1999). Although a small amount of nitrogen (~2%) may derive from elsewhere in the body (e.g. urea, nucleic acids, ammonia), approximately 98% of a human's nitrogen isotope composition lies within its proteins (Allison 1957; Schoeller 1999). Human breast milk is an exception to this and will be discussed in the next section.

Protein is primarily synthesized from 20 essential and non-essential amino acids, however the functions of these amino acids during protein synthesis can be altered due to age, fasting, and health-status (Allison 1957; Beisel 1975; Young and el-Khoury 1995; Hatch 2012; Bechard et al. 2012). This is due to disruptions in the maintenance of whole-body mixed proteins and becomes an important factor when trying to understand the

implications and interpretations of dietary intake and disease, especially in younger groups (*ibid*). In order to understand these ramifications, an understanding of protein synthesis and nitrogen excretion is warranted.

After ingestion, dietary protein is hydrolysed (broken down with water) into amino acids and polypeptides (amino acid chains that form part of a protein molecule), which are then absorbed and routed for protein synthesis (anabolism). Any excess (~25%) is routed to the liver and other similar tissues (transamination) for metabolic use and breakdown (catabolism) (Allison 1957; Linder 1985; Macko et al. 1986). Within the liver, the excess amino acids are broken down (deamination) into carbohydrates for energy or other bodily functions, and ammonia (NH_3), which is further converted to urea and primarily excreted through urine via the kidneys (*ibid*) (Figure 3.5). When the body is in equilibrium, there is a metabolic balance of anabolism and catabolism in order to ensure tissue maintenance. Because the majority of nitrogen in the body is present in protein, these metabolic processes have a profound influence over a person's overall $\delta^{15}N$ average value.

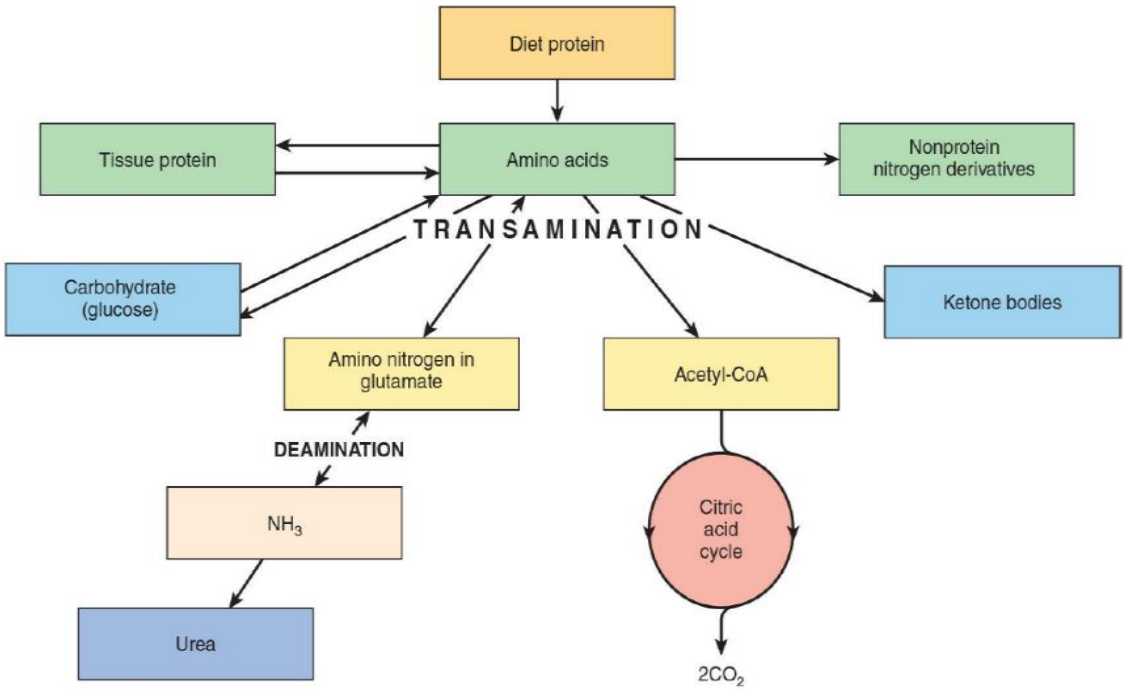

Figure 3.5 Diagram of amino acid metabolism, major metabolic pathways, and end products (Bender and Mayes 2018:343).

Unfortunately, a discrepancy exists regarding the means by which human and animal tissues are enriched in ^{15}N during the metabolic process, and remains relatively ambiguous (Hedges and Reynard 2007; Hatch 2012). When a human or animal expels nitrogen in the form of urea (the primary byproduct of protein metabolism), it is assumed that a preferential excretion of light nitrogen (^{14}N) occurs, leaving the remaining tissues to yield higher $\delta^{15}N$ values (Ambrose 1991; 1993). However, considerable variation exists in $\delta^{15}N$ values within and between species in trophic level systems, which cannot be explained by the normal nitrogen metabolism model (Ambrose 1991; Sponheimer et al. 2003a; 2003b; Robbins et al. 2005; Hedges and Reynard 2007). This has led to two schools of thought: 1) The greater the protein consumption, the higher the ^{15}N enrichment; and 2) The greater the protein consumption, the lower the ^{15}N enrichment (Sponheimer et al. 2003a; 2003b; Robbins et al. 2005). Sponheimer and colleagues (2003 a; 2003b) conducted controlled feeding studies on herbivores (llamas, alpaca, cattle, goats, and horses) offering high and low-protein diets to measure nitrogen fractionation, excretion, and diet-tissue spacing. Their results showed the animals on the lower protein diets excreted more ^{15}N enriched faeces in comparison to their high-protein diet consumers; high protein consumers excreted the majority of their nitrogen through ^{15}N depleted urine, yielding higher $\delta^{15}N$ values; and that physiological disruption via growth, nutritional stress, or disease status could alter $\delta^{15}N$ values (Sponheimer et al. 2003a; 2003b). In their controlled feeding study of 21 species of mammals and birds, Robbins and colleagues (2005) stressed that the variation they observed in $\delta^{15}N$ values between trophic level groups reflected the quality of the dietary protein, and that the actual quantity of protein intake accounted for within-group effects; i.e. nitrogen excretion increases when the protein intake exceeds a consumer's inherent biological requirement. Ultimately, the complexity of a foodweb, coupled with inherent biological variability and agency, leads to an ambiguity in the modelling of nitrogen enrichment in tissues. This underscores the need for employing flexible models of interpretation that are based on local dietary isotope baselines. Moreover, underlying human biovariability is also highly affected by factors beyond dietary consumption, which is now discussed.

3.7 BEYOND THE FOOD CHAIN

As previously discussed, factors like climate, environment and anthropogenic intervention can cause significant variations in $\delta^{15}N$ values at the first trophic level. To compound matters, disruptions and alterations due to certain biological stressors will lead to further metabolic changes of both $\delta^{13}C$ and $\delta^{15}N$ values.

3.7.1 Breastfeeding and weaning processes

Breastfeeding and weaning studies account for perhaps the greatest amount of investigations using temporal variations of $\delta^{15}N$ values in humans. Palaeoepidemiological studies regarding fertility rates were an early focus in examining the shift from hunter-gatherer to agricultural societies, and stable isotope analyses were used as a proxy for birth-spacing (Buikstra et al. 1986; Stuart-MacAdam 1995; Tsutaya and Yoneda 2015). Hunter-gatherer populations tend to breastfeed for longer durations, and therefore have lower fertility rates due to lactational amenorrhoea (Stuart-MacAdam 1995; Chowdhury et al. 2015; Victora et al. 2016). Fogel and colleagues (1989; 1997) first demonstrated through fingernail and bulk bone collagen analyses of ^{15}N that modern and archaeological infants have higher $\delta^{15}N$ values (on average +2.4‰) than their mother during breastfeeding, which subsequently decline through the weaning process until parity with the maternal values is reached. This phenomenon was later replicated in studies by Fuller and colleagues (2006) who showed there was a trophic-level shift in both $\delta^{15}N$ (+2-3‰) and $\delta^{13}C$ (+1‰) values sampled from the fingernails and hair of modern mother-infant breastfeeding pairs. The primary interpretation is that breastmilk is enriched in ^{15}N comparatively to the mother's overall diet, and has been used with relative frequency as the primary model for studying breastfeeding and weaning processes in the past (Katzenberg 1993; White and Schwarcz 1994; Katzenberg et al. 1996; Schurr 1998, Herring et al. 1998, etc.). Additionally, rapidly declining $\delta^{13}C$ values may show a stronger association with the introduction of solid foods into the infant weaning diet (Fuller et al. 2006).

Despite the fact that studies using carbon and nitrogen stable isotope analyses to interpret breastfeeding and weaning are firmly established within the bioarchaeological literature, several confounding factors and potential problems exist in their analysis and interpretation. Many researchers have treated weaning as an event, rather than a

process, which is a fundamental misunderstanding of the practice that can lead to misinterpretation (Millard 2000; Katzenberg et al. 1996; Tsutaya and Yoneda 2015). The following problematic assumptions put forward by Katzenberg et al. (1996), and Reynard and Tuross (2015) also highlight room for interpretive error:

- The collagen samples from non-adults who died of unknown causes will accurately reflect the diet and physiology of a healthy infant
- Breastmilk is largely assumed to have a fixed isotope compositions for all females
- All adults and non-adults have the same diet-tissue spacing through time
- The $\delta^{15}N$ values of a breastfeeding female's milk and bone collagen are different
- Infants are weaned with foods that have lower $\delta^{15}N$ values than breastmilk

Many of these assumptions are precarious, and recent research has already shown interpretations based upon these hypotheses are inherently flawed. Herrscher and colleagues (2017) analysed the isotopic composition of breastmilk and fingernails over 34 weeks and noted that the difference between the $\delta^{15}N$ values of the mother-infant pairs was less than reported in the bioarchaeological literature, and that breastmilk became depleted in ^{15}N in comparison to the mother's fingernails. They also noted that the $\delta^{13}C$ values of breastmilk were highly variable during the breastfeeding and weaning periods, and was likely influenced by physiological stress due to lactation or motherhood rather than diet (Herrscher et al. 2017). Similar variable patterns were also noted in incremental analyses of deciduous teeth in comparison to bulk bone collagen samples, which underscores the need to deepen our understanding of isotope variation and fractionation in growing and stressed bodies (King et al. 2017).

3.7.2 Water Stress

In their studies looking at the effect of climate on nitrogen isotope ratios in Africa, both Heaton and colleagues (1986) and Sealy and colleagues (1987) noted distinctive intraspecific variations in the $\delta^{15}N$ values of similar species in different climates. They interpreted the unusually high $\delta^{15}N$ values from animals in arid environments as a byproduct of water stress; a physiological means of water retention that leads to a shift in nitrogen isotope mass balance and ultimately, the excretion of more ^{14}N in urea

(Schoeninger and DeNiro 1984; Ambrose 1991; Tieszen 1991). With the increased excretion of the lighter nitrogen isotope, more of the heavier nitrogen isotope is retained within animal and human tissues, leading to a higher $\delta^{15}N$ value that may be interpreted as marine exploitation in diet. This has also contributed archaeologically in the interpretation of water management in areas experiencing droughts or environmental change (Ferrio et al. 2005; Riehl et al. 2008; Riehl et al. 2014).

3.7.3 Nutritional Stress

Studies on modern avian tissues and other animal taxa suggest fasting and starvation will result in an enrichment in ^{15}N (Hobson et al. 1993; Martinez del Rio and Wolf 2005; Fuller et al. 2005; Hatch et al. 2006; Mekota et al. 2006; Hatch 2012). Inadequate dietary protein intake can result in a negative nitrogen balance, which may lead to protein catabolism - the breakdown of extant protein structures into simple amino acids with amine groups that disproportionately retain ^{15}N, and then repeats the transamination and demamination processes (see 3.6.4; *ibid*). This is incited by the body's allostatic responses to maintain homeostasis (McEwen 1998; 2010). Allostasis is a form of metabolic adaptation and compensatory response within the body due to episodic stressors, but if an organism's allostatic load is stretched beyond the body's limits, the organism will go into allostatic overload, which can create lifelong impairments to metabolic function (i.e. compromised immunity) (*ibid*). This recycling of bodily tissues through protein catabolism as an allostatic response will result in ^{15}N enrichment beyond the dietary level (Biesel 1975; Hobson et al. 1993; McEwen 1998; 2010; Fuller et al. 2005).

Interestingly, not all animal studies show parity with this effect, further compounding the complexity of nitrogen isotope mass balance and nutritional intake (Hatch 2012). This discrepancy may be due to an inter- and intra-species 'Threshold Effect' mitigating the onset of ^{15}N enrichment in the face of nutritional stress (Hatch 2012:344-345). Extant nitrogen isotope mass balance models suggest that ^{15}N enrichment will only occur when the body passes a threshold resulting in severe loss of protein; usually after lipid breakdown (Martinez del Rio and Wolf 2005). This may explain why, despite fasting, certain species of animals with ample fat distribution (e.g. polar bears, dolphins, musk-

oxen, hibernating animals) showed little or no ^{15}N enrichment (Hatch 2012). This threshold effect and the body's preference to breakdown lipids first was replicated in humans by Owen and colleagues (1998), and may account for some of the $\delta^{15}N$ variation seen in past populations undergoing known periods of famine; especially children with notably less fat reserves (Tomkins et al. 1983; Beaumont and Montgomery 2016). Nutritional stress can produce a reduction in $\delta^{13}C$ values as well. Fats are already depleted in ^{13}C relative to diet due to a secondary biochemical fractionation, therefore when the body breaks down fat (lipolysis) to meet energy needs, the overall ratio of the body's carbon pool can also be affected (Krueger and Sullivan 1984; Lee-Thorp et al. 1989; Mekota et al. 2006; Ohkouchi et al. 2015). This would lead an individual suffering from famine to display the catabolic effects of a tertiary biochemical fractionation process, demonstrating an opposing covariance in their nitrogen and carbon values (i.e. $\delta^{15}N$ values increase as $\delta^{13}C$ values decrease) (Krueger and Sullivan 1984; Lee-Thorp et al. 1989; Mekota et al. 2006; Ohkouchi et al. 2015; Beaumont and Montgomery 2016). Conversely, humans experiencing an anabolic state exhibit a reduction in ^{15}N enrichment (Macko et al. 1986; Fuller et al. 2004; Hatch 2012). Anabolism is a metabolic mechanism to build complex structures (e.g. proteins) from simple molecules within the body. During periods of active growth, amino acids are preferentially routed for protein synthesis, eliminating the need for trans- and deamination via the metabolic pathways of the liver, other splenic tissues, kidneys, etc. (Minagawa and Wada 1984; Fuller et al. 2004; 2005; Hatch 2012). This anabolic state alters nitrogen diet-tissue spacing by reducing the kinetic fractionation effects, which in turn lessens nitrogen excretion and yields lower $\delta^{15}N$ values. This phenomenon has been observed in modern and archaeological studies of pregnant women and non-adults experiencing growth spurts (Beckett et al. 1997; Boisseau et al. 2002; Fuller et al. 2004; Waters-Rist and Katzenberg 2010; D'Ortenzio et al. 2015). It should be noted that $\delta^{15}N$ values from archaeological bone collagen in pregnant women and growing non-adults tends to obscure shifts to anabolic, or even catabolic, states due to the wide temporal resolution and problems of equifinality presented in the tissue; therefore sampling of incremental tissues such as hair and dentine are best (Nitsch et al. 2010; Waters-Rist and Katzenberg 2010; Beaumont et al. 2013a; D'Ortenzio et al. 2015; Beaumont and Montgomery 2016).

TABLE 3.4 – The effects of protein catabolism and anabolism on $\delta^{15}N$ values (from Allison 1957; Fuller et al. 2004; Fuller et al. 2005; Hatch et al. 2006; Mekota et al. 2006; Waters-Rist and Katzenberg 2010; Petzke et al. 2010; Hatch 2012; Beaumont and Montgomery 2016).

	METABOLIC CAUSE	EFFECT	OBSERVED IN
CATABOLISM	Net protein loss incites breakdown of extant tissues for energy and new protein synthesis	Negative nitrogen balance; $\delta^{15}N$ values increase	Starvation and Eating Disorders, Malnutrition, Weight Loss, Morning Sickness
ANABOLISM	Tissue growth reroutes amino acids direct to tissue for protein synthesis, bypassing trans- and deamination	Positive nitrogen balance; $\delta^{15}N$ values decrease	Lactation, Weight Gain, Pregnancy, Puberty, Growth Spurts

3.7.4 PATHOPHYSIOLOGICAL STRESS

Other forms of pathologically-induced physiological stress, or pathophysiological stress, including infection and trauma, can cause significant fluctuations in $\delta^{15}N$ values, and in extreme circumstances, $\delta^{13}C$ values. Pathogenic microorganisms elicit defensive reactions in the body that incite specific and consistent patterns of metabolic processes (Beisel 1975; 1977; Tomkins et al. 1983). During an infection, the immune system requires a large amount of protein to mount an effective defensive response, which will result in an overall negative nitrogen balance despite concurrent anabolic and catabolic systems (Beisel 1975; 1977; Tomkins et al. 1983; McEwen 1998; 2010; Pietzke et al. 2010). The overall nitrogen loss paired with the increase in metabolic energy needed for heightened immune function can push the body into allostatic overload, inciting a concurrent breakdown in both muscle mass and fat reserves that occurs at an exponential rate in comparison to dietary restriction, with lasting, and sometimes fatal consequences (*ibid*). During the initial phases of illnesses such as infections, trauma, neoplastic diseases, etc., the body rapidly enters a negative nitrogen balance for the reasons mentioned above, but depending on the severity of the health insult and an organism's allostatic load, the body may slowly recover or, if the person is chronically ill, they may enter into allostatic overload and a state of chronic cachexia (Biesel 1975; 1977;

McEwen 1998; 2010; Kotler 2000; Evans et al. 2008; Bechard et al. 2012; Reitsema 2013; D'Ortenzio et al. 2015). Cachexia is described as a systemic wasting disease that occurs after fat stores are depleted, and incites skeletal muscle catabolism to maintain an effective level of immune function (Evans et al. 2008). Cachexia is a secondary byproduct of an underlying disease process (e.g. leprosy, tuberculosis, cancer) that incites inflammation, loss of appetite, fatigue, impairs anabolic function, endocrine dysfunction, and anaemia; all of which will have a deleterious effect on a person's normal nitrogen balance (Kotler 2000; Evans et al. 2008; Petzke et al. 2010; Reitsema 2013; D'Ortenzio et al. 2015; Figure 3.6). Over time, individuals in a state of cachexia will reach the threshold where muscle is depleted to the extent of systemic failure and significant organ impairment, which can show a decrease in $\delta^{15}N$ values; not because of anabolic processes, but simply because there is no further energy or protein left to synthesise regardless of dietary intake (Beisel 1975; 1977; Tomkins et al. 1983; Tayek and Brasel 1995; Hatch 2012). Further, these processes will show exaggerated effects in younger cohorts (Gandra and Scrimshaw 1961; Wilson et al. 1961; Tomkins et al. 1983; Donovan et al. 1990; Coss-bu et al. 2001; Bechard et al. 2012). Additionally, if an individual suffers from a condition that incites bone resorption (e.g. osteolysis from a disease process), the breakdown of bone apatite into the body's metabolic pool can incite a tertiary fractionation factor, which could, in principle, change a person's $\delta^{13}C$ value (Krueger and Sullivan 1984; Lee-Thorp et al. 1989; Katzenberg and Lovell 1999). Numerous other diseases including paralytic conditions, sarcopenia, osteoporosis, osteopenia, renal disease (e.g. amyloidosis and nephritis), liver disease, etc. can also alter the body's metabolic processes and subsequent diet-tissue spacing (White and Armelagos 1997; Evans 2010; Petzke et al. 2010; Reitsema 2013).

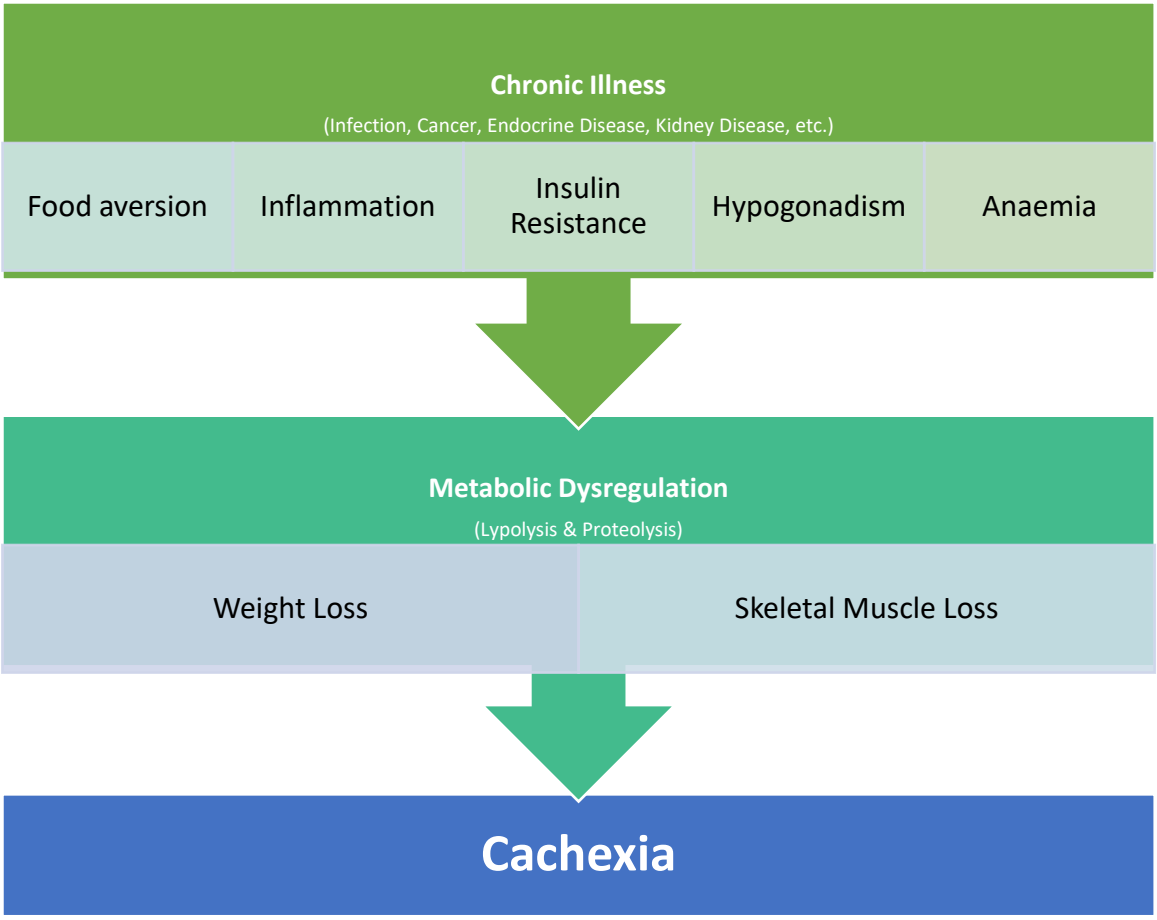

Figure 3.6 Flow chart of the cachexia process (created from information from Kotler 2000 and Evans et al. 2008: 794)

Despite examining the remains of people who died, few studies have used carbon and nitrogen stable isotopes as a proxy to examine these pathophysiological factors, or the body's metabolic response to disease (Reitsema 2013). However, of those that have been published, their potential for interpretation of these fundamental biological processes is invaluable to stable isotopy in archaeology.

3.8 Carbon and Nitrogen Stable Isotopes in Palaeopathology

A vital aspect of isotopic analysis within archaeology that is understudied is the link between stable isotopes in association with pathophysiological disruptions in the body, specifically to contextualise isotope values in archaeological human remains showing signs of pathology. The majority of published studies looking at carbon and nitrogen isotopes from archaeological human skeletal remains use the palaeodietary model as the

default interpretive framework. Although this can be extremely useful in palaeopathological studies of nutritional stress and famine, it is not wholly applicable to archaeological skeletal remains displaying signs of pathology such as infection, complications from trauma, or conditions that result in cachexia. Despite this, most carbon and nitrogen isotope studies use pathological bone changes to interpret archaeological diet and status (e.g. access to foodstuffs), neglecting to acknowledge the metabolic changes that will affect how carbon and nitrogen are routed during different disease processes. This presents a fundamental flaw in the use and interpretation of these systems when viewing the mortal remains of people with skeletal manifestations of disease, and highlights the need for a better understanding of pathophysiology in order to make meaningful and accurate interpretations about peoples of the past.

Early studies looking to interpret pathology using carbon and nitrogen isotopes as a proxy were formative in presenting pathological data alongside isotopic data, but failed to consider some biological implications. For example, Wright and White (1996) published one of the first studies to examine the health impact of civilisation change using carbon and nitrogen stable isotopes. Their study looked at evidence of pathological stress, including childhood indicators of stress (cribra orbitalia, porotic hyperostosis, linear enamel hypoplasia), metabolic deficiency (scurvy) and infection (periostitis), and compared the individuals' carbon and nitrogen values to view diachronic patterns of human biology (*ibid*). Their results did not yield any significant pattern, but several methodological and interpretive issues abound; notably the attempt to establish a link between isotope values of adult bone collagen and healed childhood lesions, which would have been obscured isotopically due to normal metabolic turnover. Nonetheless, it helped pave the way for subsequent investigations into palaeopathophysiology and stable isotopes.

Another study by White and Armelagos (1997) noted the presence of higher $\delta^{15}N$ values in female Nubian mummies with osteopenia in comparison with males. White and Armelagos linked these elevated nitrogen isotope ratios with dietary insufficiency for the women that died in the third decade of life, and menopause for the women who died in the fifth decade of life (*ibid*). However, it is more likely that the higher $\delta^{15}N$ values are

because osteopenia is often accompanied by sarcopenia (skeletal muscle wasting), which would facilitate protein catabolism and recycling of proteins into the larger metabolic pool (Evans 2010). Interestingly, White and Armelagos (1997) did not observe the same level of ^{15}N enrichment in the males with osteopenia, therefore other metabolic reactions, such as sex-linked hormonal changes may have contributed to the muscle and bone wasting observed in the females. Although White and Armelagos' (1997) studies are unable to be revisited, their contribution in establishing a link between isotope values and palaeopathology is instrumental.

Although not palaeopathological per se, Katzenberg and Lovell's (1999) study on stable isotope variation in pathological bone solidified the link between pathophysiological stress and ^{15}N enrichment. Even though they acknowledged that the ^{15}N enrichment was likely due to catabolism of extant proteins and the individual was 'wasting' at the time of death, they focused on nutritional insufficiency rather than the cachectic state associated with HIV/AIDS and infection (Katzenberg and Lovell 1999: 322-323). Further, Katzenberg and Lovell (*ibid*) demonstrated that in wasting diseases where bone apatite is being broken down (e.g. post-paralytic atrophy), ^{13}C enrichment is also possible.

Beaumont and colleagues' (2013a) seminal paper on the analysis of carbon and nitrogen isotope ratios from incremental dentine firmly cemented the link between physiological stress and the interpretation of carbon and nitrogen stable isotopes from archaeological remains. Their research demonstrated the dynamic complexity of metabolic processes governing carbon and nitrogen isotope ratios in organic dental tissues of individuals with documented periods of famine (*ibid*). Although Beaumont and colleagues' (2013a; 2013b; 2015; 2016; 2018) work primarily focuses on physiological disruptions caused by famine and nutritional stress, it has significantly demonstrated that carbon and nitrogen isotope interpretation is not just a matter of marine vs. terrestrial or breastfed vs. weaned, and that the processes that influence human metabolism in the present and past should be given more attention in interpretation. Concurrent to Beaumont and colleague's 2013 publications and shortly thereafter, other scholars were noting similar phenomenon and interpretive concerns.

Reitsema's (2013) paper looking at pathophysiological approaches to carbon and nitrogen stable isotope analysis in palaeopathology stands out as an essential work for advocating interpretations beyond the default palaeodietary models; demonstrating the interrelatedness of nutrition, disease, and the effects of pathology-influenced isotope fractionation on the body. Wheeler and colleagues (2013) were able to demonstrate this further through the analysis of multiple tissues from a 2-3 year-old child from Egypt. Their findings, contextualised with palaeopathological evidence of numerous episodes of non-accidental trauma, were able to link changes in carbon and nitrogen values with fluctuations in bone metabolism incited by the body's healing response (Wheeler et al. 2013). Similarly, Olsen and colleagues (2014) found that individuals with skeletal lesions linked to inflammatory processes and new bone formation (infection, trauma, osteoarthritis) show significant intraskeletal differences in carbon and nitrogen values depending on the proximity to the bone lesion, whereas metabolic conditions such as rickets and osteomalacia did not show significant intraskeletal differences due to the systemic nature of the diseases.

Carbon and nitrogen stable isotope analyses of other incremental tissues have highlighted further benefits of analyses that yield longer temporal results than bulk collagen. Webb and colleagues (2015) analysed increments of hair from Peruvian mummified remains to evaluate both carbon and nitrogen isotope values, as well as the glucocorticoid hormone, cortisol, in order to view life course particulars before death. Their findings were able to distinguish migrants and associate certain pathophysiological stressors before death, data that would have been masked by bulk collagen sampling or macroscopic analyses alone. Further taking a life course approach, D'Ortenzio and colleagues (2015) measured carbon and nitrogen isotope ratios from increments of hair from modern and archaeological individuals with chronic pathological conditions and cachexia, and compared their incremental $\delta^{15}N$ values with modern cadavers that had died suddenly. The outcome of the study showed that $\delta^{15}N$ values were steadily elevated by >1‰ before death in individuals with long-term pathological conditions and cachexia, whilst cadavers that died suddenly showed no significant changes (*ibid*).

Investigations into the complexities of breastfeeding, pathophysiological stress, and the benefits and disadvantages of different tissues have sparked a debate with potential for new, innovative questions. In their papers, King et al. (2017; 2018) compared bulk collagen and collagen from incremental dentine to evaluate weaning practices and attempt to longitudinally discriminate weaning from stress. Their research acknowledged the high degree of variability that can confound isotope interpretation in the face of human biocultural variation, and warned of the dangers of becoming too entangled in the idiosyncratic trajectories that analyses of incremental dentine yields (*ibid*). In response, Beaumont and colleagues (2018) argue that although breastfeeding and weaning varies in response to culture and environment, that the data underpinning bulk collagen analyses may be fundamentally flawed by not accounting for nutritional deficiencies and pathophysiological processes in early life. Beaumont et al. (2018) evaluated bulk bone collagen and collagen from incremental dentine from the same individuals from Raunds Furnells, and argue that collagen extracted from dentine versus collagen extracted from bone may differentially reflect dietary intake depending on an individual's nutritional or pathophysiological stress status. They posit that during periods of extreme stress, bone may not actually be forming, and is therefore not a reflection of diet, weaning status, or health status (*ibid*). This hypothesis has since been supported and furthered by Matsubayashi and Tayasu (2019), who looked at femoral cortical bone. Their research found that only the perimedullary bone showed metabolic turnover of collagen, with the midcortical and pericortical sections only reflecting bone collagen that had formed decades earlier in some cases (*ibid*). This certainly sparks an interesting theoretical development, which necessitates past studies of diet and bone apatite to be re-evaluated.

Additional syntheses solidifying the link between isotope analyses and pathology are crucial to encouraging more rigorous interpretations. Recently, a special issue of Bioarchaeology International focussed on stable isotope advances in bioarchaeological studies of stress and disease, which continues to underscore the vital link between isotope studies and pathophysiology (Reitsema and Holder 2018). Special thematic issues such as these, as well as syntheses such as Richards and Montgomery's (2012) review

and suggestions for linking palaeopathology with isotope studies in the future are instrumental in moving the field forward.

3.8.1 What is stress? The importance of distinguishing between starvation and disease

From recent research, it is clear that the importance of pathophysiology over the governance of metabolism needs to be considered more in the interpretation of carbon and nitrogen stable isotopes. However, the interpretation in palaeopathological and broader bioarchaeological literature also needs some fine-tuning; particularly with regard to the term 'physiological stress'. Physiological stress is not an apt enough term to describe variations in isotope values because the range of stressors encompassed by the term is too vague; i.e. physiological stress could be induced by sport, mental health, children, disease, famine, pregnancy, environment, etc (Klaus 2014; Temple and Goodman 2014; Reitsema and McIlvaine 2014). Although the effects can sometimes look similar isotopically, changes in nitrogen and carbon metabolism due to episodes of nutritional stress vs. a cachectic state from chronic disease have variable responses in terms of timings and magnitude and should not be conflated (Biesel 1975; 1977; Evans et al. 2008; Hatch 2012). According to Evans and colleagues (2008) it is imperative to differentiate systemic pathological conditions such as cachexia (sometimes termed as 'wasting' in palaeopathological or stable isotope literature) with nutritional deficiency. Nutritional stresses like famine, anorexia, or deficiencies are due to decreased energy intakes and can be remedied by nutritional intervention; whereas a chronic disease can incite persistent inflammation with concurrent fat and skeletal muscle breakdown to a much greater extent, and cannot always be remedied by nutritional intervention (Biesel 1975; 1977; Tayek and Brasel 1995; Evans et al. 2008). These differences in magnitude will have a subsequent effect on an individual's potential for allostasis (stability through adaptation), convalescence, and overall carbon and nitrogen isotope ratios (Biesel 1975; 1977; McEwen 1998; 2010).

Unfortunately, issues of equifinality in bulk stable isotope analysis mask the minutiae of the life course in order to discriminate between nutritional and cachectic stress.

However, more incremental analyses of human tissues are the best way to overcome this issue and will further aid in associating the timings and subsequent effects of pathological lesions on human skeletal remains. Despite some researchers warning about putting too much importance on the idiosyncratic trajectories of incremental data (see King et al. 2018; Beaumont et al. 2018), if the skeletal remains bear signs of pathophysiological processes or nutritional stress, it is worth utilising this method to distinguish between diet and metabolic disruption.

Figure 3.7 Schematic diagram of the biological interdependence between nutrition and disease (Ulijaszek 2018:11)

It is important to acknowledge that the life course approach to stable isotope analysis still involves a level of ambiguity when attempting to distinguish carbon and nitrogen isotope ratios linked to pathophysiological processes vs. episodes of nutritional stress, especially in deceased children; i.e. a combination of factors likely contributed to their death (see Figure 3.7). However, looking at incremental data and skeletal signs of early nutritional stress may elucidate patterns of immunology over the life course. Early nutritional stress can increase allostatic loads, which will dampen immune effects, thereby making some individuals more prone to certain infectious diseases (McEwen 1998; 2010; Katona and Katona-Apte 2008). For example, leprosy is a disease that is both heavily influenced by nutritional status, but can also affect nutritional status during its post-incubation period (Beisel 1975; 1977; Ulijaszek 2018). One potential way to address

this is to take a life course approach by using carbon and nitrogen isotope ratios of incremental dentine as a proxy that can be tied to specifically-timed pathological lesions. For example, by looking at non-specific indicators of childhood nutritional 'stress' (e.g. linear enamel hypoplasia), we may be able to link a metabolic disruption in growth during certain developmental stages and view if it bears any influence over the onset of disease manifestation later in life (Barker 2012; Filipek-Ogden 2012; Gowland 2015; Yaussy et al. 2016). By combining incremental isotope data, developmental timings of pathologies, and clinical and metabolic data, these ambiguities may start to dissipate and a more refined picture of the effects of systemic illness will arise thereby moving beyond the default dietary model.

3.9 The use of Oxygen and Strontium in Bioarchaeology

The movement of people throughout time and space is an important cultural universal intrinsically linked to economy, settlement, disease, conflict, forced migration, marriage, kinship, ethnicity, identity and agency, natural disasters, etc. Analyses of oxygen and strontium isotopes in bioarchaeology can aid in helping to contextualise the impetus for these types of mobility drivers at both the individual and population level in the past. It is worth mentioning that isotopic analyses of lead from human tissues also shows great promise in migration and disease studies of the past, however, it is beyond the scope of this research.

Compared to carbon and nitrogen, isotopic studies of oxygen and strontium have less of a presence in the archaeological literature. This may be due to a variety of factors including methodological advances, indistinct geographical patterning, and overall time and costs. Several studies, however, have shown the meritorious benefits of employing these isotope systems on their own, or in tandem. The following sections further reviews the applications of oxygen and strontium isotopes in archaeology, discuss the benefits and limitations of their use, and review their applications and potential in a palaeopathological framework.

3.10 Oxygen Isotopes

Oxygen isotope ratios have geographical variations that can be used to ascertain evidence of mobility from the bioapatites of bones and teeth (Schwarcz et al. 2010; Pollard et al. 2017). Oxygen isotopes are expressed as $\delta^{18}O$ values, which are derived from the ratio of $^{18}O/^{16}O$ relative to a standard (Pollard et al. 2017). $\delta^{18}O$ values climactically vary within rainwater depending on latitude, altitude, temperature, precipitation, distance from the coast, and season due the Rayleigh fractionation system of isotope enrichment/depletion (Knudson 2009; Knudson et al. 2012; Schwarcz et al. 2010; Lightfoot and O'Connell 2016). The Rayleigh fractionation system is used to account for the isotopic differentiation of meteoric water within the different air masses due to evaporation, condensation, and precipitation; i.e. evaporation of seawater or groundwater results in the preferential loss of the lighter isotope (^{16}O), whereas precipitation preferentially excretes the heavier isotope (^{18}O) (Katzenberg 2011; Lightfoot and O'Connell 2016; Pederzani and Britton 2019; Figure 3.8). This leaves the highest $\delta^{18}O$ values located in coastal, low latitude regions due to the condensation of ^{18}O in precipitation, and spectrally decreases in $\delta^{18}O$ values as latitude, altitude, and distance from the coast increase (Longinelli 1984; Schwarcz et al. 2010; Katzenberg 2011; Pederzani and Britton 2019; Figure 3.8). These factors are combined to establish broad geographical distributions of precipitation values ($\delta^{18}O_{ppt}$), which is used as a proxy to estimate human ingestion of local meteoric water (from drinking water or food) (*ibid*). It is of note that these patterns are not temporally consistent due to temperature fluctuations, therefore past climate data must be contextualised with interpretations of $\delta^{18}O$ values (Daux et al. 2005; Lightfoot and O'Connell 2016; Pederzani and Britton 2019).

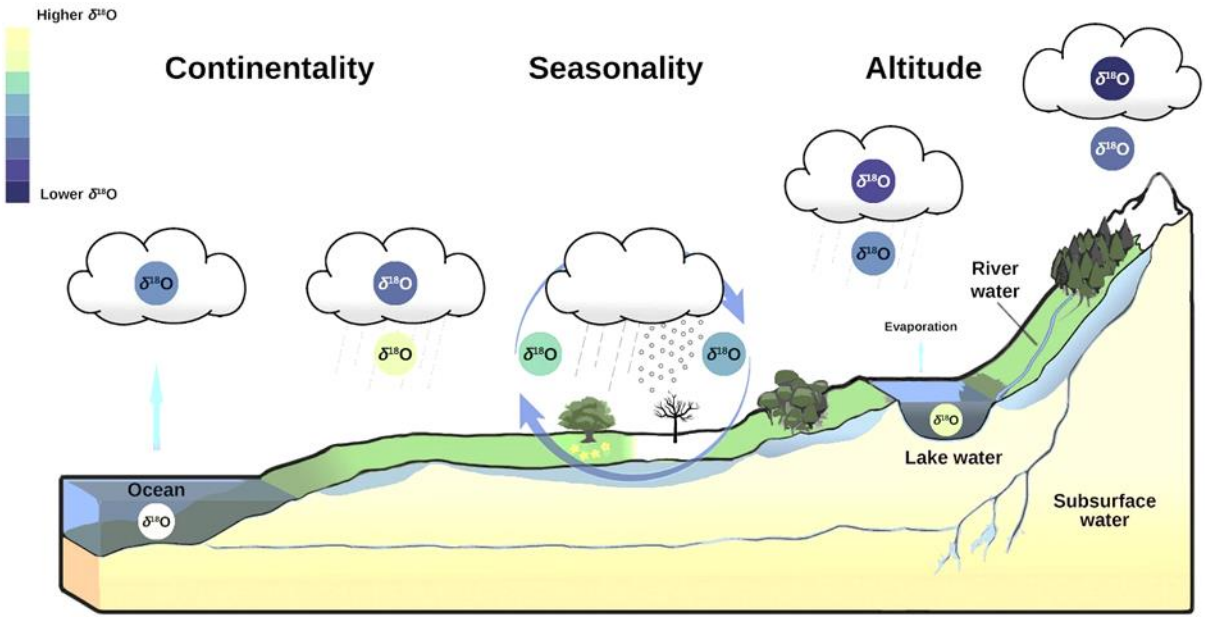

Figure 3.8 Diagram of the Rayleigh fractionation system and the variables that will affect $\delta^{18}O$ values in evaporation, condensation, and precipitation (Pederzani and Britton 2019: 79).Open Access.

The isotopic composition of oxygen in the bioapatites of bones and teeth is resultant from body water composition, which in turn is ultimately derived from the local available water sources (Longinelli 1984; Schwarcz et al. 2010). Normally, the $\delta^{18}O$ values of groundwater are a reflection of $\delta^{18}O_{ppt}$, however, variations may exist due to evaporation patterns, irrigation systems, geological disruptions, alterations in river flow, etc. (Gat 1971). Although not tied to a specific location, drinking water values are largely still a representation of certain climate zones (Schwarcz et al. 2010). This allows researchers to use oxygen isotope ratios to discriminate whether individuals found in burial assemblages show isotopic compositions in their bioapatites similar to local aquifers.

Bioapatite $\delta^{18}O$ values are measured as either phosphate ($\delta^{18}Op$) or carbonate ($\delta^{18}Oc$) and reported relative to Vienna Standard Mean Ocean Water (VSMOW)(see section 3.2; Chenery et al. 2012; Pollard et al. 2017). After the ingestion from drinking water, food intake, and inhaled atmospheric O_2, oxygen undergoes a mass-based, temperature-dependent fractionation to be in equilibrium with body water, with a preferential enrichment of ^{18}O, and preferential excretion of ^{16}O for exhalation (Longinelli 1984; Luz et al. 1984; Bryant and Froelich 1995). After fractionation, the structural carbonate and phosphate components of bioapatite form in isotopic equilibrium with body water values

creating a relative spacing of c. +8‰ and +12‰ (Luz et al. 1984; Bryant and Froelich 1995; Chenery et al. 2012; Pederzani and Britton 2019). Based on this pattern of spacing, several linear regression equations exist to convert structural carbonate and phosphate components into predicted drinking water values (Longinelli 1984; Luz et al. 1984; Daux et al. 2008; Chenery et al. 2012). Analyses of structural carbonate oxygen isotope ratios are more frequent due to the simpler methodologies, lessened costs, and greater measurement precision; however, the biochemical bond between phosphate and oxygen is much stronger and unlikely to be affected by diagenetic alteration, which allows for deeper time perspectives and encompasses smaller margins of analytical error (Longinelli 1984; Luz et al. 1984; Pollard et al. 2011; Chenery et al. 2012; Snoeck and Pellegrini 2015; Pellegrini and Snoeck 2016). Additionally, more data exists linking bioapatite $\delta^{18}O_p$ values with modern drinking water values ($\delta^{18}O_{DW}$) in humans and animals (Longinelli 1984; Luz et al. 1984; Bryant and Froelich 1995; Daux et al. 2008). Chenery and colleagues (2012) posit that it is possible to convert structural carbonate values from enamel into phosphate values with relatively low margins of error, and from there, make a further conversion into predicted drinking water values for comparative purposes. Although their equations show strong linear relationships (+1‰, 2σ), others argue that using multiple equations to convert data increases uncertainty and can lead to misinterpretation (Pollard et al. 2011; Brettell et al. 2012). For this reason, it is essential to first compare the δ^{18}Op values measured from humans before attempting to compare origins based on predicted drinking water values (Pollard et al. 2011). It is also worth noting that since bone bioapatite is subject to lifelong remodelling and post-depositional diagenesis, analyses of δ^{18}Oc from bone samples is not advised (Schwarcz et al. 2010; Chenery et al. 2012; Lightfoot and O'Connell 2016). Tooth enamel, in contrast, is almost entirely inorganic with a high crystallinity index (and therefore less likely to undergo diagenetic changes) making it the ideal tissue for the measuring either δ^{18}Oc or δ^{18}Op values, and the most robust for signalling drinking water values reflective of childhood residence (Schwarcz et al. 2010; Chenery et al. 2012; Pederzani and Britton 2019).

3.10.1 Pathophysiological and anthropogenic variations in δ^{18}O values

Like other isotope systems, local baselines must be established to comprehend a normal range and distinguish outliers. For oxygen isotopes, this may include sampling environmental water, precipitation, and local fauna (Darling 2004; Schwarcz et al. 2010; Lightfoot and O'Connell 2016). Lightfoot and O'Connell (2016) highlight several confounding factors that may affect 'local ranges' when using this data to compare human bioapatite. In their global survey on published oxygen isotope values, they found there were many sources of 'isotopic noise' that affected the oxygen isotope ratios within a population, and that differences of up to ~3‰ within groups better encompasses a normal population distribution (*ibid*). Some of these sources of 'isotopic noise' include pathophysiological influence or anthropogenic practices (*ibid*).

Drinking water is the largest component of bioapatite $\delta^{18}O$ values, and its reflection in bones and teeth is presumably not affected by metabolic disruption in the same way as carbon and nitrogen stable isotopes (Pederzani and Britton 2019). However, others advise caution in dismissing concomitant pathophysiological processes from the overall isotopic composition of bioapatite. In a controlled study on mice with sickle-cell anaemia, Retisema and Crews (2011) found that mice who were pathophysiologically stressed yielded significantly lower $\delta^{18}O$ values in comparison to their healthy controls. In contrast, Warinner and Tuross (2010) found slightly higher $\delta^{18}O$ values in a pig undergoing controlled nutritional stress. Additionally, there is little consensus in the timings of the mineralisation processes and the means by which the body subsequently accumulates its isotope composition in bioapatites, creating further ambiguity over the physiological minutiae involved in the isotopic reflection of these tissues (Lightfoot and O'Connell; Pederzani and Britton 2019). This certainly merits more investigation as our understanding of the extent of metabolic fractionation of oxygen with regard to pathological influence has not been fully explored (Reitsema and Crews 2011; Lightfoot and O'Connell 2016).

Depending on the age at death or tooth type, the influence of breastfeeding and weaning practices, such as the use of paps and whey, may cause alterations to the overall oxygen isotope composition of human bioapatites. Human breastmilk is enriched in ^{18}O due to

fractionation processes that create equilibrium with body water and preferentially excrete ^{16}O through exhalation (Bryant and Froelich 1995). Because the liquid portion of human breastmilk is composed of body water, infants consuming breastmilk will show higher $\delta^{18}O$ values in their bioapatite of up between 0.5-3‰ in comparison to the 'local range' (Wright and Schwarcz 1998; 1999; Tsuyata and Yoneda 2015; Lightfoot and O'Connell 2016). Issues of disease, starvation, and breastfeeding call into question the faithfulness of oxygen isotope ratios to mirror local drinking water values across an isoscape in individuals that died at a young age or were subject to pathophysiological or nutritional stress (Pederzani and Britton 2019). If these factors can alter oxygen isotope composition, then choosing elements for analysis from individuals under these stressors can skew the distribution of data, creating artificial parameters that are not reflective of local ranges or practices.

Beyond *in vivo* fractionation, other anthropogenic behaviours can alter the isotopic composition of drinking water. Culturally-mediated factors over ingested fluids such as boiling, brewing, dairying, stewing (dependent on time), and distillation will cause evaporative effects on $\delta^{18}O$ values, yielding significantly more ^{18}O enriched water (up to 16‰ in wine) (Lin et al. 2003; West et al. 2007; Chesson et al. 2010; Brettell et al. 2012; Meier-Augenstein et al. 2012; Lamb et al. 2014; Tuross et al. 2017; Figure 3.9).

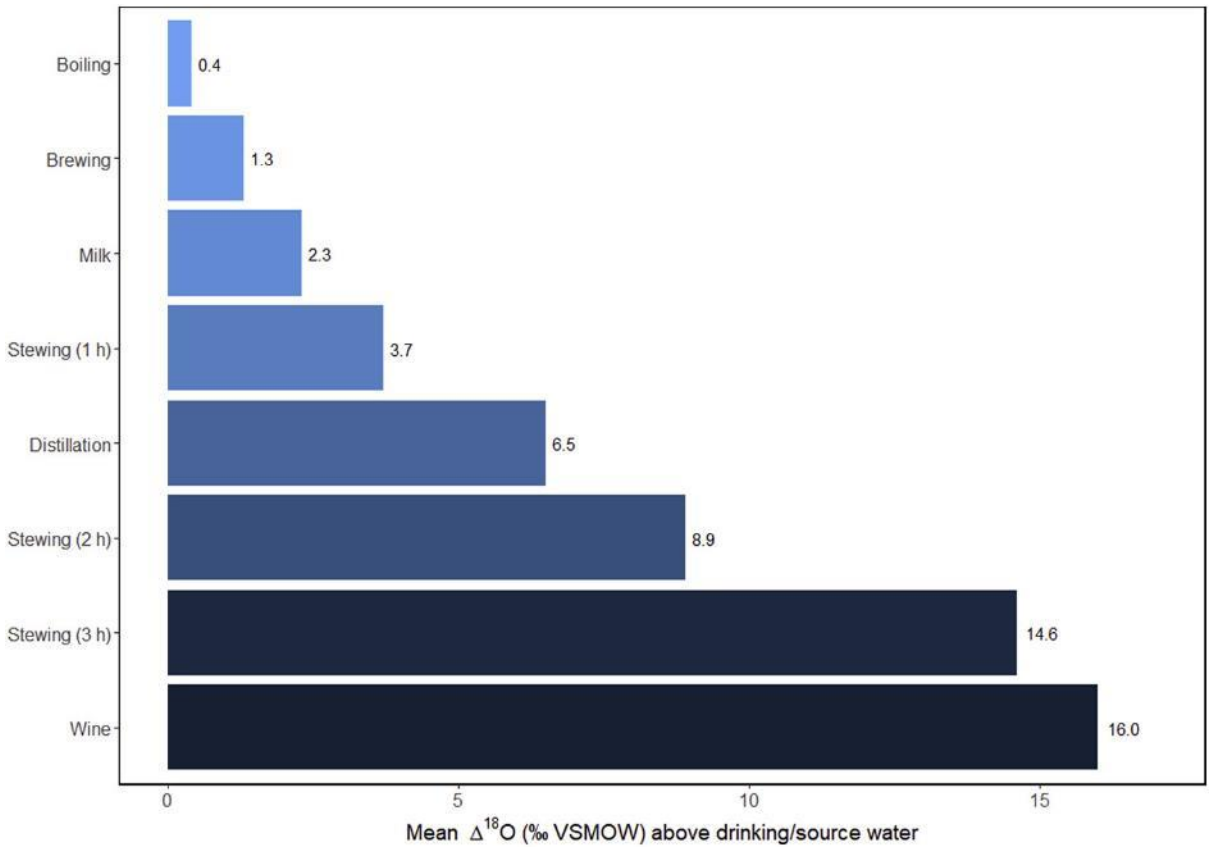

Figure 3.9 Enrichments in ^{18}O above drinking water, precipitation, or source water (Pederzani and Britton 2019: 94).Open Access.

For example, a study on Medieval oxygen isotope values by Brettell and colleagues (2012) examined the $\delta^{18}O$ values of ale, 'wort drinks' and teas, and pottage, all of which were likely consumed by Medieval children. Their study found that boiling water and cooking of foodstuffs in sufficient quantities can heavily alter an individual's oxygen isotope composition making it appear as if they had spent their childhood in warmer climates. For this reason, it is imperative that conversion equations (such as Daux et al. 2008, eq. 6) account for culturally-mediated factors over oxygen isotope values, and that contextual sources regarding the preparation of food be consulted, if available. Another way to mitigate the influence of pathophysiological or anthropogenic factors is to either treat each population as a discrete data set, or better, use multi-isotope systems to help define locality. By incorporating other individuals that are from known areas, outliers may also be better discriminated.

3.10.2 The use of oxygen isotopes in palaeopathology

Because of the non-specificity of oxygen isotope data, few studies have used them in isolation to look at the relationship between palaeopathology and mobility. However if the underlying geology is fairly homogenous, the use of strontium as an accompanying isotope system is inappropriate. Despite some of the ambiguities of oxygen data, some research has demonstrated the movement of people in association with disease or trauma using oxygen isotope ratios. At the Mesoamerican site of Teotihuacan, osteological analyses revealed many of the sacrificial victims found in the Moon Pyramid and Feathered Serpent Pyramid belonged to high-status individuals potentially from Guatemala, and $\delta^{18}O$ values demonstrated that individuals whose mandibles had been used as warrior trophies originated in three different locations around the Mayan stronghold (White et al. 2002; 2007; Spence et al. 2004). This treatment of individuals that are non-local to the site is thought to demonstrate power over both the body-politic and outlying socio-political identities. In a study using oxygen isotope ratios as a means to identify migration in the Dakhleh Oasis, Egypt, Dupras and Schwarcz (2001) were able to identify individuals with skeletal changes consistent with leprosy who had originated elsewhere to the region. Further analysis by Groff (2015) later expanded these findings by comparing $\delta^{18}O$ values of intratooth enamel samples and bone apatite, revealing that four out of the six individuals with leprosy found at the Dakhleh Oasis migrated into the area, possibly for the healing properties of alum. Prowse and colleagues (2007) analysed the teeth from 61 individuals buried at Isola Sacra, and found that approximately one-third yielded $\delta^{18}O$ values inconsistent with Rome. The large number of migrants represented in the burial population was interpreted as a possible reflection of the high mortality rate associated with hyperendemic falciparian malaria, which that they would have encountered upon migrating into the city (*ibid*). Interestingly, despite the influx of non-locals within the burial populations at Isola Sacra, the population does not appear to increase over time, which is also a byproduct of migration to areas with endemic diseases with substantial mortality rates (*ibid*).

As oxygen is often used in conjunction with strontium, a further section below details how they have been used together to elucidate the ways in which mobility history can be used in conjunction with palaeopathology.

3.11 Strontium Isotopes

Since the formative paper by Ericson (1985) first proposed the potential for the measurement of strontium isotope ratios as a biogeochemical tracer to assess human migration, studies using radiogenic strontium isotope analysis have overwhelmingly aided in creating models of movement, trade, settlement, and culture exchange in archaeological contexts. Strontium is a trace element found in most rocks and minerals, as well as aquifers, saltwater, soils, plants and animals/humans and its ratios vary geographically dependent on the age of the underlying bedrock (Slovak and Paytan 2011). Strontium has four stable isotopes (^{84}Sr, ^{86}Sr, ^{87}Sr, ^{88}Sr), of which, ^{87}Sr is radiogenic and formed by the radioactive decay of ^{87}Rb (see section 3.2) (Coelho et al. 2017). Because of its large atomic mass, the ratio of $^{87}Sr/^{86}Sr$ does not alter through biogeochemical reservoirs, and any variation that may occur is normalised during mass spectrometry (Slovak and Paytan 2011; Coelho et al. 2017). The ratios of strontium within living human tissues are ultimately derived from food and water consumption. Because $^{87}Sr/^{86}Sr$ is relatively constrained through the foodchain and does not undergo fractionation after consumption, archaeologists can use radiogenic strontium isotope ratios to model provenance and first-generation migration patterns (*ibid*; Figure 3.10). Notably, the measurement of $^{87}Sr/^{86}Sr$ in human tooth enamel can provide direct evidence for childhood residence and reflect mobility throughout an individual's first two decades of life (Katzenberg 2008; Evans et al. 2012). Previous studies looking at $^{87}Sr/^{86}Sr$ from bone have highlighted post-mortem diagenetic issues, namely leaching of strontium from the surrounding soil into human remains, therefore enamel serves as the ideal tissue to reflect childhood origins, whereas bone will reflect the burial locale (Sealy et al. 1991; Budd et al. 2000; Trickett et al. 2003; Burton and Price 2013). Additionally, human enamel does not undergo remodelling after formation, whereas bone is a dynamic tissue that remodels throughout the life course introducing more opportunity for issues of equifinality;

Figure 3.10 Diagram illustrating the relatively static nature of strontium ratios through biochemical processes (Coelho et al. 2017: 46). Reproduced with permission from Elsevier (License 4983030360317).

especially if an individual has moved (Bentley 2006). One exception to this may be the measurement of strontium in cremated remains. Recent methods have explored the use of cremated remains in strontium isotope analysis, with promising results (Snoeck et al. 2016). During the cremation process, bone becomes highly calcined with a high crystallinity index in comparison to unburnt bone, thereby encapsulating the *in vivo* strontium ratios (Thompson 2005; Snoeck et al. 2016; Snoeck et al. 2018). This new development will likely transform the utility of cremated remains in biomolecular archaeology and elucidate large swathes of prehistorical chronologies. Although radiogenic strontium isotope ratios are a robust means of assessing childhood geographic

origins, some interpretive factors must be considered. For example, movement is only visible if an individual's $^{87}Sr/^{86}Sr$ ratio differs from locally measured radiogenic strontium ratios. If they show homogeneous ratios with the local burial environment, it may mean that they are local to the area, or conversely, migrated from a region with a similar underlying geology. In places such as Britain, large geographical areas have consistent geological terra, therefore many individuals who yield strontium isotope ratios similar to their burial locale may in fact have moved from an area from further afield without significantly changing their radiogenic strontium isotope composition (Ericson 1985; Bentley 2006; Evans et al. 2010; Evans et al. 2012). Moreover, an individual may have consumed food from two or more different geological areas that produced a mixed $^{87}Sr/^{86}Sr$ value comparable to their burial location. One way to address this issue is to create $^{87}Sr/^{86}Sr$ mixing models ($^{87}Sr/^{86}Sr$ vs. 1/Sr) or employ a dual isotope system (e.g. oxygen) to help refine local bioavailable and geological strontium baselines (Price et al. 2002; Montgomery et al. 2007; Montgomery 2010; Burton and Price 2013; Burton and Hahn 2016).

Establishing an individual's mobility history is constrained by another significant variable; issues of origin. Strontium isotope analyses are only able to exclude individuals from their burial environment, but the data on its own will not reveal an individual's geographic origins (Montgomery 2010). After a 'non-local' member is identified within a burial population, it is also not possible to estimate the time that has lapsed since moving to the new locale (*ibid*). Conventionally, the default interpretation has been to suggest an immigrant is likely from the nearest terrain with similar strontium isotope ratios, however given the homogeneity of widespread geological terra, and the lack of comprehensive coverage in large-scale maps of biosphere strontium isotope ratios, the closest locale may not reflect a person's true origins (Evans et al. 2010). Recently, the availability of new online isotope platforms such as IsoArch (isoarch.eu) and the BGS Biosphere Isotope Domain for Great Britain (https://www.bgs.ac.uk/products/geochemistry/BiosphereIsotopeDomainsGB.html) have been published as a means to assist researchers in defining potential locations of origins, not just exclusions. But even so, the impetus for movement and identity is always

contextually based, therefore the biocultural archaeological context must be at the forefront of any interpretations.

3.11.1 Developing local $^{87}Sr/^{86}Sr$ baselines

The determination of local ecological and geological baselines is a key part of mobility analyses. An accurate knowledge of the range of $^{87}Sr/^{86}Sr$ ratios for a particular locale as well as local animals/humans is integral to using the method as a biological proxy for any patterns of migration, culture exchange, or even the spread of disease (Burton and Price 2013; Burton and Hahn 2016). A broad range of $^{87}Sr/^{86}Sr$ signatures can be estimated based upon the type of underlying geology; i.e. older geological formations such as continental granites with high Rb/Sr ratios will generally yield $^{87}Sr/^{86}Sr$ ratios well above 0.710, whereas the young geological formations such as Icelandic basalts can yield ratios below 0.704 (Burton and Price 2013; Evans et al. 2010, Table 3.5). Therefore areas underlain with geological formations such as Cretaceous chalk (e.g. Winchester) provide strontium isotope ratios ranging from approximately 0.708-0.709, whereas regions with geologically mixed terra (e.g. Norwich with Cretaceous chalk and Quaternary sands, gravel, silts, and clays) would provide human strontium isotope ratios that would plot along a mixing line between the highest (Quaternary sands and gravel) and lowest (Cretaceous chalk) strontium sources (Evans et al. 2010; Table 3.5).

Table 3.5 - Average $^{87}Sr/^{86}Sr$ ratios sampled from material with varying chronological lithologies (Data from Evans et al. 2010:3).

Age of underlying lithology	*Average 87Sr/86Sr*	*Material Sampled*
Igneous England Palaeozoic	0.7070	Plants
Igneous Scotland Tertiary	0.7075	Plants
Cretaceous Chalk	0.7076	Soil Leach
Cretaceous Chalk	0.7079	Water
Cretaceous Chalk	0.7082	Plants
Cretaceous Chalk	0.7082	Dentine
Cretaceous Greensand	0.7084	Bone, Water, Soil Leach, Plants, Dentine
Igneous Mesozoic, Scotland	0.7085	Bone, Water, Soil Leach, Plants, Dentine
Carboniferous Limestone	0.7089	Soil Leach
Carboniferous Limestone	0.7092	Plants
Carboniferous Limestone	0.7094	Dentine
Palaeogene	0.7093	Dentine
Cambrian, Scotland	0.7096	Bone, Water, Soil Leach, Plants, Dentine

Triassic	0.7097	Water
Jurassic Clay	0.7097	Plants
Proterozoic Dalradian	0.7097	Plants
Triassic	0.7097	Dentine, Soil Leach, Water
Devonian, Scotland	0.7098	Dentine, Bone, Water
Carboniferous Limestone	0.7099	Water
Strontian Granite	0.7099	Plants
Igneous, S Wales Palaeozoic	0.7100	Plants
Devonian, W Devon	0.7101	Plants
Ordovician, Wales	0.7105	Plants
Carboniferous Grits., N Devon	0.7107	Water
Proterozoic, N Loch Shin	0.7112	Plants
Proterozoic, Appin Gp	0.7113	Plants
Cambrian, Wales	0.7115	Water
Carboniferous Grit, Midlands	0.7116	Water
Silurian, Wales	0.7117	Plants
Proterozoic, Torridian	0.7121	Plants
Ordovician, N Wales	0.7121	Water
Devonian, N Devon and Hereford	0.7121	Water, Plants
Silurian, Wales	0.7123	Water
Dartmoor granite	0.7125	Plants, Water
Carboniferous Grits, S Devon	0.7126	Water
Proterozoic, Dalradian	0.7127	Plants
Ordovician, C Wales	0.7130	Water
Proterozoic, S Loch Shin	0.7165	Plants, Water
Cairngorm, Etive & Angus granites	0.7175	Plants, Water, Bone
Archaean; Lewisian	0.7198	Water
Proterozoic hotspots	0.7222	Plants

Although underlying geology-type can provide a general approximation of $^{87}Sr/^{86}Sr$ ratios, disparate values from geological to bioavailable strontium can and do occur (Price et al. 2002; Bentley 2006; Burton and Price 2013). Therefore, in order to establish a confined baseline of radiogenic strontium ratios that accurately reflects locality, $^{87}Sr/^{86}Sr$ should be measured from both geological and multiple bioavailable strontium sources, such as a range of small local animals whose diets will reflect place-specific averages (Price et al. 2002; Bentley 2006). From there, distribution maps that highlight spatial variations between $^{87}Sr/^{86}Sr$ across regions, countries, and continents can be created (Evans et al. 2010; Voerkelius et al. 2010; Figure 3.11). Improving the resolution of location-specific strontium isotope ratios is also of particular importance because 95% of $^{87}Sr/^{86}Sr$ human enamel ratios fall within a confined range of 0.7047-0.7190, with a mode of 0.7092 (Burton and Price 2013). This margin of difference (0.0143) in radiogenic strontium isotope ranges in human enamel underscores the necessity for developing local $^{87}Sr/^{86}Sr$

baselines using all sources of bioavailable strontium from modern and archaeological flora and fauna in order to avoid gross misinterpretations of locality.

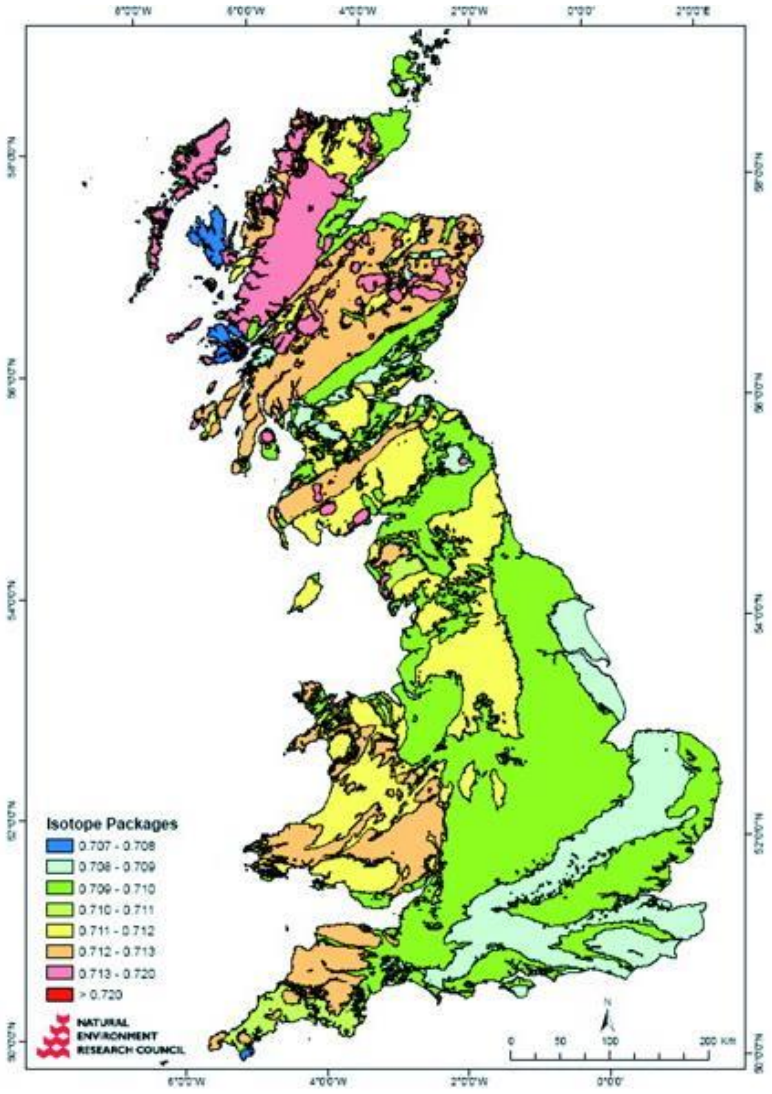

Figure 3.11 Map of biosphere strontium isotope variation across Britain (Reproduced with the permission of the British Geological Survey ©UKRI. All rights Reserved).

