An Isotopic Study of Environmental Influences on Early Anglo-Saxon Health and Nutrition

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ABSTRACT

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Ellen Jean Kendall

Early life has long been associated with high vulnerability to morbidity and mortality, risks which are reduced in infancy and early childhood through strategically high levels of parental or alloparental investment. More recently, theories have emerged addressing the manner in which poor health and nutritional stress during early life may impact upon the future health of those who survive this period. Thus, the significance of early life health and parental care extends far beyond the domain of childhood studies and may provide insight regarding population-level biocultural responses to environmental pressures throughout the lifecourse.

Skeletal and environmental data indicate that the 5-7th century cemeteries at Littleport and Edix Hill (Barrington A), Cambridgeshire represented similar communities with contrasting environments and states of population health. High prevalence of skeletal stress markers at Littleport suggests a community coping with unusual levels of biological stress, potentially a consequence of the endemic malaria historically known to be present in the Fens. In contrast, Edix Hill was an upland site which exhibited lower skeletal stress marker prevalence comparable to wider British data for the early medieval period. Early life stress levels and nutrition at Littleport (n=30) and Edix Hill (n=29) were investigated through carbon and nitrogen stable isotope analyses from incrementally-sampled deciduous and permanent molar dentine to identify variability in patterns relating to survivorship, sex, status, and disease. Meaningful variation in isotopic values within and between populations and sub-cohorts was observed. Isotopic data for Littleport demonstrated patterns of recurrent nutritional stress and health inequality between subpopulations during childhood, which were not mirrored at Edix Hill. These patterns are interpreted as consistent with the presence of malaria in the early medieval Fens. Characterisation of such inter-individual and inter-population variability should be a focus of future interdisciplinary archaeological childhood studies.

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DECLARATION

I, Ellen Jean Kendall, confirm that the work presented in this thesis is my own. Where information has been derived from other sources, I confirm that this has been indicated in the thesis.

STATEMENT OF COPYRIGHT

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For Ross, Lucie, and Elias

"I have learned that to be with those I like is enough."

1. Introduction

This study intends to provide insight into early medieval childhood health in two populations which appear to have experienced differing types and degrees of threats to health. The effects of diet and health in early life, particularly during gestation and the first few years, are a profound predictor of health throughout the lifecourse (Wadhwa *et al.*, 2009; Godfrey *et al.*, 2010). Understanding of this formative period is of paramount importance for understanding larger population dynamics, and for assessing the impacts of local disease ecology. Breastfeeding is perhaps the most crucial adaptive behaviour of early life, not only to immediate child survival, but to reduction of long-term disease risk for both mothers and infants (Gluckman *et al.*, 2008; Stuebe, 2009). The wetland Fens of eastern England present an intriguing case study for the reciprocal interaction between breastfeeding behaviours and disease ecology writ large, having a long-standing reputation for malarial insalubrity and extreme infant mortality which extends into the poorly-documented early medieval period (West, 1973; Sneddon, 2006; Noetzel, 2014). Accordingly, this study hopes to illuminate the specific concerns associated with the diet, health, and development of medieval Fenland children, especially the potential presence of endemic malaria, and to add to the limited store of extant knowledge on health in this time and place.

1.1 Background and topical context

The Fens comprise a vast area of former English wetland within a post-glacial basin occupying much of Lincolnshire and Cambridgeshire, and parts of Norfolk and Suffolk (Clayton, 2000). Prior to their drainage in the 17th to 19th centuries, the English Fens represented a unique and vibrant ecosystem, being the largest area of natural wetland in Britain, with distinct zones of silt with marine influence and river-fed freshwater peat fen (Sturt, 2006). The complex and co-evolving relationship between the landscape and its human inhabitants over time created a juxtaposition of diverse issues such as otherness and insularity, endemic disease and material wealth, and piety and nonconformity (Rippon, 2009). Despite this unique character and the widely acknowledged role of eastern England as the early focus of population movement and the purported *Adventus Saxonum*, the people of the early medieval Fens themselves have remained somewhat mysterious. Use of the term "Anglo-Saxon" in the present study is not intended to imply ethnic origin and is used as a descriptor of chronological

period only. A paucity of excavated early medieval settlement sites, and variable skeletal preservation due to the ubiquitous issues of groundwater and soil acidity, has certainly led to a greater emphasis on place than on people. Fortunately, publications within the last decade have begun to address the importance of the Fens for understanding human-environment interactions (Rippon, 2009; Oosthuizen, 2016; Ash, 2017; Huisman, 2017).

Long viewed as a marginal and fearsome environment of miracles and monsters by outsiders, the legacy of the Fens as a place "as richly English as any other part of England…also compellingly and hauntingly *strange*" was perceived as late as the 1980s (Swift, 1985: 7). Little is known about perceptions of the Fens, or of Fenlanders themselves, in the early post-Roman period. However, by the 7th and 8th centuries AD, the Fens had begun to draw pious settlers such as Guthlac, Pega, Etheldreda, and Wendreda to found hermitages and monastic houses within the Fens (Farmer, 1985). Chroniclers of these religious incursions, such as Felix, were predominantly negative in their depictions of the Fens (O' Sullivan, 2013). Writing in 1940, H.C. Darby uncritically stated that

> ...when St. Guthlac came to Crowland, he found the place infested with devils of various kinds. The incredible nature of these tales is irrelevant to the argument; the spirit which created them is an indisputable fact. They show something of what the barrier of the fens meant in the lives of people at this time. Only familiarity with the region in its most sombre aspects can do justice to the fears of these early settlers. Many centuries did not assuage their horror; and at last the horror of the fen passed into tradition – so deeply was it grounded in the Saxon mind. Nor were all the horrors imaginary. they had indeed a very substantial foundation; for the Fenland was a pestilential place "oft-times clouded with moist and dark vapours ...Ague and malaria with their hallucinations made the life of the fenman very miserable; and it is little wonder that St. Guthlac "was greatly troubled within him about the undertaking he had begun, namely to dwell there alone in the wilderness" (Darby, 1940: 8-9).

Stenton (1971: 50), like Darby, also depicts the early medieval Fen as a vast frontier which prevented the expansion of Mercian hegemony through its vacant impenetrability. The description of the Fenlands as a place of emptiness, pestilence, and horror is somewhat at odds with the alacrity with which religious pilgrims colonised the Fens to found monastic houses. Furthermore, archaeological evidence refutes the suggestion by early chroniclers that these ecclesiastical opportunists arrived in an unpopulated and barren landscape. Significant levels of continuity in settlement are demonstrated for the silt Fens and the islands of the peat Fens between the Roman and

post-Roman periods, with additional evidence for a prosperous economy based on fen resources and a system of common grazing rights. (Oosthuizen, 2011; Oosthuizen, 2016).

How should a modern observer reconcile these divergent and mutually-exclusive views: that the Fens were a sparsely-populated and disease-ridden landscape, versus a prosperous and well-populated region which drew many non-local settlers due to its natural resource wealth? For the period of drainage, colonising narratives by non-native Fenlanders intent on transformation of the landscape can be paralleled by the rationales used by British colonisers of foreign lands, and thus these descriptions and characterisations are acknowledged to be suspect by several researchers (Lindley, 1982; Evans, 1997; Butlin, 1998). Should early medieval accounts of the Fens also be approached with caution? The answer to this question is almost certainly yes. Rippon (2009) notes the disconnect between religious depictions of marshlands and the archaeological evidence for exploitation of the rich resource base of the Fens. He also points out the rhetorical advantages of portraying the Fens as a deserted wasteland, creating an ideal backdrop for Christian asceticism and miracle stories, while in practice enjoying the wealth generated by establishment of estates in wetland areas (Rippon, 2009). We must therefore assume that the colonising narrators of both the early medieval and post-medieval periods share some part of this agenda in common. This narrative is unlikely to reflect the identity and perceptions of landscape held by Fenland-born communities. Furthermore, the early medieval accounts of fevers such as malaria within the marshes at this time demand a population density sufficient to support endemicity. Thus, we must take a critical view to understanding the lives of early medieval Fenlanders.

While taking with some caution the accounts of religious chroniclers in regard to the nature of the landscape and its people, the issue of malaria must nonetheless be grappled with. *Plasmodium vivax,* a tertian form of relapsing malaria, was first positively identified in British populations via blood microscopy in the late 19th century, though it was known as "marsh ague" or simply "marsh fever" for centuries preceding this. *P. vivax* is a less virulent parasite than its close relative *P. falciparum* but can be deeply debilitating through its ability to hibernate in a dormant state within the human liver, re-emerging seasonally to cause relapsing infections and anaemic states. It is unknown when malaria first arrived on British shores, but descriptions in the 10th century of

"*lencten adl*", a vernal febrile illness, in Bald's Leechbook (cf. Cockayne, 1865) have long been thought to be consistent with the seasonal pattern of *P. vivax* infection. A study by Gowland and Western (2012) analysed geographic distributions of cribra orbitalia, a skeletal lesion commonly associated with anaemias, in early medieval archaeological populations throughout eastern England. This data was compared with historically-documented distributions of the anopheline mosquito vector, topographical features of the landscape, and reported prevalence of ague. A significant correlation was found between cribra orbitalia and historically low-lying, marshy locales offering prime habitat to mosquitoes. The authors consequently hypothesised that endemic malaria played a causal role in the observed geographic distribution of cribra orbitalia.

P. vivax infection is disproportionately deleterious to the health of pregnant women and young children, due to the high demands of growth and suboptimal immune function of both groups. Breastfeeding and weaning patterns represent an important area of study for understanding the health of these vulnerable groups, as breastfeeding exerts a profound effect on the long-term health and demography of human populations, both past and present (Dettwyler and Fishman, 1992; Jackson and Nazar, 2006; Horta *et al.*, 2007). While breastfeeding should be viewed as a complex series of negotiations between mother and child, cultural norms and family needs, local environment and ecology are powerful influences on infant feeding practices and should be included in a holistic model (Moffat, 2001). Following the critique of Temple and Goodman (2014), use of the term "stress" is here meant to indicate threats to physiological homeostasis, with "health" representing a broader, more overarching state of wellbeing. These terms are not interchangeable, as skeletal markers of stress may indicate adaptation, and paradoxically, good health (Wood *et al.*, 1992). Skeletal and environmental data indicate that the 5th to 6th century cemeteries at the Fen island of Littleport and the upland site at Edix Hill (Barrington A), Cambridgeshire represent two populations of similar material culture but contrasting environments and health. A stark disparity in the prevalence of skeletal stress markers, particularly cribra orbitalia (Malim *et al.*, 1998; Gowland and Western, 2012), between the two sites supports the depiction of Littleport as a site coping with high levels of biological stress, potentially a consequence of endemic malaria due to the marshy environs. In contrast, Edix Hill appears to have suffered fewer challenges to skeletally-reflected health, having lower-than-average prevalence of pathology and stress markers for the period, and therefore serves as an

ideal foil to Littleport. Due to these differences, it is hypothesised that breastfeeding duration would be more variable, and potentially longer, at Littleport than at Edix Hill in response to the specific disease burden in the Fens. Working from the hypothesis of Gowland and Western (2012) that high cribra orbitalia prevalence is predictive of the presence of endemic malaria in eastern England, this study intends to investigate the role of environment and disease ecology in influencing infant diet and maternal and childhood health in two early medieval populations using carbon and nitrogen stable isotope analysis.

1.2 Research aims and objectives

Aims:

- To investigate whether environmental disease burdens in the Fens, suggested by skeletal markers of stress, produced meaningful differences in breastfeeding behaviours between and within the communities at Littleport and Edix Hill
- To investigate the possible impacts of malarial infection on mothers and children at Littleport

Objectives:

- To identify the duration and pattern of phases of infant feeding (exclusive breastfeeding, complementary feeding, and termination of breastfeeding) through high-resolution carbon and nitrogen isotope analyses of incrementally-sampled permanent first and second deciduous molar dentine and the construction of individual-specific isotope profiles
- To identify the range of meaningful inter-individual and inter- and intra-group isotopic variability, particularly:
    - between populations, due to differing strategies in higher and lower risk environments
    - between males and females, thus potentially identifying previously obscure sex-differences in feeding strategies in early infancy
    - between individuals dying during childhood and individuals who survived to adulthood, highlighting potential differences in frailty

- between individuals who exhibit skeletal stress markers and those who do not

- To align isotopic data with the known pathophysiology of *P. vivax* infection to attempt to interpret characteristic longitudinal data shifts consistent with malarial infection in children and pregnant women.

1.3 Thesis structure

The second chapter of this thesis will explore theoretical principles underlying isotope biogeochemistry and an overview of applications to human palaeodietary research. It offers a full review of previous research on infant palaeodiet, from early forays into trace element analyses of bone to the present high-resolution analyses of dental tissues. This chapter also identifies gaps in the current state of knowledge regarding the physiological factors influencing isotopic patterning and suggests directions for future research which will enrich our understanding of early life diet and health.

Chapter Three is entitled "Biological Impacts of Breastfeeding". This chapter critiques a mismatch between clinical lactation research and assumptions prevalent in current infant palaeodietary studies, arguing that a robust understanding of clinical and anthropological literature on the biological norms and cross-cultural variability of breastfeeding are an essential prerequisite to interpreting infant palaeodietary data. Consequently, it presents an extensive summary of lactation physiology, biobehavioural dynamics, the role of breastfeeding in immune ontogeny and long-term host defence, and the role of nutrition throughout the complementary feeding period.

The fourth chapter, "Childhood in the Early Medieval Fens," surveys relevant evidence for the conditions under which children in the early medieval Fens may have been born, cared for, and raised. Care is here broadly defined as provision for another of what is necessary for health and welfare (Southwell-Wright *et al.*, 2016), which would include common child-rearing behaviours necessary to survival such as breastfeeding, hygiene, clothing and housing, as well as more ephemeral behaviours necessary to thriving, such as education and affection. This chapter will review environmental, historical, and archaeological evidence for the lives of children in the early medieval period. It will also present evidence for the disease ecology of the pre-drainage Fens, with a special emphasis on endemic malaria and clinical evidence for its pathophysiological effects on pregnant women, infants, and children.

Materials and Methods, the fifth chapter, will provide background information about the setting and history of at Littleport and Edix Hill. Further, it will provide a rationale for selection of individuals at the sites, and a range of background information about the burial context and osteobiography of the sampled individuals. It also outlines methods used in the study for the sampling, preparation, and analysis of dentine. Finally, it includes technical specifications for mass spectrometry and statistical methods employed in the analysis of data.

The results chapter will follow as the sixth chapter and will present the data obtained through analyses of all individuals sampled. Data in this chapter will be reported exhaustively, aiming to provide biographical data for individuals, as well as in aggregation based on cohort membership by site, sex, and age-at-death.

The discussion (Chapter 7) will interpret the dentine data from the results chapter preceding and contextualise the findings with comparative regional and national bone and dentine data for the early medieval period. It will evaluate the data obtained in light of known clinical, anthropological, and archaeological data, and assess the import of findings to the research questions and hypothesis.

The conclusion to the thesis will review the purpose and findings of the study, identify its contributions to the field, and underscore inherent limitations to interpretation. It will also make recommendations for future isotope or bioarchaeological research which could help to clarify issues around infant feeding research generally, and specifically produce greater contributions to understanding wetland human disease ecology in a British context.

2. Isotopes and Breastfeeding Studies

2.1 Introduction

This chapter aims to broadly explore the principles of isotope biogeochemistry and their applications to the study of ancient human diet and health. Despite the importance of mobility studies to the contextualisation of dietary data, the analysis of isotopes used in the estimation of geographical origin are outside of the scope of this thesis and will not be discussed at any length. The focus of this chapter will remain on familiarising the non-expert reader with the theory and research context of palaeodietary methods drawn upon in this study. Consequently, this chapter aims to review the use of isotope biochemistry for the identification of the diets and health of children in the past, with a particular focus on applications of carbon ($\delta^{13}C$) and nitrogen ($\delta^{15}N$) stable isotope biogeochemistry.

2.2 Basic principles of isotope biogeochemistry

In order to understand palaeodietary method, it is first important to understand the isotopic systems which form the foundation of this type of research. Isotopes are variations in the atomic composition of an element. While the number of protons present, or atomic number, defines the identity of the element and does not vary within an element, the number of neutrons may vary, producing variation in the mass number (determined by the number of protons and neutrons in the nucleus) of an element (Gross, 2004). These variants in the mass number of elements define their isotopes, which are known by their mass number (Brown and Brown, 2011, p. 80). For example, the three naturally-occurring variants of carbon have mass numbers of 12, 13, and 14; giving rise to carbon-12, carbon-13, and carbon-14, respectively known as ^{12}C, ^{13}C, and ^{14}C.

Some isotopes are radioactive, and undergo a process of measurable decay, such as ^{14}C. Carbon-14 decays during the life cycle of organisms but is maintained in equilibrium with atmospheric values through replacement, and its known rate of decay following the death of an organism is utilised in radiocarbon dating (Taylor, 2001). Other isotopes are stable, or not subject to a process of decay. Stable isotopes may be further subdivided into radiogenic and non-radiogenic isotopes. Radiogenic isotopes are produced by the decay of radioactive isotopes and have multiple applications in archaeological and natural sciences (Beard and Johnson, 2000). An example of this

would be radiogenic strontium-87, an alkali earth metal which is produced by the decay of radioactive rubidium-87 (Sealy *et al.*, 1995). Relative abundance of ^{87}Sr to ^{87}Rb is used in the dating of geological formations, while the distinctive relationship between the ratio of radiogenic ^{87}Sr to non-radiogenic ^{86}Sr and the geography of underlying geology is frequently employed in archaeological studies of human or animal mobility (Bentley, 2006).

Stable isotopes may also be described as "light" or "heavy", with differences in mass having a significant and direct effect on the properties and behaviours of isotopes within biological systems and their chemical processes (Brown and Brown, 2011: 81). Change in the relative abundance, or ratio, of one isotope to another during chemical reactions is known as *fractionation*. Equilibrium fractionation occurs when isotopes are exchanged between substances in chemical equilibrium, and the importance of temperature to equilibrium fractionation has been exploited in reconstruction of palaeoclimatic conditions (Sharp, 2007: 14). Kinetic fractionation involves unidirectional movement of isotopes, with a preference towards reaction of lighter isotopes over their heavier counterparts due to the greater readiness of lighter isotopes to break their chemical bonds. This generally results in a measurable enrichment of the lighter isotope in the product of the reaction, relative to the original substrate (Brown and Brown, 2011: 81). The principles of kinetic fractionation in living organisms form the basis of palaeodietary studies in archaeology, with fractionation of carbon and nitrogen isotopes being the pertinent process for the current study.

2.2.1 The role of carbon and nitrogen stable isotopes in foodwebs

An understanding of the effects of kinetic fractionation on organisms past and present, and the isotopic composition of their tissues, is essential to the reconstruction of past foodwebs, and to the contextualisation of human and animal behaviour within those foodwebs. While proportional abundance of isotopes in natural reservoirs is well-characterised, the behaviours of isotopes within biological systems, which create the isotopic variability exhibited within ecosystems, are governed by the chemical properties of those isotopes within the context of the environmental niches occupied by living organisms (Smith, 1972). In biological systems, the relative enrichment of lighter isotopes which occurs as a product of kinetic fractionation produces measurable change from the characteristic ratios of baseline environmental reservoirs, with increasing stepwise fractionation occurring at each hierarchical stage of consumption in an

ecosystem, or trophic level. Thus, the isotope ratios of organisms along the food chain are determined by both the ratios of their environmental inputs, and further fractionation resulting from *in vivo* metabolic activity (Minagawa and Wada, 1984). Identification of isotopic patterning in ancient tissues may be subsequently used as a direct "natural tracer" of diet but may also be used to infer indirect implications of diet, such as landscape use and resource management (Sealy, 2001, p. 269).

The processes of photosynthesis and tissue synthesis in plants and animals alter existing environmental proportions of carbon and nitrogen isotopes through varying mechanisms along the food chain. Knowledge of the natural proportional abundance of carbon and nitrogen isotopes presents a baseline against which deviation in living organisms from natural abundance may be quantified as a measure of fractionation. The lighter stable isotopes of both carbon and nitrogen are both more naturally abundant. The lighter isotope ^{12}C represents 98.93% of carbon atoms, with 1.07% of carbon being ^{13}C, and ^{14}C present at only one part per trillion (de Laeter *et al.*, 2003). For nitrogen, ^{14}N accounts for 99.64% of all nitrogen atoms, while ^{15}N only represents 0.36% of nitrogen (Bowen, 1994).

Analysed stable isotope compositions are expressed as deviations from known natural abundance in an internationally-agreed reference standard, which for carbon is the marine fossil Peedee Belemnite (PDB), now replaced by the equivalent Vienna PDB (VPDB) due to depletion of PDB (Hut, 1987). PDB/VPDB, as the standard, has a $\delta^{13}C$ of zero, resulting in negative relative values for most naturally-occurring materials (van der Merwe, 1982). Variation from the standard values is notated as a delta value (δ) expressed in parts per thousand (per mil, or ‰), which for carbon is calculated as:

$$\delta^{13}\text{C}\ (‰) = \left(\frac{R_{sample}}{R_{standard}} - 1\right) \text{x } 1000$$

where *R* represents the isotope ratio $^{13}C/^{12}C$ (Coplen, 2011). As δ values are measures in the relative proportions expressed in the ratio of $^{13}C/^{12}C$, increases in this value denote increases in the heavier isotope ^{13}C, while decreases in this value denote relative depletion of ^{13}C (Peterson and Fry, 1987). Measurements for nitrogen are reported similarly:

$$\delta^{15}\text{N}\ (‰) = \left(\frac{R_{sample}}{R_{standard}} - 1\right) \text{x } 1000$$

where *R* represents the isotope ratio $^{15}N/^{14}N$. For nitrogen the reference standard is atmospheric N_2 (AIR) (Sealy, 2001).

2.2.2 Trophic level shifts

The isotopic composition of human and animal tissues is shaped by the foods that they consume, in what has been colloquially termed the "you are what you eat, plus a few per mil" principle (DeNiro and Epstein, 1976). While simplistic acceptance of this principle has been greatly questioned in favour of acknowledging a more nuanced and complex relationship between diet and isotopic composition of organisms (Schoeller, 1999; Fuller *et al.*, 2004; Fuller *et al.*, 2005), a general correlation is seen between trophic level and isotopic enrichment of heavier isotopes in animals, with higher $^{15}N/^{14}N$ and $^{13}C/^{12}C$ ratios seen in carnivores than in herbivores, which in turn have higher $^{15}N/^{14}N$ and $^{13}C/^{12}C$ ratios than the plants that they consume (Sealy, 2001). Processes of fractionation in plants and animals differ in mechanism and pathway between elements creating variation in the directionality and magnitude of isotopic shifts. There is great variation in the carbon isotope ratios among plants, with only modest increases occurring with increasing trophic level; while significant variations are seen between soil, plant, and animal nitrogen isotope ratios, with greater trophic level increases in evidence among animals (Ambrose, 1993; O'Connell *et al.*, 2012). Figure 2.1 shows a simplified model of stable carbon and nitrogen isotope ecosystem distributions.

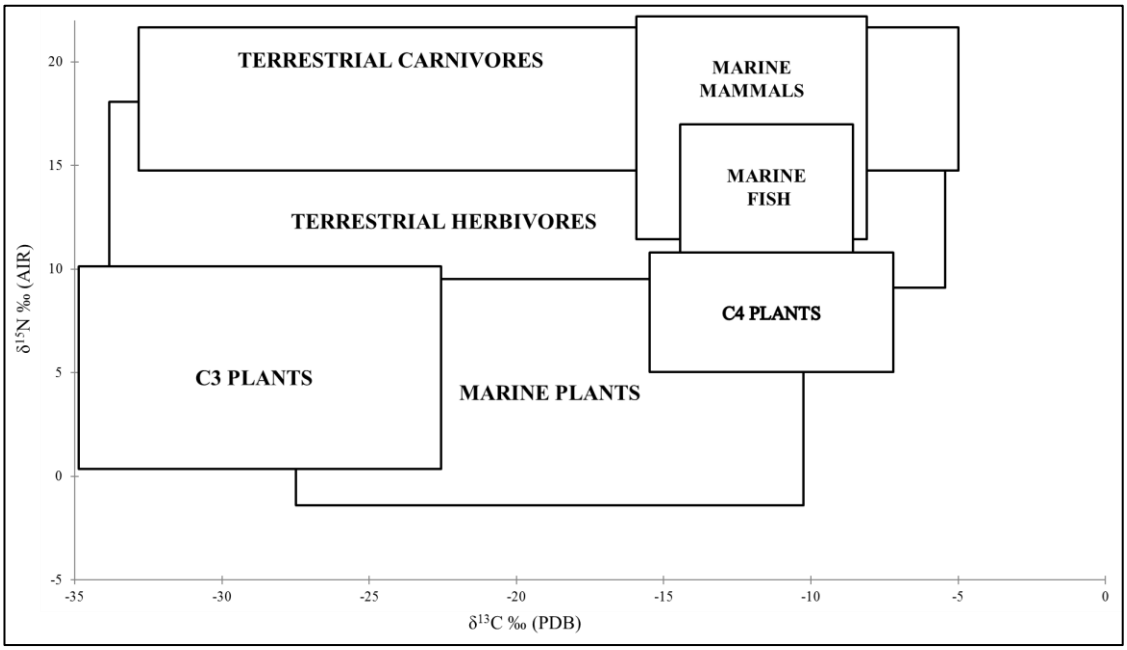

Figure 2.1. Simplified stable carbon and nitrogen isotopes ecosystem distribution (adapted from (Ambrose, 1993, p. 87).

In animals, trophic level effects from herbivores to carnivores are estimated to amount to a shift of +0.5 to 2‰ for carbon and +3 to 5‰ for nitrogen (van Klinken *et al.*, 2000; Wada *et al.*, 2013). Differences between diet and tissue isotope ratios, known as diet-tissue spacing, commonly notated as $\Delta_{diet\text{-}tissue}$, are highly variable not only between species within a shared environment, but also between tissues within a species (DeNiro and Epstein, 1981). Despite the fact that the carbon and nitrogen isotope composition of an animal's tissues are directly influenced by the diet of that animal, different tissues from a single organism produce variable isotopic values (Lee-Thorp *et al.*, 1989). Differences in isotopic composition between tissues within an organism and between organisms sharing a common diet can be understood as reflecting differential routing of dietary components (i.e. proteins, lipids, carbohydrates) as well as metabolic processes which may introduce fractionation of dietary isotope inputs (DeNiro and Epstein, 1981; Ambrose, 1993). These dietary inputs become degraded into their component substances – carbohydrates are broken down into sugars, proteins into amino acids, and fats into glycerol and fatty acids – which may be preferentially routed into varying metabolic pathways (Schwarcz, 2000). Considerable variability is observed in the isotopic composition of individual amino acids, which are drawn from dietary proteins and are allocated within the body to the formation of new tissue proteins (Macko *et al.*, 1987; Hare *et al.*, 1991).

Amino acids may be classified as essential or non-essential. Essential amino-acids cannot be synthesised by the body and must be obtained through diet, while non-essential amino-acids may be either synthesised *in vivo* from biochemical antecedents or drawn directly from the diet (Schwarcz, 2000). While the isotopic composition of essential or non-essential amino-acids which are acquired directly from food may undergo very little modification within the body, the synthesis of non-essential amino-acids within the body from dietary precursors is thought to introduce fractionation leading to tissue enrichment (Ambrose, 1993). The unique characteristics of different amino acids influence how isotopes are selected for or discriminated against during the processes of synthesis and transamination, which in turn alters the subsequent isotopic composition of proteins. Thus, the relationship between the isotopic composition of animal tissues and diet is complex. Variations in the isotope composition of tissues, specifically collagen, will be discussed at greater length below.

2.2.3 Isotopic variability and the biochemistry of collagen

The variability in diet-tissue spacing observed not only between species sharing the same diet, but also between tissues within a single organism has thus been interpreted as occurring due to preferential routing of various components of the diet, in the case of carbon. As protein represents the only significant source of dietary nitrogen, the assumption that collagen straightforwardly represents the proteinaceous portion of the diet is more likely to be valid, unlike carbon, for which the dynamics are more complex (Richards and Hedges, 1999). Variability in spacing between the isotopic compositions of tissues within an organism is referred to as an isotopic offset. Offsets are not unique to animals; plants have also been demonstrated to be anatomically variable in their biochemical composition, with significant variability observed between whole plant, leaf, and seed in carbon isotope analyses (Ambrose and Norr, 1993). However, the issue of diet to tissue offsets in animals has produced a large body of studies attempting to characterise, quantify, and model these offsets, which are isotope-, species- and tissue-specific (e.g., Jim *et al.*, 2006; Hedges and Reynard, 2007; O'Connell *et al.*, 2012; France and Owsley, 2015). For carbon and nitrogen stable isotopes, keratinised tissues such as hair, nails, or claws, as well as collagen present in bone or tooth dentine are believed to accurately reflect protein inputs within the diet (Chisholm *et al.*, 1982; Ambrose and Norr, 1993). Similarly, the mineral apatites of bone and teeth have been demonstrated to reflect carbon isotopic inputs which are representative of the whole diet (Jim *et al.*, 2004). Regardless of species, adipose tissues generally are 5 to 8‰ lower than average whole-organism values for $\delta^{13}C$, while bone mineral $\delta^{13}C$ values are conversely 3 to 10‰ higher than those of collagen, demonstrating the need to appropriately isolate and purify the desired biochemical fraction of tissues prior to analysis (Ambrose, 1993).

Recognition of tissue offsets is integral to ensuring the validity of comparative measures of isotopic composition in living organisms. While ecological studies may be required to quantify offsets between muscle, blood plasma, hair, bone, claw, or teeth, in archaeological analyses, the tissues available for analysis are rarely so diverse. In practical terms, this means analyses of ancient tissues will nearly always consist of bone, teeth, or in the rarer case of mummification or serendipitous preservation, hair or nails. While the limited range of tissues typically present in the archaeological record is less challenging than the wider range of tissues analysed in ecological studies, the

discrepancy between tissues commonly surviving within the archaeological record and tissues accessible to researchers through ethical means in modern human populations creates obstacles to clear and full understanding of the processes which alter and integrate isotopic input from the diet into human tissues. While fingernails and hair are the most common analytical targets in modern human studies and are known to reflect dietary protein inputs, offsets between diet and keratin and diet and bone or tooth collagen differ, due to differences in their amino acid composition (O'Connell *et al.*, 2012).

Researchers have attempted to reduce uncertainties surrounding tissue offsets in human remains through archaeological and modern multi-tissue analyses. O'Connell and Hedges (1999b) compared paired archaeological bone collagen and hair keratin data from 23 individuals, finding that bone collagen values were higher by approximately +0 to 1‰ for $\delta^{13}C$ and +0 to 2‰ for $\delta^{15}N$, relative to hair keratin values. This study acknowledged inherent limitations to interpretation imposed by the scope of unknowns for archaeological samples. The issue of collagen-keratin tissue offsets was revisited by O'Connell *et al.* (2001) in a study comparing modern bone collagen, hair keratin, and nail keratin of living people of known health and diet. The study found that comparing bone collagen data to hair keratin data for the same individuals reflected consistently higher values for bone collagen over hair keratin: the authors reported that collagen $\delta^{13}C$ was +1.4‰ higher than hair keratin $\delta^{13}C$, while bone collagen $\delta^{15}N$ was +0.86‰ higher than hair keratin values for the same individuals. Even within keratinised tissues there is some variation, as the same study found no offset for $\delta^{13}C$ between hair and nails, but alternately found that $\delta^{15}N$ for nail keratin values was higher by +0.65‰, relative to hair keratin (O'Connell *et al.*, 2001). As bone and dentine survive more frequently in the archaeological record than the hair or fingernails for which modern data exists, interpretation of past resource exploitation from the known (diet-keratin spacing) to the inferred (diet-collagen spacing) must take account of these differences.

Collagen is the most abundant protein present in mineralised tissues, representing 20% of bone and dentine by weight (Ambrose, 1993). While it is not the sole protein represented in skeletal tissues, with osteocalcin and others also present, it is ubiquitous enough that all other proteins in bone or dentine are referred to under the umbrella term of "noncollagenous proteins." To isolate the organic component of bone or tooth dentine for analysis, dilute acids such as hydrochloric acid (HCl) or the less-commonly

used ethylenediaminetetraacetic acid (EDTA) are used to dissolve and discard or eliminate the mineral component of samples. Following this step, the organic component of bone or dentine is subsequently gelatinised, freeze-dried, and analysed via mass spectrometry (discussed more fully in Chapter 5, Materials and Methods). In some studies, the proteinaceous product of demineralisation in skeletal tissues is consequently denoted "collagen" with quotation marks in respect of the presence of other noncollagenous proteins within this substance, and the fact that archaeological proteins are likely to be degraded. However, for the purposes of simplicity and clarity, this organic component of bone will be henceforth referred to only within this study as collagen, with collagen in its pure form referred to explicitly as such. The ubiquity and persistence of collagen in archaeological skeletal tissues, as well as its resistance to diagenesis and alteration of biogenic isotope composition after death, make it the preferred tissue fraction for palaeodietary study (Ambrose, 1993; Schwarcz, 2000).

Like all proteins, collagen is composed of amino acids. Essential amino-acids make up only 12% of collagen, and in light of the disparity in favour of non-essential amino-acids Ambrose (1993) proposes that the consistently-observed enrichment of collagen in heavier isotopes, relative to diet, may stem from recycling and synthesis of non-essential amino-acids drawn from all areas of diet, rather than strictly from the protein component. Diet to collagen spacing for $\delta^{13}C$ is generally estimated at +5‰ for humans, with an observed range of +3 to 6‰ for animals (Lee-Thorp *et al.*, 1989; Bocherens and Drucker, 2003; Lee-Thorp, 2008). Similar isotopic spacing is seen between diet and collagen for the $\delta^{15}N$ of human populations, for whom consistent trophic level effects of +2 to 6‰ are observed (Bocherens and Drucker, 2003; Hedges and Reynard, 2007; Lee-Thorp, 2008).

Protein in both tissues which remodel (bone) and tissues which do not alter their composition after formation (dentine, hair, and nails) provide, where available, a range of data which cover different portions of a person's lifecourse at varying resolution. This means that where the most commonly-represented tissues in the archaeological record are present, the skeletal remains of adult individuals preserve a static and high-resolution record of childhood diet via their teeth, which form between birth and roughly 23 years of age (Al Qahtani *et al.*, 2010), and also a highly time-averaged record of diet and health incorporating contributions spanning adolescence to the period of death (Hedges *et al.*, 2007). The variable time resolution offered by these methods

allows archaeologists to interrogate resulting data for a range of research questions appropriate to that precision. Understanding of the behavioural and physiological dynamics of biogeochemistry together with knowledge of the dynamics of skeletal tissue allows archaeologists to attempt to quantify and understand the nature of diet and dietary transition in the past.

2.3 The carbon cycle

The following section will discuss the sources and behaviours of carbon stable isotopes within ecosystems and will examine how they may be used as an indicator of environment and behaviour within foodwebs.

2.3.1 C_3 and C_4 plants

Carbon isotope patterning for plants and animals within a biosphere is fundamentally governed by the processes of kinetic fractionation associated with photosynthesis, in which the primary source of carbon is atmospheric carbon dioxide (CO_2), which recent studies estimate at a $\delta^{13}C$ value of −8‰ (van Klinken *et al.*, 2000). This atmospheric value has not been constant over time, but has been influenced by anthropogenic fossil fuel consumption, which has changed the atmospheric $^{13}C/^{12}C$ ratios relative to the −6.5‰ $\delta^{13}C$ value estimated for the mid eighteenth century (van Klinken *et al.*, 2000). All plants take in CO_2 by diffusion through leaf pores, a process which favours more rapid diffusion of the lighter isotope ^{12}C. Enzymes involved in photosynthesis also preferentially select for ^{12}C, providing an additional layer of discrimination against ^{13}C (Sealy, 2001). The advantages given to ^{12}C over ^{13}C during photosynthesis lead to lower $^{13}C/^{12}C$ ratios in the tissues of plants than in ambient air (Farquhar *et al.*, 1989). It has consequently been said that "…isotopically light carbon is a characteristic signature of life" (Sharp, 2007, p. 153). However, the extent of discrimination against ^{13}C, and the consequent effects upon plant $^{13}C/^{12}C$ ratios, depends upon the photosynthetic pathway used.

The vast majority of plants photosynthesise through the C_3, or Calvin-Benson, pathway; named for the three-carbon compound produced (Sealy, 2001). C_3 plants – a group which includes nearly all temperate terrestrial and most marine plants, as well as all major food crops domesticated by the ancient Near East civilizations apart from millet – discriminate most strongly against ^{13}C, and have $\delta^{13}C$ values of approximately −26‰, with a range −22 to −34‰ (Smith, 1972; Sealy, 2001). Ribulose bisphosphate carboxylase (Rubisco) is the primary enzyme involved in C_3 photosynthesis and is

responsible for the high level of fractionation between atmospheric CO_2 and the tissues of C_3 plants (Farquhar *et al.*, 1989). A smaller, but culturally significant, number of plants use the C_4, or Hatch-Slack, pathway, which initially produces a four-carbon compound. This group primarily consists of tropical grasses, including important food crops such as sugar cane, millet, and maize. The primary enzyme responsible for fractionation of carbon during the photosynthetic process in C_4 plants is phosphoenolpyruvate carboxylase (PEP), which also discriminates against ^{13}C, but to a lesser extent than Rubisco (Farquhar *et al.*, 1989). This produces plant tissues with mean $\delta^{13}C$ values of approximately −13‰, and a range of −8 to −16‰ (Sealy, 2001). A third group of terrestrial plants, called Crassulacean acid metabolism (CAM) plants are adapted to alternate between both C_3 and C_4 photosynthetic pathways, depending on environmental conditions. These plants consequently may have $\delta^{13}C$ values which may be reflective of either pathway, or alternately they may appear intermediate. CAM plants as a group primarily comprise cacti and other succulents, as well as pineapples (Ambrose, 1993), and do not apply to the current study.

In addition to differences in photosynthetic pathway, geographical factors may influence the lability of carbon isotope composition of plants and consumers. van Klinken *et al.* (1994) noted systematic variability in $\delta^{13}C$ between northern and southern European locations, due to the effects of temperature and humidity on fractionation, with a trend towards lower $\delta^{13}C$ in locations of lower temperature and higher humidity and higher $\delta^{13}C$ under conditions of greater aridity and temperature. The effects of temperature have been demonstrated by other studies examining macroclimatic shifts over long periods of time, as well as microclimatic seasonal shifts (Becker *et al.*, 1991; Aucour *et al.*, 1993; Loader and Hemming, 2001; Hedges *et al.*, 2004). A similar, but more localised, eco-spatial effect has been observed for plants and animals occupying an arboreal environment. Termed the "canopy effect", recycling of fractionated, plant-respired CO_2 in areas of heavy vegetation, may cause a disparity between plants and animals inhabiting and feeding in forest floor, canopy, and open environments (van Klinken *et al.*, 2000). This effect results in lower $\delta^{13}C$ values of atmospheric CO_2 in densely wooded areas, and has been observed in studies analysing plants, animals, and humans in these environments, relative to their open-dwelling counterparts (Ambrose and DeNiro, 1986; van der Merwe and Medina, 1989; van der Merwe and Medina, 1991; Drucker *et al.*, 2008).

2.3.2 Carbon isotope distribution in aquatic foodwebs

In the same way that photosynthetic pathways of plants produce distinctive isotopic patterning to those plants, so in turn do the values of those plants influence the tissue values of the animals which consume them. In contrast to the dynamics of terrestrial ecosystems, the carbon reservoir of marine aquatic environments is not atmospheric carbon dioxide but bicarbonate (HCO_3) dissolved in ocean water, which has a $\delta^{13}C$ of ~0‰ (Smith and Epstein, 1971). Marine foodwebs are highly variable but are primarily based on C_3 plants, which have an average value of −19‰ due to photosynthetic fractionation (Smith, 1972; Chisholm *et al.*, 1982; Ambrose and Norr, 1993). The fractionation factor for terrestrial and marine C_3 plants is thus similar; though $\delta^{13}C$ values will differ due to the $\delta^{13}C$ values of their respective carbon sources (Chisholm *et al.*, 1982). Plants using the C_3 pathway form the foundation of marine ecosystems, with widely varying $\delta^{13}C$ values (range −39 to −11‰) characterising the range of plants from estuarine sea grasses to marine algae (Farquhar *et al.*, 1989; Ambrose, 1993). However, it is marine animals, rather than plants, which tend to be most important in human diets; and these have much more constrained variability than that of marine plants, with mean values of −15.6 ±1.6‰ (Ambrose, 1993; Sealy, 2001). The similarity between the $\delta^{13}C$ values of marine animals and those of C_4 plants in geographic areas where both are expected to contribute substantially to human diet can make the interpretation of resource consumption based on carbon isotopes alone difficult, if not impossible, requiring the use of complementary data such as nitrogen or sulphur isotope ratios in order to distinguish between the two dietary resources (Ambrose, 1993). The ability to distinguish between marine and terrestrial reservoirs gains heightened significance in terms of ^{14}C dating, as dates based on analysis of carbon drawn from a marine reservoir may appear up to 400 years older than comparable terrestrial samples and will require a correction factor in order to interpret dates correctly (Pollard and Wilson, 2001).

Consumption of animals from freshwater aquatic environments such as lakes and rivers can also require some disambiguation when interpreting carbon isotope ratio data. The complexities of aquatic isotope systems are greater than those of terrestrial systems, particularly in the case of freshwater plants where multiple, and potentially fluctuating, sources of carbon are available (Katzenberg and Weber, 1999). These complexities result in a broad and highly variable range of $\delta^{13}C$ in freshwater consumers, which may overlap with terrestrial values (Schoeninger and DeNiro, 1984; Privat *et al.*, 2002;

Müldner and Richards, 2005). Though progress in understanding the role of freshwater protein resources to human $\delta^{13}C$ collagen values has continued since Ambrose's statement, the similarity between the carbon isotope ratios of freshwater and terrestrial animals in many cases renders them indistinguishable through carbon analyses alone, requiring additional data from sulphur or nitrogen isotopes to clarify consumption patterns (Privat *et al.*, 2007). As with the marine reservoir, a freshwater reservoir effect based on the dissolution of ancient carbonates may alter the ^{14}C values of freshwater animals and humans who consume them, creating disparities between real and measured age (Philippsen, 2013). The bone collagen $\delta^{13}C$ values of animals such as migratory birds and fish, who spend portions of their life cycle in both marine and freshwater environments, have been found to occupy an intermediate niche between animals feeding exclusively in marine or terrestrial environments, in some cases overlapping with terrestrial values, depending on the feeding pattern associated with their migratory activity (Schoeninger and DeNiro, 1984; Fuller *et al.*, 2012; Robson *et al.*, 2012). The ability to identify a characteristic carbon isotope fingerprint is further limited by poor preservation of fish remains in the archaeological record, requiring the use of complementary isotopes such as nitrogen and sulphur to better understand exploitation of an important dietary resource to many human groups (Nehlich, 2015).

2.3.3 Fractionation of carbon isotopes in humans consuming a terrestrial diet

Though the carbon isotope ratios of animals relate to their diet, fractionation alters the straightforward correlation between consumed foods and tissues. Broadly observed trends tend towards larger diet to tissue shifts for herbivores than for carnivores, and smaller shifts for animals of small body size than for large animals (Schwarcz, 2000). As moderately-sized, omnivorous consumers, $\delta^{13}C$ diet to collagen spacing is low for humans. Knowledge of environment-specific endmembers to expected ranges may guide archaeologists to interpret carbon isotopic data. For instance, human bone collagen $\delta^{13}C$ values ranging from −20 to −21‰ in terrestrial Holocene Europe would indicate a diet based on C_3 plants or C_3-consuming animals, while a measured value of approximately −12‰ in human bone collagen would indicate a diet based almost exclusively on marine foods, given an absence of C_4 plants (Schulting and Richards, 2002). However, human diet is rarely derived from a single source, and estimation of resource exploitation remains complex. Fogel *et al.* (1997) succinctly described the difficulties of reconstructing past human omnivory, which cannot be assumed to be

based on ease of resource availability. Consumption of a range of foods with differing isotopic values, each forming a different proportion of the diet, will produce "scrambling" of isotope values reflecting contributions from the whole diet (Hedges and Van Klinken, 2000, p. 213). The mixing models, often complex, associated with assessment of scrambling are of great value to analyses of tissues reflecting the whole diet, in addition to those formed via "routing," preferential allocation of carbon from a single dietary component, which is reflected in the isotopic composition of collagen (Hedges and Van Klinken, 2000; Schwarcz, 2000; Fernandes *et al.*, 2014). As the dynamics of scrambling and multi-tissue isotopic offsets are complex, this section will concentrate on the mechanisms and extent of fractionation between diet and collagen in humans which are most relevant to the present study.

All fractionation between diet and tissue occurs as a result of metabolic processes within the body. These processes – deamination, transamination, and amino acid synthesis – are believed to account for most ^{13}C enrichment in human collagen (Ambrose, 1993; Ambrose and Norr, 1993). The extent of fractionation is not equal for all food types; shifts between high-protein diets and collagen, where preferential use of exogenous amino acids may decrease the extent of enrichment, are smaller than those observed for protein-poor diets, where *de novo* synthesis of amino acids and recycling of carbon from non-protein dietary fractions results in greater fractionation (Ambrose, 1993; Schwarcz, 2000; Jim *et al.*, 2006). The theoretical nature of knowledge regarding the causes of fractionation, and the variability of its magnitude, in humans has led van Klinken *et al.* (2000) to recommend avoiding use of the term "trophic level effect" in reference to diet to tissue shifts in carbon.

Variability in shifts in the carbon composition of human tissues then chiefly occur as an outcome of the balance between intake, metabolic activity, and excretion of carbon. While intake is accounted for by diet, and metabolic activity encompasses the transformative and synthetic activities of human tissue production and maintenance, both of which involve significant variability, the pathways of carbon excretion are limited. The primary routes through which carbon is excreted from the body are the exhalation of CO_2, and to a much lesser extent other wastes such as urea or exfoliated epithelial cells (Schwarcz, 2000). Roughly 90% of carbon is excreted via exhaled CO_2, which experimental animal studies have demonstrated is broadly representative of the whole diet in carbon composition (Hedges and Van Klinken, 2000; Schwarcz, 2000;

Hatch *et al.*, 2002). Non-ruminant data from other studies suggests that exhaled CO_2 may be 1-2‰ lighter than diet (DeNiro and Epstein, 1978; Tieszen and Fagre, 1993; Perkins and Speakman, 2001). This ambiguity in the dynamics of carbon isotope mass balance would benefit from further investigation in future experimental study. Despite the uncertainties present regarding the details of mechanism involved in human carbon isotope mass balance, the extent of diet to tissue shifts in collagen is well-characterised, if variable, and has been frequently applied alongside shifts in nitrogen to understanding diet in past societies occupying terrestrial ecosystems (e.g., Richards *et al.*, 1998; Jay and Richards, 2006; Mays and Beavan, 2012; Knipper *et al.*, 2013).

2.4 The nitrogen cycle

This section will describe the ways in which nitrogen stable isotopes are assimilated and fractionated within foodwebs, including variation based in dietary trends and physiological processes.

2.4.1 Nitrogen uptake in terrestrial plants

While carbon isotopes are integrated into the tissues of terrestrial plants through CO_2 exchange as a part of respiration and photosynthesis, nitrogen is accessed at the base of the food chain through the roots of plants. They may do this in one of two ways. Plants possessing symbiotic prokaryotic nitrogen-fixing bacteria in their root systems derive nitrogen through bacterial conversion of gaseous atmospheric nitrogen, which has a $\delta^{15}N$ value of approximately 0‰, into a solid form of ammonia (NH_3 or ammonium, NH_4^+) through the use of an enzyme known as nitrogenase under anaerobic soil conditions (Shearer and Kohl, 1986). Examples of plants which access soil nitrogen through the auspices of residential symbiotic bacteria include legumes, some shrubs, and trees such as alder. Nitrogen-fixing bacteria are also present in the soil as a diverse range of free-living organisms (Kennedy and Tchan, 1992). Non-nitrogen fixing plants may derive their nitrogen through the services of these free-living bacteria, or they may derive their nitrogen through ammonium (NH_4^+) produced by the decomposition of organic matter into soil. This ammonium may then further transform by the process of nitrification into soil nitrate (NO_3^-), which is then assimilated by non-nitrogen fixing plants (Canfield *et al.*, 2010). Both processes of nitrogen derivation involve some fractionation. However, plants possessing nitrogen-fixing bacteria have tissues which are less enriched in ^{15}N than plants receiving their nitrogen via soil nitrification, as the

former have $\delta^{15}N$ values which are close to atmospheric nitrogen (van Klinken *et al.*, 2000).

In addition to the intrinsic dynamics of the nitrogen cycle, the expected values of plants may be affected by extrinsic factors, such as anthropogenic activity and meteorological change. The agricultural practice of manuring, like simple decomposition of other organic matter, produces soil which is enriched in ^{15}N relative to atmospheric nitrogen, as manure produces $\delta^{15}N$ values which reflect the values of the producer's diet (Hedges and Reynard, 2007). Thus, plants which grow in soil which has been manured or has had non-manure organic fertilisers applied will have significantly higher $\delta^{15}N$, relative to non-manured plants in the same environment, with further effects up the food chain for the consumers of such plants (Bogaard *et al.*, 2007; Fraser *et al.*, 2011). Water stress may also play a part in plant nitrogen balance. While relatively low $\delta^{15}N$ values characterise the isotopic patterning of plants and animals inhabiting moist and temperate ecosystems, higher $\delta^{15}N$ values are characteristic of hot and, especially, dry environments (Shearer and Kohl, 1986; Ambrose, 1993). The effects of water stress have been demonstrated to create greater rises in $\delta^{15}N$ in animals than have been observed for plants (Heaton, 1987). The relationship between water stress and increased $\delta^{15}N$ in plants has been suggested to result from a more "open" nitrogen cycle in plants experiencing (Handley *et al.*, 1999), or alternately to be caused by evaporative losses of ^{15}N-depleted ammonia in soil (Schwarcz *et al.*, 1999). The greater ^{15}N enrichments observed for humans and animals occupying arid environments are believed to result from a combination of consumption of plants sharing their environment and increased output of isotopically-light urea (Hartman, 2011). The intra- and inter-ecosystem variability in measured $\delta^{15}N$ of plants and animals demonstrates the impacts of climate, habitat, and plant behaviours relating to nitrogen fixation, all of which imply the importance of approaching analyses of similar organisms within different ecosystems with the benefit of baseline environmental data specific to each ecosystem (Ambrose, 1993).

2.4.2 Trophic level effects

As with carbon, the trophic level shifts which occur between the diet and tissues of a consumer are believed to result from metabolic activity relating to amino-acid synthesis. The lower vibrational frequency of the heavier ^{15}N leads to amine groups containing ^{14}N being preferentially selected for the processes of transamination and deamination,

resulting in excretion of isotopically-light waste (Adams and Sterner, 2000; Ambrose, 2000). In animals, these kinetic isotope fractionation effects associated with the formation of tissue are compounded by the activities of urea production and kidney function (van Klinken *et al.*, 2000), and in humans around 80-90% of nitrogen intake is excreted through urea, with ammonia and creatinine in urine, faecal compounds of nitrogen, perspiration, and sloughing of epithelial cells playing minor roles (Hedges and Van Klinken, 2000). Some researchers have argued that the synthesis and excretion of ^{15}N-depleted urine is principally responsible for overall body pool ^{15}N enrichment (Peterson and Fry, 1987), while others have suggested that the influence of excreta alone on nitrogen isotope mass balance represents an insufficient explanatory mechanism for tissue ^{15}N enrichment, though clearly playing a role (Schoeller, 1999; Hedges and Van Klinken, 2000). It is also theorised that the excretion of urea during periods of water stress may explain some of the variability within trophic levels, due to increased urea and decreased water excretion in urine during these periods (Hartman, 2011). Regardless of precise mechanism, the cumulative effect of this ^{15}N enrichment at each trophic level throughout the food chain results in a stepwise increase in δ^{15}N values of about 3-4‰ (Minagawa and Wada, 1984; Brown and Brown, 2011).

2.4.3 The nitrogen cycle in aquatic environments

Nitrogen isotopes may be used to distinguish between marine and terrestrial plants, as the δ^{15}N values of oceanic plants are based on the balance between nitrification and denitrification in that environment, with a bias towards denitrification (Sealy, 2001). Consequently, the δ^{15}N values of marine plants are approximately 4‰ higher than terrestrial plants, although it should be noted that these higher marine δ^{15}N values are not observed in environments where the contributions of nitrogen-fixing blue-green algae lead to near-parity with terrestrial plants (Ambrose, 1993). As with carbon, nitrogen stable isotope values are strongly tied to latitude, with higher δ^{15}N values observed for plants and animals living in the middle latitudes than is exhibited for species living at higher latitudes (Fuller *et al.*, 2012). Decomposition of nitrogenous matter in the ocean may also result in increases of 5-10‰ with increasing depth (Peterson and Fry, 1987). Marine animal δ^{15}N values are much higher than those of animals living in temperate terrestrial systems, due to a combination of the generally higher δ^{15}N of marine plants and also the much longer food-chains within marine environments, relative to terrestrial environments (Richards and Hedges, 1999; Sealy,

2001). This characteristic ^{15}N enrichment of marine animals is reflected in their bone collagen, and also the bone collagen of humans consuming significant proportions of their diet from marine sources, making distinction of these consumers theoretically straightforward at the extreme ends of a wholly marine or wholly terrestrial diet. As the only significant marine energy sources in humans are protein-based and collagen reflects protein inputs (Ambrose, 1993), marine inputs should be highly visible in human collagen data. However, human diets are generally diverse, and mixed contributions from dietary sources bearing different $\delta^{15}N$ values will add some ambiguity.

Salinity outside of a purely marine environment is another factor which may alter $\delta^{15}N$ values of plants and animals and may have implications for the interpretation of resource exploitation. Salt-loving (halophyte) C_3 plants have been demonstrated to have elevated $\delta^{15}N$ values in saline environments such as salt marshes (Cloern *et al.*, 2002; Britton *et al.*, 2008). Coastal salt marshes have frequently been used for grazing of terrestrial animals such as cattle and sheep in Britain and are an important consideration when differentiating between food source utilisation in past humans, although it is unclear whether elevated $\delta^{15}N$ values in animal collagen arise solely from the elevated values of plants, or from a form of salt-induced water stress associated with consuming these plants (Britton *et al.*, 2008).

While nitrogen isotope values provide an important indicator of marine resource usage, they may also help to distinguish freshwater fish consumption from terrestrial omnivory. Freshwater food chains, like marine food chains, are longer and contain more trophic levels than those of terrestrial ecosystems. While $\delta^{13}C$ collagen values associated with consumption of freshwater fish may not be distinguishable from those based on consumption of terrestrial proteins, humans most commonly consume carnivorous fish which will be reflected in higher $\delta^{15}N$ values which are often comparable to those of marine fish (Fry, 1991; van Klinken *et al.*, 2000; Privat *et al.*, 2002). Thus, significant consumption of freshwater proteins may result in $\delta^{13}C$ collagen values consistent with consumption of terrestrial protein, alongside elevated $\delta^{15}N$ values. While understanding of this isotopic patterning is helpful to the interpretation of resource exploitation and diet, similar patterning may be observed for individuals experiencing physiological fractionation of nitrogen associated with systemic stress (relevant to the present study and discussed more in the section following), and thus this

interpretation should be made cautiously, particularly in cross-sectional study where change to individuals' isotopic profile over time is not accessible.

2.4.4 Nitrogen isotopes in humans

Roughly 98% of nitrogen in human tissues exists as proteins and amino acids, with one significant exception: about 20% of human milk nitrogen is present as non-protein nitrogen (Donovan *et al.*, 1991; Schoeller, 1999). The strong relationship between dietary protein and body proteins, particularly collagen, has formed the basis of palaeodietary reconstruction. Although the principles of trophic level increase within foodwebs are well-characterised and accepted, Hedges and Reynard (2007) contended that the precise mechanisms of ^{15}N enrichment in humans are ambiguously supported, with disagreement concerning the strength of the relationship between dietary protein intake and higher collagen $\delta^{15}N$. Some studies have attempted to quantify diet to body tissue spacing through experimental study. Some studies have found consistent levels of diet-tissue spacing, with diet to hair spacing of +4.3‰ for $\delta^{15}N$ (Schoeller *et al.*, 1986; Minagawa, 1992). However, other studies have demonstrated considerable variability. Yoshinaga *et al.* (1996) carried out isotopic analysis of food and hair samples for 49 males in Papua New Guinea over a two-year period, finding an estimated diet-keratin spacing of +5–6.9% for $\delta^{15}N$. A later study by Hedges *et al.* (2009) observed a smaller average increase in hair $\delta^{15}N$ of +4.1‰, relative to total diet for their rural Fijian sample. O'Connell *et al.* (2012) more recently attempted to dispel some of the uncertainties surrounding body-diet spacing in a controlled diet study analysing red blood cells (RBCs) in fresh blood samples from 11 individuals over a 30-day period. Through comparison of RBC data and known offsets between other tissues, the study estimated a diet to keratin spacing of +5-5.3‰, falling between the published measurements of Hedges *et al*. and Yoshinaga *et al*. The O'Connell study also inferred diet-collagen spacing of approximately +6‰, larger than previous bone studies have suggested; leading the authors to caution that palaeodietary studies may be overestimating the importance of higher trophic-level foods in past human diet.

2.4.5 Physiological fractionation of nitrogen

Fractionation of nitrogen stable isotopes may also result from processes other than dietary change; physiological processes may also alter $\delta^{15}N$ values in humans and should be considered as potential causes of observed changes in value. These non-dietary physiological effects may result in changes from the nitrogen balance achieved

during homeostasis to anabolic or catabolic states. Anabolic states result in positive nitrogen balance, where more nitrogen is retained from inputs for the synthesis of new tissues than is excreted, reducing $\delta^{15}N$ values (Waters-Rist and Katzenberg, 2010). Negative nitrogen balance occurs during catabolic states, where intake of nitrogen is outstripped by demand, and existing tissue is broken down to meet systemic demands. This recycling of tissue occurs during periods of systemic stress, usually stemming from nutritional inadequacy or disease, and leads to a rise in $\delta^{15}N$ values (Mekota *et al.*, 2006). These deviations from homeostasis may remain invisible within highly time-averaged, synchronic analyses, but have nonetheless been the subject of investigation in both modern and archaeological populations.