3.11.2 Environmental and anthropogenic factors affecting radiogenic strontium ratios

Despite underlying bedrock geology being the principal control for strontium isoscapes, dynamic environmental niches and anthropogenic factors can alter the expected signatures for a given area. In certain climatic zones, such as areas inundated with rainfall and sea spray; dry, arid climes; or regions in a catchment area for volcanic ash; resultant atmospheric particles can have varying effects over local bioavailable strontium isotope

ratios. Further, there is evidence for anthropogenic factors such as manuring, importation of foodstuffs, and breastfeeding affecting the expected $^{87}Sr/^{86}Sr$ ratios in humans (see below).

(I) Maritime Environments and Precipitation

In most conditions, sources of strontium come from the underlying geology, which then becomes bioavailable through soil leach and then further into the foodchain via plants and animals. Maritime climates with high precipitation, however, evince another pattern. In coastal areas and maritime environs saturated with rainfall (e.g. Western Britain), the strontium isotope ratios of precipitation and atmospheric seaspray is sourced from seawater (fixed at 0.7092), and will influence the strontium isotope ratios of soil, plants, and animals, yielding a mixed $^{87}Sr/^{86}Sr$ ratio between geologically derived and seawater end members (Montgomery et al. 2003; Montgomery and Evans 2006; Bentley 2006; Montgomery et al. 2007). Unlike carbon and nitrogen isotopes, consumption of marine proteins do not significantly contribute to the strontium isotope ratios in human tissues, however if marine foods and/or plants were used as fertiliser for terrestrial crops, the subsequent human strontium signature of plants will be altered (Montgomery et al. 2007).

(II) Atmospheric Dust

Atmospheric dust from Aeolian settlements, including sands, silt, loess, and volcanic ash have varying effects on bioavailable strontium and their presence is heavily dependent on a multitude of factors, including precipitation, topography, forest density, etc. (Bentley 2006; Montgomery 2010; Frei and Price 2012). Depending on the climatic circumstances and wind direction, the accumulation of atmospheric dust from afar can bear a heavy influence over the strontium composition in local vegetation (*ibid*). Apart from evidence in Greenland ice cores, however, there is a paucity of information regarding the long-term accumulation of atmospheric dust and how it contributes to the strontium constituency of the soils, and therefore more research is warranted (Lupker et al. 2010). If noteworthy climatic events such as volcanic eruptions occurred during an individual's life and they lived within a region that may be affected by these deposits, it

may serve as an alternative explanation for incongruent strontium isotope ratios within a dataset.

(III) FERTILISER

Modern fertilisers contain significantly higher strontium isotope ratios that may create stark differences between the $^{87}Sr/^{86}Sr$ ratios of geological and bioavailable strontium (Bentley 2006; Frei and Frei 2011). Böhlke and Horan (2000) first reported that soils with $^{87}Sr/^{86}Sr$ ratios similar to seawater values may become enriched through fertilisation, however, in their survey of strontium isotopes from Danish surface waters, Frei and Frei (2011) found that the influence of modern fertilisers was minimal. Although the influence of modern fertilisers may not bear great influence over bioavailable strontium sources in the past, it may affect any modern floral and faunal samples that are used to develop local baselines (Bentley 2006). This again underscores the need to use a variety of modern and archaeological sources to develop a robust baseline that accurately reflects locality.

(IV) DIET

There is a general presumption that individuals in the past consumed primarily local, terrestrial foods. In modern contexts, this is certainly not the case as items like bottled water and substantial quantities of foodstuffs are imported from various places around the world, which in turn affects modern sampling techniques and interpretations (Coelho et al. 2017). In the past, the majority of foodstuffs would have been derived locally for most archaeological populations, however, if an individual did consume imported food, or subsisted heavily on a marine diet, their $^{87}Sr/^{86}Sr$ ratios could be significantly altered (Sealy et al. 1991; Burton and Wright 1995; Wright 2005; Price and Gestsdottir 2006; Slovak et al. 2009; Montgomery 2010). Sealy and colleagues (1991) first noted the dietary influence over the $^{87}Sr/^{86}Sr$ ratios of human bone, particularly the effect of marine diets over human strontium isotope compositions. For humans subsisting on marine resources or using large quantities of sea salt, their $^{87}Sr/^{86}Sr$ ratios will more reflective of diet rather than underlying geology (Sealy et al. 1991; Wright 2005; Price and Gestsdottir 2006; Slovak et al. 2009). Similar to the influence of seaspray on strontium isotope ratios, the

consumption of these marine sources can shift $^{87}Sr/^{86}Sr$ ratios to that of seawater values. For coastal or island populations, it is therefore best practice to develop local baselines on the human data itself (Wright 2005; Montgomery et al. 2007).

Because human enamel is the preferred tissue for measuring $^{87}Sr/^{86}Sr$, it is important to consider the biocultural influences that would have influenced tooth development, including breastfeeding and weaning. Deciduous or early developing permanent teeth (e.g. first and second incisors, canines, and first molars) will reflect the bioavailable strontium during crown mineralisation, including breastmilk if the individual was breastfed (Sillen and Smith 1984; Mays 2003; Bentley 2006; Nehlich et al. 2009; Slovak and Paytan 2011). If the individual has moved during the breastfeeding period, or is weaned on foodstuffs significantly different from their mother's or wetnurse's diet, an individual may yield an anomalous $^{87}Sr/^{86}Sr$ ratio for population, or that is inconsistent with ratios from their other teeth (*ibid*). For example, Nehlich et al. (2009) measured the strontium isotope ratios of Neolithic peoples in modern-day Germany for evidence of expansion and settlement. The individuals that were shown to be of non-local origins mainly comprised the children, including several infants. The disparity between the radiogenic strontium isotope ratios of their burial location vs. their tissues was interpreted as reflecting a maternal influence from breastfeeding, although peculiarly, there were no females of childbearing age represented in the burial population leading the researchers to believe that this was a special burial place, or that the children were taken unaccompanied by their mothers (*ibid*). Because imported foodstuffs, marine diet, and to some extent fertilisers, can skew modern faunal data, it is crucial to sample both modern and archaeological sources of bioavailable strontium in order to create a more precise range for humans and consider these environmental and anthropogenic factors in any consequential interpretations.

3.11.3 Strontium metabolism in humans

The metabolism of bioavailable strontium is complex and multifactorial, but in general, an individual's strontium isotope composition is a direct reflection of diet. Strontium is subsumed into the body as a non-nutritive trace element through the ingestion of food,

water, and aerosols (Dahl et al. 2001; Nielsen 2004; Bentley 2006; Montgomery 2010). Because of their similarity in chemical properties and atomic radius, strontium readily substitutes for calcium in the crystalline structures of hydroxyapatite and is therefore found primarily in calcified structures (i.e. bones and teeth); although in significantly less quantities (i.e. only 0.035 of its calcium content) (*ibid*). Strontium is customarily absorbed via the gastrointestinal tract (through the jejunum), placental transfer, and/or breastmilk; widely disseminated throughout the body through protein binding in blood plasma or serum; and is preferentially excreted (relative to calcium) through the renal system (Nielsen 2004). The strontium composition of the human skeletal and dental bioapatites reflect a broad average of all dietary components (*ibid*). The body's strontium composition is mainly derived from vegetables and cereals, and undergoes a process of biopurification where the ratio of Sr/Ca decreases by a factor of five (~20%) up each trophic level (Burton and Wright 1995; Nielsen 2004; Bentley 2006). However, in addition to high calcium intake, protein-rich diets will also cause the preferential excretion of strontium, therefore it is not an adequate reflection of trophic level (Burton and Wright 1995). Consequently, individuals with higher plant intake tend to show higher Sr/Ca ratios, and individuals with higher meat intake, show lower Sr/Ca ratios. Further, because the majority of strontium from animal sources is found within bone, and humans don't customarily consume bones, the variance of strontium concentrations within the human body is considerably narrower in comparison to herbivores or primary producers (Burton and Wright 1995; Bentley 2006; Montgomery 2010). Strontium concentrations are also relatively similar throughout skeletal tissues, with enamel yielding slightly higher concentrations than bone and dentine (Montgomery 2010). It is thought that the concentrations of strontium, measured in parts per million (ppm), also vary geographically, however whether that variability stems from geological or bioavailable strontium sources remains unanswered (*ibid*). 'Normal' strontium concentrations in animals and humans range from approximately 50-300 ppm, with herbivores yielding higher concentrations due to their subsistence strategy (*ibid*:328).

3.11.4 Strontium ratios in enamel: biomineralisation and developmental timings

Most archaeological studies using strontium isotope ratios sample tooth enamel due to its resistance to diagenetic change. Strontium is incorporated into enamel from the bloodstream during the ameloblastic secretion and maturation phases (Müller et al. 2019). Strontium isotope ratios and concentrations reflect a composite of the dietary strontium intake during the timings of the enamel biomineralisation process, which occurs over a number of months-years in a multi-directional prismatic formation (Fincham et al. 1999; Reid and Dean 2006; Müller et al. 2019; Figure 3.12). Because enamel biomineralisation is not strictly linear or appositional, Montgomery and Evans (2006) recommend the use of bulk sampling from core enamel for the measurement of $^{87}Sr/^{86}Sr$ in order to obtain a dietary average over the course of enamel development. Sequential microsampling along the *striae of Retzius* to measure changes in strontium isotope ratios has been suggested as a means of showing intratooth variation linked to diet and movement (Dolphin et al. 2005; Richards et al. 2008), however Müller and colleagues (2019) were able to show a consistent decrease in the Sr/Ca ratios of modern and archaeological samples between the enamel secretion and maturation phases, demonstrating that intratooth variability along the *striae of Retzius* is more to do with discrimination against Sr (relative to Ca) during the biomineralisation process rather than cultural factors.

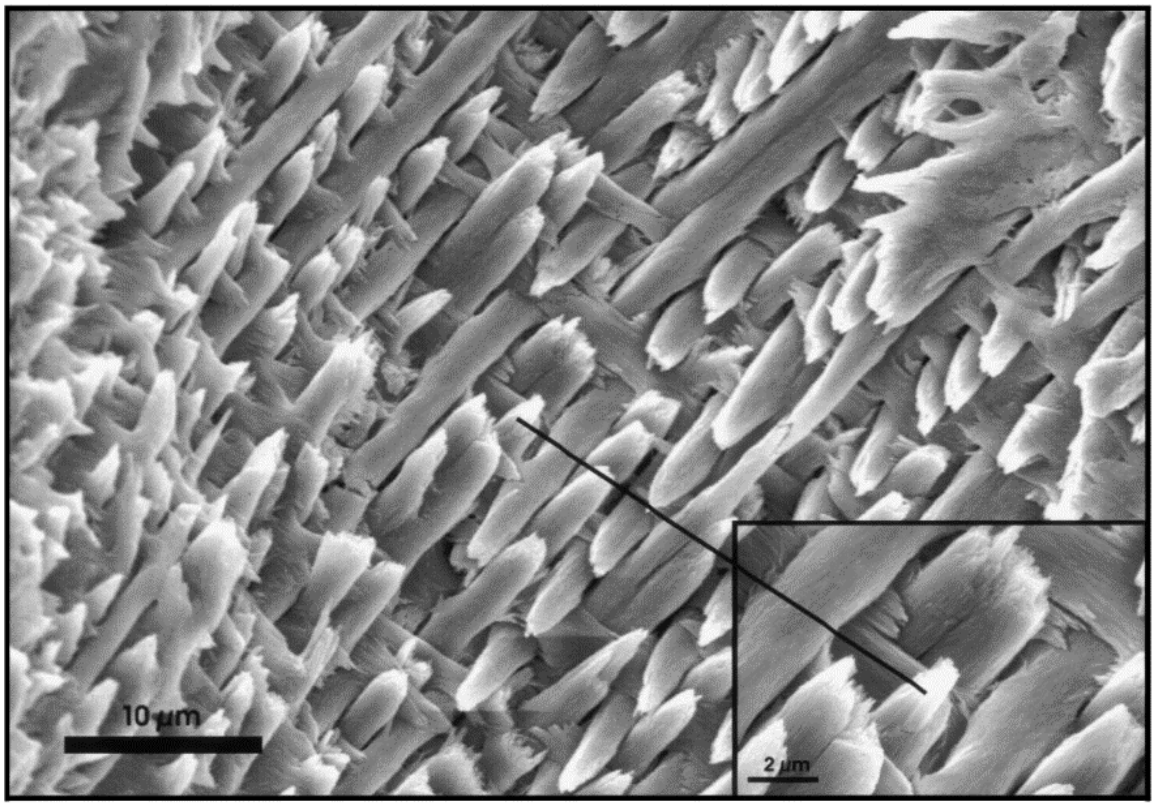

Figure 3.12 Biomineralisation of enamel highlighting a prismatic structure during formation (Fincham et al. 1999:271). Reproduced with permission from Elsevier (License 4983031281283).

The period of time a bulk enamel sample will represent is dependent on the tooth type. All deciduous begin to form in utero, with some crowns completing before birth (AlQahtani et al. 2010; Table 3.6). With the exception of the deciduous second molars, the $^{87}Sr/^{86}Sr$ ratios of deciduous teeth will almost exclusively come from placental transfer. The earliest permanent tooth crown that begins to mineralise is the first permanent molar (c. 4.5 months after birth), and will present of composite of $^{87}Sr/^{86}Sr$ from breastmilk and local foodstuffs if the individual is being breastfed/weaned. It is worth noting that the growth trajectories of children are much more expeditious in earlier years, which may reduce the time periods in which enamel subsumes its strontium isotope ratios and concentrations (Dahl et al. 2001; Neilsen 2004). After enamel secretion and maturation, enamel will not remodel and will therefore reflect an average strontium isotope ratio confined to the crown's developmental timeline (Table 3.6). Depending on the research question, specific teeth can be targeted for sampling to

investigate different time periods within an individual's life. It should always be borne in mind, however, that the methods for all isotopic analyses are destructive and sampling strategies should be devised to maximise data whilst minimising destruction.

TABLE 3.6 – Median developmental timings of deciduous and permanent dental crowns (data compiled from Moorrees et al. 1963; AlQahtani et al. 2010).

MANDIBLE		**MAXILLA**	
TOOTH	**DEVELOPMENTAL TIMING OF ENAMEL CROWN**	**TOOTH**	**DEVELOPMENTAL TIMING OF ENAMEL CROWN**
DECIDUOUS 1ST INCISOR	c. 28 weeks in utero – birth	Deciduous 1st incisor	c. 28 weeks in utero – 1.5 months
DECIDUOUS 2ND INCISOR	c. 28 weeks in utero – 1.5 months	Deciduous 2nd incisor	c. 28 weeks in utero – 4.5 months
DECIDUOUS CANINE	c. 30 weeks in utero – 10.5 months	Deciduous canine	c. 30 weeks – 7.5 months
DECIDUOUS 1ST MOLAR	c. 28 weeks in utero – 7.5 months	Deciduous 1st molar	c. 28 weeks in utero – 6 months
DECIDUOUS 2ND MOLAR	c. 30 weeks in utero – 10.5 months	Deciduous 2nd molar	c. 30 weeks in utero – 7.5 months
PERMANENT 1ST INCISOR	6 months – 3.5 years	Permanent 1st incisor	4.5 months – 4.5 years
PERMANENT 2ND INCISOR	7.5 months – 3.5 years	Permanent 2nd incisor	10.5 months – 5.5 years
PERMANENT CANINE	9 months – 5.5 years	Permanent canine	7.5 months – 6.5 years
PERMANENT 1ST PREMOLAR	2 years – 6.5 years	Permanent 1st premolar	2.5 years – 6.5 years
PERMANENT 2ND PREMOLAR	3.5 years – 6.5 years	Permanent 2nd premolar	3.5 years – 6.5 years
PERMANENT 1ST MOLAR	4.5 months – 3 years	Permanent 1st molar	4.5 months – 3.5 years
PERMANENT 2ND MOLAR	2.5 years – 8 years	Permanent 2nd molar	2.5 years – 8 years
PERMANENT 3RD MOLAR	8.5 years – 14 years	Permanent 3rd molar	8.5 years – 14 years

3.12 STRONTIUM AND OXYGEN ISOTOPE ANALYSES IN PALAEOPATHOLOGY

Isotope-based studies to elucidate human movement, and the impetuses for it, are increasingly becoming a major focus in bioarchaeological research (Dupras and Schwarcz 2001; Price et al. 2002; Bentley 2006; Montgomery 2010; Pederzani and Britton 2019). Radiogenic strontium and stable oxygen isotope analyses from human tooth enamel have the potential to determine whether an individual spent their childhood locally in relation to the surrounding area where they were buried and relative to other members of the burial population. Conventionally, archaeologists have used these isotope systems to

study mobility within and between populations, but in combination with palaeopathological analyses, greater potential exists to investigate relationships between disease, trauma, and movement (Richards and Montgomery 2012). Migration is a 'potent' factor in the origin and evolution of disease and trauma, and is heavily imbued with concepts of social identity; societal, cultural, and economic development; and individual and global health (Wilson 1995:39; Carballo 1998; Mascie-Taylor and Krzyżanowska 2017). Despite this, little research has focussed on the interrelatedness of the mobility histories of people in the past and palaeopathology, partly because of the consequential limitations to modelling both (Richards and Montgomery 2012; Reitsema and Holder 2018).

Although not disease-specific, much of the literature looking at migration and palaeopathology focusses on 'otherness' through studies of conflict. Tung and Knudson (2008) examined 31 trophy heads from the Wari Empire (600-1000 AD) in Conchopata (Peru) in order to view the origins of the crania in comparison to local peoples. They found that almost all of the trophy heads belonged to adult males, with a high percentage (42%) exhibiting perimortem cranial trauma. Further isotopic analysis on five individuals indicated that at least three of the trophy heads likely belonged to nonlocals perceived as enemies (*ibid*). Migration can also be the cause or result of intergroup and intragroup strife, leading to executions and mass graves. Chenery and colleagues (2014) conducted strontium and oxygen isotope analyses on 10 decapitated skulls as a subset of 51 individuals discovered in a Saxon-Viking mass grave (10^{th} - 11^{th} centuries AD) in Weymouth (UK). Their results showed that the group was nonlocal to Britain, and were likely members of a Scandinavian Viking raiding party that was captured and executed locally (*ibid*). Excavations of the Driffield Terrace site in York (UK) also revealed unusual Roman period (1^{st} - 4^{th} centuries AD) execution cemeteries, with more than half of its all-male burials showing evidence of decapitation (Muldner et al. 2011; Montgomery et al. 2011). Results of two different strontium and oxygen isotope studies on 24 individuals from Driffield Terrace show that the majority of individuals (n=17) are non-local to the area, and their all-male composition may have been due to a punitive military (or possibly gladiatorial) connection (*ibid*). In pre-Hispanic Central California (6^{th}- 16^{th}

centuries AD), Eerkens and colleagues (2016) used similar techniques to analyse the remains of seven unrelated men with perimortem trauma buried in a collective grave, concluding that although they were not immediately local, they were perhaps from an adjacent raiding party in competition over neighbouring territory. In the Neolithic period, mobility isotope analyses on multiple mass graves evince contrasting patterns. Strontium and oxygen isotope analyses on Neolithic (5200-4800 BC) mass graves from central Europe (Germany and Austria) reveal the local communities were on the receiving end of perimortem trauma, possibly due to exogenous raids (Bentley et al. 2008; Teschler-Nicola 2012).

One of the obvious links in viewing disease and mobility in the past is elucidating and accounting for the spread of infectious diseases, as infections rely on some form of contact between humans to spread to other geographical areas, whether it be via trade, war, as a result of leisure opportunities, or settlement-based (Wilson 1995). However, it is not possible to account for how long someone has lived with a disease, and therefore impossible to verify whether a disease was present within a person before they moved, or if they encountered it in transit or post-migration (Roberts et al. 2013; Kendall et al. 2013; Quinn 2017; Redfern et al. 2018). Therefore, viewing immigration and disease transmission as a cause-and-effect determinant oversimplifies the nature of heterogenous biocultural landscapes and disease ecologies, and can sometimes provide fuel for onerous debates over contact and immigration in the past (e.g. the origins of syphilis; see Powell and Cook 2005).

Further, asserting immigrant status with disease presence can also lead to controversial interpretations. For example, Inskip and colleagues (2015) presented an interesting case-study on an Early Medieval (5^{th} – 6^{th} century AD) individual with leprosy from Great Chesterford (Essex, UK). In their study, they proposed the individual was of non-local origins and possessed a strain of leprosy (3I) that had been previously identified in Denmark and Sweden, therefore positing that he was perhaps an individual of Scandinavian origin that brought the disease to Britain. However, the individuals from Denmark and Sweden found with similar leprosy strains were from notably later contexts

(c. 12th - 14th centuries AD), and while the Great Chesterford individual's strontium and oxygen isotope ratios ($\delta^{18}O_P$ = 16.6‰, $^{87}Sr/^{86}Sr$ = 0.7088 to 0.7101) could feasibly be obtained in some areas of Denmark, they are also wholly consistent with the Great Chesterford region (expected $\delta^{18}O_P$ = 17.2‰, ±1.3 2σ; $^{87}Sr/^{86}Sr$ = 0.7080 and 0.7100). This more likely indicates that the 3I leprosy strain was present in Britain for c. 800 years prior to the Scandinavian comparative data.

Researchers can, however, view disease at the case study or population level to challenge past conceptions about social identity and disease or employ a thematic approach to reveal big picture changes in time and space. One of the earliest examples of connecting disease to mobility and social identity, was the 'hare-lipped priest' published by Müldner and colleagues (2009:1130). Excavations of individuals from Whithorn Priory (Scotland) revealed that one individual (SK8) buried amongst the clerics, possibly with ecclesiastical artefacts, possessed a cleft-palate and subsequent isotope analyses revealed that he had spent his childhood local to the area (*ibid*). This study showed that although biblical passages forbade people with visible disabilities from entering the priesthood, that local custom and social identity superseded biblical scripture. Commingled remains from the Byzantine monastery of Mount Nebo, Jordan (4th – 7th centuries AD) also revealed an individual with a unique developmental abnormality (Kesterke and Judd 2019). The skull of an adult male displayed changes consistent with Paget's disease of bone (PDB), with strontium and oxygen isotope analyses indicating that he spent his childhood in the Mount Nebo region (*ibid*). The aetiology of PDB is currently unknown, but current hypotheses point to a combination of gene variants and a slow viral infection, and this study provides evidence for the earliest case of PDB in the Middle East, thereby helping to track its presence over time (*ibid*). The unique burial of a young female with paraplegia was found within a Bronze Age tomb at Tell Abraq (UAE), with skeletal changes consistent with paralytic poliomyelitis (Schrenk et al. 2016). In this instance, isotope analyses indicated that she arrived at Tell Abraq after the age of 15, and that her immigration into the region put her at increased immunological risk to a disease she would have otherwise encountered in childhood (*ibid*). In this instance, the authors were able to use the pathogenesis of the disease to support their interpretation, in that

exposure to polio normally occurs in infancy and those exposed to the virus later in life go on to develop paraplegic disabilities (*ibid*). Further, if the young female buried at Tell-Abraq had developed paraplegia previous to migrating to the region, movement over a large desert landscape would be less likely (*ibid*). This understanding of disease processes integrated with mobility isotope analyses help towards integrating past lifeways and social processes at a larger level. Thematic approaches beyond case studies also help to signify the importance of biocultural frameworks within multi-isotope studies.

Despite being fraught with difficulty and a multitude of considerations, the intersections of pathology, mobility, and social identity can help towards a better understanding of past experiences of disease at the sociocultural and ecological levels. One of the earliest studies to look at population-level movements and its links to disease was conducted by Chenery and colleagues (2010) who used a multi-isotope approach to look at a Roman period (2nd century AD) mass burial pit in Gloucester. Their study found considerable dietary and mobility diversity within the sample, and concluded that because the individuals had perished indiscriminately, that they likely encountered a catastrophic disease event; e.g. the plague (*ibid*). Kendall and colleagues' (2013) study on a known Medieval period (14th century AD) Black Death mass burial yielded similar results, highlighting the extent to which highly virulent pathogens can change and shape socio-political climates and demography. Another multi-isotope study by Turner and Armelagos (2012) looking at pathological links to differences in diet and childhood at Machu Picchu (Peru) revealing that primary factors that may have contributed to skeletal lesions associated with anaemia were origin-based, rather than a result of nutritional deficiency; i.e. those who originated from the arid coasts showed a significant difference with regard to early metabolic health status, but not diet. In contrast, the Early Medieval Bowl Hole cemetery (7th - 9th centuries AD) at Bamburgh Castle in Northumbria (UK), revealed a majority of individuals from all ages and sexes to have childhood origins consistent with Scandinavia, the Southern Mediterranean, and North Africa (Groves et al. 2013). When examining nonspecific lesions of childhood stress with residential origins, Groves and colleagues (*ibid*) found that those local to the site and the UK in general, revealed more indicators of poorer health in childhood, as opposed to the immigrant populations. At the

outset, this suggests that immigrants may have possessed better childhood nutritional health than those who grew up in the UK. However, it always must be borne in mind that the aetiologies of skeletal lesions are complex and multifactorial, and may in fact represent the 'healthier' individuals as non-survivors would have died before these pathological lesions had time to manifest (Wood et al. 1992). It is also important to keep in mind that most diseases leave no trace on the skeleton, and that burial contexts can further aid in the understanding of intersections of sociocultural identity, disease, and mobility. Gregoricka and colleagues (2017) studied individuals buried at a post-Medieval (17^{th} – 18^{th} centuries AD) cemetery site in Poland, where six individuals showed a 'deviant' anti-vampire mortuary treatment, but yielded no obvious pathological skeletal lesions. They examined strontium ratios and oxygen values from enamel samples and found that although afforded a deviant burial treatment, they were members of the local community. This outcome led Gregoricka and colleagues to conclude that their post-mortem sociocultural identity was likely imbued by a disease process that was not reflected in the skeleton (e.g. cholera, tuberculosis, or another pathological condition that created 'otherness' in the Drawsko community) (*ibid*). These studies highlight the interrelatedness and dynamic nature of disease ecologies and social identity on overall health and mobility.

Very little research has been done to link evidence for specific infectious diseases and mobility histories using radiogenic strontium and oxygen stable isotope analyses. Roberts and colleagues (2012) were the first to analyse individuals buried at the Late Medieval (14 - 16^{th} centuries AD) Augustinian Friary cemetery at Hull Magistrates' Court (UK) who had skeletal lesions consistent with treponematoses (tertiary-quaternary syphilis) to to examine the extent of mobility in individuals who died with a specific, communicable disease. Their study looked at enamel samples from 12 individuals (six showing skeletal lesions consistent with syphilis, six without skeletal lesions - controls) and found 4/12 individuals likely originated from outside the UK, two of which had lesions consistent with treponematosis, ultimately concluding that disease status is not demonstrably linked to immigration (*ibid*). In her doctoral study, Quinn (2017) looked at the mobility histories of 21 individuals from Roman period (1^{st} – 5^{th} centuries AD) sites in England

(York, Winchester, Baldock, Easington, Cirencester, and Poundbury) who possessed bone changes consistent with tuberculosis. Quinn found that about one-third (6/21) of the individuals with bone changes consistent with tuberculosis spent their childhoods outside of their respective burial regions, but concluded that linking mobility with disease transmission is challenging due to its multifactorial nature, and that larger, more encompassing studies in future were necessary to move big picture interpretations forward (*ibid*). Redfern and colleagues (2018) crafted a holistic approach to viewing migration and health-statuses in 151 individuals with isotope data. They took a multifactorial statistical approach to viewing differential disease categories and mobility histories, and found lesions associated with non-specific infections and metabolic deficiencies markedly increased from the Iron Age to the Romano-British period, due to an influx of people from the wider Roman Empire creating more diversity in population composition (*ibid*). They also acknowledged the nature of disease in their interpretations - not as a static component associated with a migratory event (e.g. Roman invasion), but as something that affects communities bidirectionally, insofar that every community is its own heterogeneous disease environment that will respond to, and affect, populations invariably (*ibid*). This big picture approach using strontium and oxygen isotope values and palaeopathological data demonstrated large cultural changes without sacrificing the nuances of unique human migratory and disease experiences, and presents a robust framework for exploring disease and mobility for future research.

3.13 Summary and Future Directions in Isotope Analyses and Palaeopathology

This chapter reviewed the basic principles of carbon, nitrogen, oxygen, and strontium isotope systems; the complex ways in which they interact with human metabolic processes and how these processes are affected by pathophysiological processes; and ways in which archaeologists have viewed radiogenic and stable isotopes in bioarchaeology and palaeopathology. Over the past 40 years, huge methodological and subsequent theoretical leaps have been achieved, but currently stable isotope analyses are at the precipice of further theoretical advances; namely the integration and

intersections of physiological variance, social identity, and disease ecologies. In order to keep this exciting momentum in motion, several future directions will aid in helping this field further develop.

Very few studies of pathology and its effect on carbon and nitrogen stable isotopes have been conducted with a view to the past. Previously, this was partly due to large issues of equifinality, but the development of isotope analysis of incremental dentine has broadened the scope to look how disease affects the body isotopically through lived experiences. Better ascertainment of the timings of bone lesions (i.e. when they occurred in a person's life), in conjunction with variances in carbon and nitrogen incremental profiles, will aid in the understanding of carbon routing and nitrogen recycling within the human body. Elucidating the effects of specific pathological processes on carbon and nitrogen metabolism would be another huge step forward. By looking at incremental tissues using both clinical studies and archaeological examples, some of the quagmires present in the interpretation of carbon and nitrogen may also be better resolved. For example, by studying diseases or conditions that involve cachexia and bone wasting (e.g. atrophy/disuse, cancer, leprosy, tuberculosis, etc.) using incremental tissues in clinical settings and with archaeological comparatives may yield more concrete patterns of carbon and nitrogen variability over time and different pathogeneses.

With regard to radiogenic strontium and stable oxygen isotope analyses, issues of disease timing in the body and manifestation in the skeleton can prove difficult if focussing on factors of disease transmission. However, big picture studies of health and disease ecologies could provide a more beneficial way to view links between palaeopathological evidence and the mobility of people in the past. Redfern and colleagues (2018) have introduced a multifactorial research framework that looks at overall population health and mobility, without foregoing or fixating too much on individual-level health-status. Studies like this can be further expanded to look at migrant vs. local disease risks through the intersections of early life stress, mobility, disease susceptibility, sex, age, and early mortality. Incorporating further data to view large-scale movements through space and time, and the development of user-friendly platforms, such as BGS' Biosphere Isotope

Domains GB, or open-access databases such as IsoArcH, highlight the potential for the combination of larger data sets with disease dynamics, which in turn could reveal major palaeoepidemiological patterns.

Employing multi-isotope systems in conjunction with one another could also facilitate a more holistic view of past responses to disease at the biological and social levels. By viewing isotope systems through the lenses of specific disease processes, resultant data could help to identify allostatic thresholds and shape disease landscapes. For example, using carbon and nitrogen isotope values from incremental dentine profiles could help distinguish if a person experienced a pathophysiological disruption prior to, or subsequent to, moving to a specific region. Moreover, the fluidity of movement and subsequent funerary treatment in the face of disease could help contextualise the sociocultural responses to biological and pathogenic processes.

3.13.1 Conclusions and Interpretive Considerations

This chapter has discussed the application of basic principles, methods, key theories, and specific studies of radiogenic and stable isotopes in bioarchaeology, including in palaeopathological studies. The nexus between these specialties within the broader frameworks of archaeology and palaeopathology shows great potential, and provides key areas that show particular promise if developed further. These new developments that allow researchers to delve into individual life histories and physiological processes are not without further interpretive considerations and acknowledgments. One major consideration is addressing the need for large-scale intersectional and collaborative studies to view biological and social phenomena in the past using radiogenic and stable isotope analyses as a major, but not sole, line of evidence. Humans are not a closed system and by incorporating more intersectional bioculturally driven approaches in isotope analyses, interpretations can be suggested that minimise the over-interpretation of data, while accounting for biological and social change. Isotope analyses cannot reveal the long-term biological and social impacts of disease on a society on their own, and must always be contextualised, which includes a widespread acknowledgement of the fact that we are using the remains of people that have died. Therefore the influence of

pathophysiological processes should be at the forefront of isotope data interpretations. If these considerations are applied to new methodological and theoretical approaches, and exercised with caution so as to ethically justify and not 'over sample' human remains, the potential to make major contributions to our understanding of disease in the past and its longitudinal effects on the present are boundless.

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CHAPTER 4. MATERIALS AND METHODS

4.1 INTRODUCTION

This chapter provides information about the skeletons accessed for this research and the subsequent methods employed for analysis. The Materials section (4.2) gives a brief summary of the sites chosen for study, including their historical and archaeological contexts, and the excavated skeletons, with a breakdown of basic osteobiographical information related to the individuals selected for sampling for destructive analyses. The subsequent Methods section (4.3) details the specific procedures and techniques used to generate the data, including osteological and palaeopathological methods used to estimate age at death, biological sex, and disease, the Index of Care framework as a clinical evidence-based care model, radiogenic and stable light isotope methods for mobility and dietary information, amelogenin peptide methods to identify ambiguously sexed individuals, and the summary statistics and graphical representations used to interpret results.

4.2 MATERIALS

4.2.1 BROAD ARCHAEOLOGICAL CONTEXT

This section provides a brief background for the two archaeological sites used for this study, as well as for the individuals selected for study from these sites. The sites chosen for this research were the St. Mary Magdalen leprosarium's North Cemetery in Winchester, Hampshire, England and the St. John at the Castle Gate/ Timberhill parish cemetery in Norwich, Norfolk, England (Figure 4.1). Both sites were major administrative centres in Early-Late Medieval England (Shepherd Popescu 2009: 49; Ayers 2011; Lavelle and Roffey 2015:18), and were broadly contemporaneous during the construction and usage of the cemeteries (i.e. 9^{th} – 12^{th} centuries AD). These sites were specifically chosen due to their excavations revealing human skeletal remains with pathological skeletal lesions diagnostic of leprosy, as well as for their contextual differences (i.e. leprosarium cemetery vs. a parish cemetery).

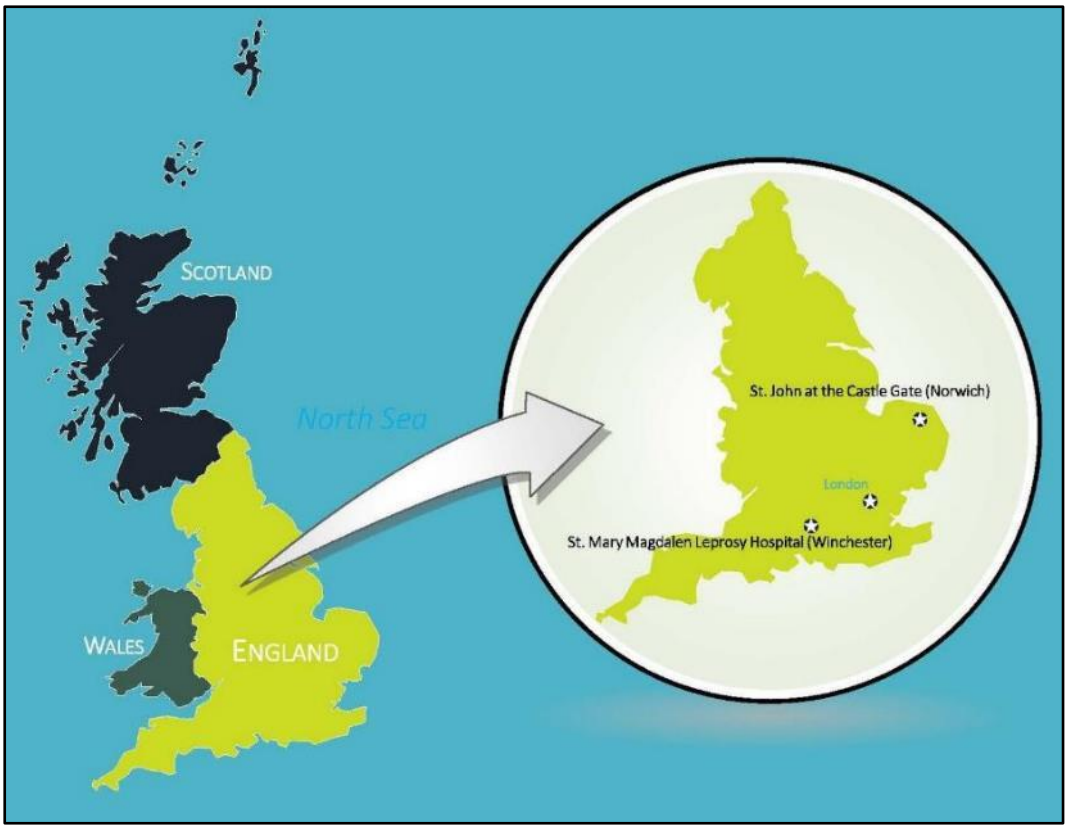


Figure 4.1 Location map of the sites selected for study in England, with London marked for geographical reference. Author's own.

4.2.2 St. Mary Magdalen leprosarium (Winchester, Hampshire, England)

(i) Location

The site of the St. Mary Magdalen leprosarium lies on Magdalen Hill alongside major roadways to London, approximately one mile east of the city of Winchester, England (Roffey and Marter 2012). Winchester is located on the South Downs where the local geology is dominated by Cretaceous Chalk and deposits of Oligocene and Eocene sands, clays, silts, and gravel found in the south of the site, and a small area to north, all within a 10km radius. There is also Gault Clay and Upper Greensand formations located approximately 25km to the east of the site on the western margin of the Weald (British Geological Survey 2007; Figure 4.2).

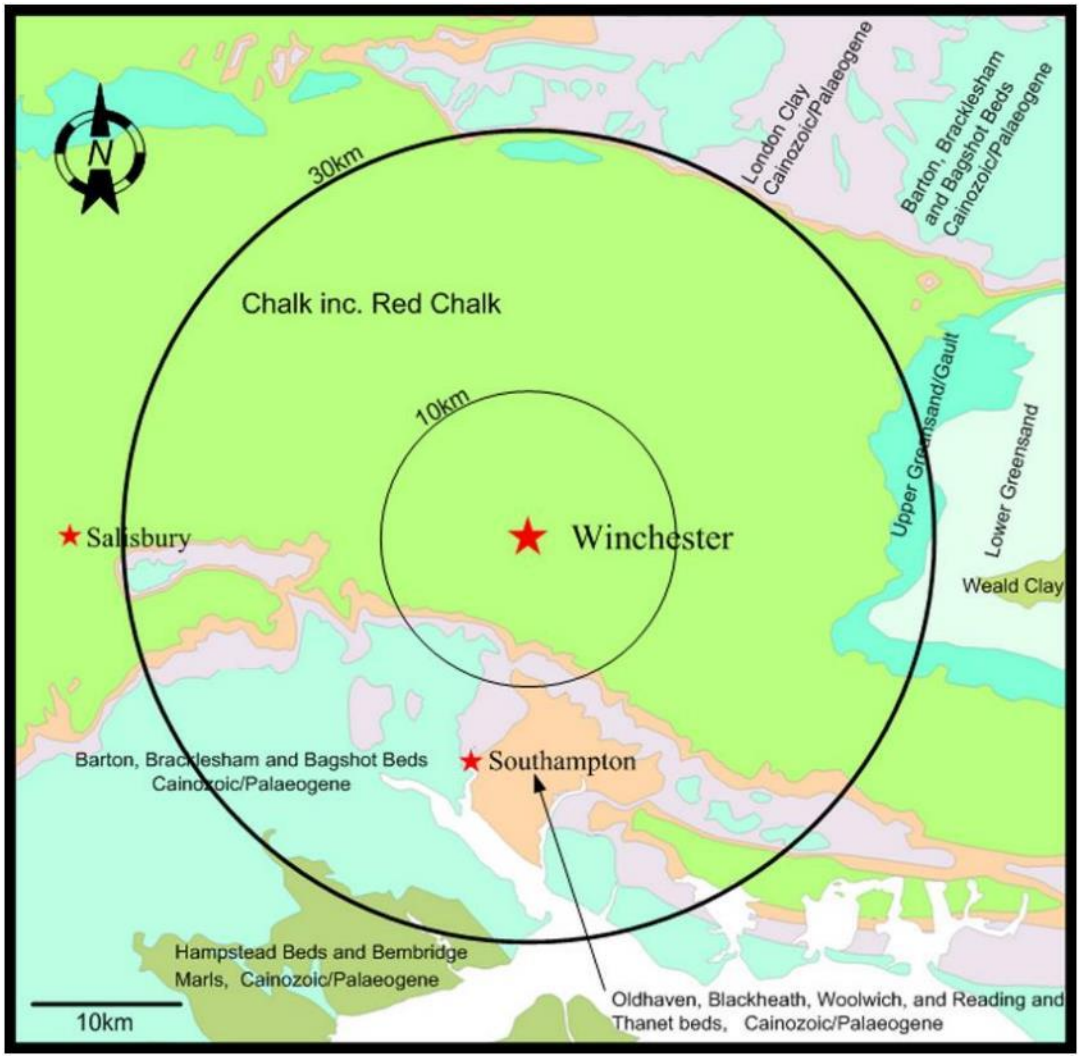


Figure 4.2 Location of Winchester and underlying bedrock geology (Eckardt et al. 2009:2818). Reproduced with permission from Elsevier (License 4983051239898).

(II) EXCAVATIONS

The excavations of St. Mary Magdalen leprosarium reveal a long and varied history. the UK TV station's Channel Four's Time Team first investigated the site in the year 2000, focusing on the later part of the site's history, which in turn laid the foundation for a more in-depth excavation project of the leprosarium. Larger excavations of the leprosarium and its subsequent post-Medieval structures began in 2008 by a team from the University of Winchester and are ongoing. These excavations have yielded several discrete burial sites, including distinctly separate cemeteries: the North Cemetery, and the South Cemetery.

Figure 4.3 Aerial view of the excavations of the St. Mary Magdalen leprosarium. The North Cemetery is located in the centre, and underlies most of the later masonry phases. Courtesy of Dr. Katie Tucker.

Within the North cemetery (Figure 4.3), 38/44 individuals showed skeletal indicators of leprosy (~86%), of which approximately 53% were adolescents (Table 4.1). These individuals were buried with their heads to the west and arranged within anthropomorphic graves containing head niches and earthen pillows (Roffey and Tucker 2012). According to Roffey (2012), this particular manner of inhumation was normally reserved for high-status ecclesiastical burials. Other clues for social identity were also discovered within the burial contexts, such as a pilgrim badge buried with Sk. 27 from the shrine of St. James in the Santiago de Compostela Cathedral in Spain (Figure 4.5), and modified, flanged feeding bowls buried with Sk. 19 who exhibited the most advanced bone changes of lepromatous leprosy found at the site (Roffey and Tucker 2012; Roffey et al. 2017). The careful construction and arrangement of the burials and included burial

Figure 4.4 Sk. 27 in situ with associated scallop shell signifying his pilgrimage to the St. James Shrine at the Santiago de Compostela Cathedral in Spain (Roffey et al. 2017: https://doi.org/10.1371/journal.pntd.0005186. Open Access).

goods are unusual for leprosaria cemeteries (e.g. Chichester) and may support a level of care and compassion not often associated with these contexts (Roffey and Tucker 2012).

In contrast, the South Cemetery shows a different burial alignment that corresponds to the orientation of the mid-12th century masonry foundations, and comprises more haphazard burial arrangements (e.g. multiple or truncated burials) (Roffey 2012; Roffey and Tucker 2012).

TABLE 4.1 - Individuals from the North cemetery with skeletal evidence for leprosy. Data are given as reported by Roffey and Tucker (2012) and Tucker (2012).

INDIVIDUAL	AGE	SEX	BONES AFFECTED BY LEPROSY
SK. 7	26-35	Male	Facial bones, Feet, Legs
SK. 8	9-11	Male*	Facial bones, Hands, Legs
SK. 9	19-25	Male	Hands, Feet, Legs
SK. 14	16-18	Male?	Facial bones, Hands, Feet, Legs

SK. 15	19-25	Male	Hands, Feet, Legs
SK. 16	19-25	Male	Facial bones, Feet, Legs
SK. 18	14-19	Male?	Facial bones, Hands, Feet, Legs
SK. 19	25-30	Male	Facial bones, Hands, Feet, Legs
SK. 20	46+	Female	Hands, Feet, Legs
SK. 21	19-25	Male?	Facial bones, Hands, Feet, Legs
SK. 22	'Adult'	?	Feet, Legs, No skull or hands
SK. 23	26-35	Male	Hands, Feet, Legs, No skull
SK. 24	46+	Female	Facial bones, Hands, Feet, Legs
SK. 25	17-19	Male	Facial bones, Hands, Feet, Legs
SK. 26	19-25	Male	Facial bones, Feet, Legs
SK. 27	25-30	Male	Feet, Legs
SK. 28	13-16	?	Facial bones, Feet, Legs
SK. 29	19-25	Male	Facial bones, Hands, Feet, Legs
SK. 30	?	?	Feet, Legs, No skull
SK. 31	45+	Female	Facial bones, Hands, Feet, Legs
SK. 32	General Adult	?	Hands, Feet, Legs, No skull
SK. 36	26-35	Ambiguous	Facial bones, Feet, Legs
SK. 37	13-16	?	Feet
SK. 38	19-25	Female?	Feet, Legs
SK. 39	19-25	Male	Facial bones, Feet, Legs
SK. 40	26-35	Male	Facial bones, Feet, Legs
SK. 41	13-16	?	Facial bones, Feet, Legs
SK. 42	26-35	Male	Facial bones, Hands, Feet, Legs
SK. 43	46+	Female	Hands, Feet, Legs

SK. 45	13-17	?	Facial bones, Feet, Legs
SK. 46	16-19	Male	Facial bones, Feet, Legs
SK. 48	36-45	Male	Facial bones, Feet, Legs
SK. 49	26-35	Male	Feet, Legs
SK. 52	13-15	?	Facial bones, Hands, Feet, Legs
SK. 53	26-35	Male	Facial bones, Feet, Legs
SK. 54	13-15	?	Facial bones, Hands, Feet, Legs
SK. 55	26-35	Male	Feet, Legs
SK. 56	16-18	Male	Facial bones, Hands, Feet, Legs

**Sex attributed through aDNA analysis (Taylor et al. 2013).*

Documentary evidence from the Winton Domesday first references St. Mary Magdalen as a functioning leprosarium in 1148, but the North Cemetery and several structures underlay 12th century AD masonry foundations suggesting that the leprosarium was functioning prior to the Winton Domesday reference (Roffey and Marter 2012; Roffey and Tucker 2012; see Figure 4.3). These structures include a small masonry chapel with timber outbuildings (*ibid*). The construction of new masonry structures in the 12th century overlying the earlier contexts would have effectively decommissioned the North cemetery, thereby confining the North cemetery to the Early-Late Medieval transition (Roffey 2012). Calibrated radiocarbon dates from the St. Mary Magdalen leprosarium site corroborate these archaeological findings, showing several pre-Norman conquest and transitionary dates for the North Cemetery, and a firm post-Norman date for the South Cemetery (Table 4.2). This evidence therefore identifies St. Mary Magdalen as the oldest leprosarium site in England (Roffey and Tucker 2012; Roffey and Marter 2012).

TABLE 4.2 - Calibrated radiocarbon dates and SNP types for individuals buried at the St. Mary Magdalen leprosy hospital site.

INDIVIDUAL	CEMETERY	CAL. ^{14}C DATE (95% CI)	SNP TYPE	REFERENCE
SK. 8	North	AD 1010-1160	2F	Roffey and Tucker 2012; Taylor et al. 2013
SK. 9	North	AD 890-1040	N/A	Roffey 2012; Roffey and Marter 2012

SK. 14	North	AD 995-1033	2F	Schuenemann et al. 2013; Taylor et al. 2013
SK. 27	North	AD 1020-1162	2F	Roffey et al. 2017
SK. 2	South	AD 1268-1283	3I	Schuenemann et al. 2013
SK. 5	Chapel	AD 1290-1410	N/A	Roffey and Tucker 2012

Further aDNA analyses of leprosy genotypes using single-nucleotide polymorphisms (SNP) reveal the presence of two separate leprosy SNP types (2F, 3I) within the North Cemetery during this time (Roffey and Tucker 2012; Roffey and Marter 2012; Taylor et al. 2013; Schuenemann et al. 2013; Roffey et al. 2017; Schuenemann et al. 2018). SNP type 3I likely originated from Central Asia and has previously been identified in individuals from Early Medieval contexts (6^{th} – 11^{th} centuries AD) within England, Sweden, Hungary, Czechia, and Turkey (Inskip et al. 2015; Inskip et al. 2017; Donoghue et al. 2015; Economou et al. 2013; Schuenemann et al. 2013). This SNP type is thought to be the type responsible for almost all European cases (both ancient and modern) (Monot et al. 2009; Economou et al. 2013). SNP type 2F is thought to be a precursor strain that travelled from the Middle East to India and Southeast Asia (Monot et al. 2009; Economou et al. 2013; Schuenemann et al. 2013; Taylor et al. 2013; Mendum et al. 2014; Schuenemann et al. 2018). 2F was previously identified within Italy (6^{th} – 8^{th} centuries AD), Scandinavia (12^{th} – 14^{th} centuries AD), and Ireland (12^{th} – 13^{th} centuries AD), and is largely interpreted as evidence of broad geographical trade networks (Economou et al. 2013; Mendum et al. 2014; Schuenemann et al. 2018; Taylor et al. 2013). The presence of both of these SNP types within the St. Mary Magdalen leprosarium therefore may also reinforce 'Anglo-Scandinavian' links and links further afield within Winchester during this time.

By the 14^{th} century AD, the building was partly rebuilt and it is believed that during this time the site ceased to function as a leprosarium. Remains recovered from the Chapel during the earliest part of this restructuring reaffirm this cut-off date and appropriation as a parish site (1290 – 1410 calAD; 95% probability) (Roffey and Tucker 2012; Table 4.2). Archaeological evidence suggests complete disuse as a hospital by the 15^{th} century AD, with the site repurposed into an almshouse in the 16^{th} century AD. This was followed by the use of the site as a military base during the English Civil War (1642-1651), a prison for

Dutch prisoners during the 17th century AD, and a First World War base (Roffey and Marter 2012; Roffey and Tucker 2012).

4.2.3 St. John at the Castle Gate/ Timberhill (Norwich, Norfolk, England)

(i) Location

The location of the St. John at the Castle Gate/Timberhill cemetery site lies under the modern-day Castle Mall Shopping Centre, immediately southwest of the gates of Norwich Castle (Shepherd Popescu 2009). The local geology of Norwich (c. 30km) lies at the confluence of two underlying bedrock geologies. The site is characterised by Cretaceous chalk to the immediate south and to the west, surrounded by undifferentiated Neogene-Pleistocene gravels, sands, and clay sediments (Figure 4.5; British Geological Survey 2007). The city of Norwich itself is bordered by Mousehold Heath to the north-east, and the St. John at the Castle Gate/Timberhill cemetery site itself lies at the end of the Ber Street ridge to the south, which is underlain by both the gravels, sands, and clays of the Norwich Crag Formation and the Beeston chalk (*ibid*).

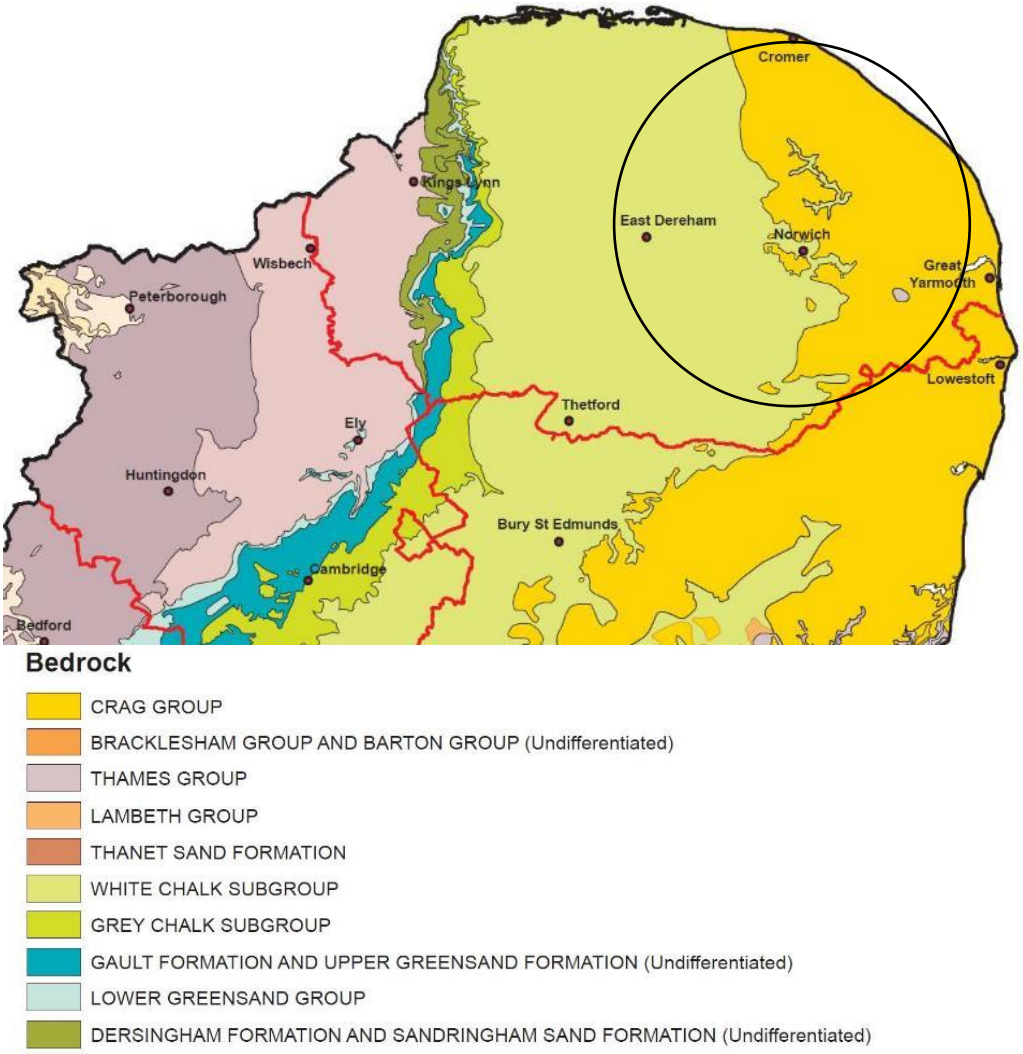


Figure 4.5 The local geology of Norwich within a 30 km radius. Modified from Geo-East: www.geoeastevents.org.uk. Public Domain.

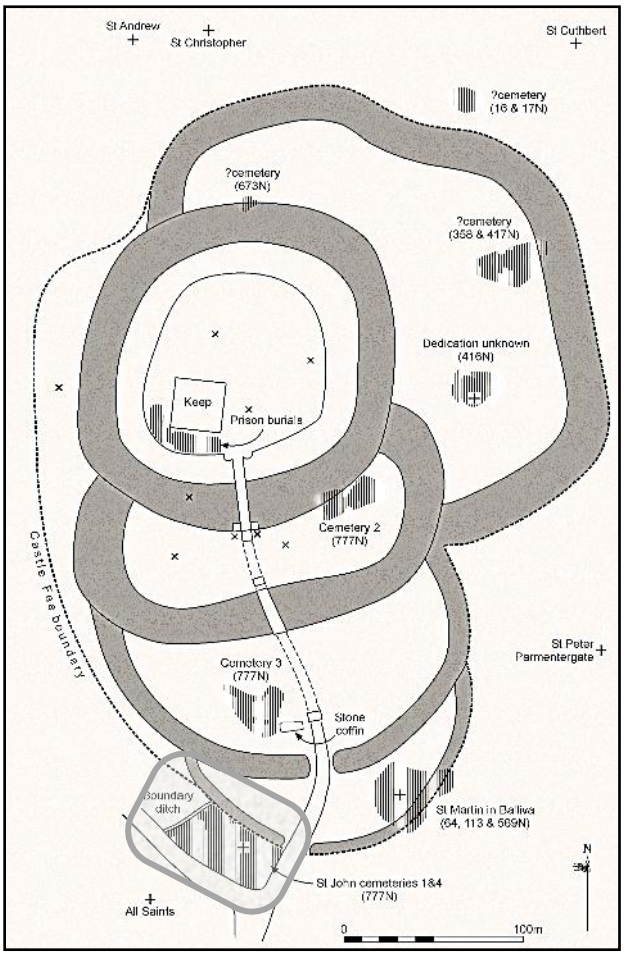


Figure 4.6 Plan showing St. John the Baptist Church and cemetery location. Modified from Norfolk Archaeological Unit 2009: https://doi.org/10.5284/1000173 - Open Access.

(II) EXCAVATIONS

Norfolk Archaeological Unit undertook major excavations of the Castle Mall structures from 1987-1991 (Shepherd Popescu 2009: 1-17; Norfolk Archaeological Unit 2009; Figure 4.6). During the course of these excavations, evidence for the earthworks of both a Norman period castle and late Saxon town were discovered. One of the areas of focus was the St. John at the Castle Gate church and parish cemetery, which was estimated to have an 11th century AD date based on stylistic similarities to other structures with known dates (*ibid*). The earliest documentary mention of this site was in AD 1157 in the *Liber Cartarum et Placitorum*, which noted a church called (Ecclesia) Sancti Johannis ante portam Castelli or 'St John at the Castle Gate', but an extant structure was most likely in place before (Shepherd Popescu 2009: 267-268; Popescu 2016: 8). The church in its

current incarnation remains standing and is now referred to as St. John the Baptist/Timberhill (Figure 4.7). Although at least six cemeteries were discovered during the excavations at Castle Mall, the cemetery associated with St. John at the Castle Gate church (Cemetery 4) yielded a significant number of individuals (34% of the observable burial population) with skeletal signs of leprosy (Anderson 1996; Anderson 2009: 228-231; Shepherd Popescu 2009: 268-271).

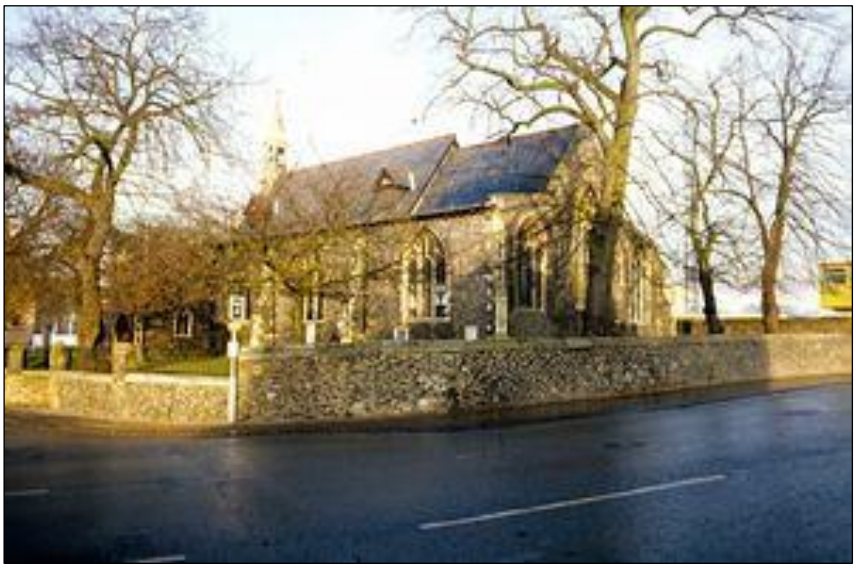


Figure 4.7 The Church of St. John the Baptist/Timberhill as it stands today (Norfolk Archaeological Unit 2009: https://doi.org/10.5284/1000173. Open Access).

Excavations of the St. John at the Castle Gate/Timberhill cemetery revealed the remains of at least 149 adults and 35 non-adults buried in a 'fan shape' (Shepherd Popescu 2009:124; Figure 4.8). The burials were aligned south-west to north-east in the west of the cemetery, east-to-west at the centre, and north-east to south-west in the east (Norfolk Archaeological Unit 2009; Figure 4.8). Skeletal analyses of the human remains from this site reported that 35/184 individuals possessed skeletal lesions consistent with leprosy (Anderson 1996; 2009: 228-231). Out of these 184 individuals, 92 had foot bones, 101 had hand bones, and 103 had facial bones available for assessment, and only 48 individuals had all three areas preserved (*ibid*). Of the 35 individuals that showed skeletal lesions consistent with leprosy, 24 (23.3 % of the observable population) were reported

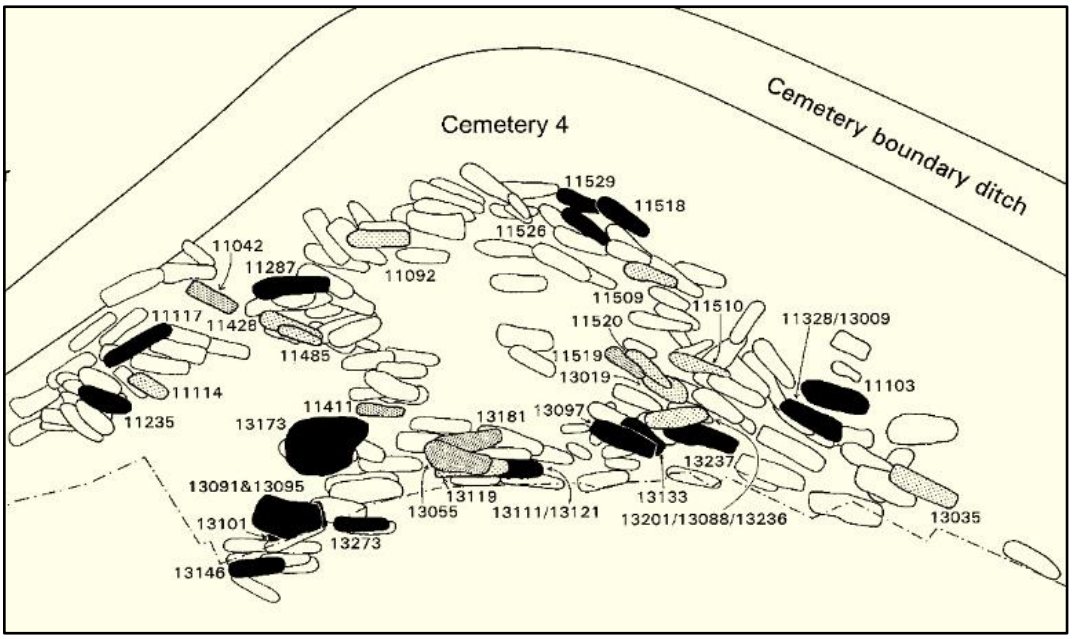


Figure 4.8 Schematic layout of burials of Cemetery 4 at the Church of St. John at the Castle Gate/Timberhill. Burials in black denote skeletons with skeletal lesions diagnostic of leprosy and shaded burials represent possible leprosy. Modified from Norfolk Archaeological Unit 2009: https://doi.org/10.5284/1000173. Open Access.

to have skeletal lesions pathognomonic of lepromatous leprosy (Anderson 2009: 228-231).

TABLE 4.3 - Individuals buried at St. John at the Castle Gate/Timberhill with skeletal evidence of leprosy (Anderson 1996; Anderson 2009:228-231). Data are given as reported by Anderson (1996; 2009).

INDIVIDUAL	SEX	AGE	BONES AFFECTED BY LEPROSY
10189/11440	F	30-35	Facial bones, Feet, Legs, No hands
11042	F	Adult	Hands, no other elements
11092	F?	30-35	Facial bones, Hands, No feet
11103	F	18-30	Facial bones, Feet, Legs
11114	F?	MA	Facial bones, No other elements
11117	M?	16-20	Facial bones, Hands, Feet, Legs
11235	F	16-25	Facial bones, No feet or legs
11287	M?	18-30	Facial bones, Hands, Legs, No feet
11328/13009	X	12-16	Facial bones, Legs
11411	?	16-20	Feet, Legs, No other elements
11428	M?	Adult	Legs, No hands or skull
11485	?	Adult	Feet, Legs, No other elements
11509	M?	Mature to older adult	Feet, Legs, No other elements
11510	M?	18-30	Hands, Feet, Legs, No skull
11518	X	12-16	Facial bones, Hands, Feet
11519	F	Adult	Hands, Feet, Legs, No skull
11520	M	Mature	Hands, Feet, Legs, No skull

11526	X	15-18	Facial bones, Legs, No other elements
11529	M	Adult	Feet, Legs, No other elements
13019	X	15-16	Feet, Legs, No other elements
13035	F	20-30	Facial bones, Hands, Legs, No feet
13055	X	16-19	Facial bones, Feet, Legs
13091	X	15-16	Hands, Feet, Legs, No skull
13095/13082	X	?	Facial bones, No other elements
13097	F	Adult	Facial bones, Feet, Legs
13101	X	13-16	Facial bones, Feet, Legs, No hands
13111/13121	F	18-30	Facial bones, Hands, No feet
13119	F?	25-30	Facial bones, Hands, Feet, Legs
13133	?	16-20	Feet, No other elements
13146	M?	20-23	Facial bones, Feet, Legs
13173	F	Mature adult	Hands, Feet, Legs, No skull
13181	M	31-49	Facial bones, Legs
13201/13088/13236	M?	Mature adult	Facial bones, Feet, No hands
13237	F?	20-25	Hands, Legs, No other elements
13273	?	Mature adult	Facial bones, No other elements
13044*	M	18-25	Facial bones, Hands

**Present in Anderson 1996, but absent in Anderson 2009*

Radiocarbon dates from 18 individuals from St. John's indicated that the cemetery was in use for a limited time period with a start date of cal AD 980-1030 (95% probability) and a terminus of cal AD 990-1050 (95% probability), representing one or two generations who lived just before the Norman Conquest in 1066AD (Bayliss et al. 2004; Bayliss et al. 2009:243). Bayliss and colleagues (2004) further suggested the internment period may have occurred in the post-Norman period due to a potential marine reservoir effect reflected in elevated carbon and nitrogen isotope ratios derived from bone collagen. However, several assumptions in Bayliss and colleagues' (2004) study created a greater margin of interpretive error for these dates. These include targeting samples from bones without signs of leprosy, with the assumption that the absence of pathological changes in bone equated to the absence of pathological influences on isotope ratios or within the body (Bayliss et al. 2004: 568). This is a misunderstanding of the complex interactions between metabolism and infection (Roberts and Brickley 2018), as well as the degree of precision needed when accounting for dietary particulars in bone collagen (Beaumont et al. 2018). Additionally, despite extensive evidence of pig remains associated with the site, the consumption of pork was discounted from consideration as a possible contributive factor to the isotope ratios due to an assumption that people with leprosy would not be afforded good quality meat (Bayliss et al. 2004:571), to which contrasting evidence does

exist (Rawcliffe 2006:213-225; Roffey et al. 2017). In spite of these overlooked factors, subsequent re-evaluation by Bayliss and colleagues (2009) stated that overall knowledge about the dietary effects on the correction for radiocarbon measurements rendered their offset interpretations unreliable, which favoured the original 10th – 11th centuries dates for the cemetery (246).

Of particular note is the archaeological context associated with the St. John's cemetery. St. John at the Castle Gate/Timberhill lies within the boundaries of the developing town, and was considered a parochial cemetery rather than a leprosarium cemetery (Anderson 1996; Anderson 2009: 231; Popescu 2016:8). The high number of individuals with leprosy represented (c. 34% of the observable burial population) is the highest for any Medieval parish cemetery not associated with a leprosarium (*ibid*). Of further significance is that a notable number of the individuals with skeletal lesions consistent with leprosy are adolescents (>40%), similar to the St. Mary Magdalen leprosarium in Winchester (Figure 4.8). Anderson (1996) hypothesizes that St. John's may have been the local parish for people living with and without leprosy, indicating a level of care and tolerance in the local community for people with the disease during this time. Many of the burials within the St. John at the Castle Gate cemetery had earthen pillows to support the heads or feet, flints supporting the skulls, and were buried in wooden coffins or above beds of pebbles (Shepherd Popescu 2009: 266-269). These burial practices show parity with the nearby, largely contemporaneous Farmer's Avenue cemetery (890 - 1060 cal AD), suggesting that St. John's could have been used as an overflow cemetery for Farmer's Avenue at the end of the 10th century AD (*ibid*). Additionally, analyses of non-metric skeletal traits in both cemeteries suggest a familial link between Farmer's Avenue and St. John at the Castle Gate, which may indicate a shift of the Farmer's Avenue church and cemetery to the St. John's parish due to expansion of Norwich Castle (Anderson 2009:235-236).

However, the shift towards St. John's as a consequence of leprosy cannot be ruled out (Anderson 2009:236). Although there is a lack of associated infirmary structures, both the location of St. John's at the Castle Gate and its access to a secure freshwater source is mirrored in leprosaria after the expansion of the Norwich Castle walls in the 12th century AD (Rawcliffe 2006: 312-313; Shepherd Popescu 2009: 269). It could therefore be that St.

John at the Castle Gate may have served as an early leprosarium community, and any associated timber buildings could have been destroyed by later building works. In the post-Conquest period, at least five leprosaria were constructed at each of the city's entrances (Figure 4.9), which may also indicate that leprosy was common and increasing in caseload in Norwich during the Early-Late Medieval transition (Anderson 1996; Shepherd Popescu 2009: 268-271).

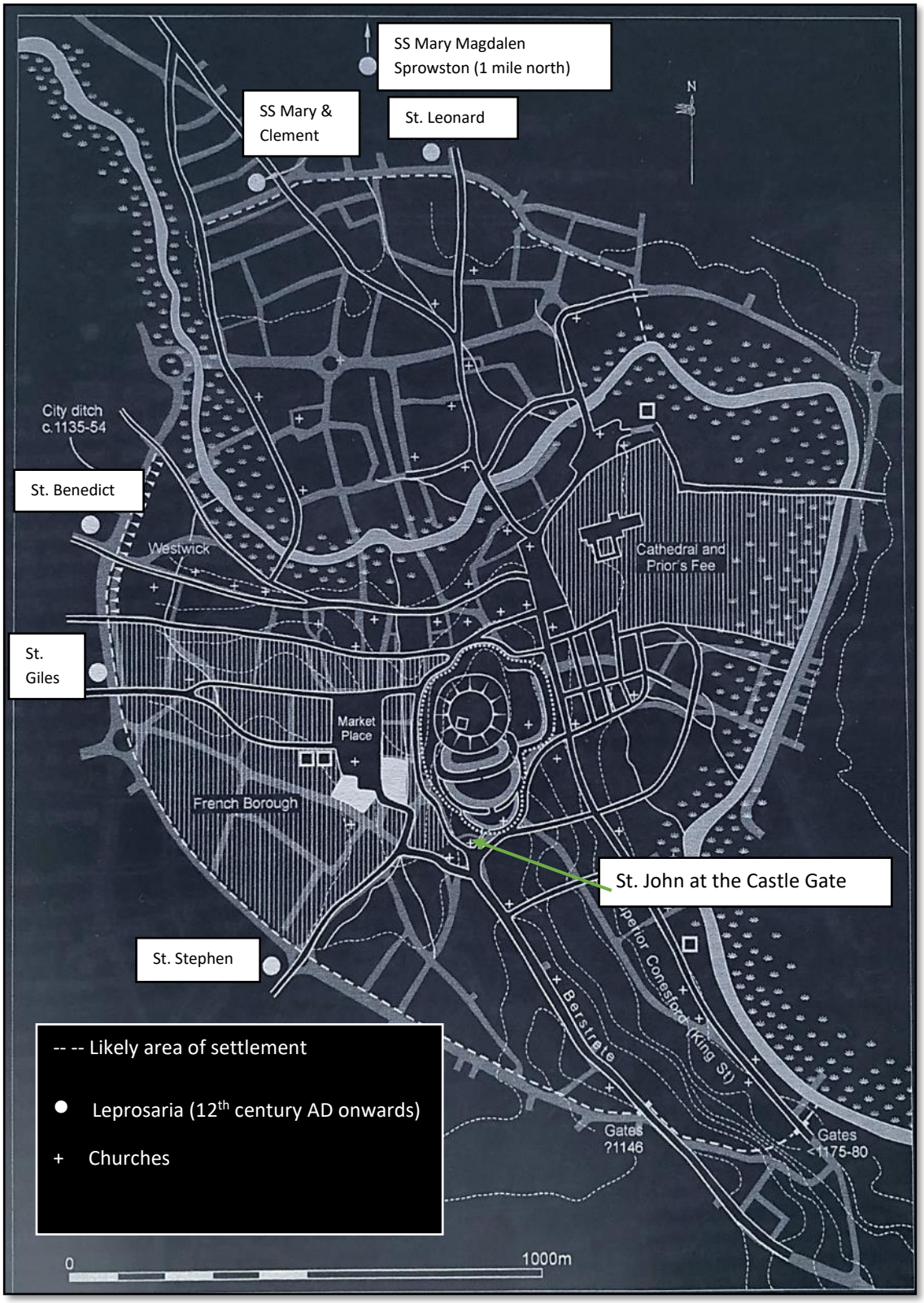


Figure 4.9 St. John at the Castle Gate/Timberhill in relation to other leprosaria surrounding the city from the 12th century AD. Modified from Popescu 2016: 17.

4.2.4 Individuals Selected for Study and Sampling Strategies

To assess the biological and social impacts of leprosy on individuals in Medieval England, this research strategically focussed on adolescents with leprosy due to their high representation at both sites (>40%), their unique social status in the Medieval period, their general underrepresentation in bioarchaeological studies (Gilchrist 2012: 34-37; Cochelin 2013; Lewis 2016), and the potential for their dental tissues to provide complete life history data (from <1 year to death). In order to identify these individuals, the published and unpublished site reports (Anderson 1996; Tucker 2012) from the St. Mary Magdalen leprosarium site and St. John at the Castle Gate/Timberhill were consulted and a list of 36 skeletons from both sites that met the selection criteria was compiled for biomolecular analysis.

These criteria included:

- the individuals showed diagnostic signs of lepromatous leprosy,
- they possessed teeth for analysis,
- and they died before their third molars were fully formed in order to capture full life history data (c. birth to death).

Permissions with ethical justifications were sought and obtained in writing from the curators of the human skeletal remains to analyse the skeletons from these sites, and to take two tooth samples from each individual for subsequent biomolecular analyses. The Codes of Ethics (at the time 2010) and the Codes of Practice (at the time 2010) from the British Association of Biological Anthropologists and Osteoarchaeologists (BABAO)(https://www.babao.org.uk/publications/ethics-and-standards) were strictly adhered to in the sampling from the individuals selected for study. This project also went through the ethical approval process at Durham University. Unfortunately, seven individuals were excluded from St. John at the Castle Gate/Timberhill due to curatorial issues (e.g. missing at the time of request, or osteological data not corresponding to the data in Anderson's (1996) site report; i.e. missing relevant bone elements, older individuals in boxes that were labelled as children, or individuals that had no skeletal

signs of lepromatous leprosy). Ultimately, 29 individuals from the St. Mary Magdalen leprosarium (n=19) and the St. John at the Castle Gate/Timberhill cemetery (n=10) were sampled for this thesis (Tables 4.4, 4.6).

TABLE 4.4 - Brief summary of the sites and individuals selected for study

SITE	ST. MARY MAGDALEN	ST. JOHN AT THE CASTLE GATE/TIMBERHILL
LOCATION	Winchester, Hampshire	Norwich, Norfolk
CEMETERY TYPE	Leprosarium	Parish cemetery
TIME PERIOD	9^{th} – 12^{th} centuries AD	10^{th} – 11^{th} centuries AD
NUMBER OF INDIVIDUALS	19	10
SEX	14 Males 5 Unsexed	4 Males 2 Female 4 Unsexed
AGE RANGES	8.5 – 25	10 – 25

After skeletal and dental development is complete, it becomes more difficult to pinpoint and evaluate particulars about a person throughout the life course, including changes in diet and mobility histories, and metabolic alterations due to pathophysiological stress. Strontium and oxygen isotope analyses can reflect residential locales during the enamel forming period for a given tooth, and carbon and nitrogen stable isotope analyses of incremental dentine can reveal diet and patterns of pathophysiological and nutritional stress at a sub-annual scale until tooth development is complete (Montgomery 2010; Beaumont et al. 2013a; 2013b). With this in mind, the enamel from the most recently developed molar teeth were chosen for strontium and oxygen isotope analyses to reveal whether the adolescent's most recent $^{87}Sr/^{86}Sr$ and $\delta^{18}O$ values were consistent with their burial place, and dentinal collagen from both the canine teeth and most recently developed molars were selected to provide carbon and nitrogen isotope data ($\delta^{13}C$, $\delta^{15}N$) that spans from the first year of life to the point of death.

Although the earliest forming permanent teeth are usually the central incisors, their growth and development were, in some instances, affected by leprosy. Therefore, samples were preferentially taken from a canine (C), and a second (M2) or third (M3)

molar, depending on the age of the individual, i.e. if the individual was not old enough to have developed an M3, an M2 was selected. These teeth respectively develop from 7.5-10.5 months ± 1.5 months to 13-14.5 years ± 6 months, 2.5 ± 6 months to 15.5 years ± 6 months, and 8.5 years ± 6 months to 21-23 years ± 6 months (Moorrees et al. 1963; AlQahtani et al. 2010). The age ranges encompass both the difference between males and females, i.e. female teeth tend to initiate and complete quicker, and the difference between maxillary and mandibular tooth development, i.e. maxillary teeth tend to initiate and complete formation at a slightly quicker rate (*ibid*). The preference for a tooth from the maxilla or mandible was guided based upon which tooth sample would cause the least amount of damage to the tooth (e.g. a loose tooth was selected over a tooth still in occlusion within the alveolar bone), over the position in the mouth. For example, for one individual from St. John at the Castle Gate/Timberhill (Sk.13121), the latest forming molar tooth and canine could not be removed without damaging the bone, so a maxillary first molar (M1) and a second premolar (PM2), which were loose in the individual's box, were chosen as substitutes. The maxillary M1 develops from 4.5 months ± 1.5 months to 12.5 years ± 6 months, and the maxillary PM2 develops from 2.5 years ± 6 months to 15.5 years ± 6 months (Moorrees et al. 1963; AlQahtani et al. 2010).

An additional component of sample selection was the presence of the lepromatous form of leprosy on the skeleton (see Chapter 2). Determining disease presence in human skeletal remains can be complex and misleading due to overlaps in skeletal responses from different diseases and unknown periods of chronicity (Ortner 1991; Wood et al. 1992). Despite the possibility that other individuals within the cemetery may have possessed a less severe/high resistant form of leprosy that did not elicit skeletal changes (e.g. the tuberculoid form), only individuals with pathognomonic, or diagnostic, signs of the disease were selected to avoid including individuals who may not have had leprosy during their lifetimes within the sample (e.g. Figure 4.10). as described in Chapter 2, these pathognomonic signs of lepromatous leprosy include rhinomaxillary changes: widening and remodelling of the nasal aperture, resorption of the anterior nasal spine, abnormally porous and/or new bone formation on the oral and nasal surfaces of the palatal bones, destruction of the inferior nasal conchae and vomer, abnormal porosity and resorption of the alveolar process with potential loss of the anterior maxillary

incisors. These changes are alongside acro-osteolysis and concentric atrophy of the hand and foot bones: destruction and remodelling of the phalanges, metacarpals, and metatarsals, 'knife-edge' or mediolateral remodelling of the metatarsal shafts, 'volar grooving' or resorptive grooves on the palmar surfaces of the hand phalanges caused by flexion contractures, tarsal fusion and dorsal exostoses; and subperiosteal new bone formation on the distal shafts of the tibiae and fibulae (Møller-Christensen and Andersen 1953; Møller -Christensen 1961; 1978; Andersen and Manchester 1987; 1988; 1992; Andersen et al. 1992; Andersen et al. 1994). Although subperiosteal new bone formation on the tibiae and fibulae are common in skeletons showing lepromatous forms of leprosy, its presence without any concomitant changes to the facial, hand, or foot bones was not enough to warrant inclusion for this study due to the multifactorial aetiologies associated with subperiosteal new bone formation alone (Ortner 2003; Klaus 2014; Filipek and Roberts 2018). It is worth mentioning that four Winchester individuals (Sk. 8, Sk. 28, Sk. 52, Sk. 56) with pathognomonic signs of lepromatous leprosy also show (rare) evidence for leprogenic odontodysplasia, which is the concentric constriction and dysplastic development of the anterior maxillary dentition caused by leprosy contraction in early childhood (Danielsen 1968; 1970; Southam and Venkataraman 1972; Reichart 1976; Roberts 1986; Matos and Santos 2013; Jain 2017). The development of leprogenic odontodysplasia and the pathological skeletal lesions associated with lepromatous leprosy are presumed to commence at approximately the same time (Ortner 2008), potentially revealing a more defined chronology for the onset of skeletal changes and the time elapsed before death. A full list of the individuals that met the criteria for sampling is provided below (Table 4.5).

Figure 4.10 Skeleton 28, aged 12.5-13.5 years old, from the St. Mary Magdalen leprosarium exhibiting evidence of rhinomaxillary changes, including 1.) flattening of the nasal bones, 2.) widening and remodelling of the nasal aperture, 3.) resorption of the anterior nasal spine, and 4.) resorption of the maxillary alveolar process. This individual also shows leprogenic odontodysplasia (encircled), or arrested development of the maxillary anterior dentition, which may occur in individuals with the disease.

4.3 Methods

4.3.1 Overview of the methods used

This section details the range of methods used to analyse the individuals selected for study.

4.3.2 Osteological Methods

Skeletons were accessed and analysed at their respective curating institutions: the St. Mary Magdalen at the University of Winchester and the St. John at the Castle Gate/Timberhill at Norwich Castle Mall Museum. The 29 skeletons selected were laid out in standard anatomical position to view skeletal preservation of the bones and teeth and the distribution of pathological lesions. Information for age and sex of the individuals was initially taken from the original site reports (Tucker 2012; Anderson 1996) and verified macroscopically. This was done in order to ensure individuals matched the original descriptions and fell within the age ranges set out in the parameters of this study. Standard osteological methods as recommended by Standards For Recording Human Remains (Brickley and McKinley 2004) and Standards for Data Collection from Human Skeletal Remains (Buikstra and Ubelaker 1994) were employed in the original analyses, and were verified and altered where necessary in this study (see Tables 4.5; 4.6).

(I) SEX ESTIMATION

Sex estimation in bioarchaeology is based on a suite of sexually dimorphic traits that develop in the pubertal and post-pubertal period (Shapland and Lewis 2013; 2014; Lewis et al. 2016; Arthur et al. 2016; Lewis 2018). Selected individuals were first assessed using Shapland and Lewis' (2013; 2014) osteological methods for evaluating pubertal stages (e.g. canine development and iliac crest ossification and fusion), and only individuals who were categorised as having reached or surpassed the 'peak height velocity' (PHV) stage of their pubertal growth spurt were re-assessed for sex. Other osteological techniques for estimating the pubertal stage of an individual were not used due to the effects of leprosy on or around the areas under evaluation (e.g. the hamate, the radius, the phalanges, etc.). For individuals who had achieved or surpassed their pubertal growth spurts, Phenice's (1969) method for examining pubic bone morphology and Acsadi and Nemerskeri's (1970) method for scoring the sciatic notch were used to record the os coxae. Sexually dimorphic characteristics of the skull, including the nuchal crest, the mastoid process, the supraorbital margin, the prominence of the glabella, and the mental eminence were scored using the methods outlined in Acsadi and Nemerskeri (1970). If an individual's pelvic and skull morphology was not in agreement, a greater weight was given to the features of the os coxae due to their function in parturition. Following

assessment, individuals were assigned one of the following sex classifications: Male (M); Possible Male (M?); Ambiguous or Indeterminate (i.e. pre-pubertal) (A); Possible Female (F?); and Female (F).

(ii) Age at death estimation

Dental eruption and development stages were recorded using the methods set out by AlQahtani and colleagues (2010; Figure 4.11), in order to ascertain the approximate age at death. Selected canine and molar teeth were extracted manually from either the maxilla or mandible, which allowed the root development stage to be recorded. This made a notable difference and allowed for refinement of the previous age assessments of the individuals selected for study, mainly reducing the age ranges previously reported by Roffey and Tucker (2012) and Tucker (2012), and Anderson (1996). Epiphyseal fusion times based on information in Scheuer and Black (2000) were cross-referenced with the dental eruption and development data to view whether the developmental timings corresponded. Long bone length was not employed as it is of limited use after the age of three (Brickley 2004) due to differential growth trajectories predicated on a combination of genetic and nutritional factors. In instances where the dental development and eruption timings and the epiphyseal fusion timings were incongruous, with no overlap, a special note was made and the dental age was recorded as the more robust age indicator over the skeletal age due to the lessened influence of external factors on dental growth and development when compared to bones (Brickley 2004; AlQahtani et al. 2010).

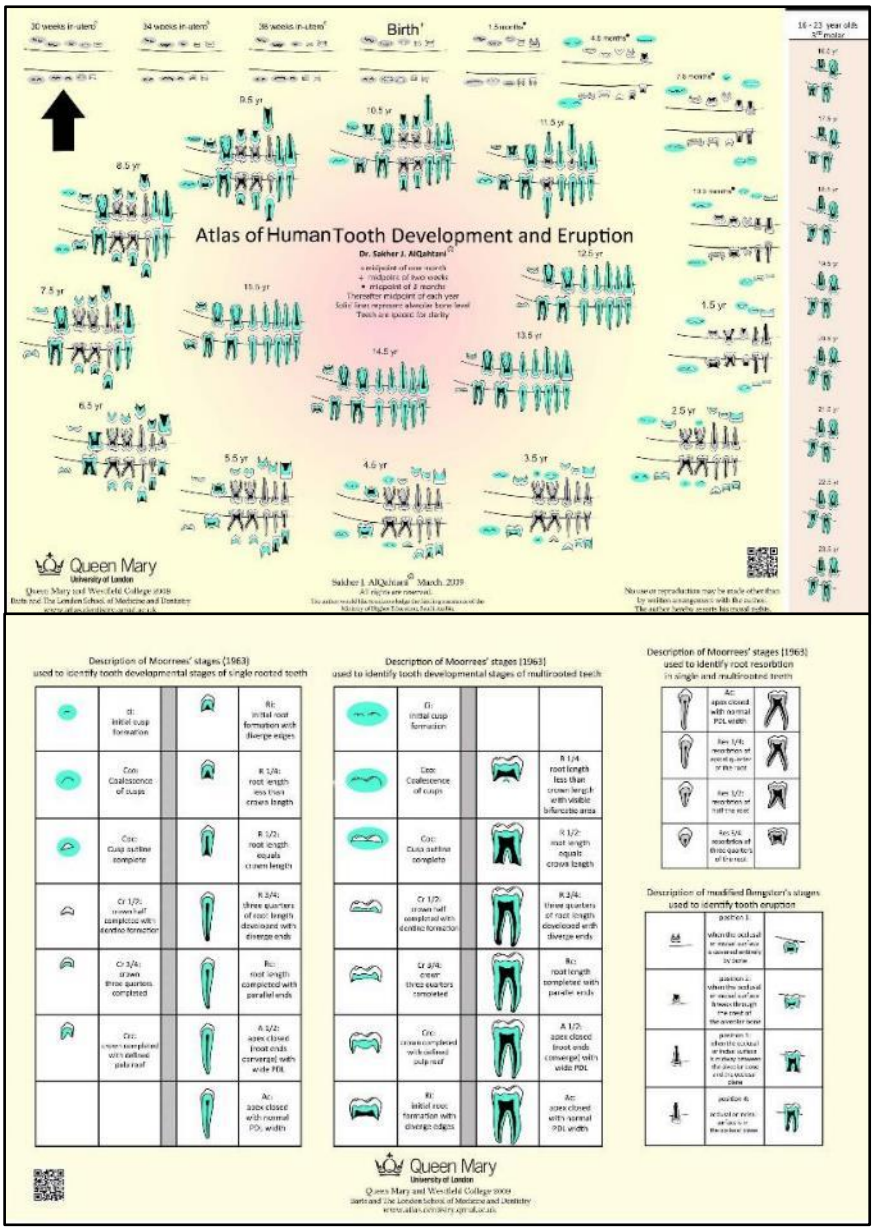


Figure 4.11 Diagrammatic representations of dental development and eruption patterns from AlQahtani 2010. Reproduced with permission from John Wiley & Sons (License 4983071227990).

Individuals who revealed third molars with closed apices were excluded in this research as their biological ages were outside of the scope of this project. Other osteological methods for age estimation such as the degeneration of the auricular surface (Lovejoy et al. 1985), the degeneration of the pubic symphysis (Brooks and Suchey 1990), dental wear (Brothwell 1981), cranial suture closure (Meindl and Lovejoy 1985), and the cartilaginous ossification of the sternal end of the fourth rib (Iscan et al. 1984; Iscan et al.

1985) were not considered for this study as they were developed for assessing the age of individuals once growth and development is completed.

TABLE 4.5 - Individuals and teeth sampled in this study with revised ages and sexes.

SITE	INDIVIDUAL	SEX	AGE (IN YEARS)	TEETH SAMPLED
ST. MARY MAGDALEN	Sk. 8	Male	Tooth formation 8.5-9.5 years; skeletal age 7-8	Maxillary Left Canine Maxillary Left M2
ST. MARY MAGDALEN	Sk. 9	Male	22.5-23.5	Maxillary Right Canine Maxillary Right M2
ST. MARY MAGDALEN	Sk. 14	Male?	16.5-17.5	Maxillary Right Canine Maxillary Left M3
ST. MARY MAGDALEN	Sk. 15	Male	20.5-22.5	Mandibular Left Canine Mandibular Left M3
ST. MARY MAGDALEN	Sk. 16	Male	18.5-21.5	Maxillary Left Canine Maxillary Right M3
ST. MARY MAGDALEN	Sk. 18	Male?	Tooth formation 15.5-16.5; skeletal age inconsistent with premature fusion and unfused bones (<14)	Mandibular Right Canine Mandibular Right M3
ST. MARY MAGDALEN	Sk. 21	Male?	Tooth formation 18.5-22.5; skeletal age 16-19	Maxillary Right Canine Maxillary Left M3
ST. MARY MAGDALEN	Sk. 25	Male	Tooth formation 18.5-20.5; skeletal age 17-19	Mandibular Right Canine Mandibular Right M3
ST. MARY MAGDALEN	Sk. 26	Male	22.5-23.5	Mandibular Right Canine Mandibular Left M3
ST. MARY MAGDALEN	Sk. 27	Male	22.5-23.5	Mandibular Right Canine Mandibular Right M3
ST. MARY MAGDALEN	Sk. 28	Ambiguous	12.5-13.5	Maxillary Left Canine Maxillary Left M2
ST. MARY MAGDALEN	Sk. 29	Male	17.5-19.5	Maxillary Right Canine Maxillary Right M3
ST. MARY MAGDALEN	Sk. 39	Male	16.5-17.5	Maxillary Right Canine Maxillary Right M3
ST. MARY MAGDALEN	Sk. 41	Ambiguous	13.5-15.5	Maxillary Right Canine Maxillary Right M3
ST. MARY MAGDALEN	Sk. 45	Ambiguous	Tooth formation 15.5-16.5; skeletal age 10-12	Mandibular Left Canine Mandibular Left M2
ST. MARY MAGDALEN	Sk. 46	Male	16.5-17.5	Maxillary Left Canine Maxillary Right M3
ST. MARY MAGDALEN	Sk. 52	Ambiguous	12.5-13.5	Mandibular Left Canine Mandibular Left M2
ST. MARY MAGDALEN	Sk. 54	Ambiguous	Tooth formation 14.5-15.5; skeletal age 9-11	Mandibular Right Canine Mandibular Right M2
ST. MARY MAGDALEN	Sk. 56	Male	16.5-17.5	Maxillary Right Canine Maxillary Right M2

ST. JOHN	Sk. 11117	Male	16.5-18.5	Mandibular Right Canine Mandibular Right M3
ST. JOHN	Sk. 11287	Male	22.5-23.5	Maxillary Left Canine Maxillary Left M3
ST. JOHN	Sk. 13009	Ambiguous	14.5-16.5	Maxillary Right Canine Maxillary Right M2
ST. JOHN	Sk. 11518	Ambiguous	Tooth formation 10.5 - 11.5; skeletal age 13-15	Mandibular Right Canine Mandibular Right M2
ST. JOHN	Sk. 11526	Ambiguous	12.5-13.5	Maxillary Right Canine Mandibular Right M3
ST. JOHN	Sk. 13035	Female	18.5-20.5	Maxillary Left Canine Maxillary Left M2
ST. JOHN	Sk. 13044	Male	15.5-16.5	Mandibular Left Canine Mandibular Right M2
ST. JOHN	Sk. 13101	Ambiguous	14.5-16.5	Maxillary Left Canine Maxillary Left M3
ST. JOHN	Sk. 13146	Male	18.5-20.5	Maxillary Right Canine Mandibular Left M3
ST. JOHN	Sk. 13121	Female	18-23.5	Maxillary Left PM2 Maxillary Left M1

TABLE 4.6 - Summary of osteological methods.

METHOD	REFERENCE	CATEGORY
DENTAL ERUPTION AND DEVELOPMENT	Moorrees et al. 1963; AlQahtani et al. 2010	Age
EPIPHYSEAL FUSION	Scheuer and Black 2000	Age
SEXUALLY DIMORPHIC CHARACTERISTICS OF THE OS COXAE	Phenice 1969 in Buikstra and Ubelaker 1994; Acsadi and Nemerskeri 1970	Sex
SEXUALLY DIMORPHIC CHARACTERISTICS OF THE CRANIUM	Acsadi and Nemerskeri 1970	Sex

(III) OTHER OSTEOLOGICAL DATA

Other information, such as metric and non-metric traits, were not collected for this study. These data have been noted in published and unpublished osteology reports by the original scholars who first studied the skeletons (Tucker 2012; Anderson 1996), but this was beyond the focus of this study. Additionally, stature was not calculated as part of this study due to the biological immaturity of the majority of individuals being analysed.

(IV) PALAEOPATHOLOGICAL ANALYSIS

Evidence for pathological conditions and trauma were noted in the individuals selected for study in order to contextualise their lived experiences. This was conducted by comparing abnormal (pathological) changes of bones with their antimeres, if present, as well as normal comparative bones (i.e. non-pathological archaeological and clinical examples). This was done to confirm the changes were abnormal so as not to over-inflate disease presence in the individuals studied. The pathological conditions diagnosed were assigned to eight palaeopathological categories: dental disease (e.g. dental caries, enamel hypoplasia, leprogenic odontodysplasia), joint disease (e.g. Schmorl's nodes, secondary osteoarthritis, spondylosis), infectious disease (e.g. leprosy, tuberculosis, subperiosteal new bone formation possibly indicating inflammation), metabolic disease (e.g. cribra orbitalia, rickets, porotic hyperostosis), congenital disease (e.g. vertebral border shifts, cleft neural arches, etc.), 'other' (e.g. neoplasms, circulatory disturbances, etc.), and trauma (e.g. fractures, amputations, etc.). The abnormal bone changes were then described in detail using recommended terms suggested by Ortner (2012) and Lovell (2000), and following the step-by-step guidance for recording abnormal bone changes in Roberts and Connell (2004; Table 4.7).

TABLE 4.7 - Step-by-step guidance for recording based on Roberts and Connell (2004).

- **Bone or tooth affected and side of the body**
- **Part and aspect of the bone or tooth affected**
- **If the lesion is forming, resorbing, or mixed**
- **The nature of subperiosteal bone formation (e.g. woven, lamellar, or mixed)**
- **Any of evidence of healing around an area of bone resorption or trauma**
- **How the lesion is distributed throughout the body**
- **A list of differential diagnoses for the abnormal bone changes**

Special attention was paid to record any skeletal manifestations associated with skeletal signs of lepromatous leprosy including concentric wasting of the hand and foot bones, leprogenic odontodysplasia, and rhinomaxillary syndrome as described by Møller-Christensen (1961; 1978; see Chapter 2), Andersen and Manchester (1987; 1988; 1992)

and Ortner (2008: 198-206). Disease categories, diagnoses, and differential diagnoses for concomitant pathological changes were compiled by consulting a variety of texts (e.g. Resnick and Niwayama 1995; Ortner 2003; Lewis 2017; Roberts and Manchester 2005; Aufderheide and Rodriguez-Martin 1998). These concomitant changes were considered when interpreting the isotopic data.

4.3.3 Index of Care Methods

The Index of Care framework was accessed online (IndexofCare.org) and applied to one individual (Sk. 19) from the St. Mary Magdalen leprosarium in Winchester. This was to determine whether health-related care within a leprosarium could be supported for this individual. Sk. 19 was specifically chosen from an Index of Care point of view based on their advanced skeletal changes associated with long-term lepromatous leprosy. Due to the unique severity of Sk. 19's skeletal changes, they were not included in the analyses that used destructive methods in this research, but the clinical and functional impacts of their skeletal changes and the broader implications for these at St. Mary Magdalen were worth considering. Sk. 19 was a c. 25 year-old male excavated from the North Cemetery, whose burial context revealed evidence of shrouding (e.g. a copper alloy shroud pin) and pottery vessels adapted for feeding (Roffey and Tucker 2012). The remains of Sk. 19 showed rhinomaxillary changes, such as complete resorption of the maxilla extending back to the first molars, and resorption of the hard palate with porosity of the remaining bone (Figure 4.12).

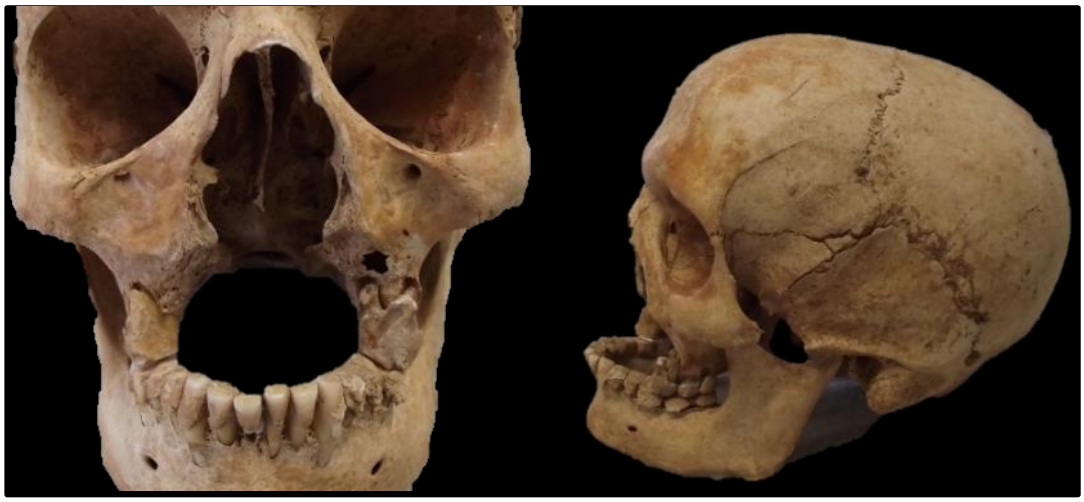


Figure 4.12 Skull of Sk. 19 demonstrating advanced rhinomaxillary changes including widening and fusion of nasal bones, widening and remodelling of the nasal aperture, resorption of the anterior nasal spine and alveolar process of the maxilla, and complete resorption of the hard palate.

The Index of Care framework is part of a 'bioarchaeology of care' methodological approach for identifying and interpreting evidence of health-related care in the past (Tilley and Cameron 2014; Tilley 2017; Tilley and Schrenk 2017). The web-based application (IndexofCare.org) provides a case-specific, multi-staged platform to evidence medical and social responses and practices in the past with a view to establishing a person's likelihood for the need for healthcare provision, defining their level of disability (if any) through the biocultural construction of past lifeways, defining and setting out parameters for health-related care, acknowledging interpretive limitations and recognising that the experience of disability and disease will be individually unique, and comprehending both individual and group agency, i.e. understanding that the provision of health-related care are deliberate, or purposeful, and actions in which both the caregiver and care-recipient participate, and that they exist within a community that facilitates this experience in some way (*ibid*). The success of this model is achieved through the consideration and evaluation of specific clinical aspects of any disease needing or requiring health-related care provision. This is achieved through a cross-disciplinarily (clinical and social) assessment of the nature and impact of disability and the caregiving response, thus providing a cogent manner to order and record evidence, observations, inferences, and interpretations, in a manner that facilitates analysis and

report production. This in turn provides a line of reasoning in the analytical process that can offer a more objective and replicable basis for the interpretation of health-related care provision in the past that has not been possible before (Tilley and Cameron 2014: 5-6).

This is achieved through a four-stage approach (Fig 4.13):

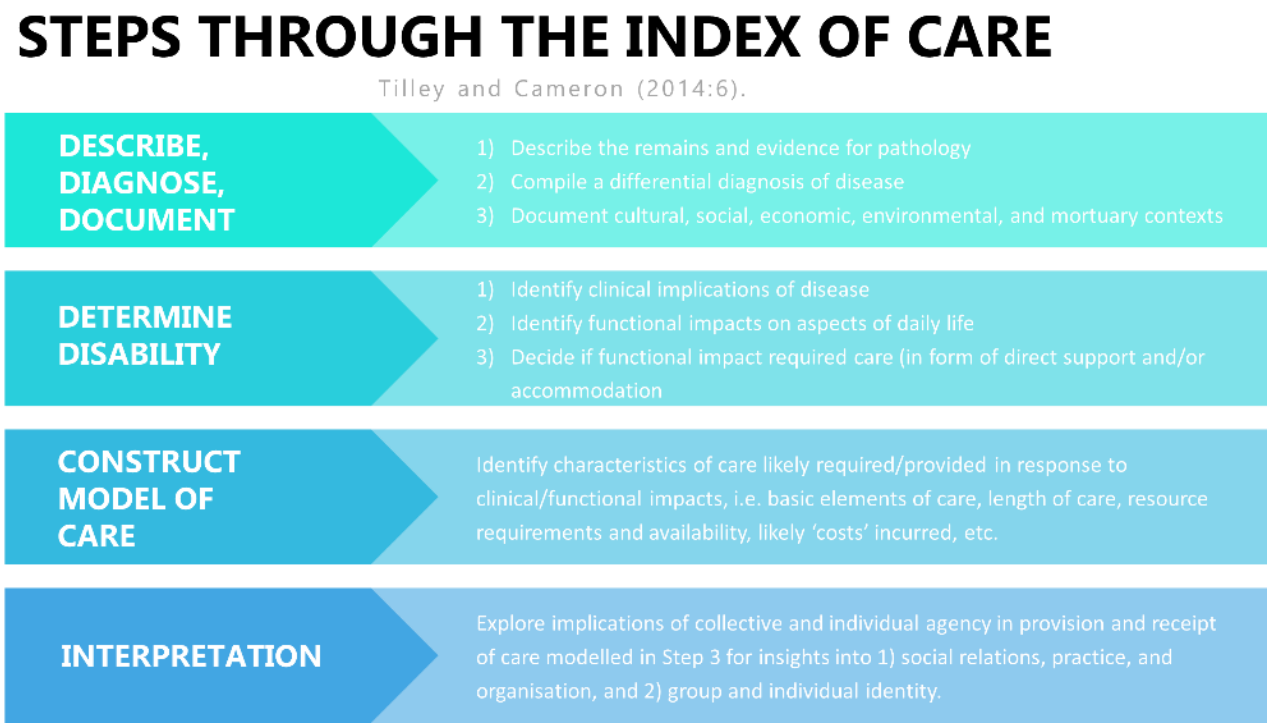


Figure 4.13 The structure and methods within the Index of Care framework. Author's Own.

Once these steps are navigated, the Index of Care web-based platform generates a series of tables that can facilitate analysis and identify areas where health-related care may be needed in order to survive with a disease. It does not, however, produce answers regarding the specificities of care (e.g. what type of care was given, how a person was treated, etc.), or the full impact of the person's experience of, in this case, lepromatous leprosy. The ambiguities in the resultant data can make interpretations contentious and more open to scrutiny, but the model is currently the only framework for the examination of health-related provision using a bioarchaeological proxy (Tilley 2017). Although it will not inform us about the unique experiences of the other people in the

leprosarium, it may provide a line of evidence to support whether health-related care could be provided, beyond general assumptions.

4.3.4 Isotope Methods

The current optimal biological material for the analysis of strontium and oxygen isotopes in archaeological skeletal remains is core tooth enamel, due to the fixing of its elemental composition during childhood development, and its resistance to diagenetic alteration after death (Budd et al. 2000; Montgomery 2010). In order that carbon and nitrogen stable isotope analyses produce results with a high temporal resolution in exploring palaeodietary patterns and/or periods of physiological stress, regular increments of unexposed dentine are ideal to use, and preferred over bone collagen (Beaumont et al. 2013a; Beaumont et al. 2018). Both enamel and dentine have the potential to represent snapshots of averaged strontium, oxygen, carbon, and nitrogen isotope values during the dental developmental processes (Budd et al. 2000; Beaumont and Montgomery 2015). Furthermore, because teeth form and develop at relatively known rates, it is possible to select teeth in order to examine particular stages of growth and development (see iv-Estimating Timings of Tooth Growth).

(i) Initial isotope sampling

Teeth were sampled in the Archaeological Isotope and Peptide Research Laboratory (AIPRL), following in-house risk assessments and laboratory protocols. Before and between sampling, all surrounding benches and equipment were cleaned with kimwipes and deionised water. Metal dental tools were rinsed three times with deionised water, submerged in beakers of Decon90 and ultrasonicated for 5 minutes, rinsed three times with deionised water, and dried with acetone to avoid cross-contamination. All teeth were initially cleaned using a Marathon-7 power unit with hand piece, and rotary dental instrument attachments including tungsten carbide dental burrs and diamond circular saws (Figure 4.14) to remove any exogenous adherents. Tooth samples were examined for linear enamel hypoplasias, dental calculus, and other dental anomalies (e.g. enamel pearls) which was then measured and recorded. Calculus was removed with a clean dental pick and placed in pre-labelled 1.5 ml Eppendorf safe lock microtubes. One half of

the tooth was used to sample approximately 10 mg and 20mg chips of core enamel for strontium and oxygen isotope analyses, respectively. Enamel was sampled with a clean diamond circular dental saw from just above the enamel-dentine junction (EDJ) (Figure 4.14). The enamel chips were then cleaned with a smaller tungsten carbide dental burr to remove any adherent dentine. Enamel samples were weighed on an HR-200 digital balance and placed in 1.5 ml Eppendorf safe lock microtubes for strontium and oxygen isotope analyses.

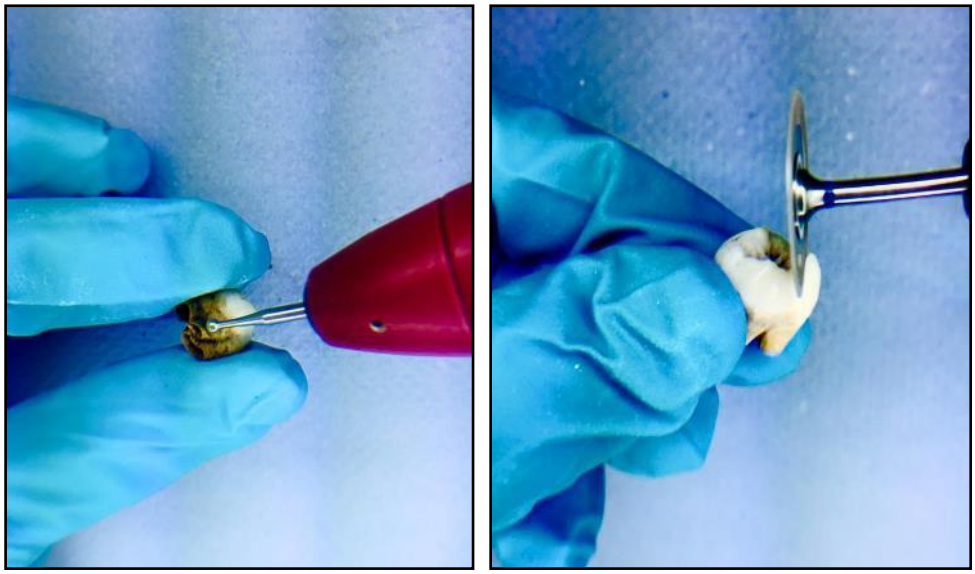


Figure 4.14 Cleaning the tooth crown and root with a dental hand piece and tungsten carbide dental burr (Left), and removing a chip of enamel along the enamel-dentine junction with a diamond circular saw (Right).

The remaining half of the tooth was retained for carbon and nitrogen stable isotope analyses of incremental dentine. All chips of core enamel prepared for strontium and oxygen isotope analyses were transferred to the class 100, HEPA-filtered laboratory facilities at the Natural Environmental Research Council (NERC) Isotope Geosciences Laboratory (NIGL) at the British Geological Survey, Keyworth, Nottinghamshire, UK for further preparation and analysis.

(II) RADIOGENIC STRONTIUM AND STABLE OXYGEN ISOTOPE METHODS

STRONTIUM

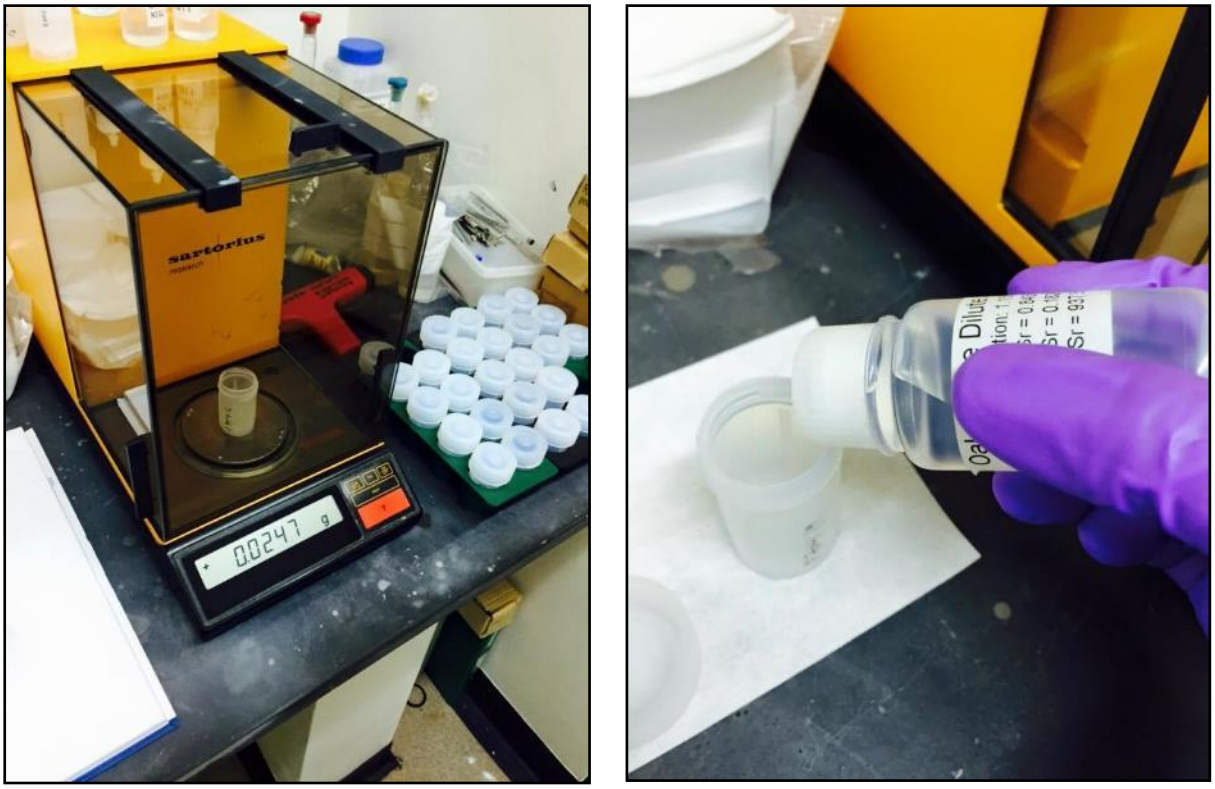
For strontium isotope analysis, enamel samples were transferred to the Radiogenic Isotope Clean Room Complex at NIGL. Gloves and full personal protective clothing were worn throughout the entirety of the subsequent processes to control for contamination. The core enamel samples were removed from their sealed tubes and placed into 10 ml clean Pyrex beakers (Figure 4.15). The beakers were filled with approximately 1- ml of deionised ultrapure water and placed on a 65°C heated block within a laminar flow hood for one hour (Figure 4.15).

Figure 4.15 Ultrapure water added to enamel samples (Left) and samples on heated block in the laminar flow hood (Right).

After 60 minutes, enamel samples were removed from the heated block and rinsed twice with ultrapure water. The samples were re-submerged with deionised ultrapure water, covered with Parafilm, and placed in an ultrasonic bath for 3 minutes to remove any further particle contaminants (Figure 4.16). Once out of the ultrasonic bath, samples were rinsed once again with ultrapure water and then left to dry out on the 65°C heated block within the laminar flow hood. After a further 60 minutes, sample beakers were removed from the laminar flow hood and covered with Parafilm before weighing.

Figures 4.16 Parafilmed enamel samples in the ultrasonic bath.

Beakers with samples were moved to the NIGL Sample Weighing Room where the samples were transferred into clean Teflon beakers and weighed using a Sartorius digital laboratory balance (Figure 4.17). Weights were recorded and Teflon beakers were labelled with a unique sample and in-house batch numbers.



Figures 4.17 Weighing clean enamel samples in Teflon beakers (Left) followed by adding strontium spike solution (Right).

After the samples were weighed, labelled, and logged, between three and four drops of Oak Ridge Dilute Strontium Spike (concentration 1.1593 ppm; 87Sr/86Sr = 0.182448) was added to each sample (Figure 4.17) to determine the strontium concentrations. One drop of the spike solution was also added to each of three empty Teflon beakers to be used as blanks. The blanks were run in tandem with the samples to estimate the blank contribution of the laboratory procedure.

Samples were transported back to the NIGL Radiogenic Isotope Clean Room Complex where c. 2ml of 8M ultrapure nitric acid was added to the samples and left over night to dissolve and evaporate to dryness on using welled hotplates, to minimise sample cross contamination, at c 105°C (Figure 4.18).

Figure 4.18 Enamel samples in Teflon beakers within on the hot plate following the addition of 8M ultrapure nitric acid.

The enamel samples were removed from the heated blocks and approximately 1 ml of Quartz distilled 6M HCl was added to each beaker, including the blanks, to convert the samples to chloride form. Samples were placed back into a 105°C heated block within the laminar flow hood and left to dry out overnight. After approximately 12 hours, samples were removed from the heated blocks, and 2 ml of 2.5M HCl was added to each beaker and left for approximately 30 minutes to fully dissolve the samples. One ml of each sample was loaded onto an Eichrom AG 50w-X8 resin columns (Figure 4.19). These columns selectively retain cations on their surface so that the elements are chromatographically separated. The remaining sample retained in a 1.5 ml Eppendorf safe lock microtube. Samples were then given a 1 ml wash of 2.5M HCl acid solution to guarantee all of the sample adhered to the resin. 48 ml of 2.5M HCl was then added to the column to wash off unwanted cations such as calcium.

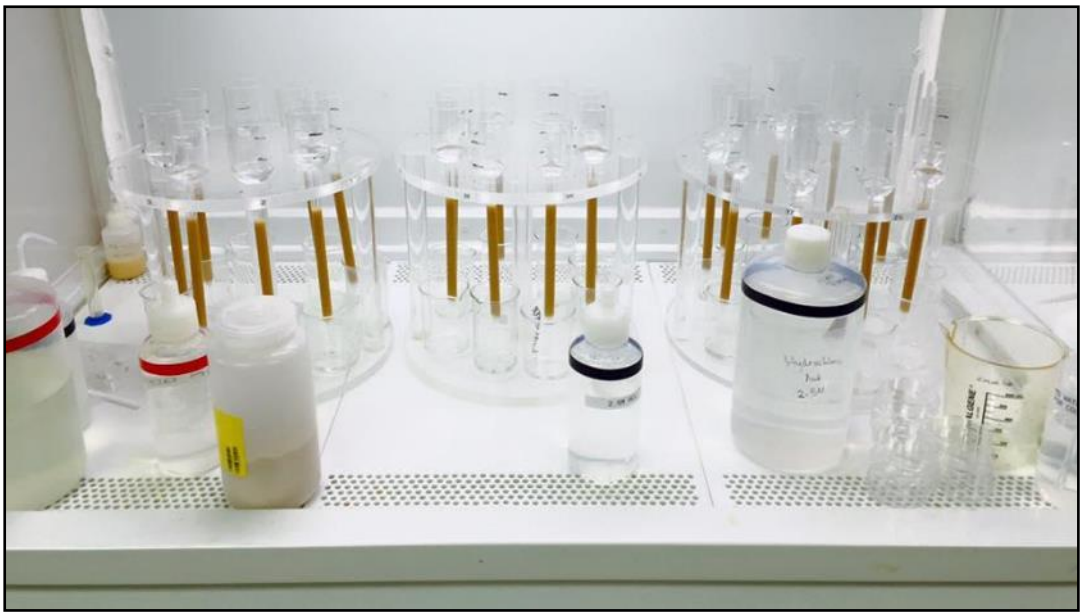


Figure 4.19 Eichrom AG 50w-X8 resin columns for column chemistry. Strontium samples were collected in the beakers below.

The empty sample Teflon beakers were cleaned of residual sample solution by rinsing with deionised ultrapure water and then leaching with approximately 1 ml of 6M HCl at 65°C for approximately two hours. Once the 2.5HCl has passed through the ion exchange column, the cleaned sample beakers were placed under the appropriate columns and 11 ml of 2.5M HCL acid solution was added to the columns. This washed the strontium off the column. These beakers were then returned to the hot place and the samples evaporated to dryness.

The strontium was loaded onto Rhenium filaments (after the method of Birck 1986) (Figure 4.20). The strontium isotope compositions and concentrations were measured using Thermal Ionisation Mass Spectroscopy (TIMS) with a Thermo Triton Multi-Collector Mass Spectrometer. The international standard for $^{87}Sr/^{86}Sr$, NBS-987, gave a value of 0.710251 ± .000005 (n=19, 2) during the analysis of these samples. Procedural blanks gave values of less than 100 picograms (pg).

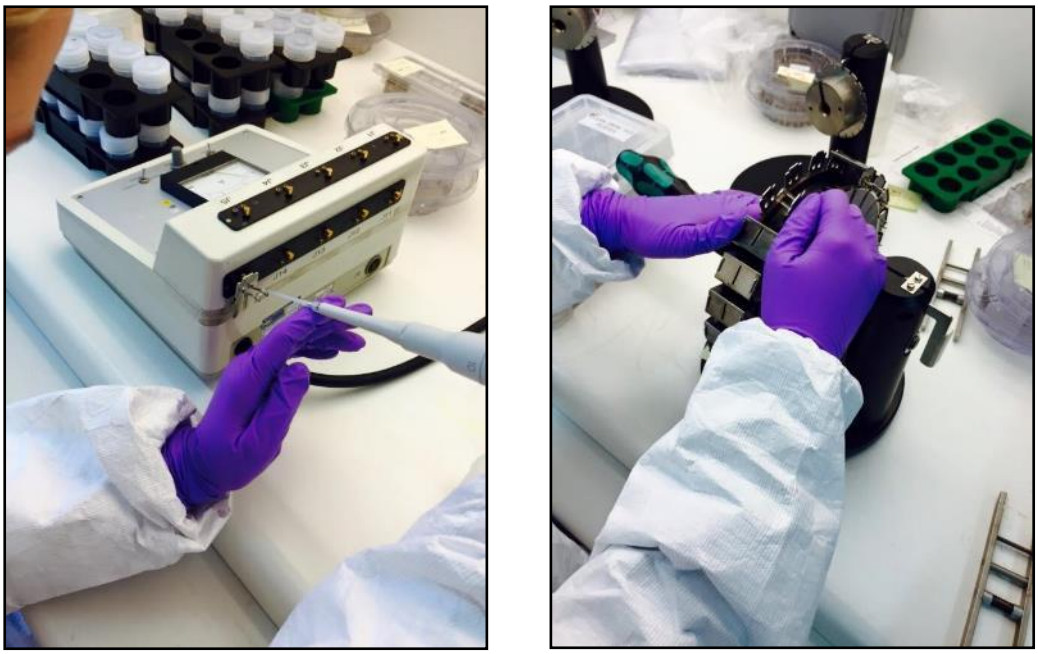


Figure 4.20 Loading isolated strontium onto rhenium filaments (Left), and loading filaments into the turrets for thermal ionisation mass spectrometry (Right).

OXYGEN

At the NIGL facilities at the British Geological Survey, enamel samples were ground into powder using a pestle and mortar covered with Parafilm. The pestle and mortar were cleaned with acetone and Kimwipes between each sample (Figure 4.21). Crushed enamel was put onto a Whatman grade B-2 3”x 3” weighing sheet, and then transferred into new 1.5 ml Eppendorf safe lock microtubes.

Figure 4.21 Powdering enamel with mortar and pestle for oxygen stable isotope analysis, protected by Parafilm.

Approximately 3 mg of powdered enamel (~5-8% carbonate) were loaded into a glass vial and sealed with septa (partitions used to seal the samples). The vials were then transferred to a heated block at 90°C on the GV Multiprep system. Afterwards, the vials were emptied and three drops of anhydrous phosphoric acid was added. The resultant CO_2 was cleaned up and collected cryogenically for 14 minutes and then transferred to a GV IsoPrime Dual Inlet Mass Spectrometer. Any deviations from the isotopic ratios ($^{13}C/^{12}C$, $^{18}O/^{16}O$) were calculated to the Vienna Pee Dee Belemnite (VPDB) scale using a within-run calcite laboratory standard (Keyworth Carerra Marble, KCM) calibrated against SRM19 NIST reference material, and are reported using delta notation in parts per mil (‰). $\delta^{18}O_{(C)VPDB}$values were normalised to Standard Mean Ocean Water (VSMOW) using the equation from Coplen (1988)(VSMOW=1.03091 x $\delta^{18}O_{(C)VPDB}$+30.91). Analytical reproducibility for this run of laboratory standard calcite (KCM) was 0.09‰ (1σ, n = 6) for $\delta^{18}O_{VSMOW}$ and ± 0.05‰ (1σ, n = 6) for $\delta^{13}C_{VPDB}$. The analytical reproducibility of the enamel, based on average of five duplicate pairs was ± 0.07‰, 1σ for $\delta\ ^{18}O_{(C)VSMOW}$ and ± 0.04‰, 1σ for $\delta\ ^{13}C_{VPDB}$.

$\delta^{18}O_{(C)VSMOW}$ were converted into $\delta^{18}O_{(P)VSMOW}$ using the regression formula of Chenery et al. (2012): $\delta^{18}O_p$ = 1.0322(± 0.008) x $\delta^{18}O_c$ -9.6849 (± 0.187), with an associated error of ±0.29 ‰, 1σ. The conversion to phosphate values was carried out to make the data more

comparable to oxygen isotope data reported in other studies and baselines (e.g. Evans et al. 2012).

$\delta^{18}O_{(C)VSMOW}$ were also converted to drinking water values ($\delta^{18}O_{DW}$) using the conversion formulae in Chenery et al. (2012), based on Daux and colleagues' (2008) Equation 6: $\delta^{18}O_{DW} = 1.590 \times \delta^{18}O_{(C)VSMOW} - 48.634$, ± 0.4‰, 1σ.

The conversion to drinking water values can involve larger uncertainties (a cumulative error of ± 1 ‰, 2σ)(Chenery 2012; Pollard et al. 2011), and therefore $\delta^{18}O_{DW}$ reported for this thesis are only used as a general guide to compare against modern drinking water values.

(III) Carbon and Nitrogen Isotope Analyses

For carbon and nitrogen stable isotope analyses of incremental dentine, 20 tooth samples were selected from 10 individuals from the St. Mary Magdalen leprosarium site (Table 4.8). St. Mary Magdalen was selected preferentially over St. John at the Castle Gate/Timberhill to examine whether any dietary consistency exists within the individuals buried in the leprosarium. Due to financial constraints, only ten individuals (20 tooth samples) could be selected for this analysis, and any other teeth that were originally selected for sampling were earmarked for return to the curating institutions. A canine, and a second or third molar (dependent on age) were selected to enable the construction of complete life history profiles (birth to death) from the incremental dentine sections.

Table 4.8 - Individuals from St. Mary Magdalen leprosarium selected for carbon and nitrogen stable isotope analyses of incremental dentine.

INDIVIDUAL	SEX	AGE	TEETH SAMPLED	CONCOMITTANT PATHOLOGICAL CONDITIONS	OTHER NOTES
SK. 8	Male	8.5-9.5	Maxillary left canine Maxillary left M2	Linear enamel hypoplasia; leprogenic odontodysplasia; cribra orbitalia	Skeletal age indicates 7-8 years
SK. 9	Male	22.5-23.5	Maxillary right canine Maxillary right M2	Linear enamel hypoplasia; osteochondritis dissecans	Asymmetrical dental calculus
SK. 15	Male	20.5-22.5	Mandibular left canine	Linear enamel hypoplasia; lytic	Potential Tuberculosis or

			Mandibular left M3	spinal lesions; cribra orbitalia; spina bifida occulta; spondylolysis	Mycotic Infection? Bilateral Os Acromiale
SK. 18	Male	18.5-19.5	Mandibular right canine Mandibular right M3	Linear enamel hypoplasia; cribra orbitalia; possible scurvy; extremely thin cortical bone	Delayed Growth – Unfused ilium <14 years; Prognathic Maxilla may indicate long-term leprosy; Asymmetrical dental calculus
SK. 27	Male	22.5-23.5	Mandibular right canine Mandibular right M3	Linear enamel hypoplasia;	Heavy asymmetrical dental calculus
SK. 28	Ambiguous	12.5-13.5	Maxillary left canine Maxillary left M2	Linear enamel hypoplasia; leprogenic odontodysplasia; cribra orbitalia; spina bifida occulta; osteochondritis dissecans	Prognathic Maxilla may indicate long-term leprosy
SK. 45	Ambiguous	15.5-16.5	Mandibular left canine Mandibular left M2	Linear enamel hypoplasia;lytic spinal lesions; rib lesions	Delayed Growth - Skeletal age indicates 10-12 years; Tuberculosis or Mycotic Infection?; Asymmetrical dental calculus
SK. 52	Ambiguous	12.5-13.5	Mandibular left canine Mandibular left M2	Linear enamel hypoplasia; leprogenic odontodysplasia; cribra orbitalia; lumbar ribs (l1); lumbarisation of s1	Prognathic Maxilla may indicate long-term leprosy; asymmetrical dental calculus
SK. 54	Ambiguous	14.5-15.5	Mandibular right canine Mandibular right M2	Linear enamel hypoplasia	Delayed Growth - Skeletal age indicates 9-11 years; Asymmetrical dental calculus
SK. 56	Male	16.5-17.5	Maxillary right canine Maxillary right M2	Linear enamel hypoplasia; cribra orbitalia; leprogenic odontodysplasia; visceral rib lesion (left t10);	Asymmetrical dental calculus

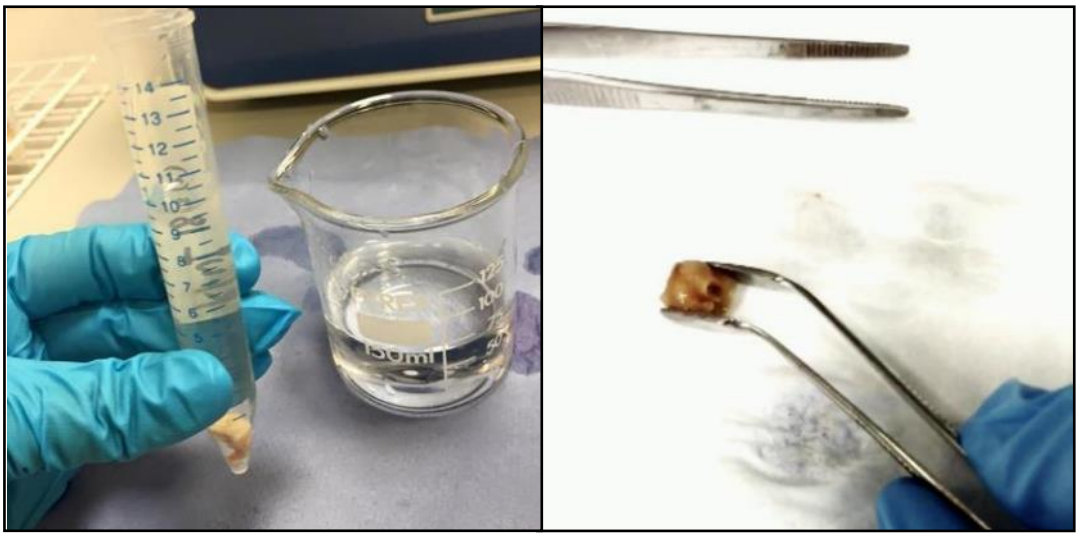
	congenital absence of 1 lumbar vertebra

Tooth Preparation

Canines and portions of molars that were not sampled for strontium and oxygen isotope analyses were further prepared for carbon and nitrogen stable isotope analysis of incremental dentine using Method 2, as outlined in Beaumont et al. (2013a) and briefly outlined here. Using a dental saw attachment for the micromotor hand piece and a fluorescent magnifying lamp, the teeth were bisected mesio-distally. In one instance (Sk. 8), only the second molar crown and first quarter of the root was available so the entire tooth was demineralised. Using the dental saw and dental burrs, most of the remaining enamel was carefully removed so as not to breach the enamel-dentine junction (EDJ). Metal attachments were sterilised between each tooth by submerging the saws and burrs in a beaker of Decon-90, and then placing them within an ultrasonicator for five minutes to remove any further particle contaminants. Following cleaning between each tooth, all metal tools were dried with acetone.

Tooth Demineralisation

Tooth samples were prepared for collagen extraction using the modified Longin method without ultrafiltration, in accordance with the methods built upon by O'Connell and Hedges (1999; Longin 1971). Longitudinally sectioned teeth were first weighed and then placed into a 15ml test tube. Approximately 5-10ml of cold 0.5M HCl was added to each tube to fully submerge the tooth sample in order to demineralise the sample, and then a marble was placed on the tube to prevent contamination. The samples were placed in a refrigerator at 4 degrees celsius and the 0.5M HCl was changed weekly until demineralisation was complete and the tooth samples were soft and pliable (Figure 4.22). The molar tooth samples took approximately eight weeks to fully demineralise, and the canine samples took approximately six to eight weeks. Samples and acid washes were logged on in-house tracking sheets.



Figures 4.22 The bisected right mandibular third molar of Sk. 18 demineralising in 0.5M of cold HCl (Left), and the left maxillary second molar of Sk. 8 after the demineralisation process (Right).

TOOTH SECTIONING

Once the tooth samples were fully demineralised and pliable, they were transversely sectioned. The demineralised teeth were first rinsed three times with ultrapure water and weighed on an HR-200 digital balance to calculate the collagen yield after extraction. The demineralised tooth was then measured with a sterile metal ruler to determine the number of sections in the tooth. The tooth was placed parallel to the metal ruler and transversely sectioned from the crown to the apex into 1mm slices with a clean scalpel, as recommended in Method 2 of Beaumont et al. (2013a) (Figure 4.23). In preparing for collagen extraction the sections were placed in a microtube and immersed into 0.5M HCl.