Anabolic states most commonly result from physiological events associated with increase in body mass, such as growth spurts or pregnancy. Fuller *et al.* (2004) analysed incrementally-sampled hair from ten modern pregnant women, comparing $\delta^{15}N$ and $\delta^{13}C$ values prior to conception and throughout pregnancy to birth. They found that while $\delta^{13}C$ values remained stable, a drop in $\delta^{15}N$ was observed for all subjects (range 0.3-1.1‰) during pregnancy, which inversely correlated with weight gain during the same period (Fuller *et al.*, 2004). D'Ortenzio *et al.* (2015) found a similar drop of approximately 1‰ in an analysis of hair from two pregnant subjects. Nitsch *et al.* (2010) attempted to demonstrate a "pregnancy effect" in an analysis of rib collagen from males and females from an 18th and 19th century London population, finding no significant differences in $\delta^{15}N$ value based on sex or parity status. The authors concluded that the potential for discerning a pregnancy effect on $\delta^{15}N$ in bone collagen was low, due to the time resolution of bone collagen data and the limited scale and duration of the hypothesised effect (Nitsch *et al.*, 2010). Periods of intense growth also produce anabolic states, due to the increase in tissue-building activities associated with new body mass. Sears *et al.* (2009) demonstrated this growth effect on nitrogen isotope ratios in an experimental study of seabird chicks, finding that $\delta^{15}N$ values declined by about 1‰ during periods of intense growth, rising again when growth slowed. The effects of growth on nitrogen ratios are particularly significant for infant palaeodietary studies, where non-adult $\delta^{15}N$ values commonly decline below those of adults following completion of weaning, and should factor into weaning models (Millard, 2000). Concern with the implications of a nitrogen growth effect for diet to tissue isotope spacing in non-adults prompted Waters-Rist and Katzenberg (2010) to analyse nitrogen

ratios in long bone collagen from individuals ranging from 7 to 19 years of age in a protohistoric Canadian ossuary sample. Like the earlier study by Nitsch *et al*., Waters-Rist and Katzenberg found that bone collagen did not reflect differences in $\delta^{15}N$ which might suggest significant shifts based on anabolic metabolism. Great potential exists to revisit some of these issues, particularly growth effects, using higher resolution methods, such as the incremental dentine methods used in the present study.

In contrast to the physiologically normal events of growth or pregnancy with which anabolism is associated, catabolic states are primarily associated with poor health. Nutritional stress, or malnutrition, is the most frequently-studied of the potential causes. The influence of nutritional stress on the development of catabolism has been demonstrated in a number of controlled animal studies, using a variety of species ranging from pigs to penguins, in recent decades (e.g., Hobson *et al.*, 1993; Gaye-Siessegger *et al.*, 2004; Cherel *et al.*, 2005; Warinner and Tuross, 2010). An experimental study by Kempster *et al.* (2007) sampling blood, liver, muscle, and feather tissue in nutritionally-stressed juvenile song sparrows and controls, found that $\delta^{15}N$ and $\delta^{13}C$ values did not differ between the two groups. The authors suggested that catabolism may not occur along a continuum of nutritional stress but may signify a threshold of metabolic stress below which isotopic shifts are negligible. The possibility of such a threshold should be explored further in future experimental studies as a topic of high relevance to palaeodietary reconstruction.

The potential for nutritional stress or disease to alter nitrogen balance and produce catabolic states in humans has received some limited experimental study but is an increasing area of research among palaeopathologists and palaeodietary researchers. Fuller *et al.* (2005) analysed hair samples grown prior to pregnancy and during pregnancy from eight women suffering from nutritional stress stemming from hyperemesis gravidarum (extreme morning sickness). The study found that $\delta^{13}C$ values were stable throughout the study period, but that $\delta^{15}N$ values increased by up to 1‰ (Fuller *et al.*, 2005). Mekota *et al.* (2006) observed a similar trend for $\delta^{15}N$ in analysed hair of six individuals suffering from anorexia nervosa, finding a rise of up to 1.5‰ during times of stress. In contrast to the finding of stable carbon isotope ratios in the Fuller study, $\delta^{13}C$ were found to correlate with supply of dietary protein, falling during nutritional stress, and rising during periods of weight gain and recovery, when adequate nutrition was reintroduced (Mekota *et al.*, 2006). A third study by Neuberger *et al.*

(2013) analysed hair from 16 malnourished autopsied individuals concurred with the study by Mekota *et al.*, finding a consistent pattern of isotopic opposing covariance, with rising $\delta^{15}N$ values and a corresponding decrease in $\delta^{13}C$ during the same period. While the relationship between carbon isotope balance and malnutrition requires further clarification, the relationship between increasing $\delta^{15}N$ values and nutritional stress appears consistently demonstrated by the limited range of human studies, and also forms a predominant trend within animal studies.

Infection or chronic diseases may also induce catabolic states which are reflected in cachexia, or wasting, and do not stem from changes in dietary intake. Cancer (e.g., Pisters *et al.*, 1993; Tisdale, 2002) and comorbidity of tuberculosis and AIDS (e.g., Paton *et al.*, 2003) are perhaps the most well-known diseases affecting protein metabolism, but potential exists for other forms of physiological distress to disrupt nitrogen balance, including infectious disease (Webb *et al.*, 2015), bone fracture, and potentially sustained psychological stress (Wheeler *et al.*, 2013). D'Ortenzio *et al.* (2015) analysed hair from four modern cadavers, two of which had cancer, and 10 archaeological individuals with recorded causes of death derived from parish records. The authors found that pathological conditions such as bone fracture, cancer, or infection were strongly associated with increases in nitrogen isotope ratios of approximately 1‰ (D'Ortenzio *et al.*, 2015). Altered nitrogen metabolism may also be reflected in isotopic values of collagen. Studies by Katzenberg and Lovell (1999) and Olsen *et al.* (2014) have both demonstrated differences in nitrogen metabolism reflected in bone collagen from pathologically-altered sampling sites, relative to non-pathological bone sampled from the same individuals. This is believed to result from increased turnover at the lesion site, and to reflect altered nitrogen balance associated with infection.

Vivax malaria (*P. vivax*) is an infectious disease relevant to the present study (discussed at greater length in Chapter 4) and is known to cause cachexia and malnutrition (Rogerson and Carter, 2008; Monteiro *et al.*, 2016). However, little is known about its effects on nitrogen balance. A single study by Yohannes *et al.* (2011) analysed carbon and nitrogen isotope ratios in the feathers of wild-caught migratory birds infected with *P. ashfordi* and *P. reticulum* species of malaria and controls, finding no apparent correlation between infection and altered isotope balance. However, the species of the *Plasmodium* genus vary greatly in their pathophysiology and potential for

cachexia, with “benign” chronic pathogens such as *P. vivax* greatly outstripping the deadly *P. falciparum* due to the greater secretion of tumour necrosis factor (TNF), a cause of anorexia and cachexia (Williams *et al.*, 1997). Consequently, malaria is one of many infectious diseases with the potential to alter nitrogen metabolism and palaeodietary data, which should be considered alongside ecological data. It is hoped that the present study may offer some new evidence towards identifying characteristic isotopic patterning.

2.5 Use of human teeth in high-resolution palaeodietary research

Isotopic analysis of human teeth has offered unprecedented opportunities to understand the time-sensitive dynamics of dietary transition. This section will discuss important principles regarding the development of human dental tissues and their analysis within archaeology. As palaeodietary research has matured as a discipline, the uncertainties regarding rates of collagen turnover and the low time resolution associated with analyses of bone have prompted development of higher resolution methods. While archaeological studies have occasionally analysed tissues which form over weeks or months, such as fingernails or hair, these methods are restricted in application by the paucity of these tissues’ representation within burial environments. Consequently, the use of hair and fingernail analyses in archaeology has primarily been limited to mummified remains. By contrast, human teeth are formed over a longer period than fingernails or hair, representing several years of formation time on average. However, like tree rings, the enamel and dentine of teeth are formed and mineralised along advancing fronts, represented by incremental structures, until the full tooth is complete. These incremental structures, which are preserved in the completed tooth and are visible through microscopy, provide a means of improving time resolution through the alignment of spatial and temporal dimensions, as an alternative to bulk sampling of bone, enamel, or dentine.

2.5.1 Odontogenesis

The timescales of dental development, or odontogenesis, are extremely well-characterised overall as they have formed a major focus of age estimation techniques in bioarchaeological studies, as well as the forensic sciences (Liversidge *et al.*, 2010). While some inter-population variability is observed for dental development, which is genetically-linked, particularly in respect to the timing of tooth eruption, the timescales of development and formation of teeth themselves are relatively constrained. In contrast

to appositional bone growth and the fusion of epiphyses, two markers of increasing skeletal growth and maturation, dental development is relatively unhindered by the problems of poor health and malnutrition, which may significantly affect the appearance of skeletal maturity by inducing growth arrest and delayed epiphyseal fusion (Elamin and Liversidge, 2013). It is important, however, to distinguish between tooth eruption, which may be influenced by early life nutrition (Oziegbe *et al.*, 2010), and the formation and development of teeth, which appear to be more robust against environmental influence. Thus, dental development timelines represent the most reliable current age estimation technique for non-adult skeletal remains.

Humans, like most mammals, possess two successive dentitions. The first or primary set of dentition is deciduous and begins to form by the sixth week *in utero* (Al Qahtani *et al.*, 2010). Table 2.1 shows estimated dental development timescales for British populations. The early formation period of deciduous teeth *in utero* allows researchers to selectively sample those tissues for isotopic analysis in order to elucidate information about maternal health and diet through its reflection of the uterine environment. The secondary, or permanent, dentition begins to form around the time of birth. The primary dentition is complete between 2-3 years of age, followed by several years of apparent oral stasis, during which the permanent dentition continues to form within the alveolus of the maxilla and mandible. By 5-6 years of age, the first permanent molars emerge, initiating the period of mixed dentition in which children will possess both deciduous and permanent teeth in occlusion. During the period of mixed dentition, as the permanent teeth form and begin to move upward in the jaws, the roots of the overlying deciduous teeth are resorbed. As their roots are resorbed by the action of odontoclasts, these deciduous teeth are exfoliated and replaced by the emergence of the underlying secondary dentition. By the time of alveolar eruption, the roots of newly erupted permanent teeth will have achieved roughly half of their eventual length, with the root continuing to form after eruption until its full length has been reached and apex closure has occurred (Al Qahtani *et al.*, 2010).

Early attempts at improving the time resolution of isotopic tooth data in enamel (e.g., Wright and Schwarcz, 1998; Balasse, 2002), dentine (e.g., Balasse *et al.*, 2001; Balasse and Tresset, 2002; Fahy *et al.*, 2014), or both (Wright and Schwarcz, 1999; Dupras and Tocheri, 2007) utilised the known developmental parameters of teeth alongside bulk sampling methods in order to create an overlapping timeline of isotope data for sampled

animals and humans. However, as the drive for higher-resolution sampling has gained momentum, more sophisticated methods utilising the incremental structures of both enamel and dentine have attempted to create individual isotopic profiles with increasingly fine time sensitivity. The sections which follow will outline the basic principles of enamel and dentine development and microstructures.

Deciduous Dentition			
Tooth	Age of initiation	Age at crown completion	Age at apex completion
i_1	−0.3±0.04	0.25±0.04	2.5
i_2	−0.3±0.04	0.3±0.04	2.5
c	−0.2±0.04	0.6±0.04	3.5
m_1	−0.2±0.04	0.9±0.04	3.5
m_2	−0.2±0.04	0.9±0.04	3.5
Permanent Dentition			
Tooth	Age of initiation	Age at crown completion	Age at apex completion
Maxillary			
I_1	0.5±0.13	4.5	9.5
I_2	0.9±0.13	5.5	10.5
C	0.6±0.13	5.5	14.5
PM_1	2.5	6.5	13.5
PM_2	3.5	6.5	14.5
M_1	0.3±0.13	3.5	9.5
M_2	2.5	8.5	15.5
M_3	8.5	14.5	21.0-23.0
Mandibular			
I_1	0.6±0.13	3.5	8.5
I_2	0.6±0.13	3.5	8.5
C	0.9±0.13	5.5	13.0
PM_1	2.5	6.5	13.5
PM_2	2.5	6.5	14.5
M_1	0.3±0.13	3.5	10.0
M_2	2.5	8.5	15.5
M_3	8.5	14.5	22.0-23.0

Table 2.1. Ages of dental development for British populations. All ages are approximate and reported in years ±0.5 year, unless otherwise noted. Data is taken from Beaumont and Montgomery (2015), after Al Qahtani *et al.* (2010). I/i=incisor, C/c=canine, PM=premolar, M/m=molar.

2.5.1.i Enamel

Enamel has been heavily exploited in archaeological biogeochemistry, primarily studies of migration, due to its inherent resistance to diagenetic alteration provided by its

structure. However, it has also been an object of interest to high-resolution studies within archaeology and biological anthropology. Development of enamel is carried out by specialised cells in the enamel epithelium, known as ameloblasts, and takes place within two phases of mineralisation activity: secretion and maturation. Ameloblasts secrete enamel matrix on a continuous, but irregular basis, with the rate of secretion varying daily (Lucas, 2004). Within the first phase of mineralisation, thin, widely-spaced immature and fragile apatite crystallites known as prisms are secreted by the ameloblasts into a matrix which is composed of approximately one-third each organic, mineral, and water components (Hillson, 2005). During the second phase, maturation, the organic component of enamel matrix is broken down and replaced spatially and chemically by the growth and expansion of the previously seeded enamel prisms, creating a very hard tissue characterised by its densely-packed crystalline structure (Smith, 1998). Mature enamel is composed of approximately 96% mineral by weight (Hillson, 2005, p. 155), with a dense crystalline structure which accounts for its hardness and resistance to environmental damage and chemical exchange.

A significant lag between the timescales of enamel secretion and maturation places limitations on the applicability of high-resolution sampling based on incremental enamel structures. The timescales of maturation are of greatest interest for archaeological biogeochemists, as the final isotopic composition of mature enamel will be proportionately reflective of the body pool at the time of input. For enamel, which alters significantly from its initial matrix to mature enamel, the vast majority of material contributed to the final mature enamel is integrated in the maturation stage (Antoine *et al.*, 2009). However, as incremental structures of enamel relate to the secretory rather than the maturation stage, the chronology of isotopic inputs is unclear. Studies attempting to microsample enamel along a developmental gradient have achieved extremely mixed results. Work has primarily been accomplished through microdrilling/milling or through laser ablation mass spectrometry, with a higher volume of work being produced by the latter technique. Studies which have utilised microdrilling techniques have principally analysed carbon and oxygen stable isotope ratios (e.g., Wurster *et al.*, 1999; Wright, 2012), while laser ablation techniques have sought to improve understanding of the relationship between tooth development, diet, and trace mineral distributions in enamel (e.g., Kang *et al.*, 2004; Dolphin *et al.*, 2005; Hare *et al.*, 2011; Müller *et al*., 2019).

2.5.1.ii Dentine

Dentine is the tissue of most relevance to the present study. It accounts for most tooth material and has a composition similar to bone, with 72% of weight being accounted for by hydroxyapatite mineral and 20% being organic, 18% of which is collagen (Hillson, 1996). Like enamel, dentine is formed in stages of secretion and maturation along incremental lines, analogous to those found in enamel. These structures in dentine are formed as part of a two-stage process of secretion and maturation which is carried out by cells known as odontoblasts (Lucas, 2004). Short term incremental bands, like the cross-striations of enamel, are known as von Ebner's lines and represent the daily rhythm of secretion (Hillson, 2005). Andresen's lines represent the longer-period record of dentine secretion and are analogous to the striae of Retzius found in enamel. Like the striae of Retzius, the periodicities of Andresen's lines are consistent throughout a single tooth, and between all teeth belonging to that individual, and may be calculated through a count of the von Ebner's lines existing between these longer-term increments.

Predentine, the substance initially secreted by odontoblasts, consists of a collagenous organic matrix seeded with short apatite crystals (Beaumont *et al.*, 2013). This initial matrix is secreted in the cuspal region of the tooth at a steady rate of 4-6μm per day in permanent teeth (Dean and Scandrett, 1995). On average, the bands enclosed between Andresen's lines represent 20-30μm of dentine (Hillson, 2005), which suggests approximately 5 days of formation time per band. Dentine formation is accomplished in a process of retreat away from the enamel-dentine junction (EDJ) in cuspal dentine (in root dentine this process similarly proceeds from the cement-dentine junction) towards the pulp cavity. The organic matrix of predentine is approximately 90% collagen, with the vast majority of that being Type I collagen (Goldberg *et al.*, 2011). Unlike enamel, the two-stage process of secretion and mineralisation in dentine is carried out closely in tandem along the well-defined boundaries of the Andresen's lines, with the processes of secretion and maturation completed within 3-8 days (Beaumont *et al.*, 2013).

2.5.2 Issues in dental microsampling

Difficulties with time resolution are attendant on each of the potential target tissues of teeth. Enamel sampling is complicated by the timescales of the two-phase process: while the timing of mineralisation is well-characterised and more or less directionally linear, the maturation process appears to follow different and less predictable patterning which does not follow the incremental structures (Smith and Tafforeau, 2008). This lack

of correlation between observable microstructure and the timeline of isotopic inputs creates significant obstacles to research on the transitions of early life diet, which require reliable and accurate time-sensitive data. The disconnect between the timelines of incremental structures and final tissue formation in human enamel are the primary reason that microsampling of enamel was rejected as an approach by this study. By contrast, dentine analyses may offer an avenue of research which provides greater confidence in the validity of estimated dietary timelines. However, the precision of dentine analyses is currently constrained by the analytical demands of existing mass spectrometric technology. In the present, archaeological dentine increments which reliably produce sufficient collagen for mass spectrometry have been estimated at a minimum sample size of 1mm of dentine, which in permanent teeth represents time-averaged data covering a period of at least 9 months (Beaumont *et al.*, 2013). In deciduous teeth resolution is better, representing approximately 3 months of life (Beaumont and Montgomery, 2015). However, as deciduous teeth are generally only present in the jaws of individuals who died in childhood, the improved resolution offered by deciduous teeth is likely to limit high-resolution analyses at present to a subpopulation which may differ from the wider population (cf. Wood *et al.*, 1992).

This lack of precision attached to current analysis of permanent teeth, during a process in which significant dietary shifts may be expected to take place within a much shorter timeframe, raises some concerns about the ability of archaeological studies using these methods to accurately describe the timing of early life dietary transitions. Nonetheless, the life-history approach offered by incremental dentine methods offers a vast improvement over the potentially much poorer resolution of bone collagen sampling and is again an improvement on previous studies using much lower-resolution incremental sampling (cf. Fuller *et al.*, 2003; Eerkens *et al.*, 2011). Furthermore, this area of research is on the cutting edge of method development, with continuing efforts to explore improvements to microsampling methods (Burt and Garvie-Lok, 2013; Beaumont *et al.*, 2014; Guiry *et al*., 2016; Czermak *et al*., 2018).

Incremental sampling of dentine for palaeodietary study in humans has predominantly, but not exclusively, focused on dietary transitions of childhood, such as the patterns of breastfeeding and its cessation, due to the chronological period represented by dental development. The heavy bias toward the study of infant diet is not exclusive to increased-resolution human studies but is also demonstrated in studies in

which researchers have applied biogeochemical method to the elucidation of weaning patterns in animal domesticates, particularly cattle (Balasse and Tresset, 2002). Notable exceptions to this focus on mother-infant dietary relations have been demonstrated by studies which have concentrated on broader questions of overall population dietary transitions and environmental resource exploitation (Montgomery *et al.*, 2013), intra-population variation and differential resource access during early adulthood (Eerkens *et al.*, 2016), or the use of high-resolution sampling of animal teeth as a proxy for seasonal palaeoclimatic fluctuation (Kirsanow *et al.*, 2008). Still, the use of high-resolution sampling in biogeochemical research remains largely driven by a core agenda of addressing the nature of early life diet and its impacts on health.

2.6 Breastfeeding and isotopes

The sections following will outline the context of developing thought in infant palaeodietary studies, the analytical methods which have been used to address questions around early life dietary transitions, and the future of the discipline.

Interest in early life diet and health has almost entirely focused on transitions associated with breastfeeding and weaning. Trophic level increases have been observed in the isotope ratios of tissues of breastfeeding infants, as the human milk they are consuming is a product of their mother's body. Experimental studies of modern mother-infant pairs sampling fingernails or paired fingernails and hair samples, which are tissues whose isotopic composition primarily reflects the protein content of diet (Fuller *et al.*, 2006a), have demonstrated elevated $^{13}C/^{12}C$ and $^{15}N/^{14}N$ ratios among exclusively breastfed infants relative to those of their mothers. The demonstrated magnitude of this "breastfeeding effect" has varied. An early study by Fogel *et al.* (1989), which examined longitudinal data from a single mother-infant pair alongside a larger cross-sectional cohort, found that the fingernails of exclusively breastfed infants had $\delta^{15}N$ values that were on average 2.4‰ higher than those of their mothers, with no parallel increase in $\delta^{13}C$. The same study also analysed archaeological bone from two prehistoric North American sites, finding a consistent $\delta^{15}N$ increase of 2.5-4‰ for infants at one year of age, relative to adult bone analysed from the same sites. A more recent re-examination of the breastfeeding isotope effect by Fuller *et al.* (2006a) analysed longitudinal fingernail and hair data from eight mother-infant pairs and found a comparable trophic level shift for $\delta^{15}N$, with a ~2-3‰ (range 1.7-2.8‰) increase for infants over maternal values which began soon after birth, and peaked just before the

onset of, or during the early stages of, weaning onto alternate food sources. In contrast to the Fogel *et al.* (1989) study, Fuller *et al.* (2006a) also found a $\delta^{13}C$ trophic level shift of approximately 1‰ for exclusively breastfed infants. Lesser shifts were observed for partially breastfed infants, with relative increase of $\delta^{15}N$ and $\delta^{13}C$ values of ~1‰ and ~0.5‰, respectively, with no elevation of values of any magnitude seen for non-breastfed subjects (Fuller *et al.*, 2006a). In all cases of infant breastfeeding, values declined over time to parity with maternal values as breastmilk was removed from the diet. The authors observed that carbon values declined more rapidly during the weaning process than nitrogen values during the same period, and that this lag might suggest that a shift towards maternal $\delta^{13}C$ values in infants may represent a better indicator of the introduction of solid foods, while decreasing $\delta^{15}N$ may be regarded as indicative of decreasing breastmilk intake. As the study analysed fingernails and hair, tissues which form over a short period and thus record high time-resolution data, it is unclear whether the same effects will be observable in commonly-preserved archaeological remains, such as teeth, as the present state of sampling methodology does not allow for such high resolution.

While the existence of a trophic level effect seen between comparable maternal and offspring tissues demonstrated by such studies is undisputed within bioarchaeology, the mechanisms of this effect remain unclear and subject to conflicting assumption. Proportional enrichment in the heavier isotopes of carbon and nitrogen seen between mother and infant has been interpreted as occurring due to fractionation at the maternal level, with milk synthesis by the breast resulting in a ^{15}N-enriched fluid, relative to her own tissues (Reitsema, 2015). Alternately, other studies such as the Fuller *et al.* (2006a) research have assumed metabolic fractionation at the infant level and have compared formula milk $\delta^{15}N$ and $\delta^{13}C$ values to offspring values, while not performing similar analyses for human milk, instead analysing solid maternal tissues exclusively. This neglect of human milk-tissue differences in studies which seek to define parent-infant spacing may stem from the assumption that human milk $\delta^{15}N$ and $\delta^{13}C$ values are in equilibrium with other maternal tissues (Reynard and Tuross, 2014).

However, the assumptions that isotopic shifts in human infants results universally from maternal metabolic activity during milk synthesis, or alternately, that keratinised or skeletal maternal tissues provide a valid isotopic proxy for infant dietary composition may both be called into question by existing research. Simultaneous $\delta^{15}N$ analysis and

amino acid quantification of infant hair and maternal milk in human subjects has demonstrated milk-infant hair spacing, suggesting that metabolic fractionation occurs in the infant's body, increasing hair $\delta^{15}N$ values by approximately 3‰ relative to maternal milk (Romek *et al.*, 2013). This evidence dispels the notion that enrichment of infant tissues in heavier isotopes is an uncomplicated reflection of maternal metabolic activity. This finding of fractionation at the offspring level dovetails with an ecological study by Jenkins *et al.* (2001) examining the relationship between maternal plasma, milk, and offspring plasma isotope compositions in eleven mammalian species, which demonstrated that while offspring plasma $\delta^{15}N$ and $\delta^{13}C$ are higher than maternal milk values, maternal milk values themselves reflect lower $\delta^{15}N$ and $\delta^{13}C$, relative to maternal plasma. These lower values were interpreted as being the cause of a smaller than expected $\delta^{15}N$ difference between maternal and offspring plasma of $+0.9\pm0.8$‰, and in some species no measurable increase in values (0.0 ± 0.6‰) over maternal plasma $\delta^{13}C$ (Jenkins *et al.*, 2001). The magnitude of increase was found to be species-dependent, with no clearly discernible pattern of broader ecological niche differentiation (*i.e.* herbivores vs. carnivores, hibernator species vs. non-hibernator species, etc.) demonstrated, leading the authors to emphasise the importance of environment, diet, and species-specific biology to understanding the fractionation processes driving mother-offspring isotopic differences.

Difficulties with the comparison of data created by analysis of ephemeral tissues such as modern hair or fingernails with the data of more time-averaged ancient tissues such as bone or teeth (Bocherens *et al.*, 2014), become yet more complex when the mechanisms driving observed fractionation between mother and infant tissues are poorly understood. Reynard and Tuross (2014) have pointed out that an additional assumption in palaeodietary breastfeeding studies is that milk isotope ratios will remain static over time. While this is an inherent hazard of inferring breastfeeding activity from the reference of maternal-infant tissue spacing, it is also highly unlikely to be the case. A recent longitudinal study of a single mother-infant pair, analysing fingernails and milk samples from the 4^{th} to 34^{th} week of breastfeeding, reflected variability of milk carbon stable isotope values not tied to maternal diet, as well as lower $\delta^{15}N$ and $\delta^{13}C$ values of milk relative to maternal and offspring keratin (Herrscher *et al.*, 2017). This fits with what we know of the macronutrient composition of human milk (discussed in Chapter 3). Milk composition is variable, but largely species-specific and adapted to

growth patterns: human infancy is adapted to optimal outcomes based on a long period of dependency with slow growth and a high demand for lipids to aid postnatal neurological development (Dewey, 2009). Consequently, human milk is relatively low in protein (with a bias in favour of whey), while being high in fat and carbohydrates (with fat and lactose providing ~90% of milk calories) (Butte *et al.*, 1984). There is, on weight of evidence, every reason to question that human milk values ought to reflect a trophic level shift.

These studies have suggested a couple of points which should be considered when interpreting breastfeeding and weaning patterns from human tissues:

1. Breastfed infants may, but will not certainly, have $\delta^{15}N$ and $\delta^{13}C$ values which are higher than those of their mothers, evidencing a form of trophic level effect.
2. The "you are what you eat, plus a few per mil" principle should be used with extreme caution in infant feeding, as while the existence of maternal-offspring enrichment appears to be valid in most cases, the full dynamics regulating trophic level shifts are currently unclear.

2.6.1 History of infant palaeodietary research

The study of breastfeeding and early life dietary practices in past populations has developed over several decades spanning the transition from the late 20th to the early 21st century. During that time, the focus of research, the questions posed, and the methods used to address them have shifted. The developments of the field, range of available methods, and current limitations have been reviewed ably elsewhere (Reitsema, 2013; Humphrey, 2014; Reynard and Tuross, 2014; Tsutaya and Yoneda, 2015; Britton, 2017). Early studies addressing "weaning" characterised breastfeeding status largely as an either/or proposition, estimated through population-level averaging and cross-sectional analyses of bone. Initially, these consisted of trace element analyses, which were rapidly displaced by the ascendance of carbon and nitrogen analyses of rib collagen. Although cross-sectional bone analyses remain common, in recent years the development of higher-resolution methods in human teeth and a longitudinal life-history approach to population studies have begun to address and ameliorate the biases inherent to the cross-sectional approach. It is currently unclear whether studies utilising incremental analyses of dental tissues will eventually supplant bone analyses in the

realm of infant palaeonutrition, but the continued development of microsampling techniques, the increased understanding of the importance of early life nutrition to overall population health, and an effusion of recent publications utilising these newer techniques to address past breastfeeding practices suggest that microsampling of incremental tissues may well represent the future direction of the discipline. The following sections will outline and discuss the development of early life nutrition and breastfeeding studies in archaeology, beginning with the origins of infant palaeonutrition in the broader context of population palaeonutrition.

2.6.1.i Trace element studies

Early attempts to elucidate infant feeding studies in the past emerged alongside interest in illuminating overall past dietary patterns, and particularly in reference to understanding the relationship between subsistence strategy, fertility, and breastfeeding patterns. These early studies focused on trace element analysis in bone, chiefly applying the analysis of strontium and calcium ratios (Sr/Ca). Strontium in the natural environment is distributed throughout the biosphere through degradation of underlying geological formations into the soils, and is further disseminated by streams and groundwater, where it is taken up by the root systems of plants, and their subsequent consumers throughout the food chain (Price, 1989). Due to the very small differences in relative mass between its isotopes, strontium is not readily fractionated, and ascends throughout the trophic levels of the food chain without significant alteration (Price *et al.*, 2002). The chemical similarities of strontium to calcium allow strontium to be readily integrated into bioapatites of humans and animals. Humans take in strontium through consumption of plant foods and animal products, with plant foods having the highest Sr/Ca ratios (Sillen and Kavanagh, 1982). The relationship between strontium uptake in plants and animals has been formerly applied as a proportional measure of meat versus plant consumption in past studies (e.g., Price and Kavanagh, 1982; Schoeninger *et al.*, 1984; Price, 1985), but this has been questioned as problematic by more recent studies (Burton and Wright, 1995; Burton *et al.*, 1999).

While strontium is environmentally present in the biosphere in measurable concentrations taken in through food and water sources, discrimination and regulation of strontium absorption relative to other elements takes place within the adult gut, the placenta and also at the mammary level (Dahl *et al.*, 2001; Pors Nielsen, 2004). By contrast, infants and young children demonstrate poorly developed gut discrimination

against absorption of strontium, as discrimination is a progressive and age-linked development (Pors Nielsen, 2004). Analyses of umbilical cord sera and colostrum have clarified that while the maternal body positively discriminates in favour of calcium, using active transport, strontium is less prioritised, depending instead on a concentration gradient for transfer across membranes within the placenta and mammary gland (Rossipal *et al.*, 2000).

Knowledge of the dynamics of strontium and calcium in biological systems has been applied to the study of infant palaeonutrition as a means of addressing the timing of transition from the phase of exclusive breastfeeding to the period of complementary feeding. Some researchers have consequently argued that since Sr/Ca ratios are low in human milk, and gut discrimination against strontium underdeveloped in the young, ossified tissues should provide a sensitive indicator of the period of exclusive breastfeeding (Mays, 2003; Humphrey *et al.*, 2007; Humphrey, 2014). However, strictly speaking, this method cannot address the elimination of human milk from the diet, as it relies on markers of presence or absence of solid foods in the diet, rather than positive indicators of the consumption of human milk. While studies have attempted to use Sr/Ca ratios in this way, particularly in reference to stepwise changes in child population Sr/Ca ratios which correlate with increasing age prior to decreases related to the development of gut discrimination, the primary application of Sr/Ca ratio analysis is in its ability to classify whether or not infants appear to have been exclusively breastfed, and to what age. Despite this significant limitation, Sr/Ca ratio analyses in bone have received a moderate amount of application within infant palaeodietary studies (Sillen and Smith, 1984; Hühne-Osterloh and Grupe, 1989; Grupe and Bach, 1993; Mays, 2003).

Emerging recognition of problems inherent to analyses of Sr/Ca in bone, beyond simple mortality bias, appears to have restricted the breadth of application of this method to the characterisation of past breastfeeding patterns. Strontium and calcium ratios in bone, despite the early promise of their usefulness to the elucidation of weaning patterns in archaeological populations, have failed to receive extensive usage in infant palaeonutrition studies. This may be due in part to the subsequent primacy of carbon and nitrogen stable isotope analyses in infant palaeonutrition. However, concerns about the risks of bone diagenesis and the differential distribution of strontium within the human skeleton are likely to have played a more primary role in the narrow

application of this technique. Some researchers have suggested that diagenesis may be ameliorated through the use of solubilising solutions to extract diagenetic strontium, leaving behind only biogenic material (Sillen, 1986; Price *et al.*, 1992). Unfortunately, subsequent studies attempting to measure the effectiveness of solubilising diagenetic material and found such protocols ineffective (Tuross *et al.*, 1989; Trickett *et al.*, 2003).

Alternatives to removing diagenetic material have been suggested, such as screening protocols for the assessment of diagenesis, allowing for the exclusion of samples deemed to be diagenetically altered prior to analysis (Nelson *et al.*, 1986; Grupe and Piepenbrink, 1989; King *et al.*, 2011). However, other researchers have discouraged this approach, pointing out that where structural replacement of the bioapatite lattice has occurred, biogenic material may not remain, and chemical diagenesis may not always be readily apparent. For instance, typically-applied measures for evaluating diagenesis, such as crystallinity indices or calcium-phosphorus (Ca/P) ratios, may fail to indicate whether Sr content of bone (and hence Sr/Ca) reflects biogenic or diagenetic values, and thus should not be relied upon as proof of biochemical integrity (Burton *et al.*, 1999). Consequently, strontium and calcium ratio analyses in bone are now largely viewed as problematic. Sampling of bone for strontium and calcium ratios is further complicated by the finding that strontium is unevenly distributed within the skeleton, leading to a potential for sampling bias to skew interpretations (Grupe, 1988; Dahl *et al.*, 2001). Recognition of the difficulties associated with the analysis of Sr/Ca ratios in bone as a source of data on early life diet has led to an interest in less inherently problematic target materials. Tooth enamel, which is recognised as a more diagenetically-resistant tissue, due to its large crystals and low porosity, has become the gold standard of analytical materials. A comparison of diagenetic alteration of dentine and enamel by Budd *et al.* (2000) found that dentine, as well as bone, is highly diagenetically-vulnerable with high variability and little predictability, while core enamel was found to be remarkable in its maintenance of biogenic integrity. The authors consequently advised future researchers to avoid sampling both archaeological bone and dentine in favour of dental enamel.

Trace element analyses of enamel to address early life diet transitions have largely eschewed a cross-sectional bulk analysis approach utilised by bone analyses, instead applying high-resolution ablation techniques to the mapping of trace element distribution within incremental structures in enamel. To date, this has been

accomplished using both human and non-human analytical materials, including teeth from controlled experimental primate studies (Humphrey *et al.*, 2008b; Dirks *et al.*, 2010) and modern human teeth from groups of known dietary or developmental parameters (Dolphin *et al.*, 2005; Humphrey *et al.*, 2007; Humphrey *et al.*, 2008a). However, these methods have not been used topically to address archaeological populations. Instead, researchers appear to have prioritised the advancement of physiological knowledge and method development over applied study. The critique of Sillen *et al.* (1989) may thus have been taken to heart:

> …problems, affecting both isotopic and trace-element studies, mainly are due to the peculiar interdisciplinary nature of the field, rather than to any technological barrier. With minor exceptions, techniques developed largely in other sciences have been grafted on to archaeological problems. This no longer suffices because gaps remain in the scientific grounding of these techniques that need to be addressed before more complicated archaeological questions can be resolved. While the necessary studies may seem to be of little immediate anthropological interest, they are vital if continued progress in palaeodietary research is to characterize the years ahead (Sillen *et al.*, 1989: 504).

Consequently, methodological studies seeking to map trace element distributions in teeth (e.g., Kang *et al.*, 2004; Dolphin *et al.*, 2005; Alvira *et al.*, 2011; Hare *et al.*, 2011) have not sought to specifically address biocultural issues of infant nutrition or childhood diet. Instead, they have focused on building an understanding of the relationship between dental development and human biochemistry which produces the temporo-spatial differentials of tooth chemistry, an area which is at present poorly understood.

Although strontium and calcium ratios have received the greatest attention in trace element research on infant nutrition, measurement of relative abundance of other trace elements, such as barium, has been attempted. Barium, like strontium, is an alkaline earth metal readily integrated into skeletal bioapatites and has been measured relative to calcium in enamel in human, hominin, and primate models (Austin *et al.*, 2013). Also, like strontium, barium is subject to a principal of biopurification, where lower concentrations of barium are measured with each increase of trophic level, which is developmentally-acquired (Humphrey, 2014). Transport of barium across the placenta is very low, and higher levels are received via human milk, although changes to Ba/Ca are individual-specific as human milk concentrations vary (Austin *et al.*, 2013). Changes to Ba/Ca during the weaning process are expected to be much greater than those of Sr/Ca, as the body discriminates much more efficiently against barium than against strontium (Metcalfe *et al.*, 2010). Austin *et al.* (2013) have published the sole extant study using

Ba/Ca ratio data from microsampled tooth enamel as an indicator of weaning transitions in modern children, captive macaques, and a single juvenile Neanderthal. Temporospatial mapping of Ba/Ca patterning in both enamel and dentine revealed a high level of correlation across regions forming during the same time period. The findings of the Austin *et al.* (2013) paper ran counter to expected patterning during weaning; the data suggested a relative rise in Ba/Ca from *in utero* levels, with a subsequent drop in Ba/Ca during weaning. Tsutaya and Yoneda (2015) have interpreted the increase in Ba/Ca in breastfed infants, where a trophic level biopurification effect would have predicted a drop, as a reflection of bioavailability rather than increased Ba concentrations. The correlations described between Ba/Ca ratios in modern human and macaque dentine and enamel microstructures are intriguing, as they suggest that, in the case of barium at least, enamel microstructures may provide a reliable guide to the chronology of inputs. This is significant, as barium is known to be vulnerable to post-mortem diagenesis (Ericson, 1993). Thus, for all trace element analyses, the future of the method is likely to emerge from enamel analyses, and in improved understanding of the timescales of biochemical inputs. The emergence of high-resolution elemental enamel analyses represents the cutting edge of methodological development, and it is currently unclear whether this technique will offer an exciting new avenue of dietary data while avoiding the weaknesses of bone analyses, or whether it will simply offer different challenges.

2.6.1.ii Carbon and Nitrogen

Following on from early attempts at understanding past breastfeeding patterns through analysis of trace elements in bone, by the close of the 1980s, researchers began to apply their knowledge of trophic level effects in carbon and nitrogen stable isotope foodweb distributions to the issue of infant palaeonutrition. This specific and directional application of emerging methods in biogeochemistry developed in parallel with an unprecedented interest in the archaeology of children and these should be seen as related developments. The historian John Boswell (1984) had noted both a surge in interest in the lives of past children among 20th century historians, and a frustrating paucity of documentary evidence with which to address questions arising from that interest. By the late 1980s, Grete Lillehammer (1989) published her seminal work "A child is born. The child's world in archaeological perspective," acknowledging the place of the child in historical research, and arguing that children merited equal attention

within archaeology, and that osteological evidence offered promising avenues of future inquiry. This increased interest in the lives and health of children as cultural participants among historians, archaeologists, and bioarchaeologists has alternately fuelled and been fuelled by developments in palaeodietary study, and most markedly in the area of past infant diet.

Analyses of bone

Rather than an explicit focus on the lives and health of children for their own sakes, early studies linked the importance of the weaning process with infant mortality and the implications of each for maternal fecundity and population demography. The undisputed presence of children in the archaeological record is acknowledged to be at odds with their nearly total lack of impact upon broader archaeological thinking, a problem which continues to the present (Lillehammer, 2010). As a result, early research can arguably be said to have focused not on children themselves, but on the child-shaped spaces which they occupied in ancient society. This is in evidence in osteodemographic publications of the 1980s and early 1990s where breastfeeding duration was deemed significant as a potential indicator of fertility, which itself was valued as a measure of changes to subsistence strategy (e.g., Buikstra *et al.*, 1986; Holland, 1989; Bentley *et al.*, 1993).

In the same year as Lillehammer issued her call to recognise the importance of children in the past, Fogel *et al.* (1989) published the earliest study to apply nitrogen stable isotope analysis of bone collagen and knowledge of trophic level effects to the detection of past breastfeeding patterns. In addition to the methodological work carried out using fingernails from modern mothers and infants, which was discussed earlier in this chapter, the researchers also analysed archaeological bone from pre- and post-horticultural populations in order to identify the impact of subsistence strategy on breastfeeding patterns in those populations, finding an age-related increase in $\delta^{15}N$ values, but no corresponding increase in $\delta^{13}C$ values. These discoveries thus confirmed the biochemical effects of breastfeeding on the collagen composition of infants and supported the use of nitrogen stable isotope analyses as a valid approach for examining past dietary patterns of young children.

Within the archaeological component of the study, Fogel *et al.* (1989) set a pattern for cross-sectional population weaning studies in years to follow. In these, bone is sampled from non-adults from a range of ages, and data obtained from these analyses is

plotted by age and isotope against a standard of adult values from the same population, creating an artificial aggregate population through which to formulate a weaning curve (Figure 2.2). Since breastfed infants and children have $\delta^{13}C$ and $\delta^{15}N$ collagen values which are elevated, relative to the values of their mothers, cross-sectional bone studies have used mean adult bone values as the standard from which relative enrichment is measured. The highest point in an aggregate weaning curve is deemed to be the point at which solids are introduced, and the age at which plotted child values fall within the range of adult values is assumed to be the age at which cessation of breastfeeding occurs within that population (Reynard and Tuross, 2014). This technique, and the inherent model associated with the use of this methodology, has represented the dominant mode of infant palaeodietary research for over 20 years.

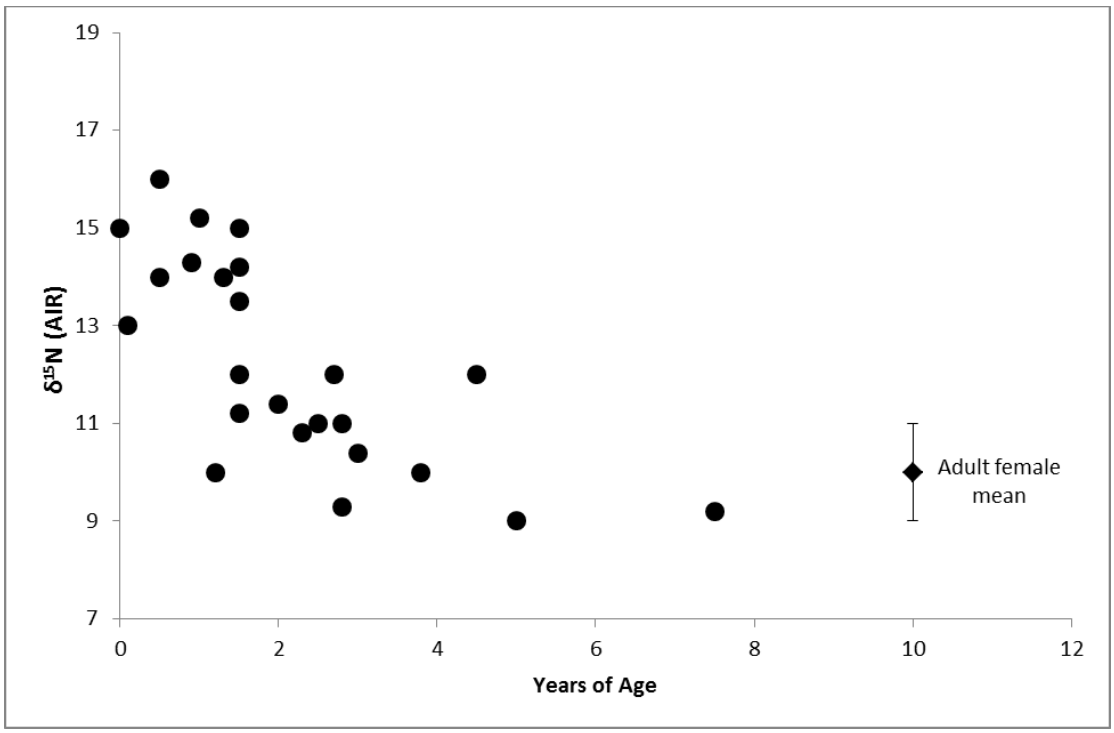

Figure 2.2. Mock weaning plot in a cross-sectional (rib collagen) $\delta^{15}N$ model

The findings of Fogel *et al.* (1989) offered an unprecedented and tantalising quantitative means of accessing information about past infant feeding patterns, as well as spurring additional work towards understanding both age-related isotopic patterning in archaeological populations and the bi-directional impacts of subsistence on population breastfeeding patterns. Katzenberg (1993) analysed carbon and nitrogen stable isotope data from three Canadian populations spanning protohistoric and historic periods and found that for all populations, independent of the variability of diet seen between populations, significant correlations existed between age of less than two years

and higher bone collagen $\delta^{13}C$ and $\delta^{15}N$ values were found. This was attributed to the consumption of ^{13}C-enriched foods during early childhood, and in the case of $\delta^{15}N$, to breastfeeding. During the immediate period which followed the Fogel *et al.* (1989) study, this trend towards examination of the relationship between breastfeeding patterns and subsistence strategies, particularly agriculture, continued as a strong theme which encompassed prehistoric North American maize agriculturalists (Katzenberg *et al.*, 1993; Schurr, 1997), the diets of North American Plains villagers (Tuross and Fogel, 1994), the importance of maize agriculture to Mayan diet (White and Schwarcz, 1989), and transitions in crop utilisation among Sudanese Nubians between the Meroitic to Christian periods (White and Schwarcz, 1994), among others.

A prominent exception to the broader preoccupation with prehistoric changes to subsistence was the palaeodietary investigation into the 19th century historic population interred at St. Thomas's Anglican churchyard in Ontario, Canada (Katzenberg and Pfeiffer, 1995; Herring *et al.*, 1998; Katzenberg *et al.*, 2000). Completed over several years and producing several papers, this study aligned historical documentary evidence with carbon and nitrogen isotope analyses of bone and known foodstuffs to take a comprehensive approach to addressing dietary patterns within this population. Aside from the unique opportunity to apply multiple lines of evidence offered by a historic population, the papers which emerged from the St. Thomas's study diverged from previous published work in two ways. Firstly, they were the first to explicitly suggest that $\delta^{13}C$ increases of approximately +1‰ could be attributed solely to a breastfeeding effect (Katzenberg *et al.*, 2000). Like the original Fogel *et al.* (1989) study, previous studies had alternately found no relationship between higher $\delta^{13}C$ and age (White and Schwarcz, 1989; White and Schwarcz, 1994), had exclusively focused (or reported) on $\delta^{15}N$ values (Schurr, 1997), or had interpreted higher $\delta^{13}C$ values among infants and young children as being solely due to consumption of specific weaning foods such as maize (Katzenberg, 1993; Katzenberg *et al.*, 1993).

Secondly, the formulation of "weaning age" as an event, popular among archaeologists and stable isotope researchers, was overtly deconstructed and addressed for the first time by Katzenberg *et al.* (1996). The terms "weaning" and "weaning age" were identified as representing a problematic tendency within archaeology to view "weaning" (i.e. the cessation of breastfeeding) as an event, rather than a process which unfolds gradually over time, beginning with the transition from exclusive breastfeeding

to the introduction of a complementary diet, and continuing through an often lengthy transitional phase of dual dependence prior to the final cessation of breastfeeding. Katzenberg *et al.* (1996) drew particular attention to this presumption of weaning-as-event as a characteristic feature of the published literature on nonspecific skeletal stress markers in past populations, where it was commonly assumed that "weaning stress" was implicated in the development of skeletal markers of developmental arrest, particularly enamel hypoplasia (cf. Cook and Buikstra, 1979; Goodman *et al.*, 1984; Corruccini *et al.*, 1985), without clarification regarding which stage of weaning was expected to produce such stress. Wood *et al.* (1992) had previously addressed the issue of non-representative selection of skeletal populations, more commonly known as mortality bias, or the "osteological paradox", and this was acknowledged by Katzenberg *et al.* (1996) as presenting a particular challenge to the interpretation of breastfeeding patterns in past populations where the group of study was by definition abnormally frail. This, the authors reasoned, presents a significant limitation to the interpretation of past breastfeeding patterns based on analyses of bone, as it is possible that frailer children experienced different patterns of weaning, or even that the pattern of weaning may be complicit in child mortality. A more complete discussion of the assumptions surrounding stress and the putative "weanling's dilemma" and an evaluation of these assumptions in light of extant clinical and anthropological data appears in Chapter 3. The ambiguity of language surrounding "weaning;" which may alternately imply the introduction of complementary foods, the final cessation of breastfeeding, or the entirety of the process; and the implied synchronicity of the term "weaning age" in reference to what is nearly always a diachronic process has been challenged by many other anthropologists and archaeologists since that time (e.g., Herring *et al.*, 1998; Schurr, 1998; Millard, 2000). However, these terms and implied concepts continue to be used without definition or qualification, an issue which could be easily ameliorated through clarification and precision of language.

By the outset of the 21st century, carbon and nitrogen analyses of bone were well-established as the preferred method for assessing past breastfeeding patterns. In contrast to the formative publications of the 1990s, papers published after the year 2000 lack the concerted focus on agricultural transitions, particularly those of North American populations, characteristic of the earlier period. This may in part be due to studies appearing during this period which failed to confirm a long-held hypothesis: namely,

that adoption of agriculture or sedentism resulted in shorter breastfeeding duration (e.g., Sellen and Smay, 2001; Schurr and Powell, 2005; Clayton *et al.*, 2006). However, a review of the broader bioarchaeological literature of this period alongside the palaeodietary literature suggests that a trend towards the adoption of a life history approach with a high value for the social context of past lives (Larsen, 2002; Wright and Yoder, 2003; Knudson and Stojanowski, 2008) developed during the first ten years of the 21st century. This influence was bidirectional. Palaeodietary data had become integral to informing interpretations of archaeological population health, particularly within the realm of nutritional deficiency. Equally, the subtle shift towards an appreciation of the qualitative nuancing provided by the acknowledgement of social meaning and status and away from simple quantification of diet which is in evidence during this period is a clear indication of how interrelated research trends between bioarchaeology and its palaeodietary subfield were. Resembling the increased profile given to breastfeeding studies by the raised status of children in archaeology during the 1980s and 1990s, the increasing appreciation for a life history model bolstered interest in characterising the lives experienced by past children.

Interest in purposefully targeting multiple and discrete phases of the lifecourse led to early attempts at distinguishing the dietary patterns through stable isotope analysis of selected elements and fractions of human bone. While the idea of sampling skeletal tissues forming at different developmental stages of the lifecourse originated much earlier (e.g., Sealy *et al.*, 1995), Bell *et al.* (2001) was the earliest to apply bone density fractionation, a technique developed to measure bone maturation in ecological studies, as a possible means of sampling archaeological human bone in order to characterise changing diet over the lifecourse through variable rates of turnover throughout the skeleton. Jørkov *et al.* (2009) similarly explored the feasibility of sampling select portions of the skeleton in order to produce early life $\delta^{13}C$ and $\delta^{15}N$ data for adults and children, finding that while values obtained from rib and femur collagen were highly variable, $\delta^{13}C$ and $\delta^{15}N$ data obtained from the inner petrous bone of the skull was comparable to values obtained from first molar dentine. Thus, the petrous bone, which does not remodel after the age of approximately two years of age, may represent a record of early diet comparably secure to that offered by analysis of teeth (Jørkov *et al.*, 2009). It is important to note that during this decade, the first investigation striving for increased-resolution sampling using analyses of teeth was undertaken by Fuller *et al.*

(2003). However, despite the increased value for longitudinal perspectives on health and diet evidenced by these early forays into increasing resolution, high-resolution sampling (discussed at length in the following section) did not become a characteristic feature of palaeodietary study at this juncture.

In contrast to the emphasis on the relationship between prehistoric population subsistence strategy and breastfeeding patterns which characterised the formative infant palaeodietary studies of the late 20th century, research published in the first decade of the 21st expanded in both geographical scope and topical emphasis. Contextual culture-specific approaches to the interpretation of infant feeding patterns, with broader application of stable isotope analysis to historic and proto-historic populations, prevailed more heavily than in the previous decade. After the year 2000, an increased focus on ecological perspectives; particularly the impacts of disease ecology and overall population nutrition on childhood health, was in evidence. This concern with disease ecology was also manifest within these studies as a reflection of perceptions within bioarchaeology of breastfeeding as an activity with implications for host defence and nutrition (Dittmann and Grupe, 2000; Schurr and Powell, 2005; Jay, 2009), or alternately, malnutrition (Keenleyside and Panayotova, 2006; White *et al.*, 2006; Walker *et al.*, 2009). In terms of geographic and chronological breadth, studies of the early 2000s addressed populations as diverse as medieval Europe (Dittmann and Grupe, 2000; Mays *et al.*, 2002; Privat *et al.*, 2002; Richards *et al.*, 2002), Postclassic Mayan communities in Belize (Williams *et al.*, 2005; White *et al.*, 2006), Roman and post-Roman Britain (Fuller *et al.*, 2006b), Imperial Rome (Prowse *et al.*, 2008), Iron Age Britain (Jay *et al.*, 2008), pre-and post-agricultural eastern North America (Schurr and Powell, 2005), medieval Nubians in Sudan (Turner *et al.*, 2007), the early Christian community in Rome (Rutgers *et al.*, 2009), and prehistoric Pacific islanders (Kinaston *et al.*, 2009). By this period, most bone studies exercised a standard methodology, with the majority of novelty in publications arising from the interpretive insights that unprecedented sources of data brought to their respective research areas. However, this is not without exception. Kinaston *et al.* (2009) took a unique approach to $\delta^{13}C$ and $\delta^{15}N$ analyses by attempting to use breastfeeding-related isotopic trends as a method of identifying neonates in a sample of perinatal infants at prehistoric Vanuatu, an approach which was ultimately unsuccessful, but which identified a pattern of increased $\delta^{15}N$ ultimately attributed to intrauterine stress.

While the first decade of the 21st century represents a period in which carbon and nitrogen isotopic analyses had expanded to become the dominant source of quantitative dietary data for early childhood and had been employed in a range of periods and geographical contexts, the breadth of application which infant palaeodietary research received during this period may be regarded as somewhat limited relative to the decade to follow. In contrast to the low volume of studies and stable pattern of isotopic palaeodietary research published from 1990-2010, a substantial increase in the volume of infant palaeodietary research post-2010 has reflected an increased level of interest generally in children (Figure 2.3). This expansion of research may be seen as part of a larger enthusiasm for investigating childhood diet, and not merely as a result of improvements in method. Although the largest relative growth in publications has occurred in the area of tooth analyses, and specifically the advances of incremental sampling of teeth, which may be attributed to the improvements in sampling and chronological resolution achieved in the present decade, the concurrent and continuing increase in carbon and nitrogen studies in bone, which have not altered appreciably in method over the last few decades, suggests that the primary driver of increased study is topical interest and accessibility of analysis, not alterations to method.

This increased interest has been fuelled by new understanding of the importance of child health for long-term and population health offered by revolutionary theories such as the Developmental Origins of adult Health and Disease (DOHaD) introduced by Barker *et al.* (2002) (also cf. Barker, 2004), as well as emerging research on the role of the infant gut microbiome to health and immunity (Bäckhed *et al.*, 2015; Bode, 2015), and the essential role played by breastfeeding in the establishment and maintenance of active host defence (Garofalo, 2010; Riskin *et al.*, 2012; Cabinian *et al.*, 2016) (see Chapter 3 for a review of the clinical evidence regarding immunological effects of breastfeeding). Alongside these advances in clinical medicine which have underlined the importance of studying early life care as an essential component of understanding population health, a wealth of 21st century archaeological scholarship focusing on the lives of children as active cultural participants (e.g., Kamp, 2001; Baxter, 2005; Lewis, 2007; Hadley and Hemer, 2014), has both reflected and fuelled an unprecedented appetite for improved insight into the lives of this understudied and essential foundation of society.

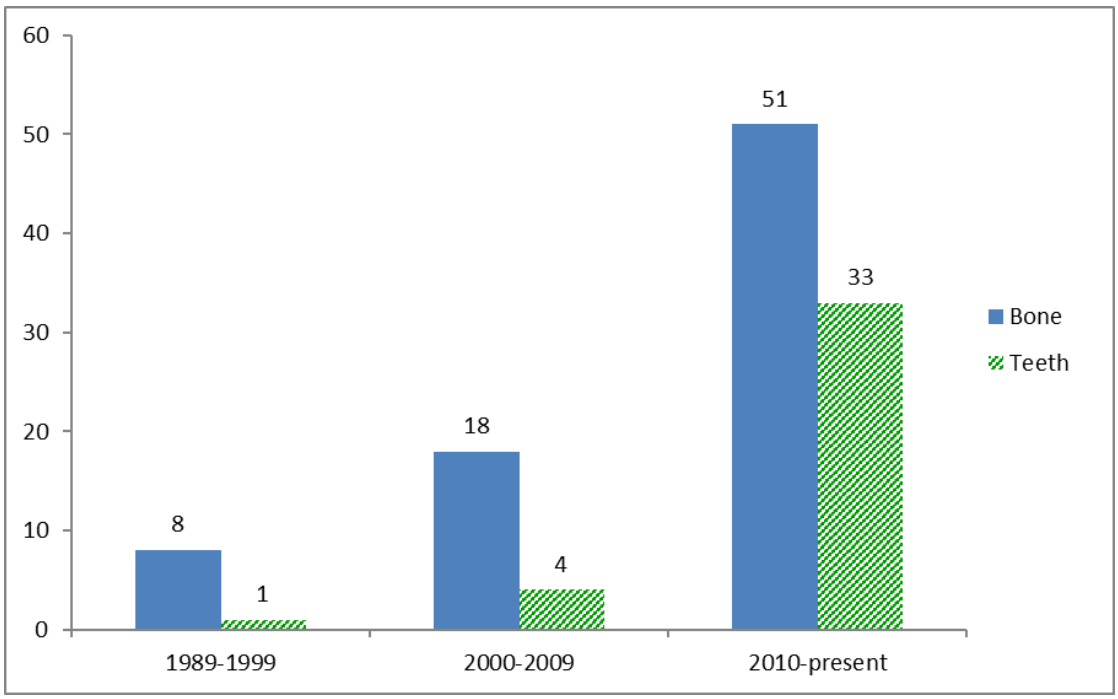

Figure 2.3. Number of Published Infant Palaeodietary Studies Using Carbon and/or Nitrogen by Decade (data obtained via Google Scholar search June 12, 2018, search terms "archaeology", "weaning", "stable isotope", "collagen", "infant diet", and "breastfeeding")

Despite the profusion of publications emerging from the infant palaeodietary subdiscipline, an increasing geographic bias is evident in recent decades. While nearly all early studies were centred on North American populations, the dominance of North American datasets waned by the outset of the 21st century. Some have attributed this profusion of New World research, followed by a sudden decline, to the impacts of the 1990 Native American Graves and Repatriation Act (NAGPRA), which has led to the cataloguing and return of Native American human remains previously held by museums and repositories. Rose *et al.* (1996) reported that significant quantities of isotopic data were collected as an integral part of the repatriation process itself, while Katzenberg (2001) describes the impacts of repatriation legislation as a de facto prohibition on destructive sampling of Native American human remains, narrowing the field of opportunity in isotopic research in North America. This has led to alternate means of accessing prehistoric dietary data, such as the use of domesticated dog remains as palaeodietary proxies (Guiry, 2012), which are useful for understanding population diet but cannot provide data on breastfeeding practices. While a small number of New World-based palaeodietary studies have continued to emerge after 2010 (e.g., Leventhal *et al.*, 2011; Eerkens and Bartelink, 2013; Tessone *et al.*, 2015), the vast majority of research has focused on Old World populations.