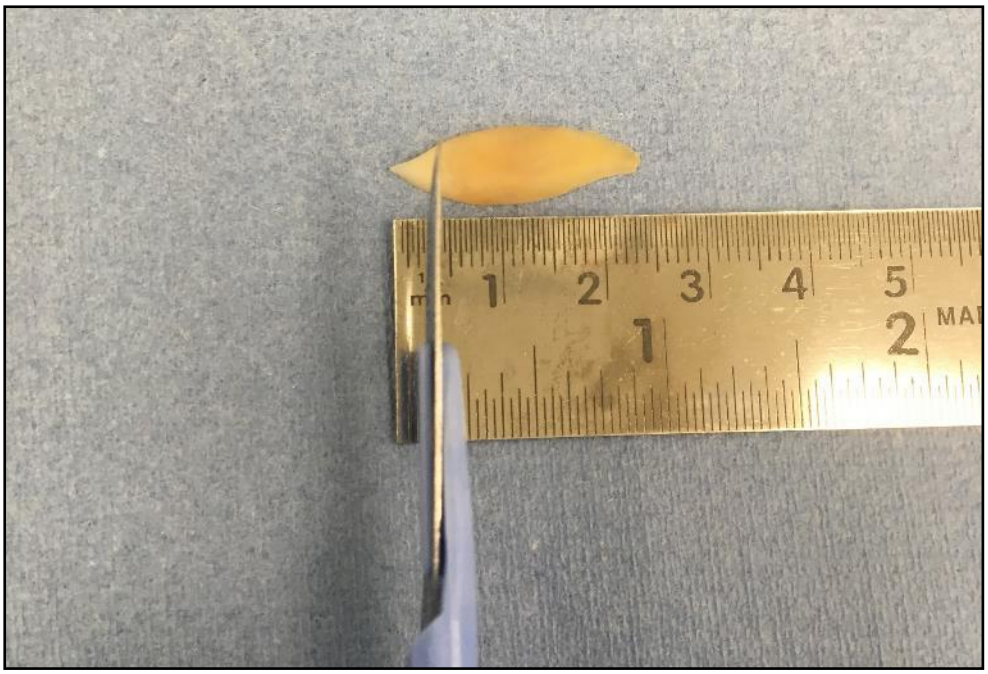


Figure 4.23 Longitudinal sectioning of a canine tooth with a scalpel following Method 2 of Beaumont et al. (2013a).

COLLAGEN EXTRACTION

Once 1mm sections were obtained, each microtube was then pipetted with a pH3 solution of 0.5M HCl and ultrapure water. The samples were then put into a Techne DB-3D heated block to denature at 70°C for 24 hours, and then centrifuged to separate any remaining debris (Figure 4.24).

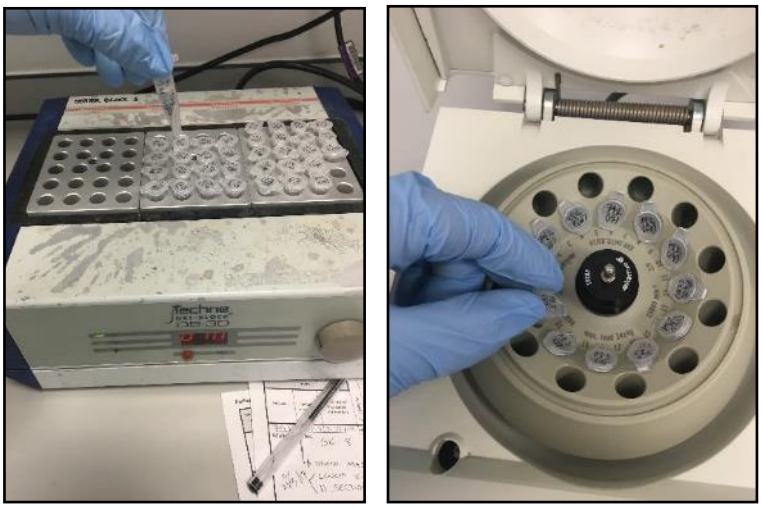


Figure 4.24 Samples within microtubes denaturing within a heated block (Left), before being centrifuged to separate any further debris (Right).

Afterwards, the samples were sealed and frozen before being lyphilised (freeze-dried) to remove the remaining water. The microtubes were freeze dried with their lids open, but covered with strips of punctured Parafilm to prevent the collagen from expanding outside the microtube and contaminating adjacent samples. Once lyphilised, extracted collagen was removed from the microtubes with sterile metal tweezers and weighed on aluminium foil on a Denver microbalance to calculate the total collagen yield. After weighing, subsamples of the collagen were weighed in duplicate in tin capsules for mass spectrometry. Using sterile metal tools, a minimum of 0.5 mg of collagen was taken from the overall sample where possible, placed within 6x4mm tin capsules, and weighed on microbalances to ensure an optimal range of collagen was obtained for the mass spectrometer in accordance with recommendations in Beaumont et al. (2013a) (Figure 4.25).

Figure 4.25 Placing collagen within a tin capsule (Left), and weighing the tin capsule on a microbalance to ensure an adequate sample weight (Right).

Collagen weighing was carried out at both the Stable Isotope Laboratory at the School of Archaeological and Forensic Sciences, University of Bradford (molar collagen) and at the Archaeological Isotope Peptide Research Laboratory in the Department of Archaeology, Durham University (canine collagen). After weighing, tin capsules were folded into small spheres with metal tools. The tools were cleaned with acetone between each sample to

prevent cross-contamination. Samples within the tin capsules were deposited within plastic annotated trays and recorded using in-house sample tracking sheets. The samples were interspersed with blank capsules, and both in-house and international standards used as measures of quality control (See V. Quality Controls).

Collagen from the dentine samples was measured in duplicate when possible, but in instances where the collagen yield from the dentine slice was low (i.e. in the initial coronal sections or the apices), only a single sample could be produced. In instances where the weight of the sample was suboptimal, the collagen yield and other quality controls were cross-referenced in order to ensure the reliability of the data. Carbon and nitrogen isotope compositions from collagen samples were measured in duplicate by combustion through a Thermo Flash EA 1112 elemental analyzer with the introduction of separated N_2 and CO_2 to a Finnigan Delta XL Plus isotope ratio mass spectrometer via a Conflo III interface at the School of Archaeological and Forensic Sciences, University of Bradford. The samples were compared to reference gasses (N_2 and CO_2) prior to combustion, and then calibrated to procedural blanks and analytical standard reference material. Results were calculated to the Pee Dee Belemnite marine limestone deposit for carbon and Ambient Inhalable Reservoir (AIR) for nitrogen, and reported using the delta notation (δ) in parts per mil (‰). Blank tin capsules and both in-house laboratory and international standards (Table 4.9) gave an analytical reproducibility of ±0.25‰ (1σ) for the in-house standards, and ±0.2‰ (1σ) or better for the international standards used.

TABLE 4.9 - Carbon and nitrogen isotope ratios for international and in-house standards used in this thesis.

STANDARD	***TYPE***	***$\delta^{13}C$‰***	***$\delta^{15}N$‰***
IAEA 600	International	-27.77± 0.04‰	1.00± 0.2‰
N1	International		0.43± 0.2‰
N2	International		20.41± 0.2‰
CH3	International	-24.72± 0.2‰	
FISH GEL	In-House	-15.52	14.45
BOVINE LIVER SERUM	In-House	-21.59± 0.25‰	7.65± 0.25‰

(IV) ESTABLISHING TIMINGS OF TOOTH GROWTH AND DEVELOPMENT

In order to link the isotope ratios from enamel and increments of dentine to an approximate biological age or age range, the developmental timings of the sampled teeth must be established. Dental growth and development is regarded as the most reliable age estimation method for individuals who died before their teeth reached full maturity (Smith 1991; Cardoso 2007; Conceição and Cardoso 2011). This is mainly due to the fact that dental tissues, namely enamel, dentine, and cementum, grow incrementally with little to no remodelling, and are primarily genetically-driven, rather than environmentally influenced (Dean 2000; Dean and Scandrett 1995; Dean and Scandrett 1996; Dean et al. 2014). It is worth mentioning however, that these periods of growth and development can be punctuated by alternating patterns of slow growth, followed by periods of faster catch-up growth within and between teeth (Avery 1991; Dean 2000; Dean and Cole 2013). Nevertheless, by using the average timings of these dental tissues and their overall development, isotope data can be linked to approximate biological age ranges (Moorrees et al. 1963; Dean and Scandrett 1995; Dean 2000; AlQahtani et al. 2010; Dean and Cole 2013).

For example, using the tooth growth and development ranges from AlQahtani et al. (2010) (Table 4.10), the crown of a mandibular third molar initiates (CI) at approximately 7.5 ± 0.5 years, the crown completes (CRC) at a maximum age of 17.5 ± 0.5 years, and the root apex closes (AC) by 23 ± 0.5 years. This means that a core enamel sample for strontium and oxygen isotope analysis will represent an average isotope composition from between the ages of 7.5 ± 0.5 years to 17.5 ± 0.5 years of that person's life. If the tooth grows from 7.5 ± 0.5 years to 23.5 ± 0.5 years, the total growth period for the tooth is approximately 16 years. If the tooth measures 10 mm in length from the coronal dentine to the apex (root tip), a 1mm increment will represent $1/10^{th}$ of 16 years, or a 1.6 year average of the individual's carbon and nitrogen isotope values. It is crucial to note that whilst dentine is deposited incrementally, it is not deposited in transverse layers (Dean and Scandrett 1995), therefore transverse sectioning of dentine will only represent running averages and not discrete time periods in a person's life.

TABLE 4.10 - Growth and development rates of teeth (both sexes) used in this research (Moorrees et al. 1963; AlQahtani et al. 2010).

TOOTH	MINIMUM AGE AT CROWN INITIATION (CI)	MAXIMUM AGE AT CROWN COMPLETION (CRC)	MAXIMUM AGE AT APEX COMPLETION (AC)	TOTAL GROWTH TIME (YEARS)
MAXILLARY CANINE	7.5 months ± 1.5 months	6.5 years ± 6 months	16.5 years ± 6 months	15.9
MAXILLARY PM2	2.5 years ± 6 months	8.5 years ± 6 months	15.5 years ± 6 months	13
MAXILLARY M1	1.5 months ± 1.5 months	4.5 years ± 6 months	12.5 years ± 6 months	12.4
MAXILLARY M2	2.5 years ± 6 months	8.5 years ± 6 months	17.5 years ± 6 months	15
MAXILLARY M3	7.5 years ± 6 months	16.5 years ± 6 months	23.5 years ± 6 months	16
MANDIBULAR CANINE	7.5 months ± 1.5 months	7.5 years ± 6 months	15.5 years ± 6 months	14.9
MANDIBULAR M2	2.5 years ± 6 months	9.5 years ± 6 months	17.5 years ± 6 months	15
MANDIBULAR M3	7.5 years ± 6 months	17.5 ± 6 months	23.5 ± 6 months	16

(v) ADDITIONAL QUALITY CONTROLS FOR CARBON AND NITROGEN STABLE ISOTOPES

In addition to the in-house and certified international standards measured in the mass spectrometers, the following quality control parameters were also used before any interpretations were made.

ELEMENTAL MASS PERCENTAGES

According to Ambrose (1993) and van Klinken (1999), the percentage of archaeological collagen should be relatively similar to modern collagen percentages, which are

approximately 45% carbon and 18% nitrogen. van Klinken (1999) has proposed an acceptable carbon range of 30-50%, and an acceptable nitrogen range of 10-18% in archaeological collagen. Too little may suggest advanced diagenesis, whereas too much may suggest other protein contaminants (*ibid*). Carbon and nitrogen percentages are given in Manuscript 5.4 within the results chapter, and also available in the appendix of this thesis (see APPENDIX: DATA).

Collagen Yields

Type 1 collagen comprises 89% of organic composition of dentine (Goldberg et al. 2011). The collagen yield derived from the demineralisation and subsequent extraction process serves as a major quality indicator to ensure that the tooth has not been significantly altered by diagenetic change. Diagenetic changes in the isotopic composition of collagen must be considered before any meaningful interpretations can be made (Ambrose 1990; Ambrose 1993). The collagen yield is calculated by dividing the mass of the collagen by the original mass of the sample. Ambrose (1990) and van Klinken (1999) suggest a minimum of 1.2% and 0.5-1% collagen yield from each sample, respectively, in order to verify the quality of the tooth sampled. The collagen yield from both the molar and the canine tooth samples ranged from 1.5%, in the instance of an incomplete canine from Sk. 8, to 26.19% (See Table 4.11).

Table 4.11 - Collagen yields from tooth samples.

INDIVIDUAL	TOOTH	SAMPLE MASS (MG)	DRY COLLAGEN MASS (TOTAL)	YIELD (COLLAGEN/DRY WEIGHT X100)
SK. 8	Maxillary Left Canine	338.4	5.36	1.50%
	Maxillary Left M2	1010	45.4	4.50%
SK. 9	Maxillary Right Canine	437.4	46.31	10.59%
	Maxillary Right M2	715	79.35	11.10%
SK. 15	Mandibular Left Canine	418.9	51.82	12.37%
	Mandibular Left M3	611	58.81	9.63%
SK. 18	Mandibular Right Canine	544.5	98.176	18.03%

	Mandibular Right M3	568	38.14	6.70%
SK. 27	Mandibular Right Canine	503	81.83	16.26%
	Mandibular Right M3	805	71.82	8.92%
SK. 28	Maxillary Left Canine	329	38.87	11.81%
	Maxillary Left M2	593	84.96	14.33%
SK. 45	Mandibular Left Canine	333.4	58.92	17.67%
	Mandibular Left M2	660	51.57	7.81%
SK. 52	Mandibular Left Canine	400.6	75.81	18.92%
	Mandibular Left M2	615	54.35	8.84%
SK. 54	Mandibular Right Canine	519.5	136.06	26.19%
	Mandibular Right M2	671	92.22	13.74%
SK. 56	Maxillary Right Canine	349.3	86.45	24.75%
	Maxillary Right M2	639	93.54	14.64%

C:N RATIOS

Another quality control for collagen extraction is the measurement of the ratio of carbon to nitrogen (C/N). C/N ratios in isotopic analyses are used to confirm that the archaeological collagen is not contaminated with other organic materials (DeNiro 1985; van Klinken 1999). Collagen has a characteristic amino acid composition, which can be altered diagenetically by the burial environment (DeNiro 1985; van Klinken 1999). Researchers suggest that the measurements of the ratios of atomic carbon to nitrogen within a collagen sample can act as a proxy for the quality of the amino acid composition (DeNiro 1985; Smith et al. 2009).

$C/N_{atomic} = (\%C/\%N) \times (14/12)$

DeNiro (1985) suggested a C/N ratio for modern humans and animals of 2.9-3.6 as acceptable quality, whereas van Klinken (1999) has suggested a narrower range of 3.1-3.5. Any sample that gave a C/N ratio that fell above 3.6 was excluded. C/N ratios are listed in Manuscript 4 in the results chapter, and in the appendix (see APPENDIX: DATA).

It is worth noting that the preceding quality controls for collagen were developed using bone collagen, which is subject to greater diagenetic alteration than dentine encapsulated by enamel (Kendall et al. 2018). Although there is currently no evidence to suggest that the Type 1 collagen within bone and dentine shows any significant difference, Beaumont and colleagues (2018) recently highlighted that archaeological collagen from dentine is a more robust protein for carbon and nitrogen isotope analyses.

4.3.5 Amelogenin Peptide Methods

Five individuals (Sk. 28, Sk. 41, Sk. 45, Sk. 52, Sk. 54) from St. Mary Magdalen were chosen for amelogenin peptide extraction to identify their biological sex. These individuals were chosen because their pre-pubertal status prevented sex estimation from their skeletal remains. Amelogenin peptide extraction is an accurate, minimally destructive method for sex determination through the surface acid etching of tooth enamel, with subsequent identification of sex chromosome-linked isoforms of enamel forming protein through nanoflow liquid chromatography mass spectrometry (nanoLC-MS; Stewart et al. 2016; Stewart et al. 2017). This method was carried out to examine whether adolescent females were represented in St. Mary Magdalen leprosarium's North Cemetery. Previous osteological analyses (Table 4.1) suggest that only three older females (46+) were present in the entire cemetery, with the possible exception of Sk. 38, whose remains were unavailable for examination at the time of analysis. The unsexed individuals from St. John at the Castle Gate/Timberhill could not be analysed with this method due to financial constraints. The amelogenin peptide extraction was performed on portions of abraded second molar enamel following the methods set out in Stewart et al. (2017). The enamel etch area was washed with 3% H_2O_2 for 30 seconds, followed by a rinse with ultrapure water. Enamel etching was carried out using 80 µl 10% HCl for two minutes, after which, the first etch was discarded, and a second two minute etch performed and retained. The peptides in the etch solution were bound to C18 resin ZipTips conditioned three times with 10 µl 100% acetonitrile and 0.1% formic acid. The peptides were eluted off the C18 resin using 4 µl of elution buffer (60% acetonitrile/0.1% formic acid). The peptide samples were then frozen at -18°C and freeze-dried.

Samples were transferred to the School of Pharmacy and Biomolecular Sciences, University of Brighton where they were dissolved in 12 µl 0.1% trifluoroacetic acid (TFA) in water, and centrifuged for 5 minutes to remove particulates and transferred to 10 µl glass autosampler vials. Approximately 5 µl of sample was analysed by reverse-phase nanoLC-MS with a liquid chromatograph (nanoRS U3000; Thermo Fisher Scientific) coupled to a hybrid quadrapole orbitrap mass spectrometer (Q Exactive; Thermo Fisher Scientific). The data were searched against the human proteome (UniportKB, 10/15) with MaxQuant v 1.5.1.2 using default search settings with methionine oxidation as a variable modification, unspecific digestion mode and a minimum peptide length of 6 (Stewart et al. 2017).

4.3.6 Summary Statistics and Data Presentation

Isotopic data was input into Microsoft Excel spreadsheets, and means and standard devations were calculated using built-in formulae. Scatterplots were generated to visually present the data for all isotope systems, and box and whisker plots were used to visualise carbon and nitrogen variation within the leprosarium sample. Summary statistical data are presented in Manuscripts 5.2, 5.3, and 5.4 of the Results section (see Chapter 5). Carbon and nitrogen sample sizes were too small to carry out any meaningful statistical analyses, but further studies may enable this in future.

4.4 References

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CHAPTER 5. RESULTS

This results chapter comprises four manuscripts prepared for publication. Each manuscript lists the provisional manuscript titles, co-authors/supervisors, the publication the manuscript has been prepared for, and denotes whether it has been submitted for review or accepted for publication. Each manuscript was drafted by the first author with comments and suggested edits from the co-authors/supervisors.

MANUSCRIPT 5.1: ALLOPARENTING ADOLESCENTS: EVALUATING THE SOCIAL AND BIOLOGICAL IMPACTS OF LEPROSY ON YOUNG PEOPLE IN EARLY-LATE MEDIEVAL ENGLAND (9TH – 12TH CENTURIES AD) THROUGH CROSS-DISCIPLINARY MODELS OF CARE.

Accepted for Publication in *The Family in Past Perspective: An Interdisciplinary Exploration of Familial Relationships Through Time*. Kendall, E. and Kendall, R. (eds.).

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Figure 5.1.1 Impaired young person being moved by cart and receiving charity. Reproduced with permission from the British Library. © British Library Board: Add. 42130 f186v.

INTRODUCTION

The majority of historical sources describe past attitudes towards people with leprosy as negative, focussing on ostracism and damnation, and this is thought to have impacted on the care that sufferers received. More recent historical and archaeological evidence challenges this longstanding perspective, portraying a very different view of care for those with this potentially debilitating and disfiguring disease (Roberts 2002; Rawcliffe 2006; Roberts 2013; Roberts 2018; Roberts 2020:280).

This paper aims to explore the social and biological impacts of leprosy on adolescents in Early-Late Medieval England (9th – 12th centuries AD). The intersection of youth, chronic infection, aspects of care (inclusive of medical, surgical, and daily support), and cultural identity has only been tangentially explored in the past (e.g see Redfern and Gowland 2011; Roberts and Bernard 2015; Lewis 2017). Studies that integrate these entwined themes can, however, provide a more holistic view of societal responses to wider encultured disease identities.

This study utilises multiple lines of evidence for medical care and social treatment to evaluate the validity of dominant historical narratives about leprosy, i.e. that people in the past with leprosy were not cared for or treated well. In order to achieve this, the notion of past requirements of care and treatment through an alloparental model will be introduced. This is followed by a review of the existing historiographical evidence for medical care for young people in the Medieval period to better understand systems of care provision and parental reactions to their sick children at this time. Discussions of leprosy in young people in the present and past will help contribute to longitudinal views of the biological impacts of leprosy and help the necessity for care in relation to certain pathological responses (i.e. the manifestation of lepromatous leprosy). To apply this framework to the past, palaeopathological and archaeological evidence from adolescent individuals excavated from the North Cemetery of St. Mary Magdalen leprosy hospital will be analysed. The presence of both leprosy and alloparental care for adolescents in the Early-Late Medieval transition at this hospital is demonstrated. Finally, the construction of a theoretical model of required clinical care and provisions, such as the

Index of Care framework, helps interpret the evidence for care in alloparental institutions such as leprosaria.

The treatment of people with leprosy in the Medieval period is often cited as a justification for the continuing stigma and community expulsion of family members with leprosy in some parts of the world (World Health Organization 2015). Therefore, it is worth examining the social milieu of this disease in which young people with leprosy in the past lived, and the models of care and treatment that may be interpreted from these data in order to dispel this longstanding stigma.

A Note About Terminology

This research considered a variety of sources for determining the biological and social age ranges for adolescence, including:

1. the Early Medieval (5^{th} – 11^{th} centuries AD) social classifications of youth (e.g. i.e. *puerita*: 7 -14, and *adolescentia*: 14 – 28) (Sharpe and Seville 1964:49; Gilchrist 2012:34; Cochelin 2013:3-6);

2. the age that a person was considered an 'independent adult' in the lay population of the Medieval period in England (i.e. 25 years; Cochelin 2013; Lewis 2016:139);

3. and modern biological definitions of adolescence (10-25 years; Patton et al. 2016; Sawyer et al. 2018).

With these definitions in mind, individuals aged c. 10 -25 years at death were included in this paper to encompass both the biological and Medieval social designations of adolescence. Other broad social terms such as 'young' and 'youth' are used interchangeably with adolescents as they are versatilely applied within the cultural understandings of the Medieval life course (Gilchrist 2012: 1-11, 34-35; Mays et al. 2017).

Considering the Role of Alloparenting in the Leprosarium

Since the publication of the contentious work *Centuries of Childhood* by Ariès (1962), many scholars have pushed an agenda for and against the assertion that "in Medieval society, the idea of childhood did not exist" (128). This has inspired a broad range of studies within both history and archaeology to reassert the visible and tangible concepts

of youth in the past, along with the provision of care for children (Demaitre 1977; Kroll 1977; Kroll and Bachrach 1986, Lewis 2016; Dawson 2017; Lewis 2017). One aspect that has not been thoroughly investigated, however, is the perception of the sick child, and the provision of care and associated medical/surgical treatment under an alloparental umbrella within an institutional setting.

Alloparenting is defined as the provision of care for young individuals by persons other than their biological parents (Kenkel et al. 2016). The practice of alloparenting is a cultural universal in both the human and many animal species, and remains an important aspect of caregiving in modern societies (Sear and Mace 2008; Kenkel et al. 2016). Most studies focus on models of alloparenting in breastfeeding, infancy studies, maternal bonding, kinship attachment, nursery/pre-school settings, and schools (Ahnert 2005; Quinlan and Quinlan 2008; Sear and Mace 2008; Bogin et al. 2014), and very few studies examine the effects of long-term healthcare provision in an alloparental institution; i.e. in hospitals for chronically ill children (Youngblut 1999; Zaslow 2006). This may be, in part, due to modern systems by which parents remain involved in limited periods of care during a child's convalescence. Despite this, some modern studies have highlighted that young people receiving care and treatment (in both medical and guardianship contexts) from alloparents and alloparental institutions report a similar quality of life, level of happiness, and health outcomes to those within comparable biological parental units (Lloyd 2012; Kenkel et al. 2016).

In view of this, it is worth considering the alloparental model for Medieval monastic hospitals, including leprosaria such as the St. Mary Magdalen leprosy hospital in Winchester, Hampshire, UK. For example, the discovery of a relatively large number of adolescents (n=23) linked to this leprosy hospital context raises questions concerning who was providing these young people care and the nature of the care received. Writings about the presence and status of adolescents within monastic contexts mainly centre on young people who were given to the monastery, termed oblates (literally meaning "the ones offered"), and their treatment as adoptive and communal family members in both sickness and health (Cochelin 2020:550-553). However, the provision of care and treatment of adolescents within linked monastic contexts such as Medieval leprosy

hospitals is poorly documented. It is therefore worth exploring the evidence for medical care for children in the Medieval period to help construct more holistic views of how young people with leprosy would have been treated within these institutions by their caregivers, or alloparents.

Medical Care for Medieval Children – An Index of Effort

In order to determine whether care and treatment for leprosy (medical and spiritual), as opposed to expulsion, would be a motive for a young person's entry into a leprosarium, we must first assess whether parents sought medical care for their sick children from monastic institutions. Medical provisions, including palliative and interventional care, for sick children in the past are not a large focus in medico-historical writings, which has led to assumptions that physicians from antiquity to the Medieval period did not offer these options for younger patients (Demaitre 1977; Kroll 1977; Kroll and Bachrach 1986; Gordon 1991). This long-standing belief is partially due to the paucity of historical documents detailing the lives of sick children, thereby inferring that they were not important. Common medical practice in the Medieval period was heavily reliant on home-based, folk-medicine; i.e. at-home treatments focussing on local herbal remedies, hygiene, and dietary corrections (Demaitre 1977; Kroll and Bachrach 1986; Newman 2007:71-72). Only in serious circumstances (e.g. chronic infections, plague, dysentery, severe trauma, paralysis, blindness, etc.) were children taken by their parents to physicians or monastic hospitals (Kroll 1977; Kroll and Bachrach 1986; Gordon 1991; Rawcliffe 2006:291-292; Newman 2007:38,41,71-72).

Monastic physicians during this period wrote of their difficulties in diagnosing and treating children, complaining that the young could not adequately vocalise their symptoms and that the conventional diagnostic methods of the time (e.g. taking the pulse and uroscopy – observing the urine) were effectively useless due to a lack of knowledge about childhood conditions (Demaitre 1977). For this reason, Early Medieval physicians and hospital facilities explicitly limited themselves to the treatment of childhood diseases only after parental medical care options were exhausted (Demaitre 1977; Kroll and Bachrach 1986; Gordon 1991). Kroll and Bachrach (1986) conducted a review of historical references from the Early Medieval period in Europe (pre-1100),

revealing 64 of 371 instances in which parents brought their sick children to monastic sites or shrines, sometimes involving upwards of several hundred kilometres travel (*ibid*). Both direct costs for transport (e.g. draft animals, cart, food for the journey), labour loss, and subsequent donations/payments to hospitals in the form of money, land, animals, the children themselves, etc., and indirect costs (e.g. anxiety, stress) involved in the process were analysed (Figure 5.2.2; Demaitre 1977; Kroll 1977; Kroll and Bachrach 1986; Gordon 1991). This study found no significant difference in the social class (nobility, townspeople, and peasants) of the children brought to monastic institutions for medical provision, suggesting that such care was accessible to all children brought by their parents. Boys, however, were brought to receive medical attention 1.4 times more than girls. This does not necessarily mean that girls were not valued as much as boys as sex-linked frailty also seemed to be significant factor, with 87.5% of the child and adolescent deaths from illness occurring in boys (*ibid*).

Figure 5.1.2 Direct and indirect costs involved with seeking medical or surgical care for children in the Early Medieval period (after Demaitre 1977; Kroll and Bachrach 1986; Gordon 1991; Rawcliffe 2006).

MEDIEVAL LEPROSARIA- CARE OR CONFINEMENT?

Evidence embedded in the recorded lives of saints, homilies, and other ecclesiastical documents indicate the lengths parents went to secure treatment for their children, but do not discuss many details of the nature of this care. Both documentary and archaeological evidence for daily life within leprosy hospitals is scarce, and most previous research has relied upon syntheses of geographically and chronologically broad and anecdotal data sets, leading to interpretations that these institutions were neglectful communes for expelled and unwanted individuals on the fringes of society, with negligible care after admittance (Brody 1975: 68-75; Richards 2000: 48-53; Orme and Webster 1995: 24-31). However, this is at odds with the *Rule of St. Benedict* – the primary operational framework for monastic hospitals and leprosaria in England from the mid-10th century AD.

From 964 AD, major Benedictine reforms codified by Bishop Aethelwold of Winchester required all monastic institutions to operate under the *Rule of St. Benedict*, mandating they establish an infirmary and place the care of children and elderly above and before all else, especially with regard to adequate provisions (beds, warmth, clothing, baths, etc.) and a balanced diet (Orme and Webster 1995: 17-23; Clarke 1931:57-58; Rawcliffe 2006: 322-377; Roffey 2012). The implementation of the *Rule* also extended to monastic leprosaria (Rawcliffe 2006: 322-337; Roffey 2012), and references within the *Rule's* chapters reveal particulars about the admittance and long-term care for young people within the leprosarium. For example, Chapter 59 details the process for transferring young people into a monastic institution (i.e. oblation), specifying that parents were obliged to offer their children in person and sign a parental contract for transfer, which may have taken significant effort with direct and indirect costs (Clark 1931: 86-88; Kroll 1977; Demaitre 1977; see above). Additionally, Rawcliffe (2006) cites several examples of wealthy parents (e.g. Brian de Insula, Elinald of Clare) specifically (and very publicly) founding leprosaria for the admission and care for their infected children, as well as the desperation of parents to ensure their beloved children were given a space within a Medieval leprosarium after home-care solutions were exhausted (130-131, 292-293). These accounts imply that parents were concerned about the well-being and care for their leprous children, and were not seeking their admittance into a leprosaria as a means of familial expulsion.

Chapter 36 in the *Rule of St. Benedict* specifically directs monasteries to welcome and treat the sick with the same care and compassion as they would treat Christ, and to give them special allowances with regard to normally prescribed bathing and food routines; i.e. feeding them meat instead of fish, and not fasting if they are seriously ill (Clarke 1931:56-57). In Chapter 37, this regulation is further extended to oblates stating, "on no account let the strictness of the *Rule* in respect to food (i.e. fasting and prescribed monastic diets) be held to as regards [young people], but let there be gentle consideration exercised in their case and let them anticipate the regular hours for meals (Clarke 1931: 58)." The *Rule* also mandated that young people and the infirm have a special caregiver (i.e. alloparent) to oversee their needs within the institution (Clarke 1931:51-52; Orme and Webster 1995:17). Although it is not possible to ascertain the extent to which the *Rule* was strictly adhered to, there is no reason to doubt that these components would have been part of a leprosarium's operational framework. Rawcliffe (2006) extensively makes the case for English leprosaria as beneficial places to be for those with leprosy, and underscores the level of social and cultural support required to maintain these institutions. Leprosaria and people with leprosy were highly favoured beneficiaries of kings, queens, noblemen, bishops, and abbots, who paid patronage in the forms of monetary compensation, donations of food, land, and palliative care (e.g. publicly washing feet and cleaning sores) (*ibid*: 302-314). People with leprosy were venerated as holy penitents, and, in return for a benefactor's patronage, daily prayers were offered by the leprosarium to secure the benefactor's place in heaven (Roberts 1986; Rawcliffe 2006: 322-377). Further, in the post-conquest period (1066 AD onwards), Huggon (2018) estimates approximately 1100 hospitals operated in England and Wales, with approximately one-third functioning as leprosaria, which either suggests a major public health crisis, and/or lends support that leprosaria were not unpleasant places, offering some stability and protection during major cultural and political transitions. Beyond these examples, specifics regarding individual levels of care with a leprosarium are absent, so in order to assess the types of care required for individuals with leprosy, we must explore the biological impacts of the disease.

LEPROSY IN YOUNG PEOPLE – PRESENT AND PAST

Leprosy, also known clinically as Hansen's Disease or more recently, Mycobacterial neurodermatosis (Butlin and Lockwood 2020), is a bacterial infection caused by either *Mycobacterium leprae* or *Mycobacterium lepromatosis*. Clinically, leprosy is a disease of the peripheral nervous system, affecting the skin, extremities, vocal and respiratory tracts, mucous membranes, eyes, and kidneys (Walker and Lockwood 2006). Once a person is infected, the mycobacterium multiplies slowly, leading to a long incubation period ranging from one to twenty years (World Health Organization 2019). The 'type' of leprosy a person develops manifests is encompassed within a broad immune spectrum, ranging from the highly resistant paucibacillary or tuberculoid form to the low resistant multibacillary or lepromatous form (Walker and Lockwood 2006; Lastoria and Abreu 2014). Leprosy has a long biological and social history, and today can be associated with stigma and isolation in endemic areas. Although notions of leprosy invoke anachronistic images of Medieval Europe, the disease is still very much part of the infectious landscape with approximately 200,000 new people diagnosed with leprosy in 2018 (World Health Organization 2019).

Leprosy can affect individuals of all ages, but leprosy in younger people is considered rare, likely due to the lengthy incubation periods associated with the manifestation of the disease. Since 2005 the proportion of children (<18) infected with leprosy out of the total infected population is between 9-35.5% (Butlin and Withington 2018). In comparison to adults, children are at increased risk of developing the more severe, lepromatous form of leprosy and subsequent permanent disabilities, with some infected communities showing 80.5% of children displaying multibacillary signs and symptoms (*ibid*). Although the incidence rates of leprosy diagnoses in children have halved from 2005-15 (407,791 to 210,740), issues with delayed diagnosis, inadequate nutrition, immunodeficiencies, and endocrine system disruption as a consequence of puberty, complicate elimination efforts (Davey and Schenck 1964; John et al. 2005; Butlin and Withington 2018). In these endemic communities where leprosy notably affects children, treatment, monitoring, and de-stigmatisation are of utmost importance for familial, community, and hospital care networks (Butlin and Withington 2018). Although leprosy and subsequent care is readily documented for children today, historical and archaeological evidence of leprosy

in young people is much more scant due to a lack of detailed sources and archaeological contexts.

Identifying Leprosy in the Past

In current popular mind-sets, the idea of leprosy during the Medieval period invokes images of stigma and expulsion. However, the assumption of a widespread hostility towards people with leprosy in the past is primarily anecdotal, and largely an artefact of conquest and racism in later colonial years (Rawcliffe 2006:13-29; Edmond 2006:61-109). Aggressively deleterious isolation campaigns led by Albert Ashmead in the late 19th and early 20th centuries led to worldwide legislative changes and helped to solidify an ingrained prejudice against people with leprosy, past and present (Ashmead 1895; 1897a; 1897b; 1897c; 1897d; 1899; 1901a; 1901b). Many sources trace leprosy back to the medical treatises of Hippocrates and Galen, and in stories of moral character found within the Bible (Adams 1868; Browne 1975; Johnston 2006). However, the 'leprosy' described in these and other Classical sources depicts a different condition (e.g. a range of skin diseases such as vitiligo, eczema, psoriasis, and impetigo) to the bacterial infection we now ascribe as leprosy. Some descriptions of the modern-day iteration of leprosy can be found in ancient texts of India and China, and in the Roman Empire in the 1st century AD, prompting some scholars to hypothesize its spread from the East along the Silk Road as a consequence of Roman Trade (Bhishagratna 1963; McLeod and Yates 1981; Mark 2002; Binder 2018). Early Roman (1st century AD) medical scholars described leprosy as a skin condition causing white patches on the skin, and, although today the signs of leprosy often begin with a skin lesion, the ensuing pathogenesis is more like the Roman descriptions of the disease *elephantiasis graecorum* (Demaitre 2007: 86-91). Following the split of the Roman Empire, these Roman medical sources fell out of favour in the West, but continued to develop within the Byzantine Empire in the East. Empirical Arabian medical practices surpassed Western superstitions as a means of treatment, culminating in a shift of medical thought at the School of Salerno. This primarily occurred when Constantine the African (a North African Benedictine monk and physician) translated Arab texts into Latin in the late 11th century AD (Conrad et al. 1998: 140-141; Demaitre 2007: 86-91; Miller and Nesbitt 2014: 21-22). In the course of these

translations, the extreme form of *elephancia* (what we now know as lepromatous leprosy) was translated and named *lepra*, thereby connecting the clinical condition with the moral condition pervasive in Medieval thought (Rawcliffe 2006:76; Demaitre 2007:87). Because this nexus occurred in the late 11th - early 12th centuries AD, the importance of understanding leprosy as a social condition in England in the pre-Conquest era is important to gauge a more accurate societal response to the disease.

BODIES OF EVIDENCE - ARCHAEOLOGICAL EXAMPLES OF LEPROSY IN YOUNG PEOPLE

At present, only a handful of archaeological skeletons of young people displaying bone changes of leprosy exist. Isolated reports of leprosy in children and adolescents have been recorded in Scotland (2280-1970 BC), Italy (2nd – 3rd centuries AD), Turkey (8th – 10th centuries AD), Czechia (9th – 10th centuries AD), in Northern England (10th century AD), Croatia (10th – 11th centuries AD), and Sweden (10th – 12th centuries AD) (Roberts 2007; Mays 2007; Economou et al. 2013; Rubini et al. 2014; Donoghue et al. 2015; Bedic et al. 2019). The inclusion of these young people within the normative cultural and burial practices for the groups associated with these sites may suggest that notions of stigma associated with leprosy were not as commonplace in the past as they are today. In further support of this view, Roberts (2002) surveyed 41 archaeological sites from the Roman to post-Medieval periods in Britain that yielded individuals with skeletal lesions consistent with leprosy, and found that 36/41 sites revealed individuals buried within the normal confines of their communities. The remaining five examples were attached to leprosy hospital sites, which tended to produce multiple individuals with leprous bone changes (*ibid*). Although leprosy hospital sites usually of the Late Medieval period (i.e. post-12th century AD) tend to produce higher numbers of skeletons showing leprosy (e.g. Magilton et al. 2008:11-12, 95), other Early Medieval contexts that reveal high percentages of individuals with leprosy within normal contexts do exist. For example, Anderson (1998) and Shepherd Popescu (2009) have reported a significant number of skeletons with leprosy (23%), including adolescents, at the Late-Saxon Timberhill site in Norwich, Norfolk, England (980-1050 AD). Communally inclusive burial contexts such as this should be borne in mind when considering the social reactions to young people with

leprosy in the past, but in order to assess levels of care and treatment, leprosaria contexts also need to be viewed.

Adolescents in Medieval Leprosaria

Danish physician Møller-Christensen first detailed the bone changes associated with lepromatous leprosy in the human remains excavated from the Late Medieval (1250-1550 AD) Naestved leprosy hospital site in Sjaelland, Denmark (Møller-Christensen 1961). Møller-Christensen noted that just under 20% of young people with leprosy (<18 years) displayed skeletal lesions indicative of lepromatous leprosy, which he interpreted as evidence of a high degree of endemicity of the disease during this time (Møller-Christensen 1961; 1978). Approximately 20% of all burials from the cemetery of the Late Medieval St. James and St. Mary Magdalene leprosy hospital (12^{th} – 15^{th} centuries AD) in Chichester, Sussex, England displayed skeletal lesions consistent with leprosy; however, none of the 104 children and adolescents demonstrated any diagnostic bone changes indicative of leprosy (Lewis 2002; Lewis 2008:174-176). This may indicate a change in medical provision by increasing diversity amongst the generally infirm in the Later Medieval period, rather than remaining a purpose-built institution for people with leprosy. Conversely, it may indicate changes in medical practice and a declining ability to distinguish the clinical signs and symptoms associated with leprosy from other conditions. It is also important to consider the Osteological Paradox (Wood et al. 1992), which acknowledges that not everyone who contracts a disease will manifest skeletal lesions and that individual responses to the disease are complex.

In contrast to St. James and Mary Magdalene, the North Cemetery from the St. Mary Magdalen leprosy hospital (9^{th} – 12^{th} centuries AD) in Winchester (Figure 5.1.3) reveals the highest prevalence (~86%; 38/44) of individuals displaying signs of lepromatous leprosy from any cemetery or leprosarium site. Within this cemetery were a significant number of adolescents (~58%), most of whom showed diagnostic evidence for lepromatous leprosy (Roffey and Tucker 2012; Table 5.1.1).

TABLE 5.1.1 – Adolescents excavated from the North Cemetery of the St. Mary Magdalen Leprosarium (c. 9^{th} – 12^{th} centuries AD; Winchester, UK).

INDIVIDUAL	AGE (YEARS)	SEX	BONES AFFECTED BY LEPROSY
SK. 8	8.5-9.5	Male	Facial bones, Hands, Legs
SK. 9	22.5-23.5	Male	Hands, Feet, Legs
SK. 14	16-19	Male?	Facial bones, Hands, Feet, Legs
SK. 15	20.5-22.5	Male	Hands, Feet, Legs
SK. 16	18-25	Male	Facial bones, Feet, Legs
SK. 17	18-25	Female	No leprous changes
SK. 18	14.5-16.5	Male?	Facial bones, Hands, Feet, Legs
SK. 19	c. 25	Male	Facial bones, Hands, Feet, Legs
SK. 21	19-25	Male?	Facial bones, Hands, Feet, Legs
SK. 25	17-19	Male	Facial bones, Hands, Feet, Legs
SK. 26	18-25	Male	Facial bones, Feet, Legs
SK. 27	22.5-23.5	Male	Feet, Legs
SK. 28	12.5-14.5	?	Facial bones, Feet, Legs
SK. 29	18-25	Male	Facial bones, Hands, Feet, Legs
SK. 37	13-16	?	Feet
SK. 38	18-25	Female?	Feet, Legs
SK. 39	18-25	Male	Facial bones, Feet, Legs
SK. 41	13-16	?	Facial bones, Feet, Legs
SK. 45	15.5-16.5	?	Facial bones, Feet, Legs
SK. 46	16-19	Male	Facial bones, Feet, Legs
SK. 52	12.5-13.5	?	Facial bones, Hands, Feet, Legs
SK. 54	14.5-15.5	?	Facial bones, Hands, Feet, Legs
SK. 56	16.5-17.5	Male	Facial bones, Hands, Feet, Legs

THE LEPROSY HOSPITAL OF ST. MARY MAGDALEN (WINCHESTER, UK)

The St. Mary Magdalen leprosy hospital is presently the oldest documented leprosy hospital in Britain. Documentary evidence from the 1148 Winton Domesday reference it

was in operation as a leprosarium under Bishop Richard of Illchester, but timber structures that underlie the 12th century masonry and subsequent radiocarbon dates (Late-9th to mid-12th centuries AD) indicate earlier establishment and use (Roffey and Tucker 2012; Roffey 2012). Within the site are separate cemeteries that can be associated to particular chronologies; the North Cemetery, which is associated with the pre-12th century AD timber phase, and the South Cemetery, which is associated with the post-12th century AD masonry phase. Despite the clear association with the building phases of the hospital, there is also an archaeological distinction in the burial treatments of individuals between the North and South Cemeteries, which may reveal shifts in cultural attitudes post 12th century AD. Prior to the excavations at St. Mary Magdalen, leprosaria were thought to be a Norman development (Roffey 2012; Roffey 2017), and although the establishment of leprosaria within Britain sharply increase from the 12th century AD (Roberts 1986), the presence of a leprosarium that predates the Norman Conquest is noteworthy.

HANDLED WITH CARE – THE BURIAL CONTEXTS

The skeletons excavated from the North Cemetery of St. Mary Magdalen leprosy hospital were very well- preserved and revealed an unusually high prevalence of skeletons with lesions consistent with or diagnostic of lepromatous leprosy (38/44), of which, 58% were adolescents (n=22) (Table 5.1.1). The majority of those buried in the North Cemetery were interred in single, anthropomorphic graves with westward-facing head niches and earthen pillows (i.e. inner ledges to elevate their heads), with the exception of Sk. 14 (radiocarbon dated cal AD 995-1033) who was buried in a coffin (Roffey and Tucker 2012). The anthropomorphic grave cuts and head niches are normally reserved for high-status ecclesiastical sites and show a considerable degree of care and effort went into creating a final “resting place” for them (Roffey and Tucker 2012). Some of the graves within the North Cemetery also contained burial goods, which is a relatively rare phenomenon in Christian cemeteries, but does help to highlight social and individual identities. For example, Sk. 27 was buried with a pilgrim badge that he presumably obtained from the shrine of St James, in the Santiago de Compostela Monastery in Spain (Roffey et al. 2017), and Sk. 19 was buried with adapted feeding elements (e.g. a

modified feeding bowl) associated with the likely difficulties (e.g. loss of hand function due to flexure contractions and resorption; Figure 5.1.6) this particular individual had with eating, suggesting a level of individualised, palliative care at St. Mary Magdalen (Roffey et al. 2017; Roffey and Tucker 2012).

This care in the burial of individuals with leprosy in the North Cemetery appears to dissipate in the South Cemetery. Here, the burials associated with the 12th century AD masonry phases are on different alignments and comprise more haphazard burial treatments for all individuals (e.g. multiple and truncated burials with no anthropomorphic grave cuts), implying some form of cultural change (Roffey and Tucker 2012). The individuals in the South Cemetery also revealed a lower prevalence rate of lepromatous leprosy (40%) (*ibid*), perhaps indicating that the response to leprosy led to a less-severe form during later periods was less-severe (e.g. Tuberculoid leprosy), or that leprosy as a disease was more poorly identified after the Norman Conquest. It may also be a reflection of the decline of leprosy from the 14th century AD onwards, possibly due to the rise of other infectious diseases such as the Black Death and tuberculosis (Manchester and Roberts 1989; Manchester 1991; Roberts 2002; Crespo et al. 2019; Roberts 2020: 291-301).

Figure 5.1.3 a) Location of St. Mary Magdalen Leprosy Hospital (Winchester) and b) aerial view of the excavations of the North Cemetery.

In order to elicit skeletal changes as a consequence of a pathological stimulus, a person must have a disease for long enough before death for the bone changes to occur (Wood et al. 1992). Therefore, when viewing the skeletal indicators of a chronic infection, we must bear in mind that the individual's immune system was strong enough to fight the acute stages of the disease for some time before the hard tissues were affected in the later chronic stages (*ibid*). This associated chronicity of disease implies that the individuals possessed a better health status in order for the inflammatory response associated with immune function to be prolonged. This is especially pertinent when viewing the severe and potentially debilitating bone changes associated with lepromatous leprosy. Many of the skeletal lesions present in the child and adolescent skeletons from the North Cemetery of St. Mary Magdalen are highly consistent with lepromatous leprosy. These include pathognomonic rhinomaxillary changes (resorption of the anterior nasal spine, remodelling of the nasal margins, abnormally porous/new bone formation on the oral and nasal surfaces of the palatal bones, destruction of the inferior nasal conchae and vomer, abnormal porosity and resorption of the alveolar process (Figure 5.1.4), acro-osteolysis/resorption and concentric atrophy of the hands and feet (destruction and remodelling of the phalanges, metacarpals, and metatarsals), mediolateral remodelling of the metatarsal shafts, resorptive grooves on the palmar surfaces of the hand phalanges (or termed volar grooving – Andersen and Manchester 1987) caused by flexion contractures, tarsal fusion and dorsal exostoses (Andersen and Manchester 1988, and subperiosteal new bone formation on the distal shafts of the tibiae and fibulae. Four individuals (SK. 8, SK. 28, SK. 52, SK. 56) also showed (rare) evidence for leprogenic odontodysplasia, which is the concentric constriction and dysplastic development of the anterior maxillary dentition caused by leprosy contraction in early childhood (Danielsen 1970; Reichart 1976). The development of leprogenic odontodysplasia and the pathological skeletal lesions associated with lepromatous leprosy are presumed to commence at approximately the same time (Ortner 2008), revealing a more defined chronology for the onset of skeletal changes and the time elapsed before death.

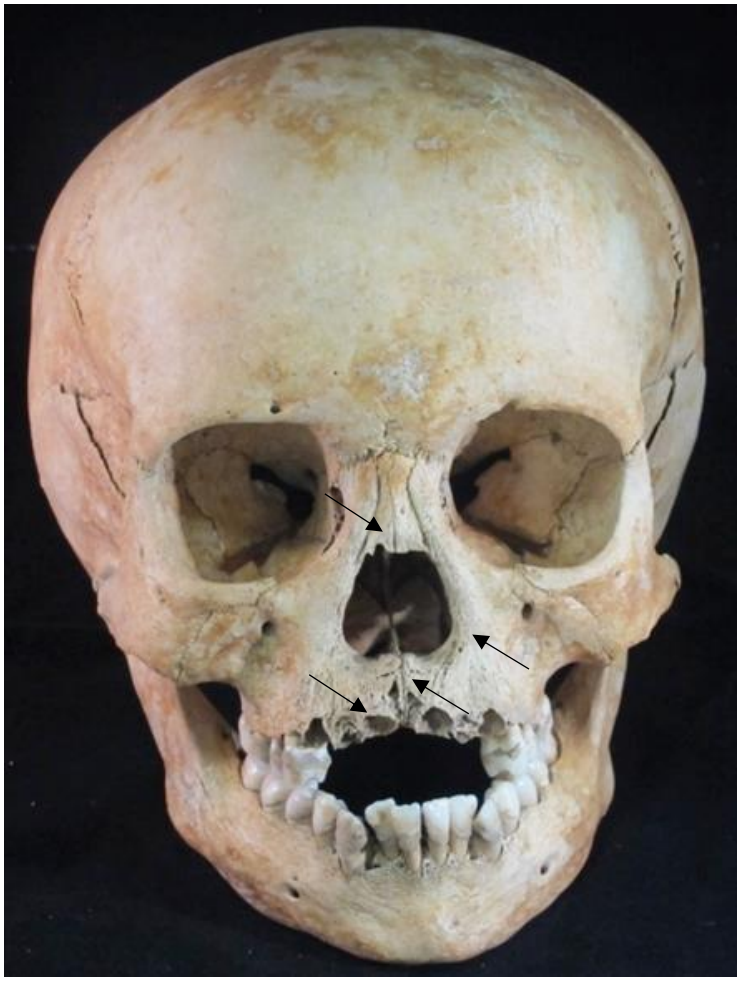

Figure 5.1.4 Skull of Sk. 52 from the St. Mary Magdalen Leprosy Hospital (Winchester) displaying evidence of rhinomaxillary syndrome including rounding of the nasal aperture, resorption of the anterior nasal spine, recession of the alveolar margin, and widening and flattening of the nasal bones.

Although St. Mary Magdalen was a dedicated facility for individuals with leprosy, it is important to note that a human body experiencing leprosy is also open to other health problems, and there were several concomitant pathologies identified in the skeletons excavated that showed signs of leprosy. All of the adolescents from the hospital possessed at least one non-specific indicator of childhood stress (linear enamel hypoplasia and cribra orbitalia), and eight of them yielded a higher dental development age in comparison to their skeletal age; both these observations potentially indicate arrested development as a consequence of the disease but other aetiologies could also be important, for example a poorly balanced diet. Other comorbidities included, pathologically induced fractures, possible tuberculosis or mycotic infections, residual

rickets, osteoporosis, and a person with evidence of a lower leg amputation, likely the result of a disease affecting the leg. Amputation is particularly relevant because amputations are an indicative aspect of interventional palliative care (Roffey and Tucker 2012; Tilley 2017). All individuals also displayed moderate to high levels of dental calculus formation (i.e. mineralised plaque), which is commonly found amongst modern lepromatous leprosy patients and may be an indicator of poor oral hygiene resulting from inflammation of the oral cavity, mouth-breathing due to facial paralysis and/or chronic inflammation of the nasal passages, or a softer, mushy diet (Reichart 1976; Ogden and Lee 2008; Souza et al. 2009; Rawlani et al. 2011; Roffey et al. 2017).

Bioarchaeological Interpretations of Care and Treatment of Leprosy

In order to examine care and treatment in the past, bioarchaeologists customarily study the treatment of the dead through burial contexts, pathological lesions, and evidence of palliative care (Roberts 2018). The Index of Care Framework (Tilley and Cameron 2014; Tilley 2017) has been used more recently to provide a holistic platform in which to assess the care (clinical and communal) a person would need to survive in a society based on the pathological lesions present on the skeleton, and what might have been provided. Roberts (2017) applied this Index of Care to a male aged 25-35 who had bone lesions related to leprosy and who was buried in the cemetery of St. James and Mary Magdalene leprosy hospital at Chichester (12^{th} – 15^{th} centuries AD). On the basis of the bone lesions and their chronicity (e.g. rhinomaxillary syndrome, acro-osteolysis of the hands and feet, tibial and fibular subperiosteal new bone formation), Roberts (*ibid*) demonstrated that he would have likely required significant personal palliative care, but that there was insufficient contextual data to indicate that he actually received that care within the leprosarium (*ibid*). Indeed, the process of using the Index of Care in an archaeological context can be fraught with uncertainty due to the incomplete nature of the data. Notwithstanding, if the Index of Care framework can stand to scrutiny in terms of the data, analysis, and interpretation, whilst acknowledging the inherent limitations, it might be used to support the historical evidence that people with leprosy were indeed cared for in the past. Given that this person (C148), amongst others buried at the site, dates from a period that allegedly was at the height of leprosy isolation (Roberts 1986; Roberts

2002), it is worth investigating if this, and other, models apply to individuals from earlier contexts to explore whether a continuity of care exists.

Facing Lepromatous Leprosy – An Index of Care

As Tilley (2017: 11-12) asserts, provisioning for those affected by illness is a common human behaviour through time, but is accompanied by physical and psychological stress for the caregiver. Therefore, the skeletal remains of individuals who are supported during chronic, debilitating illnesses also reflect the willingness, experience, knowledge, beliefs, politics, economic status, and compassion of the caregivers, and societal responses, to diseases during their lives (*ibid*). However, crafting a framework that supports these variables with a view to the past is not without complications (Tilley and Schrenk 2017; Tilley 2017). In order to lessen these complications, a bioarchaeology of care methodological approach was developed to provide a multi-staged, case-based research framework to demonstrate whether care was provisioned for an individual, or if they were left without medical and societal support (Tilley and Cameron 2014). Following the methods of Tilley and Cameron (2014) and Tilley (2017), the Index of Care online platform (indexofcare.org) was applied to Sk. 19 (the individual at St. Mary Magdalen with the most severe and likely disfiguring changes) to assess the clinical impacts and functional implications of his experience. In doing so, the research tested whether a model of care could be constructed, and whether broader implications regarding whether a group agency model of provision could be ascertained. The results of this assessment are presented in Tables 5.1.2-5.1.4.

Sk. 19 was a c.25 year-old male excavated from the North Cemetery, whose burial context revealed evidence of shrouding (i.e. a copper alloy shroud pin) and pottery vessels adapted for feeding (Roffey and Tucker 2012). The remains of Sk. 19 showed bone changes diagnostic of advanced lepromatous leprosy. These include rhinomaxillary changes such as flattening, fusion and resorption of the anterior of the nasal bones, rounding, thickening and resorption of the margins of the nasal aperture, complete resorption of the maxilla, including the anterior nasal spine, back to the first molars, and resorption of the hard palate with porosity of the remaining bone (Figure 5.1.5).

Figure 5.1.5 Skull of Sk. 19 demonstrating advanced rhinomaxillary syndrome including widening and fusion of nasal bones, widening and remodelling of nasal aperture, and complete loss of the anterior nasal spine, alveolar process of the maxilla, and hard palate.

Other significant bone changes include changes to the hands such as concentric diaphyseal remodelling of the mid-shafts of the metacarpals; sharp-edged “scooped-out” lesions around the metacarpal heads; flattening of the first metacarpal heads, subperiosteal new bone formation on the metacarpal shafts with a probable fracture of the right fifth metacarpal, partial and/or complete resorption of the distal phalanges; concentric diaphyseal remodelling of the mid-shafts of the proximal phalanges; and volar grooving of the proximal phalanges indicating flexion contractures (Figures 5.1.6-5.1.8).

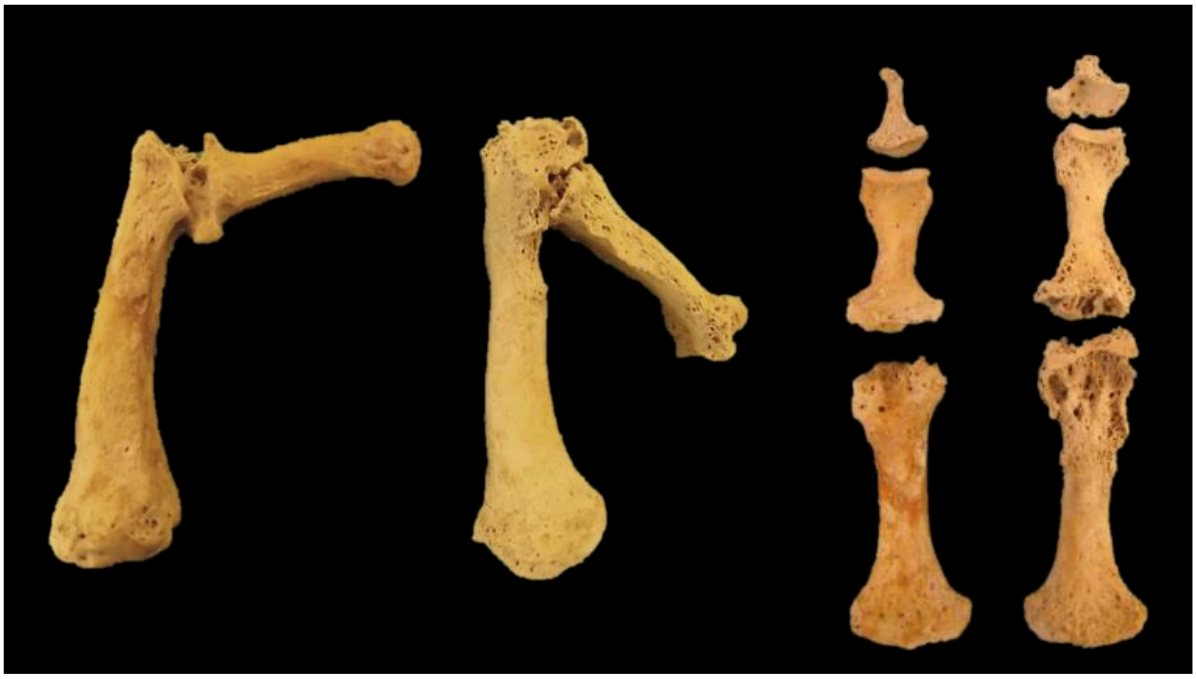

Figure 5.1.6 Hand phalanges of Sk. 19 showing volar grooving likely indicative of long-term flexion contractures.

Changes to the lower limbs and feet included resorption of the bones of the right foot to the proximal bases of the metatarsals (Figure 5.1.7), destruction and fusion of the cuneiforms, cuboid, and navicular, porosity of the posterior of the right calcaneus, as well as lamellar and woven subperiosteal new bone formation along the tibial and fibular diaphyses.

Figure 5.1.7 Right foot of Sk. 19 demonstrating bone resorption to proximal metatarsals and fusion of tarsals.

The left foot was absent and there was diffuse lamellar, woven and subperiosteal new bone formation on the tibia and fibula, showing a tapering towards the distal ends of the fibular diaphysis. The distal epiphyses of the tibia and fibula are absent antemortem, and the distal diaphysis of the tibia is flattened with rough, porous cortical bone and the remains of the medullary cavity in the centre. The bones are not atrophied but the cortical bone is greatly thinned and they are ankylosed at the distal end by bony bridging. This appears to be a deliberate amputation (Figure 5.1.8).

Figure 5.1.8. Left lower limb of Sk. 19 showing amputation at the distal end.

Although it is currently not possible to know whether Sk. 19, and indeed the other individuals found at St. Mary Magdalen were long-term patients or simply buried there at death (as suggested by Roberts 2017 in her study), the combination of skeletal lesions, demographic makeup, and burial and archaeological context indicate that people buried there had advanced signs of lepromatous leprosy by the Late Saxon period in Winchester, and that leprosy was affecting a large portion of younger individuals buried at St. Mary Magdalen leprosarium. As previously mentioned, these individuals were buried in a manner usually reserved for high-status ecclesiastical sites, but with the retention of burial goods indicating aspects of individual identity (e.g. pilgrim badges, individual feeding implements) (Roffey and Tucker 2012).

The clinical impacts comprised within Stage 2 of the Index of Care framework indicate that all bodily systems/function (Figure 5.1.9) could have been affected by lepromatous leprosy. Further, aspects of daily living as detailed in the Index of Care framework (Table 5.1.2) indicate that this man likely required assisted care based on his bone changes (Figures 5.1.5-5.1.8).

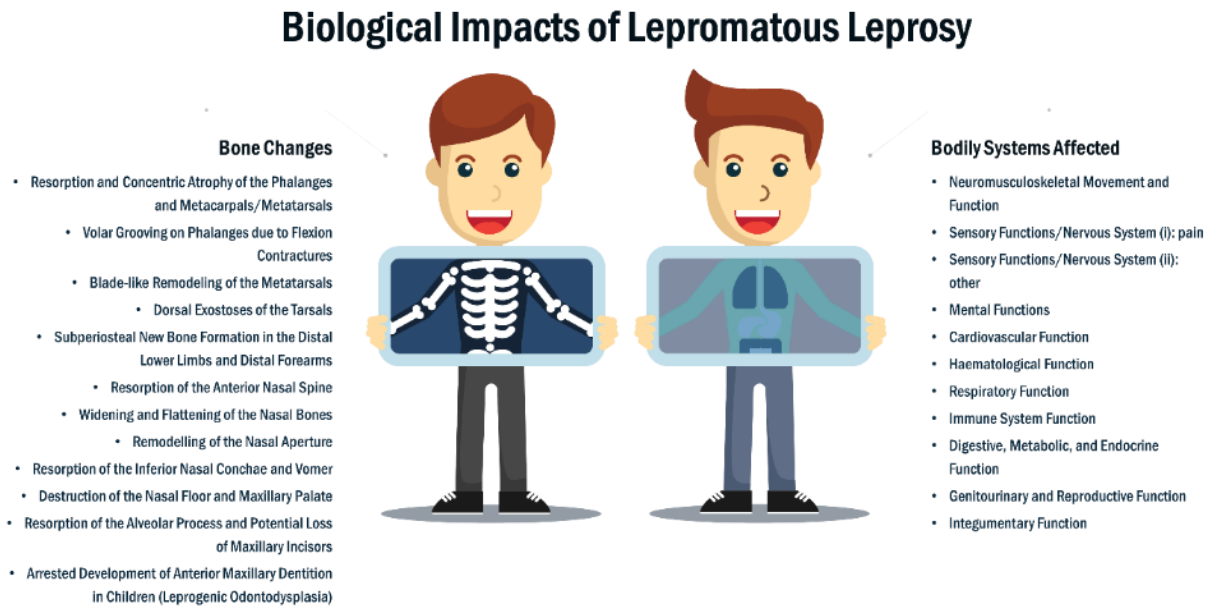

Figure 5.1.9 Biological consequences of having lepromatous leprosy, including skeletal changes and body systems affected (Walker and Lockwood 2006; Ortner 2008; Lastoria and Abreu 2014). Author's own.

TABLE 5.1.2 – Impact of Leprous Bone Changes on Aspects of Daily Living for Sk. 19 and individuals with similar bone changes when they were alive.

Activities of Daily Living

BASED ON OBSERVED BONE CHANGES ASSOCIATED WITH LEPROMATOUS LEPROSY

	CAPABLE	INCAPABLE	UNKNOWN	OBSERVATIONS
Self-provisioning: ability to manage access to food and drink unaided (e.g. independently access nearby sources of food and water).		X		Likely incapable. Sk. 19 was missing left and most of right foot. Hand function would have largely ceased due to prolonged flexion contractures and finger resorption as skeletally evidenced.
Self-feeding: ability to physically eat and/or drink without assistance (i.e. to convey food and drink to mouth).			X	Grave goods indicate individual, specialised feeding utensils, that they would not be able to make themselves, but were likely adapted to enable self-feeding. Despite these feeding implements, with the severity of the maxillary destruction, they may have still required assistance with drinking and eating.
Managing basic personal caring for self: (e.g. washing, toileting, preserving skin integrity; treating infection, managing infection risk).		X		Mobility and hand function would have been incredibly impaired. Bilateral flexion contractures of the hands, amputation of left foot and resorption of right foot would have likely necessitated help in management of basic personal care.
Basic object manipulation: ability to manage items in the immediate environment; includes self-dressing, item retrieval, etc.			X	Likely loss of hand and sensory functions would complicate basic object manipulation, self dressing, and retrieval of items. Sk. 19 would possibly have needed assistance for many of these, but the extent to which he needed assistance cannot be determined by his skeletal lesions
Mobility over limited distance: ability to move unaided over short distances (e.g. inside/around dwelling, out of range of potential hazards)		X		The loss of Sk. 19's left foot, almost complete resorption of right foot, and compromised hand function would likely require some form of aid to move any distance.
Control over body position: ability (re)position body parts as desired without assistance (e.g. to sit up and transfer body weight from a reclining position unaided).			X	This cannot be ascertained with the skeletal evidence available and is therefore, unknown.

TABLE 5.1.3 – Model of care considering the probability of need for direct support required from other people to survive with the bone changes present in Sk. 19.

Need for Direct Support

Probability of Support Needed from Others

COMPONENTS OF CARE PRACTICE	NEED FOR CARE	COMMENTS	EFFORT/RESOURCES INVOLVED
Provision of food and water	Probable	Buried with specialised feeding equipment	Work undertaken to gather, cook, and provision food and water for individuals.
Maintaining normal body temperature	Probable	Was found in a leprosy hospital cemetery, with the interpretation they received care there.	Effort undertaken to provide shelter, clothing, bed, and warmth to individual.
Facilitation of comfort, rest, and sleep	Possible	The bone changes make it unlikely for Sk. 19 to be able to build his own bed, weave a blanket, or start a fire on his own.	A bed, blanket, and shelter would have likely been supplied, potentially long-term.
Maintaining/assisting mobility	Probable	Sk. 19 had significantly compromised mobility, and would have needed assistance in the most basic tasks.	Sk. 19 needed substantial assistance to be mobile, and would have likely required aid to travel any distance.
Monitoring health status	Probable	Had evidence of amputation, implying palliative care and possible surgical care was available to him at one point.	Likely palliative provisions including hygiene and bandaging supplied for this person to avoid further infection.
Maintenance of personal hygiene/protection of integument	Probable	Without hand/foot function, would probably need assistance with hygiene and self-care.	Sk. 19 likely needed assistance with hygiene and protection of sores/open wounds (e.g. amputation) and dressings to avoid subsequent infection.
Physical manipulation/postural adjustment	Possible	No evidence of decubitis lesions indicates no long-term pressure/bed sores	Sk. 19 may have needed assistance in/out of bed without the full use of his hands and feet.
Maintenance of physiological functioning	Unknown/Probable	It is unknown what specific of medical provisions were specifically available in the St. Mary Magdalen leprosarium.	It is likely that some medical and food provisions (medical and nutriment) contributed to individual physiological functioning
Specific intervention(s) and technologies	Probable	Evidence for the amputation of left foot	Would have required skilled medical practitioners to treat.

TABLE 5.1.4 – Model of care considering the probability of need for accommodation and support across multiple divisions.