Old World infant palaeodietary studies have been chronologically and geographically diverse, with Asian populations ranging from Anatolian (Pearson *et al.*, 2010; Pickard *et al.*, 2016) to Far Eastern (Choy *et al.*, 2010; Waters-Rist *et al.*, 2011; Tsutaya *et al.*, 2015; Xia *et al.*, 2018) and European assemblages varying from the prehistoric (Howcroft *et al.*, 2012; Ash *et al.*, 2016), to Classical antiquity (Nehlich *et al.*, 2011; Redfern *et al.*, 2012; Schmidt *et al.*, 2016), the medieval (Mays, 2010; Bourbou *et al.*, 2013; Burt, 2013; Giuffra and Fornaciari, 2013; Haydock *et al.*, 2013; Kaupová *et al.*, 2014), and postmedieval (Nitsch *et al.*, 2011; Beaumont *et al.*, 2012). The historic populations of medieval and postmedieval Europe and Asia have achieved particular prominence in recent work; this may be due in part to the advantages offered by extant documentary evidence to complement isotopic data.

Trends observed in recent publications have built upon those of previous decades. In apparent response to prior critique, nearly all research in the present makes explicit acknowledgement of weaning as a process, rather than an event, regardless of whether this acknowledgement is reflected within interpretation of the data. In many cases, this recognition of process is accompanied by a further recognition that the process of weaning, and the period of complementary feeding, may be in many cases gradual and lengthy and involve considerable variation within and between cultural groups (Choy *et al.*, 2010; Howcroft *et al.*, 2012; Tessone *et al.*, 2015). This growing appreciation of the variability and biocultural implications of individual patterning of breastfeeding and weaning practices within societies, fuelled by the increasing popularity of the life history approach, has resulted in a rapid growth of higher-resolution studies which use longitudinal, rather than cross-sectional datasets, to characterise individual-level shifts in diet.

In addition to the positive advantages of newer longitudinal methods for assessing intra-individual variability, recognised pitfalls of the more traditionally-widespread cross-sectional approach may also be motivating the steeply-increasing use of high-resolution methods for analysis of past breastfeeding patterns. All isotopic analyses employing a cross-sectional sample of juvenile bone in order to characterise population norms suffer from the common problem of mortality bias. This issue, which has been considered from not only the first critique by Wood *et al.* (1992), as well as discussed at length since that time by several reviews (cf. Reitsema, 2013; Reynard and Tuross, 2014; Beaumont *et al.*, 2015; Kendall, 2016), has also been acknowledged as an

inherent limitation by many of the studies using cross-sectional methods, albeit with varying levels of concern towards the risk of mortality bias as a confounder (Dupras *et al.*, 2001; Mays *et al.*, 2002; Waters-Rist *et al.*, 2011). The "snapshot" of data provided by a single age-at-death isotopic value derived from a victim of child mortality should be considered suspect as a measure of norms of care within a population; such data do not give a true estimate of relative relationship to maternal values, nor to relative change prior to death. Additionally, the known effect of systemic stress on $\delta^{15}N$, in particular, suggest that analyses which sample the individuals within a population who are most likely to be stressed, without benefit of longitudinal data with which to differentiate dietary change from pathological change, should be suspect (Beaumont *et al.*, 2013; Beaumont *et al.*, 2015).

In keeping with the value for life history and individual variability expressed by recent research, it is important to acknowledge the extent to which the use of an aggregate approach may obscure intra-individual variability, and thus calls into question the validity of these methods for characterising past dietary transitions which are inherently variable and observed as a function of relative enrichment to a heterogeneous maternal standard (Jay *et al.*, 2008; Reynard and Tuross, 2014; Kendall, 2016). Recent studies comparing cross-sectional analyses of bone with incremental dentine analysis have called the use of bone analyses for weaning study into question, concluding that high levels of adult dietary variability may preclude interpretation of breastfeeding and weaning patterns (King *et al.*, 2018b) and that bone collagen presents unreliable record of diet and physiology for stressed individuals, such as non-survivors (Beaumont *et al.*, 2018). Recognition of the limitations created by potential non-representativity of data in cross-sectional studies together with demands for increased resolution and identification of intra-individual patterns of variability, which may be regarded as a development resulting from the popularity of the life history approach, have led to the development and increasing usage of high-resolution analyses of teeth, which will be discussed in the section to follow.

High resolution studies of teeth

Wright and Schwarcz (1998) carried out the earliest study attempt to increase the resolution of infant palaeodietary data using teeth. This study applied $\delta^{13}C$ and $\delta^{18}O$ data (use of oxygen stable isotopes will be discussed in the next section) from bulk-sampled enamel carbonate to the elucidation of breastfeeding patterns in a prehistoric

sample spanning multiple cultural transitions in Guatemala, albeit continuing to utilise the cross-sectional model prevalent at the time through its emphasis on population-level patterning. Systematic trends in $\delta^{13}C$ and $\delta^{18}O$ values were demonstrated to occur with increasing age of formation in the permanent first molars, premolars, and third molars analysed, interpreted as signs of the onset and progression of weaning (Wright and Schwarcz, 1998). Two particularly pioneering elements of this study were the researchers' recognition of the potential of analysis of teeth from adult individuals as a means of obtaining information on childhood diet while circumventing mortality bias, and the innovative analysis of multiple teeth with overlapping ages of formation in order to produce a timeline of data. A paper published the year following by Wright and Schwarcz (1999) applied similar methodology to the analysis of bulk crown dentine in the teeth of individuals from the same Guatemalan study population, but, in contrast to the earlier study, tracked individual trajectories of change between the selected teeth. The resolution offered by this method was low, suggesting only that solid foods were introduced at an unspecified point prior to two years of age, while the interpretation of the final conclusion of weaning was limited to an estimation of it occurring "…at an older age, perhaps four years" (Wright and Schwarcz, 1999: 1159). Another significant drawback of the method included the need for destructive analysis of multiple teeth, presenting bioarchaeologists with an ethical dilemma. Despite the limitations of resolution offered by these formative analyses of dental tissues, Wright and Schwarcz (1999) offered the first indication of the potential for longitudinal analysis, and in doing so additionally demonstrated within their sample the potential to demonstrate substantial variability of not only breastfeeding patterns, but complementary diet.

Fuller *et al.* (2003) pushed the development of increased-resolution study further in the first incremental analysis of dentine, which derived $\delta^{13}C$ and $\delta^{15}N$ data from deciduous and permanent human dentine at medieval Wharram Percy. This study sectioned deciduous second molar, permanent canine, and permanent third molar dentine into crown, cervical root, and apical root segments, while sectioning a further sample of permanent canines and third molars into crown, cervical root third, middle root third, and apical root third dentine parts for analysis. Like the previous cross-sectional studies, Fuller *et al.* (2003) was focussed on "weaning age" as an event, also using rib collagen data as supporting evidence, estimating this milestone to have taken place by approximately two years of age. Mortality bias was considered and addressed

in the study as a commonly raised concern of studies based on infant remains, but ultimately rejected as an issue based on agreement between data derived from non-adult rib collagen and deciduous second molars. The authors reasoned that survival beyond the cessation of breastfeeding demonstrated by second molar data indicated that breastfeeding practices were unlikely to be implicated in mortality. This reasoning unfortunately failed to recognise that as deciduous molars are only present in individuals dying prior to adolescence (Al Qahtani *et al.*, 2010), this may be indicative of morbidity, if not mortality, which has the potential to have influenced breastfeeding pattern and duration in these children. Application of the longitudinal aspect of Fuller *et al.* (2003) to the purposes of interpreting past breastfeeding patterns was limited by the size (n=8 individuals) of the study sample as well as the primary emphasis on methodological demonstration of generally decreasing values with increasing age of formation. This study may thus be seen as something of a cross-sectional/longitudinal hybrid: informed by the ethos of contemporary cross-sectional study but providing the necessary groundwork for future study in incremental dentine analysis by confirming that human dentine records a temporospatially-sensitive record of diet.

As with the studies by Wright and Schwarcz (1998; 1999), the study by Fuller *et al.* (2003) offered improved, but not high, time resolution of data, with increments representing in excess of one year of life in deciduous teeth, and several years for permanent teeth. Subsequent research using dentine did not improve upon this resolution; either using bulk dentine in conjunction with rib collagen data in reliance on a cross-sectional approach to characterising breastfeeding patterns (Clayton *et al.*, 2006), or applying the bulk sampling, multiple-tooth approach pioneered by Wright and Schwarcz to appraise population, rather than individual patterning (Dupras and Tocheri, 2007). The exception to this period of methodological stasis was an unpublished master's dissertation by Holt (2009), which improved upon Fuller *et al.* (2003)'s resolution by bisecting both crowns and roots of first and second deciduous molars, as well as first permanent molars, creating four increments. This sampling method represented a significantly improved optimum, in reference to earlier work, of approximately nine months of life for the deciduous molars and eighteen months for the permanent molars.

However, despite the importance of the work by Fuller *et al.* (2003), significant uptake of high-resolution dental methods through incremental dentine sampling did not

occur until the present decade. Eerkens *et al.* (2011) published their microsampling protocol based on six individuals from a central Californian prehistoric population, using first permanent molars from five individuals, and first, second, and third permanent molars from a sixth individual in order to construct a dietary timeline from birth to early adulthood. In sampling only permanent teeth, Eerkens *et al.* (2011) were able to bypass not only the pitfalls of sampling victims of infant mortality, but also childhood mortality, by selecting for teeth present in adults. Not only was the total departure from sampling non-adult remains for the estimation of dietary transitions a noteworthy change, but the study was also able to increase the number of increments to 5-10 per tooth, producing a resolution of approximately a year per increment as an optimum (for 10 increments, reflecting $1/10^{th}$ formation time). While this resolution is not greatly superior to that offered by the method of Fuller *et al.* (2003), due to the longer formation time of permanent molars, relative to the period of development of deciduous molars sampled by the Fuller et al. study, the sampling method of Eerkens *et al.* (2011) offered a clear advance over prior methods through increasing the possible number of increments per tooth, and also through demonstrating the validity of studying childhood dietary patterns through analysis of teeth from adults.

The impact of Eerkens *et al.* (2011) was not immediate and has not been universally pervasive. In addition to the continuation of cross-sectional bone analyses, analyses of teeth employing lower resolution methods and an aggregate population approach have continued to appear (e.g., Howcroft *et al.*, 2012; Reitsema *et al.*, 2016). Eerkens and Bartelink (2013) published a further study applying their methods to a larger sample (n=17), which improved the potential for resolution further by sampling demineralised dentine sections from cusp to apex at 1-2mm intervals, rather than a set number of 10 increments. In practice, this did not produce higher resolution within the second study. However, permanent tooth size in humans is highly variable, with genetic and environmental components (Dempsey and Townsend, 2001), and sampling of larger teeth would result in higher resolution. In addition to the contributions made by these researchers to improved resolution in incremental sampling of permanent dentine, the papers published by Eerkens *et al.* (2011) and Eerkens and Bartelink (2013) made significant use of cross-cultural and relevant culture-specific ethnographic data to inform the interpretation of their datasets.

Despite the impact of work by Eerkens and associated researchers, perhaps the greatest strides forward in method in recent years have come out of concurrent work at the University of Bradford by Beaumont *et al.* (2013), which examined infant feeding patterns in urban London and Irish workhouse cemetery populations affected by the Great Irish Famine of the 1840s. Like the work of Eerkens *et al.* (2011) and Eerkens and Bartelink (2013), this paper by Beaumont and colleagues shared a strong emphasis on elucidation of normative childhood dietary transitions based on sampling of adult "survivors" and integration of supplementary data sources, such as historical documentation. However, in addition to an emphasis on the importance of basing interpretation of culture-specific dietary norms on survivor data, Beaumont et al. also emphasised sampling of children as non-survivors to define differences in feeding pattern that may have occurred throughout the population. Furthermore, because the study population was known to represent Famine victims and survivors, Beaumont et al. could identify non-dietary patterns of change through co-variance in longitudinal $\delta^{13}C$ and $\delta^{15}N$ patterns, highlighting for the first time in infant palaeodietary study what had been demonstrated through hair study previously: the complicating and potentially confounding role of nutritional and systemic stress in palaeodietary study. Demonstration of physiological, rather than purely dietary, sources of variation have underlined the importance of longitudinal data in the interpretation of infant palaeodietary patterns, and it is probable that past weaning studies have conflated physiologically-elevated $\delta^{15}N$ values with indicators of trophic level and weaning status, due to the limitations of cross-sectional data.

Work by Beaumont and colleagues has also continued to improve sampling resolution and further understanding of the responsiveness of $\delta^{13}C$ and $\delta^{15}N$ values recorded in human dentine to physiological disequilibrium. Beaumont *et al.* (2013) decreased required sample size from 1-2mm increments of dentine to a maximum of 1mm throughout the tooth, by removing contaminants from samples using centrifugation, rather than yield-depleting ultrafiltration, within the collagen extraction procedure in order to increase collagen yield from smaller samples. Additionally, further work has offered the ability to distinguish $\delta^{13}C$ and $\delta^{15}N$ values from antenatally-forming dentine, reflective of the uterine environment and maternal values, from postnatally-forming dentine through the sampling and analysis of demineralised and lyophilised, but not denatured, cuspal dentine in deciduous teeth (Beaumont *et al.*,

2014). The high level of comparability between data derived from filtered and unfiltered samples, and denatured and undenatured collagen, reduces the amount of unprocessed dentine required for mass spectrometric analysis and suggests that higher resolution in future may be possible. Beaumont and Montgomery (2015) have also published a simple standard for assigning estimated age-at-formation to incremental dentine samples, a method of resolving a widely acknowledged problem in incremental analyses, which will facilitate and enhance cross-comparability of incremental dentine data.

Increased understanding of the physiological effects and trends discernible in high-resolution incremental dentine data has led Beaumont *et al.* (2015) to openly question the assumptions of the cross-sectional weaning model used in analyses of bone. Instead, the complexity of not only breastfeeding dynamics, but also physiological alteration to $\delta^{13}C$ and $\delta^{15}N$ values in early childhood, has been outlined through a re-evaluation of variability of prior incremental dentine data. Finally, the merits of longitudinal interpretive models have been extolled for distinguishing between effects of dietary and physiological shifts which have previously been interpretive as solely dietary in origin. The need for interpretive models for incremental dentine data which recognise and encompass physiological factors such as nutritional stress, has been re-emphasised by a recent paper by Beaumont and Montgomery (2016) which observed dentine $\delta^{15}N$ value increases during periods of nutritional stress within their Irish Famine populations, as well as corresponding $\delta^{13}C$ decreases during periods where carbon-depleted body fat was catabolised in place of dietary inputs. This study is the first in an archaeological population with known nutritional stress to demonstrate the characteristic isotopic covariance of catabolism previously observed in controlled experimental studies (e.g., Cherel *et al.*, 2005; Mekota *et al.*, 2006), and raises weighty questions about the validity of past attributions of isotopic ratio data to dietary factors alone, particularly in the case of cross-sectional analyses.

Several reviews have appeared in recent years, confirming the increasing awareness among researchers of physiological, as well as dietary, factors in shifts to early childhood carbon and nitrogen ratios, and the potential of high-resolution dentine studies to successfully navigate these newly-recognised obstacles to interpretation of carbon and nitrogen isotope ratios (Reitsema, 2013; Reynard and Tuross, 2014; Tsutaya and Yoneda, 2015). Despite such publications, application of the ideas and methods

offered by the work of Eerkens, Beaumont, and colleagues has been uneven, despite substantial increases in the prevalence of incremental dentine studies. Burt and Garvie-Lok (2013) published an alternative method of microsampling demineralised dentine which departed from the serial approach offered by Eerkens *et al.* (2011) and Beaumont *et al.* (2013) by using a punch technique to remove discrete samples from modern dentine formed before and after the demarcating neonatal line, in an attempt to circumvent the time-averaging represented by horizontal sampling of parabolic dentine incremental structures in the incremental method. While this study acknowledged the expediency of a longitudinal approach to addressing past breastfeeding patterns and recognised antenatally-forming dentine as a potential reflection of maternal health, it failed to equally recognise the potential for postnatal carbon and nitrogen ratios to equally reflect states of health beyond diet. Subsequently, the microsampling approach of Burt and Garvey-Lok has not proved as popular as the serial approach, potentially due to the less labour-intensive nature of serial sampling. To date, the Burt/Garvie-Lok method has not been utilised by further studies beyond a single application by Burt (2015), analysing a sample of exclusively deciduous teeth from medieval Fishergate, York.

Despite this singular attempt to increase sampling precision, dentine analyses published in recent years have almost universally employed incremental sampling, rather than discrete microsampling. These recent studies have demonstrated significant advantages over previous lower-resolution or cross-sectional research, in many cases identifying isotope patterning which was previously invisible or inaccessible through cross-sectional methodology relying exclusively on non-adult sampling. Montgomery *et al.* (2013) successfully applied high-resolution incremental sampling of permanent first and second molars, in a small sample from Neolithic Shetland, to the detection of fluctuations between marine and terrestrial protein inputs in childhood diet which were not otherwise evident in adult bone collagen data, nor in bulk dentine sample data. Small but significant sex-based differences in the $\delta^{13}C$ and $\delta^{15}N$ data of M1 incremental dentine profiles were observed in Henderson *et al.* (2014)'s analysis of a poor 18-19th century London cemetery population. These differences, made apparent through the longitudinal nature of incremental sampling, would not have been likely to be exhibited by more time-averaged rib collagen data, nor through the analysis of non-adult individuals, for whom morphological sex estimation is questionable.

The importance of viewing non-adults as a source of essential, but non-normative, data on the dietary patterns of cultural groups highlighted by Irish Famine incremental data was further supported by an analysis of a medieval Nubian cemetery sample by Sandberg *et al.* (2014), which explicitly compared cross-sectional $\delta^{13}C$ and $\delta^{15}N$ data from non-adult rib collagen to $\delta^{13}C$ and $\delta^{15}N$ data from incremental dentine analysis of permanent first molars and canines in order to examine the relationship between breastfeeding patterns and childhood morbidity and mortality. The study found a broad agreement between data derived from dentine forming in different teeth at the same age but observed discrepancies between cross-sectional and longitudinal $\delta^{13}C$ and $\delta^{15}N$ profiles within the population which were interpreted as earlier cessation of breastfeeding among survivors of childhood (Sandberg *et al.*, 2014). However, within this study the potential of physiological stress to mask and mimic continued breastfeeding through elevated $\delta^{15}N$ values among non-survivors was not considered, demonstrating the mixed penetration achieved by recent critiques of established infant palaeodietary theory. Similarly, incremental dentine studies by van der Sluis *et al.* (2015) and Yi *et al.* (2018), which claimed to identify shifts consistent with the end of the weaning process, failed to recognise opposing covariance of carbon and nitrogen profiles consistent with a period of stress, rather than dietary change. More positively, a recent study by Craig-Atkins *et al.* (2018) tapped into interest in variability, analysing deciduous teeth from non-survivors afforded different types of burial during the Christian early medieval period in Britain. The study found qualitative differences in patterning between children buried in child-specific clusters, relative to burials interspersed with adults, suggesting disparities in life experiences and care (Craig-Atkins *et al.*, 2018). Despite mixed evidence regarding uptake of advances in method and theory, increasing awareness of the importance of survivor data to the illustration of broad population norms and variability, and the analysis of non-survivor data as a measure of potential deviation from these norms, has been uniformly positive.

In summary, carbon and nitrogen stable isotope analyses for the investigation of past infant and childhood dietary patterns have undergone, and continue to undergo, significant changes in past decades. These developments may be broadly characterised as mirroring general trends in bioarchaeology: originating in a strong disciplinary emphasis on population-level quantitative data, leading to the development of a normative cross-sectional model; followed by a shift towards more individual-sensitive

measures of variability through a life history model and the nascent development of longitudinal methods; and finally to the present, where rapidly-expanding infant palaeodietary research is expected to be informed by multi-disciplinary bases of evidence, to the enrichment of interpretative robusticity. With the advent of ever-increasing time-precision in $\delta^{13}C$ and $\delta^{15}N$ data, the discipline has been prompted to examine its understanding of the interplay between intrinsic and extrinsic factors affecting stable isotope ratios in human remains. As should be expected, a lag exists between the development of innovative methods, and the penetration of new interpretive models and critiques of theory, and it is anticipated that both interpretive and methodological shifts will continue to develop rapidly. Carbon and nitrogen stable isotopes have achieved an unparalleled primacy in the examination of past breastfeeding patterns, which has yet to be challenged by other biogeochemical methods, but is complemented by several other analyses, including oxygen stable isotopes, which will be briefly discussed in the section to follow.

2.6.1.iii Oxygen

While oxygen stable isotopes have been most frequently employed within archaeology for studies of climate change or migration, they have also received some limited use within infant palaeonutrition research. Breastfeeding studies analysing oxygen stable isotopes share some similarities and differences with carbon and nitrogen isotope studies attempting to characterise population breastfeeding patterns. As is the case with carbon and nitrogen isotopes, the proportions of oxygen isotopes in food and drink are altered in the human body by metabolic processes, produce increasing enrichment of ^{18}O at each consumer level. However, unlike carbon and nitrogen isotopes in collagen, oxygen isotopes are analysed within the carbonate or phosphate moieties of bone or tooth apatite and do not represent dietary protein intake. Rather, $^{18}O/^{16}O$ ratios in humans are primarily reflective of and derived from their water intake, are measured relative to a standard of mean ocean water (VSMOW) and are closely aligned to geoclimatic and seasonal factors such as precipitation and temperature. The greater propensity of ^{16}O to evaporate, and ^{18}O to condense, leads to relative enrichment of ^{18}O in the precipitation and surface waters of warmer climates. This phenomenon decreases in higher latitudes, where surface waters are reflective of the ^{18}O-depleted rainfall which occurs.

Human $\delta^{18}O$ body water values, while broadly reflective of the values of local drinking water sources at the time of tissue synthesis, do not represent a simple equivalent to meteoric water values. As is the case with carbon and nitrogen stable isotopes, metabolic processes alter drinking water composition to produce higher $\delta^{18}O$ values in the body water pool from which tissues are synthesised. In the case of oxygen isotopes, relative enrichment of the heavier isotope within the body pool principally results from discrimination against ^{18}O and in favour of ^{16}O as exhaled water vapour (Bryant and Froelich, 1995; Kohn, 1996). As human milk is synthesised from the maternal body pool, providing the primary or sole water source for breastfed infants, the tissues of those infants reflect $\delta^{18}O$ body water values which are higher than not only their mothers', but also to a greater degree to the meteoric water values from which their mothers' body water is derived. This relative enrichment of ^{18}O was documented in an early study comparing the $\delta^{18}O$ urine from breast- and formula-fed infants, finding that while the urine of formula-fed infants was enriched in ^{18}O relative to the local tap water used in formula preparation, the urine of ^{18}O of breastfed infants was enriched further still, relative to the formula-fed infants (Roberts *et al.*, 1988). This study thus demonstrated the importance of recognising maternal metabolism as an intermediary source of additional fractionation between local drinking water values and breastfed infants, as well as the validity of using oxygen isotope analyses as an indicator of breastfeeding status. The magnitude of enrichment between mother and offspring has been estimated by some as representing an approximately 2-3‰ increase in $\delta^{18}O$ (Tsutaya and Yoneda, 2015), but archaeological studies have typically found a more modest 0.5-1.2‰ rise in values (Wright and Schwarcz, 1998; Wright and Schwarcz, 1999; Dupras and Tocheri, 2007; Britton *et al.*, 2015).

In addition to the metabolic fractionation of drinking water which studies have attempted to characterise through the calculations of tissue offsets in skeletal tissues, the isotopic composition of local drinking water may also be altered and become enriched in ^{18}O prior to consumption through anthropogenic activities which may produce fractionation, such as the evaporative impacts of food preparation. This has been termed the "stewing and brewing" effect and represents a significant challenge to infant feeding studies in pre-documentary cultures, where ^{18}O-enriched boiled foods or brewed beverages may contribute significantly to water intake for young children and produce shifts in $\delta^{18}O$ enamel values by +2.3‰ (Brettell *et al.*, 2012). It has been argued that

this intrinsic layer of uncertainty regarding the human cultural behaviours which may contribute to higher $\delta^{18}O$ in young children is insignificant in comparison to the magnitude of natural environmental variation of oxygen isotopes in humans, and that it is this variability which has led to limited applications of oxygen stable isotope analyses to the assessment of breastfeeding patterns in past populations (Tsutaya and Yoneda, 2015). While environmental variation produces differences in oxygen isotope ratios to a much greater extent than breastfeeding, Brettell *et al.* (2012) has demonstrated that stewing effects in cooked foods, such as the +10.2‰ shift produced by pottage cooked for several hours, should not be disregarded as a significant source of variability in human $\delta^{18}O$ values.

Regardless of the constraint to further study, the range of published oxygen isotope breastfeeding studies in archaeology has been, like the number of published trace element weaning studies, limited in scope. Following the earliest attempts by Wright and Schwarcz (1998; 1999) to interrogate breastfeeding patterns in past populations using oxygen isotope analyses, published studies using this method have been few, with most infant palaeonutrition studies relying on carbon and nitrogen stable isotope analyses. Studies utilising oxygen isotope analyses have all done so as part of a multi-isotope approach: alongside analyses of carbon from enamel carbonate (Wright, 2012), as an accompaniment to carbon from bone collagen (White *et al.*, 2004), or as a complementary analysis to pre-existing or concurrent carbon and nitrogen analysis (Dupras and Tocheri, 2007; Britton *et al.*, 2015). It is perhaps not surprising that oxygen is not used as a sole indicator of weaning status when the complexity of interpreting oxygen variability in human remains is considered. Diagenesis has also remained a concern. Acknowledgement of the greater vulnerability of bone, particularly in regard to carbonate, has produced a preference for enamel analyses, with only a single study (Britton *et al.*, 2015) opting to analyse bone using the more diagenetically-resistant phosphate oxygen. Wright (2012) used a sequential microdrilling method to analyse oxygen and carbon stable isotopes from enamel carbonate in the only high-resolution study weaning to date. The analyses sampled molars and premolars from the Classic period Guatemalan site and produced increasing $\delta^{13}C$ values consistent with maize-based weaning diet over time. However, $\delta^{18}O$ patterns did not follow an expected pattern of gradual decline over time as might be expected for the weaning process; instead, a pattern of undulating fluctuation within the longitudinal data suggested that

seasonal climatic variation exerted a more significant influence over $\delta^{18}O$ shifts than breastfeeding (Wright, 2012). While in many respects $\delta^{18}O$ analysis offer a promising means of assessing breastfeeding status through indications of water source, one which complements other biogeochemical palaeodietary methods, non-dietary fractionation factors greatly complicate the interpretation of breastfeeding patterns through this method.

2.6.2 Emerging analyses and future directions

While carbon and nitrogen stable isotope analyses have produced the largest body of data within infant palaeodietary studies, attempts have been made to supplement this data with other methods and isotopic analyses. In addition to the trace element and oxygen stable isotope analyses discussed in previous sections of this chapter, recent years have seen other types of analysis emerging. Sulphur isotope ratios ($\delta^{34}S$), which like strontium ratios arise from underlying geology and geographically-specific environmental inputs, do not vary systematically between mothers and breastfed infants but may provide a means of distinguishing between terrestrial, marine, or freshwater proteins consumed as weaning foods (Tsutaya and Yoneda, 2015). Analysis of $\delta^{34}S$ from bone collagen (Nehlich *et al.*, 2011) and incremental dentine and bone collagen (Howcroft *et al.*, 2012) have been used alongside $\delta^{13}C$ and $\delta^{15}N$ analysis as a multi-isotope approach, offering the promising ability to further nuance understanding of weaning foods. Calcium isotope ratio ($\delta^{44/42}Ca$) analysis in bone has been applied by Reynard *et al.* (2013) as another method of identifying patterns of milk consumption in archaeological populations. The study produced mixed results, and the researchers concluded that the relationship between dietary calcium and bone calcium isotope ratios were complex, suggesting the need for further work with modern controlled samples. In addition to experimental work using newer isotopic techniques, new approaches to existing techniques are also being explored for their potential to improve understanding of infant diet. Reynard and Tuross (2014) suggest that individual amino acid analysis, particularly $\delta^{15}N$, may clarify understanding the trophic levels of past infants, which would build upon emerging work based on modern populations (e.g., Romek *et al.*, 2013; Tea *et al.*, 2013). Additionally, recent work by Gröcke *et al.* (2017) demonstrating a trophic level effect for hydrogen isotopes suggests that these, too, may offer a profitable avenue of future investigation for breastfeeding research.

The current state of the art in infant palaeodietary studies promises an exciting and productive future for the discipline. The study of early childhood diet and its impacts of health and society in the past have developed away from their very early beginnings as an outgrowth – and in some respects, disciplinary afterthought – of wider palaeodietary study. Models and methods have shifted away from an aggregated, synchronic view of dietary practice to a much more nuanced, multifactorial biocultural model of parent-child interaction which is examining and describing variability as an essential and meaningful feature of practice. In the context of these developments, the sporadic inclusion of interdisciplinary perspectives from anthropology, history, immunology, biochemistry, ecology, and other relevant disciplines is greatly encouraging for the future, and it can only be hoped that conversance with these evidence bases will become the expected standard for palaeodietary researchers studying children. Better understanding of interactions between human diet and physiology are also necessary for future development, particularly in the case of carbon and nitrogen stable isotope analyses, where the precise mechanisms of fractionation between mothers, their milk, and their infants are still poorly characterised and understood. However, the rapid growth of breastfeeding research in archaeology, as well as the growth of palaeodietary study and archaeological science generally, suggests that clarification and improved understanding of isotope and breastfeeding biology through higher-quality and controlled study will remain high on the research agenda for future.

3. Biological Impacts of Breastfeeding

3.1 Introduction

Many palaeodietary studies have asserted that the protective benefits of breastfeeding diminish beyond the period of exclusive breastfeeding, and that "passive immunity" is lost beyond this stage (e.g. Katzenberg *et al.*, 1996; Dittmann and Grupe, 2000; Humphrey *et al.*, 2008; Sandberg *et al.*, 2014). Further, linkage of longer periods of breastfeeding in non-industrialised societies with malnutrition and disease in past anthropometric research has served to crystallise an inferred causal relationship between "prolonged" breastfeeding and increased risk of child stunting and malnutrition (Victora *et al.*, 1984; Brakohiapa *et al.*, 1988; Briend and Bari, 1989). While the most current body of clinical research does not support either of these assumptions, they continue to be implicit in some archaeological interpretations of past infant feeding and disease patterns. The disparity between current clinical evidence and the prevailing discourse implies a deficit in understanding of the highly-evolved mechanisms involved in lactation physiology, which should form a basic prerequisite for study and interpretation of past breastfeeding patterns.

While a working understanding of the basic biomechanics and health impacts of breastfeeding *must* be achieved by researchers wishing to interpret past childhood dietary patterns with any validity, a full specialist and in-depth understanding of a topic as biologically complex as the interplay between nutrition and human immune function is not likely to be a feasible goal for most bioarchaeologists. Regardless, understanding of how breastfeeding works as a physiological system is necessary to understand the behaviours analysed in the present study, and inform conclusions. This chapter accordingly aims to provide a simplified and targeted review of salient and up-to-date clinical and epidemiological evidence for the biomechanics of breastfeeding and the relationship between human milk, host defence, and infant nutrition.

3.2 Breastfeeding physiology

The most basic level of understanding, and one rarely achieved among non-specialists, regarding breastfeeding as a system is knowledge of how milk is actually produced. The process of initiation and continuance of milk production is known as *lactogenesis*. This process is observable as occurring in distinct phases.

3.2.1 Lactogenesis I

The initiation of milk production (lactogenesis) in mothers begins well before birth, during the second trimester of pregnancy. The first stage of lactogenesis, termed *Lactogenesis I* (LI), begins at around 24 weeks gestation, as glandular tissue achieves differentiation of epithelial cells into functioning milk-producing cells, called lactocytes (Hassiotou and Geddes, 2013). LI in late pregnancy is driven by increasing levels of a triad of hormones - prolactin, oestrogen, and progesterone (Hassiotou and Geddes, 2013). Following achievement of LI, production of prolactin in the pituitary is high. Prolactin is a protein hormone essential to the establishment of milk production in the early period of lactation (Lawrence and Lawrence, 2011: 66). During late pregnancy, approximately 30 millilitres of viscous *colostrum* (early milk) may be produced per day (and reabsorbed into the bloodstream following non-removal) (Kent, 2007).

3.2.2 Lactogenesis II

The onset of the second phase of milk production, called *Lactogenesis II* (LII) typically occurs 48-72 hours after birth, as progesterone levels decline (Hassiotou and Geddes, 2013). LII is marked by the emergence of copious milk production, colloquially described as milk "coming in" (Lawrence and Lawrence, 2011: 68). The composition of milk during LII shifts significantly (discussed below in section 3.3.3) between the phases of colostrum, transitional milk, and mature milk, with considerable variation in the timing of transitions. Immunologically-rich thick yellow colostrum produced in late pregnancy and the early postpartum period is replaced by a more fluid transitional milk, which is typically present from the fifth day to the second week postpartum (World Health Organization, 2009; Ballard and Morrow, 2013). Transitional milk is an intermediate substance which is produced as mammary secretion shifts between the production of colostrum and mature milk (Neville and Morton, 2001; Lawrence and Lawrence, 2011: 68). Milk is considered fully mature in composition by 3-6 weeks postpartum (Ballard and Morrow, 2013; Hassiotou and Geddes, 2013).

The provision of secreted milk to offspring is complex, requiring an effective milk ejection reflex in the mother (commonly known as *letdown*), a fully-functioning sucking reflex in the newborn, and correct positioning and attachment of the infant to the breast (Quandt, 1995; Watt and Mead, 2013). Breastfeeding is fundamentally a hormonally-mediated activity produced by the mechanical stimulus of suckling. Two primary hormones produced by the pituitary drive this process: prolactin and oxytocin (Neville

et al., 2002; World Health Organization, 2009). Release of oxytocin, which is also implicated in bonding between mother and child, is not only stimulated by sensory feedback from suckling, but may also be initiated by pre-feeding psychological stimuli, such as thought, sight, or sound of offspring (Jelliffe and Jelliffe, 1978: 22; World Health Organization, 2009). Psychological factors, such as stress, may conversely block the letdown reflex through the inhibiting action of adrenaline (Jelliffe and Jelliffe, 1978: 22; Quandt, 1995).

Sustained breastfeeding is entirely dependent on the establishment of an adequate maternal milk supply, which itself relies on maintenance of *sufficiently elevated* prolactin levels during the first few weeks following birth. While secretion of prolactin is immediately stimulated by suckling at the breast, prolactin levels return to baseline levels within two hours of a feed (Quandt, 1995). Hence, during LII, frequent suckling is a prerequisite to maintaining milk production. Sucking reflexes may be impaired in premature, low birth-weight, or otherwise unwell newborn infants (Jelliffe and Jelliffe, 1978: 14); it is likely that, in the past as in the present, these individuals would have required special care and feeding and measures to promote milk supply. However, while the "supply and demand" model of short-term lactation is well accepted, the precise neuroendocrine mechanisms through which this early relationship promotes longer-term lactation sufficiency in humans remain somewhat theoretical. Studies utilising non-ruminant animal models, which are believed to more closely resemble human lactation processes than ruminant models, have established that successful lactation during the LII phase is both biologically and behaviourally-mediated (Sakai *et al.*, 1985; Theil *et al.*, 2006).

3.2.3 Lactogenesis III

The third phase of lactogenesis (LIII) is less commonly discussed than LI and LII, as it represents the maintenance of established breastfeeding, rather than a period of change. However, in representing the bulk of lactation, it is significant for not only infant but also maternal health, as well as fertility patterns. LIII begins at approximately ten days postpartum and represents a transition from endocrine-mediated lactation to autocrine (local) control (Neville and Morton, 2001; Jones and Spencer, 2007). While prolactin secretion, stimulated through sucking stimuli, is essential for further mammary development during the endocrine-controlled phase of LII, it is not sufficient to sustain milk supply in the postpartum mother-infant dyad. Effective drainage of milk by, and

transfer to, the offspring are also required, as autocrine feedback mechanisms also operate to communicate supply and demand to the maternal body (Peaker and Wilde, 1996; Jones and Spencer, 2007).

Variations in prolactin are not only driven by feeding pattern but are also associated with an underlying circadian rhythm. Prolactin concentrations in both plasma and milk increasingly rise throughout the day, with the highest levels observed overnight (Hill *et al.*, 1999). Some researchers have suggested the circadian rhythm of prolactin concentrations are an artefact of infant feeding patterns (Cregan *et al.*, 2002), while others have demonstrated that the observed rhythms occur independently of feeding patterns and persist throughout lactation (Stern and Reichlin, 1990). Regardless of the direction of causality, the association between nocturnal elevation of prolactin and night-time breastfeeding has been shown to be an important predictor of prolonged postpartum infertility in well-nourished populations (Elias *et al.*, 1986; Ellison, 1995).

In general, high-demand exclusive breastfeeding during the first months of life suppresses the ovulatory and menstrual cycle, leading to temporary infertility in the mother – termed *lactational amenorrhea* (LA). The duration of LA may vary considerably among women, with some resuming reproductive cycling within months and others experiencing suppression until full weaning from the breast, depending on feeding frequency and maternal hormonal response (Diaz *et al.*, 1989; Ellison, 1995). However, in most mothers, LA may persist until solid foods are introduced, reducing feeding frequency and hormonal inhibition below an individual threshold for resumption of ovulation and menstruation (World Health Organization, 1999; Simondon *et al.*, 2003). This behaviourally-mediated infertility has heavy implications for the relationship between breastfeeding patterns and birth spacing in past populations, which has fuelled much of the interest in breastfeeding patterns among palaeodemographers.

3.3 Breastfeeding and host defence

Some researchers have explicitly suggested that due to the weight of evidence for the immunomodulating and metabolic impacts of human milk, it is important for public health or patient education programs to counter the prevailing view of breastmilk as "merely nutrition" (Ballard and Morrow, 2013: 54). It is equally vital that researchers working in the field of infant palaeonutrition share this understanding of the significance of consuming human milk in the early years of life for individual and

population health across the life course. As such an understanding of breastfeeding as a physiological modulator in early life is key to understanding its importance in the lives of the children analysed in the present study, an evidence-based review of human immune development, and the role played by breastfeeding, will be undertaken in the sections following.

3.3.1 Immune ontogeny in early life

Host defence in humans comprises the use of barrier, innate, and acquired mechanisms. Anatomical and physiological barrier mechanisms such as intact skin, mucociliary clearance, low stomach pH, and *lysozyme* (an antimicrobial enzyme) present in tears and saliva, provide the first line of defence against infection (Guyton, 1971: 71; Turvey and Broide, 2010). The importance of barrier mechanisms is attested by high vulnerability to infection where these structures are compromised. Due to the high efficacy of external barriers, most infections occur at the internal threshold of entry, within the mucosal surfaces of the respiratory, gastrointestinal, urinary, and reproductive tracts; it is here that the secondary defences of innate and acquired immune response become activated (Husband and Gleeson, 1996; Dwivedy and Aich, 2011).

3.3.1.i Innate immunity

Innate immune responses are based on discrimination between self and non-self (infectious) biomolecules, with further differentiation between normal self and abnormal products of self (e.g. infected host cells or cancer cells). Recognition of markers of microbial non-self, or more commonly termed *pathogen-associated molecular patterns* (PAMP), triggers activation of *phagocytosing* (cell-devouring) or *cytolysing* (cell-dissolving) white blood cells (WBCs or leukocytes) such as monocytes, macrophages, neutrophils, dendritic cells, and natural killer cells (Goenka and Kollmann, 2015). This type of defence represents the component of the innate response known as *cellular immunity*.

Other members of the innate immune system are not cells, but rather proteins found in extracellular fluids, and thus are termed components of *humoral immunity*. Elements of humoral immunity include various peptides and proteins offering bioactive protection, through disruption and damage of pathogen cell membranes or chemoattractant activity. Antimicrobial peptides known as defensins, which act as membrane disruptors in the presence of pathogens, play a critical supporting role within the internal and external barriers (such as skin and mucosa) where they reside (Ganz,

2003). The *complement system* may also become initiated by recognition of PAMP. Complement is comprised of blood plasma proteins, which recruit inflammatory cells and bind to pathogen surfaces, damaging pathogenic cell membranes or marking them for phagocytosis (*opsonisation*). Chemokines, a small type of *cytokine* (cell-signalling protein) are found throughout the body and may opsonise foreign molecules, increase inflammation, or exert powerful antimicrobial activity (Dürr and Peschel, 2002).

This approach to host defence is not without cost, as while pro-inflammatory (Th1) cytokines may upregulate the immune response, leading to increased blood flow and the recruitment of phagocytes, inflammatory activity carries both high metabolic costs and risk of damage to healthy cells through strategies such as systemic pyrexia (fever) (Goldszmid and Trinchieri, 2012). Further, undernutrition during the critical window of childhood immune development is theorised to bias long-term immunity towards an innate response, increasing risks of inflammatory disease in adulthood, which is exacerbated by nutritional abundance (Bourke *et al.*, 2016; McDade *et al.*, 2016). The cost-benefit comparison of innate and adaptive responses should be viewed in terms of the ecological niche in which they are developed. For instance, while adaptive responses carry a very high *developmental* cost, their maintenance and activation costs and risks of collateral damage are low. Conversely, innate responses exact low developmental costs alongside moderate maintenance costs and risk of collateral damage and high *activation* costs (McDade *et al.*, 2016). Thus, an early bias towards innate response to acute infection during the critical window of childhood – with a gradual shift towards cooperation with the adaptive response – provides short term benefits in survivorship which must be balanced with the developmental costs of longer-term immunoregulatory strategies which avoid chronic inflammation and impairment of growth and later fecundity.

What the innate immune system lacks in terms of specificity of attack, and presents in costs, it more than compensates for in terms of speed, as response is not constrained by a demand for specific recognition of new pathogens. This advantage is considerable, with innate immune responses being initiated within minutes of exposure, in contrast to the time-lag of days after first exposure required by the adaptive immune response (Parham, 2009: 9; Warrington *et al.*, 2011). The early evolutionary emergence of innate immunity in multicellular life has caused some to view the innate response as primitive. However, researchers now dispute this view of innate immunity as crude, arguing that

advances in the field of immunology in recent decades have shown innate immunity to play a complex and key role in not only overall host defence, but also the development of adaptive immunity (Beutler, 2004; Akira *et al.*, 2006)

3.3.1.ii Adaptive immunity

While immune response in early life is heavily biased towards innate mechanisms in place at birth, immune systems are not tidily discrete, as the innate immune system works in partnership with adaptive responses. Adaptive (or acquired) immunity provides a form of defence triggered by recognition of molecules specific to the foreign agent (or *antigen*) encountered (Fearon, 1999; Pancer and Cooper, 2006). A primary advantage to this specificity over the simple self/non-self discrimination of the innate response is the ability to retain long-term immunological memory of previous antigenic encounters. This immunological memory ensures progressive advancement of host protection over time, and enables the success of medical advances, such as immunization (Janeway, 2001). The learning curve of adaptive immunity is steep, with the onset of postnatal immune-education occurring rapidly as the neonate encounters dietary and pathogenic antigens within the mucosa. Adaptive immunity, like innate immunity, contributes both cellular and humoral elements, with each working cooperatively with existing innate responses.

Adaptive immunity is mediated by two types of lymphocytes, *T cells* and *B cells*, which perform functions of cellular immunity. Triggered by antigenic recognition, memory T cells perform either helper or *cytotoxic* (cell-killer) functions. *Helper T cells* are a crucial axis of adaptive immune response, as they coordinate the adaptive immune response by stimulating B cell maturation and differentiation, as well as recruiting macrophages through cytokine secretion (Alberts *et al.*, 2002). They may also trigger activation of *cytotoxic T cells*, which provide defence against intracellular pathogens and malignant cells, and act by releasing cytotoxic granules. These granules, called *perforin* and *granzymes*, cause lysis and perforation of the cell membrane and cell death, respectively. Cytotoxic T cells, like helper T cells, may also secrete cytokines with anti-infective activity.

Sensitisation to antigens through interaction between T cell receptors and an *antigen-presenting cell* (or APC; these are primarily dendritic cells) allows naïve T cells of both types to become activated and differentiated into one of two types: *effector T cells*, short-lived but fully functional migratory cells acting as primary responders to

infection, or *memory T cells*, longer-lived veterans of previous infection which proliferate rapidly upon repeat exposure to antigens (Sallusto *et al.*, 1999). Neonates possess a higher overall abundance of T cells than adults, with increases throughout infancy and a decrease to adult numbers by early childhood (Goenka and Kollmann, 2015). However, these T cells are not only of the naïve type but are also phenotypically-biased towards Th2 (anti-inflammatory) cytokine production, leading to greater susceptibility to intracellular pathogens, such as viruses, during infancy (Dowling and Levy, 2014). Several researchers have suggested that breastfeeding promotes T cell development, noting that breastfed infants have thymuses twice as large as those of their non-breastfed counterparts (Field, 2005; Hanson *et al.*, 2009).

Like T cells, B cells are formed from progenitor (stem) cells within bone marrow. Following maturation, they become activated and differentiated within secondary lymphatic organs, such as the spleen and lymph nodes, into non-proliferating (effector) *plasma cells* or dormant *memory B cells*, which, upon re-exposure to familiar antigens, function similarly to their T cell counterparts, and may further differentiate into plasma cells with improved antigenic affinity (Murphy *et al.*, 2017: 13). Both mature B and T cells are found within *gut-associated lymphoid tissue* (GALT) in company with cells of the innate immune system, especially within the intestinal epithelium. Plasma cells may act as an APC, activating T cells or other B cells. Nearly all neonatal B cells are of the naïve subtype, with low numbers of memory B cells (Goenka and Kollmann, 2015). In response to antigens binding to their membrane receptors, plasma cells also secrete *antibodies*, Y-shaped proteins with identical antigen specificity to those of their secreting plasma cell. Together, B cells and antibodies mediate the humoral component of adaptive immunity.

Five classes of antibody, also known as immunoglobulins (Ig), exist in humans: IgG (the most abundant antibody in human sera), IgM (the largest, most rapid responder to new infection, and first produced during foetal life), IgA (most common antibody in excreta, including human milk), IgD, and IgE (Wood, 2006: 51). The final antibody, IgE, is commonly associated with allergic response in cases of immune dysfunction but is also essential to resistance to infection (Guyton, 1971: 77). Antibody-antigen binding may initiate any of three host defence pathways: direct action towards invading substances, activation of the complement cascade, or opsonisation. Antibodies take direct action by preventing pathogens from binding to host cells through a range of

methods, which include inducing clumping of pathogens for efficient phagocytosis (*agglutination*), induced insolubility (*precipitation*), coating pathogens to block cellular attachment (*neutralisation*), and lysis of pathogenic cell membranes (Guyton, 1971: 76). Indirect action by antibodies, through opsonisation and activation of the complement cascade, lies at the interface between innate and adaptive immune systems, and thus demonstrates the collaborative and fluid nature of host defence.

Newborn infants possess functional, but naïve adaptive immune systems, alongside a significantly weaker initial innate response than is present during later life (Simon *et al.*, 2015). This weaker innate response in neonates represents an artefact of active foetal suppression of potentially-harmful pro-inflammatory responses, which may lead to growth restriction or pre-term delivery (Levy, 2007). Long-standing belief in the microbiological sterility of the womb has now been refuted, as researchers now acknowledge foetal immune responses and a uterine microbiome (Perez-Muñoz *et al.*, 2017). This bias against inflammatory response decreases over the course of infancy as innate immunity matures (Dowling and Levy, 2014). Immune "programming" is known to begin *in utero*; low-level maternal pathogen exposure has been demonstrated to make positive contributions to offspring immune response, while maternal malnutrition and exposure to toxins is similarly known to have deleterious effects on offspring immune development (MacGillivray and Kollmann, 2014; Goenka and Kollmann, 2015). It is also now known that there are sex differentials for *in utero* placental immune responses, with male offspring being at higher risk of growth restriction or pre-term birth than their female counterparts (Clifton, 2010).

However, the relative inexperience of the newborn immune response and absence of mucosal IgA makes all infants vulnerable to the wide variety of pathogens in the postnatal environment. They are heavily reliant upon the non-specific mechanisms of the immature innate immune response, and on finite reserves of placentally-derived IgG and low-affinity IgM (Field, 2005; Parham, 2009: 197). This vulnerability is exacerbated in infants born prematurely, who take longer to achieve immunocompetence than those born at full term (Parham, 2009: 197). Further, immune response is energetically expensive, subverting finite resources which would otherwise be used by the infant for maintenance and growth (McDade *et al.*, 2008). Human milk offers a tripartite solution, providing required energy, passive immunological protection through transfer of bioactive factors, and accelerated development and regulation of the

infant's own active immune response. Breastfeeding during the period of greatest vulnerability to infection thus allows for the creation of a shared immunity between mother and offspring, enhances growth potential, and reduces offspring morbidity and mortality.

3.3.2 Major bioactive constituents of human milk

The following section will give a broad summary of the major bioactive constituents of human milk. Some components of milk, such as antibodies and maternal cells, have a primarily immunological function. Still other constituents of milk play more complex and interactive roles; some traditionally thought to have simple nutritive or growth-related functions have recently been discovered to have immune bioactivity (Hanson *et al.*, 2009). Bioactive substances in human milk are numbered in the hundreds to thousands, and our understanding of how these biomolecules function continues to evolve (Ballard and Morrow, 2013). Consequently, as with the previous section on immune ontogeny and function, this will represent a greatly simplified and non-exhaustive overview of the major constituents and their implications for host defence and wellbeing.

3.3.2.i Antibodies

Antibodies were the first bioactive constituents of milk to be identified and retain the greatest popular recognition. These are produced by B cells, which migrate from the maternal gut to the mammary, providing specialised protection against pathogens in the shared maternal-offspring environment (Brandtzaeg, 2003). Human milk antibodies do not enter circulation in significant numbers, instead offering defence at the mucosal level (Van de Perre, 2003). While all the major antibody subtypes may be found in milk, the most important of these are the secretory form of IgA (sIgA), IgG, and IgM. Secretory IgA, which comprises 90% of all milk antibodies (Bernt and Walker, 2001), is of particular importance, as neonates are not thought to be capable of producing this antibody in early life (Battersby and Gibbons, 2013). Independent IgA production develops rapidly during infancy but is not believed to reach adult levels until as late as 4-6 years of age (Weemaes *et al.*, 2003). Milk sIgA is adapted to be particularly hardy, resisting proteolytic destruction within the digestive tract, and being found intact in infant stool (Lönnerdal, 2016). IgM is also abundant in early lactation, and provides a less antigenically-specific, though still significant, form of mucosal defence (Hassiotou *et al.*, 2013).

Conflicting results have been found regarding the role of breastmilk antibodies in the development of an active immune response. Some studies have found higher salivary (infant-derived) IgA and IgM levels in formula fed infants than those of breastfed infants, demonstrating an earlier and higher rate of antibody production in non-breastfed children (Gleeson *et al.*, 1986; Gleeson *et al.*, 1995). Others have found a reversed trend, indicating earlier mucosal immune development for breastfed infants, due to stimulating factors in milk (Ogawa *et al.*, 2004; Piirainen *et al.*, 2009). Paradoxically, IgA levels may be higher in both healthy, well-nourished children (as a sign of robust immune function) and immunologically-stressed children (in response to microbial exposure). As other researchers have noted that elevated levels of salivary IgA are associated with chronic undernutrition and impaired growth in developing countries (Cunningham-Rundles *et al.*, 2005; Miller and McConnell, 2012), maternal milk may provide cushioning against excessive strain during the early life, energetically-expensive immune system.

The mechanisms of protection provided by sIgA are predominantly biased towards neutralisation of pathogens, and thus passive protection within the mucosa (Parham, 2009: 194). Secretory IgA in human milk has a particularly high affinity for a broad array of pathogens, with a specificity that endures throughout lactation (Hanson *et al.*, 2009). Such passive protection from pathogenic attachment to the mucosal epithelium accounts for the reduced rates of urinary, upper respiratory, and gastrointestinal infections in breastfed children, without the notable disadvantages of inducing inflammatory activity, risk of tissue damage, or excess energy expenditure (Hanson *et al.*, 2001; Hanson and Korotkova, 2002). IgG, by contrast, binds to pathogens in the lumen of the intestinal, respiratory, or urogenital mucosa before transporting them across the epithelium into the bloodstream, leading to either upregulation (e.g. B cell proliferation) or downregulation (e.g. tolerance) of active immune function. IgG becomes an increasingly important component of human milk in later lactation. This is suggested to be a change in host defence strategy away from simple antimicrobial activity and towards the promotion of independent immune function in the growing infant (Gao *et al.*, 2012).

3.3.2.ii Maternal cells

These linked processes of protection and maturation are also aided by the provision of maternal cells in milk. As breastfed offspring demonstrate tolerance to maternal human

leukocyte antigen (HLA), this does not provoke rejection and allows maternal cells in milk to function in the child's system (Campbell *et al.*, 1984; Hanson *et al.*, 2009). Roughly 80% of these cells in colostrum are macrophages, which play a significant role in supporting innate immunity (Järvinen and Suomalainen, 2002; Ballard and Morrow, 2013). These high levels in colostrum decline to a low baseline of around 2% in mature milk (Hassiotou *et al.*, 2013). *Neutrophils*, a phagocytic form of WBC, are the second most abundant cell in human milk. Their function is unclear, as they demonstrate lower adherence, responsiveness, and motility in milk than do blood neutrophils. It is suggested that their function may relate to maternal, rather than offspring, defence (Goldman and Goldblum, 1990; Field, 2005). Cell counts in milk are highly variable, especially macrophage counts, which increase during active infection in the infant (Riskin *et al.*, 2012). The mechanisms by which the maternal body increases provision of bioactive agents in breastmilk are unclear but are hypothesised to occur in response to retrograde flow of infant saliva into the mammary gland, producing reciprocal communication between the maternal immune response and offspring microbiota (Hinde and Lewis, 2015). In addition to boosting the innate immune response of breastfed infants, macrophages in milk may also spontaneously differentiate into dendritic cells which stimulate T cell activity (Ichikawa *et al.*, 2003; Jakaitis and Denning, 2014).

Lymphocytes, such as T cells, also play an important role in human milk. Although they account for only 5-10% of leukocytes in breastmilk, most are of an activated and/or memory type (Wirt *et al.*, 1992; Field, 2005). Over 80% of lymphocytes in milk are T cells, with activated cells of maternal origin thought to simultaneously compensate for deficits in neonatal T cell function, whilst promoting their maturation (Field, 2005). Most breast milk lymphocytes - the vast majority being cytotoxic T cells - take up residence in the neonatal intestines (Cabinian *et al.*, 2016). B cells in milk originate in the maternal gut, where they have been sensitised by detection and presentation of antigens, before migrating to the maternal mammary gland to produce specialised immunological protection for the offspring (Newburg, 2005). Human milk has also recently been proven to contain multipotent (precursor to multiple cell types) mesenchymal stem cells, which promote not only immune development, but also potentially produce a maternal *extra-utero* influence on offspring tissue development (Cregan *et al.*, 2007; Patki *et al.*, 2010).

3.3.2.iii Microbes

Non-human cells of maternal origin in milk are also thought to contribute to long-term offspring health and immune function. Long considered microbiologically sterile, milk is now known to provide a range of commensal, mutualistic, and probiotic bacteria derived from a maternal entero-mammary pathway to the infant gut (Fernández *et al.*, 2013; McGuire and McGuire, 2015). This paradigm shift has resulted in the rapid growth of research in the field of infant gut microbiota, and an evolving body of evidence regarding the impacts of microbiome development on long-term systemic function. The maternal microbiome is foundational to the infant microbiome, with unique populations shared by each mother-infant pair. Infant gut flora are seeded through bioactive elements passed during birth, colonisation via skin contact, and also through breastfeeding; multifactorial determinants of colonisation, such as gestational age, delivery mode, maternal diet, lactation period, geography, disease ecology, and medication all influence the infant microbiome (Gomez-Gallego *et al.*, 2016). The microbiota of milk is theorised to be facilitated by the same entero-mammary link which enables transfer of gut-derived mucosal immune factors from mother to child. This transfer is estimated to be considerable, amounting to millions of cells daily (Jost *et al.*, 2014).

Maternal microbial populations are highly variable, but nine core types of gut microbe are consistently observed in milk: *Streptococcus*, *Staphylococcus*, *Serratia*, *Pseudomonas*, *Corynebacterium*, *Ralstonia*, *Propionibacterium*, *Sphyngomonas*, and *Bradyrhizobiaceae* (McGuire and McGuire, 2015). These consistently-present microbes account for about half of milk bacteria, with the other half consisting of abundant, but inconsistently-documented bacteria, such as *Lactobacillus*, *Bifidobacterium*, and *Enterococcus*. Despite the maternal microbiome's foundational role in the microbial colonisation of offspring, maternal microbiomes are more diverse than those of milk, which in turn are more diverse than the range of gut microbiota of breastfed infants (Jost *et al.*, 2014). The role of human milk in both nurturing and constraining the microbiome of breastfed infants is demonstrated by a lower species diversity of gut flora, relative to their formula-fed peers, while having two-fold higher cell counts (Andreas *et al.*, 2015).

Prebiotic (microbe-feeding) substances in milk, such as *oligosaccharides* are considered to exercise a weighty influence on the infant microbiome. Bacterial species

adapted to metabolise these prebiotic substances in milk can thrive and outcompete other species, shifting the balance of gut flora in their favour. Conversely, other bioactive substances, such as some of the fatty acids (e.g. conjugated linoleic acid, or CLA), may inhibit the proliferation of other microbes (Kelsey *et al.*, 2006). In a comprehensive review, McGuire and McGuire (2015) reported that most studies to date find a consistently characteristic milk-microbiome associated with breastfeeding, comprising species from the genera *Lactobacillus*, *Streptococcus*, and *Staphylococcus*.

The role of the microbiome as mediators of immune function has consequently begun to emerge as an area of interest. It has generally been theorised that a characteristic breastfeeding microbiome dominated by *Bifidobacterium* and *Lactobacillus* species provides infants with significant health benefits (Brück *et al.*, 2002). For instance, supplementation of human milk *Lactobacillus fermentum* and galactooligosaccharides in a sample of formula-fed infants, aged 6 to 12 months, has been shown to reduce incidence of gastrointestinal and upper respiratory infections by 46% and 27%, respectively, compared to controls (Maldonado *et al.*, 2012). Emerging evidence also suggests that early microbiome development exerts a powerful influence over immunological and metabolic function throughout the lifecourse. The presence of a balanced array of beneficial microbes within the gut plays an essential role in activities ranging from stimulating development of intestinal microstructure, to promoting conditions hostile to pathogens in the gut, and preventing the development of inflammatory disease (Round and Mazmanian, 2009; Yasmin *et al.*, 2015). Early education of immune response and induction of tolerance and anti-inflammatory activity through the establishment of a healthy microbiome is believed to set the stage for lifelong immune response. Consequences for the development of inflammatory diseases later in life potentially stem from early microbiome dysfunction (*dysbiosis*) (Groer *et al.*, 2014). These roles are fulfilled through instruction of regulatory T cells, stimulation of cytokine production, and subsequent upregulation or downregulation of inflammatory activity and antibody production. Bacteria may also themselves synthesise peptides, proteins, and nucleotides with immunomodulatory properties (Marques *et al.*, 2010). Donnet-Hughes *et al.* (2010) has also demonstrated the ability of bacterially-loaded dendritic cells to migrate from the maternal intestines to the mammary gland, potentially working to educate the neonatal immune system and induce tolerance of commensal bacteria through milk-borne messaging.

Metabolic programming also appears to derive to some extent from the microbial populations colonising the gut. For instance, increased *Bifidobacterium* and decreased *Staphylococcus aureus* in gut flora during the first year of life are reported to correlate with lower risk of obesity in later life (Kalliomäki *et al.*, 2008). Some strains of *Bifidobacteria* and *Lactobacilli* in human milk produce CLA, which inhibits *S. aureus*; this may be one mechanism by which breastfeeding promotes healthy metabolic function (Kelsey *et al.*, 2006; Marques *et al.*, 2010). Microbiome research is still in its infancy, and the precise mechanisms and long-term impacts of microbial bioregulation still to be fully elucidated. It is clear, however, that the consumption of human milk has weighty consequences for human health in the short, medium, and long-term via its role in maintaining microbial symbiosis.

3.3.2.iv Bioactive macronutrients

A simplistic assessment of human milk might envisage a neat dichotomous partitioning between the nutrient contributions of milk, and immune factors. However, such a view would be inaccurate; not only is nutritional status now known to play a decisive role in adequacy of immune function, but many nutritive elements of milk also have direct bioactivity against pathogens.

Despite the fact that, at approximately 4% by volume, human milk is relatively low in fat by mammal standards, lipids contribute a majority of the caloric content of mature human milk, accounting for 40-55% of total energy (Hinde and Milligan, 2011; Andreas *et al.*, 2015). In addition to contributing substantially to total energy content and providing fat-soluble vitamins, some lipids, such as fatty acids and monoglycerides, have antimicrobial properties (Newburg, 2005). Like all human milk lipids, levels of fatty acids in milk are highly variable, and strongly tied to maternal diet. Variation in longer-term outcomes observed for infants receiving variable diets has led to greater interest in the functional influences of ω-3 and ω-6 fatty acids on early development. In addition to the antimicrobial activity of ω-6 CLA (mentioned in the last section), ω-3 docosahexaenoic acid (DHA) and ω-6 arachidonic acid (ARA) have received attention for their dual roles in immune and neurodevelopmental maturation (Field, 2005; Brenna *et al.*, 2007). ARA is noted to stimulate generation of both type 1 (Th1) and type 2 (Th2) helper T cells, as well as being implicated in maintenance of cell membrane integrity in major organs (Hadley *et al.*, 2016). DHA also affects T cell function and delays programmed cell death in monocytes, while being integral to development and

maintenance of membranes in the central nervous system (Gottrand, 2008; Mebarek *et al.*, 2009; Lauritzen *et al.*, 2016). Despite an evolving body of knowledge regarding the exact role of fatty acids in the development of immune function and dysfunction, it is clear at least that lipids in human milk shape immune response.

Whilst lipids account for a majority of the energy content of milk, carbohydrates in the form of lactose dominate non-water (milk is approximately 90% water by volume) milk content, at 7.2% (Hinde and Milligan, 2011). As lactose provides approximately 85% of carbohydrate content, and remains stable over time, estimates of carbohydrates in milk composition are commonly reported as lactose (Coppa *et al.*, 1999; Le Huërou-Luron *et al.*, 2010). However, the third most abundant element of solid matter in human milk (after lipid and lactose fractions) is also comprised of carbohydrates, called *human milk oligosaccharides* (HMOs). These are synthesised in the mammary gland and are present in milk in large but variable amounts, representing about 1% of milk by volume and about 10% of its caloric content (Bode, 2012).

Oligosaccharides have long been recognised to play a significant part in human health, but an understanding of the scale and mechanisms of their impact continues to evolve. Indigestible and resistant to hydrolysis in the upper gastrointestinal tract, HMOs were originally thought to function solely as prebiotic substrates for commensal bacteria. HMOs particularly promote the growth and proliferation of *Bifidobacterium* and *Lactobacillus* (Bode, 2012; Groer *et al.*, 2014). The advantage given to probiotic bacteria capable of readily metabolising the variable range of HMOs produced by each mother creates a distinctive generationally-transmitted microbiome which benefits the offspring in most cases. For instance, *Bifidobacterium infantis*, which is a highly efficient metaboliser of HMOs, is also protective against diarrhoea (Petherick, 2010). However, HMOs are now known to have multifunctional roles within the body, beyond simply feeding the microbiota. Within the gut, in addition to acting as prebiotics, HMOs are hypothesised to act as decoy binding sites for pathogens, preventing attachment to epithelial receptors. A range of studies finding evidence of decreased incidence of diarrhoeal and respiratory disease in breastfed infants and HMO-supplemented formula fed infants and mice have given this hypothesis increased biological plausibility (e.g. Morrow *et al.*, 2004; Stepans *et al.*, 2006; Arslanoglu *et al.*, 2007; Manthey *et al.*, 2014).

Researchers now postulate that the range of activities undertaken by HMOs includes not only enteric, but also extra-intestinal and systemic effects. Around 1% of HMOs are absorbed by the intestines, enter circulation, and are excreted renally (Newburg *et al.*, 2005). Detection of HMOs in urine has consequently been suggested to contribute to the lower incidence of urinary tract infections in breastfed infants (Cunningham, 1995; Arslanoglu *et al.*, 2008). HMOs may also help prevent inflammatory disease, and resulting tissue damage, through their ability to bind with not only pathogens, but also excessive leukocytes (Bode *et al.*, 2004). HMOs have also been recently shown to downregulate inflammatory activity (Wickramasinghe *et al.*, 2015; Kulinich and Liu, 2016). Recent observation of HMO activity in the blood plasma of breastfed infants supports the hypothesis that they may act as systemic immunomodulators, as *in vitro* studies have demonstrated significant activity of HMOs at low concentrations (Goehring *et al.*, 2014).

Just as human milk is relatively low in fat, it is also very low in protein, at 1.3% of total volume, representing the least abundant of the macronutrient fractions (Hinde and Milligan, 2011). Even at this low concentration, the diversity of proteins in human milk is large, comprising over 1500 different proteins (Zhang *et al.*, 2014). In contrast to other commonly-consumed animal milks, human milk is overwhelmingly biased in favour of easily-digested whey proteins (~60-80%), with a lesser fraction made up of casein (~20-40%), and a still-smaller fraction composed of milk fat globule membrane proteins called *mucins* (Le Huërou-Luron *et al.*, 2010; Wada and Lönnerdal, 2014). Proteins in milk provide essential amino acids and enzymes, which contribute to a positive nutritional state in the growing infant. However, many of these proteins and peptides also exert bioactive properties, providing innate defence as defensins, and in some cases also promoting maturation of adaptive immunity. Some of these are bioactive in their intact state and exert their functions due to their resistance to proteolysis, while others, such as α-lactalbumin, exhibit bioactivity only once protein digestion in the gastrointestinal tract releases bioactive peptides. These have been shown to have prebiotic and bactericidal activity, and to enhance phagocytosis (Lönnerdal, 2010; Wada and Lönnerdal, 2014). One such bioactive substance is HAMLET (human α-lactalbumin made lethal to tumour cells, a complex formed from partial unfolding of α-lactalbumin, which is believed to protect infants against infection and cancer (Newburg *et al.*, 2005; Chirico *et al.*, 2008; Hakansson *et al.*, 2011). The

anti-carcinogenesis activity of HAMLET may help to explaining the much lower documented prevalence of childhood cancers, such as leukaemia, among breastfed children, as well as the lower rates of breast cancer among breastfeeding mothers (Kwan *et al.*, 2004; Ip *et al.*, 2007).

The second most abundant bioactive whey protein of mature milk is Lactoferrin, representing about a third of total protein (Hanson *et al.*, 2003). Lactoferrin is an iron-binding glycoprotein, produced by neutrophils in the blood and in inflamed tissues. It sequesters free iron, making it unavailable for use by pathogens (Siqueiros-Cendón *et al.*, 2014). Another human milk protein, haptocorrin, provides similar antimicrobial protection through its parallel withholding of vitamin B_{12} during infections (Lönnerdal, 2010). In addition to this *bacteriostatic* (preventative of bacterial reproduction) activity shared with haptocorrin, lactoferrin is also actively antimicrobial across the entire spectrum of pathogenic classes. It is antioxidant, enzymatic, and a potent immunomodulator through its activation of natural killer cells, enhancement of phagocytosis by macrophages, and suppression of pro-inflammatory cytokine production (Hanson, 1999; Kanyshkova *et al.*, 2003; Wada and Lönnerdal, 2014; Lönnerdal, 2016). Lactoferrin is also involved in the education of the adaptive immune response (Siqueiros-Cendón *et al.*, 2014). Resistant to proteolysis, lactoferrin not only acts in the digestive tract but enters the circulation and can be detected in newborn blood samples within hours of feeding (Kanyshkova *et al.*, 2003). This circulatory penetration is protective against septic shock and infections of the respiratory and urinary tracts in breastfed infants (Siqueiros-Cendón *et al.*, 2014; Lönnerdal, 2016).

In addition to bioactive peptides and whole proteins, the whey fraction of human milk also contributes several enzymes with bioactivity contributing to host defence. Lactoperoxidase is a proteolysis-resistant antimicrobial produced in the salivary and mammary glands. Demonstrated to be an effective antibacterial, it provides protection in the oral and upper gastrointestinal environments. Human airway secretions also contain lactoperoxidase, and have demonstrated protection against upper respiratory infection, suggesting that breastfeeding may contribute to a reduction in morbidity (Wijkstrom-Frei *et al.*, 2003). Lysozyme, an enzyme found in milk and tears is also an effective antimicrobial. Lysozyme works alongside lactoferrin, and shares several of its characteristics. Similarly resistant to proteolysis during digestion, lysozyme is believed to protect against infection through disruption and lysis of bacterial cell walls, reduce

inflammation, and has been shown in clinical trials to provide effective protection against diarrhoea, when paired with lactoferrin (Goldman and Goldblum, 1990; Goldman, 2007; Lönnerdal, 2010; Lönnerdal, 2016). Lysozyme content rises over the course of lactation, from 2% of total protein in colostrum, to 8% in mature milk (Montagne *et al.*, 2001).

Three main forms of protein are present in the casein fraction of human milk: alpha (α-casein), beta (β-casein), and kappa (κ-casein) caseins. Of these, β-casein predominates in abundance (50% of casein content) (Migliore-Samour *et al.*, 1989). Following digestion, β-casein releases bioactive peptides which act as chemoattractants for monocytes and macrophages, enhancing the innate defences (Kitazawa *et al.*, 2007). Other peptides have been shown to improve mucosal defence in the small intestines (Jakaitis and Denning, 2014). κ-casein acts as a prebiotic and protects against bacterial infection by acting as a receptor analogue (Lönnerdal, 2010; Wada and Lönnerdal, 2014). α-casein is the least abundant of the caseins in human milk (representing only 10% of caseins) and has been primarily studied in bovine milk (Migliore-Samour *et al.*, 1989). Peptides from digested bovine α-casein (and β-casein), called casokinins, have been shown to act in vitro studies as an ACE inhibitor, protecting against hypertension and promoting cardiovascular health; it is believed that these peptides in human milk provide a similar benefit. These casokinins of bovine origin have also been shown to have immunomodulatory effects, as they regulate production of lymphocytes and cytokines and enhance phagocytotic response. Human alpha-casokinins may have similar effects (Gill *et al.*, 2000; Park and Nam, 2015).

In addition to the whey and casein fractions, bioactive proteins are also found in the membranes of milk fat globules. Human milk fat globule membrane (MFGM) proteins, such as mucins, provide host defence through their ability to bind enteric pathogens. MUC1, for example, inhibits the binding of pathogenic *E. coli* to epithelial surfaces (Goldman, 2007). Another bioactive MFGM protein in human milk is lactadherin. Like lactoferrin, lactadherin mediates recruitment of phagocytes, decreases inflammatory activity, and exerts antiviral activity, particularly against rotavirus (Goldman, 2007; Ballard and Morrow, 2013). Supplementation of infants with an MFGM fraction of bovine origin has resulted in reduced rates of infection and improved health (Zavaleta *et al.*, 2011; Timby *et al.*, 2015).

A final protein, which is greatly understudied, but which may also have significant effects on infant host defence, is osteopontin. Osteopontin is present in mature human milk at relatively high levels (~140 mg/L), and is secreted by macrophages, T cells, and epithelial cells (Nagatomo *et al.*, 2004; Lönnerdal, 2016). Among its known bioactivities are cellular immune responses, regulation of inflammatory cells, and anti-inflammatory activity (Ashkar *et al.*, 2000; Nagatomo *et al.*, 2004). A recent clinical trial that supplemented infant formula with a bovine osteopontin fraction found that, relative to a control group fed on a standard infant formula, supplemented infants had much lower serum concentrations of the proinflammatory cytokine transforming growth factor α (TGF-α). They also had cytokine and overall immunological profiles more like those of typical breastfed infants and had fewer days of illness than the non-supplemented group (Lönnerdal, 2016). This lack of dose-dependency suggests that the effects of osteopontin for immune regulation in the infant may be potent enough to impact health even at low levels in milk.

3.3.2.v Cell signalling/immunoregulatory elements

Many of the antibodies, maternal cells, microbes, and bioactive nutrients covered in the previous sections are actively involved in upregulation or downregulation of the infant immune response. These processes are primarily accomplished with the secretion of immunomodulatory signalling molecules. Cytokines, growth factors, and hormones all belong to this class of communicatory immunomodulators.

Cytokines are central to the regulation of human immunity. They orchestrate immune response by initiating pro-inflammatory or anti-inflammatory activity, inducing movement of other types of immune cells, and acting as a mediator between the innate and adaptive immune responses. While cytokines in milk are primarily produced in the mammary gland, leukocytes in human milk have also been shown to produce them (Hawkes *et al.*, 2002). Cytokines present in human milk include chemokines, such as granulocyte colony stimulating factor (G-CSF) and macrophage migration inhibitor factor (MIF), interleukins (IL), interferons (IFN), tumour necrosis factors (TNF), and granulocyte macrophage colony stimulating factors (GM-CSF), among others. These are thought to resist degradation in the digestive tract and are known to penetrate to the sub-epithelial layer where they may interact with immune cells (Garofalo, 2010; Ballard and Morrow, 2013). In addition to acting as signalling molecules enhancing short-term immune response, maternal cytokines in milk also play a key role in promoting

intestinal development and the maturation and regulation of appropriate and balanced innate and adaptive immune responses (Garofalo, 2010; Oddy and Rosales, 2010).

Like cytokines, growth factors are proteins, which promote maturation of the developing infant. While both cytokines and growth factors direct development, the former may act to inhibit or encourage proliferation; growth factors consistently promote positive growth, development and differentiation. A range of growth factors promote healthy tissue development in the breastfed child (Ballard and Morrow, 2013). For example, transforming growth factor beta (TGF-β) along with IL-10, are thought to be the primary cytokines responsible for maintaining homeostasis in the adult gut. Their abundant presence is thought to be key to the development of infant immunity through their bias toward anti-inflammatory response (Penttila, 2010; Ballard and Morrow, 2013). In addition to promoting maturation of gut mucosa, high levels of TGF-β have been correlated with reduced levels of allergy. This suggests a role in the development of oral tolerance to antigens (Oddy and Rosales, 2010).

A wide array of hormones has also been detected in human milk, including cortisol, oestrogen, progesterone, androgens, erythropoietin, insulin, leptin, ghrelin, adiponectin, and prolactin, among others (Field, 2005). Some of these are derived from the maternal bloodstream, while others are synthesised directly in the mammary gland, suggesting adaptive advantages to offspring, rather than incidental inclusion in milk. Many hormones are involved in metabolic regulation of the breastfed infant. For instance, insulin is present in significant quantities in human milk, and is believed to facilitate intestinal maturation and development of insulin tolerance, resulting in a reduced incidence of Type 1 diabetes in breastfed children (Shehadeh *et al.*, 2001; Shehadeh *et al.*, 2003). Leptin, ghrelin, and adiponectin belong to a group of hormones in human milk called adipokines which are implicated in long-term regulation of appetite and adiposity and are therefore believed to play important roles as early determinants of the lifetime risk of obesity (Savino *et al.*, 2013). The long-term effects of hormones more recently added to this group of metabolic modulators discovered in human milk, including resistin and obestatin, are still relatively poorly understood (Savino *et al.*, 2009). These form a high research priority for future studies seeking to understand population obesity, metabolic dysfunction, and their relationship to inflammatory disease.

Other milk hormones serve multiple purposes, contributing to metabolic, reproductive, or immunological development, concurrently. Cortisol is a steroid hormone which, in addition to having effects on metabolic regulation, also acts as an immunomodulator through its anti-inflammatory activity and ability to increase leukocyte production (Oddy, 2002). Despite its more common association with stress and the "fight or flight" response, healthy cortisol levels are essential to normal growth and development and may influence infant temperament (Hinde *et al.*, 2015). Erythropoietin, in addition to stimulating erythropoiesis (red blood cell formation) and providing protection against anaemia, is known to promote growth and maturation of cardiovascular, smooth muscle, and endothelial tissues, and to also regulate immunoglobulin production by B cells (Semba and Juul, 2002). It has also been shown to produce tumour regression, provide anti-inflammatory benefits, and promote wound healing (Mittelman *et al.*, 2001; Arcasoy, 2008). Prolactin, discussed at length previously for its role in lactogenesis, is another hormone playing a dual bioactive role which includes immunomodulation. In animal studies, prolactin has been shown to enhance immune response to vaccines, facilitate speedier maturation of mucosal epithelia, influence leukocyte maturation, promote T cell-dependent activation of macrophages and natural killer cell response, and stimulate increases in production and activity of antigen-specific T cells (Ellis *et al.*, 1997; Jansen *et al.*, 2015).

Sex hormones, also present in human milk, are popularly associated with their later role in driving the onset of puberty and the development of secondary sex characteristics, rather than immunomodulation. However, oestrogen, progesterone, and androgens are all present in human milk, and these are known to influence sex-based immune differentials in adults. Females have greater numbers of helper T cells and generally have a more robust immune response than their male counterparts. This benefit is offset by the greater risk of autoimmunity in the female immune response (Fish, 2008). This disparity in immune response is believed to be the result of hormonal regulation: specifically, the impacts of oestrogen and progesterone on the ability to mount a well-regulated inflammatory response to infection, and the immunosuppressive effects of testosterone. Infants, too, reflect sex differences in immune response, although there is currently a paucity of studies directly addressing sexual dimorphism in childhood immunity (Muenchhoff and Goulder, 2014). This is most prominently observed in sex differentials in risk of sudden infant death syndrome (SIDS), where the

period of peak excess male mortality is correlated with an age-related surge in testosterone production and an altered cytokine profile in male infants (Moscovis *et al.*, 2014). As cases of SIDS (and also cases of stillbirth, which also reflect a bias towards excess male mortality) are frequently associated with infection, it is suggested that testosterone levels may act to the detriment of immune regulation in the developing male infant (Blackwell, 2015). While the sex-based differences in infant hormonal profile have been demonstrated, independent of feeding regime, endogenous sex hormones in human milk are believed to be present at levels too low to exert bioactivity (Fang *et al.*, 2017). Nonetheless, as breastfeeding reduces SIDS risk (Hauck *et al.*, 2011), the role of endogenous sex hormones in human milk as immunomodulators – alongside the suite of other immunomodulatory and passive protection elements in milk – may warrant further investigation as part of much-needed research on sex differentials in childhood immune response.

3.3.3 Shifts in composition associated with stage of lactation

Human milk is not a static or homogenous product, reflecting consistent concentrations of the elements discussed in the previous section. It is dynamic, not only varying in composition between mother-infant pairs, but also between different points in a single feed, and also in response to infection in either party (Riskin *et al.*, 2012). There is even some evidence that both non-human primate and human mothers produce milk of differing compositions based on infant sex (Hinde, 2007; Powe *et al.*, 2010). Milk is also highly variable over time, changing its proportion of various bioactive constituents in predictable ways over the course of lactation. Understanding the adaptive character of breastmilk is essential to comprehending the importance of breastfeeding as a modulator of disease impact in the present study. The following sections will briefly summarise these shifts in composition and illustrate how human milk is adaptive to the needs of the infant and growing child at different stages of development.

3.3.3.i Colostrum

Colostrum, the earliest milk to be produced, serves in a multifactorial capacity. Its primary roles are to purge the bowels of meconium, to keep the newborn hydrated until the onset of copious milk production, and to act as a bolus, or loading dose, of protective immunological elements. Consequently, colostrum contains relatively high concentrations of electrolytes, whey protein, and also bioactive components such as immunoglobulins and lactoferrin. It contains low concentrations of lactose and fat, and

no casein (Neville *et al.*, 2001). The low volume of produced colostrum results in a high concentration of innate immune components, which is theorised to provide adequate surface protection to the mucosa of the respiratory and gastrointestinal tracts (Pang and Hartmann, 2007). Concentrations of sIgA in colostrum are particularly high (Chirico *et al.*, 2008; Jakaitis and Denning, 2014). Lactoferrin levels are nearly as high, (Jakaitis and Denning, 2014), as are maternal cell counts. During the early period of lactation, infants may consume billions of maternal leukocytes per 24-hour period (Ballard and Morrow, 2013). Concentrations of HMOs and lactoferrin in colostrum are also very high, (Bode, 2012; Siqueiros-Cendón *et al.*, 2014), helping to nourish and shape the infant microbiome. A unique profile of probiotic bacteria characterises colostrum, predominantly biased in favour of *Lactobacillus, Staphylococcus, Streptococcus,* and *Lactococcus* strains (Cabrera-Rubio *et al.*, 2012). Cytokines, too, are present in greater concentrations in colostrum than in later milk, particularly those associated with a pro-inflammatory response to acute-stage infection (Garofalo, 2010; Marcuzzi *et al.*, 2013).

3.3.3.ii Transitional Milk

Transitional milk, as suggested by its name, represents a brief liminal interlude between the phases of colostrum and mature milk, following the onset of copious milk production. Occupying this intermediate phase between roughly 5-15 days postpartum, the composition of transitional milk shares similarities with both colostrum and mature milk. In contrast to colostrum, transitional milk is characterised by high lactose and increasing levels of fat and caseins, alongside overall decreases in total protein and electrolyte content. The bulk of caloric contributions shifts from protein (in colostrum) to fat and carbohydrates (Neville and Morton, 2001; Senterre and Rigo, 2010). Overall volume increases during this period as the infant stomach capacity expands and produces greater demand, and the maternal neuroendocrine mechanisms are stimulated to meet supply. Consequently, increasing milk volume results in reducing, but still substantial, concentrations of nearly all immune factors.

3.3.3.iii Mature milk

Mature milk is characterised by more dilute concentrations of most bioactive factors, as overall volume sharply increases, and supply stabilises. While clear distinctions exist between the composition of colostrum and mature milk, characterising the variability of mature milk over time is less simple. Longitudinal study of changes to the composition of mature milk associated with duration of lactation are thus fraught with difficulty.

Such research often attempts to draw conclusions about long-term trends using a limited pool of participants, which may not control adequately for variation stemming from time of day, point in feed at which sampling is taken, stage of lactation, or treatment of donated milk. Despite these difficulties and the necessity of caveats when negotiating conflicts in data, milk composition is largely thought to be stable over the course of mature lactation, following the dramatic shifts of the first postpartum month, with changes to concentration of components remaining subtle and incremental (Ballard and Morrow, 2013).

Total protein levels fall dramatically from colostrum to mature milk, and then continue to decline thereafter very gradually. This parallels a decrease in protein requirements, per kilogram of body weight, required for growth after the second month postpartum (Boudry *et al.*, 2010; Lönnerdal *et al.*, 2017). Despite the decline in total protein, the nutritive value of protein in milk remains stable over time, as reflected by a consistent ratio of essential amino acids to total amino acids (Lönnerdal *et al.*, 2017). Bioactive proteins, such as lactoferrin, also decrease sharply in concentration between the colostrum and mature milk phases (Jakaitis and Denning, 2014; Siqueiros-Cendón *et al.*, 2014).

Antibody and leukocyte concentrations in milk are also thought to change significantly during the first year of life (Järvinen and Suomalainen, 2002; Chirico *et al.*, 2008; Jakaitis and Denning, 2014). However, in absolute terms these levels are still high, and biologically significant. Moreover, due to increases in the volume of milk consumed, the amounts of IgA received throughout lactation may remain similar (Hanson and Korotkova, 2002). Other antibodies do not follow this documented pattern of high levels at initiation, followed by decrease over time. IgM levels are low, even in colostrum and decrease over time, while IgG levels positively increase over lactation. This may, again, be seen as due to the need-profile of the developing infant, and the adaptive nature of human milk: IgM is produced by infants from birth, thus making supplementation somewhat unnecessary; while IgG promotes active immunological development and is not produced by infants until around 6 months of age, explaining its increasing strategic importance in milk (Lönnerdal *et al.*, 2017). A similar argument has been made for a decline in the importance of supplementary milk cytokines, where concentration of interleukins has been observed to decrease over three phases of lactation (Yilmaz *et al.*, 2007).

Constituents closely tied to metabolic health are also thought to shift over time. Concentration of HMOs declines by an average of over 50% (Bode, 2012). Further gradual declines in HMO concentration have been observed between three months to one year postpartum. However, researchers argue that these lower levels may still be biologically significant, due to higher quantities consumed by the older infant, and the consistent pathogen-inhibiting behaviours of HMOs at low concentrations (Chaturvedi *et al.*, 2001). Thus, we may consider that the lower concentrations observed for mature milk may constitute a "maintenance dose", which is necessarily less than the extremely high quantities of bioactives present in colostrum. The profile of milk bacteria also shifts from colostrum, with a reduction in species diversity observed over time. Over the course of lactation, *Lactobacillus* species expand their predominance, while also exhibiting increases in *Bifidobacterium*, *Staphylococcus*, and *Lactococcus* (Khodayar-Pardo *et al.*, 2014). Decreases were also observed for *Streptococcus*, alongside the appearance of oral flora bacteria such as *Veillonella*, *Leptotrichia*, and *Prevotella* (Cabrera-Rubio *et al.*, 2012). Concentrations of adiponectin in human milk are known to decrease by approximately 5-6% for every month of lactation (Newburg *et al.*, 2010). By 12 months lactation, erythropoietin concentrations rise to over 30x their immediate postpartum levels, (Semba and Juul, 2002). Ghrelin and leptin show similar change over time in keeping with the needs of growth (Aydin *et al.*, 2006; Schuster *et al.*, 2011). These elements influencing tissue development and metabolic programming may also be seen as meeting the needs specific to the stage of growth and development at each age.

There is a paucity of research characterising milk composition beyond the first year of life. However, a recent study by Perrin *et al.* (2017) analysed a longitudinal series of milk samples from 19 lactating mothers from 11-17 months postpartum. These were compared to donated milk bank samples from the first year of lactation, in order to assess the suitability of later-lactation donors to milk banks serving younger infants. Longitudinally-sampled milk during the study period showed no decrease over time in the concentration of elements such as fat, carbohydrates (measured as lactose), iron, and potassium. Other elements, such as total protein, lactoferrin, lysozyme, IgA, and HMOs were found to *increase* between 11-17 months, with only zinc and calcium showing a decline over time. When compared to milk bank samples from donors in the first year of lactation, concentrations of total protein, lactoferrin, lysozyme, and IgA were also found

to be higher than those of the banked samples, as well as higher than reported values for early mature milk in a review by Ballard and Morrow (2013). Only zinc, calcium, iron, and HMO concentrations were lower for the longitudinal group than for milk bank samples. These results appear to contradict earlier findings concerning the predominant trend of decline in these milk components, particularly in terms of changes to milk protein content over time, which appears to rebound between the end of the first of the year and middle of the second. However, the authors argue that the cross-sectional nature of the vast majority of milk studies has obscured real longitudinal changes to milk composition through a failure to control for much greater inter-mother variation (Perrin *et al.*, 2017). Instead, these findings suggest a trend towards stability and adaptability, rather than decline, observed for macronutrient and bioactive components in advanced stages of sustained lactation (e.g. Dewey *et al.*, 1984; Perrin *et al.*, 2013).

3.3.3.iv Involution secretion

Known changes to milk composition over time, which may result directly from a reduction in demand, are rarely discussed in bioarchaeological weaning studies. The rebound in concentrations of major constituents of milk, described by Perrin *et al.* (2017) as being time-related, may alternately be related to behavioural changes: in other words, not due solely to the duration of lactation, but to changes in composition resulting from a gradual weaning process which would be expected for children breastfeeding into the second year of life. These alterations to milk composition result from a process called *involution*, the inactivation and remodelling of organ tissue due to reduced use or disuse, which may occur after abrupt cessation of breastfeeding, or during the weaning process itself, if gradual. The process of involution has been most thoroughly documented in studies of dairy animals and mouse models, which have demonstrated differences in physiological response to gradual vs. abrupt weaning. Abrupt weaning produces a trauma-like inflammatory immune response, in order to protect the maternal body from sepsis, while gradual weaning is known to result in lower tissue stress and a more balanced maternal immunological response (Silanikove, 2014). In terms of the changes to milk itself, both sudden and gradual cessation of breastfeeding have long been known to result in elevation of lactoferrin, IgA, IgG, IgM, lysozyme, albumin, α-lactalbumin and casein content of milk, increases in the sodium and chloride concentrations, and reductions in zinc and calcium content (Hartmann and

Kulski, 1978; Garza *et al.*, 1983; Goldman *et al.*, 1983; Dewey *et al.*, 1984; Prosser *et al.*, 1984).

These changes recorded for mother-infant study cohorts in the first year of lactation parallel the characteristic increases in total and biologically active proteins observed in the second year by Perrin *et al.* (2017). Thus, it appears that the alterations to composition taking place during weaning relate to behaviourally-mediated reductions in the volume produced and downregulation of mammary activity, not to duration of lactation. The fluid produced in the most latterly stages of gradual weaning, as breastfeeding tapers to a close, is termed involution secretion, and this may be thought of as a volume-compensatory immunological "bookend" to its counterpart at the outset of lactation, colostrum. Indeed, recognition of the resemblance between the end stage product of involution and colostrum in mammals, in both gross appearance and composition, was established as long ago as the mid to late 20th century (McCance and Widdowson, 1951; Hartmann and Kulski, 1978). Densely packed with immunoprotective constituents, involution secretion protects not only the involuting mammary from infection and mastitis as secretion of milk is downregulated but boosts the level of protection available to offspring as they transition to full independence from human milk (Goldman *et al.*, 1998; Fetherston *et al.*, 2001).

3.3.4 Human milk and the changing microbiome

The human microbiome is emerging as an important area of research in human health and host defence, and the role of human milk is increasingly recognised as a key determinant of the microbiome in early life and an influence beyond that period. Thus, interest has recently been shown in characterising the timing and causes of significant transitions in the population of the microbiota. Previous studies have indicated that the introduction of solid foods, to form a complementary diet with breastmilk, was the primary catalyst of the shift from a human milk microbiome to one more similar to those of adults (Fallani *et al.*, 2011; Koenig *et al.*, 2011; Voreades *et al.*, 2014).

However, a recent metagenomic study by Bäckhed *et al.* (2015), utilising a longitudinal cohort of Swedish mothers and infant pairs (n=98) to examine formative influences on the infant microbiome, has produced findings which underline the essential role of breastfeeding in directing infant microbiota. In contrast to previous studies, this study found that development of an adult-like microbial flora was consistently associated with full cessation of breastfeeding, rather than the introduction

of complementary foods. The microbiota of children receiving complementary foods alongside continued breastfeeding, even at 12 months of age (the highest age covered in the study), still reflected a predominance of human milk species such as *Bifidobacterium* and *Lactobacillus*, in contrast to no-longer breastfed children possessing more adult-like microbial populations (Bäckhed *et al.*, 2015). These findings have since been mirrored by a smaller longitudinal study following a cohort (n=11) of Brazilian children up to the age of 11 months, in which predominantly or exclusively breastfed children (up to 6 months of age) displayed a similar resilience of microbial colonisation unperturbed by the introduction of complementary foods (Carvalho-Ramos *et al.*, 2017). These findings strongly suggest that it is the provision or withdrawal of human milk to the infant gut which exert the strongest influence of shaping the microbiome of not only early infancy, but also early childhood. As we are continuing to discover the ways in which the microbiome is a crucial mediator of short-term host defence, long-term maturation of the immune response, and metabolic function, this provides further weighty evidence against the categorisation of human milk protections as predominantly "passive" in nature.

3.3.5 Morbidity and mortality of the breastfed child

As previous sections have outlined the biological parameters of lactation, immune ontogeny, and the role of breastfeeding in host defence over time, the present section will briefly summarise evidence for the real-life impacts of breastfeeding behaviours on the morbidity and mortality of children.

The timing of initiation for breastfeeding has critical importance. Evidence strongly suggests that delaying initiation of breastfeeding beyond the first day of life results in significant increases in both infant morbidity and mortality (Clemens *et al.*, 1999; Edmond *et al.*, 2006; Mullany *et al.*, 2008; Debes *et al.*, 2013), and a systematic review and meta-analysis found a doubled risk of death when breastfeeding was delayed beyond the first hour of life (Khan *et al.*, 2015). Probable provision of potentially contaminated, alternate food sources prior to the initiation of breastfeeding fails to adequately account for this observed rise in overall infections and death among infants with delayed access to the breast. The data reveal that sepsis and respiratory infection account for a significant proportion of deaths, alongside gastrointestinal infection, and necrotising enterocolitis, which is caused by dysfunctional immune response (Edmond *et al.*, 2007; Ip *et al.*, 2007; Mullany *et al.*, 2008; Khan *et al.*, 2015). This multi-causal

distribution of excess deaths among non-breastfed infants strongly indicates that positive protections against infection and excess inflammation provided by colostrum's bioactive elements, rather than simple shielding from exogenous contaminants leading to diarrheal illness, form the basis of differences in the health and survivorship of infants accessing or denied provision of colostrum.

These differences in morbidity and mortality between breastfed and non-breastfed children, which begin during the colostrum phase, extend to other phases of lactation. A robust body of evidence exists to support current recommendations of exclusive breastfeeding for the first 4-6 (optimally six) months of life, followed by the introduction of solid foods at around six months, and continued breastfeeding alongside a complementary diet thereafter for a minimum of two years as optimal for child health. Exclusive breastfeeding for the first 6 months of life and supplementary breastfeeding up until the age of two years of age is associated with reduced incidence of morbidity and mortality associated with ear, respiratory, and gastrointestinal infections, diabetes, SIDS, and childhood leukaemia, with some protective effects continuing years beyond the cession of breastfeeding (López-Alarcón *et al.*, 1997; Kwan *et al.*, 2004; Bahl *et al.*, 2005; Ip *et al.*, 2007; Lamberti *et al.*, 2011; Strand *et al.*, 2012; Mamun *et al.*, 2015; Victora *et al.*, 2016). Comparisons of exclusively breastfed, partially-breastfed, and non-breastfed infants have revealed dose-dependent effects within the first six months of life. Partially-breastfed infants in this age group occupy an intermediate niche between fully-breastfed and non-breastfed infants, enjoying the same protective effects of fully-breastfed infants, though to a lesser extent dependent on the proportion of diet coming from human milk (Scariati *et al.*, 1997; Chantry *et al.*, 2006; Quigley *et al.*, 2007).

Despite widespread perceptions that breastfeeding practices lack importance in affluent "developed" countries due to a decreased burden of infectious disease, relative to infants in low-income nations, differences in health are observable between breastfed and non-breastfed infants in industrialised nations also. Evidence of significantly reduced morbidity from ear, respiratory, and gastrointestinal infections, SIDS, and also overall all-cause mortality beyond the neonatal period has been found in large studies of breastfed children under the age of 12 months in affluent populations in the United States, Spain, the Netherlands, and the United Kingdom (among others), relative to cohorts of formula-fed children (Dewey *et al.*, 1995; Scariati *et al.*, 1997; Chen and

Rogan, 2004; Chantry *et al.*, 2006; Paricio Talayero *et al.*, 2006; Quigley *et al.*, 2007; Duijts *et al.*, 2010; Fisk *et al.*, 2011). Breastfeeding population research on health impact in Western industrialised nations has long suffered from poor study design, especially a lack of careful "breastfeeding" group classification by level of exclusivity, which ignores dose-dependent effects. This is exemplified in research such as the notable sibling-comparison US population study by Colen and Ramey (2014), which called long-term effects of breastfeeding into question, and which classified as breastfed all infants who experienced "any breastfeeding". Few studies have differentiated between exclusive, predominant, partial, and token breastfeeding in their designation of "breastfeeding" groups (Ip *et al.*, 2007), resulting in difficulties in estimating actual impacts, and increasing the likelihood of *underestimating* benefit in wealthy nations.

Continued protective mechanisms of breastfeeding are more controversial beyond two years of age. However, researchers have pointed out that breastfeeding beyond the age of two years is greatly understudied, and the lack of evidence for benefit beyond this age does not necessarily imply a lack of continued protective activity (Chantry *et al.*, 2006). Indeed, the dose-dependent effects observed for infants who are partially-breastfed at younger ages indicates that there is no reason to assume that breastfeeding ceases to positively impact the health status of children until either breastfeeding is discontinued, or full immunocompetence is achieved (whichever occurs first). Studies examining mortality risk in children between the ages of 6-23 months have consistently found elevated morbidity and mortality in children not being breastfed during that period, particularly due to diarrheal and respiratory infection (Lamberti *et al.*, 2011; Strand *et al.*, 2012; Lamberti *et al.*, 2013; Sankar *et al.*, 2015), indicating the continued protection received by children continuing to breastfeed alongside a solid diet. Evidence beyond this period is sparse, but strongly suggestive of a continued bioactivity of breastmilk beyond the somewhat arbitrary limit of two years. The majority of research has come from populations in developing countries, where breastfeeding beyond two years of age is unexceptional. Several studies examining the health of children from the ages of 1-3 years in Guinea-Bissau, found that breastfeeding was protective against diarrheal morbidity, and also that children weaned for reasons other than perceived health and readiness – particularly maternal pregnancy – were at much higher risk of death, irrespective of age within the cohort (Mølbak *et al.*, 1994a; Mølbak *et al.*, 1997; Jakobsen *et al.*, 2003b). In one study, up to the maximum age covered by the study

(rather than the maximum age of benefit), weaned children were shown to face a higher risk of diarrheal illness, a longer mean duration of illness, and a 3.5 times higher risk of death, compared to breastfed children (Mølbak *et al.*, 1994a). This aligns with findings of reduced mortality among malnourished children up to the age of 3 years in Bangladesh (Briend *et al.*, 1988; Briend and Bari, 1989)

Findings were mixed in a 2013 review of studies on breastfeeding children up to 2 years and beyond, which underlined the lack of studies in this area and concluded that available evidence was currently insufficient to merit firm conclusions on the effects of breastfeeding beyond early infancy (Delgado and Matijasevich, 2013). A more recent study by Mattison *et al.* (2015) explicitly sought to examine child health in Tanzania for children breastfed beyond two years of age. The authors reported few indications of improved health status among children nursed beyond two, with only markers of reduced inflammation, increased height for age, and a reduced likelihood of having a physician-diagnosed infectious illness, and no trends which reached statistical significance. The authors suggested that the health impacts of breastfeeding may attenuate with age, adding the caveat that children who are weaned from the breast earlier may differ from children who are breastfed longer. This latter point is highly significant as a confounder to studying the effects of breastfeeding in older children, as mothers may intentionally breastfeed vulnerable offspring longer than their more robust or well-nourished counterparts, producing apparent correlations between longer breastfeeding and poorer health status (Simondon and Simondon, 1998; Simondon *et al.*, 2001a).

Simondon (2009) argues that the possibility of mothers buffering their sickly or malnourished children from further sickness and death through delayed cessation of breastfeeding creates a hazard to interpretation; rather than breastfeeding being a causative factor for the development of malnutrition, it is far more commonly the case that malnutrition or illness leads to delayed weaning from the breast. Such comparisons of breastfed and non-breastfed children where sustained breastfeeding in the age category of interest is *not* the cultural norm may serve to occlude the health impacts of breastfeeding beyond very early infancy and are suggested to lead to an active underestimation of the supportive role of breastfeeding for children between the ages of 18 and 30 months of age (Simondon, 2009). As evidence suggests that the bioactive elements of human milk are remarkably conserved throughout lactation, the relative

benefits of continued breastfeeding through early childhood are also extremely likely to depend, not on changes to the character of milk itself, but on the combined individual circumstances of robusticity and immunocompetence attained by the child, and on the level of challenge and risk in the surrounding environment.

Interpretation of past breastfeeding behaviours as a marker of parental investment may also be complicated by the variety of possible parental responses to environmental risk. While breastfeeding may be deemed "natural" as a definitively mammalian behaviour, in humans the parameters of breastfeeding are highly variable, mediated by culture, environment, and individual agency (Holman and Grimes, 2003; Du and Mace, 2017). Furthermore, while breastfeeding is a risk-reduction strategy, extreme infant mortality risk may also lead to parental disinvestment when cost outweighs benefit. Evolutionary anthropologists have found that parents may increase investment in moderate-risk pathogenic environments, relative to low-risk environments, but disinvest in the highest risk environments (Pennington and Harpending 1988; Quinlan, 2007). However, some variability has found between parenting strategies among groups occupying the same environment, suggesting the importance of considering culture (Hewlett *et al.*, 2000; Fouts and Silverman, 2015). The adaptive nature of breastfeeding as a biological, cultural, and individual response therefore makes the impacts of past breastfeeding behaviours difficult to ascertain, where these parameters are unknown. However, what *is* certain is that breastfeeding does improve child survival in most environments, and thus should be considered as a possible risk reduction strategy.

A 2003 study addressed the role of breastfeeding in ameliorating relative risk, comparing the mortality risk of weaned children aged 9-20 months in Guinea-Bissau living as refugees during the first three months of a war with the mortality risk of similarly weaned children in the two years prior, and with breastfed children of the same age range during both periods (Jakobsen *et al.*, 2003a). When controlling for age, a six-fold increase in mortality risk was observed for weaned children during the conflict, relative to weaned children in the years prior, while no differences in mortality were seen between the breastfed war and peacetime cohorts. Thus, cessation of breastfeeding earlier in childhood may present fewer risks in some circumstances than others; or, conversely, later cessation may offer greater or lesser levels of benefit dependent on circumstance and relative need. As immunocompetence is a gradual process, children under the age of 5 years represent the highest-risk group for infectious

morbidity and mortality worldwide, and there is evidence that elements of immunity, such as production of the Th1-mediating cytokine IL-12, may not reach full adult levels of until adolescence, impairing the ability of children of all ages to produce a robust and mature response to infection (Upham *et al.*, 2002; Dowling and Levy, 2014). It is therefore important when discussing continued benefits of breastfeeding during early childhood to be aware of environmental hazard and life history variables (including *in utero* development) which may predispose some children to greater exposure or vulnerability to infection than others. It is also essential to acknowledge the rule of maternal (and to some extent infant) agency in negotiating these case-by-case variables in decisions about the weaning process (Moffat, 2001). As continued mechanisms of protection via breastfeeding throughout lactation have both biological plausibility and limited data, and as understanding the health of children in the past – in periods where breastfeeding duration is often assumed to exceed two years of age – the question of continued health impacts of sustained breastfeeding will remain important.

Breastfeeding is known to provide resistance to specific pathogens relevant to the present research, primarily parasitic infections. Research has demonstrated that breastfeeding at the age of 12 months and beyond is protective against a range of parasitic protozoan and infections, including *Cryptosporidium*, *Giardia*, and amebiasis, all associated with diarrhoeal disease and the potential for malnutrition and impaired growth (Morrow *et al.*, 1992; Mølbak *et al.*, 1994b; Mahfouz *et al.*, 1997; Korpe *et al.*, 2013). Human milk may also offer some protections against malarial infection or the development of episodes of clinical illness. While the low observed incidence of malaria during the first six months of life is generally believed to result from placental transfer of maternal IgG, direct evidence for this hypothesis is lacking (Dobbs and Dent, 2016). Human milk is known to contain factors which have demonstrated *Plasmodium*-inhibitory activity *in vitro* or which are known to promote parasite clearance, and some of these elements act systemically, such as antibodies, lactoferrin, and TGF-β. Sinnis *et al.* (1996) found a specific role for lactoferrin in blocking malarial sporozoites from invading liver cells in a mouse model study, suggesting that this may go some way to explaining lower prevalence of malaria among neonates. This has led some researchers to consider the possibility of breastfeeding as a protective factor against malarial infection and disease (Kassim *et al.*, 2000; Riley *et al.*, 2001; Doolan *et al.*, 2009; Kangoye *et al.*, 2014). A protective effect specifically relating to breastfeeding is

suggested by Kalanda *et al.* (2005)'s finding that introduction of complementary foods (and thus displacement of maternal milk in the diet) was associated with increased risk of malarial episodes in Malawian infants. Breastfeeding of infants in endemic malarial regions may be of increased importance, since infants born to mothers infected with *P. vivax* face increase risks of *in utero* undernutrition and low birth weight and may also be born with a reduced neonatal immune response (Anstey *et al.*, 2009; Gbédandé *et al.*, 2013). As compromised immune function is both a cause and effect of malnutrition, with reciprocal feedback between the two states (Bourke *et al.*, 2016), the importance of breastfeeding to maintenance of nutritional status, health and survival in endemic malarial environments is underlined.

3.4 Complementary feeding and nutrition

Introduction of complementary foods at around six months of age (and not before four months) with continued breastfeeding, following a period of exclusive breastfeeding, is considered optimal by current recommendations (Kramer and Kakuma, 2012). The following section briefly reviews evidence for the impacts of complementary diet on growth and health.

3.4.1 The end of exclusive breastfeeding

The appropriate age at which to introduce solid foods alongside breastfeeding has been subject to some debate, as clinicians and medical bodies attempt to make comprehensive recommendations which balance the risks of premature displacement of breastmilk and increased risk of metabolic dysfunction which can occur with early introduction of solids, with the alternate risks of micronutrient deficiency which can occur when solids are excessively delayed, inappropriate, or inadequate. Early introduction of solid or non-breastmilk foods has been associated with elevated risk of disease in some studies. Researchers suggest that introduction of cow's milk or solid foods before the age of four months is associated with increased risk of childhood obesity (Seach *et al.*, 2010; Huh *et al.*, 2011; Moss and Yeaton, 2014), Type 1 diabetes (Rosenbauer *et al.*, 2008; Frederiksen *et al.*, 2013; Ierodiakonou *et al.*, 2016), and the development of autoimmune issues such as food allergy and coeliac disease (Norris *et al.*, 2005; Fiocchi *et al.*, 2006). Delay of introduction of solids has also been observed to result in reduced markers of oxidative stress (Frederiksen *et al.*, 2015), also reflective of metabolic health.

In addition to risk of metabolic dysfunction, complementary foods may introduce pathogens, increasing risk of infection. This risk is commonly cited as half of the "weanling's dilemma": infants must begin to consume solids around the middle of the first year, due to declining stores of iron and the need to supplement milk macro- and micronutrients at this juncture, but risk foodborne infections from unhygienic complementary foods, which paradoxically may negatively impact on their nutritional status (Lutter, 1992). The risks of complementary feeding in hygienically non-optimal environments may be overstated within archaeology, based on an either/or conception of breastfeeding and solid foods. Studies show that continued breastfeeding greatly mitigates the impact of foodborne illness in low-income countries. Children who continue to be breastfed alongside complementary foods commonly experience fewer and less severe health impacts from diarrhoeal illness than non-breastfed children, even beyond infancy (Betrán *et al.*, 2001; Strand *et al.*, 2012; Budhathoki *et al.*, 2016; Wright *et al.*, 2017). Filteau described this systematic overestimation of risk and underestimation of human milk as evidence that

> ...complementary foods are not themselves the major problem, and it is the active role of breast milk which is primary for maintaining gut integrity. Thus, although we may have overestimated the damage caused by weaning foods, we may have underestimated the gut protective effects of breast milk (Filteau, 2000: 567)

Thus, the most perilous period for breastfed children occurs not at the outset of the weaning process, when solid foods are introduced, but at the conclusion of the complementary feeding period, when they cease to be breastfed.

3.4.2 Adequacy of complementary foods

As cited in the "weanling's dilemma", around the middle of the first year infant body stores of iron and zinc become depleted, alongside low or reducing levels of iron, zinc, and calcium in human milk (Fewtrell *et al.*, 2007; Kramer and Kakuma, 2012). The introduction of solid foods thus becomes necessary to augment intake of these micronutrients and overall caloric intake, as the requirements of rapid growth outstrip what is available from milk alone, to prevent the development of micronutrient or macronutrient deficiencies. However, unfortified traditional weaning foods in most cultures are vegetable or cereal-based foods which have high phytate levels, interfering with absorption of iron, zinc, and calcium (Bhutta, 2000; Gibson *et al.*, 2010). Thus, complementary diets may have varying levels of benefit to the breastfed infant, depending on the foods available or offered. An optimal diet for infant health is now

considered to include a wide variety of foods from differing food groups, rather than a specialised diet of limited "infant" foods, alongside continued breastfeeding, and many authorities recommend family foods early in the complementary feeding period, including proportional inclusion of foods rich in B vitamins, zinc, and iron, such as meat, fish, or eggs (Agostoni *et al.*, 2008; Coulthard *et al.*, 2009). While the onset of complementary feeding is somewhat constrained, the length of the complementary feeding period is highly variable cross-culturally, as well as among primates (Sellen, 2009).

Breastfed children have differing growth trajectories in the first year of life than children who are formula-fed, growing less rapidly in length and weight-for-length during that time. However, while this is not reflected in shorter final adult stature, this slower early growth profile is associated with lower risks of metabolic disease associated with high protein consumption and rapid growth in the first year (Dewey, 2009). The persistence of breastfeeding in environments where complementary foods are insufficient or inappropriate, as a means of extra support, has led to widespread perception of breastfeeding as a cause of malnutrition and stunting. It is also not uncommon to hear breastfeeding by nutrient-deficient mothers cited as a cause of micronutrient deficiency in young children (cf. White *et al.*, 2006; Walker *et al.*, 2009), without further exploration of the wider implications of these deficiencies (i.e. is breastfeeding the cause of deficiency, or merely a means of transmitting pre-existing dietary deficiencies common to others in the cultural group which are also reflected in complementary foods? Or is this a reflection of differential access or behaviour?). These issues require some brief examination, considering published data on growth patterns and stunting among breastfed toddlers in low-income countries, and the known relationship between impaired immune function and nutrient deficiency (Scrimshaw *et al.*, 1968; Solomons, 2007).

Many studies in recent decades have examined the long-observed association between malnutrition and sustained (or the more value-laden "prolonged") breastfeeding in developing nations. While earlier studies suggested that breastfeeding may play a causal role in the development of malnutrition and consequent stunting (e.g. Victora *et al.*, 1984; Brakohiapa *et al.*, 1988; Thoren and Stintzing, 1988), more recent research has reached a predominant consensus that that the principle of reverse causality is in play: it appears that being small for age, malnutrition, and stunting may cause

mothers to opt for longer periods of breastfeeding to increase odds of infant survival (Marquis, 1997; Simondon and Simondon, 1997; Fawzi *et al.*, 1998; Simondon and Simondon, 1998; Habicht, 2002; Kramer *et al.*, 2011; Jones *et al.*, 2014). In fact, while breastfed infants grow more slowly in the first year of life, longer duration of breastfeeding has been associated with greater linear growth during the second year in several studies (Onyango *et al.*, 1999; Simondon *et al.*, 2001b; Habicht, 2002).

Timing, for both introduction of complementary foods and growth faltering, also form important variables in child growth and health. Observed differences between normal-birthweight breastfed infants in affluent and resource-poor nations indicate that growth faltering occurs during the period of complementary feeding, rather than the period of exclusive breastfeeding, indicating similarity of milk quality and variability of complementary food quality (Dewey *et al.*, 1992). While introduction of solid foods before six months of age has been shown to displace milk intake (Cohen *et al.*, 1994; Dewey *et al.*, 1999), the relationship between continued breastfeeding and complementary feeding after this period is less competitive. Providing that intake of more difficult-to-digest solid foods is not excessive, breastfeeding may continue to form a significant source of calories and nutrients which does not impact negatively on growth throughout the complementary feeding period (Onyango *et al.*, 1998; Dewey, 2002; Kumwenda *et al.*, 2016). Thus, it is differential *access* to adequate complementary foods – associated with variables of socioeconomic and educational status – and state of wellbeing which determines nutritional competence or deficiency, and subsequently impaired growth (Dewey *et al.*, 1992; Fawzi *et al.*, 1998; Anderson *et al.*, 2008; Krebs *et al.*, 2011). Infection, on the other hand, may cause sickness-induced anorexia (Ayres, 2013), which may exacerbate pre-existing malnutrition arising from inadequate complementary feeding. Considering its role in reducing the energy expenditure required for immune response, breastfeeding - of any duration - is more likely to provide unambiguous benefit to the sick or malnourished child than to undermine health status and survival.

3.5 Summary

Patricia Stuart-Macadam stated: “Breast milk is not a magic potion, not a panacea for all human ills. It is a vital, dynamic substance that can transmit both beneficial (such as immunoglobulins and nutrients) and detrimental (such as nicotine and alcohol) substances to the infant” (Stuart-Macadam, 1995: 27). This is an accurate assessment of

the essential nature of human milk, which is, broadly speaking, highly evolved as an adaptive nexus between mother and infant, to provide the offspring with nutrition and shared immunity which shield the infant from environmental insult and excessive growth-impairing demands. Human milk is, nonetheless, largely underestimated within bioarchaeology in both its range of bioactive elements and adaptability to the needs of the child throughout lactation.

This chapter has outlined the physiology of human lactation, the development of human immune function, and the role of human milk in facilitating host defence and maturation of independent immunity in the hopes that outlining the complexity of these issues will lead to new respect for the bioactive and adaptive nature of milk as "more than nutrition" among palaeodietary researchers. While breastfeeding is indeed not a panacea for all of human (nor infant) ills, it is nonetheless a substance which has evolved to meet the changing needs of infants and children at different developmental stages and to act as a buffer against physiological challenges during a vulnerable period of life. As children gradually develop independent biological systems for negotiating these challenges without support, the relative importance of breastfeeding diminishes. However, it is important to distinguish between declining need and declining levels of support in understanding the changing role of breastfeeding during early childhood. Improved awareness of the evolving body of clinical and anthropological evidence will enrich future bioarchaeological research on past breastfeeding patterns, and it is hoped that training in these areas will become common among palaeodietary researchers.

4. Childhood in the Early Medieval Fens

"One does not love a place the less for having suffered in it, unless it has been all suffering, nothing but suffering..." (Austen, 1998: 201).

4.1 Introduction

This chapter aims to specifically address the known environmental and cultural factors likely to have affected the health and life experiences of children in the early medieval Fens. This will be accomplished through a description of the unique natural environment of the Fenlands, its history of human habitation and past disease ecology, and an exploration of available historical and archaeological evidence for the lives of children during the early Anglo-Saxon period.

4.2 The Fenlands of eastern England

The Fenlands (or Fens) of eastern England form an area of nearly 4,000 km^2, encompassing portions of the modern counties of Lincolnshire, Cambridgeshire, Norfolk, and Suffolk (Wheeler and Waller, 2009). Prior to their transformation to arable land, which was initiated in the 17th century and completed in the 19th century, the Fens comprised a shallow inland wetland basin which adjoined the Wash estuary of the North Sea and formed the largest wetland area in England (Figure 4.1). The Fen basin resulted from glacial excavation by the Anglian ice sheet, beginning in the north and finally depositing eroded chalk beyond the limits of the basin within East Anglia (Clayton, 2000). Thus, the Fens are bounded to the north, east, and south by deposits of bedrock chalk and till, and to the west by Jurassic clays and limestone (Hall and Coles, 1994: 1).

The movement of water within the Fens has resulted in substantial deposits of overlying sediment (Warham, 2013). The Fenlands of eastern England may be described as a land shaped almost wholly by water; whether by glacial ice, or more recently through alluvial deposition during the Holocene. While the Fens have for most of known history been subject to frequent marine inundation and flooding events from their connection to the Wash, they were also freshwater-fed via the rivers Witham, Nar, Nene, Larke, Great Ouse, Little Ouse, and Welland (Godwin, 1978). These contributions from riverine and marine sources demarcated the Fens into two distinct zones: a northern area of brackish silt fen subject to tidal influences and varying salinity, and a southern region of freshwater peat fen dotted with low-lying islands.

These varying environmental zones produced distinct differences in dynamic, resources, and human settlement patterns, which will be discussed in the sections following.

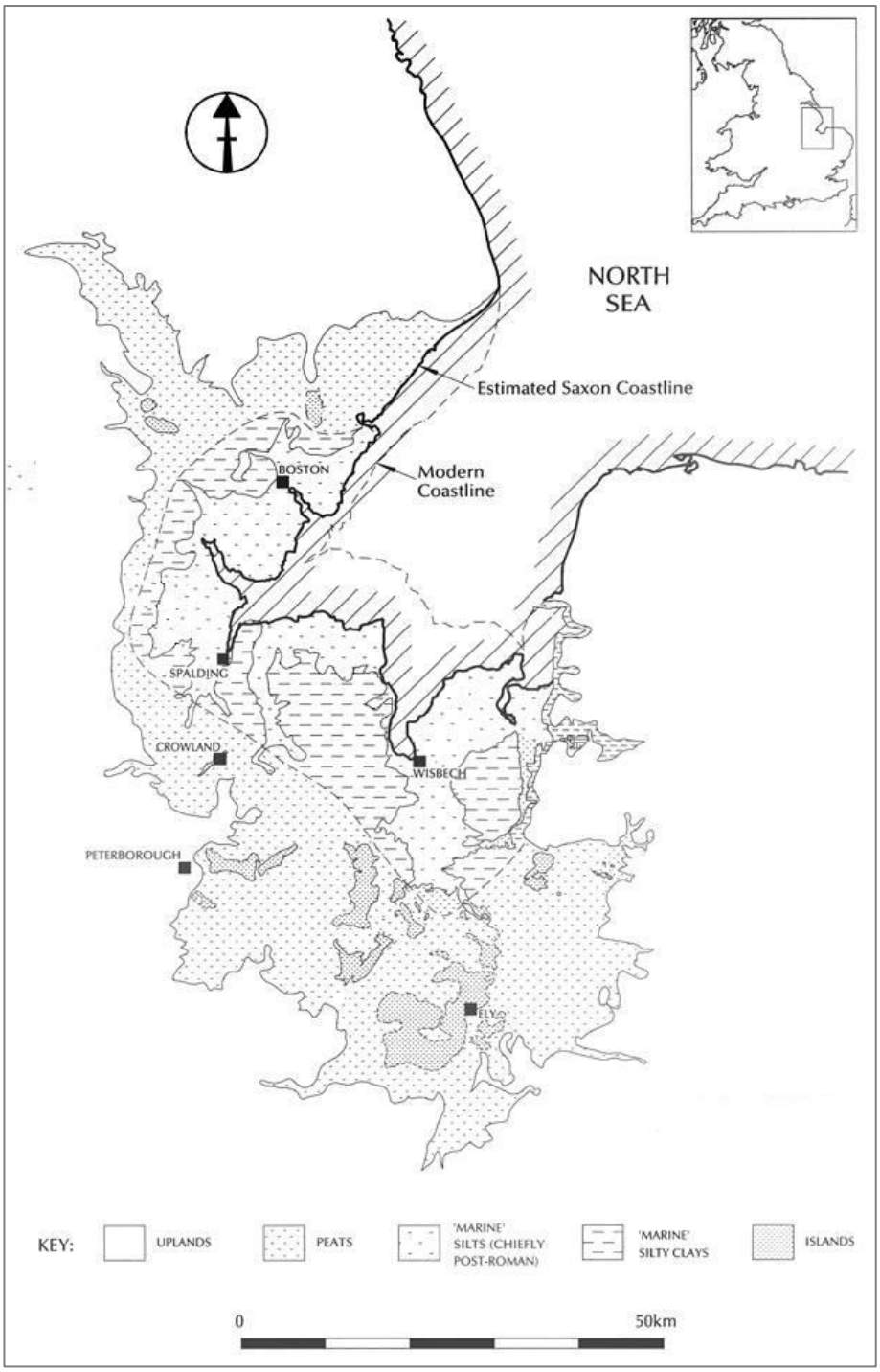

Figure 4.1 Location and topography of the early medieval Fens. Adapted from Penn (2005: 5).