Probability of Need for Support

	DOMAIN	NEED FOR CARE	COMMENTS	EFFORT/RESOURCES INVOLVED	EVIDENCE FOR EFFICACY OF CARE
	DOMESTIC	Probable	Sk. 19, and others with similar bone changes, might be able to contribute to domestic life with supported adaptations or activities.	Near the end of Sk. 19's life, they were less likely to contribute to domestic activities due to the severity of their leprosy.	Care was effective enough for Sk. 19 to live long enough to develop skeletal lesions.
	MOBILITY	Probable	Sk. 19 would have required substantial assistance to travel any distance.	If Sk. 19 had to travel with his end of life pathological lesions, he would have needed assistance to go any distance, and possibly to be transported by cart.	Unknown
	ECONOMIC	Probable	Buried within a leprosarium context with the interpretation that he was resident there, with food and shelter provided.	Food, shelter, blanket, chairs, assistance devices, etc. would be required for Sk. 19.	Care was effective enough for Sk. 19 to live long enough to develop skeletal lesions.
	BASIC LIFESTYLE	Probable	Found in context with leprosy hospital	Would have needed provision for food and water, including obtaining, cooking, probable assistance eating, probable assistance drinking, etc.	Care was effective enough for Sk. 19 to live long enough to develop skeletal lesions.
	COMMUNITY	Possible	Sk. 19 possibly needed individual or social support to participate within the community.	Any community interactions would likely require someone to assist Sk. 19 with movement and potentially verbal engagement.	Unknown

When considering components of care practice with regard to needs for direct support and accommodation (Table 5.1.2, Table 5.1.3), Sk. 19 and others with similar skeletal lesions associated with lepromatous leprosy likely needed long-term clinical, medical, and economic infrastructures to survive (see Figure 5.1.10. – Group Agency Model).

From these aspects of bioarchaeological enquiry, particularly through the Index of Care model, it can be interpreted that people with leprosy were likely provided with care at St. Mary Magdalen, and not necessarily neglected. In order to facilitate this type of care at the leprosarium level, wider economic and cultural resources are needed to enable a group agency model for health-care provision (Figure 5.1.10), meaning these provisions must have been sanctioned and supported at a wider societal and administrative level. Despite the Index of Care framework demonstrating that individuals at St. Mary Magdalen were likely cared for (in the form of medical and daily support), we must bear in mind that people do not experience disease in the same way (Ortner 1991: 7-11). For example, 10 people with leprosy can have a range of impacts from the disease that will not all be the same, or with similar levels of severity or disability. In addition, the immune spectrum of leprosy ranging from high to low resistance and other types in between will show different impacts on people today compared to pre-biotic eras.

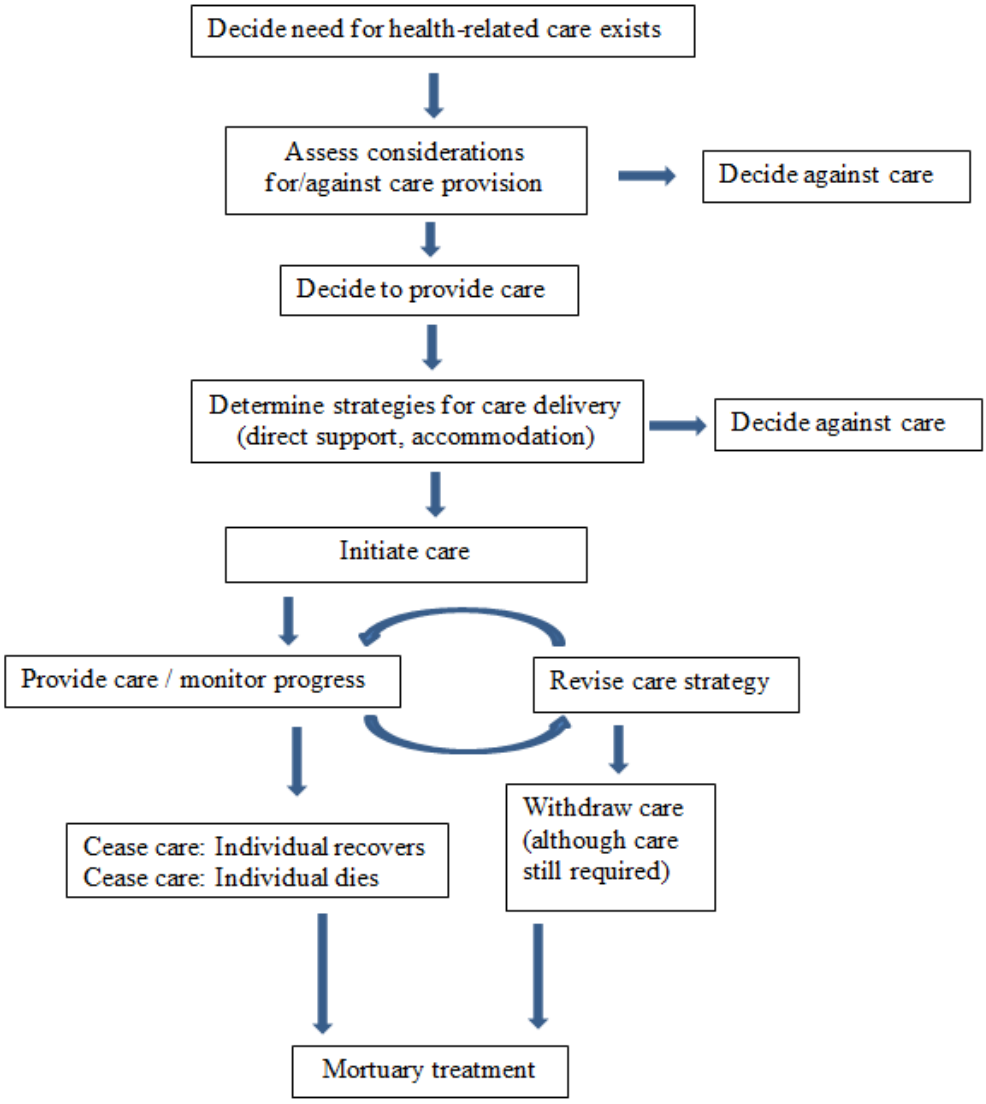

Figure 5.1.10. 'Decision Path' in the health-related caregiving process for a group agency infrastructure (From IndexofCare.org).

Discussion and Conclusions

This research aimed to explore the social and biological impacts of young people with leprosy in Early-Late Medieval England through a thematic approach of alloparental care and an evaluation of evidence for care and treatment. Historical documents detail that in seeking medical provision for their children, parents were active participants in seeking and negotiating care, and not peripheral bystanders. Benedictine Rule also likely required parental involvement upon their admission into the hospital and mandated provisions

and a duty of care once under the alloparental umbrella of the monastery. Although contemporaneous historical records do not specifically record social sensibilities towards leprosy in children, interpreting the funerary and skeletal evidence of young individuals displaying advanced signs of leprosy aid in filling in these historical lacunae. Adolescents with skeletal lesions diagnostic of lepromatous leprosy make up the majority of individuals buried in the North Cemetery at the St. Mary Magdalen leprosy hospital site and the deleterious effects of their disease status potentially affects all aspects of daily living including the way they move around, their food preparation, food consumption, etc. This may lead to significant impairment, and within in the archaeological context under study here, support the necessity for monastic-led care provisions via an alloparental/caregiver model for these adolescents.

The Index of Care Framework has previously been applied to an adult male from Late Medieval Chichester to further draw together several lines of clinical, sociological, and bioarchaeological evidence of care (Roberts 2017). This model of care was replicated for Sk. 19 from the St. Mary Magdalen leprosy hospital. The results indicate that a complex, group agency model of care likely existed to provide care for these adolescents.

Access to medical care could be difficult in the past and the overall effectiveness of conventional medical treatments likely did not inspire confidence (Kroll and Bachrach 1986). Given the tremendous efforts involved in seeking assistance for ill children, including transport, provision of care, and parental consent for the admittance of young people into these monastic facilities, our understandings should be shifted more towards a positive view of how Early Medieval parents felt towards their children. If people did not 'care' or expelled their loved ones for contracting leprosy, we would expect abandonment and isolation, not potential long-distance travel and admittance into a hospital. This demonstrates how applying multiple models of care can elucidate social responses to disease in the past, and the efforts made to help the weakest amongst them. It also reaffirms the benefits of studying youth as a vulnerable subset of society that can serve as a highly sensitive, more accurate cultural barometer (Redfern and Gowland 2011; Roberts and Bernard 2015; Lewis 2017; Mays et al. 2017).

What this model cannot tell us, however, is the lengths people would have travelled to seek this hospital care, e.g. did they travel from further afield or were they only accommodating adolescents from the local communities? Likewise, we are unable to fully demonstrate how long they spent within the leprosarium before death and what care was like after admittance, e.g. were they fed an adequate diet? In future, the inclusion of multi-isotope analyses can help to add to this growing re-evaluation of past leprosy narratives. Radiogenic strontium and stable oxygen isotope analyses can help to reveal particulars about the mobility histories of individuals to ascertain what the catchment area of places like St. Mary Magdalen in Winchester was (Evans et al. 2010; Evans et al. 2012; Kendall et al. 2013). If people were travelling far distances to the hospital, this may indicate that the treatment afforded was widely-known and that it was not a place of banishment. Likewise, similar analyses of non-leprosaria contexts (e.g. parish cemeteries) revealing individuals with leprosy may help to understand transmission dynamics and community responses. Additionally, stable isotope analyses of carbon and nitrogen from incremental dentine can reveal diets and pathophysiological reactions of young leprosy sufferers from around birth to death if the tooth is still forming, providing some indication of their lived experiences before and after admittance (Beaumont et al. 2013; Beaumont et al. 2015; Beaumont and Montgomery 2016). Because previous assertions about the way people in the past with leprosy were treated have demonstrable effects on people afflicted with the disease today, it is worth exploring these and other lines of evidence to better understand societal reactions to disease in the past and challenge commonly regurgitated and stigmatising disease narratives.

ACKNOWLEDGEMENTS

We would like to thank Dr. Joanna Moore, Professor Janet Montgomery, and the anonymous reviewer for their extremely helpful comments, and Dr. Simon Roffey from University of Winchester for access to the skeletons from St. Mary Magdalen. Additionally, we are very grateful to Drs. Ellen and Ross Kendall for inviting us to contribute to this volume.

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MANUSCRIPT 5.2: HEALTH AT HOME: MOBILITY HISTORIES OF ADOLESCENTS WITH LEPROSY FROM THE LATE SAXON (10TH – 11TH CENTURIES AD) CEMETERY OF ST. JOHN AT THE CASTLE GATE/TIMBERHILL (NORWICH, UK)

Prepared for International Journal of Osteoarchaeology

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ABSTRACT

Leprosy is one of the most notorious diseases in history, widely associated with social stigma and exclusion. This study re-evaluates the evidence for social stigma in relation to leprosy through the isotopic and palaeopathological analysis of adolescent skeletons (10 – 25 years old) from the 10th – 11th century AD cemetery of St. John at the Castle Gate in Norwich (UK). Core enamel samples from premolar and molar teeth from 10 young individuals with diagnostic lesions for leprosy were selected for radiogenic strontium ($^{87}Sr/^{86}Sr$) and oxygen ($\delta^{18}O$) stable isotope analyses. Strontium ratios did not exclude anyone from the regional range; however one individual (13121) revealed a higher oxygen isotope value, likely due to an enrichment of ^{18}O from breastfeeding. The data suggest that those with visible signs of leprosy were buried within their local community and in a normative manner, thus challenging the notion of profound stigma experienced by Medieval people with leprosy. This study underscores the importance of bioarchaeological data in challenging broad historical and archaeological narratives.

KEYWORDS: Infectious disease, Stable isotopes, Strontium, Oxygen, Disease stigma, Early Medieval

KEY SUMMARY POINTS:

- First strontium and oxygen isotope data from human tooth enamel reported for Norwich
- All individuals show strontium isotope ratios consistent with the area
- One individual shows ^{18}O enrichment potentially from breastfeeding
- No archaeological or isotopic evidence for leprosy stigma in this place and time

INTRODUCTION

Leprosy is a chronic bacterial infection caused by the pathogens *Mycobacterium leprae* or *Mycobacterium lepromatosis*. Approximately 250,000 new cases of leprosy are diagnosed worldwide each year, and the condition is associated with low socioeconomic status, a lack of access to medical care, and community stigma (Jacob and Paredes 2008; Bennett et al. 2008; Goulart and Goulart 2008; World Health Organization 2019). Leprosy is most commonly found in areas of the Indian subcontinent, the South Pacific, and Brazil, however small numbers of cases do occur in Europe and North America (World Health Organization 2019). Transmission of the disease is still not fully understood, but it is believed to spread through the inhalation of respiratory droplets (Walker and Lockwood 2006; Lastoria and Abreu 2014). Leprosy has a high infectivity, but low pathogenicity meaning up to 95% of the population is able to successfully fight off the infection through high cell-mediated immunity (CMI) (*ibid*). If the infection persists within a person, the mycobacteria multiply slowly leading to a long incubation period that can range from one to over 20 years; although studies in children reveal the incubation period may be significantly shorter (Moorthy and Desikan 2006; World Health Organization 2019). The severity of leprosy presents along a spectrum. In tuberculoid (paucibacillary) leprosy, individuals show lower numbers of bacilli, few skin lesions, and present a good immune response (Ridley and Jopling 1966; Ridley 1974). Lepromatous (multibacillary) leprosy is the more severe form of the disease, which lies at the opposite end of the spectrum, and is characterized by higher numbers of bacilli, numerous skin lesions, and a poor immune response (*ibid*). For those that develop the disease, the severity of the leprous response can have deleterious effects on the peripheral nervous system, skin, eyes, testes, extremities and can eventually harm other parts of the body including the skeleton (Walker and Lockwood 2006; Goulart and Goulart 2008; Lastoria and Abreu 2014). Its ability to affect the skeleton allows leprosy to be identified bioarchaeologically (Møller-Christensen 1961). Skeletal lesions for leprosy are generally confined to the lepromatous form, and may include destruction and loss of the facial bones (resorption of the anterior nasal spine, remodelling of the nasal margins, abnormally porous/new bone formation on the oral and nasal surfaces of the palatal bones, destruction of the inferior nasal conchae and vomer, abnormal porosity and resorption of the alveolar process), and characteristic destruction and remodelling of the hand and foot bones (acro-osteolysis

and concentric atrophy of the phalanges, metacarpals, and metatarsals) (see Møller-Christensen 1961, Andersen and Manchester 1987, Andersen and Manchester 1988; Andersen and Manchester 1992; Ortner 2008). Tuberculoid leprosy is generally confined to the soft tissues, and can only be confirmed with the aid of pathogenic aDNA analyses in bioarchaeology. However, Matos (2010) suggests that bone changes in the hands and feet can sometimes be identified, as based on his study of medical records and radiographs of patients from a 20th century Portuguese leprosarium.

Leprosy has a complex social past both in the medicohistorical and archaeological records. Leprosaria, or leprosy hospitals, were very common in Medieval Europe (12th – 14th centuries AD) leading scholars to interpret leprosy as an endemic disease during this time (Manchester and Roberts 1989; Richards 2000: 83-97; Rawcliffe 2006: 1-12; Demaitre 2007: 42-80; Moore 2008; Roberts 2002; Roberts 2018; Filipek et al. 2021). Many historical sources repeatedly cite that people with leprosy in the past were stigmatised and treated poorly, however more recent archaeological and historical evidence has challenged this for the Medieval period, particularly prior to the 12th century AD (Roberts 2002; Rawcliffe 2006: 67-78; Demaitre 2007: 99-102; Filipek et al. 2021).

Evaluating the movement of people with leprosy is critical to understanding transmission, dissemination, and potential stigma. In recent years, pathogenic aDNA analyses and palaeopathological research has helped to verify the presence of leprosy in time and space, as well as the burial treatment of sufferers, and by extension, perception of the disease by past communities (Donoghue et al. 2018; Roberts 2013). Reviews of leprosy from prehistory to the 12th century AD in Asia, Africa, and Europe, often reveal no differential burial treatment of those with the disease (Roberts 2002; Filipek et al. 2021). This suggests that the evidence for social ostracism and removal from society should be re-evaluated. This study's primary objective is to view whether any evidence of movement exists that may support or refute the hypothesis that people with leprosy in Early Medieval England were subject to exclusion from their local communities. To achieve this objective, archaeological and palaeopathological evidence is integrated with isotopic evidence ($^{87}Sr/^{86}Sr$ and $\delta^{18}O$) to evaluate whether individuals with leprosy buried

at St. John at the Castle Gate/Timberhill (Norwich, UK) were local to the area, or excluded from their geographic origins, potentially as a consequence of leprosy stigma.

Background

Historical Background of the area

Early Medieval Norwich (Norfolk, UK) developed from a series of communal settlements in the earlier Medieval period (5th – 7th centuries AD) to a more centralised mercantile centre concentrated around the Cockey streams and valleys of the River Wensum in the Middle Saxon Period (c. 7th - 9th centuries AD) (Ayers 2011; Shepherd Popescu 2009; Figure 5.2.1). In 866, the Viking "Heathen Army" invaded East Anglia and according to the *Anglo-Saxon Chronicle*, 'peacefully' settled with local inhabitants (Swanton 1998:69). Norwich was ruled under Danelaw from the Alfred-Guthrum Treaty (c. 890) until 917, when King Edward the Elder reconquered East Anglia (Davis 1955; Ayers 2011). Despite this conquest, Viking raids persisted in the area and in 1004, King Sweyn (Forkbeard) of Denmark and Norway 'came with his fleet to Norwich, and completely ravaged and burnt the town' (Swanton 1998: 134-135). After multiple campaigns, King Sweyn briefly ruled as King of Denmark, Norway, and England from 1013 until his death in 1014 (Higham and Ryan 2013). Upon King Sweyn's death, Norwich reverted back to the House of Wessex (Aethelred the Unready and Edmund Ironside) until King Cnut's (son of King Sweyn) conquest of Norwich, and subsequently the rest of England, in c. 1016 (Nelson 2000; Higham and Ryan 2013). Danish rule of England persisted until 1042 when Edward the Confessor (House of Wessex) was crowned and ruled until 1066 AD (*ibid*). After the Norman Conquest, a motte-and-bailey castle was established under William the Conqueror by c.1067-1068 (Shepherd Popescu 2009; Popescu 2016). Few records exist pertaining to Early Medieval Norwich, however its strategic location as an entry point into England, the presence of its own mint, and reference in the Book of Ely as a reputable place signifies it was likely a place of 'considerable status' during this time (Shepherd Popescu 2009: 49).

Norwich Castle Mall Cemetery Excavations

Excavations at the South Bailey of Norwich Castle for a retail centre (Castle Mall) were undertaken by Norfolk Archaeological unit from 1987-1991. During the course of the

excavations, upwards of six cemeteries were discovered, with two (Farmer's Avenue and St. John at the Castle Gate/Timberhill) radiocarbon dated to the Late Saxon period (c. 850-1060 AD) (Bayliss et al. 2009). Shepherd Popescu (2009:257) suggests the term 'Anglo-Scandinavian' is used to denote this period in Norwich, based on artefactual evidence consistent with a sustained Viking presence in the area. mtDNA evidence from the Farmer's Avenue cemetery has revealed the presence of haplotypes associated with Scandinavian groups, which aligns with the increasing presence of a Viking influence on the growing urban population of Late Saxon Norwich. The presence of an individual with a Romani mitochondrial haplotype, however, suggests further interaction beyond the nearest Scandinavian territories by the 10th century AD (Töpf and Hoelzel 2005). Of particular note is the evidence for leprosy at the parish site of St. John at the Castle Gate/Timberhill.

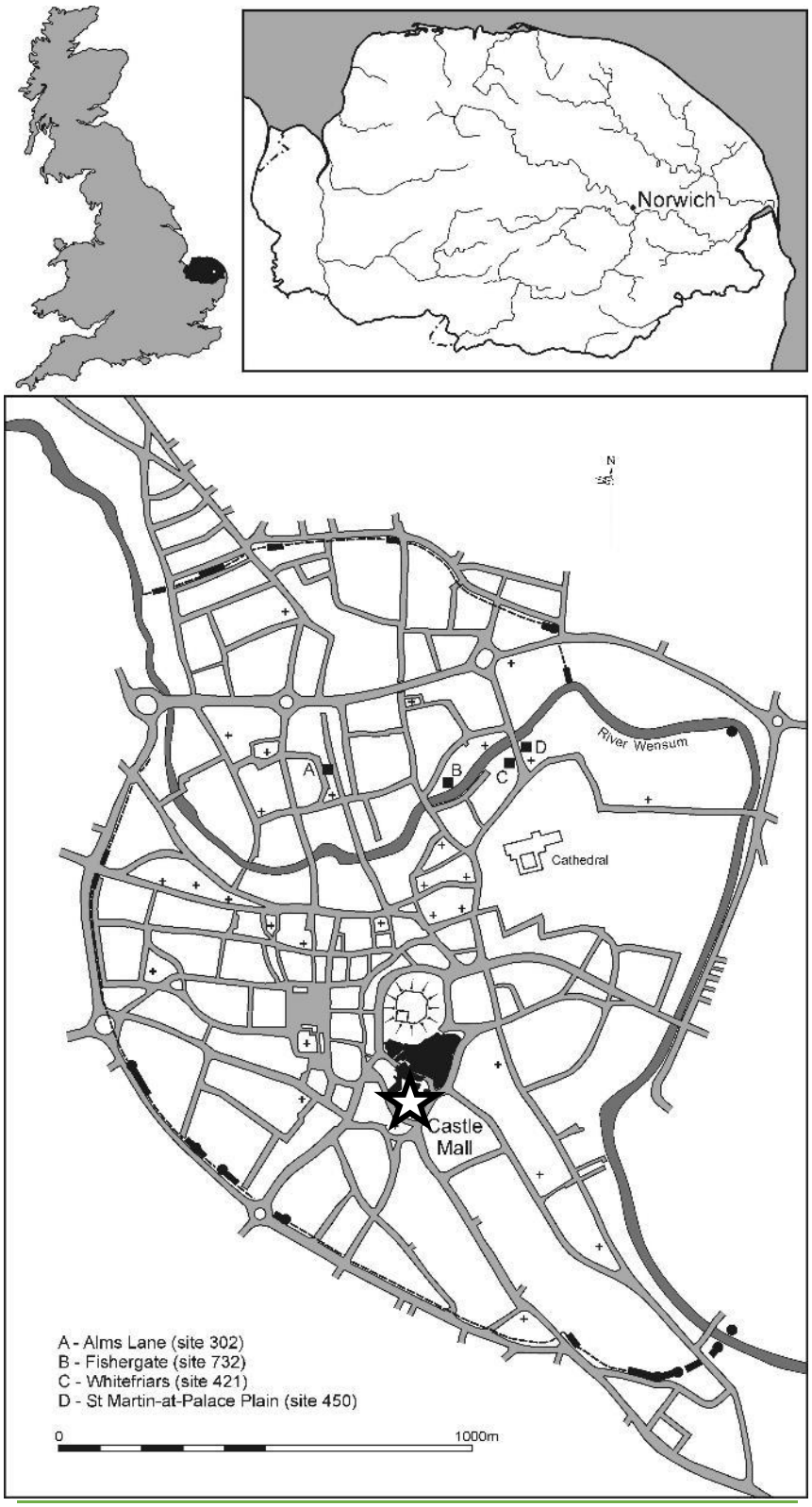

Figure 5.2.1 Map of Norwich with location of Norwich Castle Mall excavations of Farmer's Avenue and St. John at the Castle Gate (starred). (Modified from Norfolk Archaeological Unit (2009) Norwich, Castle Mall [data-set]. York: Archaeology Data Service [distributor] https://doi.org/10.5284/1000173; Open Access)

The church of St. John at the Castle Gate (now St John the Baptist, Timberhill) is estimated to have an 11th century AD foundation date based on stylistic similarities to other structures with known dates (Shepherd Popescu 2009; Popescu 2016). The earliest documentary mention of this site was in 1157 in the *Liber Cartarum et Placitorum*, which noted a church called (Ecclesia) Sancti Johannis ante portam Castelli or 'St John at the Castle Gate', however, an extant structure was most likely in place before (*ibid*). Excavations of a small part of the original cemetery revealed the articulated remains of at least 149 adults (59 males, 76 females, and 14 undetermined), and 35 non-adults buried in a 'fan shape' along the northernmost part of the churchyard, with graves aligned south-west to north-east in the western part of the cemetery, east-to-west at the centre, and north-east to south-west in the east (Anderson 1996; Norfolk Archaeological Unit 2009; Shepherd Popescu 2009; Figure 5.2.2).

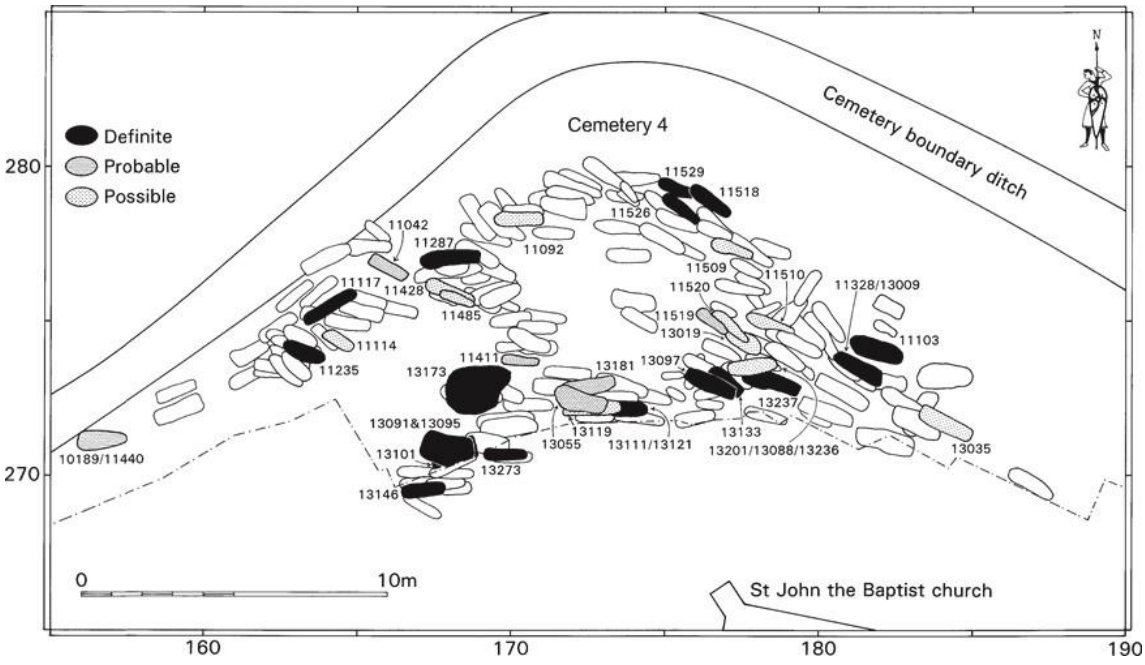

Figure 5.2.2 Distribution of individuals with skeletal evidence of leprosy at St. John at the Castle Gate. (Modified from Norfolk Archaeological Unit (2009) Norwich, Castle Mall [data-set]. York: Archaeology Data Service [distributor] https://doi.org/10.5284/1000173; Open Access)

Radiocarbon dating of 17 individuals from the site reveal that the cemetery was in use for a very short time period, commencing cal AD 980-1030 (95% probability) and ceasing in

cal AD 990-1050 (95% probability), likely representing one or two generations (Bayliss et al. 2009: 243). The site is largely contemporaneous with the Anglo-Scandinavian Farmer's Avenue cemetery (cal AD 890-1060, 95% probability), with St. John at the Castle Gate potentially serving as an extension to the Farmer's Avenue cemetery (Shepherd Popescu, 2009: 236-268). Analysis of carbon and nitrogen stable isotopes from bone collagen of individuals from both cemeteries are consistent with each other (Table 5.2.1), indicating a diet based on plants with a C_3 photosynthetic pathway, and terrestrial protein with the possible incorporation of some marine component (Bayliss et al 2004; Bayliss et al. 2009). This is substantiated by the discovery of a large number of pig, cattle, and sheep remains from the Late Saxon period of the site (Albarella et al. 2009). Bayliss et al. (2004) initially suggested the radiocarbon dates for the individuals at St. John at the Castle Gate may be slightly offset due to a potential marine reservoir effect, however subsequent re-evaluation by Bayliss and colleagues (2009) states that this model is not preferred due an unreliability in the knowledge about dietary effects on the correction for radiocarbon measurements (246). The earlier date ranges are also consistent with related archaeological findings from the site (Bayliss et al. 2009: 245).

TABLE 5.2.1 – Radiocarbon dates and mean carbon and nitrogen isotope values from bone collagen from Farmer's Avenue and St. John's at the Castle Gate, Norwich (UK). Data from Bayliss et al. (2009: 239).

CEMETERY	^{14}C (95% CONFIDENCE)	δ^{13}C‰	δ^{15}N‰
FARMER'S AVENUE	890 – 1060 cal AD	-19.3	10.7
ST. JOHN'S	990 – 1050 cal AD	-19.3	11.1

EVIDENCE FOR LEPROSY AT ST. JOHN AT THE CASTLE GATE

Diagnostic evidence for leprosy in the skeleton is dependent on several factors, including the chronicity and severity of the disease (e.g. tuberculoid vs. lepromatous), and the presence of characteristic, or pathognomonic, skeletal lesions on elements including the facial bones, and bones of the hands and feet (Møller-Christensen 1961; Andersen et al. 1992; 1994; Andersen and Manchester 1992). Out of the 184 individuals excavated, 92 had foot bones, 101 had hand bones, and 103 had facial bones available for assessment (Anderson 1996; Anderson 1998; Anderson 2009). Only 48 individuals had all three areas preserved (*ibid*). Anderson (2009) reported lesions consistent with a diagnosis of leprosy in 35 individuals (34% of the observable population), with 24 individuals (23.3 % of the

observable population) reported to have skeletal lesions pathognomonic of the condition (228-231). The prevalence rate of those with lesions diagnostic of leprosy is comparable with St. James and Mary Magdalen leprosy hospital site (12^{th} – 15^{th} centuries AD) in Chichester, at approximately 24% (Magilton et al. 2008).

Using Strontium and Oxygen isotopes to study movement of people with disease

The movement of people throughout time and space is linked to major drivers such as economy, settlement, disease, conflict, forced migration, marriage, kinship, ethnicity, identity and agency, natural disasters, etc. (Castelli 2018). Analyses of radiogenic strontium ($^{87}Sr/^{86}Sr$) and stable oxygen ($\delta^{18}O$) isotopes in bioarchaeology help to contextualise the impetus for these types of mobility drivers at both the individual and population level in the past (see Evans et al. 2006; Evans et al. 2010; Chenery et al. 2010; Montgomery 2010; Evans et al. 2012). Strontium and oxygen isotope ratios are subsumed into forming tooth enamel from the ingestion of food and water, and remain relatively unmodified throughout a person's lifetime (Montgomery and Evans 2006; Evans et al. 2010; Montgomery 2010; Evans et al. 2012). Strontium isotope ratios ($^{87}Sr/^{86}Sr$) are related to the underlying geology of an area and therefore provide a geographical cue to where a person sourced their food and water during tooth crown development (Bentley 2006). Oxygen isotope ratios ($^{18}O/^{16}O$) of tooth enamel are related to the ingestion of drinking water, and therefore indirectly reflect an area's isotopic composition of precipitation, which varies according to climate, proximity to the coast, altitude, and region (Darling et al. 2003; Chenery et al. 2010; Evans et al. 2012). Unlike strontium, oxygen undergoes a fractionation process once subsumed, therefore regression formulae must be applied to values in order to make comparisons with modern data (Daux et al. 2008; Chenery et al. 2010; Chenery et al. 2012). Based on these principles, the use of both isotope systems can reveal whether a person's strontium ($^{87}Sr/^{86}Sr$) and oxygen isotope ratios ($\delta^{18}O$) are consistent with their burial location, and by extension used to interpret whether individuals were raised locally or not.

Despite being a key factor in disease transmission and its possible consequences (e.g. disease stigma), few studies have examined the mobility histories of individuals with skeletal lesions diagnostic of specific infectious diseases (Roberts et al. 2013; Filipek-

Ogden et al. 2016; Quinn 2017; Roffey et al. 2017). This is in part due to the difficulty in ascertaining whether a person was infected with the disease before, during, or after movement to an area (*ibid*). By examining isotope ratios from younger individuals, the likelihood that their geographical origins during childhood overlap with the location where they were infected with leprosy increases due to the longer incubation periods associated with the disease. A critical but key limitation to understanding these isotope systems is that they function on a discriminatory basis, meaning they can only show whether a person's strontium and oxygen isotope ratios are consistent with their burial locations and cannot provide a definitive place of origin (Montgomery 2010; Evans et al. 2012). Similarly, large swathes of geological and climatic regions can produce similar isotope data, some culturally-mediated behaviours, certain diseases, and issues of equifinality can alter the isotopic composition of human tissues, therefore caution and consideration of these issues should be exercised in interpretations (Montgomery 2010; Brettell et al. 2012; Bogaard and Outram 2013; Reitsema 2013).

Geological Context and Meteorological Context

The local geology of Norwich (c. 30km) lies at the intersection of two lithologies: Cretaceous chalk to the immediate south and to the west, abutted by undifferentiated Neogene-Pleistocene gravels, sands, and clay sediments (British Geological Survey 2007). Norwich lies at the lowest fording point above the confluence of the River Wensum with the River Yare, about 33 km east of the North Sea. Deposits of alluvium extend to the east and were widely used for grazing at the time of the compilation of the Domesday Book (11th century AD), and heavier, more fertile soils to the west and northwest suggest the area was extensively wooded in the past (Shepherd Popescu 2009:37). The city is enveloped by Mousehold Heath to the north-east and the archaeological site itself lies at the end of the Ber Street ridge to the south, which is underlain by the gravels, sands, and clays of the Norwich Crag and the Beeston chalk (*ibid*). Expected biosphere ratios ($^{87}Sr/^{86}Sr$) for the Norwich area lie between 0.708 – 0.710 (Evans et al. 2010). A previous study on Medieval and post-Medieval faunal remains recovered from the Norwich Castle Mall excavations by Madgwick et al. (2012) provide a dentine ratio of 0.7098, which is consistent with predicted values.

The $\delta^{18}O_{(P)VSMOW}$ for archaeological humans excavated in Britain shows a mean value of 17.7‰ ± 1.4 (2σ), with west-coast, higher rainfall areas producing a mean delta value of 18.2‰ ± 1 (2σ, n=40), and east-coast, lower rainfall areas, producing a mean delta value of 17.2‰ ± 1.3 (2σ, n=83) (Evans et al. 2012). Individuals raised local to the Norwich area should fall within the east-coast, lower rainfall range (<700mm/year) (Darling and Talbot 2003). Evans and colleagues' (2012) provide a conversion of $\delta^{18}O$ to drinking water values ($\delta^{18}O_{DW}$) based on Daux et al. (2008; Eqn. 6), and estimate a mean a drinking water value of -7.5‰ ±1.8 (2σ, n=83) for lower rainfall areas, which is consistent with data produced by Darling et al. (2003).

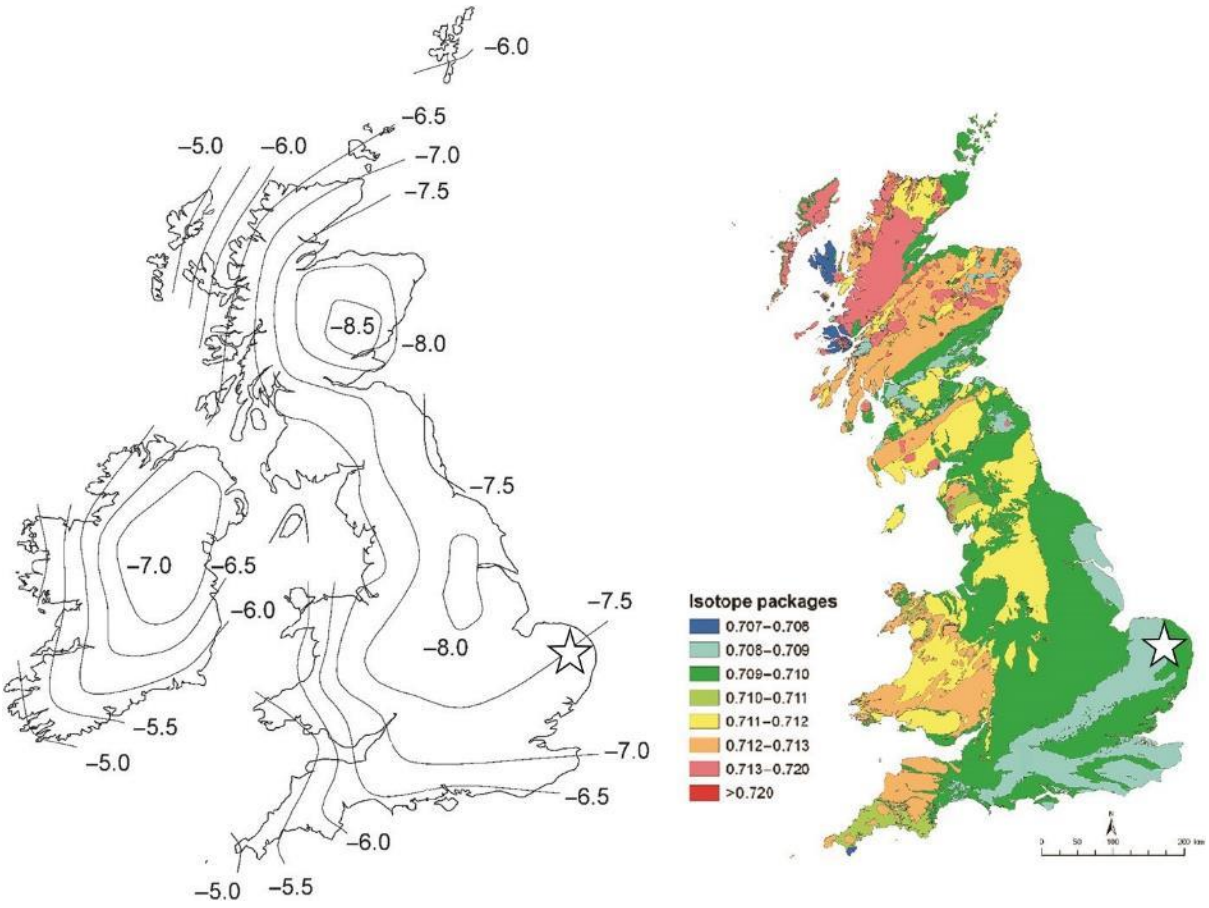

Figure 5.2.3 Oxygen contour and strontium biosphere maps with Norwich starred. Modified from Darling et al 2003:189, and Evans et al 2010:2.

Materials and Methods

Osteological Methods

Using the previously published skeletal report (Anderson 1996), skeletons were selected and re-evaluated for macroscopic evidence of leprosy at the Norwich Castle Museum. In order to analyse the individuals from this site and take tooth samples for subsequent isotopic analyses, permissions with ethical justifications were sought and approved from both from the curators of the human skeletal remains and the in-house ethics committee at the Department of Archaeology at Durham University. The Codes of Ethics (2010) and the Codes of Practice (2010) from the British Association of Biological Anthropologists and Osteoarchaeologists (BABAO)(https://www.babao.org.uk/publications/ethics-and-standards) were strictly adhered to in the sampling from the individuals selected for study. Individuals with elements showing lesions consistent with diagnostic indicators of lepromatous leprosy (see Ortner 2008) were selected for further analyses. These included rhinomaxillary changes (resorption of the anterior nasal spine, remodelling of the nasal margins, abnormally porous/new bone formation on the oral and nasal surfaces of the palatal bones, destruction of the inferior nasal conchae and vomer, abnormal porosity and resorption of the alveolar process; Figure 5.2.4), and acro-osteolysis and concentric atrophy of the hands and feet (destruction and remodelling of the phalanges, metacarpals, and metatarsals), and mediolateral remodelling of the metatarsal shafts ('knife edge' remodelling). Other lesions consistent with, but not wholly diagnostic of leprosy, were also evaluated, including resorptive grooves on the palmar surfaces of the hand phalanges (or 'volar grooving') caused by flexion contractures (Andersen and Manchester 1987), tarsal fusion and dorsal exostoses (Andersen and Manchester 1988), and subperiosteal new bone formation on the distal shafts of the tibiae and fibulae (Ortner 2008), however individuals needed to have at least one pathognomonic indicator of leprosy to be included in this study.

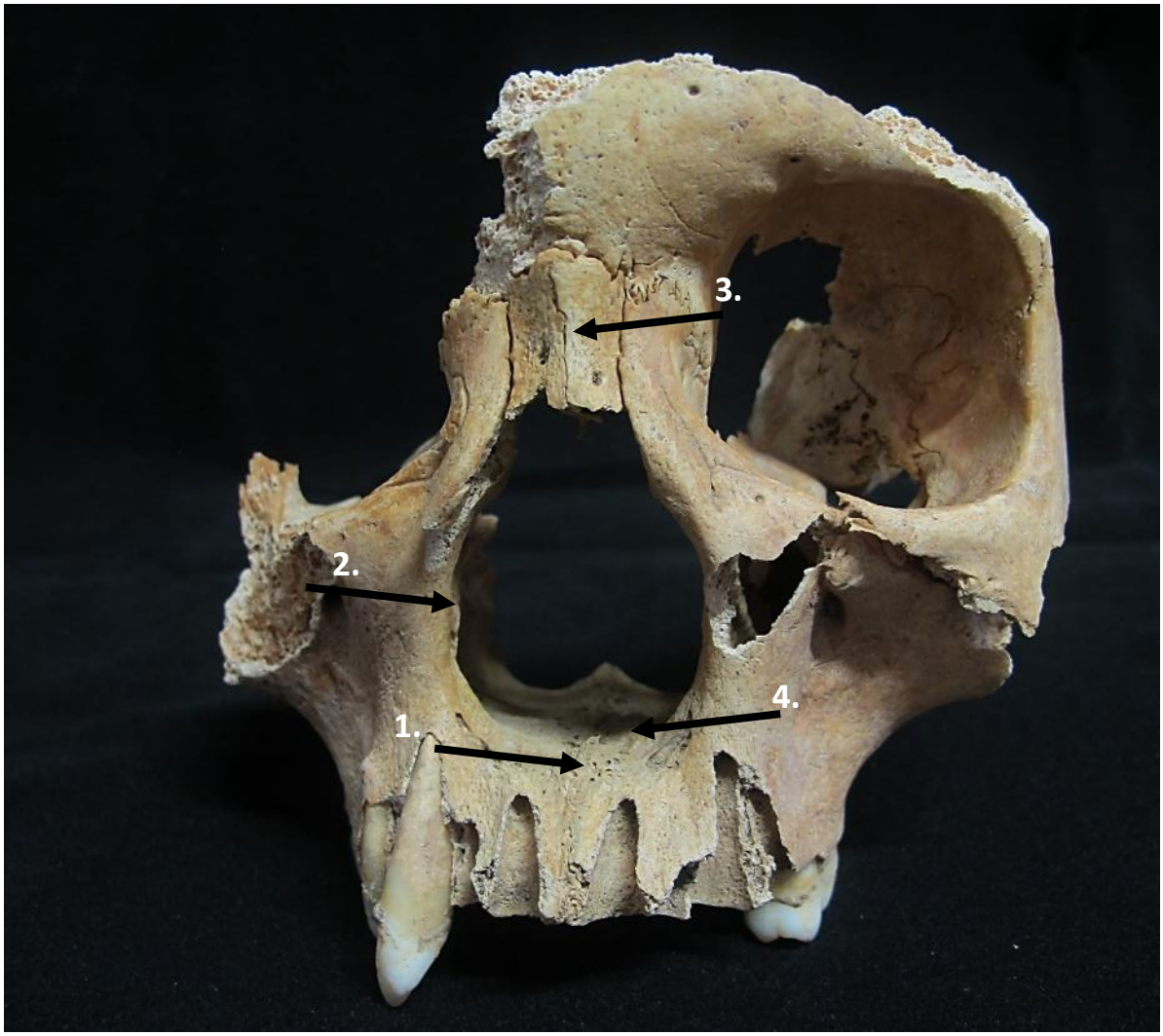

Figure 5.2.4 Facial bones of Sk. 13009 displaying evidence of rhinomaxillary syndrome including 1.) Resorption of the anterior nasal spine, 2.) Widening and remodelling of the nasal aperture, 3.) Widening and flattening of the nasal bones, and 4.) Pitting on the nasal floor.

TABLE 5.2.2 – Individuals selected for strontium and oxygen isotope analysis. Radiocarbon dates and carbon and nitrogen isotope values from Bayliss et al. (2009: 239). Tooth development timing based on Moorees et al. (1963) and AlQahtani et al. (2010).

INDIVIDUAL	SEX	AGE AT DEATH	TOOTH SAMPLED	AGE RANGE OF CROWN FORMATION	BONES AFFECTED BY LEPROSY	^{14}C DATES (2σ)	$\delta^{13}C$ (‰)	$\delta^{15}N$ (‰)
SK. 11117	Male	16.5-18.5	Mandibular RM3	7.5 – 17.5 years	Facial bones, Hands, Feet, Distal lower limbs	cal AD 990-1050	-18.2	11.4
SK. 11287	Male	22.5-23.5	Maxillary LM3	7.5 – 16.5 years	Facial bones, Hands, Distal lower limbs, Feet unavailable for observation			
SK. 13009	Ambiguous	14.5-16.5	Maxillary RM2	2.5 – 8.5 years	Facial bones, Hands, Possible feet, Distal lower limbs	cal AD 980-1030	-17	11.7
SK. 11518	Ambiguous	10.5 - 11.5	Mandibular RM2	2.5 – 9.5 years	Facial bones, Hands, Feet, Distal lower limbs	cal AD 990-1040	-19.2	11.5
SK. 13121	Female	18-23.5	Maxillary LM1	1.5 months – 3.5 years	Facial bones, All other skeletal elements unavailable for observation at time of analysis			
SK. 11526	Ambiguous	12.5-13.5	Mandibular RM3	7.5 – 17.5 years	Facial bones, Distal lower limbs			
SK. 13035	Female	18.5-20.5	Maxillary LM2	2.5 - 8.5 years	Facial bones, Hands, Distal lower limbs, Feet unavailable for observation			
SK. 13044	Male	15.5-16.5	Mandibular RM2	2.5 - 9.5 years	Facial bones, Hands			
SK. 13101	Ambiguous	14.5-16	Maxillary LM3	7.5 – 16.5 years	Facial bones, Feet, Distal lower limbs, Hands unavailable for observation			
SK. 13146	Male	18.5-20.5	Mandibular LM3	7.5 – 17.5 years	Facial bones, Feet, Distal lower limbs, Hands unavailable for observation at time of analysis			

Individuals were reassessed for age at death based on tooth development according to methods set out in AlQahtani et al. (2010), in order to target adolescents, defined socially and biologically as ranging from ages 10 – 25 years (Lewis 2016; Patton et al. 2016; Sawyer et al. 2018). Adolescent individuals were specifically selected because of their unique liminal status (Lewis 2016) and because their potential for movement is more limited due to their younger age, i.e. the older a person is the more opportunity they may

have to move (Montgomery 2010). Young people also offer a crucial insight into the social attitudes towards disease in the past and provide a 'pivotal conduit' for understanding the social and physical impacts of health within a population (Redfern and Gowland 2011: 111). A total of 10 tooth samples from adolescent individuals were selected for radiogenic strontium and stable oxygen isotope analysis (Table 5.2.3). Either molars or premolars were selected to represent the most recently formed teeth available for each individual, and only teeth with no signs of pathology were selected. Teeth were removed from their alveolar sockets by hand and photographed by the Department of Archaeology at Durham University in the occlusal, buccal, lingual and apical aspects before sample preparation for isotopic analyses.

ISOTOPE METHODS

Sections of core enamel were extracted in the Sample Preparation Laboratory in the Department of Archaeology, Durham University. Tooth sample preparation followed the guidelines of Montgomery (2002). Tooth surfaces were abraded using tungsten carbide burrs to remove exogenous material and 15-25mg of enamel was sectioned using diamond edged dental saws. All surface enamel and any adhering dentine were mechanically removed from enamel sections. Processed chips of core enamel were sealed in Eppendorf microtubes and transferred to the (class 100, HEPA-filtered) laboratory facilities at the NERC Isotope Geosciences Laboratory (NIGL) at British Geological Survey (Keyworth, Nottinghamshire, UK) for further preparation.

For strontium isotope analyses, the sections of core enamel were then further prepared and measured according to Evans and colleagues (2006). In a clean laboratory, the enamel sample was first cleaned ultrasonically in high purity water to remove dust, rinsed twice, dried down in high purity acetone and then weighed into pre-cleaned Teflon beakers. A known amount of ^{84}Sr tracer solution was added to each sample, which was dissolved in Teflon distilled 8 M HNO_3. The sample was converted to chloride using Quartz distilled 6M HCl and then taken up in 2.5M HCl. The strontium was extracted using Eichrom Dowex AG50X8 resin. The samples were loaded onto Rhenium filaments (Birck 1986) and the isotope composition and concentrations were determined by Thermal Ionization Mass spectroscopy (TIMS) using a Thermo Triton multi-collector mass

spectrometer. The international standard for $^{87}Sr/^{86}Sr$, NBS-987, gave a value of 0.710251 ± .000005 (n = 19, 2sd) during the analysis of these samples. Procedural blank values were less than 100pg.

For oxygen isotope analyses, approximately 3 mg of powdered enamel was loaded into a glass vial and sealed with septa. The vials were transferred to a hot block at 90°C on a GV Multiprep system. The vials were evacuated and 4 drops of anhydrous phosphoric acid added. The resultant CO_2 was collected cryogenically for 14 minutes and transferred to a GV IsoPrime dual inlet mass spectrometer. The resultant isotope values are reported as delta (δ) values, in parts per thousand (per mil; ‰) normalized to the VPDB scale using a within-run calcite laboratory standard (Keyworth Carerra Marble, KCM) calibrated against SRM19, NIST reference material. These ratios are converted to the VSMOW scale using the published conversion equation of Coplen (1988): VSMOW = (1.03091 x $\delta^{18}O_{VPDB}$) +30.91. Analytical reproducibility for this run of laboratory standard calcite (KCM) is 0.09‰ (1σ, n = 6) for $\delta^{18}O_{VSMOW}$ and ± 0.05‰ (1σ, n = 6) for $\delta^{13}C_{VPDB}$. The reproducibility of the enamel, based on average of five duplicate pairs is ± 0.07‰, 1σ. The carbonate oxygen results ($\delta^{18}O_{(C)VSMOW}$) were converted to phosphate values $\delta^{18}O_{(P)VSMOW}$ using the regression equation $\delta^{18}O_P = 1.0322 \times \delta^{18}O_C - 9.6849$ (Chenery et al. 2010; Chenery et al. 2012), with an associated error of ±0.29 ‰, 1σ. The conversion to phosphate values was carried out to make the data more comparable to oxygen isotope data reported in other studies and baselines (e.g. Evans et al. 2012).

The carbonate oxygen results ($\delta^{18}O_{(C)VSMOW}$) were converted to drinking water values ($\delta^{18}O_{DW}$) using Daux et al.'s (2008) equation 6 in accordance with Chenery and colleague's (2012) calculation: $\delta^{18}O_{DW} = 1.590 \times \delta^{18}O_C - 48.634$. The calculation of drinking water values can involve larger uncertainties (± 1 ‰, 2σ)(Chenery 2012; Pollard et al. 2011) and therefore drinking water values are used as general guidance only.

RESULTS

Strontium and oxygen isotope data and strontium concentrations for the 10 samples are presented in Table 5.2.3 and Figure 5.2.5. Strontium isotope ratios range from 0.7090 to 0.7101 (mean 0.7095, ± 0.0008 2σ). Strontium concentrations range between 71 ppm

and 193 ppm (mean 105, ± 38 ppm, 2σ), which are consistent within previously reported archaeological and modern teeth from Britain (98 ± 130 ppm) (Eckardt et al. 2009; Chenery et al. 2010; Evans et al. 2012; Hemer et al. 2013; Hemer et al. 2014), suggesting good preservation. One individual (Sk. 13044) failed to produce a strontium isotope ratio. The remaining nine individuals provide strontium isotope ratios compatible with the predicted ratios for the Norwich area (~0.708 – 0.710).

The $\delta^{18}O_{(P)VSMOW}$ values for the individuals range from 17‰ to 19.3‰ (mean 17.8 ± 0.6‰ 1σ), with corresponding $\delta^{18}O_{DW}$ values ranging from -7.6‰ to -4.1‰ (mean -6.3 ± 0.9‰ 1σ). Nine of the 10 individuals show $\delta^{18}O_{(P)VSMOW}$ (mean 17.7 ± 0.4‰ 1σ) and $\delta^{18}O_{DW}$ (-6.5 ± 0.6‰ 1σ) values that fall within the predicted values for the study area. One individual (Sk. 13121) is a clear outlier from the group with a high $\delta^{18}O_{(P)VSMOW}$ (19.3‰; $\delta^{18}O_{DW}$ = -4.1‰).

TABLE 5.2.3 – Strontium and oxygen isotope data, including $\delta^{18}O_P$ calculated from Chenery et al. (2012), and $\delta^{18}O_{DW}$ calculated from Daux et al. (2008) eqn. 6, in accordance with Chenery et al. (2012).

SKELETON	SR PPM	$^{87}Sr/^{86}Sr$	$\delta^{13}C_{VPDB}$ (‰)	$\delta^{18}O_{(C)VSMOW}$ (‰)	$\delta^{18}O_{(P)VSMOW}$ (‰)	$\delta^{18}O_{DW}$ (‰)(Eqn 6)
13101	120	0.7098	-12.35	26.73	17.9	-6.1
13009	193	0.7095	-12.45	25.82	17.0	-7.6
11117	81	0.7090	-12.68	26.38	17.6	-6.7
11518	87	0.7100	-12.98	26.76	17.9	-6.1
13121	71	0.7096	-12.83	28.03	19.3	-4.1
11526	128	0.7097	-13.26	26.51	17.7	-6.5
13035	84	0.7090	-13.01	26.83	18.0	-6.0
13044	FAILED	FAILED	-13.22	26.98	18.2	-5.7
13146	100	0.7101	-13.44	26.31	17.5	-6.8
11287	84	0.7090	-12.61	26.25	17.4	-6.9

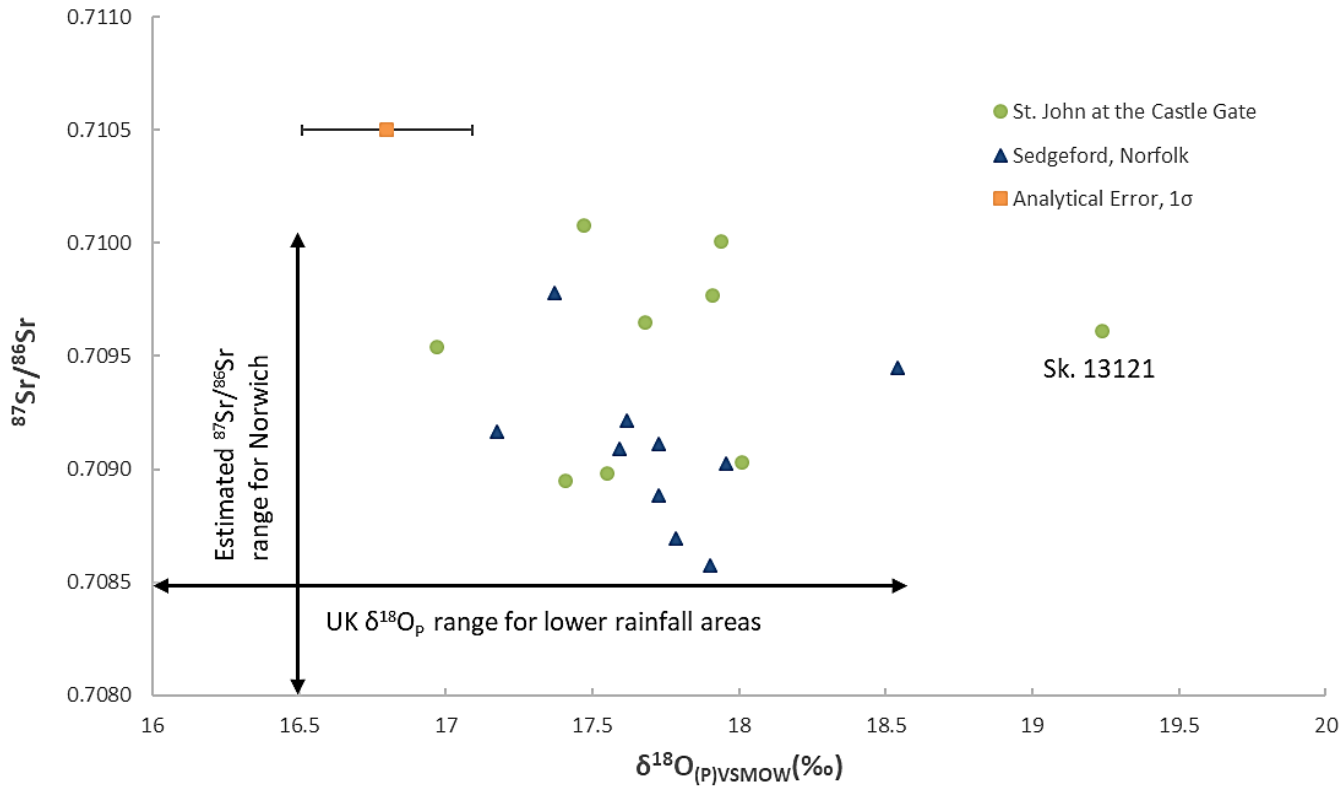

Figure 5.2.5 Strontium and oxygen isotope data from St. John at the Castle Gate (Norwich, Norfolk, UK). Also plotted are the estimated predicted ranges for Norwich from Evans et al. (2010) and Evans et al. (2012), and comparative data from the Early Medieval period (7th-9th centuries AD) local burials from nearby Sedgeford, Norfolk, UK (Haraldsson 2016). Sk. 13121 presents as an outlier likely due to an enrichment in 18O from breastfeeding during tooth formation.

DISCUSSION

One individual (13121) has an oxygen isotope ratio outside of the expected range for the burial location, likely due to an additional metabolic alteration in their $\delta^{18}O$ values from breastfeeding. The latest forming tooth available for sampling from 13121 was the left maxillary first molar, the crown of which develops between the ages of 1.5 months and 3.5 years (AlQahtani et al. 2010). The tissues of breastfed infants are often enriched in ^{18}O relative to the $\delta^{18}O$ body water values of the mother instead of from meteoric drinking water directly. This relative enrichment can result in an increase in an individual's $\delta^{18}O_{DW}$ by as much as 2-3‰ within tissues developing during the breastfeeding period (Wright and Schwarcz 1998; Fuller et al. 2006; Tsutaya and Yoneda 2015). Given that their strontium isotope ratio aligned well with others within the

cemetery, the most likely explanation is that this individual was raised locally and that their higher oxygen isotope ratio is a by-product of this biologically mediated behaviour.

This study's main objective was to view whether any evidence of movement existed to support or refute the hypothesis that people with leprosy in Early Medieval England were subject to exclusion from their local communities. Results indicate that all of the young people with lesions diagnostic of lepromatous leprosy were buried in a normative manner within a parish cemetery, and have isotope profiles consistent with the Norwich area (within 30km) and other broadly contemporaneous burials from the region (Haraldsson 2016). Our sampling strategy does acknowledge that younger people would have had less time to move from their communities, but if they were expelled as many have suggested (Browne 1975; Brody 1975: 147-157; Dols 1979; Kealey 1981:104-105; Conrad et al. 1998: 187-189; Covey 1998: 95-103; Porter 1999:121-122; Richards 2000: 48; Moore 2008: 43-60), they may be expected to be found beyond the confines of their local area.

It is worth noting that inclusion within the local geographical confines of the area does not necessarily reflect social inclusion. However, if they were socially stigmatised but not expelled from the area, we would expect their burials to show some degree of deviancy or otherness signifying this. Moore (2008:59) argues that individuals with leprosy in Medieval England were subject to such stigma that their bodies, residences, and belongings were incinerated upon their deaths. This is not supported by the archaeological and skeletal evidence, which shows that individuals with leprosy were buried with care, including earthen pillows, flint head supports, wooden coffins, burial shrouds, and beds of pebbles - all of which show parity in burial practice with nearby, largely contemporaneous cemeteries (Shepherd Popescu 2009:266-269). Furthermore, isotope data from the affected individuals is consistent with the local area indicating that notions of stigma and social exclusion have been overstated.

It is important to note, that leprosy would not have been unknown in this time and place. Several Early Medieval cemetery sites around Norfolk, as well as the remainder of Britain, have revealed skeletal remains of people with leprosy including high-status burials (e.g. Edix Hill; see Roberts 2020 – Appendix 3; Table 5.2.4). Documentary evidence such as the

Leechbooks give advice on the care and treatment of individuals with leprosy (Cockayne 1865; 1866; Mount 2016; Doyle 2017), and Fursey, an Irish missionary specifically spoke about the presence leprosy in East Anglia as early as the 7th century AD (Krusch 1902, cited in Shepherd Popescu 2009: 270). In 964, major Benedictine reforms took place under the reign of Edgar (Higham and Ryan 2013) to provide care and compassion for people with ailments, including leprosy (Orme and Webster 1995:17-20; Clarke 1931: 17-18; Rawcliffe 2006:322-337; Roffey 2012). Textual evidence from Later Medieval contexts indicates that in some instances people with leprosy were valorised for their suffering, rather than stigmatized (Rawcliffe 2006:44-102).

Although this study cannot tell us how individuals with lepromatous leprosy were treated in life as a direct consequence of their disease at this time, their inclusion within their local parish cemetery of St. John at the Castle Gate, and not outside the town, implies that their disease status did not exclude them from their local communities or normal cultural burial customs at the times of their deaths. It may be argued that the presence of individuals with leprosy at St. John at the Castle Gate and not at Farmer's Avenue suggests some degree of separation, however the latter cemetery does have an earlier start date (c. 890 AD) and archaeologists surmise St. John's may have been used as an overflow cemetery at the end of the 10th century AD due to population pressures and Castle building works (Shepherd Popescu 2009:268-269). Perhaps the increased prevalence of the disease at St. John's represents a more general increase in the disease within the population, or the presence of an early monastic hospital as a by-product of mid-10th century Benedictine reforms. Although the introduction of leprosy hospitals are generally attributed to the Normans (Orme and Webster 1995: 22-31; Roffey 2012), evidence for a pre-Norman leprosy hospital has recently been discovered in Winchester (Roffey and Tucker 2012; Roffey 2012) indicating that the disease was becoming a public health issue before the Late Medieval period (11th – 16th centuries AD).

TABLE 5.2.4 – British sites reporting skeletons with possible and confirmed leprosy prior to the 12th century AD.

SITE/LOCATION	TIME PERIOD	CITATION
EAST LOTHIAN, SCOTLAND	2280 – 1970 cal B.C.	Roberts (2007)
POUNDBURY, DORSET	4th century AD	Reader (1974)

CANNINGTON, SOMERSET	3rd – 8th centuries AD	Brothwell et al. (2000)
GREAT CHESTERFORD, ESSEX	415 – 545 cal AD	Inskip et al. (2015)
BROUGHTON LODGE, WILLOUGHBY-ON-THE-WOLDS, NOTTINGHAMSHIRE	5th – 7th centuries AD	Roberts (1993)
COLLINGOURNE DUCIS, WILTSHIRE	5th – 7th centuries AD	Dinwiddy (2016)
BECKFORD, GLOUCESTERSHIRE	6th century AD	Wells (1962)
BARRINGTON EDIX HILL, CAMBRIDGESHIRE	575 – 650 cal AD	Duhig (1998)
DUNSTABLE, BEDFORDSHIRE	650 – 670 cal AD	Beavan and Mays (2013)
BURWELL, CAMBRIDGESHIRE	7th century AD	Møller-Christensen and Hughes (1962)
ECCLES, KENT	7th century AD	Manchester (1981)
TEAN, SCILLY ISLES	7th – 8th centuries AD	Brothwell (1961)
BRANDON, NORFOLK	7th – 8th centuries AD	Anderson (2014)
ST. ANDREWS, SCOTLAND	8th century AD	Lunt (2013)
HOXNE, SUFFOLK	885 – 1015 cal AD	Inskip et al. (2017)
ST. JOHN'S TIMBERHILL, NORWICH, NORFOLK	10th – 11th centuries AD	Anderson (1996; 1998); Watson and Lockwood (2009); Bayliss et al. (2009); *This study*
ST. CATHERINE'S, NORWICH, NORFOLK	10th – 11th centuries AD	Wells (1962)
SCHOOL STREET, IPSWICH, SUFFOLK	10th – 11th centuries AD	Mays (1989)
WHARRAM PERCY, YORKSHIRE	960–1100 cal AD	Taylor et al. (2006); Mays (2007)
WINCHESTER, HAMPSHIRE	10th – 12th centuries AD	Roffey and Tucker (2012); Schuenneman et al. (2013); Roffey et al. (2017)
RAUNDS FURNELL, NORTHAMPTONSHIRE	10th – 12th centuries AD	Kerudin et al. (2019)

CONCLUSION

This study aimed to ascertain the mobility histories of people with leprosy buried at St. John at the Caste Gate in Norwich, with the objective of evaluating evidence for stigma in Early Medieval Norwich. The analysis of strontium and oxygen isotopes combined with the burial evidence suggests that young individuals with leprosy in Norwich were not ostracised from their communities of origin in the Anglo-Scandinavian period. Future studies ascertaining and comparing leprosy strain type and individual haplotypes will further aid in understanding the leprosy burden and particulars of migration and transmission in Early Medieval Britain. This study also reports the first strontium and

oxygen isotope data from people with leprosy in East Anglia. Although the study sample was small, this contributes to a growing body of research interrogating the social impacts of disease and challenges to previously held narratives about the treatments of people with leprosy in the past.

ACKNOWLEDGMENTS

Special thanks is given to Tim Pestell from the Norwich Castle Museum for allowing access to the skeletal collection, and Hilary Sloane and Carlyn Stewart from the NERC Isotope Geosciences Laboratory for their additional support. Additional thanks are given to Harald Haraldsson and the Sedgeford Historical and Archaeological Research Project (SHARP) for permitting the use of their data for comparison. KLF would also like to thank Drs. Joanna Moore and Lucie Johnson from Durham University for their helpful comments.