4.2.1 Environment

The siltlands of the northern Fens were formed by a combination of glacial alteration of the landscape and repetitive episodes of marine or freshwater flooding during the

Holocene (Penn and Lane, 2005). The silt Fens are bounded by the Wash inlet of the North Sea and are fringed by a series of coastal salt marshes. Accumulated sedimentary deposits of marine and freshwater origin are estimated to reach depths of up to 26 meters in some areas of the silt Fens (Sturt, 2006). The Fen Basin was primarily wooded until the Neolithic period. After this time, a gradual deforestation driven by rising sea levels, and aided by human clearance, occurred (Hall and Coles, 1994: 46). By the Iron Age, the region consisted almost entirely of open landscape, consisting of meadows, isolated stands of wood, and wetland. Marine inundation and deposition of alluvium, driven by sea level rises during the wet Iron Age, decreased during the drier Roman period, which saw reduced rates of water movement and the formation of silt-clogged roddons (defunct waterways). This more stable trend continued into the early medieval period, with episodes of exceptional tidal flooding occurring intermittently between the 4th and 6th centuries (Penn and Lane, 2005).

Marine flooding also had a profound effect on the development of the area beyond the silt band of the Northern Fens, the peatlands of the central and southern Fens. Inundation of seawater and sediment, which raised the ground level within the northern aspect of the Fens, also impeded river drainage, leading to areas of standing fresh water (Godwin, 1978: 5). The peat Fens were characterised by varying levels of freshwater influence, depending on ground elevation. Three major types of ground existed in this region, each with their own differing micro-ecology: low lying, permanently flooded areas comprising meres and pools, higher intermediate zones which flooded in the winter and emerged as dry land during the warmer months, and permanently dry uplands found on the islands and around the edge of the Fen basin (Oosthuizen, 2017: 3). The consistently-waterlogged areas of the central and southern Fens provided conditions for the development of a stable wetland ecology, and formation of the peat deposits which typified the sediments of the freshwater Fens.

4.2.2 Human occupation of the Fens

It is impossible, in some respects, to speak adequately of the natural environment of the Fens in their pre-drainage state without discussing the human role in actively shaping and maintaining the landscape. Humans are believed to have been present within the area occupied by the Fen basin since the Pleistocene. However, evidence for human habitation within the Fens spans roughly six millennia, with the earliest evidence for occupation occurring in the Mesolithic period (Hall and Coles, 1994: 28). A full

discussion of human-landscape interactions and exploitation of the pre-wetland landscape is outside of the scope of this thesis. Consequently, this section will provide an overview of human exploitation and management of the Fen wetlands in the periods directly preceding the early medieval period.

The Romano-British inhabitants of the Fens left major impacts on the landscape, with some still apparent into the early medieval period, as they expanded their occupation of the area due to falling water levels (Hall and Coles, 1994: 8; Hooke, 1998: 170-171). The Fen economy during this period (as in others) was centred around pastoral agriculture, with additional exploitation of Fen food, crafting, building resources, and intensification of salt-making activity (Hooke, 1998: 171; Rippon, 2000: 43-45; Hayes, 2015). In addition to their talent for opportunistic colonisation, the Romans brought with them knowledge of engineering and infrastructure. The proximity of the Pontine Marshes and their malarial influence on Rome had long since shaped Roman attitudes to wetlands, creating great ambivalence and a strong desire for reclamation. Plutarch described the determination of Julius Caesar to "...draw off the water from the marshes about Pomentium and Settia, and to make them solid ground, which would employ many thousands of men in the cultivation ..." (Dryden and Clough, 1906: 315). Consequently, under Roman influence, the Fens underwent unprecedented levels of alteration to the landscape. Roads, drains, canals such as the Car Dykes, and the construction of the trans-wetland Fen Causeway all served to manage water levels and increase transport and communication capabilities within the Fens (Rippon, 2000: 69-71). Despite the existence of palatial sites such as Stonea Grange, and evidence of villas along the Fen edge, occupation patterns in the Fens during this period appear to have remained rural (Hall and Coles, 1994: 110).

It is unclear how much population fluctuated with Roman colonisation, or alternately, continued with little more than cultural influence. Increased flooding occurring in the 4th and 5th centuries AD, alongside the contraction of the Roman Empire, may have hastened consolidation of settlement patterns. Rippon (2000, 2009) states that a failure to maintain flood defences at the end of the Roman period, alongside rising sea levels, would have led to a reversion of wetland to its natural state and abandonment of reclaimed coastal wetlands. Knowledge of changing environmental and sociopolitical conditions during the late- and post-Roman period had led the historical geographer H.C. Darby to state confidently that "in the eighth century it [the Fens] was

essentially a 'wide wilderness', a waste untilled, devoid of settled habitation" (Darby, 1934: 188). This view among archaeologists of an abandoned and bereft Fen wilderness rediscovered by Anglo-Saxon colonisers predominated among 20th century scholars (cf. Darby, 1934; Darby, 1940; Hoskins, 1969; Phillips, 1970; Stenton, 1971; Hall and Coles, 1994: 122), abetted by heavy reliance on the accounts of ecclesiastical chroniclers of the early medieval period, such as Felix (early 8th century) and Bede (c. AD 672-735), and indices such as the Tribal Hidage. Writing in the 8th century of the 7th century Saint Guthlac of Crowland (AD 674-715), the biographer Felix described the terror of the Fens:

> There is in the midland district of Britain a most dismal fen of immense size, which begins at the banks of the river Granta not far from the camp which is called Cambridge, and stretches from the south as far north as the sea....many had attempted to dwell there, but had rejected it on account of the unknown portents of the desert and its terrors of various shapes...No settler had been able to dwell alone in this place before Guthlac the servant of Christ, on account of the phantoms of demons which haunted it (Colgrave, 1985: 87-89).

As mentioned in Chapter 1, there are many reasons why the negative accounts of the Fens by religious chroniclers should be viewed with some scepticism, not least because of discordance with archaeological evidence, and the agenda being served by such narratives (discussed at greater length in Section 4.2.3).

Early medieval sources with a non-religious purpose, such as the Tribal Hidage, have also heavily informed ideas about the population of the Fens. The Tribal Hidage is thought to have been written sometime in the late 7th century AD as a catalogue of the kingdoms and holdings of England at that time, although it is unknown how far short of comprehensive coverage it falls. It represents the earliest surviving post-Roman record of polities from that period (Oosthuizen, 2017: 53-54). The name "hidage" is derived from the measurement of land based on "hides", the non-specific amount of land required to support a household. The groups thought to populate the area of interest to the present study, the peat Fens, are thought to be the *Gyrwe*, although other groups such as the *Willa* and *Wixna* have also been suggested as occupying the area (Hart, 1971; Oosthuizen, 2017: 60). Inhabiting areas from modern southern Lincolnshire southwards to the fen edge, the *Gyrwe* are described as having north and south branches, each estimated at 600 hides (Sawyer, 1998: 47). Darby's own commentary on these estimates appears grudging as he conceded that

> Mention of the "six hundred families" serves as a reminder that although most of the peat fen must have been waste, it is unlikely to have been completely without value even at this time...But even so, it is not difficult to understand that many parts in the eighth century could still be described as a "wide wilderness", devoid of settled habitation (Darby, 1940: 7-8).

Susan Oosthuizen has in recent years offered challenges to Darby's long-standing characterisation of the Fenlands as lawless and sparsely populated. Darby largely based his conclusions on the "wilderness" accounts of early medieval religious writers, and on the apparent underpopulation he identified in the 11th century Domesday survey. Darby (1936) argued that apparent increases in the wealth and population of the Fens between the 11th and 14th centuries supported the idea that the area stood nearly unpopulated during the early medieval period. Recalculating projected population densities mapped by Darby, Oosthuizen (2014) found population densities for the 11th century approaching those of the 14th century. She suggests that Darby's calculations are likely to significantly underestimate actual populations of the 11th century, by calculating average population over habitable and uninhabitable land alike. Settlement in habitable regions around Ely, for instance, appears to have accommodated over 10 tenants per square mile (Oosthuizen, 2017: 19). By the time of the Norman Conquest, certainly, populations levels in most parts of the Fens were at least as dense as in upland areas of Cambridgeshire, south of the Fens.

In addition to undermining the idea of the Fens as a lonely region, Oosthuizen also called the idea of lawlessness and cultural discontinuity during the early Anglo-Saxon period into question, going so far as to suggest a "bottom up" approach to inferring governance from property rights (Oosthuizen, 2016b: 179). The Tribal Hidage offers evidence of many distinct polities within the Fens, although it is not believed to be comprehensive. The significance of these units of administration, often linguistically signifying a landscape-based identity, lies in the control of land resources: in the Fens, these were grazing rights. Common land rights are believed to be ancient, predating the early medieval period, but were certainly established by the Anglo-Saxon period (Oosthuizen, 2011). The establishment and perpetuation of common land rights and intercommoning between communities implies continuity of recognized tradition, as well as continued recognition of formal statute and custom. The enriching effect of seasonal inundation on pastures in the intermediate flood zone of the Fens made grazing rights central to the economic function and social fabric of the area throughout the medieval period.

As in earlier periods, grazing was not the only bounty offered by the medieval Fen. The 12th century *Liber Eliensis* described the fen environs of Ely as offering terrestrial animals such as "stags, little roe-deer, goats and hares...otter, weasels, stoats, and polecats" in addition to "innumerable eels...large wolf-fish and pike, perch, roach, burbot and lampreys...salmon and likewise the royal fish, sturgeon.." from aquatic resources, and "countless geese, fig-birds, coots, divers, cormorants, herons, and...ducks" from the marsh and sky (Fairweather, 2005: 213-214). Eels were especially plentiful in the peat fens, with Bede attributing the name of Ely to "the vast quantity of eels that are caught in the marshes" (Sherley-Price *et al.*, 1990: 239). In addition to these animal resources, which were present in the early medieval period, the Fens also offered an array of edible plants, sedge, reeds, and peat. The natural environment for the inhabitants of the early medieval Fens, and the resulting dietary resources available to children and adults alike was rich indeed. However, in providing the important dietary and economic resources of meat, dairy, wool, and leather, protection of grazing land as a resource must also have formed an important priority. While flooding at low temperatures is well-tolerated by the grass species of the Fens, summer flooding may have proved catastrophic to the health of pasture land. Rippon (2000: 176-177) finds no evidence for aggressive water management in the Fens during the early medieval period, suggesting that small drainage ditches would have managed local waters at only a low level. Oosthuizen (2017: 113-119) notes that the wealth of Fenlanders was reliant on management of water abundance as a resource, with evidence for active manipulation of water by the 10th century, although it may have occurred earlier in the Anglo-Saxon period.

4.2.3 Human adaptation of the landscape

The dynamics of engagement with the landscape are likely to reflect perceptions of, and attitudes towards, these landscapes, and nowhere is this more clearly reflected than in human-landscape interactions within wetlands. (Rippon, 2000: 1) differentiates between exploitation, modification, and transformation within wetlands to describe Roman engagement with the Fen landscape, an interpretive distinction which may also be important for the early medieval period. Huisman (2017) contrasts Roman antipathy to wetland "wastes", and their desire to reclaim and tame such spaces, with their intensive exploitation of natural resources unique to wetlands. Roman disparagement of wetlands and manipulation of the water table occurred alongside profitable harvesting of valuable

assets such as salt and peat. Such ambivalence was characteristic of Roman attitudes towards the natural and the civilized and can also be observed as a feature of later colonising discourses in the medieval and postmedieval periods.

Colonising dialectics regarding management of the Fens have consistently portrayed the Fens as untouched, squandered, and existing in a wild state, prior to external intervention. This has set up a false dichotomy between unsophisticated exploiters of Fen resources (typically the native inhabitants), and outsiders wishing to use the landscape to its full and untapped potential through modification and improvement. The assumption underlying this line of argument, that native Fenlanders merely used, and did not actively manage the landscape, has been shown to be false. Godwin (1978: 10-13) has long since recognised the ecological instability of fens, and their propensity to progress through stages of ecological succession without human intervention such as strategic grazing and clearance of aquatic overgrowth. Noting that pollen data consistently suggest the Fens as existing in a stable state of open grass meadows and wetland, with only occasional stands of isolated carr woodland, Oosthuizen (2017: 90-91) infers active human management of the landscape throughout the prehistoric and historic periods of occupation. Thus, we may discard a colonising binary of exploitation/transformation, and surmise that Rippon's middle category, modification or management, may have applied to Fenlanders over the millennia. Active management of the landscape has some obvious incentives, as protection of seasonally-flooded intermediate zones for grazing, and permanently flooded zones for aquatic plant and animal resources, would have both been crucial to protecting livelihoods and a way of life. Rather than characterising Fenlanders of the peatlands as rude and unsophisticated consumers alone, we may to some extent surmise that the people of the early medieval period understood the landscape well enough to maintain its balance, while lacking the disdain for wetlands which marked Roman, later medieval, and postmedieval writers.

4.3 People of the early medieval Fens

If a paucity of evidence forces speculation regarding the attitudes of Fenland occupants towards the landscape they occupied, the uncertainty with which we are forced to approach the identity of early medieval Fenlanders is greater still. Who the people of the early medieval Fens were, and by extension, how they saw themselves, forms an important element of one of the great debates of medieval archaeology: who were the Anglo-Saxons? Or more properly, the people of post-Roman Britain? Regarding the

Fens, the ability to answer this question is hampered by the dearth of sources from any period of history written by native Fenlanders, and the readiness of non-Fenland writers to characterise the Fens and its people unflatteringly, to suit varying agendas. However, the following section will attempt to address the people of the Fens themselves; their identity, culture, and aspects of health which may have impacted on both.

4.3.1 Insularity and identity

Historically, Fenlanders have almost universally been construed as "other," in terms of their purported insularity and suspicion of outsiders, as well as their sloth, cunning, barbarousness, and lack of proper feeling. Several researchers have remarked upon the near-absence of Fenlander literature, and the fact that accounts of the Fens and their people have almost universally been written by outsiders (Evans, 1997; Ash, 2017: 17; Huisman, 2017). As most of these etic observers have either been influenced by prior exposure to external narratives or through designs upon the landscape, these reports of Fenlanders have largely been shaped in a negative fashion by attitudes to the landscape itself. Accordingly, it is difficult to discern the character of Fenland inhabitants of *any* period with any degree of confidence. However, as Edward Storey (1985) mused,

> ...are people born and bred in the Fens all that different from people who 'enter the world' in Yorkshire, Kent, or Somerset? In many ways they are - and, of course, it works in reverse. We are conditioned by the place of our birth and our upbringing, by the historical associations, the social prejudices, the local traditions and the soil... (Storey, 1985: 13-14).

While perpetuation of the false narrative of Fenland "otherness" should be carefully avoided (Huisman, 2017), it is nonetheless indisputable that the identity of any people is a dynamic property which encompasses their history, culture, and engagement with the landscape. As previously discussed in Chapter 3, cultural context is indispensable for understanding breastfeeding behaviours. In consequence, in attempting to understand the lives of early medieval Fenland children it is necessary to use such elements as are at our disposal. In the most concise terms, this means exploring evidence for the physical and cultural identity of Fenlanders.

4.3.1.i Migration

The movement of people into or around the Fens, and the openness of inhabitants to newcomers is a recurrent theme of Fen historiography. McIntosh (2007) described an active management of local identity through the definition of social and economic boundaries separating "insider" and "outsider" membership in late medieval English

market towns, and exclusion of "undesirable" incomers. While this selective acceptance or exclusion of incomers described by McIntosh belongs to a broader English trend, it is intriguing to speculate on the origins of this insularity, and whether a similar maintenance of insularity existed among early medieval Fenlanders. Huisman (2017) rejects the notion of an insider/outsider dichotomy in Fenland identity before the drainage of the later medieval period, arguing that perceptions of difference and otherness have been assigned by external narrators, and are not reflective of all past native Fenland perceptions. Regardless, the presumed migration of the 5th and 6th centuries within eastern England, and associated cultural shifts, signify a direct challenge to the identity of people resident in the Fens at that time. The controversy over continuity or colonisation during this period represents a crucial element of the enigma of early medieval Fenland identity.

In addition to being relevant to identity and culture, movement of people has immense importance for health and disease. Migrants have been shown to be at increased risk of severe disease, through changes to diet and exposure to new pathogens (MacPherson and Gushulak, 2001). During the postmedieval period, Daniel Defoe wrote of malaria and migration to the wetlands of eastern England:

> I have one remark more before I leave this damp part of the world, and which I cannot omit on the women's account, namely, that I took notice of a strange decay of the sex here; insomuch that all along this country it was very frequent to meet with men that had had from five or six to fourteen or fifteen wives; nay, and some more...That they, being bred in the marshes themselves and seasoned to the place, did pretty well with it; but that they always went up into the hilly country, or, to speak their own language, into the uplands for a wife...when they came out of their native air into the marshes among the fogs and damps, there they presently changed their complexion, got an ague or two, and seldom held it above half a year, or a year at most; And then, said he, we go to the uplands again and fetch another; so that marrying of wives was reckoned a kind of good farm to them. It is true the fellow told this in a kind of drollery and mirth; but the fact, for all that, is certainly true... (Defoe and Rogers, 1971: 54-55).

Taking Defoe's description at face value, we may infer several things regarding marsh ague, all of which accord well with clinical evidence on *P. vivax* infections. Firstly, adult natives of marshlands are likely to have been veterans of childhood infections, gaining some acquired immunity to severe disease absent in newcomers to the area. Secondly, it is also likely that in addition to the seasonality of infection, events occurring during the first year of marriage are likely to have included a first pregnancy, which would impact on overall immunity, and potentially the immunity of future

children. Pregnant new residents of marshlands appear to have been at particular risk of severe disease, underlining the known modern risks to the non-immune, pregnant women, and the unborn. Nor can it be assumed that migration from other wetlands would have been protective, as local disease ecologies may vary. For the purposes of understanding the health of children of the 5th and 6th century, the importance of being either the biological product of centuries of adaptation and natural selection in a highly specific malarial environment, or relative newcomers to the area, cannot be overstated.

4.3.1.ii The Adventus Saxonum

The nature of the *Adventus Saxonum*, or the purported Anglo-Saxon migration of the 5th and 6th centuries, represents one of the most important and contested topics in early medieval archaeology. It would be incredibly remiss to address the identity of Fenlanders of this period without exploring the immense impact that exchange of people, material culture, and ideas may have had during this period on health and identity. The following sections will provide a summary of the salient evidence from historical, archaeological, and biomolecular sources regarding demographic change in eastern England during the 5th and 6th centuries.

Every English schoolchild is taught a story of ethnogenesis which asserts that following the contraction of the Roman Empire in the early 5th century, the Britons found themselves defenceless against marauding Scots and Picts. Failing to receive aid from Rome, the British king Vortigern invited Saxons to settle as hired mercenaries and were ultimately overtaken violently by the invaders. This fondly-told national tradition stems from the historical accounts we have for an Anglo-Saxon migration by the monks Gildas (6th c.) and Bede (8th c.), and the 9th-11th century Anglo-Saxon Chronicle. Both monastic writers were writing second-hand (and in the case of Bede, who relied heavily on Gildas, third-hand) accounts, with the purpose being moral polemic and illustration of the judgement of God (James, 2001: 95). Unlike Gildas or Bede, the Chronicle assigns a firm timeline to the Germanic incursion, dating Anglo-Saxon aggression to AD 449 (Swanton, 2000: 12). All sources had an agenda of reinforcing existing religious and political structures through a narrative of conquest and English ethnogenesis. Face value of these histories as evidence of significant migration and ethnic replacement of "peoples", as envisaged through a racialist lens, has in recent decades given way to a more critical appraisal of English ethnogenesis among

archaeologists (Hamerow, 1997; Härke, 2003; Hills, 2003; Higham, 2004; Brugmann, 2011).

Archaeologists have largely moved away from culture-historical interpretations, and towards a more dynamic model of interpretation which encompasses the possibility of continuity, as well as the probabilities of cultural exchange through movement of goods and people. Scull (1995) suggests that as migration of people and ideas is a normal feature of human behaviour throughout history, there is no reason to doubt that population mobility occurred during the 5th and 6th centuries AD. Despite continuing arguments for Anglo-Saxon colonisation (cf. Thomas *et al.*, 2006; Härke, 2011), based primarily on changes to burial practice during this period, it is not necessary to attribute these changes to migration, as funerary norms are also sensitive to changes in economy, shifts in religious belief, and regional variation (Hills, 2003: 96-97). Indeed, many published examples exist of continuous use of burial grounds between the Romano-British and Anglo-Saxon periods (Lucy 2000: 150-151; Morris, 1962; Bilikowska, 1980; Smith, 1906; Lethbridge, 1938; Fennell, 1974; Crawford, 1983), and osteological studies spanning the Romano-British to Anglo-Saxon transition have found metric and morphological consistency between periods (Lloyd-Jones, 1995; Gowland, 2002). Decentralising changes to settlement strategy and population distribution in post-Roman Britain also do not require invoking invasion and ethnic colonisation (Hamerow, 1991; Williams, 2002). Here, sociopolitical and economic shifts suffice to explain alterations to settlement pattern. Furthermore, large and well-characterised sites in the east of England, such as Mucking, Essex (Jones, 1974) and West Stow, Suffolk (West, 1985; Crabtree, 1989), have shown continuity in use from the Romano-British to early Anglo-Saxon periods. Biogeochemical and biomolecular evidence for early medieval migration has also been equivocal, failing to decisively establish clear evidence of large-scale colonisation by continental migrants (Montgomery *et al.*, 2005; Hedges, 2011; Hughes *et al.*,2018).

In terms of the specific identity of the people of the Fens, the eminent archaeologist E. Thurlow Leeds commented "...of the Gyrwas, who seem to have been closely connected with the Fens, not much light can be thrown..." (Leeds, 1913: 70). However, more recently, archaeologists using a broad array of evidence have made strong arguments for stability of population within the Fens. Following Hamerow (1997), Oosthuizen (2011; 2016a; 2017: 26) sees no compelling archaeological evidence for a

break in cultural continuity in the Fens, with no significant shift in mode of living between the Roman and sub-Roman periods. Many sites in the silt and peat Fens show signs of continuous occupation throughout the transition from Romano-British to Anglo-Saxon period, and in many cases beginning as early as the Iron Age (Hall and Coles, 1994: viii). Indeed, the hilltop site at Highfield Farm, Littleport (sampled in the present study) shows continuity of use from the Neolithic period through to the 6th century AD. Linguistic evidence from place-names also is suggestive of continuity, with nearly equal retention of Brittonic and Old English elements and no systematic patterns in geographic distribution (Oosthuizen, 2016a; Oosthuizen, 2017: 33-47). A recent whole-genome study of skeletons from fen-edge early medieval Oakington (Schiffels *et al.*, 2016) showed high levels of genetic diversity not consistent with a previously-proposed hypothesis of sexual "apartheid," limiting intermarriage by native Britons and their Anglo-Saxon colonisers (e.g. Thomas *et al.*, 2006; Härke, 2011). Furthermore, Felix's *Life of Guthlac*, written in the 8th century AD, references Guthlac's encounter of Brittonic speakers in the Fens (Colgrave, 1985: 109), demonstrating the persistence of British language and culture in the early medieval Fens. The archaeological record strongly suggests that it is likely that, small-scale mobility aside, the people who occupied the Fenlands during the prehistoric and Roman periods continued to inhabit the area into the early medieval period and beyond, with significant influence from Germanic language and material culture. Archaeological evidence of potentially lengthy residence is further complemented by skeletal evidence for stress markers, showing a well-adapted population. Regarding the early medieval people of the Fens, the current weight of archaeological and scientific evidence appears to indicate a strong likelihood that the Fenlanders combined continuity of tradition with dynamic adaptation to incoming ideas, material culture, and people, as they had for many centuries prior.

4.3.2 Health and disease in the Fens

The historically-recorded social and economic changes to 5th and 6th century Britain must be assumed to have had impacts on the health of the people of Britain. However, there are also notably specific concerns for health in the historic Fens which should be addressed, and this section will address these risks to health below.

4.3.2.i Malaria

The most notorious of the risks to health in the Fens in times past was the dreaded "Fen ague", "marsh fever", "intermittent fever", or "tertian/quartan fever", as malaria was

variously known in England during the historic period. So feared was this disease that Dobson (1997: 295) records the frequency with which British vicars of the 18th century declined to reside in their marshland parishes, citing the unhealthful conditions. Understanding the historical pattern of endemic British malaria is fraught with difficulties. The scientific discoveries which lead to positive identification of the malarial parasite did not occur until the late 19th century, coinciding with the completion of drainage in the Fens and the end of endemicity. Additionally, terms such as "ague" were relatively nonspecific, also being used to describe other diseases historically prevalent in the Fens, such as typhus (Russell, 1955: 23; Howe, 1972: 91; Nicholls, 2000). Regrettably, until methods to positively identify malarial infection in skeletal material are developed, the only recourse is inference from accounts predating modern medical understanding. Nonetheless, the importance of understanding the impacts of malaria for population, and particularly childhood, health cannot be overstated.

Malaria is a mosquito-borne disease caused by infection with eukaryotic protozoan parasites of the genus *Plasmodium*. Over 200 species of *Plasmodia* are known; with 53 of these affecting mammals, and 30 specifically afflicting primates (Luo *et al.*, 2015). Four species have historically infected humans: *P. falciparum*, *P. malariae*, *P. ovale*, and *P. vivax* (Table 4.1). These are typically described by the periodicity of febrile attacks (paroxysms) as either "tertian" (*P. falciparum*, *P. vivax*, *P. ovale*), occurring every 48 hours, or "quartan" (*P. malariae*), occurring every 72 hours (Antinori *et al.*, 2012). Malaria is one of the most devastating of infectious diseases in the modern world, causing around 200 million infections per year and around 500,000 deaths (Piperaki and Daikos, 2016). The species also occupy differing ecological niches. Tertian *P. falciparum* accounts for most deaths worldwide – primarily among pregnant women and young children – and is geographically concentrated within the equatorial tropics and subtropical regions, particularly within sub-Saharan Africa (Snow *et al.*, 2005). This non-relapsing species has also been found frequently within the Mediterranean region of southern Europe but requires higher temperatures for completion of its reproductive cycle than are routinely found in northern Europe (Snow *et al.*, 2005; Gething *et al.*, 2011). Consequently, *P. falciparum* has never been able to establish endemicity in Britain. *P. ovale*, which is a relapsing form of tertian malaria, also is confined to high-temperature regions, being found almost exclusively within sub-Saharan Africa and islands of the western Pacific (Collins and Jeffery, 2005). Only

two species have ever been documented to have reached northern Europe: *P. vivax*, which has the broadest latitude distribution range of any species, and *P. malariae* (Piperaki and Daikos, 2016). These two species are easily distinguishable, however, due to the quartan periodicity of *P. malariae* and its non-relapsing form.

Species	**Periodicity**	**Habitat Range**	**Relapsing**
Plasmodium falciparum	Tertian	Tropical to Mediterranean	No
Plasmodium vivax	Tertian	Tropical to Temperate	Yes
Plasmodium ovale	Tertian	Tropical	Yes
Plasmodium malariae	Quartan	Tropical to Temperate	No

Table 4.1 Overview of human *Plasmodium* species.

Transmission of malaria is exclusive to female mosquitoes of the genus *Anopheles*. However, not all anopheline mosquitoes are capable of acting as vectors, and, of those that are, not all are equally adapted to efficient transmission of every species of malaria (Neafsey *et al.*, 2015). Five potential vector species are present in Britain: *Anopheles claviger*, *A. atroparvus*, *A. messeae*, *A. algeriensis*, and *A. plumbeus* (Lindsay and Thomas, 2001), each with its own distinct ecological niche. In terms of the historic overlap in distribution of British malaria and anopheline habitat, *A. atroparvus* (often conflated with *A. messeae* within the *A. maculipennis* complex) is widely acknowledged as the most important past and present English vector species (Nuttall *et al.*, 1901; Vaile and Miles, 1980; Snow, 1998; Lindsay and Thomas, 2001). Although *A. atroparvus* is capable of breeding in fresh water, this zoophilic (prefers livestock but will bite humans) species shows a distinct preference for breeding within the brackish waters of coastal marshes and wetlands (Kuhn *et al.*, 2003). Due to the brackish conditions and heavy reliance on pastoral agriculture in the Fens prior to drainage, it should be assumed that *A. atroparvus* would have had a still greater competitive advantage over other species than in the present. *A. atroparvus* has been shown to be a particularly poor vector for both *P. malariae* and *P. falciparum*, while *P. vivax* is transmitted readily (Shute and Maryon, 1951; Snow, 1998; Sallares, 2002: 156). Consequently, we can conclude with some confidence that these species of malaria were unlikely to be responsible for reports of intermittent epidemic ague in the Fens occurring alongside the commonplace relapsing tertian variety. Accordingly, the following sub-sections will review modern clinical data relevant to the likely impacts of *P. vivax* on early medieval Fenlanders, and historical evidence for the contours of malaria in Britain.

Clinical pathophysiology of P. vivax

The infection cycle for *P. vivax* (see Figure 4.2) requires incubation and development in both human and mosquito hosts. The cycle is initiated in humans as *sporozoites* are injected by the bite of an infected *anopheline* mosquito. This first infectious stage of the malarial parasite migrates through the bloodstream to the liver within minutes, where they invade hepatocytes and progress to either a dormant *hypnozoite* stage or an active and asexually-reproductive *schizont* (Mueller et al., 2009). Hypnozoites are exclusive to relapsing forms of malaria such as *P. vivax*, and their activation weeks, months, or years after initial infection is thought to be triggered by systemic stress or salivary proteins introduced by further *anopheline* bites (Hulden and Hulden, 2011). After six to sixteen days, mature schizonts rupture from their hepatocyte incubators into the bloodstream as *merozoites* (Mueller *et al.*, 2009).

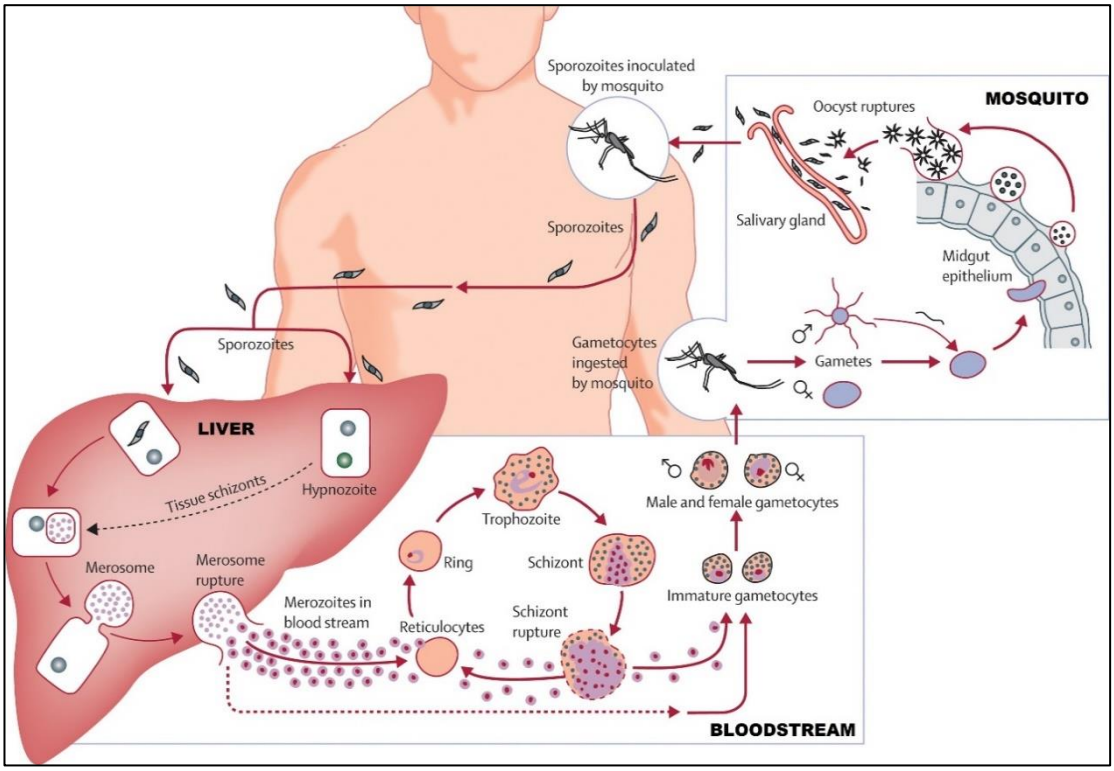

Figure 4.2 Lifecycle of *P. vivax* in humans (adapted from Mueller *et al.*, 2009: 556).

P. vivax merozoites represent the second blood stage, and these predominantly invade immature red blood cells (erythrocytes) called *reticulocytes*. Within these erythrocytes, merozoites either grow into ring-shaped *trophozoite* schizonts which reproduce further, or differentiate into *gametocytes*, the sexual form of *Plasmodium* parasites. Completion of maturation leads to rupture of the erythrocyte, releasing schizonts or gametocytes into the bloodstream. Febrile periodicity in untreated malaria

is tied to this emergence, and is species-dependent (Antinori *et al.*, 2012). Schizonts may go on to invade further reticulocytes and perpetuate the blood cycle, while gametocytes are ingested by *anopheline* mosquitoes feeding on infected individual. Gametocytes reproduce sexually in the midgut epithelium of the infected mosquito before migrating to its salivary gland as sporozoites, thus completing the infection cycle.

The high mortality rates associated with *P. falciparum* have historically biased research priorities and resources in its favour (Mendis *et al.*, 2001; Price *et al.*, 2007; Carlton *et al.*, 2011). In consequence, far less is known about the physiological impacts of *P. vivax*, which has traditionally been described as benign. Nevertheless, recent decades have seen the emergence of data suggesting that the severity of *P. vivax* impacts on health have been grossly underestimated (Galinski and Barnwell, 2008; Baird, 2009; Singh *et al.*, 2011; Baird, 2013; Val *et al.*, 2017). The hypnozoite life stage of *P. vivax* enables cycles of dormancy and relapse, allowing the parasite to create physiological impacts which far exceed those expected for low *parasitaemia* (parasite load). *P. vivax* is increasingly recognised by researchers as having severe consequences for health and survival, including multi-organ failure (Price *et al.*, 2007; Rogerson and Carter, 2008; Baird, 2009; Carlton *et al.*, 2011; Singh *et al.*, 2011). The paroxysms of *P. vivax* produce temperatures of up to 41°C, and typically follow the onset of severe chills and rigor (Karunaweera *et al.*, 1992; Karunaweera *et al.*, 2003). While these fevers may be high, and create significant distress, they are comparatively benign in relation to the potential complications of severe *P. vivax* infection.

Some manifestations of malaria, like the febrile reaction to blood-stage infection, result from immune response. *P. vivax* infection typically provokes greater inflammatory responses from the immune system than *P. falciparum*, and these may lead to pulmonary oedema, lung injury, or life-threatening acute respiratory distress syndrome (Baird, 2007; Andrade *et al.*, 2010; Sarkar *et al.*, 2010). Inflammatory response is also implicated in the development of cerebral malaria, which can lead to impaired consciousness, seizures, coma and death in children and adults (Dunst *et al.*, 2017). Other problems may arise from non-inflammatory immune responses. The spleen plays a pivotal role in the body's response to malarial infection, due to its blood-filtering and lymphatic functions. Splenomegaly (enlargement of the spleen) can occur in response to the higher workload created by the haemolytic anaemia and parasitic

invasion of *P. vivax*, with expansion of the red and white splenic pulp responsible for producing immune factors such as macrophages and B cells (Machado Siqueira *et al.*, 2012). The spleen, being a small organ of the upper abdomen, is not normally palpable on examination but becomes so during splenomegaly. Expansion and distension of the spleen is considered one of the most common signs of *P. vivax* infection and increases the risk of spontaneous rupture under mechanical stress (Kim *et al.*, 2015), which would have proved deadly before the advent of modern medicine. It is therefore not surprising that splenomegaly was commonly recorded for endemic English malaria, to the extent of being referred to by the colloquial term "ague cake" (Kuhn *et al.*, 2003: 9997).

Further complications of severe *P. vivax* malaria may result from the damage directly wrought by the presence of the *Plasmodium* parasite. Merozoite invasion of reticulocytes and resulting *haemolysis* (destruction of erythrocytes), in addition to immunological destruction of healthy erythrocytes, may lead to several negative outcomes. Haemolytic anaemia, the most common of these, is a significant contributor to morbidity and mortality among sufferers of *P. vivax*, particularly in young children (Price *et al.*, 2007; Mueller *et al.*, 2009). Thrombocytopenia (low blood platelet count) is also very common, and may result directly from coagulation with haemolysed erythrocytes, or from immune destruction (among other causes not well understood at present) (Kochar *et al.*, 2005). Coagulation of platelets with haemolysed erythrocytes can lead to thrombotic microangiopathy (damage to the endothelium of small blood vessels via clotting), especially in organs involved in the filtration of blood. Organ dysfunction or failure, particularly of the kidneys or liver, are noted as possible sequalae in *P. vivax*, and are life-threatening even in modern medical environments providing supportive procedures and drug therapy (Baird, 2007; Kochar *et al.*, 2009).

While severe disease is not common in the present, it is increasing in prevalence, due to emerging pathogen resistance to front-line drug treatments, and revealing a high potential for fatality (Rogerson and Carter, 2008; Tjitra *et al.*, 2008; Price *et al.*, 2009). Severe disease is most common in vulnerable modern populations, especially pregnant women and children, and it is here for the past that the highest probability of severe complications should be assumed. The relapsing nature of *P. vivax* infection allows for the emergence of acquired immunity and tolerance among survivors. However, this is rarely attained before the age of five years, and pregnancy-related immunosuppression may reduce the immune response of expectant mothers to infection, leading to greater

probability of severe or fatal complications in these groups. (Nosten *et al.*, 2004; Rogerson and Boeuf, 2007; Mueller *et al.*, 2009). Furthermore, pregnant women emit more heat and carbon dioxide than non-pregnant women, making them more attractive to the mosquito vector, and more likely to be bitten and infected (Lindsay *et al.*, 2000).

Micronutrient and macronutrient malnutrition are also substantial concerns for pregnant women and young children. The risk of developing moderate or severe anaemia, in particular, is much higher in these groups, due to the effects of haemolysis on iron status, and more poorly-regulated immune response (Douglas *et al.*, 2012; Castro-Gomes *et al.*, 2014). Impaired growth may also result from maternal or childhood malarial infection, beginning *in utero*. While *P. vivax* lacks the placental cytoadherence of *P. falciparum*, the greater activity of pro-inflammatory cytokines can restrict blood flow through the placenta to the developing foetus, leading to intrauterine growth restriction (IUGR) and stillbirth or low birthweight as common outcomes of *P. vivax* infection in pregnancy (Nosten *et al.*, 1999; Whitty *et al.*, 2005; Poespoprodjo *et al.*, 2008; Umbers *et al.*, 2011). In addition to increased risk of prenatal or perinatal mortality, surviving infants born with IUGR may exhibit impaired immune function during the postnatal period and over the longer term, leading to heightened vulnerability to illness (McDade *et al.*, 2001; Cromi *et al.*, 2009; Longo *et al.*, 2014). Nor does the risk of malnutrition end with birth. *P. vivax* infection is strongly associated with acute malnutrition and impaired growth in young children, even exceeding the deleterious effects of acute episodic diarrhoea on growth (Williams *et al.*, 1997; Hutchinson and Lindsay, 2006; Poespoprodjo *et al.*, 2009; Lee *et al.*, 2012). While the relationship between undernutrition and *P. vivax* infection is complex, and almost certainly bidirectional (Caulfield *et al.*, 2004; Poespoprodjo *et al.*, 2009), the parasite is known to stimulate high production of TNF-α, a pro-inflammatory cytokine, leading to anorexia and cachexia, and lending plausibility to the causal role of malaria in the development of childhood malnutrition (Williams *et al.*, 1997; Monteiro *et al.*, 2016).

Effective antimalarial treatments have existed since the 17th century, when the merits of quinine for malaria were first experienced via tinctures of Peruvian cinchona bark, variously termed “Jesuit’s bark”, “Peruvian bark”, or “cinchona bark” (Mac Arthur, 1951; Reiter, 2000). *P. vivax* proved particularly recalcitrant to cure, relative to acute species of *Plasmodium*, due to survival of its hypnozoite stage, and thus was subject to relapse even after treatment. Modern drugs such as chloroquine have been more able to

target and eliminate the hypnozoite stage, but the malarial parasite has in some areas begun to evolve resistance to these treatments also, producing disease of greater severity (Baird, 2004; Price *et al.*, 2009). Thus, the relationship between emerging treatment failure and serious complications of *P. vivax* in the present offers strong indications that severe disease would have been more prevalent in past untreated populations.

Historical evidence for malaria in Britain

The date of malaria's arrival on British shores is the topic of some disagreement among scholars, as conclusive proof of the earliest infections is lacking. Some scholars believe that malaria is likely to have entered Britain with Roman immigration during the 1st to 5th centuries AD, especially with the soldiers of the Roman army (Boyd, 1949; Sallares, 2002: 156; Sallares, 2006; Gowland and Western, 2012). Sallares (2002: 156) cites Cassius Dio's (AD 155-235) description of the suffering of the troops of Septimius Severus in Scotland's marshes, as suggestive of malaria being present during this period. It is indisputable that malaria was a familiar malady to the Romans, and present within the Pontine Marshes as well as throughout other areas of the Empire. Thus, it is likely that infected individuals arrived at some point during the Roman occupation, due to the high levels of migration to Britain.

If the role the Romans may have played in the coming of malaria to Britain is unclear, writings from the early medieval period present a stronger case for it being present by at least that time. Several researchers have commented upon the apparent familiarity of early medieval writers with an annually-recurring chronic fever cycle which they termed *lencten adl*, or "spring ailment" (Mac Arthur, 1951; Howe, 1972: 90-91; Cameron, 1993: 10). The reference to a spring seasonality is characteristic of the bimodal seasonal patterns of English *P. vivax*, in which primary infections originating in the summer or autumn were known to secondarily relapse in the early springtime (Dobson, 1980; Hutchinson and Lindsay, 2006). The term *lencten adl* was notably selected by an Anglo-Saxon translator (traditionally attributed to King Alfred) of Bede's Latin *Ecclesiastical History*, in which the miraculous healing of a long-standing recurrent febrile illness is described using the more nonspecific *febris*, or fever (Mac Arthur, 1951: 91; Howe, 1972). *Lencten adl* is also specifically addressed in *Bald's Leechbook*, in which it was advised "...for a tertian fever, let the sick drink in warm water ten sups of betony, when the fever is approaching...." (Cockayne, 1865: 135). Cameron (1993: 11) notes that malarial remedies commonly contained betony, a grassy

herb, and that these potions are as unlikely to have been effective as the magical remedies that were used alongside them. Indirect evidence for sequelae such as malarial anaemia is also present in the Anglo-Saxon period. In Felix's *Life of Saint Guthlac* the "demon" inhabitants of Crowland are described as being "...ferocious in appearance, terrible in shape with great heads, long necks, thin faces, *yellow complexions*..." (Colgrave, 1985: 103, emphasis added). This is the earliest example of a characterisation of yellow-skinned Fenlanders which remained consistent over a millennium. From this juncture through to the early modern period, Fenlanders were described as pale or sallow, as in the poem by an anonymous 19th century Fenman:

The moory soil, the watry atmosphere
With damp, unhealthy moisture chills the air.
Thick, stinking fogs, and noxious vapours fall,
Agues and coughs are epidemicall,
Hence every face presented to our view
Looks of a pallid or a sallow hue (Darby, 1940: 117).

These depictions of pale or yellowed skin suggest jaundice, a common manifestation of haemolysis in *P. vivax* infection (Rodriguez-Morales *et al.*, 2006), and provide a compelling indication of the consistent presence of malaria in the Fens from at least the early medieval period.

Climate change almost certainly played a decisive role in the undulating impact of Fenland malaria, though in ways which are currently unclear. The wetter and cooler conditions of the early post-Roman period would seem likely to facilitate breeding conditions for the vector. However, *A. atroparvus* prefers slow-moving, sunlit water with high algal content (Medlock and Vaux, 2011), and a rise in water levels may have led to more rapid currents in rivers, as well as allowing for an expansion of habitat for piscine predators of mosquito larvae. As discussed earlier in this chapter, it is also believed that population levels in the Fens were reasonably high, and reliance on a pastoral economy – providing the preferred blood source for *A. atroparvus* – was heavy. This mixed evidence for the ecological conditions supporting malarial proliferation during the early Anglo-Saxon period makes it difficult to confidently predict the impacts of the disease during this time. We simply do not have enough information regarding environmental or epidemiological variables to confidently assess relative impact over centuries of change, at present.

The malarial disease ecology of the Fens in some cases may have shielded its inhabitants from the full impacts of catastrophic events, such as epidemic disease – despite immune dysregulation resulting from malarial infection. The Black Death, which followed the famines of the early 14th century, is believed to have impacted relatively lightly upon mortality in Fenland populations, particularly in districts known to have been hit hard historically by malaria, such as Ramsey, Huntingdonshire (Darby, 1940; DeWindt and DeWindt, 2006: 44). Researchers have noted the protective effects of iron deficiency against plague mortality, and some have suggested that this may somewhat explain lower death rates among potentially deficient subpopulations (Wake *et al.*, 1974; Ell, 1984; Denic and Agarwal, 2007). *Yersinia pestis*, the causative organism of plague, requires iron to replicate and grow (Ell, 1984). Low plague mortality among Fenlanders may therefore indicate pervasive chronic anaemia stemming from malarial haemolysis and provide some justification for inferring the presence of endemic malaria in the Fens at this time. However, in our understanding of the interplay of environment, disease, and human behaviour even some geographic nuance must be applied. In contrast to the low number of deaths documented at Ramsey, Darby (1940: 152) asserts that mortality from the Black Death at Spalding in 1349 was heavy enough to disrupt maintenance of the drainage ditches which kept the waters at bay. It is plausible that the different ecologies in the silt Fens (Spalding) and the liminal brackish zone between the silt and peat Fens (Ramsey) produced differing malarial impacts, and therefore disparities in the outcome of interaction between comorbidities which may have been present in the early medieval period also.

Evidence from the postmedieval period may offer some clues as to the likely impacts of malaria on children. Early modern Fen children had a shockingly elevated risk of death, with total life expectancy in the Fenland village of Wrangle, Lincolnshire averaging a meagre 14 years, compared to the national average of 35 years (Clarkson, 1975: 50). West (1973) estimated that an astounding 25% of children died before their first birthday in the east Lincolnshire villages of Leake and Wrangle during the 16th to 19th centuries. Defoe expressed great pleasure at leaving the Fens on his tour of Britain, saying "...but for the healthiness or pleasantness of it [the town of Spalding, Lincolnshire], I have no more to say than this, that I was very glad when I got out of it, and out of the rest of the Fen country, for 'tis a horrid air for a stranger to breathe in" (Defoe and Rogers, 1971: 415). Despite arguments by some historians aligning with the

"benign" view of vivax, English malaria was certainly greatly feared by visitors and seems to have exerted a deadly influence over vulnerable populations.

Figure 4.3 Depiction of *Ague and Fever* by T. Rowlandson, London, 1788, showing embodiments of the alternating hot and cold phases of malaria (Credit: Wellcome Collection Images https://wellcomecollection.org/works/g4wfh4bq).

Dobson (1997) argued for the importance of integrating malaria as a central shaper of English wetland health and practices, saying that:

> Malaria gave rise to a peculiar demographic and social structure in the marshlands...It is difficult to separate out the chains of cause and effect or give any precision to the interdependent roles of intercurrent infections, biological immunity, nutrition, low energy levels, alcohol, narcotic substances, domestic dwellings, poor sanitation, poverty, infant care and feeding practices... (Dobson, 1997: 342-343).

She is echoed more cautiously by Sneddon (2006) who called for a nuanced interpretative model for infant mortality which integrates but decentralises malaria:

> Whilst conceding that fenland ague was, indeed, in part, responsible for the 'chronic state of ill-health' found in this region, the mechanisms producing high levels of infant mortality in the Victorian period were inevitably varied and multifaceted, and were, in fact, even in this seemingly homogenous rural-agricultural area, the result of a complex interaction of various physical, socio-economic, demographic and cultural factors (Sneddon, 2006: 94).

Biomolecular approaches to finding P. vivax

A complex interpretative model is certainly needed in order to understand the impacts of early medieval Fen malaria, and this requires better evidence for presence and prevalence. Considering the paucity of sources deriving directly from the early medieval period for the prevalence and impact of malarial infection, more direct bioarchaeological methods may offer a way forward. At present, no successful method has emerged for the detection of *P. vivax* in archaeological remains; and despite attempts to correlate malarial infection with osteological markers, malaria does not create pathognomonic lesions (Gowland and Western, 2012; Smith-Guzmán, 2015). Ancient DNA methods present great difficulties, as detection of *vivax* would require active blood-stage infection at the time of death and would fail to detect latent hypnozoite infection in the absence of hepatic tissue. Furthermore, the low parasitaemia typical of *vivax* infections offers further difficulties and requires high sensitivity. To date, studies attempting to detect all species of malaria from have achieved limited results and no successful amplifications of *P. vivax* (e.g. Sallares and Gomzi, 2001; Nerlich *et al.*, 2008; Pinello, 2008; Marciniak *et al.*, 2016). Kendall (2014; 2016) tried a different approach, seeking to detect antibodies in human bone, which might lead to the detection of malaria-specific antibodies. Again, without success, due to diagenetic factors. Other immunological studies have claimed to have identified malaria in archaeological bone via immunological dipstick or rapid tests (e.g. Miller *et al.*, 1994; Rabino Massa *et al.*, 2000; Fornaciari *et al.*, 2010), but these have been critiqued on methodological grounds due to a lack of control for diagenetic cross-reactivity (Sallares and Gomzi, 2001; Kendall, 2014).

Methods which target by-products of malarial infection, rather than antibodies or the parasite itself, may achieve better results in future. Hemozoin, an insoluble biocrystal produced by parasitic consumption of haem, is deposited in bone marrow during malarial infection and progressively accumulates with successive episodes. Hemozoin crystals, having characteristic size and shape which is specific to the species of parasite, have been described as being a promising future target in archaeological research (Setzer, 2014; Bianucci *et al.*, 2015), and have been successfully used in multi-method palaeopathological diagnosis in recent years (Inwood, 2013; Cox, 2018). Development and application of objective direct means of identifying malarial infection in

archaeological human remains will enable advances from our current understanding of malaria in England with incalculable value.

4.3.2.ii Waterborne and enteroparasitic diseases

In addition to the risk of endemic malaria, waterborne and enteroparasitic diseases are ever-present concerns of humans living in wetlands and would also have been serious issues for the health of medieval Fenland children. Some of these pathogenic organisms are naturally present in the environment, while others are zoonotic or derived from human reservoirs. Several factors make waterborne and enteric diseases worth consideration for the health of Fen children. Firstly, wet conditions and a high water table would have made the maintenance of a safe water supply, uncontaminated by faecal inputs, nearly impossible. Water, drawn from a well of the depth possible with early medieval technology, would be subject to lateral contamination from toilet deposits, and may have simply been drawn from the more obvious open sources of water (Hutchinson and Lindsay, 2006). Secondly, the emphasis on pastoralism would have provided ample sources of zoonotic pathogens to the water catchment, as deposition of animal faeces in the path of runoff can have deleterious impacts on wetland water quality (Collins, 2004; Haley *et al.*, 2009), as well as through direct contact.

Enteric infections would perhaps have represented the greatest non-malarial threat to children's health, as they do in modern developing nations. Food and water sources contaminated with human and animal faecal bacteria would have resulted in seasonal and chronic diarrhoea in some cases, having impacts on long-term growth and wellness, while other infections would have produced intermittent epidemic disease. Cholera, a gastrointestinal illness caused by ingestion of the bacterium *Vibrio cholerae*, is among the pathogens naturally found in the environment, and is indigenous to marine and brackish waters (Stewart *et al.*, 2008). Greatly feared in the Victorian period, the high epidemic mortality of cholera was found to be most severe in districts of low-lying elevation (Farr, 1852; Gilbert, 1958). A watery and brackish marsh district, totally lacking in modern water management mechanisms, such as the medieval Fenlands, would certainly have encountered outbreaks of cholera periodically, with unknown levels of fatality. Similarly, food and waterborne typhoid fever (caused by the bacterium *Salmonella typhi*), was the cause of frequent epidemics within the marshlands. Unlike cholera, diarrhoea and vomiting in typhoid are uncommon, instead producing typical

symptoms of lethargy, fever, headaches, constipation, abdominal pain, and an accompanying speckled rose-coloured rash. Mortality from typhoid fever was greatly increased by comorbidity with pre-existing malarial infection, making it particularly deadly among Fenlanders (Dobson, 1980; Nicholls, 2000).

Enteroparasitic infections are ubiquitous in past populations (Gonçalves *et al.*, 2003), with variable chronic impacts on health. Most infections would have been debilitating, rather than deadly, leading to episodic poor health and chronic nutritional deficiencies. Cameron (1993: 10) identifies sheep liver fluke (*Fasciola hepatica*) infection as a particular hazard for pastoral communities during the Anglo-Saxon period, identifying the symptoms of infection in the description of symptoms in *Bald's Leechbook*: cirrhosis, diarrhoea, vomiting, and abdominal distension. Helminth infections would also have been an issue in a rural, pre-industrial environment such as the early medieval Fens, and may frequently result in micronutrient deficiencies. Infections by fish tapeworm (*Diphyllobothrium latum*), contracted via the consumption of undercooked or raw fish, have been noted as resulting in a deficiency of vitamin B12 and megaloblastic anaemia in British individuals (Harland *et al.*, 1950). Considering the likely contribution of fish to the Fenland diet, *D. latum* infection warrants some consideration; not least as a possible contributor to high cribra orbitalia prevalence in the Fens. Other soil-borne helminthic parasites are expressive of poor sanitation, such as roundworm (*Ascaris lumbricoides*) and whipworm (*Trichuris trichiura*) and can cause gastrointestinal symptoms and anaemias through occult blood loss, especially in children (Gonçalves *et al.*, 2003). Highly persistent in the environment, both *A. lumbricoides* and *T. trichiura* are highly contagious, and are frequently found in palaeoparasitological samples (Burden *et al.*, 1976; Gonçalves *et al.*, 2003; Mitchell *et al.*, 2013). Perhaps counterintuitively, *Ascaris* infection has been shown to exert a protective effect against severe disease in malarial infection through induction of an anti-inflammatory response (Nacher, 2011). Comorbidities of waterborne, enteroparasitic, and malarial disease thus would have formed a complex network of interactive relationships in the health of Fenland children of the pre-drainage past.

4.3.2.iii Chronic zoonotic bacterial infections

A final major factor in Fenland health relevant to the disease ecology of children living during the early medieval period is the proximity to zoonotic vectors in a pastoral society, primarily cattle. Chief in importance among the possible chronic bacterial

infections are brucellosis (caused by bacteria of the genus *Brucella*), and tuberculosis (caused either by *Mycobacterium tuberculosis* or *Mycobacterium bovis*, with *M. bovis* being transmissible from cattle to humans). There is significant overlap between the clinical course of the two infections, with each causing relapsing fever, anorexia, chills, lethargy, joint pain, and weight loss (Buzgan *et al.*, 2010; Rubach *et al.*, 2013). Both also cause osteological involvement (primarily the joints of the body, including the spine) in a minority of cases, causing erosive changes to joints, and requiring differential diagnosis (Cordero and Sanchez, 1991; Roberts and Cox, 2003: 230; Mays, 2007). And finally, both are contracted through exposure to animals, their faeces, or through the consumption of infected meat or milk.

However, there are substantial differences between brucellosis and tuberculosis. *Brucella* infection is rarely fatal (Rubach *et al.*, 2013), but due to weight loss and loss of appetite is likely to impact on child growth. Untreated clinical tuberculosis, either of the human species *M. tuberculosis* or the zoonotic *M. bovis* (clinically and palaeopathologically indistinguishable), is more frequently fatal, with higher rates of mortality observed for malnourished populations (Roberts and Buikstra, 2003: 10; Cegielski and McMurray, 2004). In addition to the gastrointestinal route shared with brucellosis, tuberculosis may also be spread through the respiratory route by humans or cattle, causing pulmonary disease. Malaria and tuberculosis may also have a complex relationship. Comorbidity of malaria and tuberculosis may have substantial impacts on their prognoses. Malarial infection, whether pre-existing or acquired, has been shown to decrease humoral and cellular immune responses to *M. tuberculosis* infection and exacerbate chronic tuberculosis infection (Li and Zhou, 2013). Respiratory pathologies associated with *P. vivax* infection, and the dysregulatory effects of *hemozoin* (an insoluble biocrystal produced by *Plasmodium* metabolism of haemoglobin) on macrophage activity, may further impair immune response to tuberculosis infection (Faure, 2014).

Evidence for the presence of brucellosis and tuberculosis in early medieval British populations is variable. Biomolecular evidence for brucellosis in Britain is entirely lacking, and osteological evidence is considered questionable by some researchers (Mays, 2007). By contrast, tuberculosis is much better characterised in British archaeology. Anglo-Saxon leechbooks prescribe lichen as a remedy for *lungen adl* (lung ailment), almost certainly pulmonary tuberculosis (Cameron, 1993: 124). However,

respiratory, febrile, and cachectic complications of *P. vivax* infection strongly resemble the pathophysiology of pulmonary tuberculosis, and correct identification of the causative factor probably would have been beyond the abilities of Anglo-Saxon medicine. Bioarchaeological evidence is less equivocal. Comprehensive multiperiod surveys of British palaeopathology have found an increase in pathology consistent with tuberculosis between the Roman and early medieval period, with evidence of disease clustered in the east and south of England (Roberts and Buikstra, 2003: 132-139; Roberts and Cox, 2003: 230). Declining urbanism and a move to a more agriculturally-centred economy may well have contributed to this trend, which applies in no small measure to the rural livestock-based economy of the Fens. Consequently, zoonotic infections such as tuberculosis and brucellosis should be considered as weighty concerns for the health and survival of Fenland children during this period.