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MANUSCRIPT 5.3: DISEASE AT THE DOORSTEP: SEX AND MOBILITY HISTORIES OF ADOLESCENTS BURIED IN THE CEMETERY OF ST. MARY MAGDALEN LEPROSARIUM (WINCHESTER, ENGLAND)

Prepared for *American Journal of Physical Anthropology*

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ABSTRACT

OBJECTIVES

In exploring biological sex and geographic origins of people buried at an Early Medieval cemetery, this paper examines amelogenin proteins and $^{87}Sr/^{86}Sr$, and $\delta^{18}O$ from tooth enamel of individuals buried at the St. Mary Magdalen leprosarium (Winchester, England). The resultant data are combined with archaeological and palaeopathological evidence to provide a broader snapshot of migration and its relationship to leprosy during the Early-Late Medieval transition (9^{th} – 12^{th} centuries AD).

MATERIALS AND METHODS

Amelogenin peptides were extracted from five individuals whose biological sex could not be determined using osteological sex estimation methods due to their pre-pubertal status. Tooth enamel samples from 19 adolescents were analysed for $^{87}Sr/^{86}Sr$ ratios and $\delta^{18}O$ values to explore mobility histories.

RESULTS

Amelogenin peptides revealed three males and two females. Tooth enamel samples provided an $^{87}Sr/^{86}Sr$ ratio range from 0.7084 to 0.7103 (mean 0.7090, ± 0.0012, 2σ). $\delta^{18}O_P$ values show a wide range of 15.6‰ to 19.3‰ (mean 17.8 ± 1.6‰ 2σ), with corresponding $\delta^{18}O_{DW}$ values ranging from -9.7‰ to -4.1‰ (mean -6.3 ± 2.4‰ 2σ).

DISCUSSION

Amelogenin peptides indicate the presence of adolescent females buried at St. Mary Magdalen leprosarium. Prior to this study, only males were identified within the adolescent

population. These are the youngest confirmed females with bone changes of leprosy in the archaeological record. Results also indicated that at least 12 adolescents were born and raised locally, and seven were from further afield, including outside Britain. Since St. Mary Magdalen was a leprosarium, it is possible that these young people were taken there or travelled there voluntarily for support, including medical care. Archaeological and palaeopathological data support the notion that care was provided at this facility, and that leprosy stigma as we know it today may not have existed in this time and place.

KEYWORDS: Stable Isotopes, Strontium, Oxygen, Amelogenin Peptides, Infectious Disease, Stigma, Migration, Early Medieval

INTRODUCTION

LEPROSY IN THE PRESENT AND PAST

Leprosy, also known as Hansen's Disease, is a bacterial infection caused by *Mycobacterium leprae* or *Mycobacterium lepromatosis*. Clinically, leprosy is a disease of the peripheral nervous system that possesses the capacity to affect the skin, extremities, vocal and respiratory tracts, mucous membranes, eyes, kidneys, endocrine system, and bone (Davey and Schenck 1964; Walker and Lockwood 2006). Leprosy is considered a spectral disease meaning the 'type' of leprosy a person can develop presents along a broad immune spectrum, ranging from the high immune-resistant tuberculoid form (paucibacillary) to the low immune-resistant lepromatous form (multibacillary) (Walker and Lockwood 2006; Lastória and Abreu 2014). Lepromatous leprosy elicits pathognomonic, or diagnostic patterning of lesions that allow it to be identified in archaeological skeletons (Møller-Christensen 1961). These skeletal lesions include destruction and loss of the facial bones (resorption of the anterior nasal spine, remodelling of the nasal margins, abnormally porous/new bone formation on the oral and nasal surfaces of the palatal bones, destruction of the inferior nasal conchae and vomer, abnormal porosity and resorption of the alveolar process), and characteristic destruction and remodelling of the hand and foot bones (acro-osteolysis and concentric atrophy of the phalanges, metacarpals, and metatarsals) (see Møller-Christensen 1961, Andersen and Manchester 1987, Andersen and Manchester 1988; Andersen and Manchester 1992; Andersen et al. 1992; Andersen et al. 1994; Ortner 2008).

Leprosy has a complex biological and social history, and today can be linked with stigma and community expulsion in many endemic areas. Although leprosy is commonly associated with Medieval Europe, approximately 200,000 people are diagnosed with the infection annually (World Health Organization 2015; 2019). Once infected, leprosy mycobacteria multiply, slowly leading to a prolonged incubation period that can last of upwards to 30 years (Bhat and Prakash 2012; World Health Organization 2015; 2019). Leprosy can affect individuals of all ages, but is considered rare in younger individuals, likely due to the lengthy incubation periods associated with the manifestation of the disease (*ibid*). However, when compared to adults, non-adults that do develop leprosy are at an increased risk of developing the more severe lepromatous form potentially with subsequent permanent disabilities complicated by issues of delayed diagnosis, inadequate nutrition, immunodeficiencies, and pubertal endocrine system disruption (Davey and Schenck 1964; John et al. 2005; Butlin and Withington 2018). In situations where leprosy affects people who have not reached adulthood, medical treatment and community education related to de-stigmatisation are of equal importance for familial, community, and hospital care networks (Butlin and Withington 2018). Unsurprisingly, leprosy rates in children are higher in those who have familial contacts with leprosy (Fine 1982; Melsom et al. 1980; Jopling and McDougall 1988; Jain et al. 2002; Goulart and Goulart 2008; Lydyard et al. 2010).

Although the effects of leprosy and courses of treatment for non-adults are fairly well documented today through the World Health Organization and its affiliated charities (e.g. Lepra), archaeological evidence of leprosy in children, including how it was treated and attitudes of society to those affected, is notably absent in the published literature. Many historical sources repeatedly cite that people with leprosy in the past were stigmatised and treated poorly, however, recent evidence suggests that this interpretation is a more contemporary view (Rawcliffe 2006: 13-43; Edmond 2009: 61-109). This calls into question whether the historical and archaeological evidence of leprosy related stigma and community expulsion in the Medieval period, particularly prior to the 12th century AD, can be supported (Touati 2000; Roberts 2020: 280; Rawcliffe 2006: 67-78; Demaitre 2007: 99-102; Filipek et al. 2021). In reviews of archaeological contexts that include skeletons with evidence of leprosy from prehistory to the 12th century AD in Asia, Africa, and Europe, there is usually no differentiation in the burial treatment of those with leprosy (Roberts 2020: overview, 191-

280; Filipek et al. 2021). Further, analyses of mobility isotopes from children and adolescents with leprosy buried at the Late Saxon (10th – 11th centuries AD) cemetery parish church site of St. John at the Castle Gate/Timberhill (Norwich, England) revealed isotope ratios and values consistent with their origin within the burial location. These non-adults had not been expelled from their communities (Filipek et al. *in prep*). A further examination of unique leprosy contexts, such as leprosaria, is warranted to examine if any differences in mobility histories exist between them and non-leprosarium contexts.

The aim of this paper is to explore social reactions towards young people with leprosy during a key historical and cultural event in England: the Early-Late Medieval transition (9th – 12th centuries AD). In doing so, it examines amelogenin peptides and strontium and oxygen isotope ratios from the enamel of late forming teeth of individuals buried at the St. Mary Magdalen leprosarium (Winchester, Hampshire, England) to assess whether there is evidence of mobility in adolescents with leprosy and if so, who was moving? These data are combined with archaeological and palaeopathological evidence to provide a snapshot of migration and its relationship to leprosy.

Archaeological Context

The Magdalen Hill Archaeological Research Project (MHARP) began in 2007 with the aim of investigating the archaeological development of the St. Mary Magdalen leprosarium (Winchester; Figure 5.3.1) and its later transformations into other facilities (e.g. 16th century AD almshouse, English Civil War military camp, and a 17th century AD prison for Dutch prisoners of war). The first documentary reference to the site as a dedicated leprosarium can be traced to 1148 from the Winton Domesday with a later re-foundation c. 1180; however, archaeological evidence suggests the original foundation precedes this date (Barlow et al. 1976:90; Roffey and Tucker 2012; Roffey 2012). Archaeological evidence for these earlier phases includes a range of timber buildings with linear features, small masonry structures, and a cemetery, all of which underlay the re-foundation of the documented hospital in the 12th century (Roffey and Tucker 2012; Roffey 2012). The cemetery associated with this earlier context (North Cemetery) is archaeologically distinct from the later cemetery (South Cemetery) that is associated with the site's 12th century AD re-foundation (*ibid*). Skeletal analyses of people who were buried within the North Cemetery show over half were children

and adolescents (<25 years), and the entire burial population showed a high prevalence (~86%) of skeletal lesions diagnostic of lepromatous leprosy (Roffey and Tucker 2012; Filipek et al. 2021). Radiocarbon dates from the North Cemetery (Table 5.3.1) suggest these earlier burials likely began sometime in the 10th century AD, and excavations reveal that individuals were interred in discrete, anthropomorphic graves with westward-facing head niches/earthen pillows, demonstrating a considerable degree of care in their construction (Gilchrist and Sloane 2005: 132-133; Roffey and Tucker 2012). Burial goods were also found, which is a relatively rare phenomenon in Medieval Christian cemeteries (Roffey and Tucker 2012; Roffey 2020). For example, Sk. 27 was buried with a Pilgrim Badge from the shrine of St James at the Santiago de Compostela Monastery in Spain (Roffey et al. 2017), and Sk. 19 was buried with adapted artefacts (e.g. modified feeding bowls) associated with the likely difficulties this individual experienced with eating (e.g. impaired hand function) (Roffey and Tucker 2012; Lastoria and Abreu 2014; Filipek et al. 2021). This individual also showed evidence of a well-healed left amputation of his foot, suggesting some level of individualised, medical and palliative care at St. Mary Magdalen (Roffey and Tucker 2012; Filipek et al. 2021).

Figure 5.3.1 Location of Winchester with aerial view of excavations of St. Mary Magdalen leprosarium (inset).

In contrast to the North Cemetery, all burials from South Cemetery (post-1150 AD) are on notably different alignments and reveal more haphazard burial treatments (e.g. multiple and

truncated burials with no anthropomorphic grave cuts). This may indicate some degree of change over time in social perceptions evidenced by the differences in burial practices at St. Mary Magdalen. The individuals in the South Cemetery also revealed a lower prevalence of lepromatous leprosy (<40%) (Roffey and Tucker 2012), which is consistent with other post 12th century leprosaria contexts in England (e.g. Chichester; Lee and Magilton 2008: 263-265). This perhaps signifies that the form of leprosy present during the Later Medieval period was less-severe and therefore did not leave any skeletal lesions (e.g. paucibacillary cases), or that the ability to accurately identify leprosy waned after the 12th century AD.

It may also be a general reflection of the decline in prevalence of leprosy in the Later Medieval period (14th century onwards) possibly due to the rise of other infectious diseases, such as the Black Death and tuberculosis (Manchester 1984; Manchester and Roberts 1989; Roberts 2002; Roffey and Tucker 2012; Crespo et al. 2019; Roberts 2020: 291-301). In common with leprosaria elsewhere in England, St. Mary Magdalen (Winchester) ceased to function exclusively as a leprosarium by the 14th century AD (Roberts 2002; Roffey and Tucker 2012). Understanding the pathological conditions evident in individuals buried in these early facilities and where they may have been coming from, will help to contribute to a growing body of evidence on mobility histories and the potential care and treatment the sick received in the past.

Table 5.3.1 – Radiocarbon dates and SNP types for individuals buried at St. Mary Magdalen (Winchester, England).

INDIVIDUAL	CEMETERY	CAL. ^{14}C DATE (95% CI)	SNP TYPE	REFERENCE
SK. 8	North	AD 1010-1160	2F	Roffey and Tucker 2012
SK. 9	North	AD 890-1040	N/A	Roffey 2012
SK. 14	North	AD 995-1033	2F	Schuenemann et al. 2013
SK. 27	North	AD 1020-1162	2F	Roffey et al. 2017
SK. 2	South	AD 1268-1283	3I	Schuenemann et al. 2013
SK. 5	Chapel	AD 1290-1410	N/A	Roffey and Tucker 2012

EVIDENCE FOR LEPROSY IN THE YOUNG PEOPLE BURIED AT ST. MARY MAGDALEN

An infectious pathological stimulus will only produce skeletal changes if the affected person has the disease for long enough before death (Wood et al. 1992). This is especially relevant when viewing the severe and potentially debilitating bone changes diagnostic of lepromatous leprosy in the adolescent cohorts at St. Mary Magdalen. The pathognomonic lesions present

in their skeletons include rhinomaxillary changes (Figure 5.3.2), acro-osteolysis and concentric atrophy of the hands and feet, 'knife-edge' or mediolateral remodelling of the metatarsal shafts, resorptive grooves on the palmar surfaces of the hand phalanges (termed volar grooving – Andersen and Manchester 1987) caused by flexion contractures, tarsal fusion and dorsal exostoses (Andersen and Manchester 1988), and subperiosteal new bone formation on the distal shafts of the tibiae and fibulae. Four individuals (SK. 8, SK. 28, SK. 52, SK. 56) also showed evidence for leprogenic odontodysplasia (Figure 5.3.3), which is the concentric constriction and dysplastic development of the anterior maxillary dentition caused by leprosy bacilli infiltration into the developing tooth in early childhood (Danielsen 1970; Reichart 1976). The development of leprogenic odontodysplasia and the pathological skeletal lesions associated with lepromatous leprosy are presumed to commence at approximately the same time (Ortner 2008), revealing a more defined chronology for the onset of skeletal changes and the time elapsed before death.

Figure 5.3.2 Skull of Sk. 28 (aged 12.5-13.5 years) from St. Mary Magdalen Leprosy Hospital (Winchester, England) displaying evidence of rhinomaxillary syndrome (black arrows) including 1.) Rounding of the nasal aperture, 2.) Resorption of the anterior nasal spine, 3.) Recession of the alveolar process, and 4.) Widening and flattening of the nasal bones.

Although St. Mary Magdalen leprosy hospital was referenced as a dedicated leprosarium in the mid-12th century AD (Roffey and Marter 2012), it is important to note there were several other concomitant pathologies identified in these skeletons. All of the young people from the hospital possessed at least one non-specific indicator of childhood stress (linear enamel hypoplasia and/or cribra orbitalia), and eight yielded a higher dental development age in comparison to their skeletal age (long bone length shorter for their age, as estimated by dental development and eruption). Both these observations potentially indicate some form of arrested development as a consequence of leprosy but other aetiologies likely factor in an

aetiological list, e.g. early childhood nutritional stresses. Other comorbidities within this young cohort included: pathologically induced fractures, possible tuberculosis or mycotic infections, and residual rickets. Individuals also displayed high levels of dental calculus formation (i.e. mineralised plaque), which is commonly found amongst patients with lepromatous leprosy today and may be an indicator of poor oral hygiene resulting from inflammation of the oral cavity, mouth-breathing due to facial paralysis and/or chronic inflammation/congestion of the nasal passages, a softer pulpy diet, or a combination of the above (Reichart 1976; Ogden and Lee 2008; Souza et al. 2009; Rawlani et al. 2011; Roffey et al. 2017).

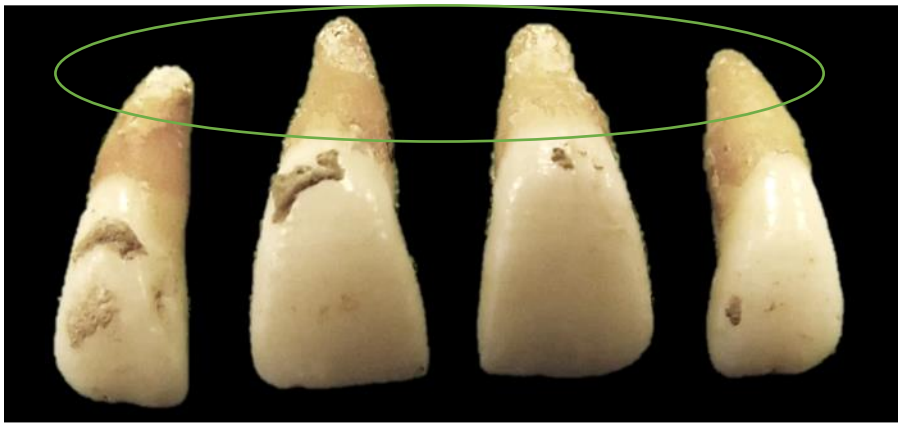

Figure 5.3.3 Leprogenic odontodysplasia (arrested development of the teeth) in the maxillary incisors of Sk. 56 due to an infiltration of Mycobacterium leprae (encircled). Based on dental development (Moorrees et al. 1963; AlQahtani et al. 2010), this arrested growth would have occurred between 6.5-7.5 years of age.

Previous pathogenic aDNA analyses on individuals buried in the cemetery associated with St. Mary Magdalen revealed the presence of two different SNP types and subtypes: 3I and 2F. Type 3I likely originated in Central Asia and until recently, was thought to be the sole strain type responsible for all European leprosy (both archaeological and modern), and Type 2F is considered the precursor strain that migrated with humans from the Middle East to modern-day India and South-East Asia (Monot et al. 2009; Economou et al. 2013; Schuenemann et al. 2013; Taylor et al. 2013; Mendum et al. 2014; Schuenemann et al. 2018). The appearance of the 2F strain at St. Mary Magdalen is of particular relevance having only been previously identified in a 7th century tomb from Italy (Belcastro et al. 2005; Donoghue et al. 2015) and concurrent Scandinavian and Irish contexts (Economou et al. 2013; Mendum et al. 2014;

Schuenemann et al. 2018; Taylor et al. 2018). The presence of this strain is linked to extended Middle Eastern trade networks, suggesting broad geographical connections in the Medieval dissemination of the disease (Economou et al. 2013; Mendum et al. 2014; Donoghue et al. 2015; Schuenemann et al. 2018). In order to view whether these younger cohorts with skeletal lesions diagnostic of lepromatous leprosy were from areas local to the leprosarium or from further afield, we applied strontium and oxygen isotope analysis of tooth enamel from late-forming teeth.

ISOTOPIC BACKGROUND

The analysis of the strontium ($^{87}Sr/^{86}Sr$) and oxygen ($\delta^{18}O$) isotope ratios of tooth enamel is a robust and well-established method for examining an individual's geographic origins (Evans et al. 2006a; 2006b; Bentley 2006; Eckardt et al. 2009; Montgomery 2010; Evans et al. 2010; Chenery et al. 2010; Evans et al. 2012; Moore et al. 2020). Tooth enamel is an avascular acellular tissue that is highly resistant to isotopic alterations both after mineralisation and in the post-mortem burial environment, making it highly suitable for multi-isotope analysis (Budd et al. 2000; Montgomery 2010; Moore et al. 2020). The strontium ratios and oxygen isotope values of enamel are ultimately subsumed through food and water during the development of the enamel of the teeth, respectively, and are linked to the geological and climactic biospheres during the enamel's mineralisation periods (Evans et al. 2010; Evans et al 2012). Strontium weathers from the bedrock into soil and subsequently through the human food chain largely unfractionated. Therefore, strontium ratios within enamel can directly reflect the geological area from which an individual derived their food and water during childhood and adolescence (Bentley 2006; Montgomery 2010; Coelho et al. 2017). Oxygen isotopes derived from enamel are a reflection of an individual's drinking water values and indirectly reflect the isotopic composition of a location's meteoric water, which varies by altitude, temperature, latitude, and other climatic factors (Darling et al. 2003; Darling and Talbot 2003; Evans et al. 2012; Pederzani and Britton 2019). Once consumed, oxygen undergoes a metabolic fractionation process, and therefore regression formulae (see methods) must be applied to values in order to make comparisons with modern data (Daux et al. 2008; Chenery et al. 2010; Chenery et al. 2012). Interpretation of $\delta^{18}O$ results must also consider the potential influence of other biological and culturally-mediated behaviours, such

as the alteration of water temperature in the preparation of foodstuffs (i.e. stewing, brewing, boiling; Brettell et al. 2012), pathophysiological influences (e.g. diabetes and anaemia; Reitsema 2013), or additional metabolic fractionations through other biological processes (e.g. ingestion of breast milk; Wright and Schwarcz 1998; Tsutaya and Yoneda 2015).

Based on these principles, both isotope systems can reveal whether strontium and oxygen isotope values from adolescents buried within this leprosarium context are consistent with their burial location when their tooth enamel was developing. Despite being a key factor in disease transmission and the subsequent social milieu surrounding disease status, few studies have examined the mobility histories of individuals with skeletal lesions diagnostic of specific infectious diseases (Roberts et al. 2013; Kendall et al. 2013; Quinn 2017; Roffey et al. 2017). This is in part due to the difficulty in establishing whether a person was infected with the disease before, during, or after moving to the area (*ibid)*. By examining isotope ratios from younger individuals, the likelihood that their geographical origins during childhood overlap with the location where they were infected with leprosy increases due to the longer incubation periods associated with the disease (Walker and Lockwood 2006) and, concurrently, the opportunity for movement during their lifetime is reduced; i.e. they had less time or ability to move before death (Montgomery et al. 2000; Evans et al. 2006a).

Characterizing the Strontium and Oxygen isotope ranges for Winchester

The city of Winchester is located on the South Downs approximately 30km north from the southern coast of England. The St. Mary Magdalen leprosy hospital site is situated approximately 1.6 km east of Winchester Cathedral, and the local geology is characterised by Cretaceous Chalk within a 10km radius (British Geological Survey 2007). Within a larger 30 km radius, the dominant bedrock geology remains Cretaceous Chalk, however deposits of Oligocene and Eocene sands, clays, silts, and gravel can be found to the south and in a small area to the north of the site, and Gault Clay and Upper Greensand formations can be found approximately 25km to the east on the western margin of the Weald (British Geological Survey 2007).

In order to establish the local range of an area, both isotope systems rely on isotopic baselines for comparison, and the geographic distributions of isotope compositions across Britain are well-evidenced (Darling et al. 2003; Darling and Talbot 2003; Evans 2006; Daux et al. 2008;

Evans et al. 2010; Montgomery 2010; Evans et al. 2012; Pellegrini et al. 2016). Data for $^{87}Sr/^{86}Sr$ values are compared against a dataset of bioavailable strontium isotope ratios for Britain provided by Evans and colleagues (2010; 2018). The expected $^{87}Sr/^{86}Sr$ values should lie between 0.7072 (value of the Cretaceous chalk) and 0.7092 (modern seawater) (Evans et al. 2010; Evans et al. 2012; Evans et al. 2018). Analyses of modern rodent teeth and a human dentine sample from the site revealed $^{87}Sr/^{86}Sr$ value ranges of 0.7077 – 0.7082, which is consistent with predicted ranges (Taylor et al. 2013).

The $\delta^{18}O$ from groundwater within Britain is primarily influenced by precipitation (Darling et al. 2003; Darling and Talbot 2003). The overall $\delta^{18}O_P$ range for archaeological humans excavated from Britain is 17.7‰ ± 1.4 (2σ), with westerly and southerly areas having higher rainfall, including Winchester, producing a mean $\delta^{18}O_P$ value of 18.2‰ ± 1 (2σ, n=40) (Evans et al. 2012). This is statistically different from easterly, lower rainfall areas that produce a mean of 17.2‰ ± 1.3 (2σ, n=83) (*ibid*). On this basis, oxygen isotope ratios may be used to discriminate broad geographical origins. Individuals raised local to the Winchester area should fall within the southerly, higher rainfall range (>700mm/year) with predicted $\delta^{18}O_P$ ranging from 17.2-19.2‰ (Evans et al. 2006b; Eckardt et al. 2009; Evans et al. 2012). Additional conversions to drinking water values ($\delta^{18}O_{DW}$) help to provide comparisons with well-documented modern groundwater values, however these conversions are associated with larger errors (±1, 2σ) and therefore should be interpreted with caution (Daux et al. 2008; Pollard et al. 2011; Chenery et al. 2012). The modern $\delta^{18}O_{DW}$ for Winchester falls between -7.0 and -5.6‰ (Eckardt et al. 2009).

Osteological Analyses and Biomolecular Methods

In order to analyse the individuals from this site and take tooth samples for subsequent isotopic analyses, permissions with ethical justifications were sought and approved both from the curators of the human skeletal remains and the in-house ethics committee at the Department of Archaeology at Durham University. The Codes of Ethics (2010) and the Codes of Practice (2010) from the British Association of Biological Anthropologists and Osteoarchaeologists (BABAO) (https://www.babao.org.uk/publications/ethics-and-standards) were strictly adhered to in the sampling from the individuals selected for study.

Individuals

Adolescents (c. 10-25 years) with recorded evidence of leprosy were selected and re-evaluated for further study at the Osteology Lab within the Department of Archaeology at the University of Winchester. Only individuals with bones or teeth showing lesions diagnostic of lepromatous leprosy (see Ortner 2008) were chosen for further analyses. A total of 19 tooth samples from 19 adolescent individuals from the North Cemetery were selected for radiogenic strontium and stable oxygen isotope analysis, four of which were also selected for amelogenin peptide analyses (Table 5.3.2). Either second or third molars with no signs of pathology were selected to represent the most recently formed teeth (at the time of death) available for each individual and to lessen the influence of biologically-mediated behaviours (e.g. breastfeeding). Teeth were removed from their alveolar sockets by hand and were photographed by the Department of Archaeology at Durham University in the occlusal, buccal, lingual and apical aspects before sample preparation. All remaining tooth sample material and archival photos are scheduled to return to the curating institution.

Age and Sex

This study chose to focus on adolescent individuals due to their liminal biological and social statuses, and general underrepresentation in previous archaeological enquiries (Lewis 2016). By looking at the adolescent experience, we can magnify the larger biological and social impacts of disease on a population, which make them a 'pivotal conduit' for understanding the overall effects of environments and cultural reactions on disease (Redfern and Gowland 2011: 111; Mays et al. 2017). Individuals were reassessed for age at death based on tooth development in accordance with the methods set out in Moorrees et al. (1963) and AlQahtani et al. (2010) and with the aim of identifying adolescents. Due to disparities between dental and skeletal ages (i.e. dental development yielding an older age than skeletal fusion), dental developmental was used as the primary age estimation method as it is largely buffered against environmental and biological stressors (Smith 1991; Cardoso 2007; Conceição and Cardoso 2011). Our study assigned individuals between the ages of 10 – 25 years at death as adolescents to align with both Medieval social definitions (Lewis 2016), and modern biological understandings of growth and development (World Health Organization 1993:1; Patton et al. 2016; Sawyer et al. 2018).

Biological sex was assigned using standard osteological methods (Acsádi et al. 1970; Phenice 1969; Buikstra and Ubelaker 1994: 15-38) on the adolescents who showed evidence of sexual dimorphism and had achieved and/or surpassed the deceleration phase of puberty, in accordance with the methods recommended by Shapland and Lewis (2013) and Lewis and colleagues (2016). For the remainder of the unsexed individuals, a previous study (Taylor et al. 2013) determined the biological sex of Sk. 8 (aged 8.5-9.5 at death) through aDNA analyses, and the remaining five individuals (Sk. 28, Sk. 41, Sk. 45, Sk. 52, Sk. 54) had their sex determined using amelogenin peptide extraction methods described by Stewart et al. (2016; 2017). The genes to form the protein amelogenin are located on the sex-linked chromosomes and can easily be extracted with minimal destruction to the tooth by performing surface acid etching of the enamel surface (*ibid*; see methods below).

Sample Preparation

Sections of core enamel for isotope analyses were extracted in the Isotope Laboratory in the Department of Archaeology, Durham University. Tooth sample preparation followed the guidelines of Montgomery (2002). Molar surfaces were first abraded using tungsten carbide burrs to remove any exogenous material and 15-25mg of enamel was sectioned using diamond edged dental saws. All surface enamel and any adhering dentine were mechanically removed from enamel sections. Processed chips of core enamel were sealed in Eppendorf microtubes and transferred to the (class 100, HEPA-filtered) laboratory facilities at the NERC Isotope Geosciences Laboratory (NIGL) at the British Geological Survey for further preparation (Keyworth, Nottinghamshire, England). The molars of unsexed individuals were retained for enamel surface acid etching for amelogenin peptide extraction.

Amelogenin peptide extraction methods

Amelogenin peptide extraction was conducted on five individuals following the methods of Stewart and colleagues (2017). A wide area of molar enamel that avoided any previous sampling was selected for surface acid etching. The enamel etch area was washed with 3% H_2O_2 for 30 seconds, and then rinsed with ultra-pure water. Enamel surface acid etching was carried out by using 80 µl 10% HCl for two minutes on the chosen area. The first surface acid etch was discarded, and a second two minute etch was performed and subsequently retained.

The peptides in the etch solution were bound to C18 resin ZipTips, and conditioned three times with 10 µl 100% acetonitrile and 0.1% formic acid. The peptides were eluted off the C18 resin using 4 µl of elution buffer (60% acetonitrile/0.1% formic acid). The peptide samples were frozen at -18°C and lyophilised.

Freeze-dried samples were transferred to the School of Pharmacy and Biomolecular Sciences, University of Brighton, Sussex, England where they were dissolved in 12 µl 0.1% trifluoroacetic acid (TFA) in water, and then centrifuged for 5 minutes to remove particulates, and transferred to 10 µl glass autosampler vials. Approximately 5 µl of sample was analysed by a reverse-phase nanoLC-MS with a liquid chromatograph (nanoRS U3000; Thermo Fisher Scientific) coupled to a hybrid quadrapole orbitrap mass spectrometer (Q Exactive; Thermo Fisher Scientific). The data were searched against the human proteome (UniportKB, 10/15) with MaxQuant v 1.5.1.2 using default search settings with methionine oxidation as a variable modification, unspecific digestion mode and a minimum peptide length of six (Stewart et al. 2017).

Isotope Methods

For strontium isotope analyses, the sectioned core enamel was further prepared and measured according to Evans and colleagues (2006). In a clean laboratory, the enamel sample was first cleaned ultrasonically in high purity water to remove dust, rinsed twice, dried down in high purity acetone, and then weighed into pre-cleaned Teflon beakers. A known amount of ^{84}Sr tracer solution was added to each sample, which was dissolved in Teflon distilled 8M HNO_3. The sample was converted to chloride using Quartz distilled 6M HCl and then taken up in 2.5M HCl. The strontium was extracted using Eichrom Dowex AG50X8 resin and the samples were loaded onto Rhenium filaments (Birck 1986). The isotope composition and concentrations were determined by Thermal Ionization Mass Spectroscopy (TIMS) using a Thermo Triton multi-collector mass spectrometer. The international standard for $^{87}Sr/^{86}Sr$, NBS-987, gave a value of 0.710251 ± .000005 (n = 19, 2sd) during the analysis of these samples. Procedural blank values were less than 100pg.

For oxygen isotope analyses, approximately 3 mg of powdered enamel was loaded into a glass vial and sealed with septa. The vials were transferred to a hot block at 90°C on a GV Multiprep

system. The vials were evacuated and 4 drops of anhydrous phosphoric acid added. The resultant CO_2 was collected cryogenically for 14 minutes and transferred to a GV IsoPrime dual inlet mass spectrometer. The resultant isotope values are reported as delta (δ) values, in parts per thousand (per mil; ‰) normalized to the VPDB scale using a within-run calcite laboratory standard (Keyworth Carerra Marble, KCM) calibrated against SRM19, NIST reference material. These ratios were normalised to the VSMOW scale using the published conversion equation of Coplen (1988): VSMOW = (1.03091 x $\delta^{18}O_{VPDB}$) +30.91. Analytical reproducibility for this run of laboratory standard calcite (KCM) is 0.09‰ (1σ, n = 6) for $^{18}O_{VSMOW}$ and ± 0.05‰ (1σ, n = 6) for $^{13}C_{VPDB}$. The reproducibility of the enamel was based on average 1σ of five duplicate pairs is ± 0.07‰. The carbonate oxygen results ($\delta^{18}O_{(C)VSMOW}$) were converted to phosphate values ($\delta^{18}O_{(P)VSMOW}$) using the regression equation $\delta^{18}O_P$ = 1.0322 x $\delta^{18}O_C$– 9.6849, which produces an associated error of ± 0.29‰, 1σ (Chenery et al. 2010; Chenery et al. 2012). The carbonate oxygen results ($\delta^{18}O_C$) were converted to drinking water values ($\delta^{18}O_{DW}$) using Daux and colleagues' (2008) equation 6 in accordance with Chenery and colleagues' (2012) calculation: $\delta^{18}O_{DW}$ = 1.590 x $\delta^{18}O_C$– 48.634. The calculation of drinking water values involves larger uncertainties (± 1 ‰, 2σ)(Chenery 2012; Pollard et al. 2011) and therefore these values are used as general guidance only.

RESULTS

The results for biological sex from the amelogenin peptide extraction of five individuals, and strontium and oxygen isotope data for all 19 individuals, are presented in Tables 5.3.2 and Figure 5.3.4. Amelogenin peptide extraction revealed three males (Sk. 28, Sk. 41, Sk. 54) and two females (Sk. 45, Sk. 52). Strontium isotope ratios range from 0.7084 to 0.7103 (mean 0.7090, ± 0.0012, 2σ). The $\delta^{18}O_{(P)VSMOW}$ values for the individuals show a wide range of 15.6‰ to 19.3‰ (mean 17.8 ± 1.6‰ 2σ), with corresponding $\delta^{18}O_{DW}$ values ranging from -9.7‰ to -4.1‰ (mean -6.3 ± 2.4‰ 2σ).

Table 5.3.2 – Adolescents selected for study with resultant amelogenin peptide data, and strontium and oxygen isotope data. $\delta^{18}O_P$ calculated from Chenery et al. (2012), and $\delta^{18}O_{DW}$ calculated from Daux et al. (2008) equation 6, in accordance with Chenery et al. (2012).

BURIAL	SEX	AGE	TOOTH	Sr PPM	$^{87}Sr/^{86}Sr$	$\delta^{13}C_{VPDB}$ (‰)	$\delta^{18}O_{(C)VSMOW}$ (‰)	$\delta^{18}O_{(P)VSMOW}$ (‰)	$\delta^{18}O_{DW}$ (EQN 6) (‰)

SK. 8	M**	DD: 8.5-9.5; SA: 7-8	L. Max. M2	81	0.7089	-12.37	26.67	17.9	-6.2
SK. 9	M	22.5-23.5	R. Max M2	102	0.7089	-12.65	26.25	17.4	-6.9
SK. 14	M	16.5-17.5	L. Max. M3	57	0.7085	-13.04	26.63	17.8	-6.3
SK. 15	M	20.5-22.5	L. Man. M3	96	0.7088	-12.88	26.77	18.0	-6.1
SK. 16	M	20.5-21.5	R. Max. M3	91	0.7086	-12.65	26.95	18.1	-5.8
SK. 18	M	DD: 18.5-19.5; SA: <14	R. Man. M3	111	0.7093	-13.66	26.68	17.9	-6.2
SK. 21	M	DD: 21.5-22.5; SA: 16-19	L. Max. M3	82	0.7084	-13.2	26.42	17.6	-6.6
SK. 25	M	DD: 18.5-20.5; SA: 17-19	R. Man. M3	56	0.7091	-13.21	27.27	18.5	-5.3
SK. 26	M	22.5-23.5	L. Man. M3	99	0.7087	-12.58	27.59	18.8	-4.8
SK. 27	M	22.5-23.5	R. Man. M3	70	0.7103	-12.79	26.09	17.2	-7.2
SK. 28	M*	12.5-13.5	L. Max. M2	73	0.7094	-12.89	27.88	19.1	-4.3
SK. 29	M	18.5-19.5	R. Max. M3	41	0.7095	-13.85	26.83	18.0	-6.0
SK. 39	M	16.5-17.5	R. Max. M3	107	0.7102	-13.44	26.7	17.9	-6.2
SK. 41	M*	13.5-15.5	R. Max M3	89	0.7086	-12.57	26.5	17.7	-6.5
SK. 45	F*	DD: 15.5-16.5; SA: 10-12	L. Man. M2	111	0.7086	-12.83	26.32	17.5	-6.8
SK. 46	M	16.5-17.5	R. Max. M3	120	0.7090	-13.12	26.18	17.3	-7.0
SK. 52	F*	DD: 12.5-13.5; SA: 9-11	L. Man. M2	69	0.7099	-13.27	28.03	19.3	-4.1
SK. 54	M*	DD: 14.5-15.5; SA: 9-11	R. Man. M2	78	0.7085	-12.21	26.09	17.3	-7.2
SK. 56	M	16.5-17.5	R. Max. M2	94	0.7087	-12.55	24.49	15.6	-9.7

* Biological sex determined by Amelogenin Peptide Extraction; **Biological sex determined via aDNA analysis (Taylor et al. 2013); DD: Dental development age; SA: Skeletal age based on epiphyseal fusion; Man: Mandibular; Max: Maxillary.

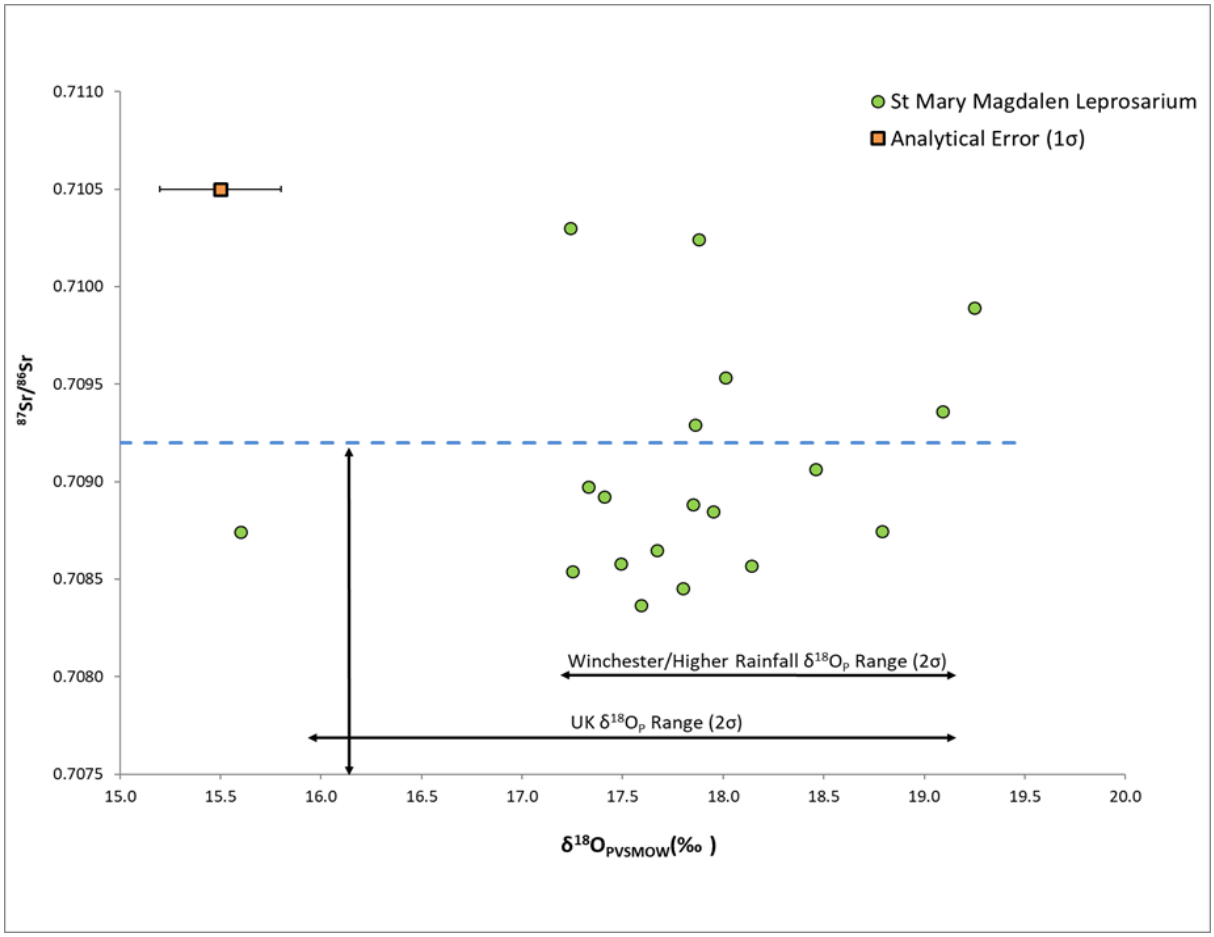

Figure 5.3.4 Strontium and oxygen isotope ratios showing mobility histories of 19 adolescents from the St. Mary Magdalen leprosy hospital, Winchester. The dashed line represents the strontium isotope ratio for modern seawater (0.7092) (Veizer 1989), and therefore the upper limit for the predicted strontium isotope ratios for the Winchester area.

DISCUSSION

The results presented here contribute to an evolving understanding of mobility and its relationship to infectious disease and medical treatment in England during the Early-Late Medieval transition (9th – 12th century AD). Particulars of early leprosaria and the people populating them are not well-documented (Rawcliffe 2006: 302-305), and this study helps to reveal further nuanced information that contributes to larger archaeological narratives about disease in the past.

Amelogenin peptide data reveal that adolescent females with lepromatous leprosy (Sk. 45, Sk. 52) were present in the North Cemetery population at St. Mary Magdalen. These individuals are also the youngest known females with lepromatous leprosy in the archaeological record. Previous to this study, only four older females (46+) had been

identified in the North Cemetery, and therefore the presence of adolescent females broadens our knowledge of the demographic make-up of those in the leprosarium community. Prior to the 15th century AD, hospitals and leprosaria were purportedly sex-specific to avoid impropriety (Rawcliffe 2006: 144-145; Magilton 2008: 57-59), with the exception of older nurses being present to provide care in male institutions (Orme and Webster 1995: 82-83; Rawcliffe 2006: 148-149; 261-262). Given the advanced signs of lepromatous leprosy in these adolescent females, it is more likely they were there for care and treatment and not there to help provide it, as some have previously suggested (Orme and Webster 1995: 22-23; 82-83; Magilton 2008: 59). In contrast, Green (1989:434) poses that in Medieval Europe, including England, "women's health was women's business," and that much of the medical care provided to females was undertaken by community midwives until the 15th century AD, when legislation was enacted to limit the role of female medical practitioners. This may explain the preponderance for male skeletons in urban hospital contexts and the introduction of mixed-sex leprosaria in the 15th century AD (Gilchrist and Sloane 2015: 205-206; Magilton 2008: 57-59). However, documentary accounts from Eadmer's *History of Recent Events in England* indicate that Archbishop Lanfranc's leprosarium were initially open to both men and women in the 11th century AD (Orme and Webster 2005: 20-23, 109-111) but these rules seem to have changed some time in the 12th century AD. This new evidence for female patients in a hospital context warrants further bioarchaeological investigations into the role and social identities of women as patients in leprosaria, which is a particularly invisible group both historically and archaeology.

Individuals with $^{87}Sr/^{86}Sr$ and $\delta^{18}O_{(P)VSMOW}$ values consistent with the study area (Winchester)

Twelve individuals have $^{87}Sr/^{86}Sr$ values between 0.7072 and 0.7092 and $\delta^{18}O_{(P)VSMOW}$ values between 17.3 – 18.8‰, compatible with residential origins on a terrain underlain by chalk with a higher rainfall area, such as Winchester (Evans et al. 2012), and comparable to locals from prior studies (Evans et al. 2006; Eckardt et al. 2009). Equivalent $\delta^{18}O_{DW}$ values based on the conversion equations in Chenery et al. (2012) provide a drinking water range of -7.0 and -4.8‰, consistent with the modern precipitation values for Winchester given in Eckardt et al. (2009). Although there are alternative areas that produce a similar combination of strontium ratios and oxygen isotope ratios within the UK and further afield, the most parsimonious

interpretation is that these individuals spent their childhoods near the St. Mary Magdalen leprosarium.

Individuals with non-local $^{87}Sr/^{86}Sr$ but $\delta^{18}O_{(P)VSMOW}$ values consistent with British ranges

Six individuals (Sk. 18, Sk. 27, Sk. 28, Sk. 29, Sk. 39, Sk. 52) show higher than expected $^{87}Sr/^{86}Sr$ values (0.7093 – 0.7103) for an area underlain by marine carbonates such as in the Winchester area. These ranges are, however, common across silicate lithologies that can be found broadly throughout England (Evans et al. 2010; Evans et al. 2012).

All six individuals reveal oxygen ratios that are consistent with British ranges reported by Evans et al. (2012), however, two individuals had higher $\delta^{18}O_{(P)VSMOW}$ values in comparison to others. Sk. 28 gave a $\delta^{18}O_{(P)VSMOW}$ value of 19.1‰ ($\delta^{18}O_{DW}$ -4.8‰), and Sk. 52 showed a $\delta^{18}O_{(P)VSMOW}$ value of 19.3‰ ($\delta^{18}O_{DW}$ -4.1‰), which hover at the 2σ range for the Britain. Higher than expected $\delta^{18}O_{(P)VSMOW}$ values may be due to a variety of scenarios: they may indicate that these individuals come from the extreme western seaboard of Britain; or they may have spent their childhood further afield (e.g. in areas around the Mediterranean Sea) (Mitchell and Millard 2009; Chenery et al. 2010; Evans et al. 2012); their 2nd molars may show an isotopic offset from an extended period of breastfeeding (Wright and Schwarcz 1998; Tsutaya and Yoneda 2015); or the majority of their childhood diet may have come from milk or heated/cooked food and drink (Brettell et al. 2012).

Individuals with $\delta^{18}O_{(P)VSMOW}$ values inconsistent with British ranges

One individual (Sk. 56) revealed an $^{87}Sr/^{86}Sr$ value (0.7087) consistent with an area underlain by chalk (limestone), but had an unusually low $\delta^{18}O_{(P)VSMOW}$ value. Sk. 56, aged 16.5-17.5, had a $\delta^{18}O_{(P)VSMOW}$ value of 15.6‰ ($\delta^{18}O_{DW}$ -9.7‰), which is more than 2σ compared to the other individuals buried at the St. Mary Magdalen leprosy hospital, and at the 3σ threshold for British ranges reported by Evans et al. (2012). Oxygen isotope ratios fall with decreasing temperatures, and increasing altitude and latitude (Darling et al 2003; Evans et al. 2012; Figure 5.3.5), which indicate that Sk. 56's early childhood was spent in an area significantly cooler than Britain. Possible areas that may accommodate the combination of Sk. 56's strontium and oxygen isotope compositions include Iceland (Walser et al. 2019), coastal areas of Denmark or possibly Norway (Price et al. 2014), Central/Eastern Europe (Lecolle 1985;

Eckardt 2009; Voerkelius 2010; Hughes et al. 2014), or regions underlain by basalts, chalks, or limestones that produce similar oxygen isotope ratios. Another possibility is that Sk. 56 may have had a pathophysiological condition that altered their normal oxygen isotope composition (e.g. end stage renal failure) (Kuo et al. 2012; Reitsema 2013). Although leprosy does affect renal function (da Silva Junior et al. 2015), it is doubtful that end stage renal failure would be the cause of the low isotope ratio seen here as life expectancy at that time would be unlikely to be long enough to reflect changes in the tooth enamel (Kuo et al. 2012; da Silva Junior et al. 2015). However, the accumulation of isotopically light metabolites within the kidneys has not been investigated in leprosy patients, and therefore this possibility cannot be wholly excluded and warrants future investigation.

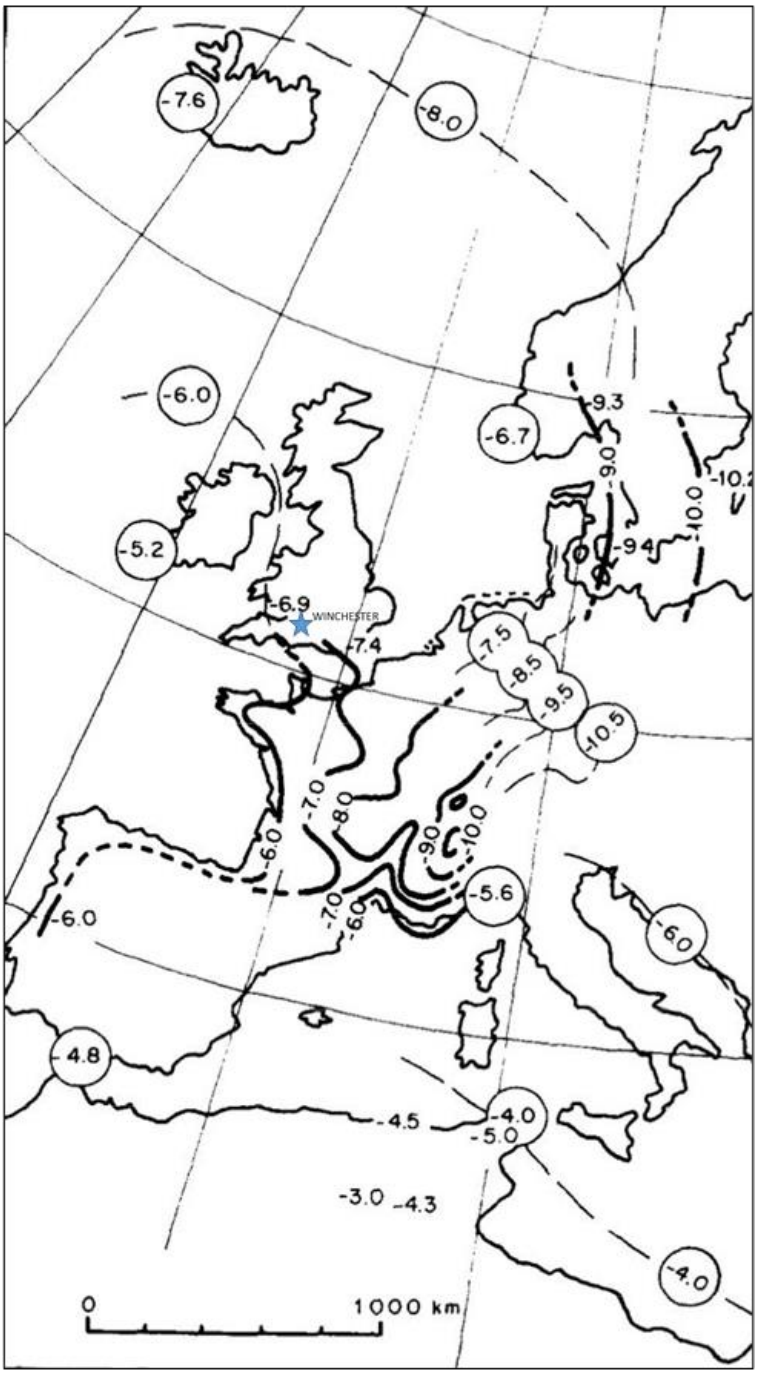

Figure 5.3.5. Oxygen isotope drinking water values from Western Europe with Winchester (starred) (Modified from Lecolle 1985).

The majority of individuals (n=12) from the sample population are considered to have been born and raised local to the Winchester area. This combined with the high number of people with lepromatous leprosy suggests that by this time, St. Mary Magdalen was functioning as a hospital for people with leprosy who resided both locally in Winchester, and non-locally (see next paragraph). This aligns with major Benedictine reforms in the 10th century AD that

necessitated monastic institutions provide a dedicated facility for care and provisions for the infirm (Dainton 1961:17-18; Orme and Webster 1995:15-18; Retief and Cilliers 2006; Huggon 2018).

Seven individuals fell outside the local ranges for strontium and oxygen isotope ratios. Four individuals revealed relatively common strontium isotope ratios for central and southern England but these were too high for Winchester, and two individuals (Sk. 28, Sk. 52) had oxygen isotope ratios that, if not a consequence of culturally-mediated behaviours, place their origins in an extreme western point in Britain or further afield (e.g. the Mediterranean). One individual (Sk. 56) had a significantly lower oxygen isotope ratios that, based on present data, is too low to have originated from Britain. These data suggest that the non-local individuals buried in the sample population did not all originate from similar geographic areas; i.e. mobility was more widespread.

Social reactions to leprosy

Previous researchers have refuted the long-held image of leprosaria as spaces for segregation and neglect. Life within the leprosarium likely offered security, medical and spiritual care, shelter and warmth, beds, food and ale, clothing, and transactional community contact (Rawcliffe 2006; Roberts 2013, 2020; Filipek et al. 2021). In order to receive these benefits within a leprosarium, a group agency model that culturally sanctioned and economically supported such an institution had to exist (Filipek et al. 2021). Further evidence with regard to burial construction (e.g. similar to those of high status ecclesiastical burials), the unique and individual burial goods (e.g. pilgrim badges and modified artefacts for feeding), and an individual who underwent a foot amputation (suggesting the existence of some level of medical and palliative care) further demonstrates that at this time and place, the idea of leprosy stigma and community ostracism was not part of the broad social milieu (Roffey and Tucker 2012; Roffey 2012; Roffey et al. 2017; Roffey 2020). The presence of both local (<30km) and non-local adolescents buried within this early leprosarium context implies that this care was extended to the local community's children with leprosy, as well as young people from the hinterland, and further afield. It is worth noting that isotope data cannot speak to the social inclusivity of individuals (local or non-local) within broader cultural contexts. For these reasons, bioarchaeologists must rely on burial practices, grave goods, and archaeological

context. As Shaw and colleagues (2016) highlight, cultural construction of social identity is not necessarily predicated on childhood origins, but is better reflected in the ways in which an individual's funerary identity is constructed. The combined funerary and isotope evidence indicate that previous notions of exclusion and maltreatment of individuals with leprosy in this period have been overstated.

We are not able to say, however, at what age these individuals travelled, whether they moved to Winchester due to its urban status as the *de facto* capital of England at the time (Roffey and Lavelle 2015), or purposefully travelled to Winchester to seek medical care for leprosy, nor can we determine how long they would have been cared for prior to death. We are also not able to ascertain whether these individuals had leprosy when they migrated, or if they developed leprosy after moving to the Winchester area. However, the long incubation period associated with lepromatous leprosy (c. 2 to 10 years, or more) (Bhat and Prakash 2012; Smith et al. 2015), their young age at death, and the advanced lepromatous skeletal lesions point towards the probability that non-local individuals likely migrated with the disease potentially to access the care and treatment provided in the St. Mary Magdalen leprosarium (Roffey and Tucker 2012; Filipek et al. 2021).

In future, analyses of carbon and nitrogen isotope values from the incremental dentine of these individuals could reveal more nuanced aspects of migration in relation to leprosy. This could go alongside considering subsequent care and treatment revealed by changes in their diet that may potentially be linked with their mobility histories, leprosarium provision, and pathophysiological stress. Further comparative studies of individuals with leprosy from the later contexts within the St. Mary Magdalen leprosarium cemetery (i.e. South Cemetery) may also indicate whether any sociocultural changes in the treatment of people with leprosy existed from the 12th century AD onwards. Lastly, further amelogenin peptide analyses would be highly beneficial in understanding the demographic variation, social identities, and access to medical care for children with leprosy in a Medieval institutional setting.

CONCLUSIONS

The aim of this study was to explore social reactions towards young people with leprosy during a key historical and cultural event in England: the Early-Late Medieval transition (9th –

12th centuries AD). This was achieved by exploring biological sex as an aspect of identity and the mobility histories of adolescents buried in the North Cemetery at the St. Mary Magdalen leprosarium in Winchester. These data were combined with archaeological and palaeopathological evidence to view the extent of the journeys these people with leprosy made to the leprosarium. Amelogenin peptide extraction demonstrated that two adolescent females were amongst the burial population, currently the youngest known females with leprosy in the archaeological record. Strontium and oxygen isotope values revealed that 12 individuals showed isotope ratios consistent with their burial environment, whilst seven individuals had origins inconsistent with the local area. Combining these results with the contextual and palaeopathological data indicates that social reactions to young people with leprosy were not necessarily negative, as previous historians suggest, and underscores the relevance and importance of using multidisciplinary and holistic approaches to understanding social reactions to disease in the past.

ACKNOWLEDGMENTS

Special thanks are given to Dr. Simon Roffey and Dr. Katie Tucker from the University of Winchester for facilitating access to the Winchester skeletons, and Hilary Sloane and Carlyn Stewart from the NERC Isotope Geosciences Laboratory for their assistance. KLF would also like to thank Dr. Lucie Johnson from Durham University for her helpful comments.

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MANUSCRIPT 5.4: FEEDING THE BODY AND SOUL: ISOTOPIC EVIDENCE OF DIET AND PATHOPHYSIOLOGICAL STRESS IN ADOLESCENTS WITH LEPROSY BURIED AT THE ST. MARY MAGDALEN LEPROSY HOSPITAL (WINCHESTER, HAMPSHIRE, ENGLAND)

Prepared for *International Journal of Osteoarchaeology, International Journal of Paleopathology, or American Journal of Physical Anthropology*

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ABSTRACT

Leprosy is an infectious disease commonly associated with the Medieval medical landscape. This study aims to examine carbon and nitrogen isotope data ($\delta^{13}C$ and $\delta^{15}N$) in young people with lepromatous leprosy from the St. Mary Magdalen leprosarium (leprosy hospital) (Winchester, Hampshire, England; 9^{th} – 12^{th} centuries AD). Incrementally sampled dentine collagen from permanent canine and molar teeth of local and non-local adolescents with incomplete dental development were chosen to capture full life-history profiles (c. 6 months old – death). Results indicate a broad trend in dietary consistency with decreasing isotopic variation in molar dentinal increments (crown to apical) suggesting a monastic 'convalescent diet', with increasing $\delta^{15}N$ variation near to the end of their lives. $\delta^{13}C$ and $\delta^{15}N$ dentine collagen profiles from canine and molar pairs also reveal periods of opposing covariance contemporaneous with the formation of linear enamel hypoplasias in early life, and highlight deviations in collagen values in co-forming tissues (i.e. rib and concurrently forming dentinal collagen) near the end of life. We attribute these variations to pathophysiological alterations in carbon and nitrogen metabolic pathways as a consequence of nutritional and pathophysiological stress. As there is little detail about what life in leprosaria was like or who populated these institutions, analyses connecting biological aspects of the disease and the 'hospital diet' are integral for understanding the wider biological and social ramifications of leprosy in the past.

KEY FINDINGS:

- $\delta^{13}C$ and $\delta^{15}N$ in dentine collagen from molar increments indicate that the diet consumed in the leprosarium was of a higher trophic level and contained more

marine resources than previously published data for Early Medieval diet for Hampshire.

- $\delta^{13}C$ shifts in non-local individuals reveal the possible timing of travel to the St. Mary Magdalen area
- Dietary variability decreases later in life and is consistent with a proscribed monastic diet, however fluctuations in $\delta^{15}N$ at end of life are possibly due to pathophysiological stress
- Pathophysiological and nutritional stress can influence $\delta^{13}C$ and $\delta^{15}N$ values in co-forming tissues and, as such, pathological status should always be considered in interpretations of $\delta^{13}C$ and $\delta^{15}N$ values

KEYWORDS: carbon isotopes, nitrogen isotopes, incremental dentine, leprosarium, Early Medieval

INTRODUCTION

Leprosy, also known as Hansen's Disease or Mycobacterial neurodermatosis (Butlin and Lockwood 2020), is an infectious disease of the peripheral nervous system caused by *Mycobacterium leprae* or *Mycobacterium lepromatosis*. The mode of transmission is not fully understood, but evidence suggests that inhalation of respiratory droplets is the most likely route of infection (World Health Organization 2019). Leprosy is classed as a 'spectral' disease (Jopling and McDougall 1988: 10-21) with infected individuals variously manifesting the infection between two polar clinical forms, depending on their host cell-mediated immunity. These range from loss of sensation and minor skin lesions (tuberculoid/paucibacillary) to major systemic complications (lepromatous/ multibacilliary), including skin ulceration, hair loss, difficulty in breathing, swallowing, and speaking, arrested development of the maxillary anterior teeth in children (leprogenic odontodysplasia), motor nerve damage and paralysis, blindness, disruption to multiple organ systems (e.g. respiratory, cardiovascular, endocrine, lymphatic, and renal pathways), and characteristic destruction of bones of the skull and extremities (Danielsen 1970; Jopling and McDougal 1988: 22-55). It is estimated that more than 95% of people are naturally immune to leprosy (Scollard et al. 2006), and therefore understanding immunological susceptibilities are vital for leprosy prevention.

LEPROSY CARE AND DIET IN THE PAST

Many have assumed that the visible and potentially disfiguring changes associated with leprosy, particularly lepromatous leprosy, led to widespread stigma towards people with leprosy in the past, leading to negligent social and medical treatment (Brody 1974: 60-71; Richards 1977:48-61; Orme and Webster 1995:24-31; Covey 1998: 90-92; Covey 2001). However, more recent scholarship suggests that past perceptions of individuals with leprosy were complex, citing evidence that their presence sometimes served as a test of Christian compassion worthy of charity and dedicated care (Touati 2000; Rawcliffe 2006:6-7, 354-357; Roberts 2013). Between the 11th to 14th centuries AD, over 300 documented leprosaria (leprosy hospitals) were established in England (Roberts 1986; Rawcliffe 2006: 418-421; Huggon 2018), leading to the inference that leprosy was a wide-scale public health issue that necessitated the foundation of these specialised institutions. Little historical evidence exists regarding what life was really like inside leprosaria, including what kind of care, if any, was provided. Rawcliffe's (2006: 311-314) comprehensive work on leprosy in Medieval England presents conflicting historical accounts of diet in these institutions. These include documentary references to familial and communal support that helped to provide care in local leprosaria by donating high-quality foods (e.g. quality meats and bread) in the 12th – 14th centuries AD, as well as contrary references documenting rotting fish as a main dietary donation in areas of England and Scotland from the 14th century (Rawcliffe 2006: 79-80). Similarly, Richards (1977:28-30) and Miller and Nesbitt (2014:121-138) mention inconsistent reports of dietary provision and care within other leprosaria in both England and Scandinavia in their reviews of social perceptions of people with leprosy in the Later Medieval and early modern periods (13th – 19th centuries AD).

Eating a well-balanced diet is an essential component of care giving and helps to ensure effective metabolic regulation and overall good health (Ulijaszek 1990; Ulijaszek 1996; Ulijaszek 2018; Wells 2016: 43-63). Bioarchaeological evidence provides a direct means of understanding whether a person's nutritional needs were met and sustained within a leprosarium. A high resolution incremental dentine analysis approach allows 'dietary biographies' to be explored and can offer a more nuanced lens through which diet related care in an Early Medieval context can be examined. This study aims to examine carbon and nitrogen isotope data ($\delta^{13}C$ and $\delta^{15}N$) in a with lepromatous leprosy from the

St. Mary Magdalen leprosarium (Winchester, Hampshire, England; 9th – 12th centuries AD) to better comprehend the 'leprosarium diet', and the potential impacts of disease on lifelong carbon and nitrogen metabolism. This is achieved through palaeopathological study and carbon and nitrogen isotopic analyses of incremental dentine.

Incremental Dentine

Assessing $\delta^{13}C$ and $\delta^{15}N$ values of incrementally forming dentinal collagen show the potential to reveal physiological alterations in people, including fluctuations in diet or metabolic perturbations caused by nutritional and pathological stress (Beaumont et al. 2013b; Beaumont et al. 2015; Beaumont and Montgomery 2015; Garland et al. 2018; Craig-Atkins et al. 2018; Crowder et al. 2019; Nicholls 2020; Walter et al. 2020; Craig-Atkins et al. 2020). To date, isotopic investigations of diet as a proxy for care and treatment within leprosaria have largely been confined to bulk bone collagen samples (Roffey et al. 2017; Brozou et al. 2019), which do not offer the same temporal resolution in which to contextualise actual lived experience (Beaumont et al. 2018; Beaumont 2020). This study therefore contributes a life history approach to further nuance these particulars and provide a more holistic view of the biological and social impacts of leprosy in the past.

The St. Mary Magdalen Leprosarium

The St. Mary Magdalen leprosarium, first documented around 1148 AD, lies approximately one mile east of the city of Winchester (Hampshire, England) (Roffey and Marter 2012; Figure 5.4.1). Ongoing excavations by the University of Winchester have revealed the presence of several chronologically distinct phases and cemeteries. The North Cemetery is found in association with early timber structures and a small masonry chapel that were originally estimated to date from the late 11th century AD. However, radiocarbon dates from the North Cemetery burials have extended the potential range of use from the late 9th to mid-12th centuries AD (Roffey and Marter 2012; Roffey and Tucker 2012; Schuenemann et al. 2013; Table 5.4.1). Overlying the earlier structures and the North Cemetery are larger masonry walls and a substantial chapel associated with the South Cemetery, suggesting a possible re-foundation of the leprosarium in the 12th century AD (Roffey and Marter 2012). Archaeological and historical evidence suggests the

site was subsequently reconfigured sometime in the 14th century AD, where it ceased to function solely as a leprosarium (Roffey 2012; Roffey and Marter 2012). Archaeological evidence also highlights a clear dissimilarity between the North and South Cemeteries with regard to burial treatment.

TABLE 5.4.1 - Calibrated radiocarbon dates and SNP types for individuals buried at the St. Mary Magdalen leprosy hospital site.

INDIVIDUAL	CEMETERY	CAL. ^{14}C DATE (95% CI)	SNP TYPE	REFERENCE
SK. 8	North	AD 1010-1160	2F	Roffey and Tucker 2012
SK. 9	North	AD 890-1040	N/A	Roffey 2012; Roffey and Marter 2012
SK. 14	North	AD 995-1033	2F	Schuenemann et al. 2013
SK. 27	North	AD 1020-1162	2F	Roffey et al. 2017
SK. 2	South	AD 1268-1283	3I	Schuenemann et al. 2013
SK. 5	Chapel	AD 1290-1410	N/A	Roffey and Tucker 2012

In the North Cemetery, individuals were buried in discrete, anthropomorphic graves normally reserved for high-status ecclesiastics with varying levels of personalized grave goods (*ibid*), whilst in the post-12th century AD South Cemetery (Late Medieval), people were buried more haphazardly in multiple, truncated internments (Roffey and Tucker 2012). A notable feature of the North Cemetery is the high number of skeletons with lesions diagnostic of lepromatous leprosy (n=38/44) and, of those, the high number of adolescents (c. 10-25 years old) with advanced signs of the disease (n=20). Previous research on individuals buried in the North Cemetery reveals some evidence of palliative care (e.g. potentially surgical foot amputation; feeding vessels) (Roffey and Tucker 2012), the presence of different genotypes of leprosy through the identification of single nucleotide polymorphisms (SNPs; 2F, 3I) representing broad geographical contact with the site (Taylor et al. 2013; Schuenemann et al. 2013; Schuenemann et al. 2018), as well as evidence for local and non-local origins of its interred who potentially and deliberately travelled to the leprosarium to seek care and treatment (Roffey et al. 2017; Filipek et al. 2021; Filipek et al. *under review*). This recent contextual, palaeopathological, and biomolecular data provide suggestions of institutional care to accommodate a wide range of individuals in the Early Medieval period. This is incommensurate with

stereotypical perceptions of leprosaria and social attitudes towards leprosy in the past (i.e. stigma), and warrants further investigation.

Figure 5.4.1 a) Location of Winchester, England, and b) aerial view of St. Mary Magdalen excavations.

Sampling and Methods

Skeletal analyses and tooth selection

This research strategically focusses on adolescents, defined as c. 10-25 years at death and in accordance with modern clinical (World Health Organization 1993; Patton et al. 2016; Sawyer et al. 2018) and Medieval social definitions (Gilchrist 2012: 34; Cochelin 2013; Lewis 2016). Adolescents were intentionally targeted for study due to: the proportionately high representation of this age cohort at the St. Mary Magdalen leprosarium who had advanced lesions diagnostic of lepromatous leprosy (>50%); their unique Medieval social status and general underrepresentation in bioarchaeological studies (see Gilchrist 2012: 34-37; Cochelin 2013; Lewis 2016; Lewis et al. 2016); and the potential for their dental tissues to provide near complete life course information (<1 year to death) (Moorrees et al. 1963; AlQahtani et al. 2010).

Permissions with ethical justifications to analyse the skeletons, and to take two tooth samples for subsequent biomolecular analyses were sought and approved both from the

curators of the human skeletal remains and the in-house ethics committee at the Department of Archaeology at Durham University. The Codes of Ethics (2010) and the Codes of Practice (2010) from the British Association of Biological Anthropologists and Osteoarchaeologists (BABAO) (https://www.babao.org.uk/publications/ethics-and-standards) were strictly adhered to in the analysis and sampling from the individuals selected for study.

An essential component of sample selection was the presence of the lepromatous form of leprosy on the skeleton. Identifying disease presence in human skeletal remains can be confounded by overlap in skeletal responses from different diseases and unknown periods of chronicity (Ortner 1991: 7-11; Wood et al. 1992). Despite the likelihood that other individuals within the cemetery may have possessed a less severe/high resistant form of leprosy that did not elicit skeletal changes (e.g. the tuberculoid form), only individuals with pathognomonic signs of the disease were selected.

These pathognomonic signs of lepromatous leprosy included:

1.) Rhinomaxillary changes: widening and remodelling of the nasal aperture, resorption of the anterior nasal spine, abnormal porosity and/or new bone formation on the oral and nasal surfaces of the palatal bones, destruction of the inferior nasal conchae and vomer, abnormal porosity and resorption of the alveolar process with potential loss of the anterior maxillary incisors (Figure 5.4.2), and

2.) Acro-osteolysis and concentric atrophy of the hand and foot bones: destruction and remodelling of the phalanges, metacarpals, and metatarsals (Møller-Christensen and Andersen 1953; Møller-Christensen 1961; 1978; Andersen and Manchester 1987; 1988; 1992; Andersen et al. 1992; Andersen et al. 1994).

Pathological lesions consistent with, but not pathognomonic of, lepromatous leprosy including resorptive grooves on the palmar surfaces of the hand phalanges (also known as "volar grooving" - Andersen and Manchester 1987), tarsal fusion and dorsal exostoses; and subperiosteal new bone formation on the distal shafts of the tibiae and fibulae were recorded, but not included in the criteria for selection due to their varied aetiology (Ortner 2003: 265-269; Ortner 2008; Klaus 2014; Filipek and Roberts 2018). Other concomitant

pathological conditions were recorded in the individuals using selected reference texts (e.g. Resnick and Niwayama 1995; Ortner 2003; Lewis 2017; Roberts and Manchester 2005; Aufderheide and Rodriguez-Martin 1998) and were taken into consideration when interpreting the isotopic data.

For sex estimation, individuals were assessed using Shapland and Lewis' (2013) methods for evaluating pubertal stages using osteological techniques (e.g. canine development and iliac crest ossification and fusion). Only individuals who were categorised as having reached or surpassed the 'peak height velocity' (PHV) stage of their pubertal growth spurt were re-assessed for sex. Other osteological techniques for estimating the pubertal stage of an individual were not used due to the potential effects of leprosy on or around the areas under evaluation (e.g. the hamate bone, the radius, the phalanges, etc.). For individuals who achieved or surpassed the PHV stage, standard methods for evaluating the sexual dimorphic characteristics of the pelvis and skull were used to assign sex (Phenice 1969; Acsádi and Nemeskéri 1970). Sex determination for ambiguously sexed individuals has been previously achieved through ancient DNA analyses (Taylor et al. 2013) or through amelogenin peptide extraction (Filipek et al. *in prep*).

A permanent canine (C), and a permanent second (M2) or third (M3) molar, depending on the age of the individual, were selected from 5 local and 5 non-local individuals determined through parallel strontium and oxygen isotope analyses (Filipek et al. *in prep*) to capture data over most of the full life course. These teeth respectively develop from 7.5 ± 1.5 months upwards to 16.5 years ± 6 months, 2.5 ± 6 months upwards to 17.5 years ± 6 months, and 7.5 years ± 6 months to 23.5 years ± 6 months, respectively (Moorrees et al. 1963; AlQahtani et al. 2010). Teeth were preferentially selected from the maxilla or mandible based upon which tooth sample would cause the least amount of damage to the bone (e.g. a loose tooth was selected over a tooth still in occlusion within the alveolar bone), rather than position in the mouth. Individuals were re-assessed for age at death based on their dental development using methods set out by Moorrees et al. (1963) and AlQahtani et al. (2010) to target individuals under the age of 23.5 years. Linear enamel hypoplasias were measured from the CEJ and evaluated using both the methods of Primeau et al. (2015). Prior to preparation for isotope analysis, the teeth were photographed by the

Department of Archaeology at Durham University and all remaining tooth sample material and archival photos are scheduled to return to the curating institution.

A full list of the individuals selected for this study is provided in Table 5.4.2.

TABLE 5.4.2 - Individuals from the St. Mary Magdalen leprosarium selected for carbon and nitrogen stable isotope analyses of incremental dentine. Geographic origins previously estimated by Filipek et al. (*in prep*) and Roffey et al. (2017).

INDIVIDUAL	SEX	AGE	TEETH SAMPLED AND DEVELOPMENTAL STAGE	CONCOMITTANT PATHOLOGICAL CONDITIONS
SK. 8 (LOCAL)	Male*	8.5-9.5	Maxillary left canine (RC ½) Maxillary left M2 (RC ¼)	Linear enamel hypoplasias (2-2.9, 5-5.9, 6-6.9 years); leprogenic odontodysplasia; cribra orbitalia; delayed growth
SK. 9 (LOCAL)	Male	22.5-23.5	Maxillary right canine (AC) Maxillary right M2 (AC)	Linear enamel hypoplasias (2-4.9, 10-10.9 years); asymmetrical dental calculus
SK. 15 (LOCAL)	Male	20.5-22.5	Mandibular left canine (AC) Mandibular left M3 (A ½)	Linear enamel hypoplasias (2-5.9 years); lytic spinal lesions (TB?); cribra orbitalia
SK. 18 (NON-LOCAL)	Male	18.5-19.5	Mandibular right canine (A ½) Mandibular right M3 (R ½)	Linear enamel hypoplasias (2-3.9, 10-10.9 years); cribra orbitalia; possible scurvy; extremely thin cortical bone; delayed growth (<14 years); asymmetrical dental calculus
SK. 27 (NON-LOCAL)	Male	22.5-23.5	Mandibular right canine (AC) Mandibular right M3 (A ½)	Linear enamel hypoplasias (3-4.9 years); asymmetrical dental calculus
SK. 28 (LOCAL)	Male**	12.5-13.5	Maxillary left canine (AC) Maxillary left M2 (R ¾)	Linear enamel hypoplasias (2-4.9 years); leprogenic odontodysplasia; cribra orbitalia
SK. 45 (LOCAL)	Female**	15.5-16.5	Mandibular left canine (AC) Mandibular left M2 (A ½)	Linear enamel hypoplasias (2-4.9, 6-6.9 years); lytic spinal and extraspinal lesions (TB?); visceral rib lesions
SK. 52 (NON-LOCAL)	Female**	12.5-13.5	Mandibular left canine (AC) Mandibular left M2 (RC)	Linear enamel hypoplasias (4-4.9 years); leprogenic odontodysplasia; cribra orbitalia; delayed growth (<11 years); asymmetrical dental calculus
SK. 54 (LOCAL)	Male**	14.5-15.5	Mandibular right canine (AC) Mandibular right M2 (RC)	Linear enamel hypoplasias (1-2.9, 4-4.9, 6-6.9); delayed growth (<11 years); asymmetrical dental calculus
SK. 56 (NON-LOCAL)	Male	16.5-17.5	Maxillary right canine (AC) Maxillary right M2 (A ½)	Linear enamel hypoplasias (2-4.9); cribra orbitalia; leprogenic odontodysplasia; visceral rib lesion (left T10)

*Sex determined by aDNA analysis (Taylor et al. 2013).

** Sex determined by amelogenin peptide analysis (Filipek et al. *in prep*).

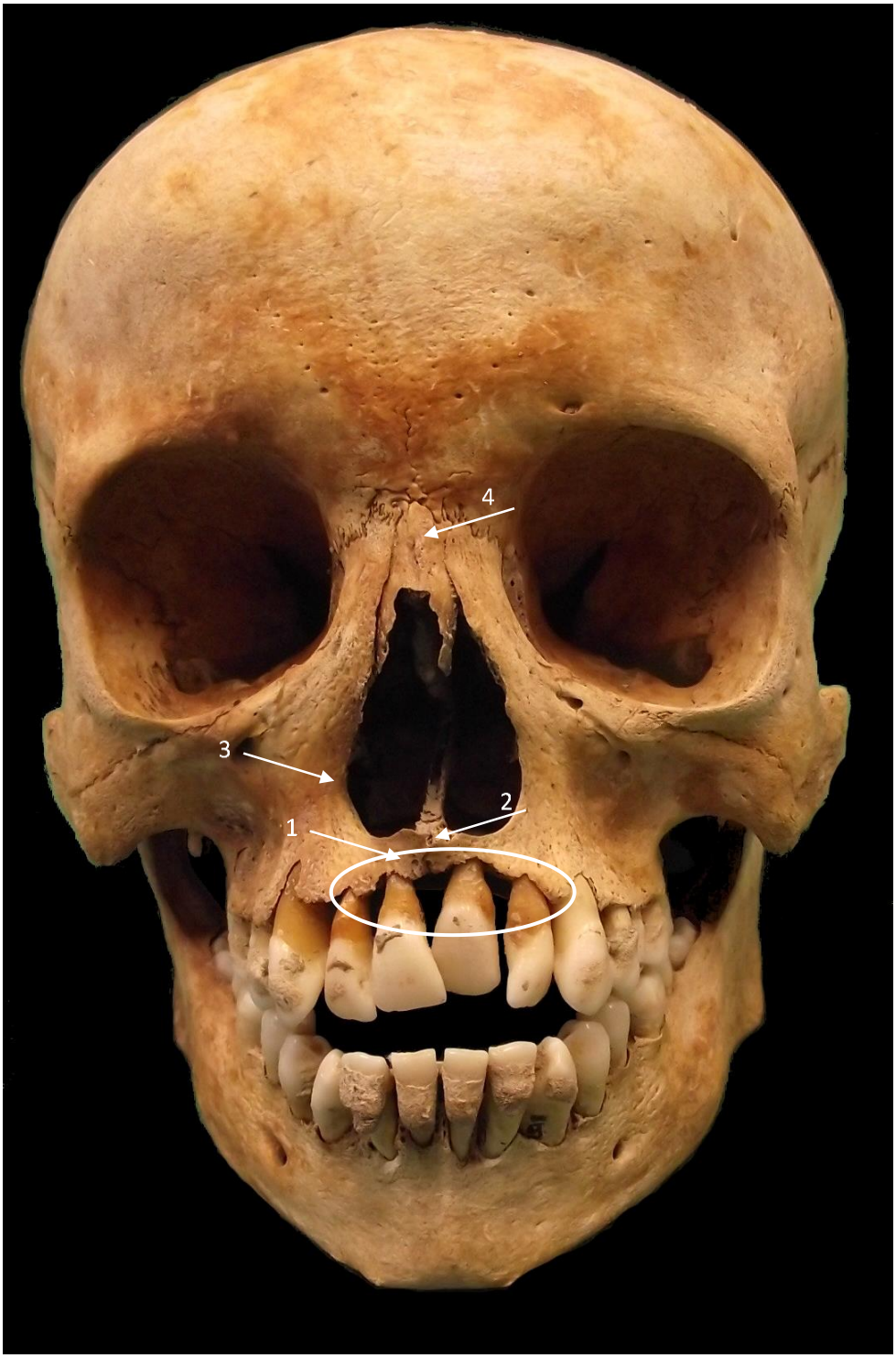


Figure 5.4.2 Skull of 16.5-17.5 year old male (Sk. 56) displaying facial signs of lepromatous leprosy. These include 1) resorption of the maxillary alveolar process, 2) resorption of the anterior nasal spine, 3) widening and remodelling of the nasal aperture, and 4) widening and flattening of the nasal bones. Sk. 56 also displays evidence for leprogenic odontodysplasia, with constriction and arrested development of the maxillary incisors (encircled).

Canine and molar teeth were prepared for collagen extraction from incremental dentine using Method 2, as outlined in Beaumont et al. (2013a) and briefly described here. Sample preparation and collagen extraction was undertaken at the Archaeological Isotope and Peptide Research Laboratory (AIPRL) at Durham University. Tooth samples were first abraded and bisected mesiodistally using a diamond-head dental saw, followed by demineralisation in 0.5M HCl at 4 °C using the modified Longin method without ultrafiltration (O'Connell and Hedges 1999; Longin 1971) over a time period of approximately eight weeks. Demineralised dentine was transversely sectioned into 1 mm increments using a sterile scalpel from dentine horn to root apex (depending on the developmental stage of the tooth). Increments of dentine were placed into Eppendorf microtubes with a pH 3 solution and denatured on a heated block at 70 °C for 24 hr, followed by centrifugation to separate any remaining debris. Afterwards, samples were sealed and frozen before being lyophilised to remove remaining water. Once lyophilised, extracted collagen was weighed to calculate the total collagen yield, and a minimum of 0.5 mg of collagen was placed within 6x4mm tin capsules and transferred to the Stable Isotope Laboratory at the School of Archaeological and Forensic Sciences, University of Bradford for mass spectrometry.

Samples were measured in duplicate where possible by combustion in a Thermo Flash EA 1112 and introduction of separated N_2 and CO_2 to a Delta plus XL via a Conflo III interface. The samples were compared to reference gasses (N_2 and CO_2) prior to combustion, and then calibrated to procedural blanks and international standards (IAEA 600, CH3, N1, and N2) and in-house analytical reference materials (fish gelatine and bovine liver serum). Results were calculated to the Vienna Pee Dee Belemnite (VPDB) for carbon and Ambient Inhalable Reservoir (AIR) for nitrogen, and reported using the delta notation (δ) in parts per mil (‰). Blank tin capsules and both in-house laboratory and international standards gave an analytical reproducibility of ±0.25‰ (1σ) for the in-house standards, and ±0.2‰ (1σ) or better for the international standards used. Recommended collagen quality parameters (DeNiro 1985; Ambrose 1990; van Klinken 1999) were used

to exclude any increment samples that did not meet the accepted carbon and nitrogen percentages, C:N ratios, or collagen yield (Table 5.4.3).

Incremental samples were assigned an average age following the methods in Beaumont et al. (2013a) and Beaumont and Montgomery (2015). The minimum age for crown initiation (CI) was subtracted from the maximum age of tooth completion depending on its developmental stage (Moorrees et al. 1963; AlQahtani et al. 2010) and divided by the number of increments obtained. It is crucial to note that whilst dentine is deposited incrementally, it is not deposited in discrete layers (Dean and Scandrett 1995; Beaumont et al. 2013a), and therefore transverse sectioning of dentine represents cumulative averages over the developmental period of the tooth and not an exact age (Beaumont and Montgomery 2015; Tsutaya 2020).

RESULTS

Carbon and nitrogen stable isotope ratios are provided in Table 5.4.3, while individual scatterplots are presented in Figure 5.4.3. Figure 5.4.4 combines molar incremental data and comparative data from bone collagen from surrounding archaeological sites in the Winchester region. Some broad trends in diet and patterns in dentine collagen profiles are presented here.

TABLE 5.4.3 - Data and quality parameters for this study.

Individual	**Tooth**	**Dentine Section Number**	**$\delta^{15}N_{AIR}$‰**	**Amt % N**	**$\delta^{13}C_{VPDB}$‰**	**Amt % C**	**C:N**
SK8	L Max C	1	nil	nil	nil	nil	nil
		2	7.8	10.8	-18.6	32.6	3.5
		3	8.5	12.8	-19.4	36.1	3.3
		4	8.7	18.7	-19.5	54.1	3.4
		5	9.4	15.2	-19.5	42.0	3.2
		6	9.8	12.8	-19.4	37.0	3.4
		7	9.9	11.5	-19.2	35.3	3.6

		8	10.7	12.4	-18.9	37.4	3.5
		9	10.4	11.9	-18.5	32.7	3.2
		10	10.6	12.1	-18.8	35.9	3.5
		11	10.2	7444.3	-18.7	21066.6	3.3
	L Max M2	1	9.6	13.8	-19.8	39.2	3.3
		2	9.5	12.1	-19.7	34.0	3.3
		3	9.3	15.0	-19.6	40.0	3.1
		4	9.4	14.8	-19.4	39.3	3.1
		5	9.6	14.8	-19.3	39.5	3.1
		6	10.0	14.9	-19.1	39.5	3.1
		7	10.6	14.9	-19.1	39.5	3.1
		8	10.8	15.6	-18.9	41.9	3.1
SK9	R Max C	1	15.6	18.2	-19.1	48.9	3.1
		2	13.7	18.1	-19.6	49.4	3.2
		3	12.5	18.0	-19.8	49.0	3.2
		4	12.5	18.3	-19.9	49.6	3.2
		5	14.0	18.3	-20.0	49.5	3.2
		6	14.6	18.2	-19.7	49.5	3.2
		7	13.3	18.2	-19.6	49.9	3.2
		8	12.3	14.3	-19.7	39.6	3.2
		9	11.9	14.6	-19.7	40.4	3.2
		10	12.1	14.7	-19.6	40.2	3.2
		11	11.9	14.6	-19.6	40.1	3.2
		12	11.5	14.6	-19.7	40.3	3.2
		13	10.9	14.8	-19.8	40.8	3.2
		14	11.0	14.6	-19.7	40.0	3.2

		15	11.1	14.7	-19.6	40.4	3.2
		16	11.5	14.6	-19.5	40.2	3.2
		17	11.6	14.7	-19.5	40.6	3.2
		18	11.7	14.4	-19.4	40.1	3.2
		19	12.1	14.6	-19.2	40.1	3.2
	R Max M2	1	12.5	14.8	-20.0	39.5	3.1
		2	12.2	11.1	-19.9	29.5	3.1
		3	13.5	15.3	-19.9	40.6	3.1
		4	14.3	15.8	-19.7	42.2	3.1
		5	13.4	15.2	-19.7	40.5	3.1
		6	12.4	15.3	-19.8	40.8	3.1
		7	12.2	14.5	-19.8	39.3	3.2
		8	12.2	15.1	-19.8	40.0	3.1
		9	12.1	15.2	-19.7	40.0	3.1
		10	11.7	14.8	-19.8	39.0	3.1
		11	11.5	15.2	-19.8	40.1	3.1
		12	11.5	14.6	-19.7	38.7	3.1
		13	11.6	11.3	-19.8	30.4	3.2
		14	12.2	14.7	-19.6	40.0	3.2
		15	12.5	14.2	-19.2	38.8	3.2
SK15	Man L C	1	11.6	13.5	-20.3	37.1	3.2
		2	10.2	13.5	-20.6	36.9	3.2
		3	10.2	13.6	-20.2	38.2	3.3
		4	10.2	13.3	-20.0	36.5	3.2
		5	10.5	13.6	-20.3	37.5	3.2
		6	10.3	13.5	-19.8	37.3	3.2

		7	10.1	13.8	-19.8	37.9	3.2
		8	10.3	13.1	-19.3	36.1	3.2
		9	10.6	13.6	-18.8	37.5	3.2
		10	11.0	13.5	-18.6	37.1	3.2
		11	10.8	13.5	-18.7	37.3	3.2
		12	10.4	13.2	-18.7	36.4	3.2
		13	10.5	13.6	-18.7	37.4	3.2
		14	10.2	13.5	-18.5	37.2	3.2
		15	10.1	13.2	-18.4	36.5	3.2
		16	9.8	11.4	-18.5	32.5	3.3
		17	10.0	11.4	-18.8	34.1	3.5
		18	10.5	12.3	-18.7	34.3	3.2
		19	7.6	9.4	-19.3	30.5	3.8
		20	10.8	10.8	-18.8	33.8	3.6
		21	10.3	13.7	-18.6	41.4	3.5
	L Man M3	1	11.3	15.0	-18.9	39.7	3.1
		2	11.7	15.0	-18.8	39.3	3.1
		3	11.6	14.9	-18.7	39.0	3.1
		4	11.6	14.7	-18.8	38.5	3.1
		5	11.8	15.0	-18.7	39.5	3.1
		6	11.6	15.1	-18.8	39.8	3.1
		7	11.8	13.3	-18.8	34.9	3.1
		8	12.0	13.6	-19.0	38.0	3.3
		9	11.6	14.2	-19.3	40.9	3.4
		10	11.7	14.3	-19.4	41.3	3.4
SK18	Man R C	1	14.3	12.2	-19.5	35.3	3.4

		2	14.4	12.7	-19.6	36.3	3.3
		3	13.6	12.8	-19.6	35.6	3.2
		4	12.3	12.9	-19.7	35.9	3.3
		5	10.7	13.0	-19.8	36.3	3.3
		6	10.7	12.9	-19.9	36.2	3.3
		7	10.2	12.8	-20.0	35.7	3.3
		8	10.3	13.1	-19.9	36.9	3.3
		9	10.6	13.1	-19.7	36.6	3.3
		10	10.2	13.0	-19.6	36.0	3.2
		11	9.6	13.0	-19.8	36.2	3.3
		12	9.6	13.1	-19.9	36.6	3.3
		13	9.4	13.0	-19.9	36.4	3.3
		14	9.7	13.0	-19.8	36.4	3.3
		15	9.9	13.0	-19.8	36.4	3.3
		16	10.0	13.0	-19.7	36.4	3.3
		17	10.5	12.9	-19.7	36.2	3.3
		18	10.7	12.8	-19.7	36.1	3.3
		19	10.5	12.6	-19.8	36.3	3.4
		20	6.4	8.1	-19.7	30.6	4.4
		21	9.4	11.4	-19.8	34.9	3.6
	R Man M3	1	10.9	11.7	-20.2	31.6	3.2
		2	10.9	14.3	-20.1	38.8	3.2
		3	11.4	14.7	-19.9	39.5	3.1
		4	11.1	14.4	-20.0	38.4	3.1
		5	10.4	13.4	-19.4	36.0	3.1
		6	10.5	10.6	-19.2	29.1	3.2
		7	11.0	14.2	-18.8	38.6	3.2

		8	11.3	11.0	-19.1	31.7	3.4
		9	11.5	12.9	-19.2	39.6	3.6
		10	12.2	12.4	-19.4	39.4	3.7
SK27	Man R C	1	10.6	14.0	-20.4	36.6	3.0
		2	12.9	14.5	-19.8	39.8	3.2
		3	12.2	14.5	-19.9	39.6	3.2
		4	10.9	14.5	-19.8	39.6	3.2
		5	10.7	14.5	-19.7	39.9	3.2
		6	10.3	14.6	-19.9	40.0	3.2
		7	9.7	14.5	-19.9	40.2	3.2
		8	9.2	14.7	-19.5	40.3	3.2
		9	9.5	14.6	-19.3	40.1	3.2
		10	9.4	14.5	-19.4	40.2	3.2
		11	9.8	14.6	-19.4	40.2	3.2
		12	9.8	14.5	-19.3	40.1	3.2
		13	10.1	14.6	-19.1	40.2	3.2
		14	9.9	14.4	-19.3	40.0	3.2
		15	10.1	14.5	-19.3	40.2	3.2
		16	10.2	14.5	-19.4	40.1	3.2
		17	10.4	14.3	-19.6	40.2	3.3
		18	10.5	14.1	-19.7	39.4	3.3
	R Man M3	1	10.2	11.8	-19.6	32.3	3.2
		2	10.4	14.6	-19.5	38.8	3.1
		3	10.6	14.1	-19.3	37.6	3.1
		4	10.4	15.0	-19.4	39.4	3.1
		5	10.3	13.9	-19.5	36.7	3.1

		6	10.3	14.6	-19.6	38.8	3.1
		7	10.6	13.2	-19.4	34.9	3.1
		8	11.0	14.6	-19.4	38.8	3.1
		9	11.4	14.5	-19.2	38.6	3.1
		10	11.2	9.3	-19.2	24.4	3.0
		11	11.6	14.4	-19.0	38.3	3.1
		12	11.5	12.3	-19.4	34.7	3.3
		13	10.9	8.3	-19.2	22.3	3.1
		14	11.7	14.3	-19.1	38.1	3.1
		15	11.8	7.5	-19.3	20.9	3.3
SK28	Max L C	1	10.6	16.3	-20.0	44.0	3.2
		2	11.4	17.3	-20.3	48.1	3.2
		3	11.6	17.7	-20.1	48.1	3.2
		4	11.7	18.1	-20.1	49.0	3.2
		5	10.5	18.1	-20.3	48.9	3.1
		6	10.3	17.9	-19.9	48.6	3.2
		7	10.6	17.6	-19.7	47.8	3.2
		8	10.2	17.8	-19.4	48.8	3.2
		9	9.9	18.0	-19.2	48.8	3.2
		10	9.5	17.8	-19.5	48.2	3.2
		11	9.4	16.2	-20.4	54.6	3.9
		12	9.7	17.8	-19.7	48.6	3.2
		13	9.7	17.8	-19.5	48.7	3.2
		14	10.0	17.8	-19.3	48.7	3.2
		15	10.5	17.5	-18.8	47.6	3.2
		16	10.4	16.7	-19.2	47.8	3.3
		17	10.9	13.0	-22.2	31.3	2.8

	L Max M2	1	10.7	13.4	-20.3	35.7	3.1
		2	10.3	14.6	-20.0	38.5	3.1
		3	10.0	14.6	-19.8	38.5	3.1
		4	9.9	14.4	-19.5	38.7	3.1
		5	9.6	14.7	-19.2	38.5	3.1
		6	9.4	14.5	-19.4	38.2	3.1
		7	9.5	15.7	-20.4	46.1	3.4
		8	9.9	15.1	-19.8	40.1	3.1
		9	9.9	14.6	-19.6	39.7	3.2
		10	10.3	14.5	-19.2	38.4	3.1
		11	10.7	14.9	-18.8	39.8	3.1
		12	10.6	14.9	-18.9	39.8	3.1
		13	11.0	14.1	-18.9	38.6	3.2
		14	11.1	14.3	-19.1	41.3	3.4
SK45	Man L C	1	14.5	17.5	-18.8	47.9	3.2
		2	13.6	17.5	-19.0	48.2	3.2
		3	13.5	18.0	-19.0	49.1	3.2
		4	12.5	17.9	-19.1	49.0	3.2
		5	11.2	18.2	-19.3	49.2	3.2
		6	10.7	17.8	-19.4	48.2	3.2
		7	10.0	18.2	-19.5	49.5	3.2
		8	9.6	14.9	-19.5	40.6	3.2
		9	9.4	18.2	-19.5	49.3	3.2
		10	9.3	18.1	-19.3	49.2	3.2
		11	9.7	18.0	-19.1	48.7	3.2
		12	9.6	18.3	-19.1	49.8	3.2

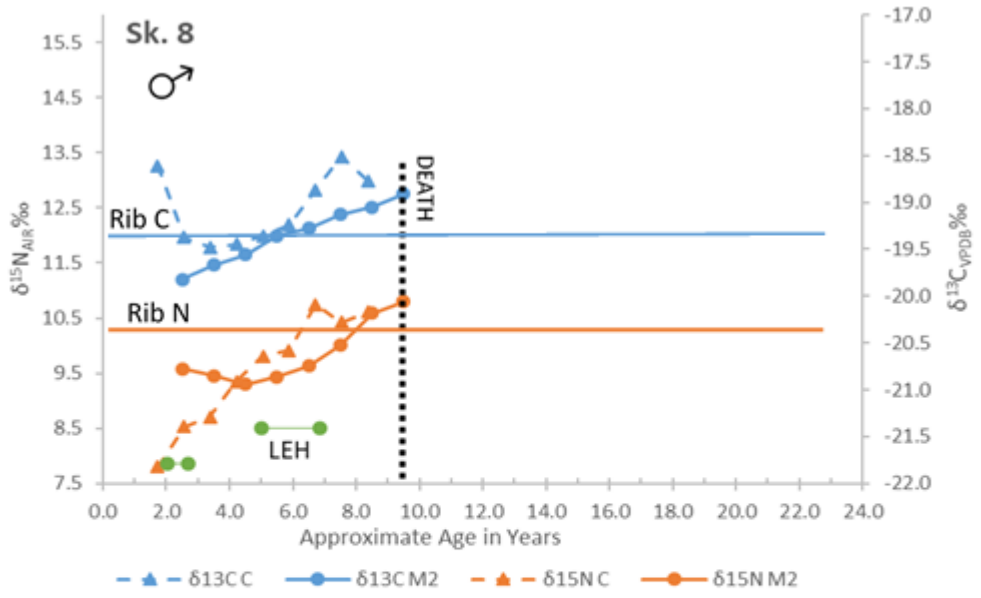
		13	9.8	18.4	-19.2	49.9	3.2
		14	9.9	18.0	-19.3	48.8	3.2
		15	10.2	17.8	-19.6	48.9	3.2
		16	10.3	17.5	-19.8	48.9	3.2
		17	10.1	16.4	-19.9	47.2	3.4
	L Man M2	1	10.1	13.9	-19.4	37.4	3.2
		2	9.5	14.4	-19.4	38.5	3.1
		3	9.3	6.1	-19.6	16.4	3.1
		4	9.5	13.2	-19.4	35.8	3.2
		5	10.0	13.9	-19.2	37.4	3.1
		6	10.1	13.5	-19.4	36.1	3.1
		7	10.4	14.1	-19.4	37.4	3.1
		8	10.6	14.4	-19.3	38.6	3.1
		9	10.9	14.4	-19.3	38.9	3.2
		10	10.7	11.6	-19.3	32.4	3.3
		11	11.0	12.6	-19.5	37.0	3.4
		12	10.6	11.3	-19.8	32.0	3.3
SK52	L Man C	1	13.6	14.5	-19.7	39.7	3.2
		2	13.6	14.5	-19.7	39.8	3.2
		3	11.7	30.4	-20.0	83.5	3.2
		4	10.8	14.5	-20.3	39.7	3.2
		5	10.7	14.5	-19.9	39.8	3.2
		6	10.5	14.2	-19.8	38.9	3.2
		7	10.3	14.5	-19.7	39.9	3.2
		8	10.6	14.8	-19.3	40.9	3.2
		9	10.6	13.6	-19.0	37.3	3.2

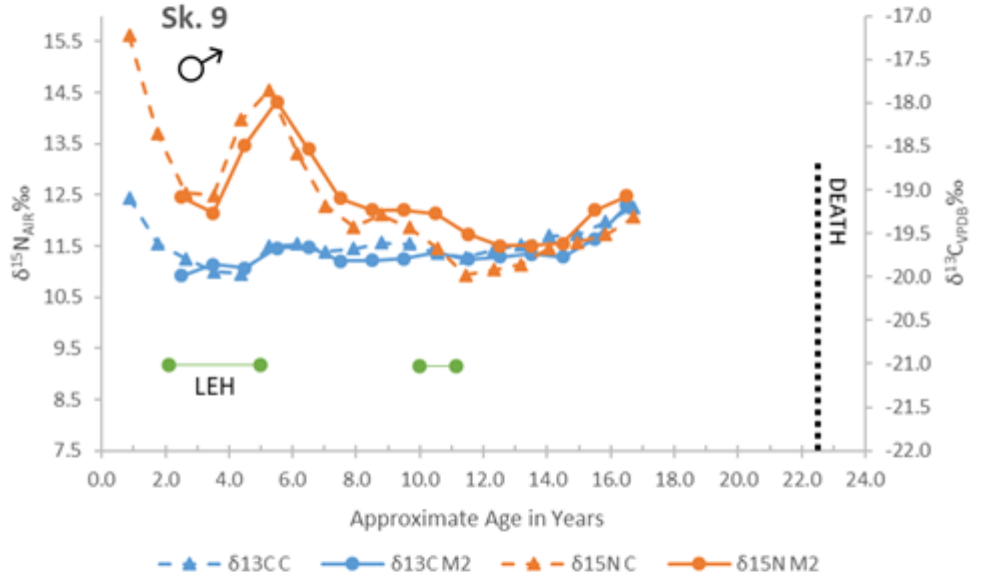
		10	10.4	14.3	-18.8	39.3	3.2
		11	10.2	14.4	-18.9	39.5	3.2
		12	10.1	14.7	-18.8	39.9	3.2
		13	10.0	14.2	-18.9	38.5	3.2
		14	10.5	14.6	-19.2	40.0	3.2
		15	9.8	13.8	-19.1	37.8	3.2
	L Man M2	1	10.9	14.5	-20.3	38.7	3.1
		2	10.4	14.6	-19.9	38.4	3.1
		3	10.3	14.8	-19.6	39.1	3.1
		4	10.6	14.5	-19.3	38.3	3.1
		5	10.7	14.4	-18.9	38.3	3.1
		6	10.6	14.7	-18.9	39.7	3.2
		7	10.7	14.7	-18.9	39.0	3.1
		8	10.9	15.2	-18.9	40.2	3.1
		9	11.3	14.7	-19.0	39.1	3.1
		10	11.6	14.7	-18.9	39.1	3.1
		11	11.5	14.5	-19.1	39.0	3.1
		12	11.1	15.0	-19.1	41.0	3.2
SK54	Man R C	1	17.7	8.7	-20.0	36.5	4.9
		2	10.8	14.6	-19.0	39.4	3.2
		3	10.0	14.6	-19.4	39.7	3.2
		4	9.6	14.6	-19.7	39.7	3.2
		5	9.7	14.5	-19.8	39.6	3.2
		6	10.0	14.5	-19.8	39.4	3.2
		7	9.7	13.8	-19.3	37.4	3.2
		8	10.1	14.6	-19.1	39.5	3.2

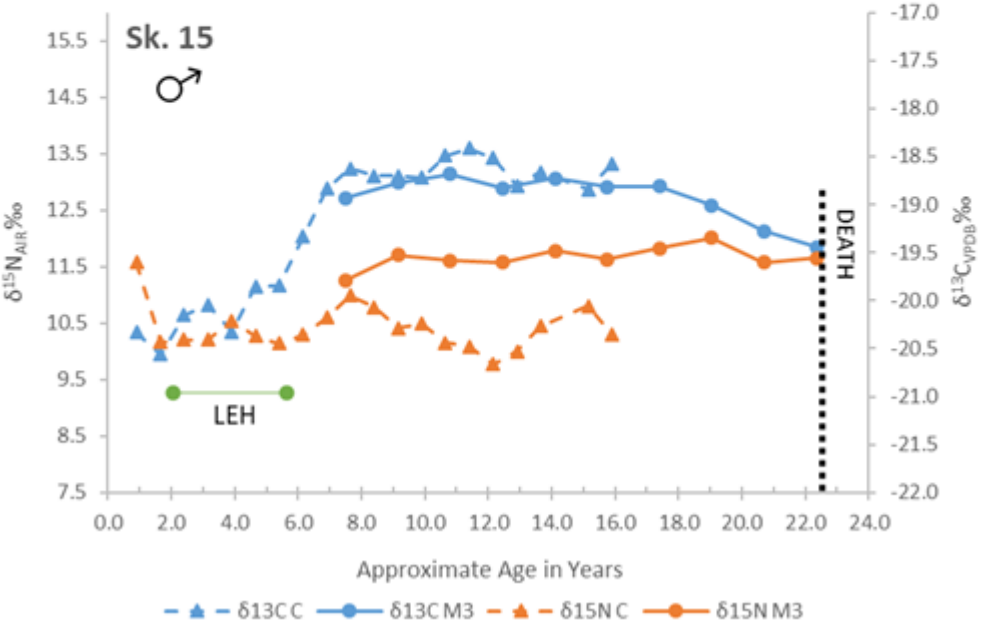
		9	10.1	14.2	-19.1	38.9	3.2
		10	10.1	14.6	-19.0	39.6	3.2
		11	10.3	12.0	-18.9	32.8	3.2
		12	10.2	17.4	-18.8	47.6	3.2
		13	9.9	13.0	-18.8	35.4	3.2
		14	10.3	15.0	-18.8	41.0	3.2
		15	9.4	54.4	-18.8	148.9	3.2
		16	9.9	15.1	-18.7	41.2	3.2
		17	9.9	15.6	-18.5	42.6	3.2
		18	9.7	15.7	-18.5	43.0	3.2
		19	9.5	15.2	-18.6	41.5	3.2
		20	9.9	14.4	-19.0	39.5	3.2
		21	9.0	5	-20.1	14	3.5
	R Man M2	1	10.0	14.3	-19.6	38.3	3.1
		2	10.0	14.5	-19.5	38.2	3.1
		3	10.0	14.7	-19.2	38.5	3.1
		4	10.2	14.6	-19.1	38.1	3.1
		5	10.1	14.1	-18.9	37.1	3.1
		6	10.3	14.9	-18.7	38.9	3.0
		7	10.3	13.5	-19.0	38.8	3.4
		8	10.3	13.5	-18.4	35.6	3.1
		9	10.1	13.8	-18.3	36.2	3.1
		10	10.5	7.0	-19.2	21.5	3.6
		11	10.4	13.1	-19.4	38.9	3.5
		12	10.6	13.7	-19.4	39.3	3.4
		13	11.0	13.6	-19.6	39.7	3.4

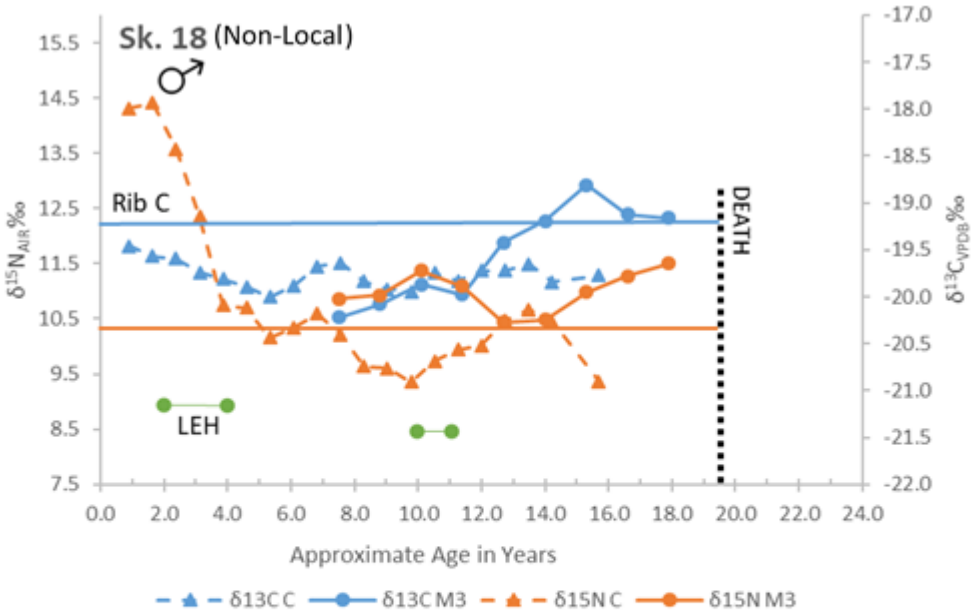
SK56	Max R C	1	13.0	6.1	-18.9	17.1	3.3
		2	12.7	14.0	-18.8	38.4	3.2
		3	12.0	15.5	-18.8	42.6	3.2
		4	11.8	16.0	-18.6	43.8	3.2
		5	11.2	12.0	-18.9	32.9	3.2
		6	10.9	9.3	-18.9	25.4	3.2
		7	10.3	16.9	-18.9	46.6	3.2
		8	10.4	15.7	-18.9	43.3	3.2
		9	10.7	17.8	-18.8	48.7	3.2
		10	10.7	17.0	-18.9	46.7	3.2
		11	10.6	14.7	-18.9	40.3	3.2
		12	10.9	14.7	-18.8	40.5	3.2
		13	10.5	13.8	-19.0	38.2	3.2
		14	10.6	13.2	-18.8	36.1	3.2
		15	10.6	14.9	-18.8	40.9	3.2
		16	10.7	13.9	-18.8	38.2	3.2
		17	11.0	9.6	-18.7	26.5	3.2
		18	11.1	13.1	-18.8	35.8	3.2
	R Max M2	1	11.5	14.4	-19.0	42.1	3.4
		2	11.7	14.9	-18.7	40.6	3.2
		3	10.8	14.5	-18.8	39.7	3.2
		4	10.5	14.7	-18.7	39.9	3.2
		5	10.8	15.0	-18.7	40.7	3.2
		6	10.9	14.5	-18.7	39.2	3.2
		7	10.8	14.8	-18.6	40.0	3.2
		8	11.0	13.7	-18.6	37.8	3.2
		9	11.2	14.6	-18.6	39.3	3.1

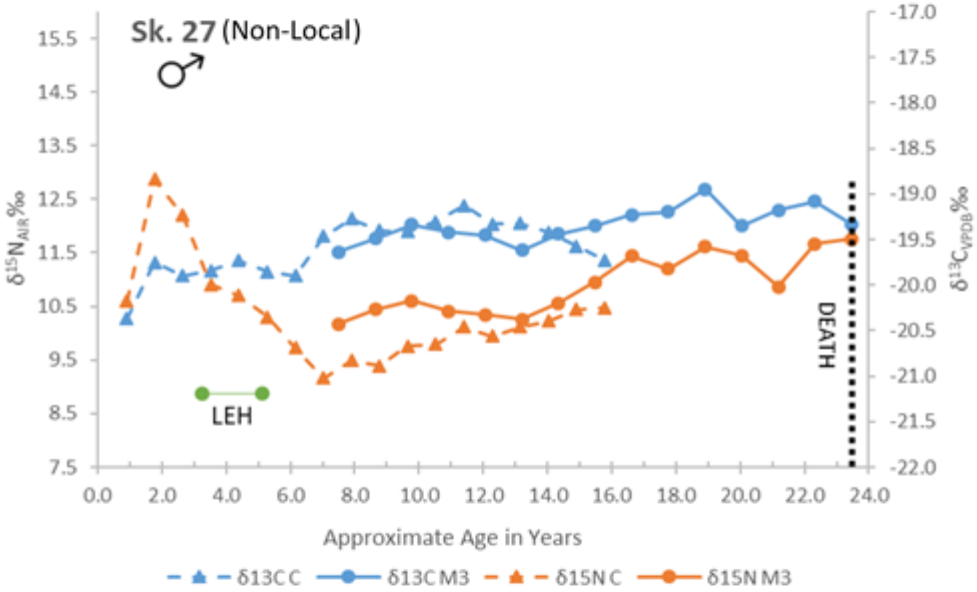
		10	11.5	14.8	-18.4	40.1	3.2
		11	11.6	14.6	-18.5	40.0	3.2
		12	11.3	14.3	-18.6	39.1	3.2
		13	11.0	15.1	-18.8	41.4	3.2
		14	10.9	13.7	-18.9	38.8	3.3
		15	10.7	12.9	-19.2	38.5	3.5

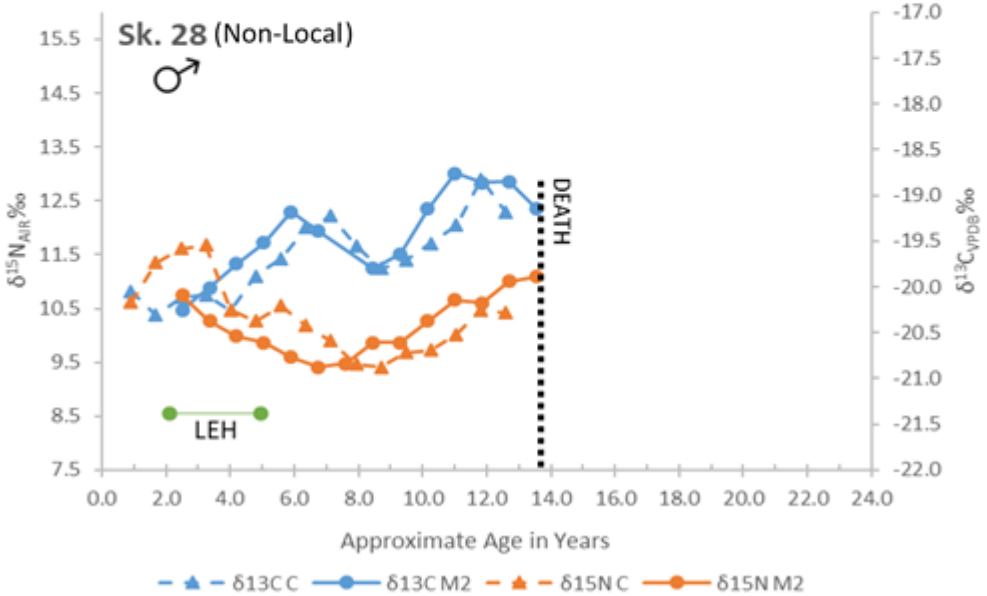

Sk. 8
♂
Rib C
Rib N
DEATH
LEH
δ15N AIR ‰
δ13C VPDB ‰
Approximate Age in Years
δ13C C
δ13C M2
δ15N C
δ15N M2

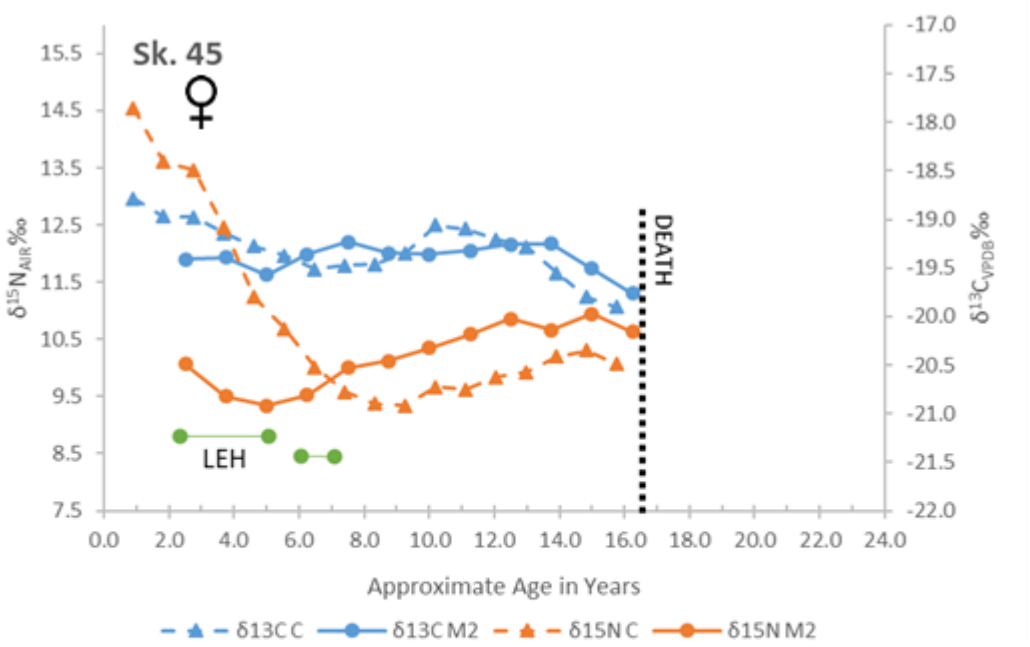

Sk. 9
♂
DEATH
LEH
δ15N AIR ‰
δ13C VPDB ‰
Approximate Age in Years
δ13C C
δ13C M2
δ15N C
δ15N M2

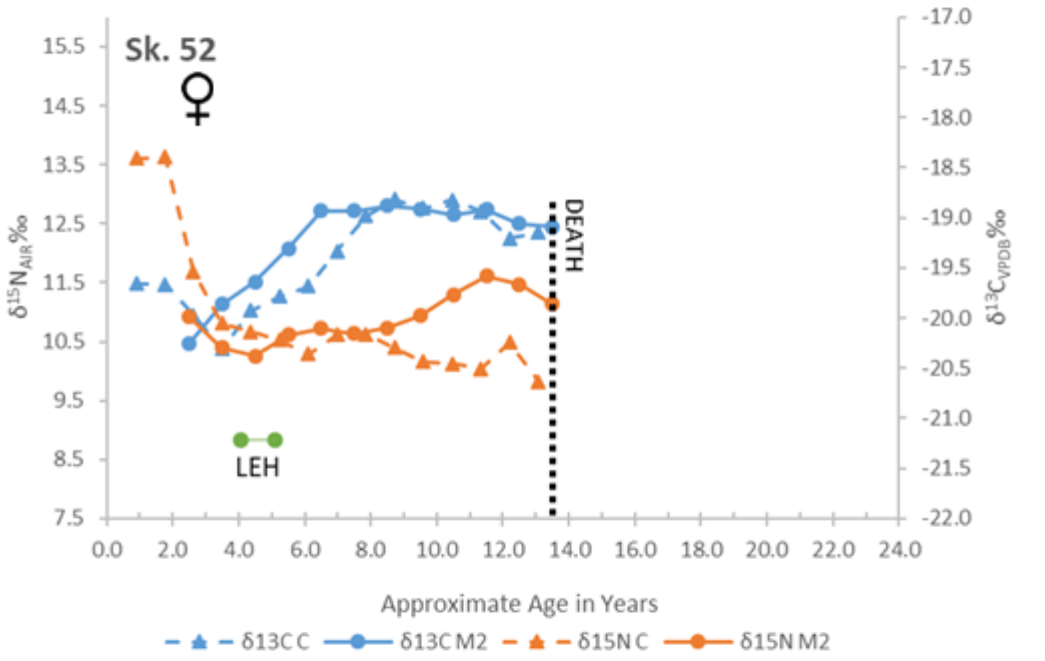

Sk. 15
♂
DEATH
LEH
δ15N AIR ‰
δ13C VPDB ‰
Approximate Age in Years
δ13C C
δ13C M3
δ15N C
δ15N M3

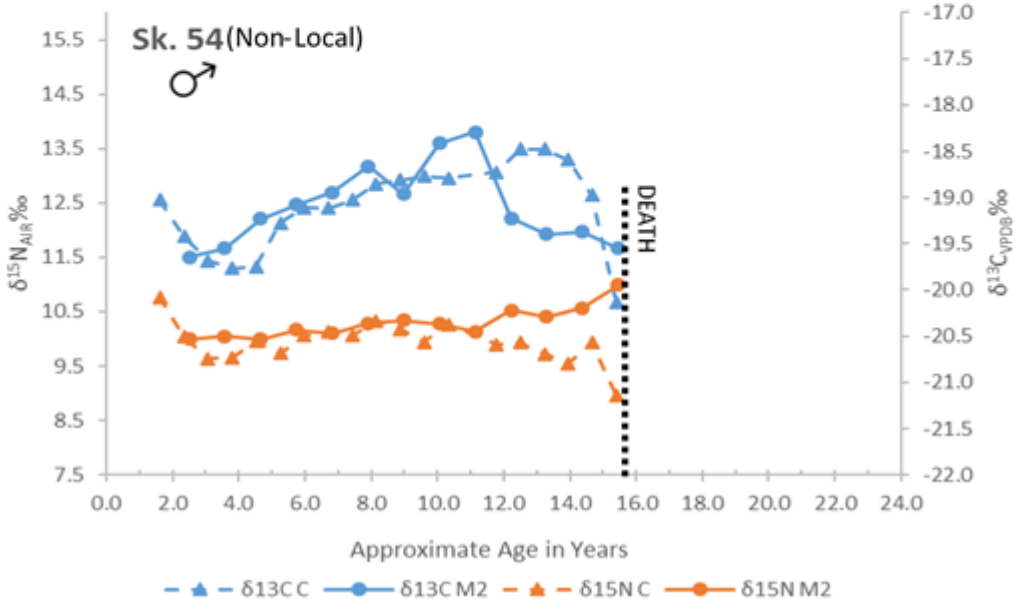

Sk. 18 (Non-Local)
♂
Rib C
DEATH
LEH
Approximate Age in Years
δ13C C
δ13C M3
δ15N C
δ15N M3


Sk. 27 (Non-Local)
♂
DEATH
LEH
Approximate Age in Years
δ13C C
δ13C M3
δ15N C
δ15N M3


Sk. 28 (Non-Local)
♂
DEATH
LEH
Approximate Age in Years
δ13C C
δ13C M2
δ15N C
δ15N M2


Sk. 45
♀
DEATH
LEH
δ15N AIR ‰
δ13C VPDB ‰
Approximate Age in Years
δ13C C
δ13C M2
δ15N C
δ15N M2


Sk. 52
♀
DEATH
LEH
δ15N AIR ‰
δ13C VPDB ‰
Approximate Age in Years
δ13C C
δ13C M2
δ15N C
δ15N M2


Sk. 54 (Non-Local)
♂
DEATH
δ15N AIR ‰
δ13C VPDB ‰
Approximate Age in Years
δ13C C
δ13C M2
δ15N C
δ15N M2

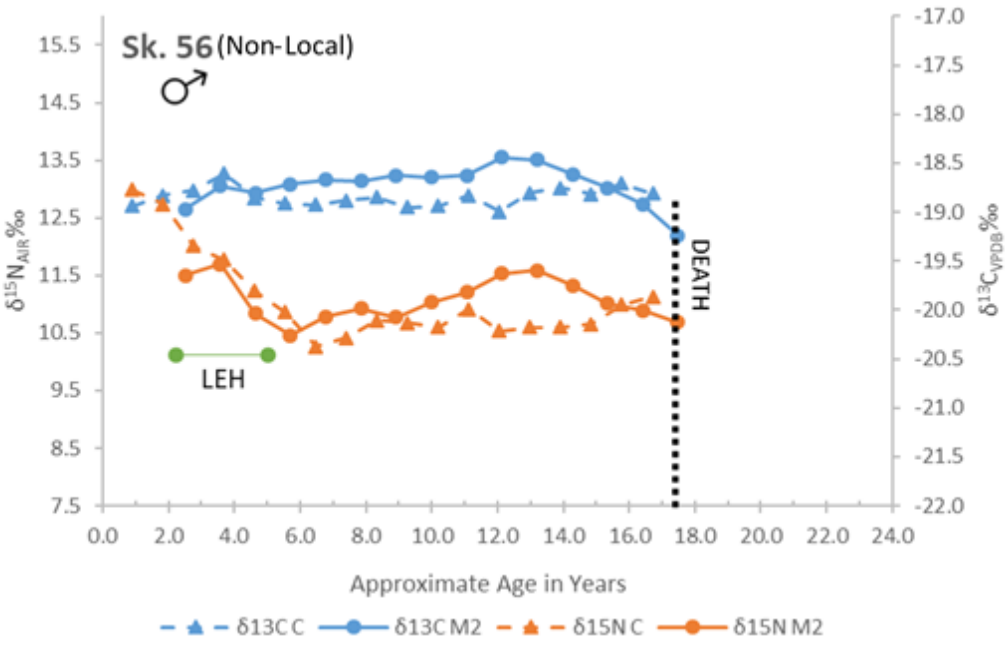
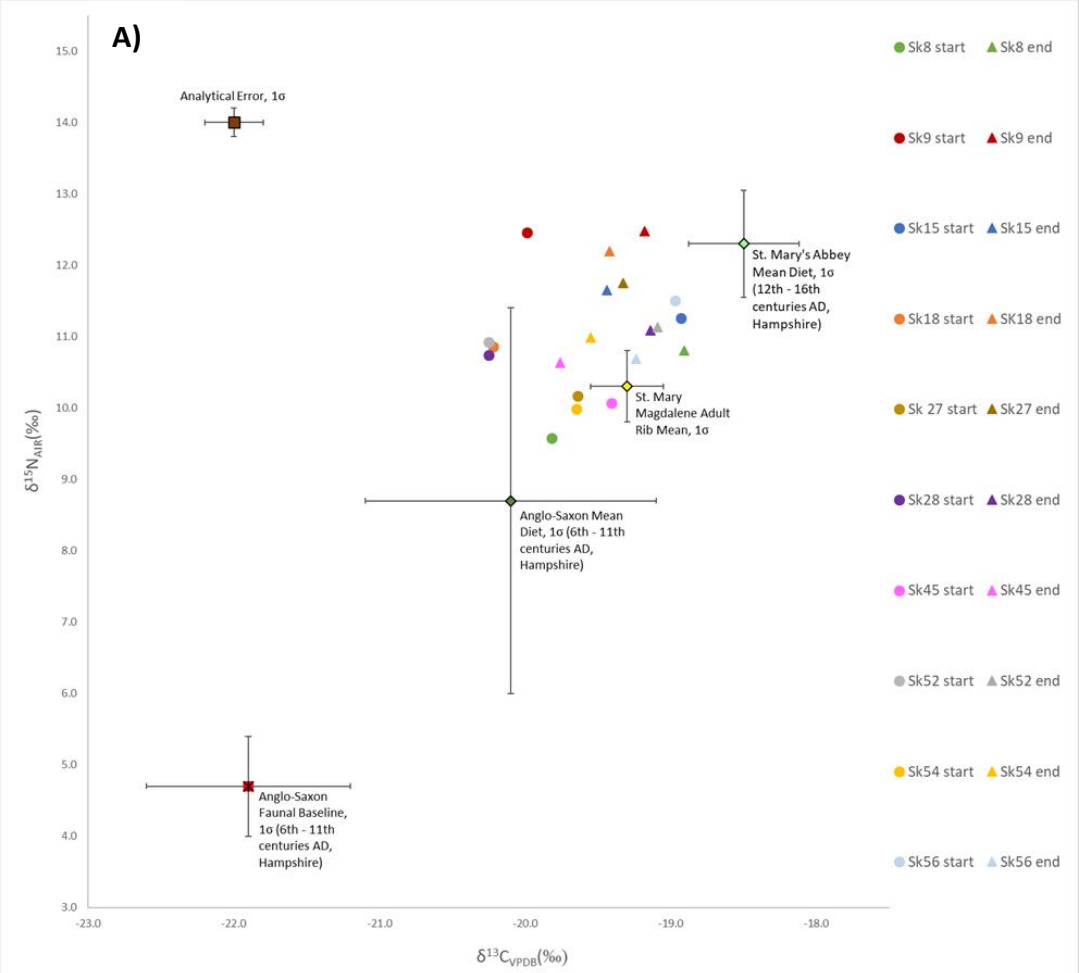


Figure 5.4.3 Life history incremental dentine profiles of $\delta^{13}C$ and $\delta^{15}N$ values from canine (triangles) and molar (circles) teeth from adolescents with lepromatous leprosy buried at St. Mary Magdalen Leprosy Hospital (Winchester, England). $\delta^{13}C$ and $\delta^{15}N$ from rib collagen (Cameron 2014; Roffey et al. 2017) are plotted for Sk. 8, Sk. 18, and Sk. 27. Also plotted are concurrent periods of linear enamel hypoplasia formation, estimated from Primeau et al. (2015).

BROAD TRENDS IN DIET AND POTENTIAL MOVEMENT TO THE LEPROSARIUM

$\delta^{13}C$ and $\delta^{15}N$ of both first and last increments of the molar teeth of local and non-locals are plotted in Figure 5.4.4 below and summarised in Table 5.4.3 above. The molar increments were plotted to avoid the influence of a breastfeeding signal in the $\delta^{15}N$ expected in the canines. The results do indicate a small shift in $\delta^{13}C$ (+ 0.4 ‰) and $\delta^{15}N$ (+ 0.6 ‰) values between the earliest and latest forming dentinal increment from both local and non-local adolescents, however, these differences are still within the normal dietary range of monastic populations reported elsewhere (Müldner and Richards 2005; 2007a; 2007b; O'Connell and Hull 2011; Kancle et al. 2018). Unsurprisingly, the biggest shifts in $\delta^{13}C$ and $\delta^{15}N$ occurred in non-locals (e.g. Sk. 18, Sk. 28, Sk. 52, Sk. 56). This is either most likely reflective of a change in diet from the place of their origin to being in the leprosarium in Winchester where they benefited from monastic provisions, i.e. a higher quality diet, or movement to the broader Hampshire area (Figure 5.4.5).

Additionally, the early forming molar increments have higher standard deviations ($\delta^{13}C$ = 0.4, $\delta^{15}N$ = 0.86, ±1σ) than the terminal molar increments that formed over the last one to two years prior to their deaths ($\delta^{13}C$ = 0.26, $\delta^{15}N$ = 0.64). Although this difference is small, it may indicate less dietary variability in line with a monastic convalescent diet. Interestingly, all of the terminal molar increments exhibit higher $\delta^{15}N$ values than the previously reported rib collagen values from adults at St. Mary Magdalen (Roffey et al. 2017; Figure 5.4.4). This difference may be due to bone collagen not representing the same age span as dentine due to metabolic disruption during bone turnover (Fleshman 2000; Weinbrenner et al. 2003; Pervanidour and Chrousos 2012; Beaumont et al. 2018; Beaumont 2020), and therefore not recording the changes in diet and physiological responses to disease in the years preceding death.


A)
Analytical Error, 1σ
St. Mary's Abbey Mean Diet, 1σ (12th - 16th centuries AD, Hampshire)
St. Mary Magdalene Adult Rib Mean, 1σ
Anglo-Saxon Mean Diet, 1σ (6th - 11th centuries AD, Hampshire)
Anglo-Saxon Faunal Baseline, 1σ (6th - 11th centuries AD, Hampshire)
Sk8 start
Sk8 end
Sk9 start
Sk9 end
Sk15 start
Sk15 end
Sk18 start
SK18 end
Sk 27 start
Sk27 end
Sk28 start
Sk28 end
Sk45 start
Sk45 end
Sk52 start
Sk52 end
Sk54 start
Sk54 end
Sk56 start
Sk56 end
15.0
14.0
13.0
12.0
11.0
10.0
9.0
8.0
7.0
6.0
5.0
4.0
3.0
-23.0
-22.0
-21.0
-20.0
-19.0
-18.0
δ15NAIR(‰)
δ13CVPDB(‰)

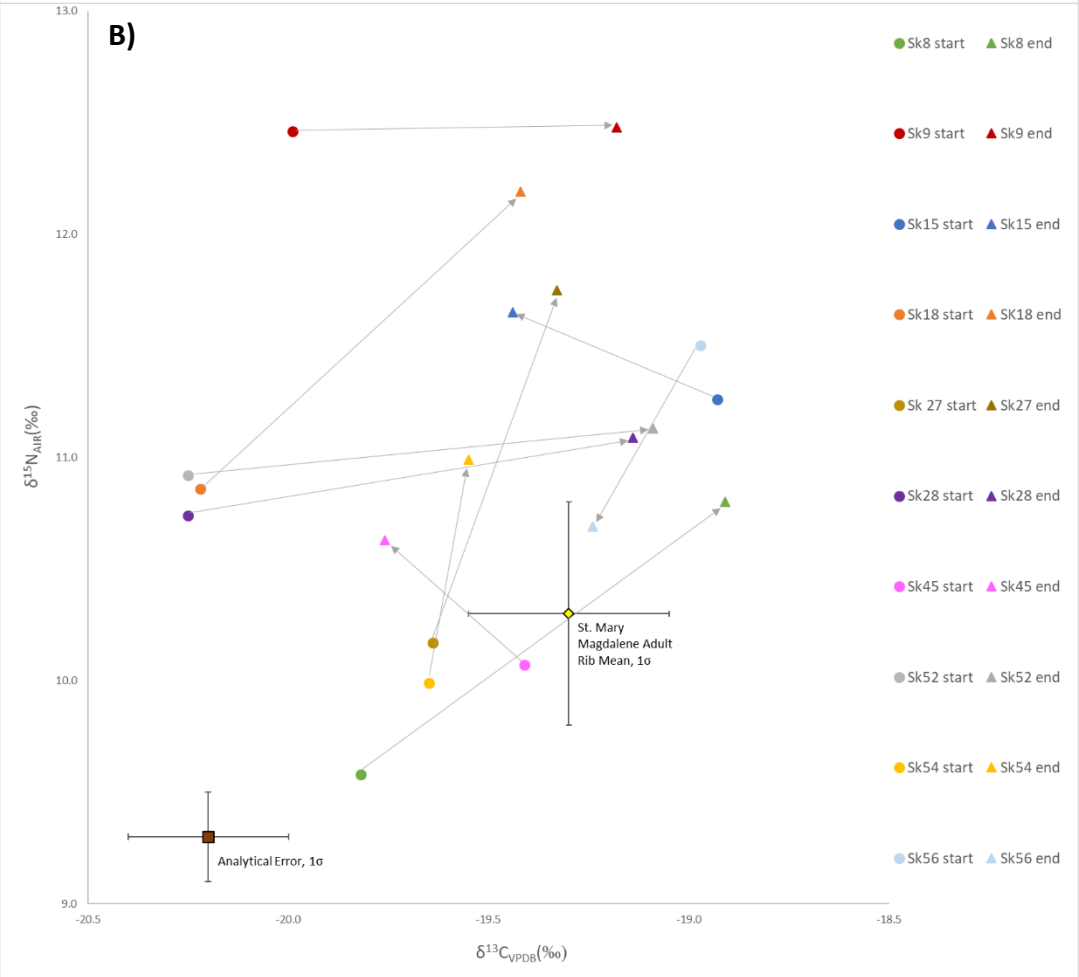

B)
St. Mary Magdalene Adult Rib Mean, 1σ
Analytical Error, 1σ
Sk8 start
Sk8 end
Sk9 start
Sk9 end
Sk15 start
Sk15 end
Sk18 start
SK18 end
Sk 27 start
Sk27 end
Sk28 start
Sk28 end
Sk45 start
Sk45 end
Sk52 start
Sk52 end
Sk54 start
Sk54 end
Sk56 start
Sk56 end
13.0
12.0
11.0
10.0
9.0
-20.5
-20.0
-19.5
-19.0
-18.5
δ15NAIR(‰)
δ13CVPDB(‰)

Figure 5.4.4 A) Scatterplot showing initial (circle) and terminal (triangle) molar increments of dentine collagen from adolescents buried at the St. Mary Magdalen leprosarium (SMM). Also plotted are data for comparison, including an 'Anglo-Saxon' (6th – 11th centuries AD) faunal baseline data from Hampshire (O'Connell and Hull 2011), the average 'Anglo-Saxon' mean diet from Hampshire (ibid), the SMM adult rib mean (Roffey et al. 2017), and the adult rib mean from the nearby high-status St. Mary's Abbey (12th – 16th century AD) mean diet (ibid). B) Scatterplot detail showing a general pattern of enrichment in ^{15}N and ^{13}C from the initial molar increment to the terminal molar increment of 10 adolescents buried at SMM. The end member values also differ to the average adult $\delta^{13}C$ and $\delta^{15}N$ rib values for the leprosarium, potentially indicating a more accurate reflection of diet at the time of death for adolescents (Beaumont et al. 2018; Beaumont 2020). The shift is likely due to increased access to higher quality foodstuffs, including animal products and marine resources. This would be in line with monastic prescriptions on diet, or a consequence of pathophysiological alterations of carbon and nitrogen metabolism due to systemic disruption as a consequence of leprosy or other diseases.