4.4 Children in the early Anglo-Saxon period

As previously mentioned in Section 2.6.1.ii, interest in infant palaeodietary trends emerged in the late 1980s alongside the birth of childhood archaeology as a discipline. From those early beginnings, recent decades have seen a proliferation of research focusing on social identity and age, with a specific emphasis on childhood and children as a demographic with qualitatively different experiences and concerns, relative to the adult population. These developments have been especially marked in Anglo-Saxon archaeology, where the social status of children, as expressed through differential funerary treatment, has produced considerable debate and discussion among early medieval scholars (Crawford, 1993; Lucy, 1994; Crawford, 2000; Gowland, 2006; Craig-Atkins, 2014; Squires, 2014).

Due to the overall lack of documentary evidence for the early Anglo-Saxon period (Yorke, 1993), little is known about the intimate details and experience of childhood during the early medieval period prior to the presumed reintroduction of Christianity to Britain at the close of the 6th century and early 7th. It is unclear whether the conceptualisation of childhood resembled the views and practices of the preceding Romano-British period, those of the middle and later Anglo-Saxon period which followed, or neither. Consequently, what is known about the social status and care of early Anglo-Saxon children must be largely inferred from earlier or later texts, or from archaeological evidence. These will be reviewed briefly in the sections which follow.

4.4.1 Social status

The social status of children in Anglo-Saxon England is perhaps the most difficult of the problems of the early medieval periods to parse, as no contemporary written sources exist. Historians following the arguments of Ariès (1965) have assumed that children in pre-modern periods were viewed as miniature adults, with childhood as a developmentally-distinct period arising only from the modern period. However, Kuefler (1991) has pointed out that both Anglo-Saxon law code and hagiography persistently treated children as qualitatively different in nature and understanding to their adult counterparts, and in need of protection. Crawford (1999: 169) also cites the commentary of the apparently much affected King Alfred, asking "what sight is more intolerable than the death of a child before its father's eyes?", as evidence that children were highly valued and evoked distinct compassionate and protective emotions in Anglo-Saxon society. It is unlikely that children, bonded to their parents by the practices of daily care, held a low value in society, especially when the marked value for ties of social and blood kinship in Anglo-Saxon society is considered (Lancaster, 1958b; Lancaster, 1958a).

Other archaeologists have tried to interpret the provision of grave goods as meaningfully expressive of social status. Some have pointed out the apparent reflection of age thresholds in the provision of mortuary furnishings, and that infants and young children are rarely accorded highly valuable grave goods on a par with adults, and often appear as accessory burials in the multiple burial rite (Crawford, 1999; Crawford, 2000; Crawford, 2007). This could be considered evidence of lower status, certainly. Crawford (2000) also has suggested that toys, perhaps the most obvious of choices for grave goods, may be apparently absent because purpose-made toys were rare, or because these are ephemeral in the burial environment. This interpretation has been countered with the suggestion that grave goods may represent achieved, rather than ascribed status. This is somewhat supported by evidence for changes to provisioning of grave goods at set age thresholds around 2-3 years (theorised to represent weaning), and again at 10-12 (representing the age of majority and onset of gendered treatment) (Crawford, 1999; Stoodley, 2000; Crawford, 2007). However, the provision of very rich grave goods to singly-buried infants too young to have achieved status somewhat undermines this interpretation and reveals grave goods to be a complex expression of social value that is, as yet, poorly understood (Stoodley, 2000). Crawford (2000) has

suggested that in the case of children from wealthy families, different rules of funerary rite may have applied, overriding age-related considerations. The context-specific ambiguity of meaning attached to funerary practices during the early Anglo-Saxon period creates major challenges to interpretation. Consequently, due to the limited scope for addressing the complexity of funerary practices and grave goods, these have been excluded from the present study. What is important here is the recognition that children were treated differently in death than their adult counterparts and are likely to have also been perceived as qualitatively different in life.

Complicating interpretation further is the much-emphasised critique that archaeologists approaching interpreting the care of "children" make significant culturally-mediated assumptions regarding categories of identity, biological, and chronological age (Lucy, 1994; Lillehammer, 2000; Sofaer Derevenski, 2000; Kamp, 2001; Gowland, 2006; Sofaer, 2006; Baxter, 2008). A modern understanding of the parameters and contours of childhood, and children's identity, is unlikely to match an early medieval mindset, creating an interpretive morass for the 21st century archaeologist. Reconciling the existence of childhood as a valid social construct, as well as a biological reality, represented by Anglo-Saxon age thresholds – in contrast to Ariès's arguments about "childhood" as a function of modernity – whilst not projecting modern assumptions as to the nature of this period requires significant nuance and caution. As care of offspring is a form of social investment implying value, and in the cases of mothers and infants, biologically mediated and motivated to a large extent, the least ambiguous indicator of the social status of children is their health and parental care. Thus, combining an understanding of archaeological evidence with documentary, osteological, and biogeochemical markers of health, disease and care can perhaps place us a step closer to translating the cultural codex of Anglo-Saxon childhood.

4.4.2 Health and parental care

Infant feeding practices and care are largely ignored by medieval writers, making the early medieval period one that remains enigmatic. British sources from the early medieval period are sparse; while medical treatises, lives of saints, poems, legends, art (*i.e. Maria Lactans*), and documents such as hospital records from continental Europe all provide clues as to attitudes towards children in during the later medieval period. During the Roman period preceding, maternal nursing among Germanic mothers was noted by writers such as the 1st century AD writer Tacitus to be nearly universal,

regardless of social status (Jochens, 1996; Benario, 1999). Occupying a poorly-illuminated cultural interval between the Romano-British and later Christian Anglo-Saxon Britain, the early (presumed) pre-Christian Anglo-Saxon period presents a dilemma: to assume continuity in ideas and practice from the Romano-British period preceding, or from the middle and later Anglo-Saxon period which followed?

4.4.2.i Romano-British precedents

Fildes (1995) argues that in preindustrial Europe, the question of whether or not a baby was breastfed or not is invalid: if a baby survived, it was almost universally the case that they were breastfed, whether maternally or by a relative or wet nurse. Babies who were unfortunate enough to be "hand fed" a solid diet or fed on animal milks in the early weeks of life would had low odds of survival, with the likelihood of death falling to 50 percent if artificial feeding was initiated at a month's age (Barness, 1987). It is therefore probably not coincidental that infant feeding vessels are commonly found at ancient Greek and Roman infant *burial* sites (Lacaille, 1950; Weinberg, 1993), rather than domestic habitation sites. While microbial contamination is a serious concern, it is now known that untreated animal milks, even when free of common pathogens, are entirely nutritionally inappropriate for human infants (Weaver and Prentice, 2003), and that the risk of death is up to four-fold higher in infants who are given animal milks or solids during the neonatal period, even when partially breastfed (Bahl *et al.*, 2005; Edmond *et al.*, 2006). Until the advent of modified infant formulas in the 20th century, feeding animal milks to infants posed a heavy risk of death through protein intoxication or hypoelectrolytemia, in addition to the lack of protection from human immunological factors (Barness, 1987; Kleinman *et al.*, 2003). Undoubtedly with these risks in mind, Aristotle wrote that babies were generally breastfed, with the relationship between lactation and amenorrhea being known, although misunderstood. It was thought milk was formed from stored menstrual blood during pregnancy, an idea which was accepted well into the 19th century as part of humoral medicine (Fildes, 1986; Aly, 1996; Obladen, 2012).

The first medical sources to pay significant attention to paediatric care derive from first and second century Rome, with non-medical sources such as Pliny the Elder and Plutarch providing supplemental commentary on contemporary infant care (Fildes, 1986). Of these medical sources, the most significant and influential in preindustrial Europe was Soranus of Ephesus's (late 1st to early 2nd century AD) *Gynaecology*, which

covered childbirth, infant management, and children's diseases. *Gynaecology* advised against the feeding of colostrum for newborns; instead a boiled honey or honey and goat's milk purge was offered, followed by mature breastmilk from a wet nurse beginning on the second day postpartum and continuing for 20 days (Temkin, 1956). Mother's milk could, it was thought, safely be fed to the infant after this point – immature human milk was thought unwholesome, and physicians who advised feeding first milk to newborns were derided by those favouring non-colostrum purges and temporary non-maternal nursing (Fildes, 1986).

The first discussions of milk quality arose at this time: the "Nail test" is widely credited to Soranus but was mentioned by Pliny the Elder a century earlier – milk which stayed motionless on fingernail was thick (bad), milk which ran off was thin (bad), whereas milk which spread gently was of an ideal quality. These erroneous ideas about "bad milk" have persisted into the present day, with far-reaching negative effects for infant nutrition (Dettwyler and Fishman, 1992; Baumslag and Michels, 1995). Milk quality, not only in its physically-observable appearance, but in its effects, was felt to be directly related to the qualities of the woman nursing (Aly, 1996). Personality traits and virtues were believed to transfer from nurse to infant through the milk, behoving careful parents to consider whether the mother were herself temperamentally suited to nursing her infant, and to be highly discriminating in their choice of a nurse if she were not (Temkin, 1956). Thus, breastfeeding was not believed to be the common, accessible, and defining feature of all mammals, but a specialised pursuit desirable and achievable only to a favoured group, a view which is still pervasive in the modern world. Rather than feeding "on demand", Soranus prescribed frequent, small feeds, albeit with an apparent understanding of the supply-demand nature of milk production and the need to stimulate production (Temkin, 1956). In arguing against feeding based on infant cues, he established a myth which is also still frequently heard repeated in the present: he claimed that although excessive crying is bad for babies, the newborn should not be given the breast simply because it cries, as crying "is a natural exercise to strengthen the lungs" (Temkin, 1956:111).

In addition to the beliefs concerning milk quality and the virtue of parent-controlled feeding which Soranus introduced or perpetuated, *Gynaecology* insisted that breastfeeding was above all debilitating and disfiguring, a fallacy which has also persisted into the modern day. Soranus explains:

> ...just as [the earth] is exhausted by producing crops after sowing and therefore becomes barren of more, the same happens with the woman who nurses the infant; she either grows prematurely old having fed one child, or the expenditure for the nourishment of the offspring necessarily makes her own body quite emaciated. Consequently, the mother will fare better with a view to her own recovery and to further childbearing if she is relieved of having her breasts distended too" (Temkin, 1956:90).

This belief in the debilitating but necessary nature of breastfeeding may go some way to explaining the popularity of wet-nursing among parents who could afford the labour of slaves or hired nurses, as well as the low regard in which breastfeeding appeared to be held in the Classical world.

Despite viewing breastfeeding as an affliction best avoided by those with the means to do so, Soranus did not advise easing the burden by introducing solid foods at an early age. Instead, he recommended introducing solid foods at around six months of age, with first foods being soft offerings such as porridges, soft-cooked egg, cereals, bread sop of milk or wine, and water and wine as supplementary drink (Temkin, 1956). Cessation of breastfeeding was then intended to occur gradually, reaching completion around 18-24 months of age (ideally in spring) if all teeth were erupted and the child was eating well. It is unlikely that these recommendations were followed with a rigid adherence to age (Fildes, 1995), and it is equally uncertain how closely they were followed in Roman Britain.

Galen of Pergamum (AD 129-c.200), a second major Classical source whose advice continued to be followed for over a millennium following its publication, disagreed with Soranus' recommendations in his *De Sanitate Tuenda* ("Hygiene") and advised maternal milk from birth, following a honey purge. The duration of breastfeeding was to be three years in total, longer than the period suggested by his predecessor (Piovanetti, 2001), but with solids being introduced at eruption of first teeth, rather than a set age, which in practical terms concurs with the guidelines proposed by Soranus regarding food (Vuorinen and Mussalo-Rauhamaa, 1995). A more adult diet of bread, vegetables, meat, and milk was suggested (with wine forbidden until adulthood) in acknowledgment of dental eruption, in contrast to Soranus' soft sops and porridges (Fildes, 1986). In both the Soranic and Galenic recommendations, the process of replacing breastmilk in the diet with solid foods was designed to be slow and gradual – occurring over a period of years, rather than the modern tenure of months. This may have appeared something of a hardship in light of Galen's other guideline – complete

sexual abstinence for the entirety of the nursing period. This ban derived from Galen's belief that the milk would become altered by intercourse, in addition to the risk of pregnancy, which would divert resources from the nursing child to the developing foetus. Whether or not this prohibition was strictly observed, Obladen (2012) concluded that the expectation of maternal abstinence, which persisted into the early 20th century, and continues to be a feature of many non-Western societies in the modern world, served to isolate mothers, create a rivalry between father and child, and provided a justification for male extramarital affairs.

4.4.2.ii Early medieval sources

In contrast to the many extant continental sources for both the Roman and medieval periods, there are few *indigenous* primary sources which directly address the care of children in early medieval Britain. Most information about the care and feeding of children has therefore had to be inferred from oblique references contained within texts on other topics. Until the process of childbirth began to be medicalised during the 18th century (Cahill, 2001), the majority of English physicians paid scant attention in their texts to infant care or feeding, as it was seen as the province of midwives and women, not of male medical writers (Fildes, 1988b). Crawford (1999) comments that during the pagan Anglo-Saxon period it is likely that maternal breastfeeding was nearly universal in England. However, references to wet-nursing were appearing in Anglo-Saxon sources by the Christian period. Bede in his *Ecclesiastical History of the English People* reproduced a *Libellus responsionum* ("book of answers") written by Pope Gregory I (c.540-604) in response to St. Augustine of Canterbury's (d. 604) letter requesting clarification on several points relating to righteous behaviour for clerics and the laity among the newly-converted English. While commenting on the matter of postpartum conjugal relations, Gregory made known his disapproval of wet-nursing:

> A man should not approach his wife until her child is weaned. But a bad custom has arisen in the behaviour of married people that women disdain to suckle their own children and hand them over to other women to nurse. This custom seems to have arisen solely through incontinency; for when women are unwilling to be continent, they refuse to suckle their children (Sherley-Price *et al.*, 1990:85).

This prohibition on marital intercourse for breastfeeding women mirrors the Classical warnings against premature diversion of maternal resources from nursling to foetus, but it is more likely to be based on a religious rationale, later stated in the *Libellus*. Gregory sternly admonishes, "Lawful intercourse should be for the procreation of offspring, and

not for mere pleasure; to obtain children, and not to satisfy lust" (Sherley-Price *et al.*, 1990:86). Since lactational amenorrhea would render a new mother temporarily infertile, then under the moral framework laid out by Gregory sexual intercourse would not only be potentially harmful to an existing infant if a new pregnancy did occur, but also sinful if the procreative potential of coitus was prevented, which was of greater concern to monastic writers who were intent on preserving the spiritual health of the newly-Christian kingdoms of the English. The language used in the *Libellus* also makes it clear that blame for "incontinence" was placed solely and unfairly on mothers, who would surely have been subject to competing pressures from motherhood, marriage, and religion. Where maternal breastfeeding did occur among elites during the Christian period, it was accompanied with significant religious prestige, as being associated with the Madonna, to whose pattern of maternity all virtuous women should aspire (Shahar, 1990).

It is unclear whether the prevalent maternal breastfeeding noted in the first century by Tacitus to be typical of Germanic populations was supplanted in high-status Anglo-Saxon families by wet nursing in the same way that other pre-Christian customs became replaced by the habits of Christian Rome, or whether the putative change to maternal nursing among high-ranking Anglo-Saxons was due to intrinsic factors unconnected to the introduction of Christianity and the re-establishment of Roman influence via the church. It is even questionable whether Tacitus may be considered a reliable or unbiased source for "Germanic" ethnography, which must not only take Tacitus' own agenda into account, but also variation in culture, time, and place – Anglo-Saxon elites may have always relied on the services of wet nurses (Kuefler, 1991). Fosterage, or direct care of a child by persons other than their parents, whether by a nurse within the parental household, or through entirely external care in another élite household to cement social ties and obligation, is documented to have been widespread later in the Anglo-Saxon period, and thus alloparental care should be expected within this socioeconomic group.

Regardless of the uncertainties of pre-Christian practice, wet nurses are mentioned in the law code of Ine of Wessex (AD 688-726) as one of the essential servants a *thegn* (thane) was allowed to take travelling with him on business for his lord (Crawford, 1999). This mention is intriguing and suggests the expectation of a highly involved role for fathers when compared to modern fathers, who would be expected to leave their families at home while on business. However, caution should be taken when drawing

conclusions about overall cultural norms, based on the recorded customs of a privileged group. For example, although wet nursing was common among the aristocracy of later medieval western Europe, due to high infant mortality and a faster return of fertility for the mother (Fildes, 1988a), these strategies are not expected to have applied or appealed to the common poor, whose resources would support fewer children. While these allowances granted to a *thegn* shed no direct light on the practices of the common people, they do demonstrate that non-maternal nursing was clearly taking place. As in other periods, most children during the early medieval period are thought to have been breastfed, whether by their mother or a nurse, an average of two to three years during this period, with some disagreement about the speed and pattern of the weaning process (Crawford, 1999). The uncertainties as to breastfeeding duration, and the length and intensity of the weaning process, particularly during the early Anglo-Saxon period, will hopefully be somewhat lessened by the present study.

Despite the paucity of sources specifically addressing the parameters of breastfeeding itself during this period, a few sources obliquely approach the care and diets of children during the later early medieval period. Aelfric's Colloquy (10^{th} c.) is a dialogue written in both Anglo-Saxon and Latin which was written in order to instruct young Anglo-Saxon boys in Latin, rather than as a book of history. However, its content provides a broad level of detail about the lives and diets of a range of individuals during the late 10^{th} and early 11^{th} centuries. When boasting of his craft, the Baker speaks of the importance of his bread to young children (Garmonsway, 1978), suggesting that their diets may have been based around the same farinaceous staples as children in later periods. Within the dialogue, relevant mention is made of the entrusting of young boys to the care and instruction of monastic communities, with a boy of unspecified age speaking of beatings as a matter of routine for misbehaviour. The same child gravely explains to his interrogator he ate moderately of eggs, vegetables, fish, cheese, and in respect of his age and position, also meat, with ale or water to drink (Garmonsway, 1978). While this document was designed for purposes other than dietary history, and further documentary sources of information regarding the care and diets of young children in Anglo-Saxon society remain sparse, we can cautiously conclude that children's diets may have introduced different food groups at different stages of development, and that by the time they were fully weaned and beyond early childhood, they consumed a diet resembling that of adults.

4.4.3 Child mortality and inclusion in the burial rite

Despite the parental investment in early medieval children through widespread breastfeeding, child mortality would have been high, as it was in the preindustrial period, and remains so in the developing world. Assessment of child mortality represents a particularly challenging problem for the archaeologist, because of a widely acknowledged disparity between expected mortality and actual representation of child burials in Anglo-Saxon cemeteries. Young children only account for 10-15% of inhumations of the 5th and 6th centuries AD, while expected mortality for juveniles in non-industrial populations should amount to as much as 50% of deaths (Crawford, 2011). Different explanations have been suggested to account for the dearth of infants and young children in the archaeological record of the pre-Christian early medieval period. Buckberry (2000) has argued that children's remains, being poorly mineralised, are more likely to be disproportionately affected by diagenesis and other taphonomic factors and less likely to survive in the environment to be represented. Other archaeologists have argued that differential disposal – burial at shallower depths, leading to truncation, or burial in a different area – may account for the dearth of young children present in early Anglo-Saxon cemeteries (Lucy, 1994; Härke, 1997; Crawford, 1999: 76; Crawford, 2011). Sayer (2014) has offered perhaps the most intriguing explanation for the paucity of children in some Anglo-Saxon cemeteries. Arguing that contemporary estimates for child mortality from developing regions such as Africa are extreme and inappropriate, he pointed out that distribution of infants is highly skewed towards underrepresentation in many small cemeteries. With no shortage of children represented in most large cemeteries, he argues that return journeys in maternal mobility at the time of marriage and birth may account for the unequal distribution. In other words, patrilocal residence combined with matrilocal burial meant that women may have been more likely to return to their natal kin network at the time of birth, and also likely to bury their juvenile offspring at centres of tribal significance to her own family also. This regionalist theory is interesting, and not incompatible with other theories regarding the underrepresentation of children.

Regardless of causes, low numbers of children's skeletal remains in many locales offer keen challenges to understanding Anglo-Saxon childhood within cross-sectional population studies. Studies of breastfeeding and weaning patterns have faced particular challenges due to this issue. It is arguable that under these circumstances, the

osteological paradox (Wood *et al.*, 1992) should be a central concern. With such a paucity of remains represented in the archaeological record for this period, individual children who are present in cemeteries are much more likely to be exceptional or abnormal in ways that may not be apparent than would be the case in contexts with greater representativity. This is particularly the case for regions such as the medieval Fens, where high stress and elevated childhood mortality may be suspected. Unfortunately, it is not possible to confirm or allay these concerns, except to seek sources of information about the lives and deaths of Anglo-Saxon children from a wider range of sources than the remains of deceased juveniles alone.

4.5 Finding the children: circumventing issues of preservation and mortality bias

If fluctuating water levels in the Fens presented no special difficulties to its inhabitants, and in some respects provided protection and prosperity to Fenlanders, the same cannot be said to be true for their effects on bone preservation and the work of archaeologists seeking to study past children. Preservation of bone is acknowledged to be generally poor in areas with fluctuating water levels, resulting in degradation and destruction (Jans *et al.*, 2002; Kendall *et al.*, 2018). In addition to diagenesis, transformation of the Fens from wetland to intensively farmed arable land has increased taphonomic damage of archaeological features and finds through drying and ploughing (Lane, 2015), which will undoubtedly have truncated or disturbed many comparatively shallowly-buried early medieval infant skeletons. It is therefore probable that the challenges facing studies of Anglo-Saxon childhood generally are writ large in the Fens, and that excavated infant skeletons represent a fraction of children actually consigned to the grave in that period. Consequently, the interpretative difficulties regarding the dangers of mortality bias are further increased as sampled non-adults may be drawn from an exceptional subcategory of an exceptional subcategory: non-surviving children whose remains persisted in the burial record.

How then to proceed with understanding patterns of childhood health and care, particularly the study of breastfeeding patterns? Analysis of teeth may offer a way forward. Teeth begin and complete their formation during the first decades of life and the study of the permanent dentition, retained throughout adulthood, allows a study of childhood health with a wider cross-section of individuals. Such samples might include both survivors and non-survivors of childhood, males and females, normative and non-normative burial types, and variable states of visible pathology. In addition to mitigating

mortality bias and issues of representativity, dentine is structurally more resistant to diagenesis than bone, and is therefore more likely to be intact even under generally poor conditions of preservation (Kendall *et al.*, 2018). Such an increase in the breadth of archaeological remains available for analysis undeniably extends the scope of the questions we can ask regarding Fenland childhood. In such an area of research, with compelling and unsolved problems such as identity, establishment of disease ecology, human-landscape interactions, and culture of care unaddressed by the historical record, additional methodologies which may make the early medieval period less obscure are essential.

5. Materials and Methods

5.1 Materials

This section will outline and summarise contextual information regarding the sites and individuals included in the present study. It will also outline the rationale used in the selection of these sites and individuals.

The primary aim of the study was to identify the potential effects of environmental pressures on childhood health and diet in the setting of early Anglo-Saxon Fenland (discussed previously in Chapter 4). Due to the limitations of scope and cost that are inherent to doctoral-level research, it was not possible to complete a comprehensive isotopic survey of Fenland burial sites during this period. Comparison and contrast of two large contemporary sites, representing Fenland and non-Fenland environments, within the Anglian region was deemed more feasible, allowing some illumination of the unique health challenges in the Fens. This was the strategy chosen for the present study.

5.1.1 Site selection criteria

The primary selection criteria for the two sites were common chronological, geographical, and quantitative features: both cemetery sites needed to be contemporary, within the same region, and be of sufficient size and demographic quality to allow for representative sampling. Secondarily, the two sites needed to be prototypically representative of the observable skeletal health patterns seen broadly in Fenland and upland environments. Acute disease processes generally do not modify the skeleton, as individuals either recover or die before changes take place, producing a false appearance of health among individuals suffering from these diseases in life (Wood *et al.*, 1992). However, chronic disease processes and the systemic stress they produce may induce alteration of the skeleton through disruption of bone metabolism, resulting in the appearance of skeletal lesions (Roberts and Cox, 2003). While certain chronic diseases may produce characteristic and diagnostically-specific patterns or type of lesion, several skeletal lesions including periosteal new bone formation, enamel hypoplasia, cribra orbitalia (hypertrophy of the superior orbit, resulting in a spongy appearance), porotic hyperostosis (hypertrophic marrow expansion of the cranial vault), decreased stature, or radiographically-visible growth arrest lines are believed to be multifactorial in aetiology, and therefore constitute non-specific markers of general

health stress which may include origins of infection or deficiency (Goodman and Martin, 2002; Larsen, 2002).

Gowland and Western (2012)'s application of GIS methodology to align palaeopathological and ecological data demonstrated a relationship between geographical environment, disease pressures, and prevalence of skeletal stress markers: specifically, the correlation between *anopheline* mosquito habitat and high prevalence of cribra orbitalia in Anglo-Saxon Fenland cemetery populations. For this reason, potential candidate sites were screened via the osteological reports in the grey literature for characteristic patterning of stress markers. While it was deemed desirable that the chosen Fenland cemetery population exhibit the high prevalence of skeletal stress markers characteristic of Fenland sites during this period, it was equally desirable that the non-Fen comparative site be representative of broader early medieval norms for health in Britain. Thus, while the Fen site should epitomise the features of interest (osteological markers of stress) characterizing Fenland populations, it was essential that the comparative site represent an average state of health during this period. Sites with an unusually high population burden of skeletally-observable communicable disease which could obscure differences in feeding and stress patterns (such as tuberculosis) were excluded from consideration as candidates for non-Fen representation.

Two sites were subsequently evaluated for comparison based on these criteria: Highfield Farm, Littleport, a former Fen island which would have sat at an interface of peat and silt fen, and Edix Hill (Barrington A), located southwest of Cambridge. Both sites conformed to selection criteria. Mortuary artefacts dated each cemetery assemblage within a use period of the 5th to 6th centuries AD (NB: whilst Edix Hill was in use from the 5th to 7th centuries AD, only burials dating from the early phase of the cemetery were selected for analysis). Spatially, if not environmentally, the sites were in relative proximity to each other (Figure 5.1). With less than 30 miles between them, this distance could have been covered on foot and watercraft in a day, and it is likely that each of the two communities would have been aware of the other. To ensure representativity of Anglo-Saxon Fenland and non-Fenland health by Littleport and Edix Hill, data on the prevalence of a series of skeletal stress markers at the sites was compared to early medieval mean prevalence for Britain (Roberts and Cox, 2003). Figure 5.2 demonstrates how closely Edix Hill conforms to mean prevalence of skeletal stress markers for the early medieval period in Britain, as well as demonstrating

Littleport's divergence from this norm – particularly in the case of cribra orbitalia (true prevalence rate 59.5%). Based on these measures of conformity to the selection criteria, the sites were selected as suitable for the purposes of the present study.

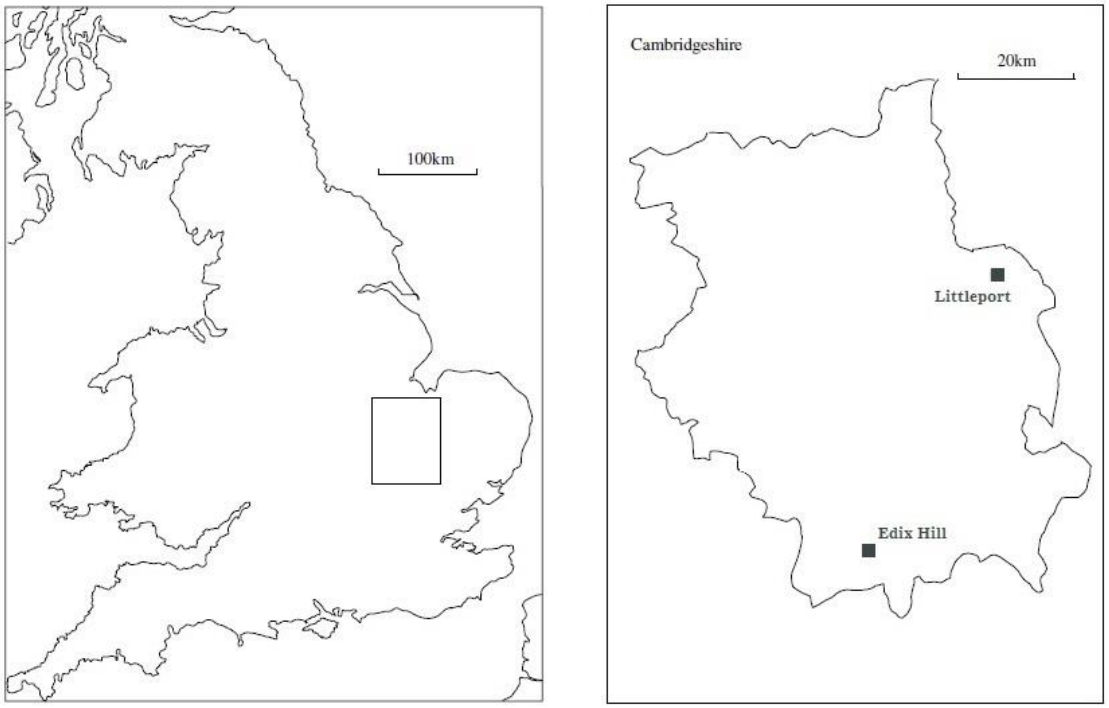

Figure 5.1 Location map showing position of study sites within modern Great Britain and Cambridgeshire. Adapted from Holt (2008, Figure 1).

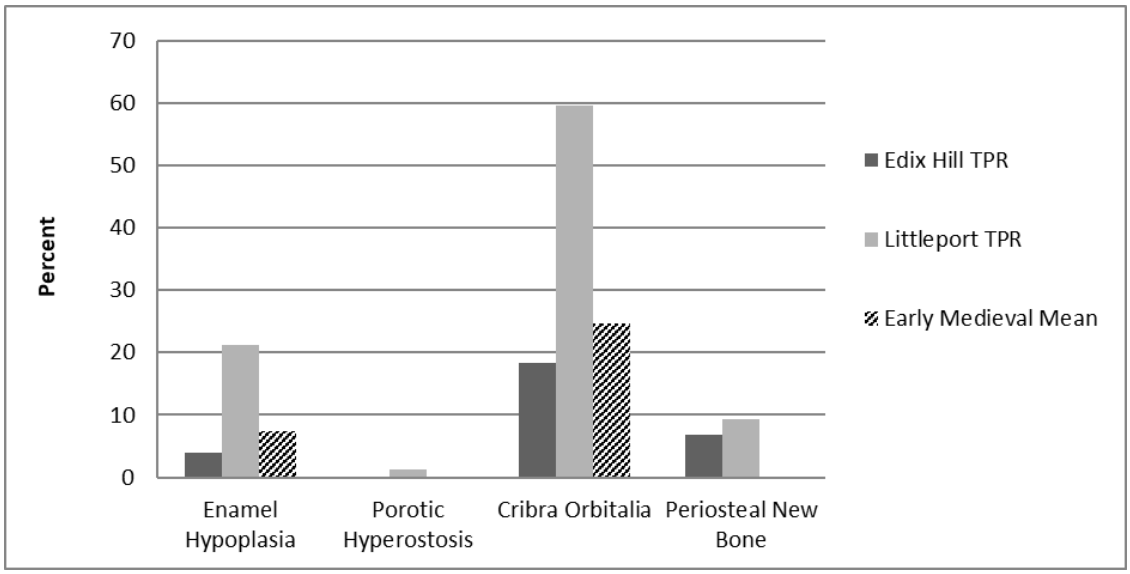

Figure 5.2 Prevalence of non-specific skeletal stress markers (expressed as true prevalence, or TPR) for the study sites and early medieval mean. Data from Roberts and Cox (2003).

In terms of overall comparative cemetery demography, the sites do not differ greatly in pattern of age representation within the cemeteries (Figure 5.3), aside from a slight shift in peak representation which occurs in the very young adult to young adult age

groups at Edix Hill, with a paucity of individuals in the mature adult age group and a secondary peak in the old adult age group, while peak representation within Littleport occurs within the mature adult age category. This may represent a differing pattern of mortality at the two sites, but in acknowledging the underrepresentation of key demographic groups, including infants and the elderly, within Anglo-Saxon populations, interpretation will not lean heavily on any assumption of comprehensive representation of the past communities of Littleport and Edix Hill within the incomplete skeletal population samples they have produced (cf. Wood *et al.*, 1992; Buckberry, 2000).

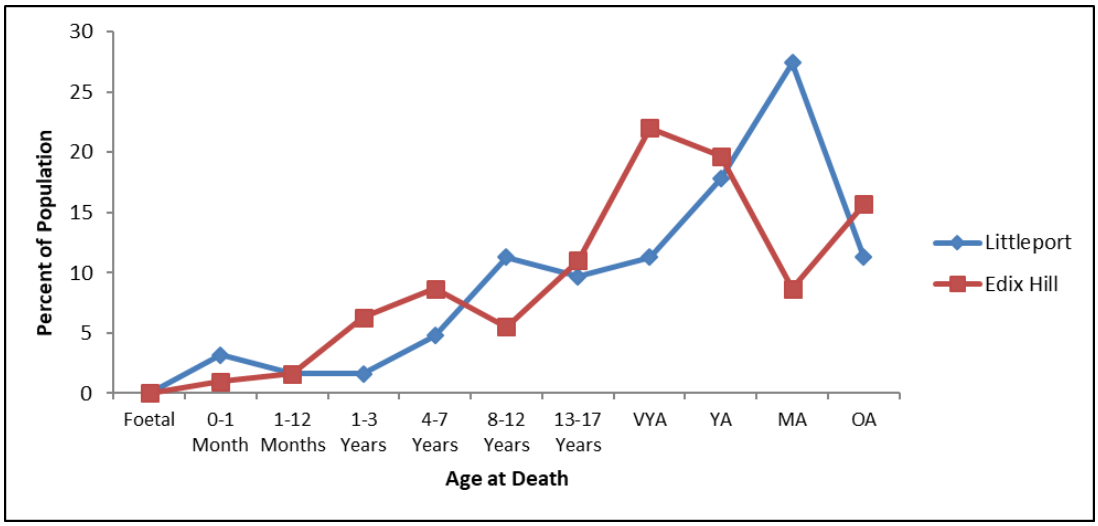

Figure 5.3 Demographic profiles of Littleport (n=62) and Edix Hill (n=127) based on age-estimable sample of population. Data compiled from Western (2008) and Duhig (1998). VYA=very young adult, YA=young adult, MA=mature adult, OA=old adult.

5.1.2 Edix Hill (Barrington A)

5.1.2.i Location

The cemetery of Edix Hill was situated on a chalk hill approximately 12 km southwest of the modern city of Cambridge (Figure 5.1), on the western border of the Cambridgeshire parish of Barrington. The hill rises to a maximum of 170 m OD, or 4 m above the surrounding low-lying plain of Gault clay and is the northwesterly culmination of an area of Lower Chalk originating near Barrington in the southeast (Malim and Hines, 1998a). This area of chalk is intersected by the Rhee tributary to the River Cam, which lies to the southeast of the site (Figure 5.4) and lies within an area triangulated by the Roman roads of Ermine Street, Worsted Street, and the putative Ashwell Street. The land surrounding Edix Hill would have been prone to flooding, due to its proximity to the river and heavy, poorly-draining sediments present, and would

have been more likely to have been used for grazing than arable agriculture (Malim and Hines, 1998a).

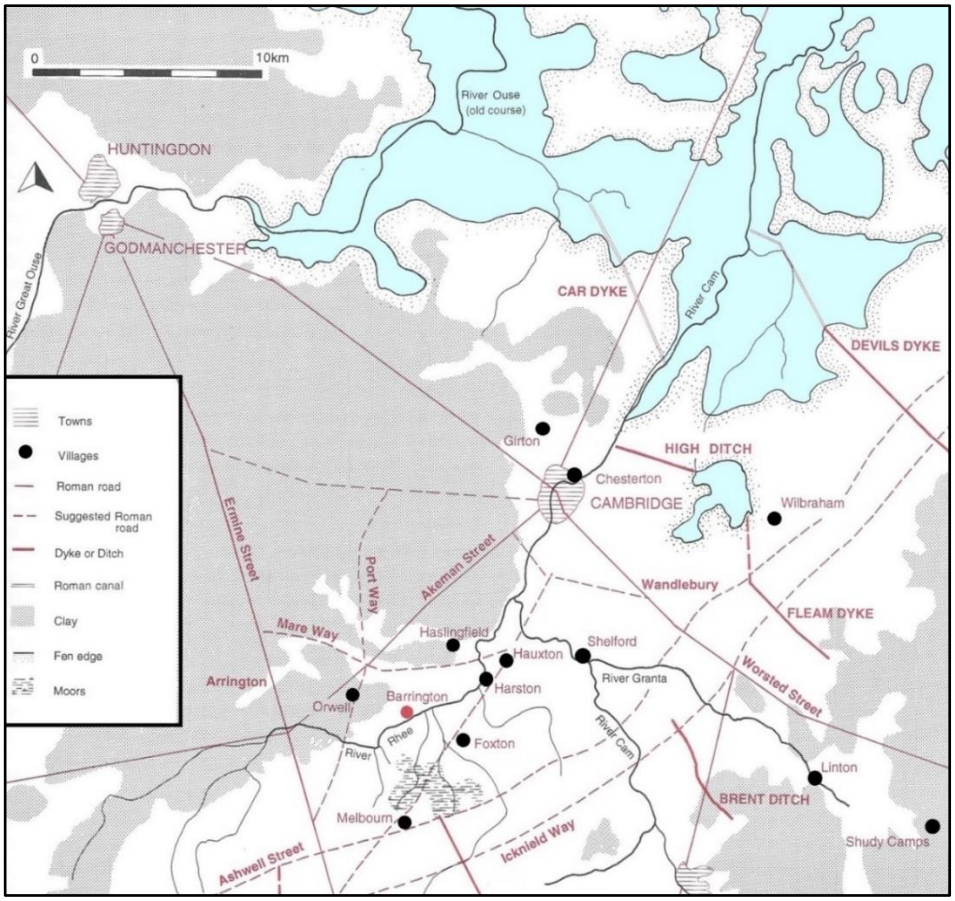

Figure 5.4 Map of the area surrounding Edix Hill. The village of Barrington is indicated with a red marker. Adapted from Malin and Hines (1998:5).

5.1.2.ii Historical setting

The cemetery site at Edix Hill (Barrington A) was first officially documented in 1860, when drainage works resulted in the discovery of human skeletal remains and artefacts (Babington, 1860). However, finds which were not documented at the time of unearthing occurred much earlier than that, such as a sword burial found in 1840 (Malim and Hines, 1998a). Two excavations were conducted in 1860 and 1861 at the site, by Thomas Bendyshe and Joseph Wilkinson respectively, which revealed 40-50 inhumations (Malim and Hines, 1998a). A second large 5-7th century Anglo-Saxon cemetery at nearby Hooper's Field (Barrington B) was excavated in 1880 after being encountered and partially-destroyed by coprolite extraction, an important local industry of the latter half of the 19th century (Foster, 1883). The relationship between the two contemporary burial grounds, and the community or communities which they may have served, continues to be unclear (Malim and Hines, 1998a).

5.1.2.iii Excavations and findings

Following the excavations of the 1860s, no further discoveries were documented at Edix Hill until 1987 and 1988, when reported finds from metal detection led to county archaeological evaluations confirming the location of the cemetery (Malim and Hines, 1998a). Long-term protection of the site was deemed desirable but unfeasible, due to the impossibility of obtaining scheduled monument status, and a lack of funding with which to persuade landowners to discontinue ploughing. Consequently, excavations to sample approximately half of the extant cemetery were undertaken by Cambridgeshire County Council Archaeological Field Unit over three seasons from 1989 to 1991 (Malim and Hines, 1998a). Excavation revealed that Edix Hill conformed to a commonly-observed pattern among Anglo-Saxon cemeteries, that of reuse of prehistoric funerary monuments (Lucy, 2000; Williams, 2011), with a late Iron Age ditch inhumation and finds relating to a Bronze Age barrow also present (Malim and Hines, 1998a). The presence of prehistoric monuments, together with the visibility of Edix Hill in a low-lying landscape, was no doubt one of the attractions leading to its continued use for mortuary purposes (Malim and Hines, 1998a).

5.1.2.iv Demographic summary

The 1989-1991 field seasons recovered the remains of 149 individuals in 115 graves. The quality of bone was generally excellent in terms of both gross and histological preservation, due in part to the favourable chalky soil common to Cambridgeshire (Kendall, 2014). Despite the high quality of bone preservation, the impacts of ploughing and other agricultural disturbance, especially to the shallower burials, resulted in truncation or disturbance of many of the burials and destruction of fragile skeletal elements, including the majority of skulls excavated (Duhig, 1998). Of the 149 individuals excavated during the 1989-1991 seasons, age could be estimated for a total of 127 individuals. Distribution of age categories (defined in Table 5.1) represented produced the demographic structure shown in Figure 5.5. Malim and Hines (1998b) estimated a minimum of 300 burials originally interred at Edix Hill, with a use-life of 150 years, suggesting that it served a community of around 50 individuals at any given time.

Age Band	Age Represented
Foetal/Neonate	Conception to Birth
Infant	1-12 months
Child	1-12 years
Adolescent	13-18 years
Very Young Adult (VYA)	18-25 years
Young Adult (YA)	25-35 years
Mature Adult (MA)	35-45 years
Old Adult (OA)	45+ years

Table 5.1 Age categories used in study

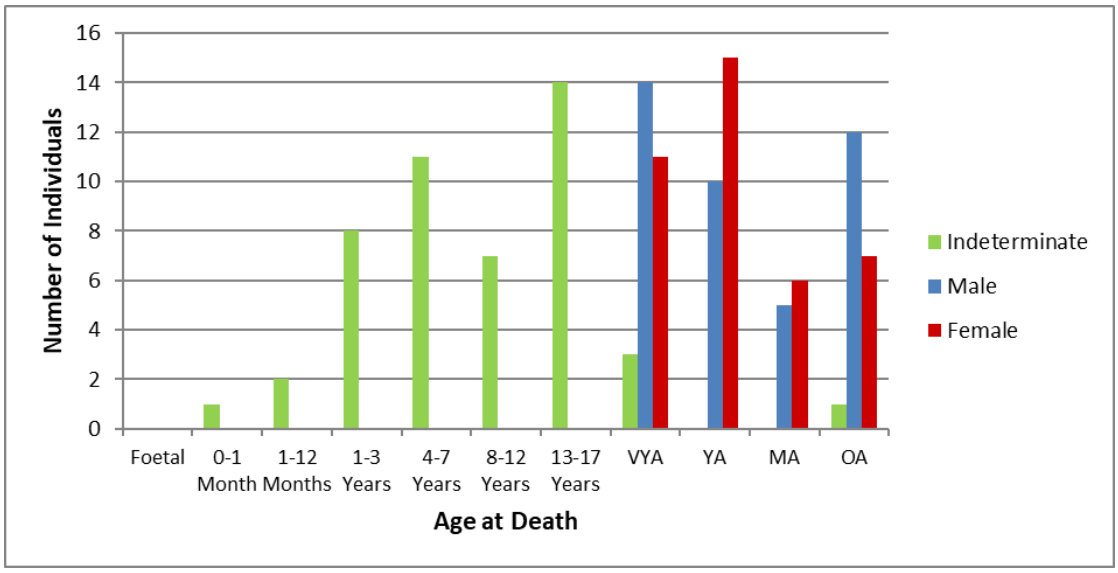

Figure 5.5 Demographic summary for age-estimable population (n=127) at Edix Hill. Data from Duhig (1998).

Reconstruction of the demographic structures of past populations from excavated skeletal populations is widely acknowledged as an intrinsically problematic and controversial undertaking (Bocquet-Appel and Masset, 1982; Van Gerven and Armelagos, 1983; Konigsberg and Frankenberg, 2002). Factors introducing bias such as variation in the burial rite, bone diagenesis, mechanical disturbance, excavation sampling strategy, accuracy of demographic analytical methods, and post-excavation curation all playing varying roles in the likeness borne by cemetery populations to the communities they served (Wood *et al.*, 1992; Duhig, 1998). This is an area of general concern, as researchers are in broad agreement that current skeletal age estimation methods for adults routinely underestimate the age of older adults (Konigsberg and Frankenberg, 1992; Martrille *et al.*, 2007; Milner and Boldsen, 2012), and that the remains of older individuals may be more vulnerable to post-mortem destruction (Walker *et al.*, 1988). The representation of the very young, like that of the very old, is a specific concern for the Anglo-Saxon period, where infants are routinely

underrepresented in skeletal populations, relative to expected child mortality levels for nonindustrial societies (Buckberry, 2000; Stoodley, 2000). Consequently, the demographic structure of the cemetery population at the sites studied is unlikely to reflect the living community during the period the cemeteries were in use, in its entirety. With the relationship between the burial grounds at Edix Hill (Barrington A) and Hooper's Field (Barrington B) also unclear, caution is doubly urged. With this caveat, the descriptive palaeodemographic structure of the skeletal population at Edix Hill will be outlined briefly for adults and children in the sections below.

Adults

Sex ratios of close to 1:1 would be expected for a rural living population of the period, which is reflected by the overall adult (n=102) sex distribution of the individuals for whom sex estimation was possible (Table 5.2), consisting of 47% females and 53% males (Duhig, 1998). The age distribution of males and females within the population followed divergent patterns. Peak representation of females was in the young adult (25 to 35-year-old) category. In contrast, male representation within the sample was bimodal in pattern; with peaks in the very young adult (17 to 25-year-old) and old adult (45+ years) age categories. Twenty percent of the adult population represented individuals surviving beyond the age of 45 years, with a full third of the skeletal sample estimated by Duhig (1998) to be over 40 years of age at the time of death. Although this is almost certainly an underestimation, due to issues of bias in age estimation methods, as well as the possibility that the unaged individuals may also belong to the oldest age category, it does suggest a reasonable expectation of longevity. It is worth noting Duhig's inconsistency in adult age reporting. While broadly reporting demographic patterns in terms of age categories (i.e. Adult 1-4, with Adult 1 being youngest and Adult 4 the oldest categories, respectively), ages of potentially spurious precision and non-discrete ranges have been used to report the estimated ages of individuals. For simplicity, and cross-comparability, the present study will align estimated ages with the larger categories with which they belong and will favour the analogous age terms found in Western (2008) (i.e. very young adult, young adult, mature adult, old adult) in preference to Duhig's numerical categories.

Sex	VYA	YA	MA	OA	Other	Total
Indeterminate	3	0	0	1	6	10
Male	14	10	5	12	7	48
Female	11	15	6	7	5	44
Total	28	25	11	20	18	102

Table 5.2 Adult age and sex distribution for Edix Hill. Data from Duhig (1998). (VYA=very young adult, YA=young adult, MA=mature adult, OA=old adult).

The health of the population is also reflected by stature. Duhig (1998) estimated the average adult stature at Edix Hill as 1.69 m (range 1.51–1.84 m), with the mean height for males reaching 1.73 m (range 1.60–1.84 m) and females attaining a mean height of 1.63 m (range 1.51–1.71 m), falling within the higher strata of expected height for the early medieval period (Roberts and Cox, 2003). It should be noted that Duhig utilised the Trotter (1970) formulae for estimation of stature, which ongoing work suggests may actually underestimate stature in Anglo-Saxon skeletal populations (Walther, 2017). As both Littleport and Edix Hill, in common with many other contemporary sites, have been calculated using these formulae, giving internal consistency, this is not a great concern for this study. However, recalculation of stature at these sites using updated methods may be of benefit in future.

Children

Demographically, the nonadult skeletal population at Edix Hill represents about a third of burials, with only the youngest age categories underrepresented, relative to expected mortality for non-industrial societies (Duhig, 1998). Infants under the age of one year are present in very low numbers (Table 5.3). The favourable conditions for bone preservation at Edix Hill do not suggest diagenetic factors in their underrepresentation, with non-normative deposition outside of the cemetery context or disturbance of shallow graves being more likely possibilities. The sex distribution of the non-adult population is unknown, due to the low reliability of methods for estimation of sex from skeletal morphology in children and adolescents (Scheuer and Black, 2000).

Age Band	Number
Foetal/Neonate	1
Infant	2
Child	26
Adolescent	14
Other immature	3
Total	46

Table 5.3 Non-adult age distribution for Edix Hill. Data from Duhig (1998).

However, the gendered characteristics of a portion of the child burials may throw some light on demographic structure represented by children to some degree. The high degree of agreement between skeletal sex and gendered grave goods among adults may lend some insight into the gender of children at the site for whom finds were provided. The conformity to the adult pattern of gendered positioning seen for the children, and the consistent relationship observed between apparently gendered grave goods and direction of flexed burials, including that of the 5-year-old SK632 (Hines, 1998), suggests that children within this community may have been attributed gendered characteristics at a young age far preceding puberty. However, as over half of the children's burials excavated at Edix Hill were devoid of grave goods, this cannot be used reliably as a proxy for non-adult sex ratios at the site. Similarly, few non-adult bones were intact enough to be measurable for the attribution of growth patterns. A single individual, SK136B, was observed to have reduced skeletal growth suggesting an age of 1.5-2 years alongside a dental age of around 5 years, suggesting significant disease or nutritional stress for that child (Duhig, 1998). However, acknowledging that the children represented within any skeletal series are unlikely to have enjoyed good health prior to their early deaths, it is difficult to estimate the normal growth patterns of surviving children from such measurements.

5.1.2.v Health

The skeletal health enjoyed by the community served by Edix Hill may be described as generally good, by relative contemporary standards. Table 5.4 summarises prevalence of skeletal markers of development, health, and occupational patterns at Edix Hill for the entirety of its use period. Skeletal bone among adults was robust, with strongly marked muscular attachments (Duhig, 1998). The pathologies most commonly observed within the skeletal population at Edix Hill were arthropathies, or degenerative joint disease, and dental disease. Both of these are ubiquitous in modern non-industrial populations, and place Edix Hill broadly within the expected spectrum of archaeological populations (Duhig, 1998).

Degenerative joint disease comprised the largest share of pathology observed within the sample, accounting for 40.5% of all pathology at the site. Degenerative changes to the joints of the spine were most prevalent, affecting 47.3% (crude prevalence or CPR) of individuals at Edix Hill (Duhig, 1998). Gamble (2011) described gendered differences between male and female prevalence of joint disease at Edix Hill, with 71%

Condition Type	% Prevalence	% of Total Pathologies Represented
Joint disease	-	**40.5**
Spinal	47.3	-
Non-spinal	25.7	-
DISH	1.4	-
Septic arthritis	0.7	-
Dental disease	-	**33.3**
Caries	3.2	-
Antemortem tooth loss	7.1	-
Non-specific stress markers	-	**7.1**
Cribra orbitalia	18.3	-
Harris lines	8.8	-
Enamel hypoplasia	10.1	-
Infection	**15.5**	**3.7**
Tuberculosis	0.7	-
Leprosy	1.4	-
Non-specific	14.2	-
Trauma/surgical intervention	**7.4**	**2.1**
Head wounds/trepanation	2.7	-
Post-cranial fractures	5.4	-
Ossified haematoma	0.7	-
Activity-related changes	-	**4.2**
Third trochanters	1.4	-
Squatting facets	4.7	-
Os acromiale	4.7	-
Osteochondritis dissecans	0.7	-
Dental parafunctional wear	2.0	-
Epigenetic traits	-	**7.4**
Metopism and wormian bones	11.5	-
Cranio-caudal border shift	6.1	-
Neural arch deficit	3.4	-
Spondylolysis	2.7	-
Septal aperture	3.4	-
Premature suture closure	0.7	-
Neoplasia	**1.4**	**0.3**
Button osteoma	0.7	-
Metastatic carcinoma	0.7	-
Osteoporosis	**0.7**	**1.3**

Table 5.4 Skeletal markers of health and activity at Edix Hill (prevalence is reported as crude prevalence, except for dental disease and cribra orbitalia (reported as TPR). Data from Duhig (1998).

of observable males showing signs of joint degeneration and 43% of observable females, while both sexes exhibited an inverse relationship between average wealth scores and prevalence of joint disease. The prevalence of degenerative joint disease falls within the high end of the range for the early medieval period, which in other populations saw decreases in degenerative joint changes from the Roman period, for both spinal and extra-spinal arthropathies (Roberts and Cox, 2003). Os acromiale, non-

fusion of the acromion of the scapula commonly associated with repetitive action, was much higher than expected at 4.7% CPR, relative to an early medieval mean of 2.6% (Roberts and Cox, 2003). This high prevalence for the period, together with the overall robusticity of bone and muscle attachments, may suggest a physically-active labouring population. Cranial and postcranial trauma, including fractures, which might also suggest activity patterns, were within expected norms for early medieval populations (Roberts and Cox, 2003).

After arthropathies, dental disease was the second-most common health issue, accounting for 33.3% of observable pathologies at Edix Hill. However, the prevalences of dental caries (3.2% TPR) and antemortem tooth loss (7.1% TPR) at the site were below mean prevalences (4.2% TPR for caries and 8% for antemortem tooth loss) reported by Roberts and Cox (2003) for the early medieval period, representing better than expected oral health for the period and potentially suggesting consumption of a diet with low cariogenicity. Evidence for dietary deficiency was sparse, with no observed cases of scurvy, rickets, or osteopenia. In addition to the lack of evidence for specific deficiency affecting the skeleton, two cases of diffuse idiopathic skeletal hyperostosis (DISH), a systemic disorder distinguished by the ossification and ankylosis of ligaments and entheses, were identified. The presence of DISH, whose aetiology is strongly associated with risk factors of protein-rich diet, sedentary activity patterns, and obesity (Mader *et al.*, 2013), provided evidence that at least some members of the population had not only adequate, but in some cases potentially excessive levels of nutrition.

Some non-specific skeletal stress markers have been interpreted in the past as an indicator of nutritional stress, particularly in the case of cribra orbitalia and its long-attributed association with iron deficiency anaemia. However, current thinking aligns the aetiology of many stress markers more closely with infectious disease insult, host-parasite interactions in childhood, or even hormonally-mediated processes of normal growth (Walker *et al.*, 2009; Papageorgopoulou *et al.*, 2011; Anthonappa and King, 2015). The prevalence rates (TPR) reported by Duhig as 18.3% for cribra orbitalia and 5.1% for enamel hypoplasia at Edix Hill, like the data reported for dental disease, align favourably with mean early medieval TPR reported by Roberts and Cox (2003) of 24.6% for cribra orbitalia and 7.4% for enamel hypoplasia. Gamble (2011) found some social dimensions to the distribution of skeletal stress markers, with a moderate positive association between wealth scores and stress marker prevalence among males, and an

extremely weak negative correlation between wealth and stress indicators among females.

Other areas of pathology, such as specific and non-specific infection, trauma, activity related skeletal changes, neoplastic disease, or epigenetic skeletal conditions, do not represent highly prevalent conditions at Edix Hill, and also fall well within expected range based on reported data by Roberts and Cox (2003). As with other findings by Gamble (2011) concerning specific pathologies such as joint disease and development of skeletal stress markers, the development of overall cumulative pathology in the Edix sample was potentially found to be drawn along lines of gender and age, with females in her study evidencing a relationship between lower wealth and greater pathology among younger women, and an association between higher wealth and lower pathology among older women. While it is difficult to make sweeping generalisations about the health of past populations from the limited data provided by skeletal populations (Wood *et al.*, 1992), we may summarise the data from Edix Hill as depicting a well-nourished, physically active community with a relatively good expectation of general health and longevity on a par with other populations of the early medieval period in Britain.

5.1.3 Littleport

5.1.3.i Location

Prior to the onset of large-scale drainage in the 17th century, Littleport rose above the peat fens as one of several low-lying islands located in the southern portion of the Fenland basin. Situated 8 km northeast of the modern city of Ely (also an island prior to drainage), the isle of Littleport lay to the south of the Old Croft River, formerly the primary collection channel of the waters of the southern Fen (Holt, 2008). Beneath the chalky till soils observed at higher elevations on the island, the geology of Littleport comprises an area of glacial sand and gravels overlying a base of Kimmeridge Clay. The cemetery at Littleport was located on a north and west-facing slope at the western aspect of the island, lying between 19 m and 6 m OD (Holt, 2008). Occupying such a low-lying position, nearly on a par with sea level itself, the Fen islands (including Littleport) were extremely vulnerable to minor changes in water level which occurred over time.

5.1.3.ii Historical setting

Littleport lies at an interface of the freshwater peat and brackish silt fens (Figure 5.6),

lying directly to the south of a finger-like projection of silt. This projection of silt suggests that although Littleport is adjacent to Ely, which lies wholly within the freshwater peat Fens, the northerly aspects of the isle of Littleport may have been subject to marine influence and had higher salinity waters.

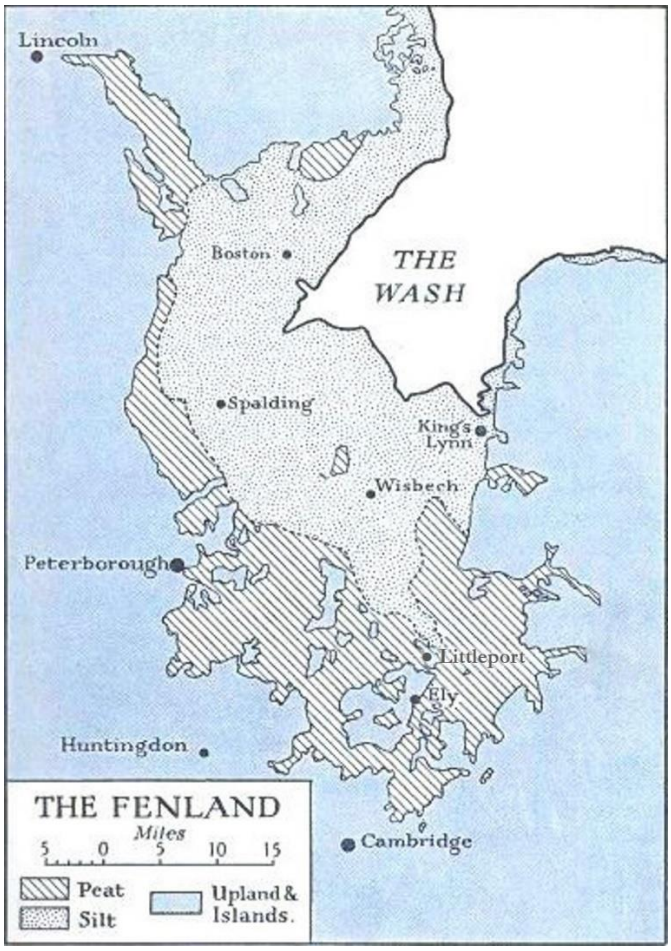

Figure 5.6 Location of the isle of Littleport at the interface of peat and silt Fenland environments prior to drainage. Adapted from Darby (1940: 5).

The probability of increased salinity in the waters surrounding Littleport, relative to adjacent insular locations such as Ely, is supported by the presence of archaeological evidence of salt making near Littleport. Briquetage and other evidence of salterns found in other sites near the Littleport cemetery, particularly along the roddon of the Old Croft River, suggests that like other Fenland communities, the isle of Littleport formed a major focus of salt making activity during the prehistoric and Roman periods (Hall and Coles, 1994). Investigations by Fowler (1950) revealed a significant Romano-British settlement at Littleport associated with these marine silts, demonstrating the island's importance as a place of settlement throughout late antiquity. Highfield Farm, a development site encompassing roughly 30 hectares to the west of the modern village,

was under investigation as early as 1996, with its inclusion in the Fenland Survey's extensive programme of fieldwalking (Hall, 1996). In subsequent years, the site was subject to further assessment and evaluation; including geophysical survey, trenching, and digging of test pits (Lucas, 1998; Dymond, 1999). After evaluation trenches encountered human remains suggestive of an Anglo-Saxon burial ground, geophysical survey was employed to estimate the full extent of the cemetery, an endeavour which was largely unsuccessful (Holt, 2008).

5.1.3.iii Excavation and findings

Full excavation of the site at Highfield Farm, Littleport occurred between 2004 and 2005 in advance of residential development. In addition to an Anglo-Saxon burial ground with finds suggesting a date of the late 5th to 6th centuries AD, excavations also revealed a range of features representing use of the site from the Neolithic onwards, with declines and upturns in activity tied to climatic conditions (Holt, 2008). Prehistoric use of the site was interpreted as ritual in nature, comprising a scatter of discrete pits containing placed animal bone (including some non-domesticated species such as wild pig and red deer), worked flints, and Neolithic Peterborough ware and Beaker pottery (Holt, 2008). Following a decline in activity during the Middle Bronze Age, an increase in archaeological activity was observed for the late Bronze age and Iron Age, during which a further series of pits and ditches were created at the site (Holt, 2008). During the Middle Iron Age, activity again declined with wetter conditions. Conditions became drier by the Romano-British period, during which an overall increase was seen in occupation of the Fen edge and island (Hall and Coles, 1994), which is reflected in an increase in evidence for field systems and other settlement features such as ditched enclosures and pits containing domestic and butchery waste at Highfield Farm (Holt, 2008). Further phases of activity were also recorded for the later medieval, post-medieval, and modern periods, but these will not be discussed here.

The early Anglo-Saxon period covered by the cemetery was climatically continuous with the drier conditions enjoyed by the Romano-British period, with settlement locations remaining overall stable along Fen edges and islands between the two periods (Hall and Coles, 1994). Atypically for a period where cemeteries are rarely found in association with a settlement (Lucy, 2000), non-funerary features were also present at the site, suggesting the need for further investigations to establish the possible presence of an associated settlement. Burials were concentrated in a southwest corner of a pre-

existing field enclosure, in an area where the underlying deposits suggest a rise in the contemporary ground level (Holt, 2008). The use of areas of higher ground for funerary sites is typical for Anglo-Saxon period cemeteries and is a point of similarity in the situations of Edix Hill and Littleport.

Both faunal and environmental assessments were undertaken at Highfield Farm, providing direct evidence for the resources available to the community served by the cemetery. Species represented within the cemetery were predominantly cattle, sheep/goat, horse, and domestic fowl, consistent with a livestock-producer site. The inclusion of these remains within the cemetery suggests the deposition of joints of meat as a part of the burial ritual (Kitch, 2008). Wild species, fish, and water fowl – readily available natural resources at the site – were significantly underrepresented in the assemblage, which has been suggested by Kitch (2008) to potentially be a result of excavation strategy, rather than a lack of exploitation of Fen resources. Environmental assessments of features of Anglo-Saxon date yielded charred grains of barley and wheat, and wild plant species which included grasses and sedge, as well as shellfish and fish bone (Martin and Rackham, 2008). Pollen analysis evidenced a nearly complete lack of tree pollens, with the exception of pine and hazel pollens, which may travel considerable distances (Wiltshire, 2008). Analysis of the soils at the cemetery also revealed a highly phosphate- and nutrient-poor composition, demonstrating that the site was not used for grazing, and may have been reserved exclusively for funerary ritual (Wiltshire, 2008). Together with faunal evidence, the botanical and pollen remains recovered paint a picture of a community practicing combined arable and livestock husbandry in a grassland landscape, formed by Romano-British field systems, within the Fens.

5.1.3.iv Demographic summary

Excavations at Highfield Farm revealed a total of 97 graves, including three horse burials. The human remains comprised 97 individuals, of which four were urned cremations and 93 were articulated inhumation burials. Seven of the inhumations were discovered on the periphery of the site, without artefactual evidence, and thus could not be confidently associated with the Anglo-Saxon phase of activity on the site (Holt, 2008). In consequence, the total number included in the osteological analysis of the Anglo-Saxon cemetery was reduced to 86 individuals. Gross preservation of the skeletal material was fair, although histological analysis showed the assemblage to have

undergone a significant and variable degree of diagenetic bone alteration (Kendall, 2014). Most skeletons were more than 50% complete, reflecting evidence of little undercutting of graves, and suggesting the use of contemporary grave markers (Holt, 2008; Western, 2008). Figure 5.7 summarises the demographic profile of the populations at Littleport for whom age could be estimated (n=62).

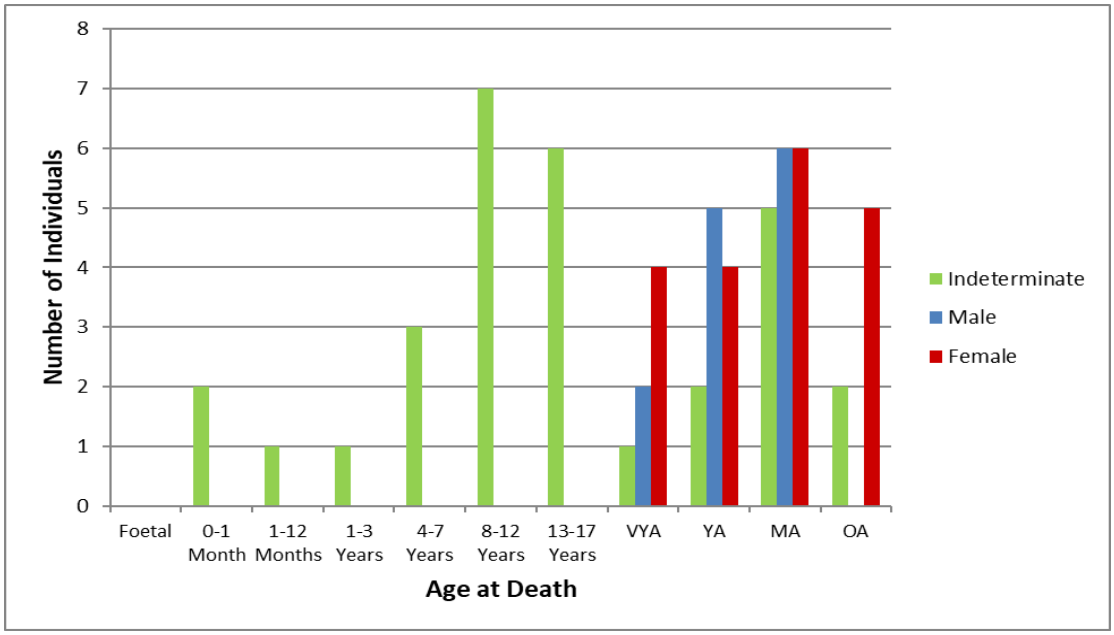

Figure 5.7 Demographic summary for age-estimable population (n=62) at Littleport. Data from Western (2008).

Adults

Table 5.5 summarises both age and sex distribution in the Littleport adult cemetery population. A male: female sex ratio of 0.76:1 was observed in the adult sample at Littleport (Western, 2008). However, the high number of individuals of all adult age categories to whom skeletal sex could not be assigned (n=24), totalling over a third of the total adult sample, must be acknowledged as potentially obscuring trends in true sex distribution. Most sexually-indeterminate individuals were also unaged, suggesting the role of preservation and post-depositional disturbance in their high numbers. Of the individuals for whom sex could be estimated, different patterns in age representation were observed. Females were fairly evenly distributed throughout all adult age categories. In contrast, male age distribution was peaked in the middle age categories of Young Adult and Mature Adult, with similar distribution between the two. Only two male individuals were represented in the youngest (VYA) age category, while they were totally absent from the oldest (OA) age group, where females were more equitably

represented. What this implies about sex and longevity at Littleport cannot be discerned, due to the small numbers of individuals represented within each age category.

Sex	VYA	YA	MA	OA	Other	Total
Indeterminate	1	2	5	2	14	24
Male	2	5	6	0	3	16
Female	4	4	6	5	2	21
Total	7	11	17	7	19	61

Table 5.5 Adult age and sex distribution for Littleport. Data from Western (2008). (VYA=very young adult, YA=young adult, MA=mature adult, OA=old adult).

Western (2008) estimated mean adult stature at Littleport as 1.68 m, with mean male height of 1.72 m (n = 11) and mean female stature of 1.63 m (n = 12), using the Trotter (1970) formulae. This stature estimation is entirely comparable with that of the Edix Hill cemetery population, placing both populations at the higher end of the spectrum of expected height for the Anglo-Saxon period. As adult stature is tied to both genetics and childhood nutrition, with nutrition playing a crucial role in enabling genetic potential (Steckel, 1995; Roberts and Cox, 2003), the relatively favourable heights achieved for the period suggest that the childhood diet at Littleport, like Edix Hill, would have compared favourably to those of their contemporaries.

Children

Table 5.6 summarises the age distribution for non-adults (n=25) at Littleport. Paralleling the age structure observed for non-adults at Edix Hill, peak representation of individuals at Littleport occurred in the Child (1-12 years) category, with a bias toward the early end of that age spectrum as expected. Underrepresentation of foetal/perinatal and infant individuals, relative to expected morality rates of young children in non-industrial settings, also mirrored trends at Edix Hill and Anglo-Saxon cemeteries more broadly. While preservation generally was poorer than that at Edix Hill, the paucity of foetal, perinatal, and infant remains cannot be interpreted as resulting from taphonomic processes, as statistical analysis of adult and non-adult preservation quality did not reveal any significant differences (Western, 2008).

In contrast to Edix Hill, no consistent pattern of status-based or age-related gendered burial treatment could be inferred for the children of Littleport. While grave goods which have been traditionally associated with gender, such as iron weaponry or large concentrations of beads, were primarily found in burial contexts containing children who had achieved middle childhood (7 years and older) at a minimum, beads were also

found in the context of a single burial of a neonate (SK4498). Half of all non-adults (12/25) were not associated with grave goods, either through total absence or through occupation of a multiple burial where extant grave goods could not be securely attributed to that individual. Four non-adults were interred in multiple burials, in total. Among burials containing grave goods, while many were found in an extended/supine position, many were also flexed, and in one case, prone (Holt, 2008). Crouched burials were the only category not represented among non-adults with grave goods. For non-adults in unfurnished graves, the full range of burial positions was represented. The lack of consistency between positioning, grave goods, and theorised markers of age-dependent gender thresholds or status places serious constraints on interpretation of these characteristics for the children of Littleport.

Age Band	**Number**
Foetal/Neonate	0
Infant	3
Child	11
Adolescent	6
Other immature	5
Total	25

Table 5.6 Non-adult age distribution for Littleport. Data from (Western, 2008).

5.1.3.v Health

Broadly, skeletal evidence of health at Littleport was comparable to that at Edix Hill. Prevalence of skeletal markers of development, health, and occupational patterns at Littleport for the entirety of its use period is summarised in Table 5.7. The image that emerges from the data is of a robust, resilient population which adapted well to various types of stress. Prevalence of most types of skeletally-visible disease was low, with the one of the most predominant pathologies observed being cribra orbitalia, which had a startlingly high 59.5% (TPR) prevalence. Current theory holds this condition to be aligned to haemolytic or megaloblastic anaemia, rather than dietary deficiency (Walker *et al.*, 2009). It is likely that the proposed role of malaria (Gowland and Western, 2012) in cribra orbitalia in Fen populations was also central at Littleport. Enamel hypoplasia (45.3% CPR) prevalence also bears witness to a population under high childhood stress.

Nevertheless, the estimated stature at Littleport does not support a characterisation of its population as nutritionally-deprived. Also supporting the nutritional adequacy of diet at Littleport is the high prevalence (81% TPR) of dental calculus (mineralised plaque) (Western, 2008), which has been associated by researchers with high-protein diets and

oral alkalinity (Roberts and Cox, 2003). To a lesser extent, non-specific "infection" as an inflammatory response resulting in the deposition of new periosteal bone, which affected just under a tenth of the population, may also be interpreted as a response to stressors at Littleport. Skeletal stress markers are known to themselves potentially act as indications of resilience, as individuals must survive long enough for skeletal modification to take place as a form of adaptation (Wood *et al.*, 1992).

Condition Type	**% Prevalence**
Joint disease	-
Spinal	25.6
Non-spinal	16.3
TMJD	1.2
Dental disease	-
Caries	5.0
Antemortem tooth loss	7.5
Calculus	81.0
Abscess	1.7
Non-specific stress markers	-
Cribra orbitalia	59.5
Enamel hypoplasia	45.3
Infection	-
Tuberculosis	1.2
Non-specific	9.4
Trauma/surgical intervention	-
Post-cranial fractures	2.3
Ossified haematoma	1.2
Activity-related changes	-
Os acromiale	2.3
Osteochondritis dissecans	1.2
Epigenetic traits	-
Metopism and wormian bones	7.0
Cranio-caudal border shift	1.2
Spondylolysis	5.8
Septal aperture	7.0
Squatting facets	1.2
Neoplasia	**4.7**
Ivory osteoma	3.5
Fibroblastic tumour	1.2
Osteoporosis	**2.3**

Table 5.7 Skeletal markers of health and activity at Littleport (prevalence is reported as CPR, except for dental pathologies and cribra orbitalia (reported as TPR).

Like Edix Hill, the population at Highfield Farm contained a significant number of individuals affected by degenerative joint disease. Both spinal and extra-spinal joint disease were very common, and increased with age (Western, 2008), suggestive of a connection with variable activity patterns and cumulative joint wear. The remarkable extent of skeletal stress markers at Littleport, together with a paucity of evidence for specific health issues and the presence of evidence consistent with a well-fed and active

lifestyle, characterise the community served by the Highfield Farm cemetery as an extraordinarily resilient population under significant stress.

5.2 Methods

The sections below will outline the methods used in the present study, including sample selection, preparation, and analytical methods.

5.2.1 Samples

The time-demands and complexity of the isotopic analysis did not allow for original osteological analyses to be carried out as a part of the larger study, and thus skeletal data for demography and palaeopathology were derived from the site reports for Littleport (Western, 2008) and Edix Hill (Duhig, 1998). Sampled teeth were selected for minimal wear, and all teeth were free of caries.

5.2.1.i Pilot study

A pilot study funded by a NERC Isotope Geosciences Facility Steering (NIGFSC) grant was undertaken in order to apply standard carbon and nitrogen stable isotope methodology to a small portion of the overall sample using a new sequential sectioning sampling technique developed by Beaumont *et al.* (2013). The end goal of observing intersite differences in weaning pattern on a small scale was intended to enable a successful application for funding to investigate health and dietary patterns on a larger scale.

Sample selection

To identify intersite differences in a small pilot sample without potentially introducing confounders through additional variables such as sex/gender or survivorship, it was decided that only adult females (n=4) would be selected for the initial phase of the study. First permanent molar dentine begins forming around the time of birth and continues to form until the root apex closes at approximately 10 years of age (Beaumont and Montgomery, 2015), providing an expected 10 years of childhood isotope data. Four mandibular M1s (Table 5.8) were prepared for dentine collagen preparation at Durham University. All teeth were abraded with a rose-headed dental burr to remove external contamination along the entire length of the tooth. The cleaned tooth was then half-sectioned with a diamond-tipped rotary dental blade, creating a longitudinal section with a single root.

Site	Skeleton	Estimated Sex	Estimated Age
Littleport	3687	Female	18-25
	4075	Female	20-30
Edix Hill	20B	Female	~25
	726	Female	17-25

Table 5.8 Individuals included in carbon and nitrogen isotope pilot study

Sample preparation

Following longitudinal sectioning, enamel overlying the dentine was removed with a diamond saw while leaving the enamel-dentine junction (EDJ) intact. Tools were cleaned with acetone between samples, and the resulting longitudinal sections placed into labelled microtubes prior to transverse sectioning. Longitudinally-sectioned teeth were embedded in a 1cm x 1cm x 10cm block composed of equal proportions of dental plaster and dental stone. Blocks contained two teeth each, positioned with root apices at the ends of the block. Once set, the block was sectioned on the transverse plane using a Buehler Isomet 1000 precision saw fitted with a diamond abrasive wafering blade (dimensions 102mm x 0.3mm), a cooling water bath, and a digital micrometer. Sections were cut into 1mm increments, beginning at the root apex, with the saw running at 100 rpm using Method 1 described by Beaumont *et al.* (2013) (Figure 5.8).

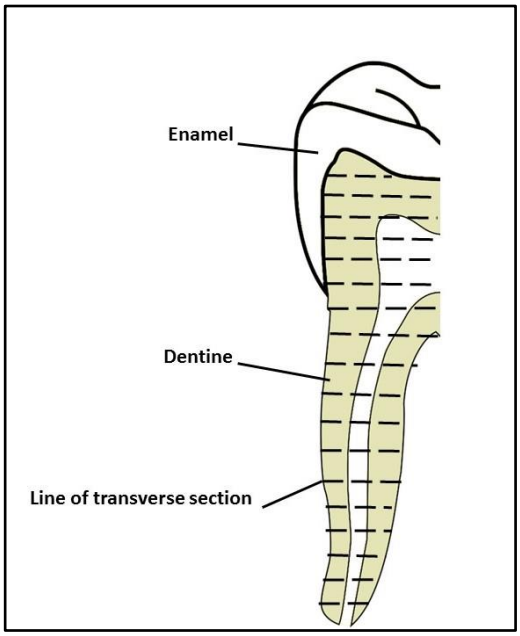

Figure 5.8 Transverse sectioning method used for incremental sampling following Method 1 of Beaumont *et al.* (2013).

Dry sections were then weighed prior to collagen extraction, which was accomplished following a modified Longin method (Longin, 1971). Dentine was demineralised with 0.5 M hydrochloric acid at 4°C over a period of approximately 3 weeks. The

demineralised sections were rinsed 3 times with deionized water and placed in sealed microtubes in a pH 3 hydrochloric acid solution at 70°C for 24 to 48 hours. The gelatinized samples were then centrifuged and decanted into preweighed vials for freeze-drying, with any remaining solids discarded. Once lyophilized, the resulting collagen was weighed into tin capsules for mass spectrometry.

5.2.1.ii Full Study

Following the success of the pilot study at producing preliminary data demonstrating the potential for variability, in terms of both isotope ratios and patterns of change in individuals within and between the two sites, a full study applying similar methods to a larger dataset was undertaken. The following section will outline both changes and continuity from the pilot phase of the study.

Sample selection

Fifty-five individuals were selected for the full phase of study at Edix Hill (n=27) and Littleport (n=28). Table 5.9 provides a demographic overview of the full sample (including pilot individuals). Detailed demographic information is found in Appendix 1.

Site	Nonadults	Females	Males	Indeterminate	Total
Edix Hill	9	9	11	0	29
Littleport	6	12	11	1	30
Total	15	21	22	1	**59**

Table 5.9 Demographic breakdown of individuals in the total (pilot and full phases) sample at Littleport and Edix Hill

Sampling was designed to select a cohort weighted towards the representation of adult survivors to demonstrate a measure of "normal" practice within that population, with some non-survivor juveniles additionally sampled to explore survivor versus non-survivor differences in patterning. The priority given to sampling adult individuals also allowed for potential identification of gender-based variation in feeding practices, due to the high degree of reliability of sex estimation in adult skeletal remains, especially where the pelvic elements are present (Meindl *et al.*, 1985; Spradley and Jantz, 2011). Whilst it is acknowledged that biological sex and gender are not interchangeable qualities within bioarchaeology, gendered care in childhood is being considered as a frequently-overlapping, linked variable to biological expression of sex at maturity. Adults of both sexes were therefore selected based on prior analysis and successful sex estimation, with any indeterminate individuals excluded. One exception was made to

this rule; at Littleport skeleton 4556, a very young adult of indeterminate sex, was included due to the identification of pathognomonic skeletal changes indicative of skeletal tuberculosis. This individual was selected in order to characterize any anomalies in profile that might be associated with a disease where nutritional stress is a known risk factor for, and also a direct outcome of, active infection (MacAllan *et al.*, 1998; Cegielski and McMurray, 2004).

However, while study design attempted to control for confounding variables such as survivorship which have been problematic for cross-sectional infant palaeonutrition studies in the past, the intent of the present study was not to replace the homogeneity of data drawn from non-survivors in the cross-sectional model with a potentially homogenous sample of survivors. Study design was therefore not designed to interrogate data to observe an assumed normative "weaning age," as has been a common goal of prior studies. Rather, an active expectation of variability drove selection of methods which would allow for characterisation of differences within and between communities, and between survivors and non-survivors, to obtain a wider understanding of the full range of responses to healthy and unhealthy children during this period. To this end, in addition to the adults selected to produce a "healthy" baseline for estimating population norms, a subsample of deciduous teeth (dm2) from non-surviving children (Table A1.1) was included at each site. Detailed demographic information regarding the adult sample from Littleport is found in Table A1.2, while Table A1.3 provides similar detail for the adults sampled from Edix Hill.

Sample preparation and analysis

Sample preparation for the full study paralleled the methods used in the pilot study, with all samples being prepared, from initial cleaning to completion of collagen extraction, in the Department of Archaeology at Durham University. Molar teeth from surviving adults in the sample (M1) were prepared by Method 1 of the Beaumont *et al.* (2013) protocol, as in the pilot. Some minor changes were made from the original prescribed method. Firstly, the study was expanded to include sampling of both maxillary and mandibular M1s, allowing a greater range of individuals to be sampled. Preference was given to the longest root (in upper molars, the palatal root), but due to preservation issues, neither upper nor lower molars were consistently used. Teeth were selected based on availability for study, with careful notation of which teeth were used to assign correct ages to data. In all cases, the most complete root present was selected. Secondly,

permanent molars were sectioned from crown to root apex, rather than beginning at the apex, as in the pilot. This small point of practice was altered due to the propensity of the final sections of the crown to detach from the block, being at that point shallowly embedded, preventing further sectioning and lowering resolution in the crown dentine, the region of greatest interest. Beginning the process of sectioning at the crown – cutting the plaster in thicknesses <1 mm until visual confirmation of the crown was obtained – was a simple alteration to practice which ensured that any potential disengagement from the block occurred during the final root portion of sectioning, where greater time averaging to data could be more comfortably entertained. Resulting 1 mm sections were individually weighed before being demineralised with 0.5 M hydrochloric acid at 4°C, refreshed as needed, over a period of several weeks.

The inclusion of deciduous (non-survivor) teeth in the full sample required the addition of Beaumont *et al.* (2013)'s Method 2 to the full phase of study in order to discretely sample the antenatally-forming portion of dentine. Following abrasion of the entire tooth to remove surface contaminants and half-sectioning and removal of the palatal root (where available), bulk enamel was removed, leaving the EDJ intact. Whole tooth sections were then demineralised with 0.5M hydrochloric acid at 4°C. Incremental sectioning took place when the tooth was mostly, but not fully, demineralised, to allow for precision cutting of sections, as sectioning at full demineralisation led to uneven cuts and tearing. Increments were cut beginning at the crown, with the dentine "horns," representing the antenatally-forming first ~0.5-1 mm of crown dentine, removed first. Subsequent sections were cut at 1 mm intervals. Cuspal dentine derived from the antenatally-forming first increment was rinsed three times in deionised water and directly lyophilised without denaturing or filtration, by the method described in Beaumont *et al.* (2014). A recent experimental study comparing the results of parallel analyses using bone pseudomorphs and the more rigorous method of demineralisation followed by denaturing and ultrafiltration found no significant difference in data produced (Sealy *et al.*, 2014). This suggests that for small samples, such as those produced by incremental sampling of dentine, the negative effects of denaturing and filtration on sample yield outweigh the potential benefit of these procedures.

Following demineralisation, 1 mm samples underwent collagen extraction via a modified Longin method (Longin, 1971). Demineralised increments were rinsed three times with deionised water and placed in sealed microtubes in a pH 3 hydrochloric acid

solution at 70°C for 24 to 48 hours. The gelatinized samples were then centrifuged and decanted into preweighed vials, with any remaining solids discarded, prior to lyophilisation. Following freeze-drying, the resulting collagen was weighed and yield percentages for each increment were calculated. All yielded collagen was weighed into tin capsules in duplicate for mass spectrometry at Bradford's Stable Light Isotope Facility.

5.2.2 Stable isotope mass spectrometry

Mass spectrometry was carried out in two separate facilities for the pilot and full phases of study, with the pilot phase analysis taking place at NIGL. Following a failure to secure funding to continue analyses at NIGL for the full study, the decision was taken to transfer the full study analyses to the University of Bradford's Department of Archaeological Sciences Stable Light Isotope Facility, with quality control measures in place to ensure consistent cross-comparability, including the use of calibration to international standards, and continuity of collagen preparation at Durham.

5.2.2.i Basic principles of mass spectrometry

Mass spectrometry is used to quantify the isotopic composition of sample materials. This is done by ionising analytes to produce differences in their mass-to-charge ratio, based on existing differences in the mass of isotopes (Gross, 2004). This leads to differing dynamics in a low-pressure system such as a mass spectrometer, which can be exploited to quantify these ions. Stable isotopes of carbon and nitrogen in archaeological materials, such as bioapatite (carbon only) and collagen (carbon and nitrogen), are analysed through isotope ratio mass spectrometry (IRMS). IRMS requires conversion of samples from a solid state (like collagen) to a gaseous state – in the case of collagen, into carbon dioxide and nitrogen gases. This is accomplished through combustion of samples at 1020°C in a compartment containing copper, nickel, and platinum wires, following which the combustion products are carried in a stream of helium over copper wires heated to 600°C to convert any remaining nitrogen oxides to pure nitrogen (Brown and Brown, 2011).

Following conversion to a purified gaseous state, the yielded carbon dioxide and nitrogen sample gases are introduced under high vacuum to an ionising filament, resulting in a focused beam of positively-charged ions (Ambrose, 1993). This beam is then accelerated through a flight tube where the ions are deflected by a curved magnetic field differentially based on their mass-to-charge ratio, allowing them to be directed to

different Faraday collectors (Figure 5.9). The collection of ions within each Faraday collector generates an electrical current which may be used to quantify the proportions of ions, and thus isotopes, within the sample (Brown and Brown, 2011). In addition to the analysis of sample materials, mass spectrometry is also used to analyse materials of known isotopic composition, or standards, to ensure the accuracy of measurement within each analytical run.

IRMS is commonly carried out in one of two forms: continuous flow (CFIRMS) or dual inlet (DI-IRMS) isotope ration mass spectrometry. In DI-IRMS, purified gaseous analytes are rapidly alternated with reference gases of known composition, CO_2 and N_2, through a system of valves, resulting in many comparative measurements of sample and reference composition. In CFIRMS, while purified gas produced from combustion of sample materials is also compared to reference gases of CO_2 and N_2, reference measurements occur in advance of sample introduction. CFIRMS is a more recent development in mass spectrometry than DI-IRMS, allowing greater efficiency than its predecessor (Oesselmann *et al.*, 2002), and is the mass spectrometry method used in both phases of the present study.

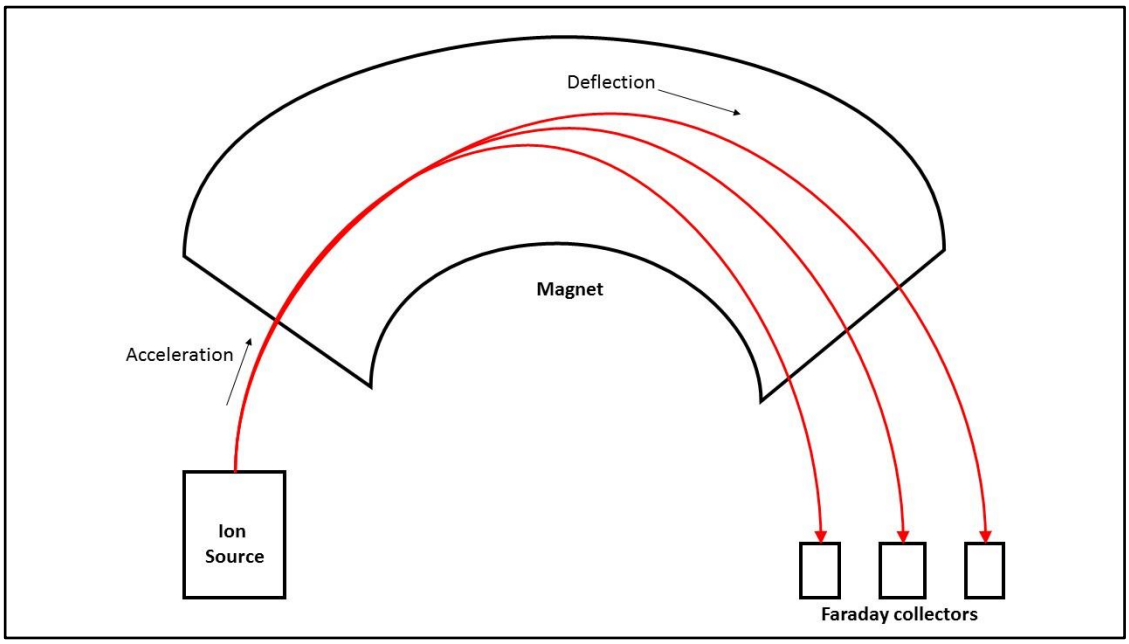

Figure 5.9 Basic components of an isotope ratio mass spectrometer

5.2.2.ii Measurement of samples

Pilot study

Mass spectrometry was carried out at the NERC Isotope Geosciences Laboratory (NIGL) at the British Geological Survey site in Keyworth using Continuous Flow

Isotope Ratio Mass Spectrometry (CFIRMS). An elemental analyser (Flash/EA) coupled to a Thermo Finnigan Delta^{Plus} XL isotope ratio mass spectrometer via a ConFlo III interface was employed for collagen analysis. After weighing samples into tin capsules, analyses were carried out in single measurements, with weighed replicates of reference material interspersed throughout the run. Empty tin capsules (blanks), were also included to correct for the effects of the capsules. Carbon and nitrogen stable isotope ratios were calibrated using in-house reference materials M1360p (powdered gelatine from BDH, expected values of −20.32 ‰ for $\delta^{13}C$, calibrated against IAEA CH7, and +8.12 ‰ for $\delta^{15}N$, calibrated against IAEA N-1 and N-2) and SADCOW (Sheffield Archaeology Department modern bovine collagen, expected values of 4.6 ‰ for $\delta^{15}N$ and −22.2 ‰ for $\delta^{13}C$). The analytical error for both carbon and nitrogen was ± 0.2 ‰ or better (1σ).

Full study

For the full phase of the study, mass spectrometry was carried out at the University of Bradford's Stable Light Isotope Laboratory in the School of Archaeological Sciences. Dentine collagen samples were weighed into tin capsules and measured in duplicate via CFIRMS using a Thermo Flash EA 1112 coupled to a Finnigan Delta^{Plus} XL isotope ratio mass spectrometer via a Conflo III interface. Where possible, sample size was targeted to the ideal range for the Finnigan, 0.5±0.1 mg. Collagen samples were interspersed within each run alongside blanks, internal standards and international standards. International standards measured were: IAEA 600 (caffeine, expected values of −27.77 ‰ for $\delta^{13}C$ and +1.0 ‰ for $\delta^{15}N$), IAEA CH3 (cellulose, expected value of −24.72 ‰ for $\delta^{13}C$), IAEA CH6 (sucrose, expected value of −10.45 ‰ for $\delta^{13}C$), IAEA N1 (ammonium sulphate, expected value of +0.43 ‰ for $\delta^{15}N$) and IAEA N2 (ammonium sulphate, expected value of +20.41 ‰ for $\delta^{15}N$). Internal reference materials analysed were: fish gelatine (expected values of −15.52 ‰ for $\delta^{13}C$ and +14.45 ‰ for $\delta^{15}N$), BLS (bovine liver standard, expected values of −21.59 ‰ for $\delta^{13}C$ and +7.65 ‰ for $\delta^{15}N$), and methionine (expected values of −26.60 ‰ for $\delta^{13}C$ and −3.00 ‰ for $\delta^{15}N$). The analytical error for both carbon and nitrogen was ± 0.2 ‰ or better (1σ).

5.2.2.iii Quality control parameters

Following extraction and analysis of samples, several measures of collagen quality are used to assess the extent of diagenetic (either degradation or contamination) effects on

collagen, and the acceptability of data obtained from these samples. Common parameters used as markers of acceptable quality include percentages of collagen yield by weight (Ambrose, 1990; van Klinken, 1999), measured carbon to nitrogen (C:N) ratios (DeNiro, 1987; van Klinken, 1999), and elemental mass percentages of carbon and nitrogen (%C and %N) within the sample (Ambrose, 1993; van Klinken, 1999). Each of these parameters will be briefly outlined in the sections following.

Yield of collagen

Collagen yield (expressed as a percentage by mass of the original sample) is used as a primary indicator of diagenesis and collagen quality. Modern bone contains approximately 22% collagen by weight, with steep climate-dependent decreases in collagen occurring after burial (van Klinken, 1999). Collagen is relatively robust, in terms of diagenetic alteration of isotopic composition, even in bones where significant diagenesis of bone mineral has occurred (Nelson *et al.*, 1986), with integrity being maintained until >99% of collagen is lost to degradation (Dobberstein *et al.*, 2009). Collagen yields of <1% of the original sample mass have been demonstrated to impact upon the other collagen quality assessment parameters, such as carbon to nitrogen atomic ratio and elemental mass percentages, with extreme values being more commonly observed in low collagen samples (Ambrose, 1990). Variable standards for the minimum collagen yield required have been proposed, with Ambrose (1993) placing a cautious lower limit of 1.2% and van Klinken (1999) recommending a more liberal boundary of 0.5-1% yield. The present study will take a middle path, using a 1% yield threshold for acceptability.

C:N atomic ratios

Carbon to nitrogen atomic ratios have also been argued to be an effective measure of contamination or degradation of collagen in archaeological samples. These ratios are calculated using the formula

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

and resulting ratios are compared to the expected ratio for diagenetically unaltered modern collagen to assess quality. The C:N ratio of modern collagen, estimated based on amino acid composition, is 3.21, while typically measured C:N ratios in well-preserved archaeological collagen samples range from 2.9-3.6 (DeNiro, 1985; Ambrose, 1993). Analyses of archaeological collagen producing ratios outside of this range are

routinely rejected as invalid by broad consensus (van Klinken, 1999; Brock *et al.*, 2010b). Some researchers have proposed more stringent standards of acceptability, with Ambrose (1993) suggesting that samples in the 3.4-3.6 range may be reflective of humic contamination, while van Klinken (1999) restricts the range of acceptability to 3.1-3.5 with judicious use of other collagen quality indicators. The present study utilises the van Klinken range of 3.1-3.5.

Carbon and nitrogen concentrations

As with collagen yield and C:N ratios, parameters of acceptable carbon and nitrogen concentrations are defined by their level of deviation from those measured in modern bone. While modern bone contains 3.5-4.5 % nitrogen by weight, well-preserved collagen from prehistoric bone or teeth will generally contain greater than 3% carbon and 1% nitrogen by weight (% gelatine in dry bone) (Ambrose, 1993). Ambrose (1990) states that modern bone collagen contains 15.3-47% carbon and 5.5-17.3% nitrogen by weight (% of combusted collagen). Acceptable composition ranges for archaeological bone collagen are estimated variously at 30-50% for carbon (Brock *et al.*, 2010a) and 11-16% (van Klinken, 1999), or up to 17.3% (Ambrose, 1990), for nitrogen. Deviation from these ranges may indicate the presence of contaminants. Percentages below the acceptable ranges may indicate inorganic contamination, whereas percentages above the optimal ranges suggest the presence of non-collagenous organics (van Klinken, 1999; Brock *et al.*, 2010b). The present study uses acceptable ranges of 30-50% for carbon and 11-17.3% for nitrogen, alongside other collagen quality indicators.

5.2.3 Assignment of chronological age to sample data

Following the method of Beaumont and Montgomery (2015), the chronological age-at-formation of each dentine increment was calculated as a proportion of overall tooth formation time. In the case of adults for whom the first mandibular permanent molars were sampled, dentine begins forming at approximately 0.3 years of age, with apex completion occurring at approximately 10 years of age (Al Qahtani *et al.*, 2010), yielding a formation time of 9.7 years. This formation time is subsequently divided by the number of increments yielded by 1 mm sampling to produce an estimate of formation time for each increment (i.e. if 20 increments are yielded by sampling, then this produces a computation of 9.7/20=0.485 years per increment for this M1). A range of chronological ages covered by each increment can then be calculated, beginning with the age of the earliest forming dentine. For instance, an M1 begins forming at

approximately 0.3 years, so the range represented by first increment will be calculated as being bounded by the endpoints of that increment. For an M1 containing 20×1 mm increments these endpoints will be 0.3 years (or 2.5 months) of age and 0.3+0.485=0.785 years (or 9.4 months) of age. Subsequent increments are calculated using the same formation time to produce chronological age calculations for each increment, and these ranges are used to plot data using a midpoint of this range. Maxillary M1s have a slightly shorter development period than their mandibular counterparts, forming between approximately birth and 9.5 years (Beaumont and Montgomery, 2015), and this variability in formation time was considered when assigning ages to mandibular or maxillary M1s.

The method for assigning age to deciduous teeth is similar, with the notable exception that the first ~0.5-1 mm of dentine (representing the cuspal dentine) in the dm2 represents a static developmental window of approximately 0.2 years to birth (Beaumont and Montgomery, 2015), requiring an anatomically-sensitive sampling method such as Beaumont *et al.* (2013)'s Method 2. All subsequent 1 mm increments were calculated in keeping with the method used for assigning age to the permanent teeth, as representing equal fractions of the remaining formation time. For dm2 teeth, this represents a period spanning from birth to approximately 3.5 years (Beaumont and Montgomery, 2015). In the case of both permanent and deciduous teeth, as increments represent a proportion of overall formation time, a greater number of increments indicates higher resolution, with deciduous teeth offering a higher level of time resolution than the permanent dentition (Beaumont *et al.*, 2015), due to the shorter total formation time relative to size.

5.2.4 Classification of profile type

Once plotted by age, data were analysed based on shape and patterning. These were categorised into three categories, based on overall appearance. The first category was parallel covariance, represented by changes to $\delta^{13}C$ and $\delta^{15}N$ which were in concert, or parallel, directionally. The second category was flat, which represented profiles which exhibited low variability in either $\delta^{13}C$, $\delta^{15}N$, or both, and an overall flat appearance. Previous research has suggested that dual flat profiles may be representative of homeostasis, while singly flat paired with labile profiles may be more indicative of stress or other processes (Beaumont, 2013; Craig-Atkins *et al.*, 2018). The third and final category was opposing covariance, which was defined as non-parallel variance in

carbon and nitrogen profiles in excess of analytical error, following characteristic patterns indicative of catabolic or anabolic shifts identified by multiple studies (Fuller et al., 2004; Fuller et al., 2005; Mekota et al., 2006; Neuberger et al., 2013). For the purposes of typology, classification was based on the predominant pattern observed in data from the first 3.5-4 years of life. This limitation was put in place to ensure a like-for-like comparison between chronological age of survivors and non-survivors, and to avoid introducing bias where the later contributions from survivors would be expected to represent a less stressful period of life.

5.2.5 Statistical analyses

Statistical analysis of relationships between the data resulting from this study and their relationships to palaeodemographic indicators was undertaken to better understand associations between demographic status, diet, and stress at these two sites. The null hypothesis consisted of the assertion that no relationship exists between palaeodemographic categories, skeletal pathology, and distribution of isotopic patterning. Due to potentially small cell counts, this hypothesis was tested using two-tailed (method of summing small p-values) Fisher's exact tests where appropriate to test independence of categorical variables such as location, presence or absence of stress markers such as cribra orbitalia, and presence or absence of opposing covariance in isotopic profile. The statistics were run using the XLSTAT 2018 statistical analysis interface with Excel. For all comparisons, an α-value < 0.05 was selected to indicate statistical significance, and a Holm-Bonferroni correction was applied to p-values.

6. Results

6.1 Introduction

This chapter presents the carbon and nitrogen isotope data results from the incremental dentine analyses of teeth from Edix Hill and Littleport. Data are presented here in graphical form, with collagen data tables for all individuals being in Appendix 1. The sections which follow are organised by the type of data (individual vs. grouped), by site, by tooth type, and additionally by sub-cohort (male vs. female) for permanent teeth. Developmental ages assigned to increments in this chapter are based on the method of Beaumont and Montgomery (2015).

6.2 Individual Results

Nearly all dentine samples produced sufficient collagen to meet the requirements for optimal sample size for the mass spectrometer (i.e. 0.5 mg). Acceptable C:N ratios were within the range of 3.1-3.5 (van Klinken, 1999), with percentages of carbon and nitrogen falling within accepted parameters (Ambrose, 1990; van Klinken, 1999; Brock *et al.*, 2010), indicating that collagen of acceptable quality had been recovered. Any analyses not falling within these ranges were excluded from inclusion in the full population data. Collagen yield from all samples retained was in the range of 7-19% of dry dentine weight. Data included here in the full analysis has therefore met all quality assurance parameters for good quality collagen, and poor collagen quality can be excluded as a factor in divergent covariance of $\delta^{13}C$ and $\delta^{15}N$ values. Data for $\delta^{13}C$ and $\delta^{15}N$ values were reported in parts per thousand (per mil or ‰), relative to standards of Vienna Pee Dee Belemnite (VPDB) for carbon and atmospheric air (AIR) for nitrogen.

6.2.1 Edix Hill results

Full data for all individuals sampled at Edix Hill appear in Tables A.1-A.29. For the total sample at Edix Hill, $\delta^{13}C$ values ranged from −21.1 to −19.6 ‰ (mean −20.4 ± 0.3 ‰), while $\delta^{15}N$ values fell between 8.6 and 15.6 ‰ (mean 11.4 ± 1.2 ‰).

6.2.1.i Deciduous second molar profiles

This section presents data for all nine non-surviving non-adults sampled at Edix Hill. Table 6.1 presents a summary of data for non-adults at Edix Hill. Incremental dentine data profiles for individuals will be presented graphically in the subsections below.

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
EH 12	−20.4 - −19.9‰	−20.1 ± 0.1‰	0.5‰	12.5 - 13.2‰	12.9 ± 0.2‰	0.7‰	Flat
EH 133	−21.1 - −20.1‰	−20.8 ± 0.4‰	1.0‰	12.1 - 13.4‰	12.8 ± 0.5‰	1.3‰	Flat
EH 178	−20.4 - −20.0‰	−20.2 ± 0.1‰	0.4‰	10.6 - 13.7‰	12.2 ± 1.2‰	3.1‰	PC
EH 352	−20.3 - −19.8‰	−20.1 ± 0.3‰	0.5‰	13.5 - 15.5‰	14.4 ± 0.7‰	2.0‰	OC
EH 447B	−20.5 - −19.9‰	−20.2 ± 0.2‰	0.6‰	11.0 - 13.2‰	12.3 ± 0.6‰	2.2‰	PC
EH 529	−20.7 - −19.6‰	−20.3 ± 0.4‰	1.1‰	11.2 - 13.5‰	12.4 ± 0.9‰	2.3‰	PC
EH 547B	−21.0 - −19.8‰	−20.5 ± 0.4‰	1.2‰	10.0 - 15.3‰	11.5 ± 1.7‰	5.3‰	OC
EH 587	−20.9 - −20.4‰	−20.7 ± 0.2‰	0.5‰	11.3 - 11.7‰	11.5 ± 0.2‰	0.4‰	Flat
EH 679	−20.2 - −19.9‰	−20.0 ± 0.2‰	0.3‰	11.4 - 12.1‰	11.6 ± 0.3‰	0.7‰	Flat
All	**−21.1 - −19.6‰**	**−20.4 ± 0.4‰**	**0.3 - 1.2‰**	**10.0 - 15.5‰**	**12.3 ± 1.2‰**	**0.4 - 5.3‰**	**-**

Table 6.1 Summary of incremental data for non-adult dm2 dentine at Edix Hill (OC= opposing covariance, PC= parallel covariance)

EH12

Full data for EH 12 are found in Table A.1 (Appendix 1). Figure 6.1 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 12 to produce a longitudinal profile.

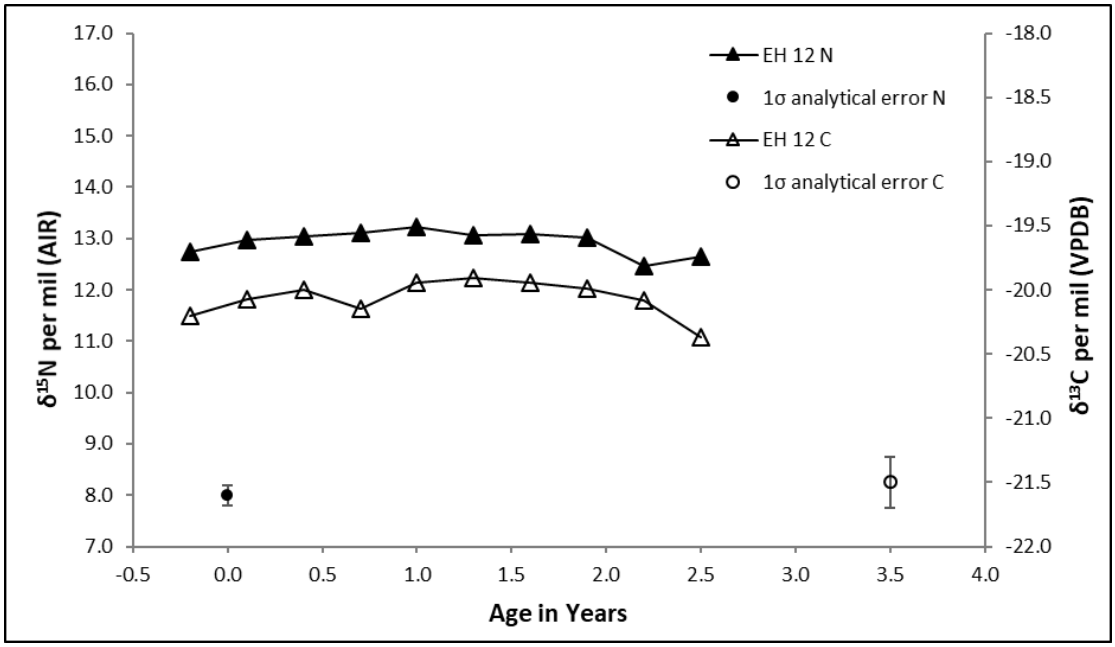

Figure 6.1 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 12

EH 133

Full data for EH 133 are found in Table A.2 (Appendix 1). Figure 6.2 shows $\delta^{13}C$ and $\delta^{15}N$ data for EH 133, plotted by age to produce a longitudinal profile.

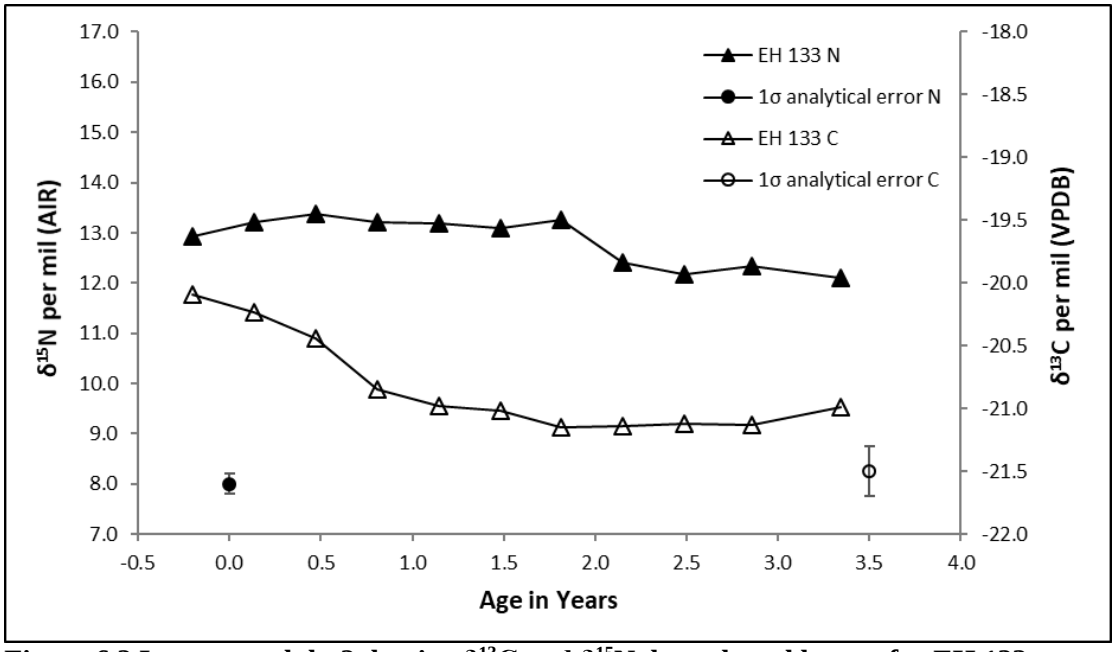

Figure 6.2 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 133

EH 178

Full data for EH 178 is found in Table A.3 (Appendix 1). Figure 6.3 shows the longitudinal profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 178.

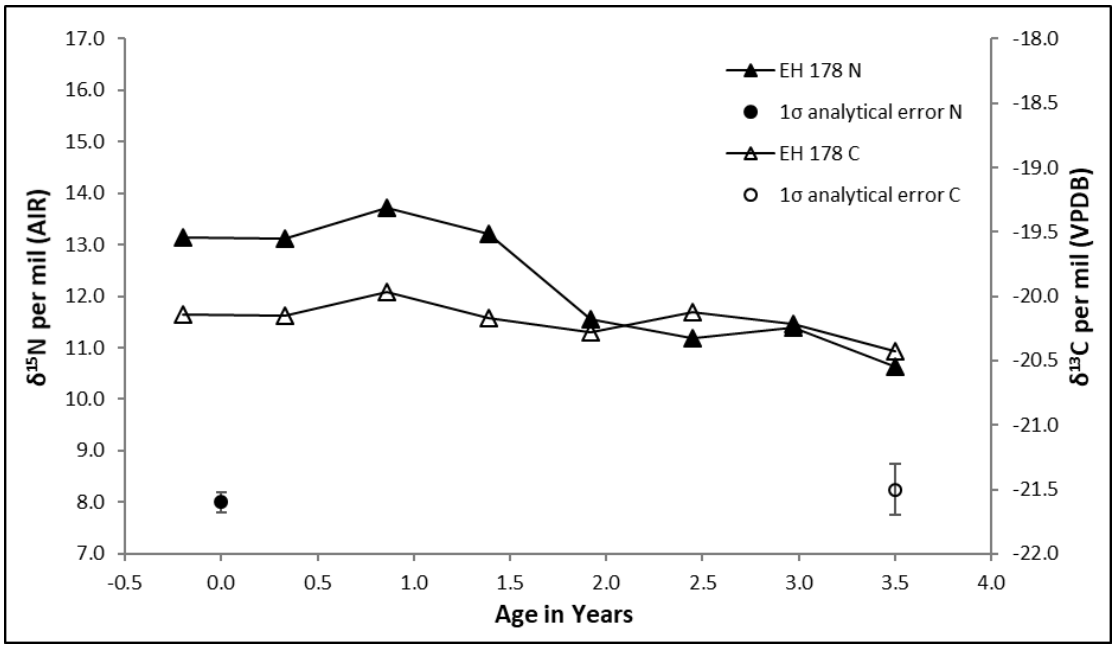

Figure 6.3 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 178

EH 352

Full data for EH 352 are found in Table A.4 (Appendix 1). Figure 6.4 shows the longitudinal profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 352.

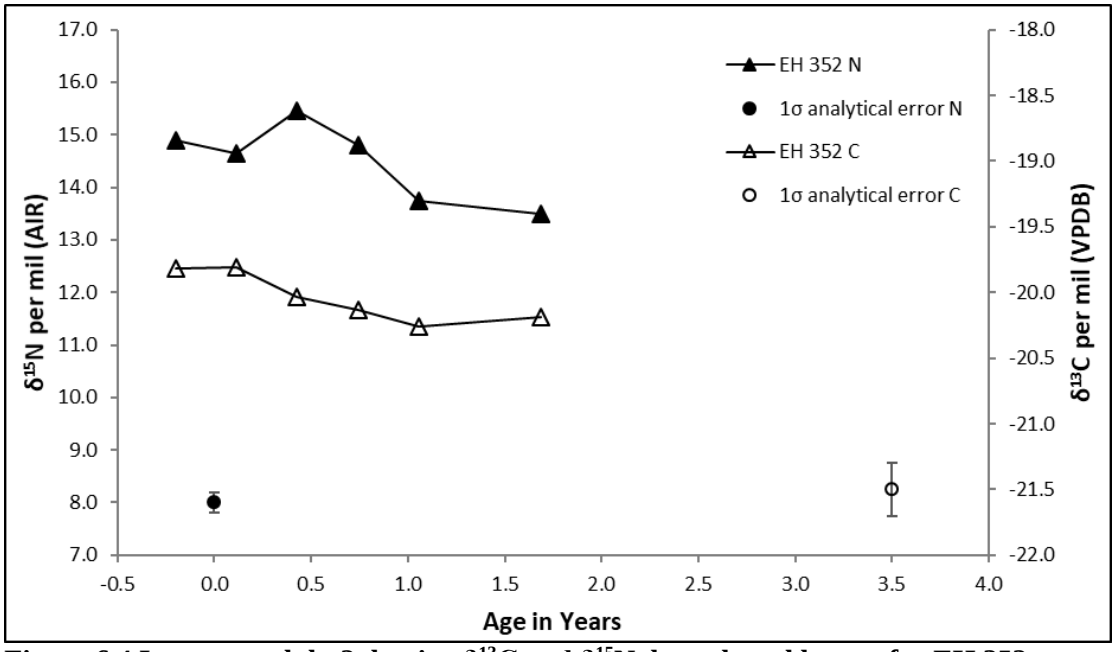

Figure 6.4 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 352

EH 447B

Full data for EH 447B are found in Table A.5 (Appendix 1). Figure 6.5 shows profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 447B.

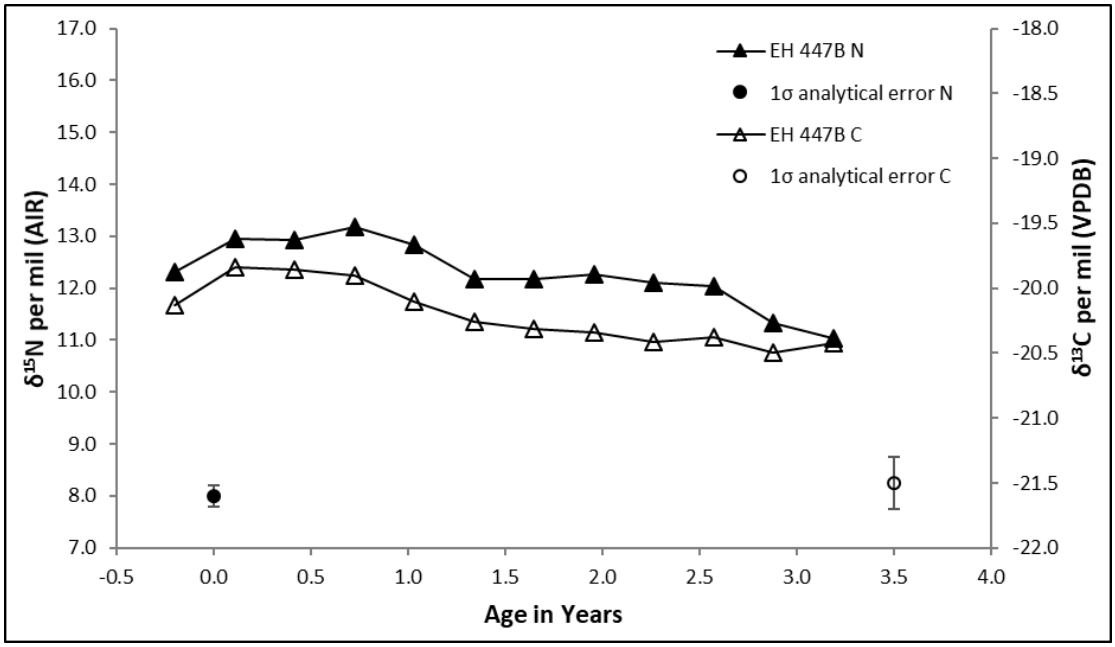

Figure 6.5 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 447B

EH 529

Full data for EH 529 are found in Table A.6 (Appendix 1). Figure 6.6 shows the longitudinal profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 529.

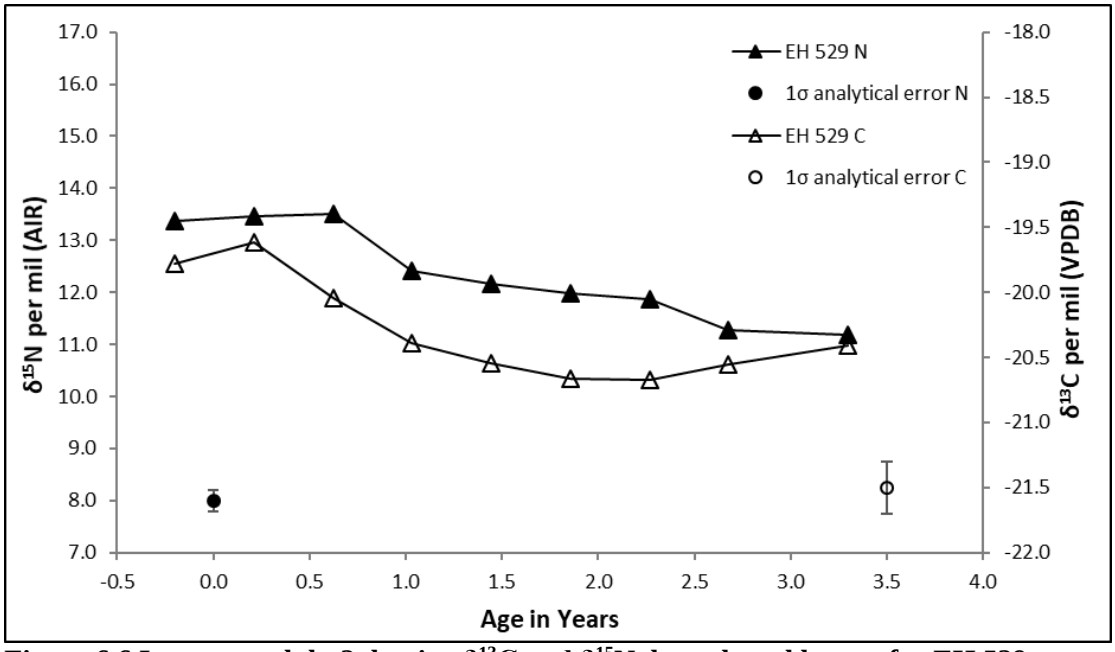

Figure 6.6 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 529

EH 547B

Full data for EH 547B are found in Table A.7 (Appendix 1). Figure 6.7 shows the longitudinal profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 547B.