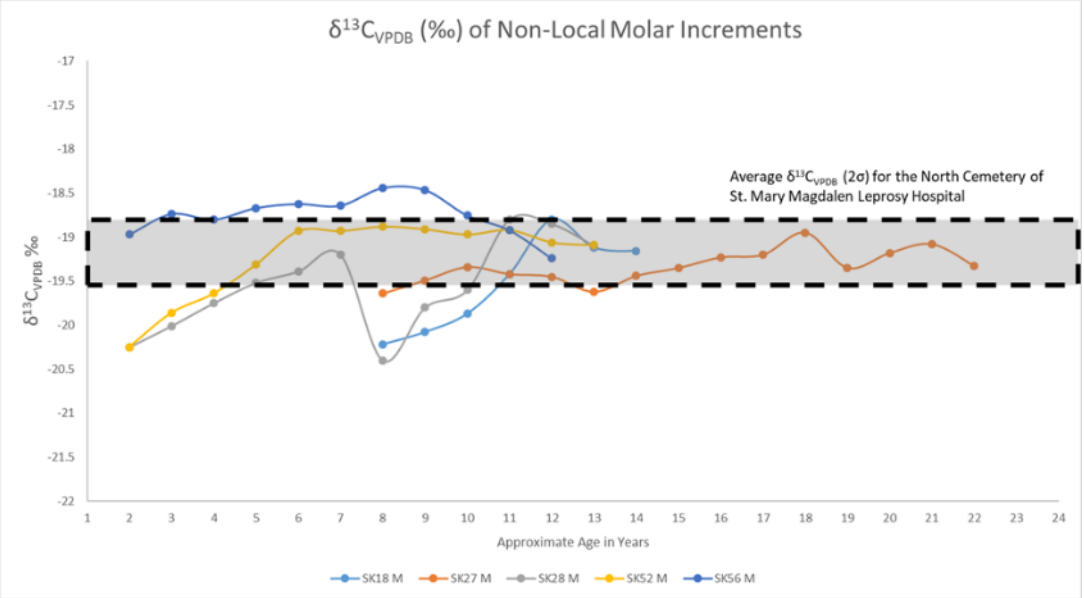
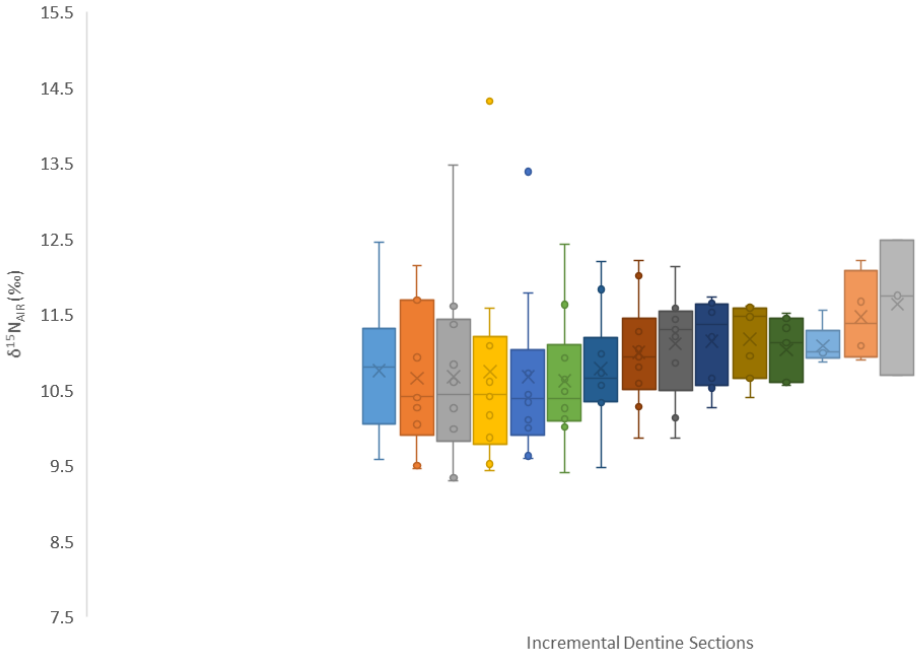


Figure 5.4.5. Scatterplot of incremental $\delta^{13}C$ values from molars of non-locals showing shifts towards the dietary average for people buried at St. Mary Magdalen leprosy hospital (boxed) These shifts may potentially signify entry into the leprosarium and how long the individuals remained there before death.

POTENTIAL INDICATORS OF PATHOPHYSIOLOGICAL STRESS

$\delta^{13}C$ and $\delta^{15}N$ values for individual molar sections were combined and are presented in box and whisker plots according to tooth section (Figure 5.4.6). These data show a general trend of reduced variability in both and $\delta^{13}C$ and $\delta^{15}N$ values between individuals as they move towards the end of their lives, possibly indicative of their monastic diet; however, $\delta^{15}N$ values begin to show greater variability in the final two increments. The increase in $\delta^{15}N$ variability near the end of their lives may be due to pathophysiological stress altering nitrogen metabolism, the presence of non-locals within the sample, or a combination of these two.



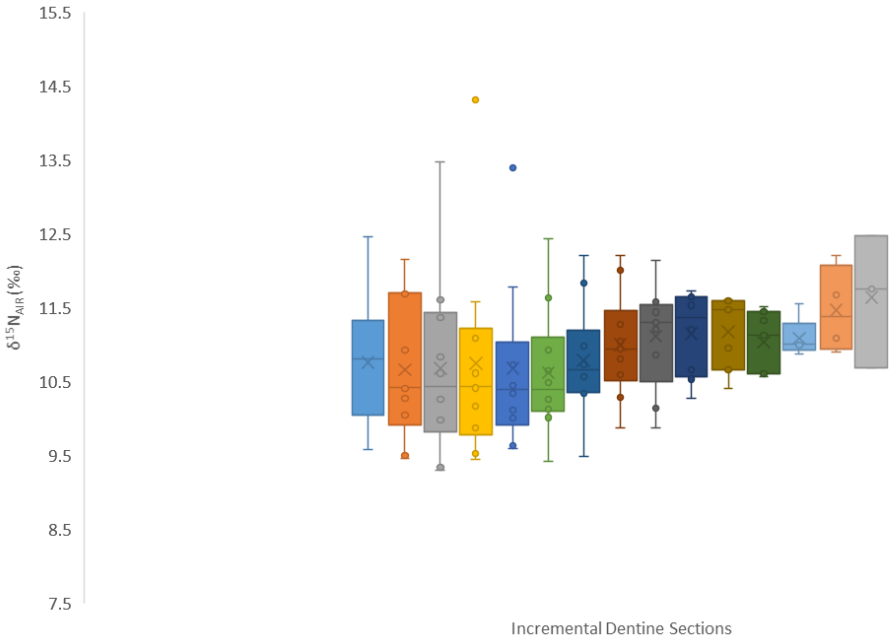


Figure 5.4.6 Box and whisker plots of $\delta^{13}C$ and $\delta^{15}N$ from molar dentine showing a reduced variability towards a "leprosarium diet". The last two molar increments illustrate decreased variation in $\delta^{13}C$ values and increased variation in $\delta^{15}N$ values, possibly due to pathophysiological stress caused by leprosy.

When viewing both canine and molar profiles, all individuals show at least one opposing covariance in carbon and nitrogen isotope ratios (i.e. a concurrent rise in $\delta^{13}C$ and a fall in $\delta^{15}N$, or vice-versa), sometimes within the same isotope of concurrently forming teeth (e.g. second molars and canines; Figure 5.4.7). Prior studies show that the presence of opposing co-variances can indicate nutritional and/or pathophysiological stress events (Beaumont et al. 2013b; Beaumont et al. 2015; Beaumont and Montgomery 2016; Craig-Atkins et al. 2018; Garland et al. 2018; Crowder et al. 2019; Walter 2020; Petersone-Gordina et al. 2020). Although the age of dentine increment formation are approximations (Beaumont and Montgomery 2015; Tsutaya 2020), previous studies of adolescents demonstrate isotopic continuity in co-forming canine-molar pairs (Beaumont et al. 2013b; Henderson et al. 2014; Beaumont and Montgomery 2015; Beaumont and Montgomery 2016; Crowder et al. 2019; Millard et al. 2020). Therefore, these deviations may be a consequence of the documented metabolic disruption associated with early life nutritional stress (Henderson et al. 2014; Beaumont et al. 2015; Garland et al. 2018;

Craig-Atkins et al. 2020) and leprosy development during periods of anabolic growth and development (Davey and Schenck 1964; Leal et al. 2003; Scollard et al. 2006; Rao 2009; Leal and Foss 2009; Singh et al. 2015; Butlin and Withington 2018).

Figure 5.4.7 Scatterplots of individuals showing isotopic disparities in $\delta^{15}N$ (all) and $\delta^{13}C$ values (Sk. 18, Sk. 54, 56) in concurrently forming canines (triangles) and molars (circles).

DISCUSSION

WERE ADOLESCENTS FED A POOR DIET IN THE LEPROSARIUM?

Isotope profiles obtained from the incremental dentine collagen of adolescents demonstrate an isotopic shift towards a more homogenous diet between individuals in the leprosarium, and one higher in animal products and/or marine food sources relative to the average carbon and nitrogen isotope ratios for the Early Medieval period and Hampshire region (O'Connell and Hull 2011). This potentially indicates that food provisions within the leprosarium were more protein-rich (e.g. meats, dairy products, fish) than those outside the hospital where the population consumed a predominantly plant-based diet (O'Connell and Hull). Diet is intimately linked to health and immune system strength, and diet is an important component of medical care today, as it was in the past (Adams 1868: 350; Johnston 2006: 261; Ulijaszek 1990; Ulijaszek 2018). Previous analyses (Cameron 2014; Roffey et al. 2017) of $\delta^{13}C$ and $\delta^{15}N$ from adult rib collagen from the individuals buried at St. Mary Magdalen leprosarium were indistinguishable from rib collagen carbon and nitrogen isotope ratios at nearby Hyde Abbey (12^{th} – 16^{th} centuries AD; $\delta^{13}C$ mean -19.5‰, $\delta^{15}N$ mean 10.4‰), indicating consistency in diet with other nearby monastic communities. However, adolescent dentine collagen data in the present study yielded slightly higher $\delta^{13}C$ and $\delta^{15}N$ means ($\delta^{13}C$ mean -19.3‰, $\delta^{15}N$ mean 11.4‰) and plot in between the dietary means for Hyde Abbey and the high-status St. Mary's Abbey (12^{th} – 16^{th} centuries AD; $\delta^{13}C$ mean -18.5‰, $\delta^{15}N$ mean 12.3‰) (Cameron 2014; Roffey et al. 2017). The possibility of a diet higher in animal products and/or marine food sources for adolescents in the leprosarium is also consistent with the c. 12^{th} century AD reports of communal donations of 'high-quality foods' to leprosaria (e.g. fresh meat, dairy, poultry, and fresh fruits) within England and Scotland (Rawcliffe 2006: 322-328). Furthermore, archaeological evidence from pre-12^{th} century AD English parish cemeteries and leprosaria suggests that individuals with leprosy were provided with a level of care and community inclusion that is inconsistent with oft-cited historical sources (Anderson 1998; Duhig 1998; Roffey and Tucker 2012; Inskip et al. 2015; Roffey et al. 2017; Filipek et al. 2021). Although a small sample of individuals were studied here, this evidence helps to contribute to a larger biocultural picture of care that challenges the view that people with leprosy were treated poorly in the past.

Can we view when adolescents entered the leprosarium?

Dentinal collagen provides a higher temporal resolution than bone collagen in isotope analysis and, as such, we may be able to use plots of incremental dentine of individuals to explore when immigration occurs in relation to death and burials (Beaumont et al. 2013a). In previous and parallel studies (Roffey et al. 2017; Filipek et al. *under review*), five individuals (Sk. 18, Sk. 27, Sk. 28, Sk. 52, Sk. 56) were deemed to be non-local to the St. Mary Magdalen leprosarium, based on strontium and oxygen isotope analyses derived from molar enamel. When viewing their incremental isotope plots, clear shifts in $\delta^{13}C$ values are apparent (Figure 5.4.5), and the timing of these shifts can be used as a potential proxy for when the individuals migrated to Winchester and were admitted to St. Mary Magdalen leprosy hospital, or lived in the broader Winchester area. Similar shifts are also evident in $\delta^{15}N$ values (Figure 5.4.3), however, these show greater variability likely due to isotopic 'noise' as a consequence of pathophysiological stress altering the normal diet-tissue space (Reitsema 2013; Reitsema and Holder 2018). One individual (Sk. 56) possibly from Scandinavia or central Europe (Filipek et al. *under review*), demonstrates a decrease in $\delta^{13}C$ around the age of 12. This individual also shows signs of leprogenic odontodysplasia, which can be approximately timed to have occurred between 6-8 years old using dental development methods (Moorrees et al. 1963; AlQahtani et al. 2010). This would indicate Sk. 56 migrated with the disease, potentially to seek care at St. Mary Magdalen leprosy hospital. Two other individuals (Sk. 28 and Sk. 52) yielded nearly identical strontium and oxygen isotope values (*ibid*), and their $\delta^{13}C$ values also show identical shifts commencing from approximately the age of 2-3 years (Figure 5.4.5). Their identical origins and early life dietary patterns warrant further biomolecular investigations to explore whether they were related to each other (kinship) and *M. leprae* strain type to see whether strain type can provide information on geographic location.

PATHOPHYSIOLOGICAL STRESS NEAR THE END OF LIFE IN NITROGEN ISOTOPE PROFILES

Recent methods pioneered by Beaumont and colleagues (2012; 2013; 2015; 2018; Beaumont and Montgomery 2015) highlight the benefits of using collagen derived from incremental dentine to signal dietary fluctuations through the life course, and reaffirm the sensitivity of these isotopic systems to metabolic stressors, including nutritional and

pathophysiological stress (Craig-Atkins et al. 2018; Crowder et al. 2019; Craig-Atkins et al. 2020; Nicholls et al. 2020; Walter et al. 2020; Petersone-Gordina et al. 2020). Whilst the results from the incremental dentine collagen profiles show a less dietary variation over time in adolescents, homogenising towards a 'leprosarium diet', terminal dentinal increments show an increased variation in $\delta^{15}N$ (Figure 5.4.6). This may be due to non-locals moving to the leprosarium during the last year of life, or that nitrogen metabolism was altered as a consequence of tissue catabolism due to leprosy dissemination. Because the $\delta^{13}C$ values continue to homogenise until death, the latter scenario is more likely.

Discrepancies in Bone and Dentinal Collagen

Although bone collagen is routinely used to measure carbon and nitrogen isotope ratios, incrementally sampled dentine collagen reflects a more detailed record of temporal changes through childhood and adolescence that may not be otherwise recorded in bone collagen values (Beaumont et al. 2013a; Montgomery et al. 2013; Beaumont et al. 2018; Beaumont 2020). For example, the $\delta^{13}C$ and $\delta^{15}N$ previously reported from the rib collagen of three individuals (Sk. 8, Sk. 18, Sk. 27) (Cameron 2014) were offset with end of life $\delta^{13}C$ and $\delta^{15}N$ values by a period of up to six years prior to death, whereas dentinal collagen recorded $\delta^{13}C$ and $\delta^{15}N$ values up to the point of death. This reveals a more accurate snapshot of both the 'St. Mary Magdalen diet' and the potential metabolic alterations consequential of pathophysiological stress. The capacity of leprosy to affect bone protein composition and metabolism is well-established (Davey and Schenck 1964; Leal et al. 2003; Scollard et al. 2006; Leal and Foss 2009; Singh et al. 2015; Scott et al. 2016; Scott et al. 2020). As such, a stress threshold in osteoblastic production may be a reason why bone collagen values derived from ribs are offset with dentinal averages that should reflect similar periods *in vivo*; i.e. odontoblasts continue to record $\delta^{13}C$ and $\delta^{15}N$ values, and are therefore a more accurate reflection of carbon and nitrogen isotope ratios values at the time of death (Beaumont et al. 2018).

Linking early life stress in enamel and the manifestation of leprosy in the dentinal collagen profiles

Incremental dentine profiles demonstrated small opposing co-variances in $\delta^{13}C$ and $\delta^{15}N$ values in conjunction with the timing of linear enamel hypoplasia in all the individuals.

The timings of these metabolic disturbances are also observed in dentine profiles elsewhere (Henderson et al. 2014; Garland et al. 2018; Crowder et al. 2019; Walter et al. 2020) and they may signify alterations in diet or rerouting of resources to where they are most needed during periods of nutritional or pathophysiological stress. Previous studies (Filipek-Ogden and Roberts 2014; Yaussy et al. 2016; Betsinger and Dewitte 2017; Yaussy and Dewitte 2018) demonstrate statistically significant associations with linear enamel hypoplasias and increased morbidity risk and early mortality across multiple archaeological sites, including those revealing individuals with leprosy. These early life 'stress' events may be a predisposing factor in the high prevalence of skeletal lesions diagnostic of lepromatous leprosy among the adolescent cohort at St Mary Magdalen. Early life nutritional status as a primary factor in leprosy susceptibility and manifestation are a more recent area of research in endemic communities today (Feenstra et al. 2011; Filipek-Ogden and Roberts 2014; Wagenaar et al. 2015; Oktaria et al. 2018; Dwivedi et al. 2019), and our data mirrors these modern clinical patterns.

Physiological disruptions in co-forming teeth

An incidental finding in this study was the unexpected isotopic disparity in co-forming teeth. Canine and molar teeth were expected to have overlapping values during the co-forming stages of tooth development, demonstrating continuity between teeth and as seen in other studies (Beaumont et al. 2013b; Henderson et al. 2014; Beaumont and Montgomery 2015; Crowder et al. 2019; Millard et al. 2020). At least five individuals (Sk. 15, Sk. 18, Sk. 52, Sk. 54, Sk. 56) show a divergence in $\delta^{15}N$ of up to 2‰ in co-forming teeth, two of whom (Sk. 18, Sk. 54) also show smaller divergence in $\delta^{13}C$ values of up to 0.9 ‰ (Figure 5.4.7). Leprosy opportunistically metabolises during anabolic phases (i.e. metabolic growth phases) and shows the capacity to disrupt nearly all body systems, consequently affecting metabolic processes and puberty in growing children (Rao et al. 1972; de Oliveira et al. 2012; Davey and Schenck 1964; Leal et al. 2003; Scollard et al. 2006; Leal and Foss 2009; Singh et al. 2015; Lastoria and Abreu 2014). These metabolic perturbations may account for both the disruption in $\delta^{15}N$ and $\delta^{13}C$ values in co-forming teeth, and the advanced stages of lepromatous leprosy present in the St. Mary Magdalen adolescents. Moreover, three individuals who show leprogenic odontodysplasia (Sk. 8,

Sk. 52, Sk. 56) demonstrate these $\delta^{15}N$ offsets that are concurrent in timing with arrested development of the anterior maxillary dentition. This potential proxy for identifying the onset of leprosy related metabolic disruption warrants further investigation. If metabolic disruptions caused by nutritional and pathophysiological stress can be identified within dentine profiles, this may give researchers more insight to the timing of diseases affecting lifelong metabolism and offer more meaningful understandings of inequity, frailty, and resilience.

CONCLUSIONS

Notions of Medieval stigma and inadequate care are often linked with present-day negative treatment of people with leprosy. Therefore, it is worth investigating if these past views are anecdotal or can be supported with archaeological evidence, thus providing a long view or deep time perspective. This study demonstrates some of the potential for using carbon and nitrogen isotope analyses of incremental dentine to understand both the biological and social ramifications of leprosy in the past. Broad trends indicate a homogenous diet over time for adolescents buried at St. Mary Magdalen leprosarium, perhaps indicating the hospital diet was higher in animal products and marine resources than the Early Medieval average in that region, and shows a dietary similarity to other high-status monastic contexts (e.g. St. Mary's Abbey). Examinations of $\delta^{13}C$ values also serve as a potential proxy for the movement to the hospital of people who were buried there.

Incremental dentine analyses allow for the visualisation of fluctuations in carbon and nitrogen isotope ratios to reveal life course perturbations that can influence these broad interpretations of diet. Detailed examination of $\delta^{13}C$ and $\delta^{15}N$ dentine collagen profiles generated from adolescent canine and molar pairs shows isotopic disparities in co-forming tissues (both between ribs and concurrently forming teeth) in $\delta^{15}N$ and (to a lesser extent) $\delta^{13}C$. This supports the suggestion that dentinal collagen is more sensitive to pathophysiological stress (Beaumont et al. 2018; Beaumont 2020; Petersone-Gordina et al. 2020), underscoring the need to consider pathophysiological stress in stable isotope interpretations *per se*. By viewing carbon and nitrogen isotope profiles of incremental dentine in conjunction with pathological conditions, we will become better at being able

to tease out the impacts of nutritional stress and disease on diet-tissue space; i.e. the difference between dietary values and consumer tissues (Beaumont et al. 2013b; Henderson et al. 2014; Garland et al. 2018; Walter et al. 2020). It is the case that too often there is a lack of consideration of the possibility that pathological lesions and the subsequent metabolic effects of physiological disruptions are not considered in the interpretation of isotopic data (Katzenberg and Lovell 1999; Reitsema 2013). This highlights the growing need of incorporating a more holistic view of the body in the interpretation of isotope values and ratios derived from human tissues in archaeological skeletons. Palaeopathology as a discipline should not be ignored.

In future, expanding the sample size to include $\delta^{13}C$ and $\delta^{15}N$ analyses of incremental dentine collagen from individuals buried within later leprosaria contexts (e.g. the South Cemetery at St. Mary Magdalen, Winchester; St. James and St. Mary Magdalene, Chichester; and St. Leonard's, Peterborough) may help to reveal further isotopic proxies for the entry of people into hospitals, and whether any temporal changes in diet exist within and between leprosy hospitals. Similarly, it is worth further exploring the biological impact of leprosy through more carbon and nitrogen isotope analyses of incremental dentine collagen to view potential patterns that may signal the 'onset' of leprosy. Comparisons of individuals with leprosy from other non-leprosaria contexts may help to further tease out isotopic signals for metabolic disruptions due to nutritional and/or pathophysiological stress. Generating sequential $\delta^{13}C$ and $\delta^{15}N$ dentine profiles from individuals showing evidence for leprogenic odontodysplasia may also contribute to solidifying the nexus between nitrogen metabolism disruption and systemic *M. leprae* dissemination through the bodies of people who contracted the infection in the past.

ACKNOWLEDGEMENTS:

Special thanks are given to Simon Roffey and Katie Tucker for granting access to the adolescents from the St. Mary Magdalen leprosy hospital, and Marise Gorton, Tommy Morgan, John Henry Jackson from Bradford University and Beth Upex and Steve Robertson from Durham University for their invaluable support in the isotope labs. Thank you to Matthew Crowther and Ellen Kendall for their helpful comments.

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CHAPTER 6. DISCUSSIONS, FUTURE DIRECTIONS, AND FINAL CONCLUSIONS

The primary aim of this research was to explore the biological and social impacts of leprosy on adolescents during the Early-Late Medieval transition through a lens of 'caring'. The resultant data generated from contextual data (historical and archaeological), palaeopathological analyses, clinical considerations, and multi-isotope models have all helped to answer achieve this aim and answer the three research questions proposed alongside the hypothetical models in Chapter 1 (Table 6.1), giving us a deeper insight into the lived experience of adolescents with leprosy in the Early Medieval period. This final chapter outlines the outcomes of the research questions addressed, provides key limitations to this study, offers future directions to further our understanding of leprosy and the ways in which people with the disease were treated in the past, and concludes with an overall summary of this research.

TABLE 6.1 – Research questions revisited

MAIN RESEARCH QUESTIONS	AREAS IN THESIS
Do we see evidence of mobility in adolescents with leprosy?	Manuscript 5.2, Manuscript 5.3
Were individuals provided medical and nutritional care within the leprosarium?	Chapter 2, Manuscript 5.1, Manuscript 5.3, Manuscript 5.4
Can we see indications of nutritional or pathophysiological stress in the dentine collagen profiles of individuals with leprosy over the life course?	Manuscript 5.4

6.1 MOBILITY AND IDENTITY ANALYSES

This study used strontium and oxygen isotope analyses to reveal mobility histories of adolescents who died with leprosy in both a 'normal' parish cemetery (Norwich, modern day Norfolk) and a leprosy hospital context (Winchester, modern day Hampshire). The results presented in Manuscripts 5.2 and 5.3 expand previous work on skeletons at Winchester (Taylor et al. 2013; Roffey et al. 2017), and have revealed marked differences between the two contexts. Manuscript 5.2 also presents the first human strontium and

oxygen isotope data for Norwich. At the St. John at the Castle Gate/Timberhill site, all of the individuals showed isotope ratios consistent with the local area and nearby comparative data. One outlier (Sk. 13121) demonstrated a higher oxygen isotope ratio than expected, however, it is likely the high values indicate a breastfeeding signal rather than evidence of non-locality due to the age of development of the tooth selected. Combining the isotopic and archaeological data from Norwich, it appears that evidence for community exclusion of people affected by leprosy is not supported. The individuals studied from St. Mary Magdalen leprosy hospital yielded more varied data, indicating that 7/19 were non-local to the Winchester area. The timing of leprogenic odontodysplasia in at least one of the non-local individuals in Winchester (Sk. 56) coincides with his molar crown development, meaning he was likely enduring the visible effects of leprosy before moving to the Winchester area. In conjunction with the archaeological data, this points to this hospital, at least, likely being regarded as a place of refuge that people with leprosy travelled long distances to for care and treatment, rather than a place of exclusion. This has been previously suggested (Roberts 1986; Rawcliffe 2006:291-301; Roffey and Tucker 2012; Roberts 2013), but has proven difficult to evidence. This study lends further support to these previously posed hypotheses, tipping the balance in favour of support, and not directed, stigma, for people with leprosy.

Regarding sex-based differences in the occurrence of leprosy in adolescents, there was little difference between both sites with a higher male:female ratio, although sex could not be determined in 4/10 individuals at the St. John parish site. Prior research has posited that hospitals and leprosaria were segregated by sex (Orme and Webster 1995: 22-23; Rawcliffe 2006: 122-123; Magilton 2008:59) and the presence of women in these spaces were generally confined to abbesses, nurses, or caregiving servants (Orme and Webster 1995: 82-83). Previously, four older females were identified in the North Cemetery at St. Mary Magdalen leprosy hospital, which could align with previous assumptions regarding the role of women in the hospital, however amelogenin peptide analyses revealing two female adolescents with advanced stages of lepromatous leprosy warrants reconsideration of these prior engendered social identities.

6.2 CARE IN THE LEPROSARIUM

To further understand what life was like inside the leprosy hospital, two approaches were taken: application of the Index of Care framework and examining aspects of the diet of those affected by leprosy. The Index of Care is a multi-staged, case-based research framework that shows the potential to demonstrate whether care was provided for a person, or if they were left without medical and societal support (Tilley and Cameron 2014). Previous research by Roberts (2017) applied the Index of Care framework to an individual with lepromatous leprosy (C148) buried in the cemetery associated with St. James and Mary Magdalene Leprosy Hospital, Chichester, in modern day Sussex (12^{th} – 15^{th} centuries AD). Roberts (*ibid*) demonstrated that the individual could have required significant palliative care to survive, but could not assert with any confidence that such care was provided within the leprosarium. This method was applied to Sk. 19 from St. Mary Magdalen due to the presence of advanced signs of lepromatous leprosy and personalised grave goods (e.g. adapted feeding implements). The results in Manuscript 5.1 also demonstrates that Sk. 19 could have needed significant palliative, economic, and communal support to survive. Although like Roberts' (2017) study, it is not possible to say for certain that Sk. 19 received this support from within the St. Mary Magdalen leprosarium, other evidence of apparent palliative care (e.g. showing evidence for an amputation), and his unique burial context with individualised grave goods increases this likelihood.

Another focus of care this study researched was diet. Diet is a key component of health and of medical care, both past and present (Ulijaszek 1990; Ulijaszek 2018). In this regard, this study presents the first carbon and nitrogen isotope data from incremental dentine from individuals buried at an archaeological leprosarium. Although both canine and molar teeth were analysed, the molars were used to understand changes in diet and better comprehend the nature of the leprosarium diet. This was to avoid breastfeeding signals from the canines confounding the data. Previous research (Cameron 2014; Roffey et al. 2017) established a dietary average for the St. Mary Magdalen leprosy hospital based on adult rib collagen and found that the $\delta^{13}C$ and $\delta^{15}N$ values were nearly identical to Hyde Abbey (Winchester), but dissimilar to the 'high-status' St. Mary's Abbey (Winchester). Manuscript 5.4 showed dentinal increments from adolescent molars

revealed a dietary shift in $\delta^{13}C$ and $\delta^{15}N$ towards the St. Mary's Abbey values between the coronal and terminal increments. This shift is not reflected in the bone collagen $\delta^{13}C$ and $\delta^{15}N$ values, likely due to the slow turnover of bone (Beaumont et al. 2018; Beaumont 2020), thereby not reflecting the diet as effectively as dentinal collagen. $\delta^{13}C$ profiles from people identified as non-locals show dietary shifts, which may potentially signal a move of the person to the leprosarium. These profiles combined with palaeopathological evidence (e.g. leprogenic odontodysplasia) suggests that at least one individual migrated after they had developed leprosy, potentially seeking care and treatment. Although small, the variation in $\delta^{13}C$ and $\delta^{15}N$ values also lessens in the molar increments through time, which may indicate a dietary consistency in the years preceding death, and therefore a better representative 'leprosarium diet'. While the variation in $\delta^{13}C$ and $\delta^{15}N$ values does lessen in the years preceding death, there is increased variation in $\delta^{15}N$ values near to the time of death, possibly reflecting recently admitted hospital patients or an interference in nitrogen metabolism due to pathophysiological stress, or both.

6.3 Life Histories and 'Stress'

Crucial but not fully understood components of contracting leprosy and its dissemination are individual immunocompetency and lifelong physiological factors (Scollard et al. 2006; Lastória and Abreu 2014). Many studies focus on genetic factors that may increase leprosy susceptibility (Santos et al. 2002; Bleharski et al. 2003; Mira et al. 2004; Zhang et al. 2009) but, more recently, studies on lifelong nutritional status and its effects on immune system development and function have increased (Feenstra et al. 2011; Wagenaar et al. 2015; Oktaria et al. 2018; Dwivedi et al. 2019). Incremental dentine profiles of archaeological skeletons offer a glimpse into an individual's lifelong nutritional status and shows the potential to reveal metabolic perturbations that can be linked to both nutritional and pathophysiological stress (Beaumont et al. 2013; Henderson et al. 2014; Beaumont et al. 2015; Garland et al. 2018; Craig-Atkins et al. 2018; Crowder et al. 2019; Nicholls 2020; Walter et al. 2020; Craig-Atkins et al. 2020). Manuscript 5.4 provides the results of palaeopathological and incremental dentine analyses undertaken on 10 individuals from the St. Mary Magdalen leprosy hospital. They suggest that early life

stress is not only skeletally present in all the individuals sampled, but align isotopically with small opposing co-variances in $\delta^{13}C$ and $\delta^{15}N$ values, suggesting physiological disruptions to carbon and nitrogen metabolism. It is important to bear in mind that although these adolescents bear lesions associated with early life nutritional or pathological stress, their immune status was strong enough to enable them to survive these early life insults. However, survival usually comes at an immunological cost, leading to a trade-off of energy expenditure for survival at the expense of acquired immune robusticity (Ulijaszek 1990; 1996; Barker 2004; Pervanidou and Chrousos 2012; Langley-Evans 2015; Wells 2016:43-63). This can have implications for later life health and a potential earlier age at death, as shown by clinical studies (Barker 2004). By verifying these early life stressors that were significant enough to disrupt normal metabolic function, this helps to understand the overall impact of early life nutritional status on immune susceptibility.

Incidentally, the dentine profiles also signalled an opposing covariance in $\delta^{13}C$ and $\delta^{15}N$ values between concurrently forming teeth later in life. This was an unexpected finding, and one that perhaps indicates when the effects of leprosy begin to cause wider pathophysiological disruption in a person's life. A potential proxy for linking leprosy related disruption to $\delta^{13}C$ and $\delta^{15}N$ values could be information about the timing of the development of leprogenic odontodysplasia (arrested tooth development of the anterior maxillary incisors) against the dentinal plots. If these $\delta^{13}C$ and $\delta^{15}N$ co-variances consistently occur with the development of this condition, we can use this co-variance in concurrently forming teeth as a proxy for understanding when physiological disruptions are occurring in other individuals, as well as how long individuals are living with the deleterious effects of the disease.

6.4 Limitations of the Research

This research contributes to the understanding of both biological and social impacts of leprosy in the past; however, it is not without significant limitations. Many of the methodological limitations are addressed in Chapters 2 and 3 (background chapters), and issues with access, sampling, and analysis are given in Chapters 4 and Chapter 5

(materials and methods, and results), and therefore major interpretive limitations will be the focus here.

One of the main limitations of this research is the sample sizes of the selected cohorts. Only 10 individuals from St. John's at the Castle Gate/Timberhill and 19 individuals from St. Mary Magdalen leprosy hospital were selected for strontium and oxygen isotope analyses (Sections 5.2 and 5.3). Although this number is not insignificant, particularly in mobility analyses where costs can prove prohibitive, the sample size only offers a glimpse rather than a broader view of disease related mobility and sets the scene for future work. Another sample size limitation was encountered with the carbon and nitrogen isotope analyses of incremental dentine (Section 5.4). Although permission was provided to apply this method to all individuals sampled, only 10 individuals were chosen, due to financial constraints, which limits the application of statistical analyses and comprehending wider patterns of dietary and pathophysiological alterations. Financial constraints also prohibited the broader application of the amelogenin peptide analyses to the adolescents from St. John at the Castle Gate/Timberhill. This would have provided more understanding of the implications of sex and social identity of the adolescents who died with leprosy within their communities, and offered a better comparative population breakdown for the St. Mary Magdalen adolescent cohort. These sample size and financial restrictions are limiting, however, these limitations offer more opportunity for further research in the future.

6.5 Future Directions

The research embedded within this thesis evidences some of the biological and social impacts of leprosy on adolescents during the Early-Late Medieval transition through a lens of care, but consequentially creates more research avenues to explore.

6.5.1 Disease related mobility and mortuary treatment

Disease ecologies and their transmission dynamics are a key component to understanding how individuals and their communities respond to infections. By expanding the datasets for strontium and oxygen isotope analyses to other early leprosy sites (e.g. sites in Uzbekistan, Israel, Turkey, Hungary, France, Italy, and the UK) and high-

status burials of individuals with leprosy (e.g. Edix Hill bed burial, Israeli tombs, Uzbek kurgans), would improve our understanding of leprosy transmission and the 'treatment' of people by communities through time, with treatment being considered in its widest sense. By viewing the mobility histories of individuals buried within cemeteries at other leprosy hospital sites (e.g. Chichester, Peterborough, Naevsted, etc.), a bigger picture of disease-related mobility may also emerge (i.e. how far were people travelling and which groups were travelling most). It would also be worth expanding the datasets of the cohorts already selected to include adults. It is possible that local adolescent populations (e.g. those buried at St. John at the Castle Gate) were children of non-locals with the disease, and this may alter our perceptions of community treatment. Lastly, applying strontium and oxygen isotope analyses to individuals with leprosy from the later-dating South Cemetery at St. Mary Magdalen may reveal if the mobility histories of individuals change from the 12th century AD, possibly due to a wider paradigm shift in the treatment of individuals with leprosy prior to the disease's 14th century AD decline.

6.5.2 Synergistic relationships between isotopes and disease

Further studies that explore the relationships between isotope values and ratios and disease, as identified in skeletons, are needed. This is particularly relevant because of the plethora of published literature on the relationships between migration and disease (see https://www.euro.who.int/en/health-topics/health-determinants/migration-and-health). Being able to provide the deep time perspective for mobility in relation to infectious disease should be one of bioarchaeology's foci. By viewing more whole life carbon and nitrogen incremental dentine profiles from adolescents with lepromatous leprosy buried in the St. Mary Magdalen leprosy hospital, as well as other leprosy hospital sites, we may better comprehend both aspects of diet and potential long-term care within the leprosarium. This study attempted to view dietary changes as a proxy for entry into the leprosarium, but the sample sizes were too small to highlight any specific pattern. Despite this, initial data presented in Manuscript 5.4 suggests this pattern may exist. It is therefore worth evidencing further to better comprehend the timing of the palaeopathological appearance of leprosy-related skeletal lesions and their relationship

with entry to leprosaria, and to view the length of time individuals spent within the leprosarium before death.

Another aspect of this research that merits further research is investigating disruptions to the normal diet-tissue space – i.e. the normal offset between stable isotope ratios of diet and consumer tissues. Divergences in the dentine collagen profiles of concurrently forming teeth signify a metabolic disruption potentially as a consequence of leprosy. It is worth trying to determine if these disruptions can be 'timed' with the development of other leprosy related lesions (e.g. leprogenic odontodysplasia). It is also worth exploring whether similar diet-tissue spacing disruptions occur with other infections that affect the metabolism of carbon and nitrogenous compounds (e.g. adolescents with tuberculosis). If similar patterns between leprosy and tuberculosis exist in adolescent dentine profiles, we may be able to further establish when carbon and nitrogen isotopes are no longer a reflection of diet alone.

6.5.3 Identity and Kinship

Lastly, by expanding the methods used in this research, we can better understand both biological and social aspects of leprosy in the past. This study was restricted in its ability to apply amelogenin peptide analyses to five individuals from St. Mary Magdalen leprosy hospital. However, the limited data produced have shown that female adolescents entered the St Mary Magdalen leprosarium and were potentially accessing care there. By expanding this method to the adolescents at St. John at the Castle Gate/Timberhill, we can better ascertain who was developing leprosy within the Norwich community and whether sex-linked differences between St. John at the Castle Gate and St. Mary Magdalen exist.

If leprosaria were indeed community-based organisations with the possibility of familial presences, as Rawcliffe (2006:312-316) has suggested, it would be beneficial to explore potential kinship structures within leprosaria cemetery populations *per se*. St. Mary Magdalen offers a unique and discrete sample cohort to understanding whether familial relationships are evident within the leprosy hospital. This study expanded the female presence within the North Cemetery at St. Mary Magdalen to seven through peptide

analysis. The North cemetery also revealed the presence of two perinates at the time of study. aDNA analyses to consider whether a familial relationship exists between the females and the perinates at this site would enhance understandings of the nature of the lives of people in the leprosarium. This would include the possibility of children being born within the leprosarium. Investigating these perinates also using bacterial DNA analysis could further help us to know whether they were possibly born with leprosy. Leprosy rarely crosses the placental barrier (Melsom et al. 1980; Jopling and McDougal 1988:46-47; Butlin and Withington 2018) but combining kinship and bacterial DNA analyses holds the potential to elucidate other biological and social aspects of leprosy in the past. aDNA analyses can also reveal whether a familial component to leprosy susceptibility exists within communities in the past as well (Abel et al. 1998; Mira et al. 2004; Alcaïs et al. 2005; Alter et al. 2008). By looking at kinship relationships at St. John at the Castle Gate, we could access epidemiological insights that potentially indicate whether leprosy was restricted to specific familial groups or more widespread in the community.

6.6 Final Conclusions

Leprosy was a known condition in the Early Medieval period in England and the Early-Late Medieval transition (Anderson 1998; Duhig 1998; Roberts 2002, 2020; Roffey and Tucker 2012; Anderson 2014; Inskip et al. 2015; Inskip et al. 2017), and the notion that people with leprosy were stigmatised in the past is not supported by this research.

Strontium and oxygen isotope analyses combined with archaeological data from St. John at the Castle Gate (Norwich, Norfolk) show that the individuals with leprosy were not exiled from their local geographic origins, and were included within community cemeteries with normal burial customs (as noted by Roberts 2020 in her global view of archaeological skeletons with leprosy). Although this line of enquiry cannot tell us definitively how individuals with leprosy were treated in life as a direct consequence of their disease at this time, their isotope data and inclusion within their local parochial cemetery of St. John at the Castle Gate matches with the normative funerary identities for the Norwich Anglo-Scandinavian culture. This implies that their disease status did not exclude them from possessing a consistent cultural burial identity at the times of their

deaths. This may therefore represent a more localised community of inclusion or even an early community leprosarium for individuals suffering from the debilitating and disfiguring changes associated with leprosy in this time and space.

In contrast, the St. Mary Magdalen Leprosy Hospital in Winchester showed evidence for the care of both local and non-local individuals of both sexes. The presence of young adolescent females with advanced forms of leprosy challenges prior notions of the role of women in these institutions (Orme and Webster 2005: 20-22). It is likely these adolescent females were patients, and not care givers implying the early leprosarium was a mixed-sex care facility. This also challenges pre-conceived notions about the roles of the older females found in the cemetery. The ages of these women led archaeologists to believe they were present in the cemetery due to their possible status as care providers (*pers. comm* – Katie Tucker), however the presence of younger females with severe bone changes associated with leprosy questions this. Additionally, the identical isotope history (mobility and diet) of one of the young females (Sk. 52) with a similarly aged young male (Sk. 28) raises the possibility that not only were females present as patients, but that young inmates travelled to the hospital together from further afield. Although we are not able to determine the specific mobility drivers that brought these individuals to Winchester, it is likely some of the individuals migrated with leprosy after the disease began to take its hold. Therefore, we cannot exclude the possibility that some may have migrated to Winchester, the *de facto* capital of England during this time, in seek of medical care and treatment.

In further support of this view, carbon and nitrogen isotope analyses suggest the St. Mary Magdalen hospital provided a good quality diet, and applying the Index of Care framework to one individual suggests a significant amount of care was likely necessary to support individuals with leprosy buried there. Additionally, a reduction in dietary variability, particularly in the non-local individuals, allows us to approximate when these adolescents potentially entered the leprosarium. These results when combined with the archaeological evidence of mortuary treatments further supports the idea that these institutions were comparatively good places to be (as noted by Rawcliffe 2006), and individuals were afforded various levels of care. This supports the notion that life inside

the leprosarium was better, particularly for people who needed long-term medical assistance. Further, the clinical and archaeological evidence intimates that this institution, including its provisions and care, required significant community and broader social support to exist. Indeed this can be further evidenced by the number of leprosaria endowed during and immediately after the Norman Conquest.

Life history dentine profiles of adolescents buried at the St. Mary Magdalen leprosy hospital indicate early life nutritional and/or pathophysiological stress, which may have increased their susceptibility to leprosy at a younger age. Variations in $\delta^{15}N$ values at the end of life may reflect pathophysiological disruptions in the end stages of leprosy, and divergences in diet-tissue spacing in concurrently forming teeth may signal when leprosy begins to affect metabolic systems. Although the long incubation period associated with leprosy cannot be wholly pinned down, these disruptions to normal carbon and nitrogen metabolic pathways visible in incremental dentine profiles can help to verify when clinical signs and symptoms of leprosy are taking its hold on the body. Further, the timing of these disruptions, as approximated from the age of tooth development paired with the timing of specific pathological lesions (i.e. leprogenic odontodysplasia) allows us, in essence, to view the chronicity of the infection in these adolescents (i.e. how long they lived with the effects of the disease).

This research offers new, evidenced-based insights into the physiologies of people with leprosy in the past, and their potential care, that run counter to existing oft-cited narratives. This is particularly important given the negative attitudes towards modern-day people with leprosy that stem from unsupported Medieval perceptions, thereby demonstrating and confirming other research that there is archaeological, palaeopathological, and isotopic potential for dispelling harmful disease myths.

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APPENDIX:DATA

STRONTIUM AND OXYGEN DATA

NORWICH

SKELETON	SR PPM	$^{87}Sr/^{86}Sr$	$\delta^{13}C_{VPDB}$ (‰)	$\delta^{18}O_{(C)VSMOW}$ (‰)	$\delta^{18}O_{(P)VSMOW}$ (‰)	$\delta^{18}O_{DW}$ (‰)(Eqn 6)
13101	120	0.7098	-12.35	26.73	17.9	-6.1
13009	193	0.7095	-12.45	25.82	17.0	-7.6
11117	81	0.7090	-12.68	26.38	17.6	-6.7
11518	87	0.7100	-12.98	26.76	17.9	-6.1
13121	71	0.7096	-12.83	28.03	19.3	-4.1
11526	128	0.7097	-13.26	26.51	17.7	-6.5
13035	84	0.7090	-13.01	26.83	18.0	-6.0
13044	FAILED	FAILED	-13.22	26.98	18.2	-5.7
13146	100	0.7101	-13.44	26.31	17.5	-6.8
11287	84	0.7090	-12.61	26.25	17.4	-6.9

WINCHESTER

BURIAL	SEX	AGE	TOOTH	Sr PPM	$^{87}Sr/^{86}Sr$	$\delta^{13}C_{VPDB}$ (‰)	$\delta^{18}O_{(C)VSMOW}$ (‰)	$\delta^{18}O_{(P)VSMOW}$ (‰)	$\delta^{18}O_{DW}$ (EQN 6) (‰)
SK. 8	M**	DD: 8.5-9.5; SA: 7-8	L. Max. M2	81	0.7089	-12.37	26.67	17.9	-6.2
SK. 9	M	22.5-23.5	R. Max M2	102	0.7089	-12.65	26.25	17.4	-6.9
SK. 14	M	16.5-17.5	L. Max. M3	57	0.7085	-13.04	26.63	17.8	-6.3
SK. 15	M	20.5-22.5	L. Man. M3	96	0.7088	-12.88	26.77	18.0	-6.1
SK. 16	M	20.5-21.5	R. Max. M3	91	0.7086	-12.65	26.95	18.1	-5.8
SK. 18	M	DD: 18.5-19.5;	R. Man. M3	111	0.7093	-13.66	26.68	17.9	-6.2

		SA: <14							
SK. 21	M	DD: 21.5-22.5; SA: 16-19	L. Max. M3	82	0.7084	-13.2	26.42	17.6	-6.6
SK. 25	M	DD: 18.5-20.5; SA: 17-19	R. Man. M3	56	0.7091	-13.21	27.27	18.5	-5.3
SK. 26	M	22.5-23.5	L. Man. M3	99	0.7087	-12.58	27.59	18.8	-4.8
SK. 27	M	22.5-23.5	R. Man. M3	70	0.7103	-12.79	26.09	17.2	-7.2
SK. 28	M*	12.5-13.5	L. Max. M2	73	0.7094	-12.89	27.88	19.1	-4.3
SK. 29	M	18.5-19.5	R. Max. M3	41	0.7095	-13.85	26.83	18.0	-6.0
SK. 39	M	16.5-17.5	R. Max. M3	107	0.7102	-13.44	26.7	17.9	-6.2
SK. 41	M*	13.5-15.5	R. Max M3	89	0.7086	-12.57	26.5	17.7	-6.5
SK. 45	F*	DD: 15.5-16.5; SA: 10-12	L. Man. M2	111	0.7086	-12.83	26.32	17.5	-6.8
SK. 46	M	16.5-17.5	R. Max. M3	120	0.7090	-13.12	26.18	17.3	-7.0
SK. 52	F*	DD: 12.5-13.5; SA: 9-11	L. Man. M2	69	0.7099	-13.27	28.03	19.3	-4.1
SK. 54	M*	DD: 14.5-15.5; SA: 9-11	R. Man. M2	78	0.7085	-12.21	26.09	17.3	-7.2
SK. 56	M	16.5-17.5	R. Max. M2	94	0.7087	-12.55	24.49	15.6	-9.7

* Biological sex determined by Amelogenin Peptide Extraction; **Biological sex determined via aDNA analysis (Taylor et al. 2013); DD: Dental development age; SA: Skeletal age based on epiphyseal fusion

Carbon and Nitrogen Data

Winchester

Individual	**Tooth**	**Dentine Section Number**	**$\delta^{15}N_{AIR}$‰**	**Amt % N**	**$\delta^{13}C_{VPDB}$‰**	**Amt % C**	**C:N**
SK8	L Max C	1	nil	nil	nil	nil	nil
		2	7.8	10.8	-18.6	32.6	3.5
		3	8.5	12.8	-19.4	36.1	3.3
		4	8.7	18.7	-19.5	54.1	3.4
		5	9.4	15.2	-19.5	42.0	3.2
		6	9.8	12.8	-19.4	37.0	3.4

		7	9.9	11.5	-19.2	35.3	3.6
		8	10.7	12.4	-18.9	37.4	3.5
		9	10.4	11.9	-18.5	32.7	3.2
		10	10.6	12.1	-18.8	35.9	3.5
		11	10.2	7444.3	-18.7	21066.6	3.3
	L Max M2	1	9.6	13.8	-19.8	39.2	3.3
		2	9.5	12.1	-19.7	34.0	3.3
		3	9.3	15.0	-19.6	40.0	3.1
		4	9.4	14.8	-19.4	39.3	3.1
		5	9.6	14.8	-19.3	39.5	3.1
		6	10.0	14.9	-19.1	39.5	3.1
		7	10.6	14.9	-19.1	39.5	3.1
		8	10.8	15.6	-18.9	41.9	3.1
SK9	R Max C	1	15.6	18.2	-19.1	48.9	3.1
		2	13.7	18.1	-19.6	49.4	3.2
		3	12.5	18.0	-19.8	49.0	3.2
		4	12.5	18.3	-19.9	49.6	3.2
		5	14.0	18.3	-20.0	49.5	3.2
		6	14.6	18.2	-19.7	49.5	3.2
		7	13.3	18.2	-19.6	49.9	3.2
		8	12.3	14.3	-19.7	39.6	3.2
		9	11.9	14.6	-19.7	40.4	3.2
		10	12.1	14.7	-19.6	40.2	3.2
		11	11.9	14.6	-19.6	40.1	3.2
		12	11.5	14.6	-19.7	40.3	3.2
		13	10.9	14.8	-19.8	40.8	3.2
		14	11.0	14.6	-19.7	40.0	3.2
		15	11.1	14.7	-19.6	40.4	3.2
		16	11.5	14.6	-19.5	40.2	3.2
		17	11.6	14.7	-19.5	40.6	3.2
		18	11.7	14.4	-19.4	40.1	3.2
		19	12.1	14.6	-19.2	40.1	3.2
	R Max M2	1	12.5	14.8	-20.0	39.5	3.1
		2	12.2	11.1	-19.9	29.5	3.1
		3	13.5	15.3	-19.9	40.6	3.1
		4	14.3	15.8	-19.7	42.2	3.1
		5	13.4	15.2	-19.7	40.5	3.1
		6	12.4	15.3	-19.8	40.8	3.1
		7	12.2	14.5	-19.8	39.3	3.2

		8	12.2	15.1	-19.8	40.0	3.1
		9	12.1	15.2	-19.7	40.0	3.1
		10	11.7	14.8	-19.8	39.0	3.1
		11	11.5	15.2	-19.8	40.1	3.1
		12	11.5	14.6	-19.7	38.7	3.1
		13	11.6	11.3	-19.8	30.4	3.2
		14	12.2	14.7	-19.6	40.0	3.2
		15	12.5	14.2	-19.2	38.8	3.2
SK15	Man L C	1	11.6	13.5	-20.3	37.1	3.2
		2	10.2	13.5	-20.6	36.9	3.2
		3	10.2	13.6	-20.2	38.2	3.3
		4	10.2	13.3	-20.0	36.5	3.2
		5	10.5	13.6	-20.3	37.5	3.2
		6	10.3	13.5	-19.8	37.3	3.2
		7	10.1	13.8	-19.8	37.9	3.2
		8	10.3	13.1	-19.3	36.1	3.2
		9	10.6	13.6	-18.8	37.5	3.2
		10	11.0	13.5	-18.6	37.1	3.2
		11	10.8	13.5	-18.7	37.3	3.2
		12	10.4	13.2	-18.7	36.4	3.2
		13	10.5	13.6	-18.7	37.4	3.2
		14	10.2	13.5	-18.5	37.2	3.2
		15	10.1	13.2	-18.4	36.5	3.2
		16	9.8	11.4	-18.5	32.5	3.3
		17	10.0	11.4	-18.8	34.1	3.5
		18	10.5	12.3	-18.7	34.3	3.2
		19	7.6	9.4	-19.3	30.5	3.8
		20	10.8	10.8	-18.8	33.8	3.6
		21	10.3	13.7	-18.6	41.4	3.5
	L Man M3	1	11.3	15.0	-18.9	39.7	3.1
		2	11.7	15.0	-18.8	39.3	3.1
		3	11.6	14.9	-18.7	39.0	3.1
		4	11.6	14.7	-18.8	38.5	3.1
		5	11.8	15.0	-18.7	39.5	3.1
		6	11.6	15.1	-18.8	39.8	3.1
		7	11.8	13.3	-18.8	34.9	3.1
		8	12.0	13.6	-19.0	38.0	3.3
		9	11.6	14.2	-19.3	40.9	3.4
		10	11.7	14.3	-19.4	41.3	3.4

SK18	Man R C	1	14.3	12.2	-19.5	35.3	3.4
		2	14.4	12.7	-19.6	36.3	3.3
		3	13.6	12.8	-19.6	35.6	3.2
		4	12.3	12.9	-19.7	35.9	3.3
		5	10.7	13.0	-19.8	36.3	3.3
		6	10.7	12.9	-19.9	36.2	3.3
		7	10.2	12.8	-20.0	35.7	3.3
		8	10.3	13.1	-19.9	36.9	3.3
		9	10.6	13.1	-19.7	36.6	3.3
		10	10.2	13.0	-19.6	36.0	3.2
		11	9.6	13.0	-19.8	36.2	3.3
		12	9.6	13.1	-19.9	36.6	3.3
		13	9.4	13.0	-19.9	36.4	3.3
		14	9.7	13.0	-19.8	36.4	3.3
		15	9.9	13.0	-19.8	36.4	3.3
		16	10.0	13.0	-19.7	36.4	3.3
		17	10.5	12.9	-19.7	36.2	3.3
		18	10.7	12.8	-19.7	36.1	3.3
		19	10.5	12.6	-19.8	36.3	3.4
		20	6.4	8.1	-19.7	30.6	4.4
		21	9.4	11.4	-19.8	34.9	3.6
	R Man M3	1	10.9	11.7	-20.2	31.6	3.2
		2	10.9	14.3	-20.1	38.8	3.2
		3	11.4	14.7	-19.9	39.5	3.1
		4	11.1	14.4	-20.0	38.4	3.1
		5	10.4	13.4	-19.4	36.0	3.1
		6	10.5	10.6	-19.2	29.1	3.2
		7	11.0	14.2	-18.8	38.6	3.2
		8	11.3	11.0	-19.1	31.7	3.4
		9	11.5	12.9	-19.2	39.6	3.6
		10	12.2	12.4	-19.4	39.4	3.7
SK27	Man R C	1	10.6	14.0	-20.4	36.6	3.0
		2	12.9	14.5	-19.8	39.8	3.2
		3	12.2	14.5	-19.9	39.6	3.2
		4	10.9	14.5	-19.8	39.6	3.2
		5	10.7	14.5	-19.7	39.9	3.2
		6	10.3	14.6	-19.9	40.0	3.2
		7	9.7	14.5	-19.9	40.2	3.2
		8	9.2	14.7	-19.5	40.3	3.2
		9	9.5	14.6	-19.3	40.1	3.2

		10	9.4	14.5	-19.4	40.2	3.2
		11	9.8	14.6	-19.4	40.2	3.2
		12	9.8	14.5	-19.3	40.1	3.2
		13	10.1	14.6	-19.1	40.2	3.2
		14	9.9	14.4	-19.3	40.0	3.2
		15	10.1	14.5	-19.3	40.2	3.2
		16	10.2	14.5	-19.4	40.1	3.2
		17	10.4	14.3	-19.6	40.2	3.3
		18	10.5	14.1	-19.7	39.4	3.3
	R Man M3	1	10.2	11.8	-19.6	32.3	3.2
		2	10.4	14.6	-19.5	38.8	3.1
		3	10.6	14.1	-19.3	37.6	3.1
		4	10.4	15.0	-19.4	39.4	3.1
		5	10.3	13.9	-19.5	36.7	3.1
		6	10.3	14.6	-19.6	38.8	3.1
		7	10.6	13.2	-19.4	34.9	3.1
		8	11.0	14.6	-19.4	38.8	3.1
		9	11.4	14.5	-19.2	38.6	3.1
		10	11.2	9.3	-19.2	24.4	3.0
		11	11.6	14.4	-19.0	38.3	3.1
		12	11.5	12.3	-19.4	34.7	3.3
		13	10.9	8.3	-19.2	22.3	3.1
		14	11.7	14.3	-19.1	38.1	3.1
		15	11.8	7.5	-19.3	20.9	3.3
SK28	Max L C	1	10.6	16.3	-20.0	44.0	3.2
		2	11.4	17.3	-20.3	48.1	3.2
		3	11.6	17.7	-20.1	48.1	3.2
		4	11.7	18.1	-20.1	49.0	3.2
		5	10.5	18.1	-20.3	48.9	3.1
		6	10.3	17.9	-19.9	48.6	3.2
		7	10.6	17.6	-19.7	47.8	3.2
		8	10.2	17.8	-19.4	48.8	3.2
		9	9.9	18.0	-19.2	48.8	3.2
		10	9.5	17.8	-19.5	48.2	3.2
		11	9.4	16.2	-20.4	54.6	3.9
		12	9.7	17.8	-19.7	48.6	3.2
		13	9.7	17.8	-19.5	48.7	3.2
		14	10.0	17.8	-19.3	48.7	3.2
		15	10.5	17.5	-18.8	47.6	3.2
		16	10.4	16.7	-19.2	47.8	3.3

		17	10.9	13.0	-22.2	31.3	2.8
	L Max M2	1	10.7	13.4	-20.3	35.7	3.1
		2	10.3	14.6	-20.0	38.5	3.1
		3	10.0	14.6	-19.8	38.5	3.1
		4	9.9	14.4	-19.5	38.7	3.1
		5	9.6	14.7	-19.2	38.5	3.1
		6	9.4	14.5	-19.4	38.2	3.1
		7	9.5	15.7	-20.4	46.1	3.4
		8	9.9	15.1	-19.8	40.1	3.1
		9	9.9	14.6	-19.6	39.7	3.2
		10	10.3	14.5	-19.2	38.4	3.1
		11	10.7	14.9	-18.8	39.8	3.1
		12	10.6	14.9	-18.9	39.8	3.1
		13	11.0	14.1	-18.9	38.6	3.2
		14	11.1	14.3	-19.1	41.3	3.4
SK45	Man L C	1	14.5	17.5	-18.8	47.9	3.2
		2	13.6	17.5	-19.0	48.2	3.2
		3	13.5	18.0	-19.0	49.1	3.2
		4	12.5	17.9	-19.1	49.0	3.2
		5	11.2	18.2	-19.3	49.2	3.2
		6	10.7	17.8	-19.4	48.2	3.2
		7	10.0	18.2	-19.5	49.5	3.2
		8	9.6	14.9	-19.5	40.6	3.2
		9	9.4	18.2	-19.5	49.3	3.2
		10	9.3	18.1	-19.3	49.2	3.2
		11	9.7	18.0	-19.1	48.7	3.2
		12	9.6	18.3	-19.1	49.8	3.2
		13	9.8	18.4	-19.2	49.9	3.2
		14	9.9	18.0	-19.3	48.8	3.2
		15	10.2	17.8	-19.6	48.9	3.2
		16	10.3	17.5	-19.8	48.9	3.2
		17	10.1	16.4	-19.9	47.2	3.4
	L Man M2	1	10.1	13.9	-19.4	37.4	3.2
		2	9.5	14.4	-19.4	38.5	3.1
		3	9.3	6.1	-19.6	16.4	3.1
		4	9.5	13.2	-19.4	35.8	3.2
		5	10.0	13.9	-19.2	37.4	3.1
		6	10.1	13.5	-19.4	36.1	3.1
		7	10.4	14.1	-19.4	37.4	3.1

		8	10.6	14.4	-19.3	38.6	3.1
		9	10.9	14.4	-19.3	38.9	3.2
		10	10.7	11.6	-19.3	32.4	3.3
		11	11.0	12.6	-19.5	37.0	3.4
		12	10.6	11.3	-19.8	32.0	3.3
SK52	L Man C	1	13.6	14.5	-19.7	39.7	3.2
		2	13.6	14.5	-19.7	39.8	3.2
		3	11.7	30.4	-20.0	83.5	3.2
		4	10.8	14.5	-20.3	39.7	3.2
		5	10.7	14.5	-19.9	39.8	3.2
		6	10.5	14.2	-19.8	38.9	3.2
		7	10.3	14.5	-19.7	39.9	3.2
		8	10.6	14.8	-19.3	40.9	3.2
		9	10.6	13.6	-19.0	37.3	3.2
		10	10.4	14.3	-18.8	39.3	3.2
		11	10.2	14.4	-18.9	39.5	3.2
		12	10.1	14.7	-18.8	39.9	3.2
		13	10.0	14.2	-18.9	38.5	3.2
		14	10.5	14.6	-19.2	40.0	3.2
		15	9.8	13.8	-19.1	37.8	3.2
	L Man M2	1	10.9	14.5	-20.3	38.7	3.1
		2	10.4	14.6	-19.9	38.4	3.1
		3	10.3	14.8	-19.6	39.1	3.1
		4	10.6	14.5	-19.3	38.3	3.1
		5	10.7	14.4	-18.9	38.3	3.1
		6	10.6	14.7	-18.9	39.7	3.2
		7	10.7	14.7	-18.9	39.0	3.1
		8	10.9	15.2	-18.9	40.2	3.1
		9	11.3	14.7	-19.0	39.1	3.1
		10	11.6	14.7	-18.9	39.1	3.1
		11	11.5	14.5	-19.1	39.0	3.1
		12	11.1	15.0	-19.1	41.0	3.2
SK54	Man R C	1	17.7	8.7	-20.0	36.5	4.9
		2	10.8	14.6	-19.0	39.4	3.2
		3	10.0	14.6	-19.4	39.7	3.2
		4	9.6	14.6	-19.7	39.7	3.2
		5	9.7	14.5	-19.8	39.6	3.2
		6	10.0	14.5	-19.8	39.4	3.2
		7	9.7	13.8	-19.3	37.4	3.2

		8	10.1	14.6	-19.1	39.5	3.2
		9	10.1	14.2	-19.1	38.9	3.2
		10	10.1	14.6	-19.0	39.6	3.2
		11	10.3	12.0	-18.9	32.8	3.2
		12	10.2	17.4	-18.8	47.6	3.2
		13	9.9	13.0	-18.8	35.4	3.2
		14	10.3	15.0	-18.8	41.0	3.2
		15	9.4	54.4	-18.8	148.9	3.2
		16	9.9	15.1	-18.7	41.2	3.2
		17	9.9	15.6	-18.5	42.6	3.2
		18	9.7	15.7	-18.5	43.0	3.2
		19	9.5	15.2	-18.6	41.5	3.2
		20	9.9	14.4	-19.0	39.5	3.2
		21	9.0	5	-20.1	14	3.5
	R Man M2	1	10.0	14.3	-19.6	38.3	3.1
		2	10.0	14.5	-19.5	38.2	3.1
		3	10.0	14.7	-19.2	38.5	3.1
		4	10.2	14.6	-19.1	38.1	3.1
		5	10.1	14.1	-18.9	37.1	3.1
		6	10.3	14.9	-18.7	38.9	3.0
		7	10.3	13.5	-19.0	38.8	3.4
		8	10.3	13.5	-18.4	35.6	3.1
		9	10.1	13.8	-18.3	36.2	3.1
		10	10.5	7.0	-19.2	21.5	3.6
		11	10.4	13.1	-19.4	38.9	3.5
		12	10.6	13.7	-19.4	39.3	3.4
		13	11.0	13.6	-19.6	39.7	3.4
SK56	Max R C	1	13.0	6.1	-18.9	17.1	3.3
		2	12.7	14.0	-18.8	38.4	3.2
		3	12.0	15.5	-18.8	42.6	3.2
		4	11.8	16.0	-18.6	43.8	3.2
		5	11.2	12.0	-18.9	32.9	3.2
		6	10.9	9.3	-18.9	25.4	3.2
		7	10.3	16.9	-18.9	46.6	3.2
		8	10.4	15.7	-18.9	43.3	3.2
		9	10.7	17.8	-18.8	48.7	3.2
		10	10.7	17.0	-18.9	46.7	3.2
		11	10.6	14.7	-18.9	40.3	3.2
		12	10.9	14.7	-18.8	40.5	3.2
		13	10.5	13.8	-19.0	38.2	3.2

	14	10.6	13.2	-18.8	36.1	3.2
	15	10.6	14.9	-18.8	40.9	3.2
	16	10.7	13.9	-18.8	38.2	3.2
	17	11.0	9.6	-18.7	26.5	3.2
	18	11.1	13.1	-18.8	35.8	3.2
R Max M2	1	11.5	14.4	-19.0	42.1	3.4
	2	11.7	14.9	-18.7	40.6	3.2
	3	10.8	14.5	-18.8	39.7	3.2
	4	10.5	14.7	-18.7	39.9	3.2
	5	10.8	15.0	-18.7	40.7	3.2
	6	10.9	14.5	-18.7	39.2	3.2
	7	10.8	14.8	-18.6	40.0	3.2
	8	11.0	13.7	-18.6	37.8	3.2
	9	11.2	14.6	-18.6	39.3	3.1
	10	11.5	14.8	-18.4	40.1	3.2
	11	11.6	14.6	-18.5	40.0	3.2
	12	11.3	14.3	-18.6	39.1	3.2
	13	11.0	15.1	-18.8	41.4	3.2
	14	10.9	13.7	-18.9	38.8	3.3
	15	10.7	12.9	-19.2	38.5	3.5