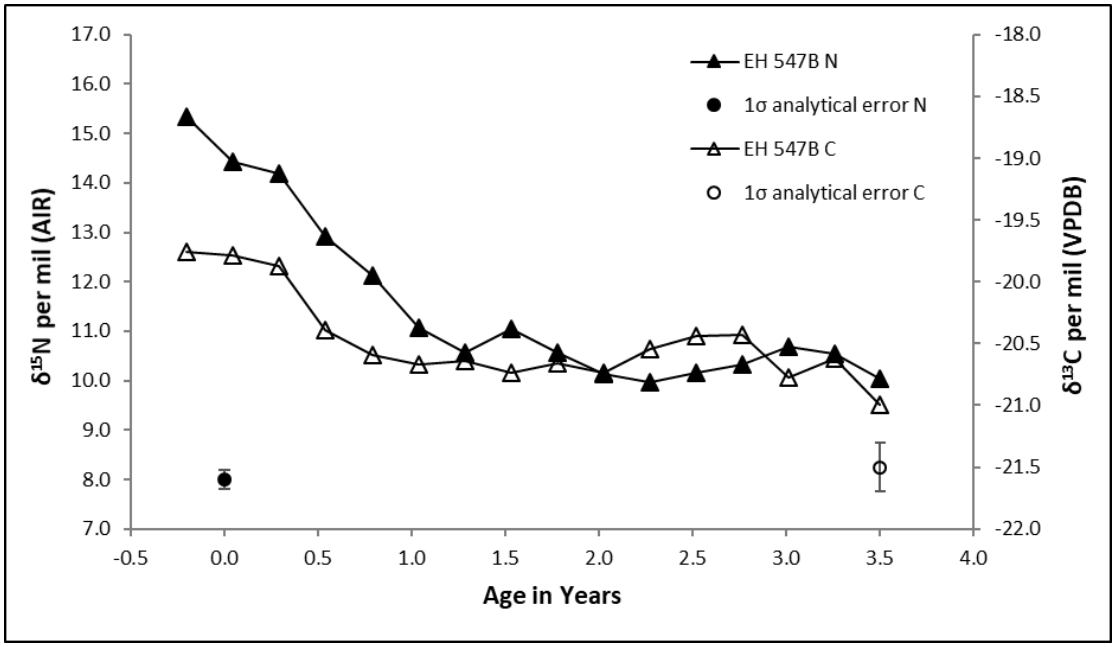

Figure 6.7 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 547B

EH 587

Full data for EH 587 are found in Table A.8 (Appendix 1). Figure 6.8 shows the longitudinal profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 587.

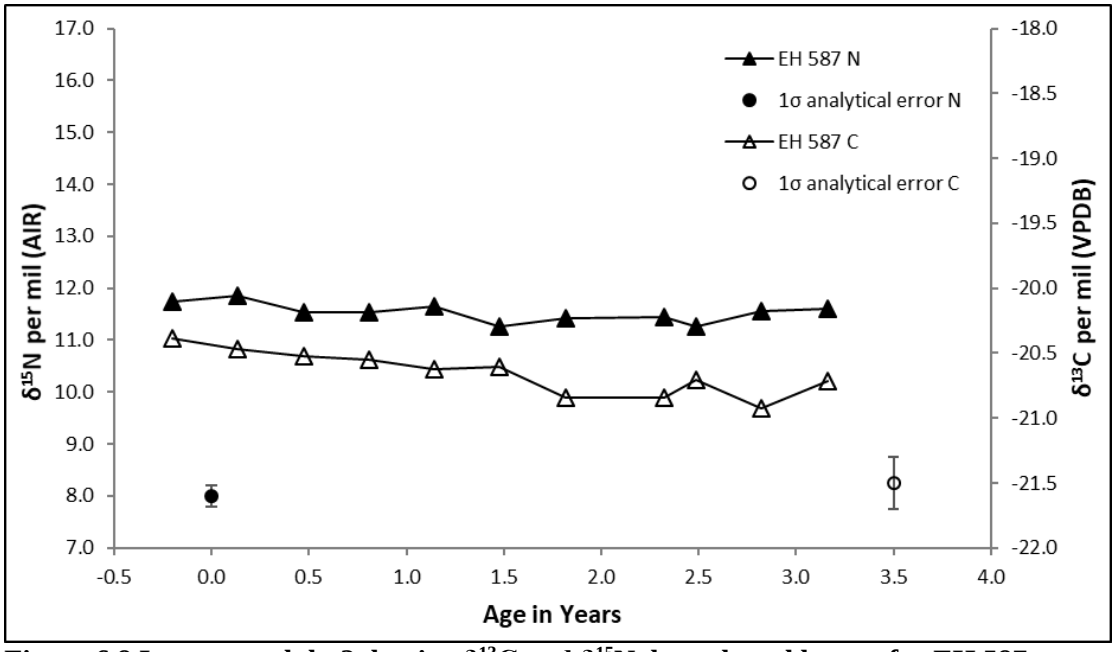

Figure 6.8 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 587

EH 679

Full data for EH 679 are found in Table A.9 (Appendix 1). The longitudinal profile of $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 679 appears in Figure 6.9.

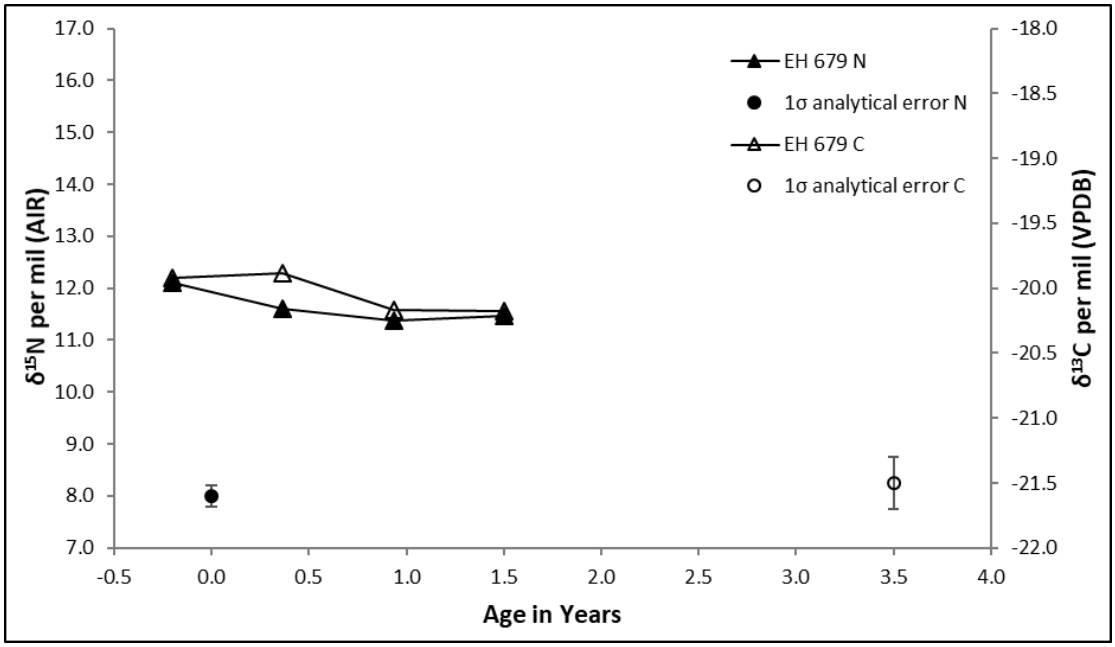

Figure 6.9 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 679

6.2.1.ii Permanent first molar profiles

This section presents data for M1 teeth at Edix Hill. Dentine collagen $\delta^{13}C$ values of the M1 sample fell within a range of −21.1 to −19.6 ‰ (mean −20.4 ± 0.2 ‰), while $\delta^{15}N$ values ranged from 8.6 to 15.6 ‰ (mean 11.1 ± 1.1 ‰). Intra-individual variation ranged from 0.4-1.2 ‰ for $\delta^{13}C$ and 0.6-4.8 ‰ for $\delta^{15}N$ over the course of M1 tooth development in adults sampled at Edix Hill. Incremental dentine data profiles for individuals will be presented graphically in the subsections below (Figures 6.10-6.29).

Males

This section presents M1 data for adult males at Edix Hill. Table 6.2 presents a summary of M1 data for adult males at Edix Hill. Individual data profiles for Edix Hill males will be presented below.

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
EH 11	−20.5 - −19.9‰	−20.2 ± 0.2‰	0.6‰	10.5 - 11.7‰	11.3 ± 0.3‰	1.2‰	Flat
EH 33	−20.8 - −20.0‰	−20.3 ± 0.3‰	0.8‰	9.0 - 10.3‰	9.6 ± 0.3‰	1.3‰	Flat
EH 112	−20.6 - −20.0‰	−20.3 ± 0.2‰	0.6‰	9.6 - 11.2‰	10.1 ± 0.5‰	1.6‰	PC
EH 125	−20.7 - −20.1‰	−20.5 ± 0.2‰	0.6‰	11.1 - 13.3‰	11.7 ± 0.6‰	2.2‰	Flat
EH 146	−20.5 - −19.6‰	−20.3 ± 0.3‰	0.9‰	10.4 - 15.2‰	11.4 ± 1.4‰	4.8‰	PC
EH 198	−20.6 - −20.2‰	−20.4 ± 0.1‰	0.4‰	10.1 - 12.2‰	11.0 ± 0.6‰	2.1‰	Flat
EH 300A	−20.5 - −20.0‰	−20.3 ± 0.2‰	0.5‰	10.2 - 14.1‰	10.9 ± 1.0‰	3.9‰	Flat
EH 453	−21.1 - −19.9‰	−20.3 ± 0.3‰	0.6‰	11.2 - 11.8‰	11.6 ± 0.2‰	1.2‰	OC
EH 578	−20.2 - −21.1‰	−20.4 ± 0.2‰	0.9‰	9.8 - 12.6‰	10.5 ± 0.9‰	2.8‰	OC
EH 626B	−20.7 - −20.0‰	−20.4 ± 0.2‰	0.7‰	10.9 - 13.5‰	11.7 ± 0.7 ‰	2.6‰	PC
EH 727	−20.6 - −20.2‰	−20.4 ± 0.1‰	0.4‰	10.2 - 14.1‰	11.6 ± 1.1‰	3.9‰	OC
All	**−21.1 - −19.6‰**	**−20.3 ± 0.2‰**	**0.4-1.2‰**	**9.0 - 15.2‰**	**11.0 ± 1.0‰**	**0.6-4.8‰**	**-**

Table 6.2 Summary of incremental data for adult male M1 dentine at Edix Hill (OC= opposing covariance, PC= parallel covariance)

EH 11

Full data for EH 11 are found in Table A.10 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 11 appear in Figure 6.10.

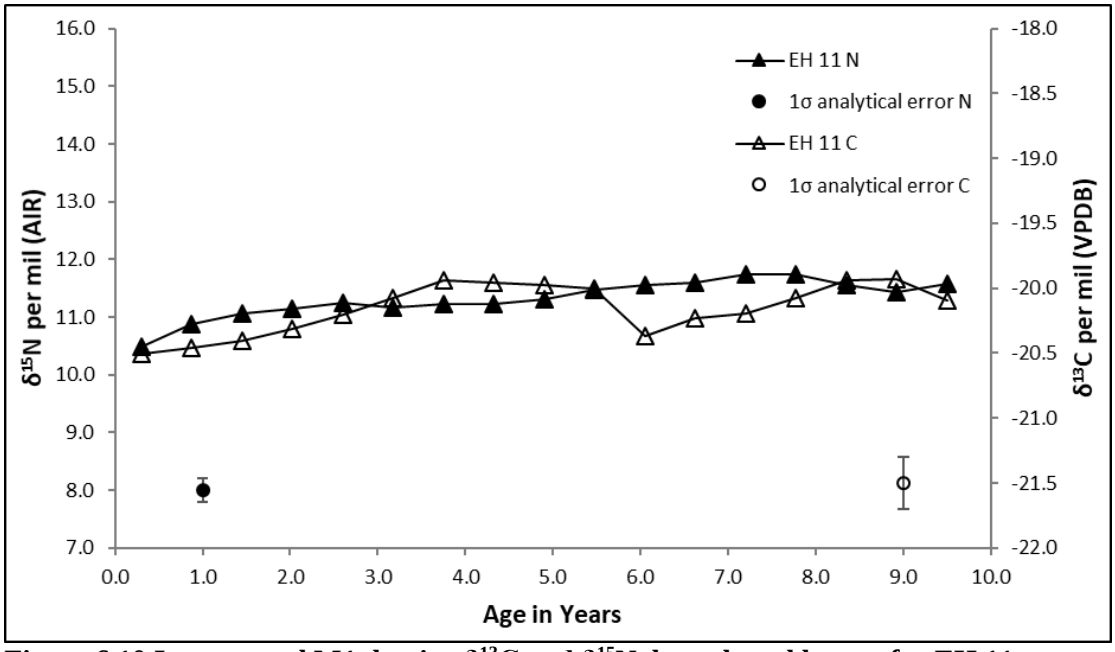

Figure 6.10 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 11

EH 33

Full data for EH 33 are found in Table A.11 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 33 appear in Figure 6.11.

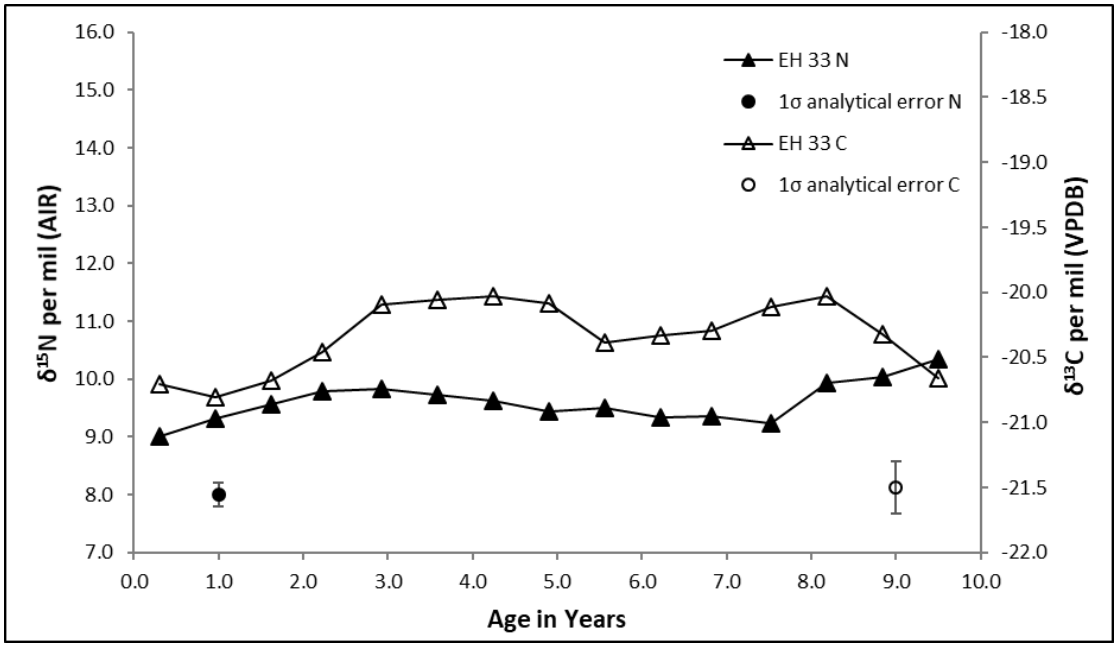

Figure 6.11 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 33

EH 112

Full data for EH 112 are found in Table A.12 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 112 appear in Figure 6.12.

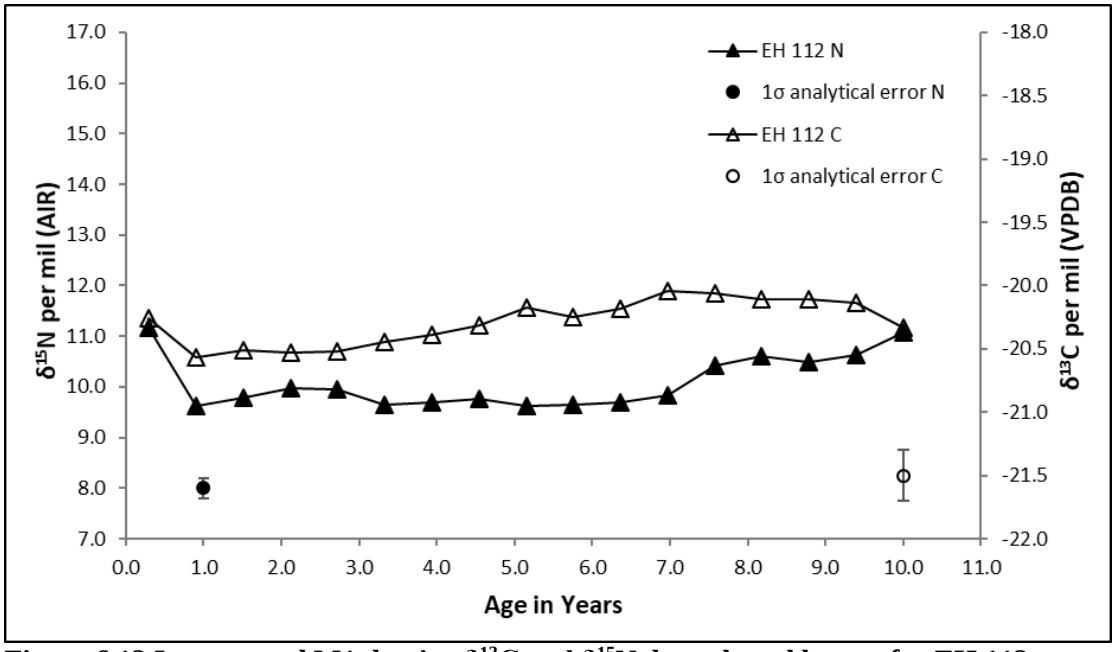

Figure 6.12 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 112

EH 125

Full data for EH 125 are found in Table A.13 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 125 appear in Figure 6.13.

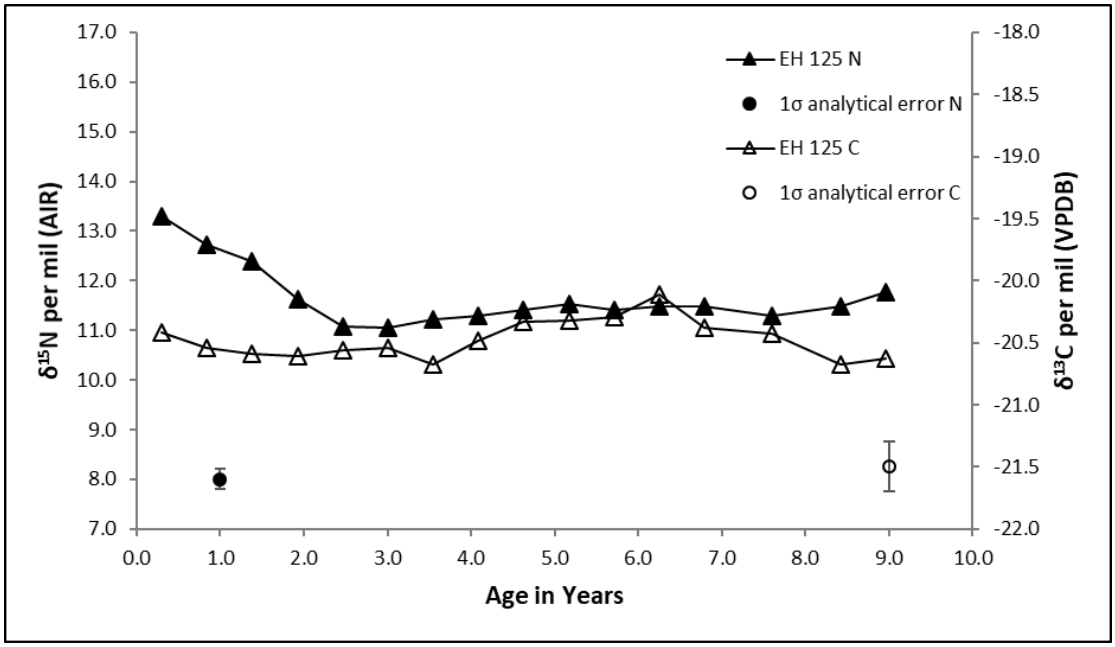

Figure 6.13 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 125

EH 146

Full data for EH 146 are found in Table A.14 (Appendix 1). Figure 6.14 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 146.

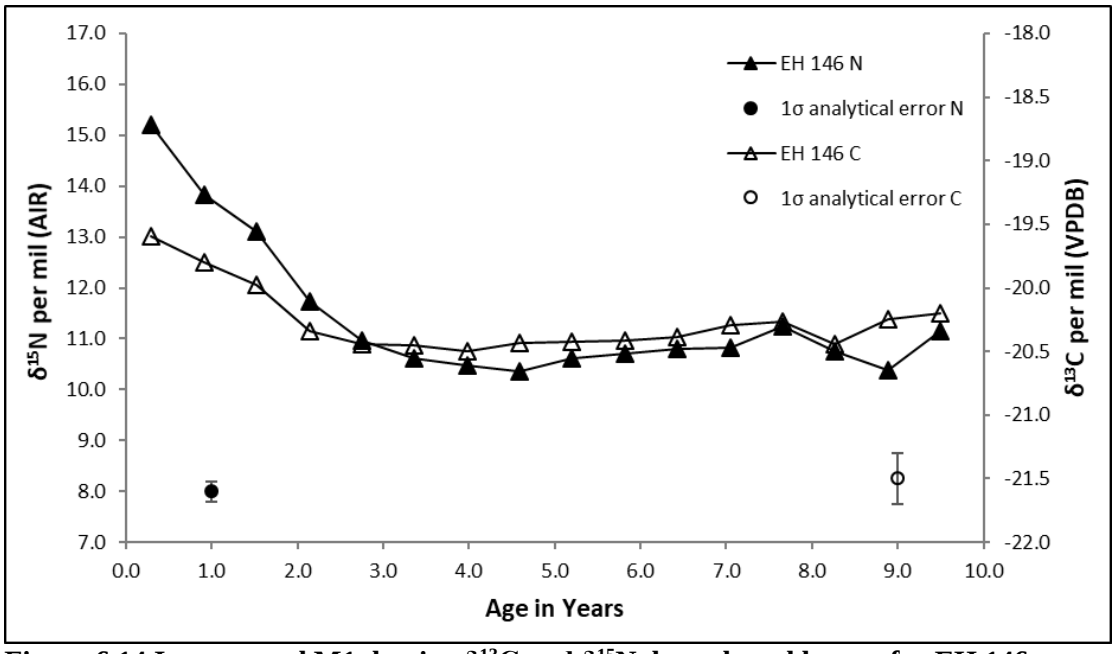

Figure 6.14 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 146

EH 198

Full data for EH 198 are found in Table A.15 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 198 appear in Figure 6.15.

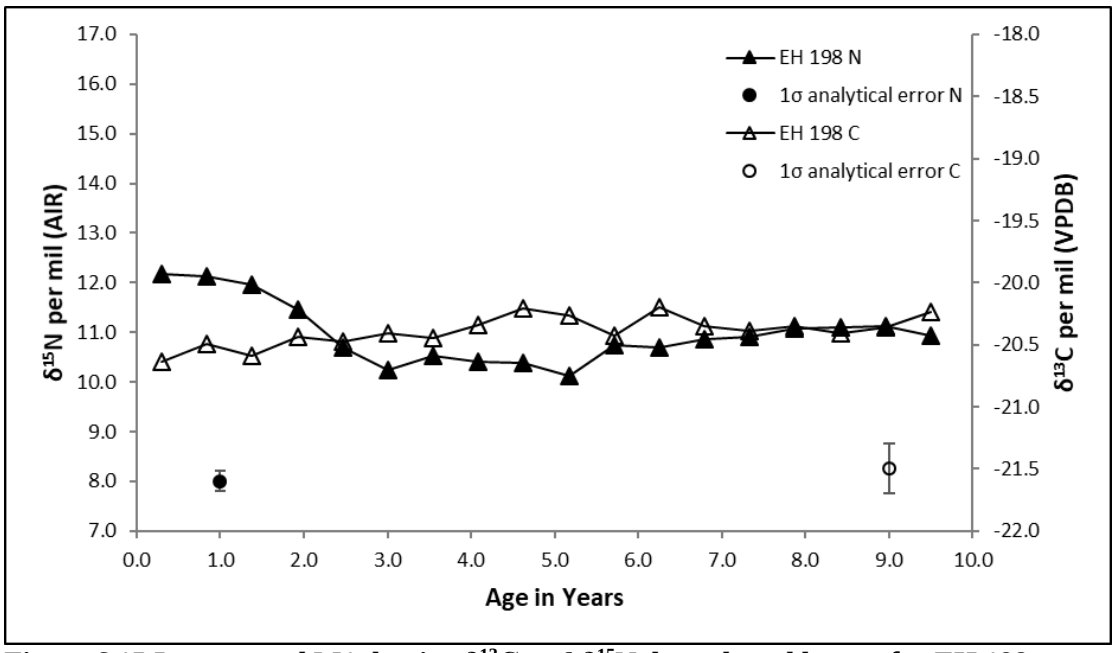

Figure 6.15 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 198

EH 300A

Full data for EH 300A are found in Table A.16 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 300A appear in Figure 6.16.

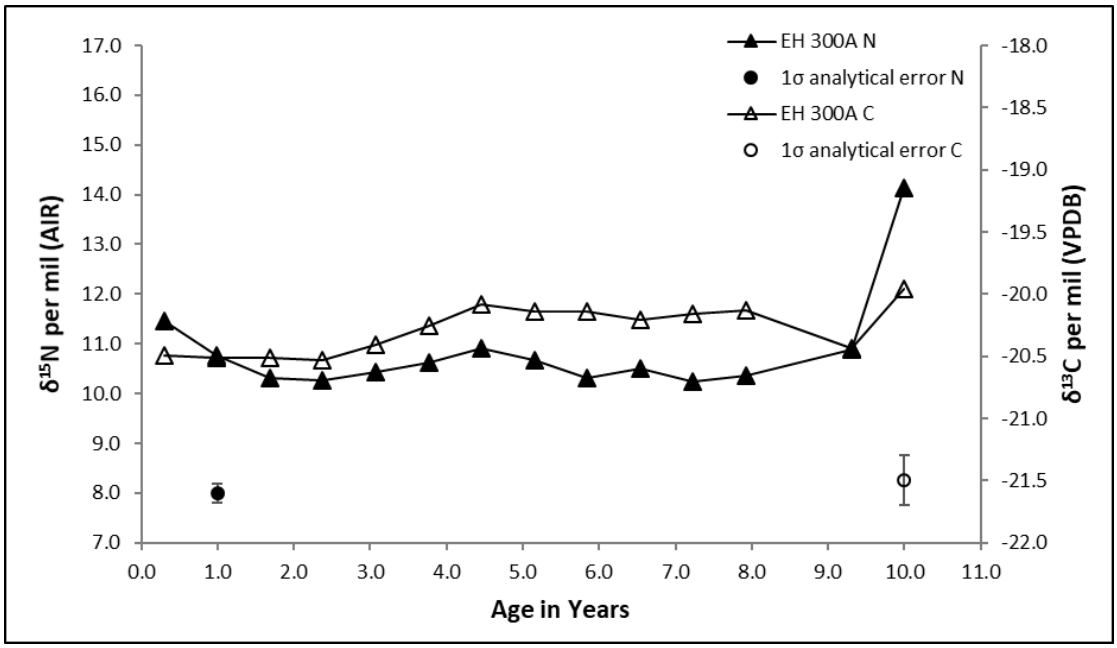

Figure 6.16 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 300A

EH 453

Full data for EH 453 are found in Table A.17 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 453 appear in Figure 6.17.

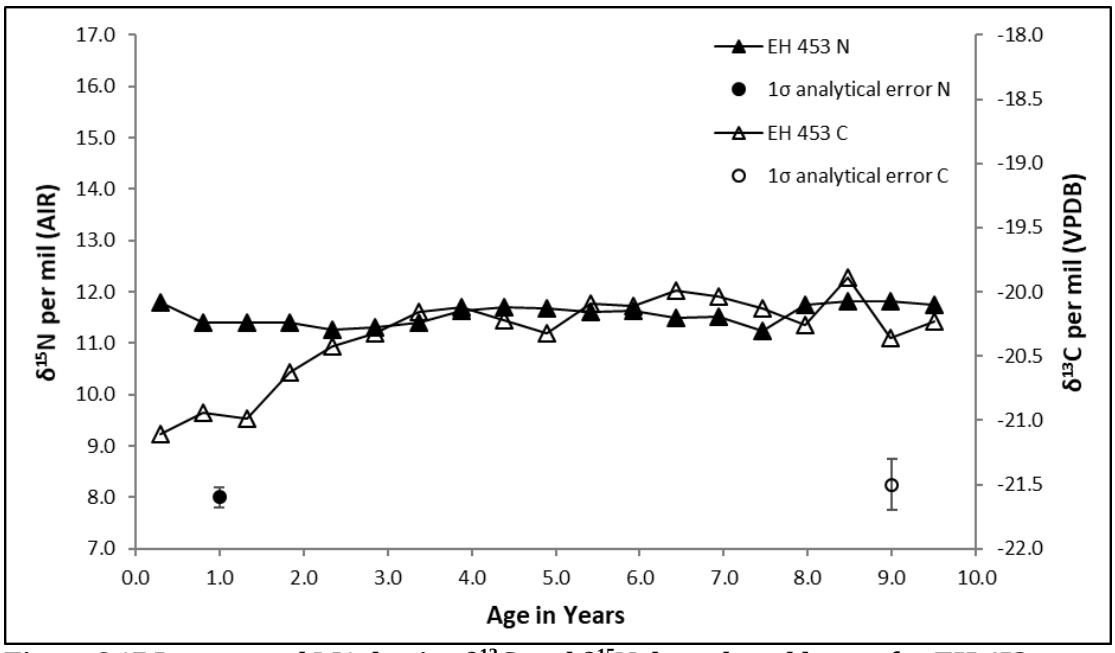

Figure 6.17 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 453

EH 578

Full data for EH 578 are found in Table A.18 (Appendix 1). Figure 6.18 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 578.

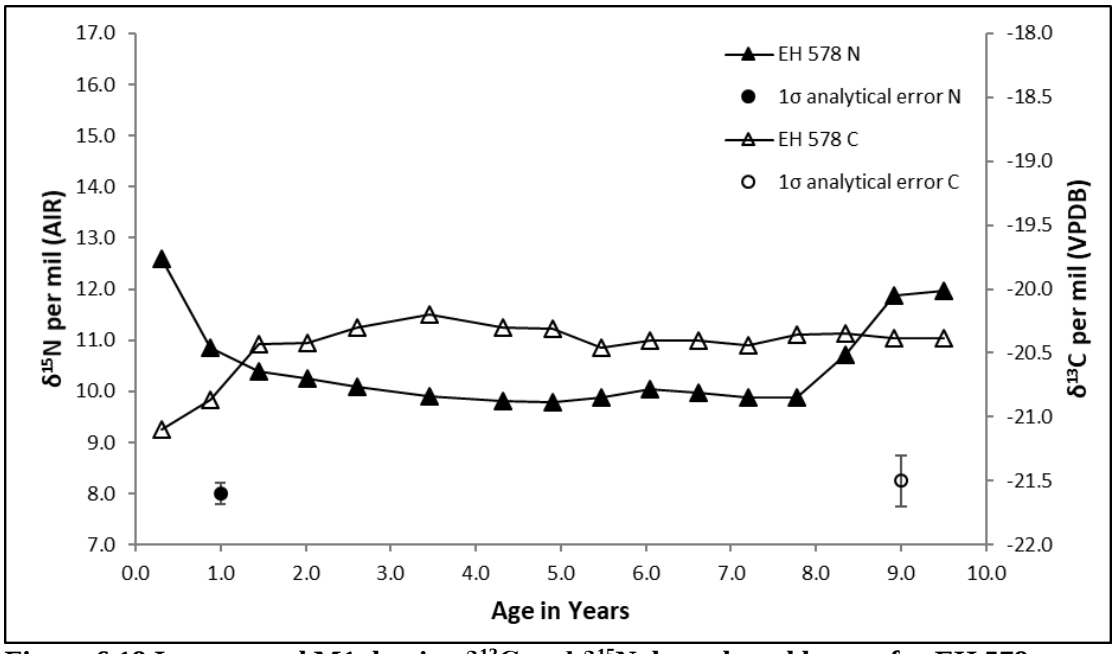

Figure 6.18 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 578

EH 626B

Full data for EH 626B are found in Table A.19 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 626B appear in Figure 6.19.

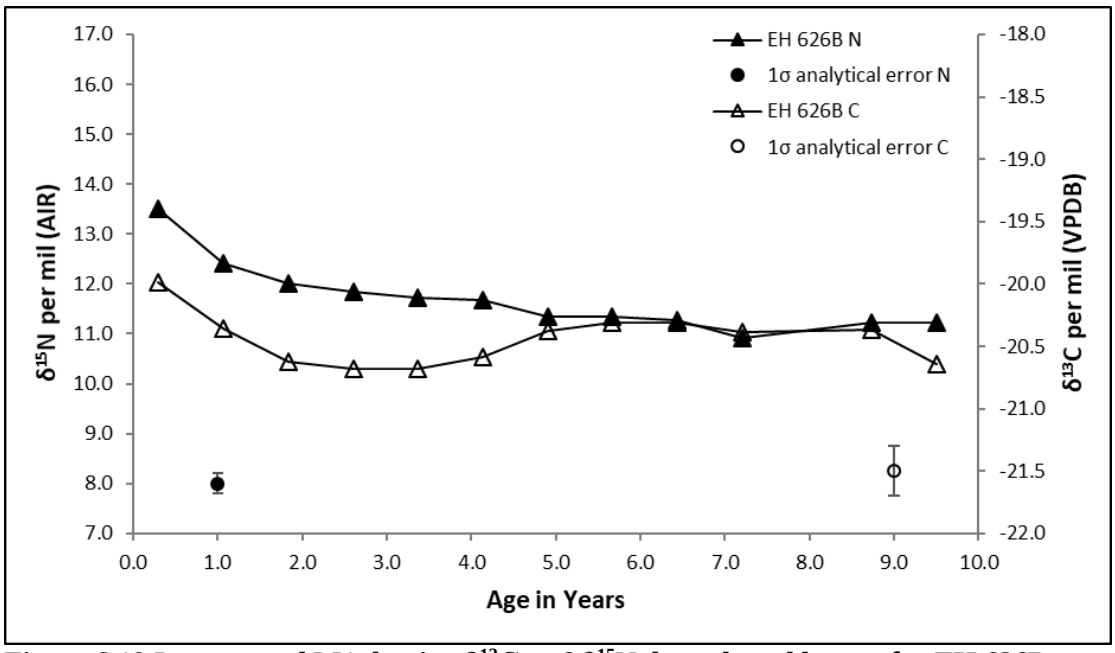

Figure 6.19 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 626B

EH 727

Full data for EH 727 are found in Table A.20 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 727 appear in Figure 6.20.

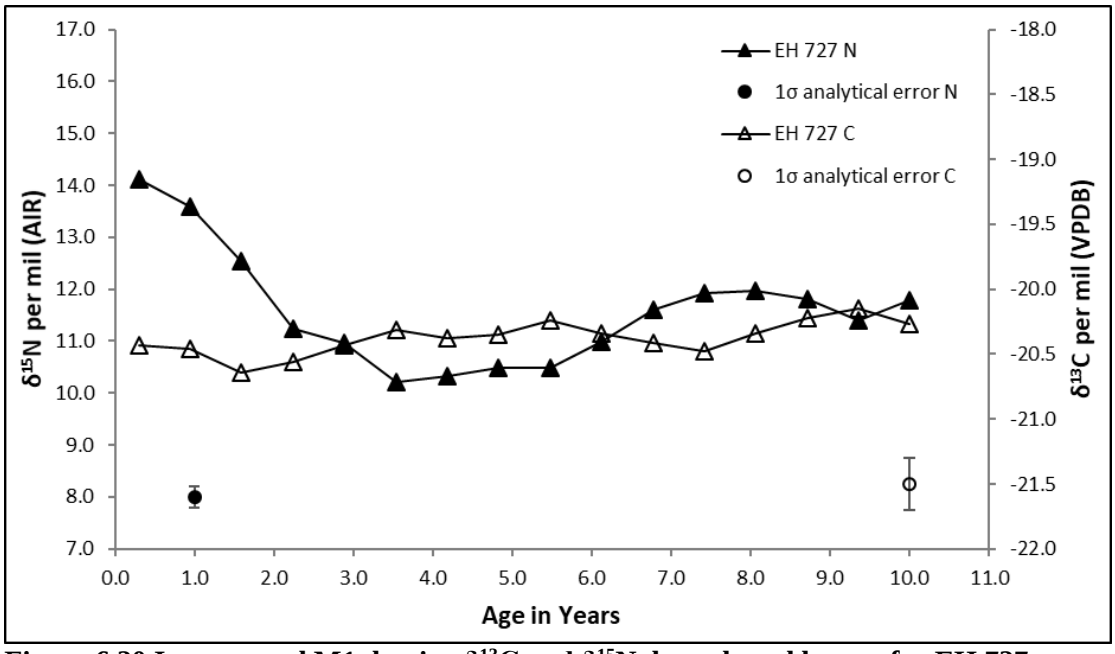

Figure 6.20 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 727

Females

This section presents M1 data for adult females sampled at Edix Hill. Table 6.3 summarises M1 data for adult females at Edix Hill. Individual data profiles for Edix Hill females will be presented in the subsections following.

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
EH 4	−20.9 - −20.2‰	−20.5 ± 0.3‰	0.7‰	11.5 - 14.0‰	12.3 ± 0.7‰	2.5‰	PC
EH 9	−21.0 - −20.0‰	−20.5 ± 0.4‰	1.0‰	10.0 - 13.6‰	10.7 ± 1.0‰	3.6‰	OC
EH 20B	−20.7 - −19.9‰	−20.5 ± 0.2‰	0.8‰	10.9 - 15.6‰	12.3 ± 1.4‰	4.7‰	PC
EH 45	−20.9 - −19.9‰	−20.2 ± 0.3‰	1.0‰	9.7 - 13.2‰	10.5 ± 1.0‰	3.5‰	OC
EH 359	−20.7 - −20.2‰	−20.4 ± 0.1‰	0.5‰	8.6 - 13.1‰	9.6 ± 1.0‰	4.5‰	PC
EH 440A	−20.6 - −20.2‰	−20.4 ± 0.1‰	0.4‰	10.9 - 12.7‰	11.6 ± 0.6‰	1.8‰	PC
EH 547A	−21.0 - −19.9‰	−20.3 ± 0.3‰	1.1‰	11.3 - 14.2‰	11.9 ± 0.8‰	2.9‰	PC

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
EH 626A	−20.9 - −20.3‰	−20.5 ± 0.2‰	0.6‰	11.3 - 12.7‰	11.7 ± 0.5‰	1.4‰	OC
EH 726	−20.7 - −20.2‰	−20.4 ± 0.1‰	0.5‰	10.2 - 12.8‰	10.8 ± 0.6‰	2.6‰	Flat
All	**−21.0 - −19.9‰**	**−20.4 ± 0.3‰**	**0.4-1.1‰**	**8.6 - 15.6‰**	**11.2 ± 1.2‰**	**1.8-4.7‰**	**-**

Table 6.3 Summary of incremental data for adult female M1 dentine at Edix Hill (OC= opposing covariance, PC= parallel covariance)

EH 4

Full data for EH 4 are found in Table A.21 (Appendix 1). $\delta^{13}C$ and for $\delta^{15}N$. $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 4 appear in Figure 6.21.

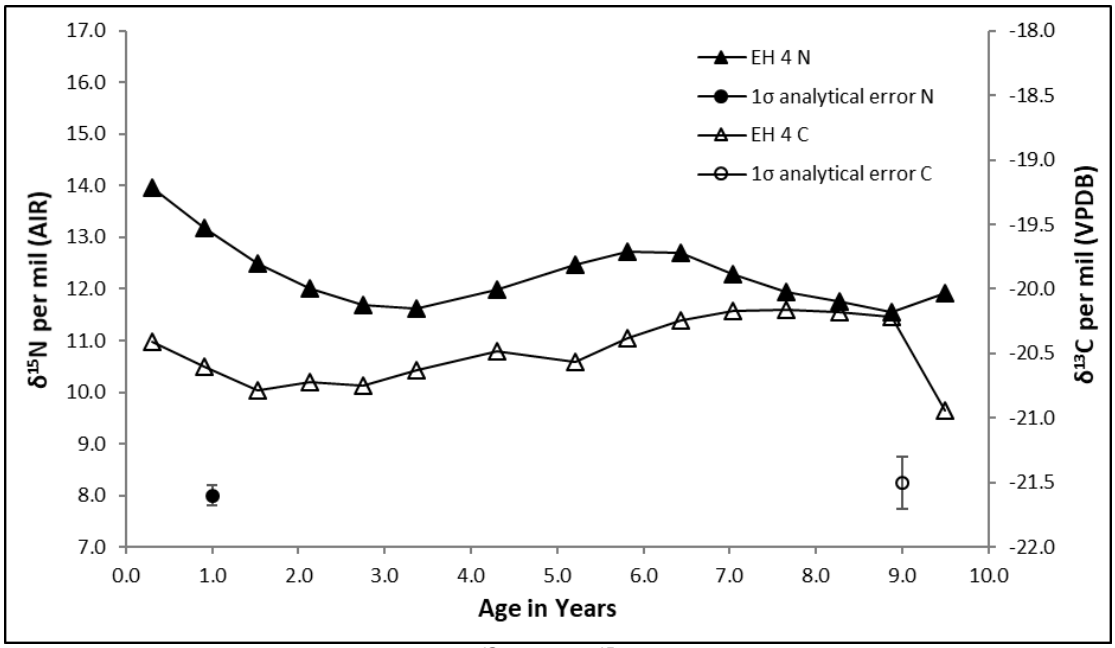

Figure 6.21 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 4

EH 9

Full data for EH 9 are found in Table A.22 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 9 appear in Figure 6.22.

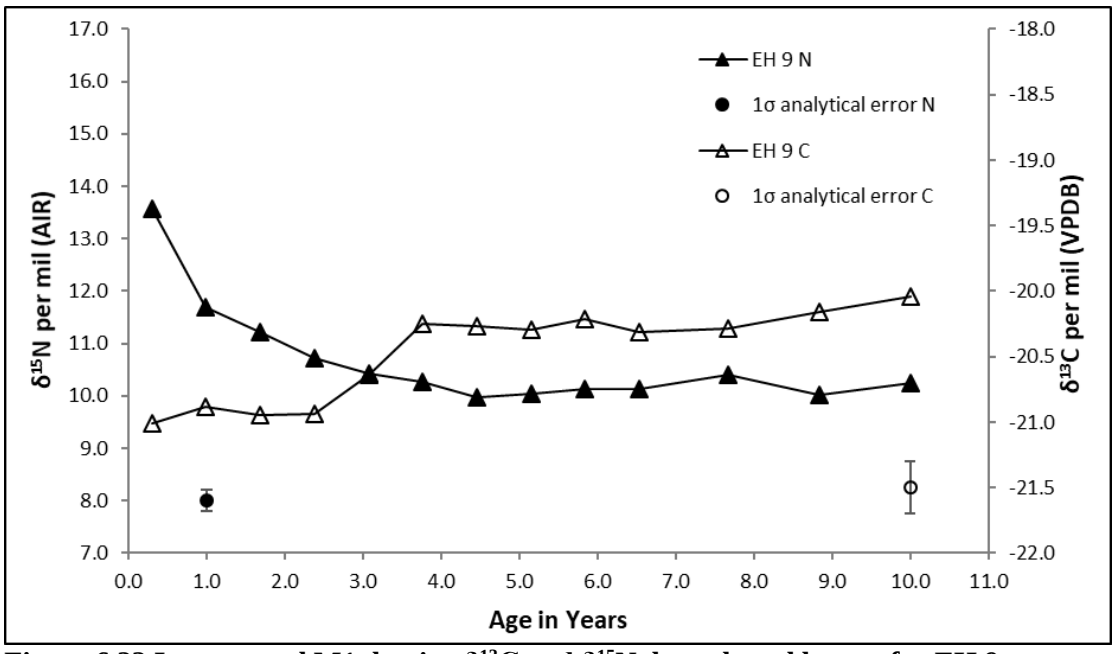

Figure 6.22 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 9

EH 20B

Full data for EH 20B are found in Table A.23 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 20B appear in Figure 6.23.

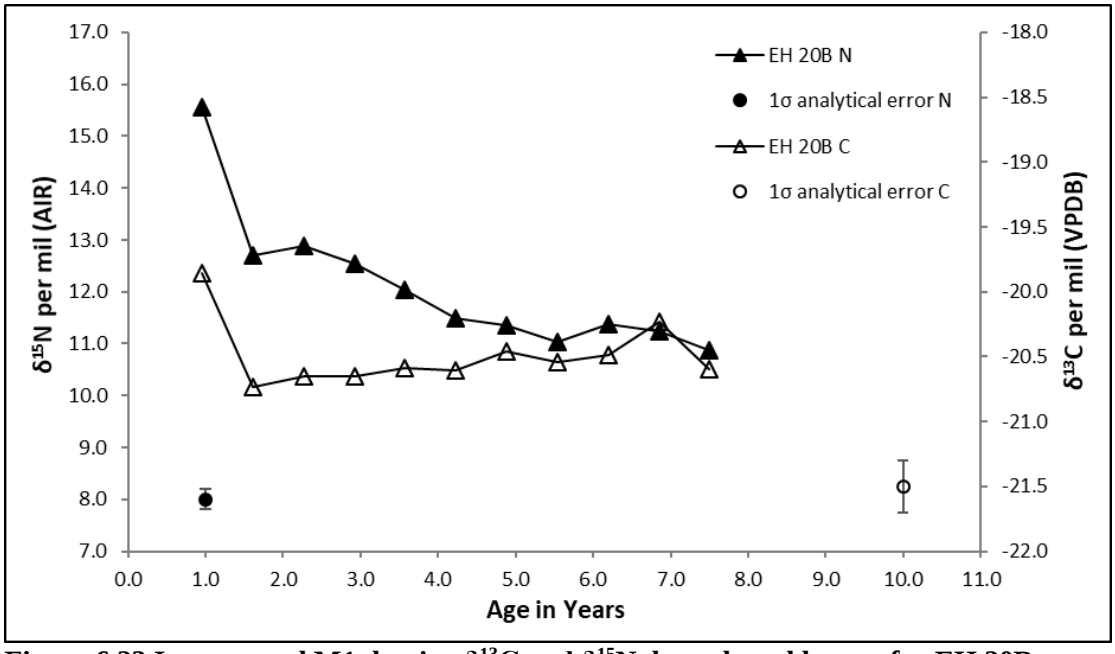

Figure 6.23 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 20B

EH 45

Full data for EH 45 are found in Table A.24 (Appendix 1). Figure 6.24 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 45.

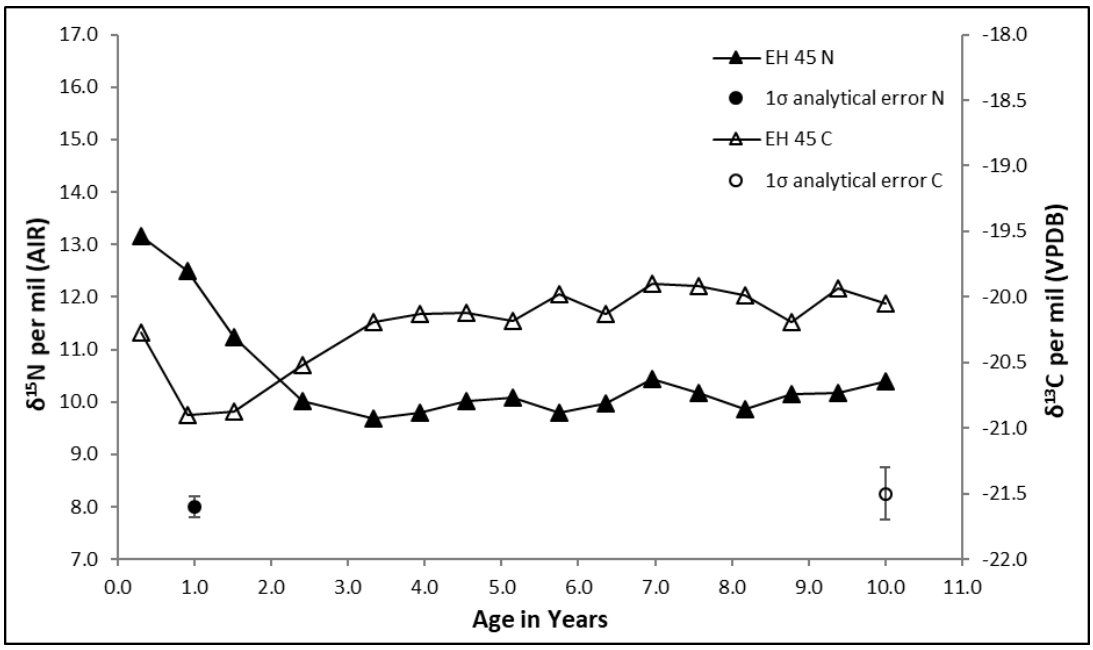

Figure 6.24 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 45

EH 359

Full data for EH 359 are found in Table A.25 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 359 appear in Figure 6.25.

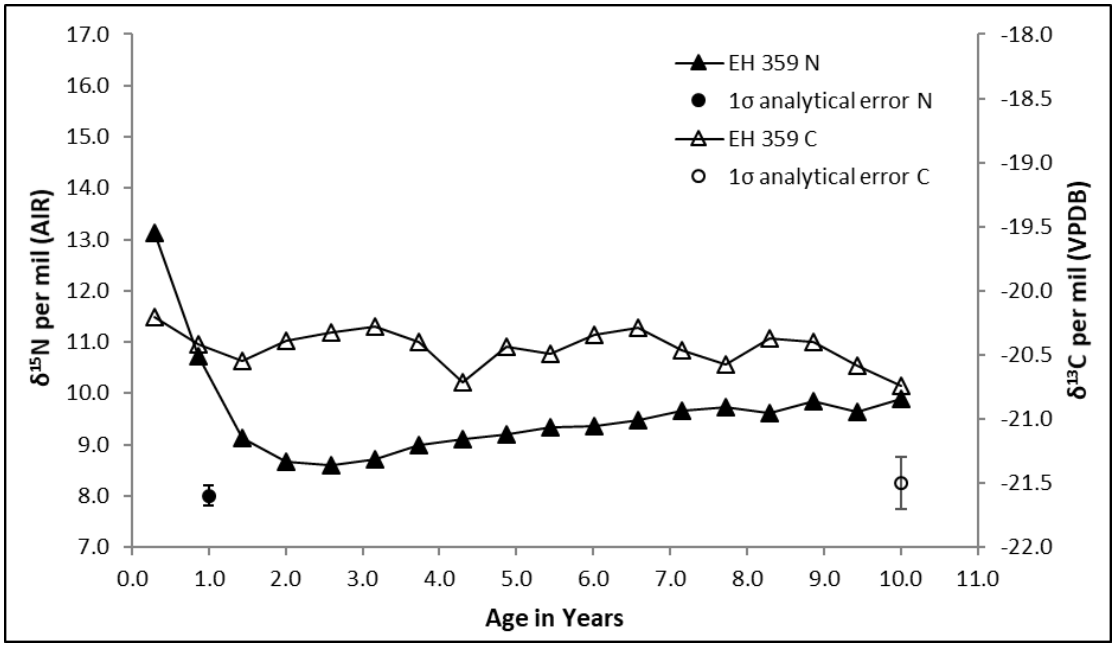

Figure 6.25 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 359

EH 440A

Full data for EH 440A are found in Table A.26 (Appendix 1). Figure 6.26 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 440A.

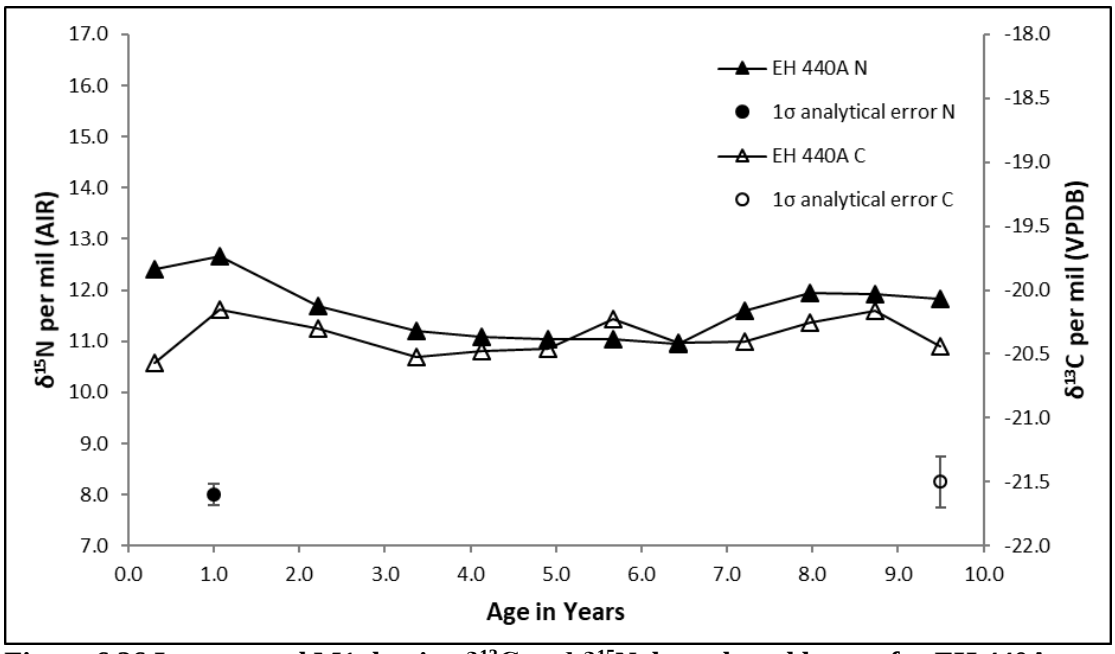

Figure 6.26 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 440A

EH 547A

Full data for EH 547A are found in Table A.27 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 547A appear in Figure 6.27.

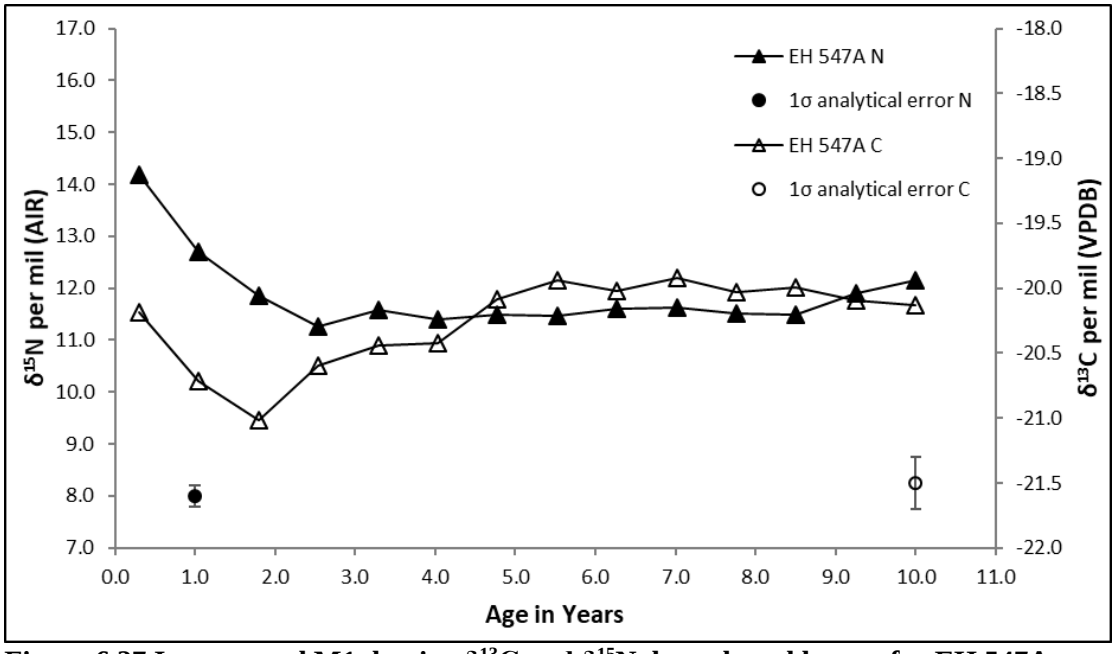

Figure 6.27 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 547A

EH 626A

Full data for EH 626A are found in Table A.28 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 626A appear in Figure 6.28.

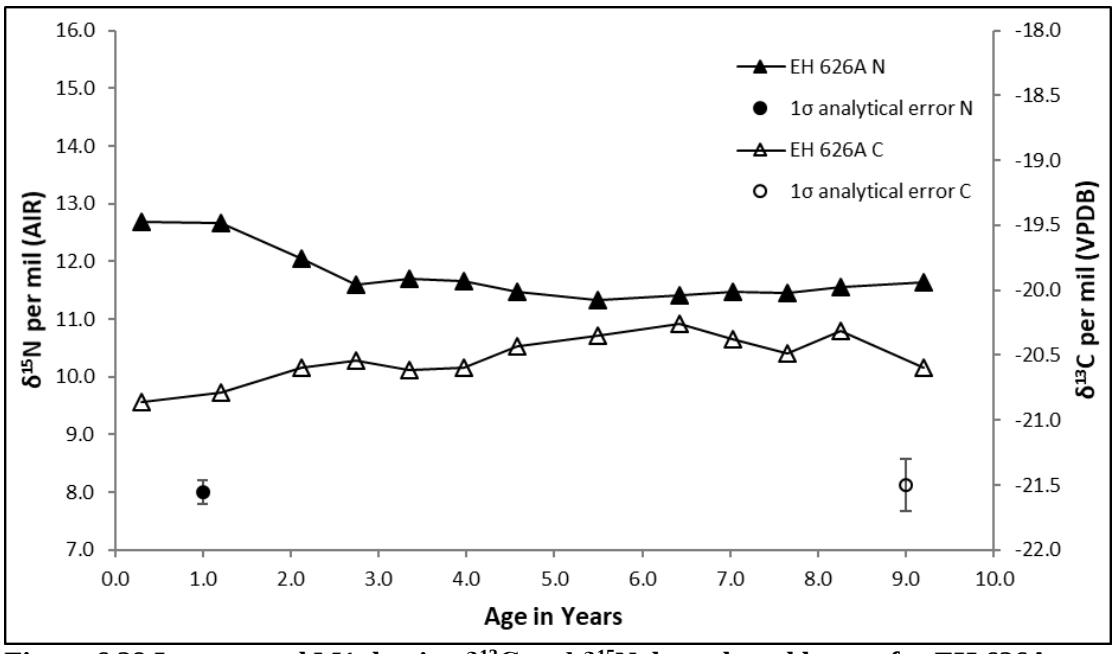

Figure 6.28 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 626A

EH 726

Full data for EH 726 are found in Table A.29 (Appendix 1). Figure 6.29 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 726.

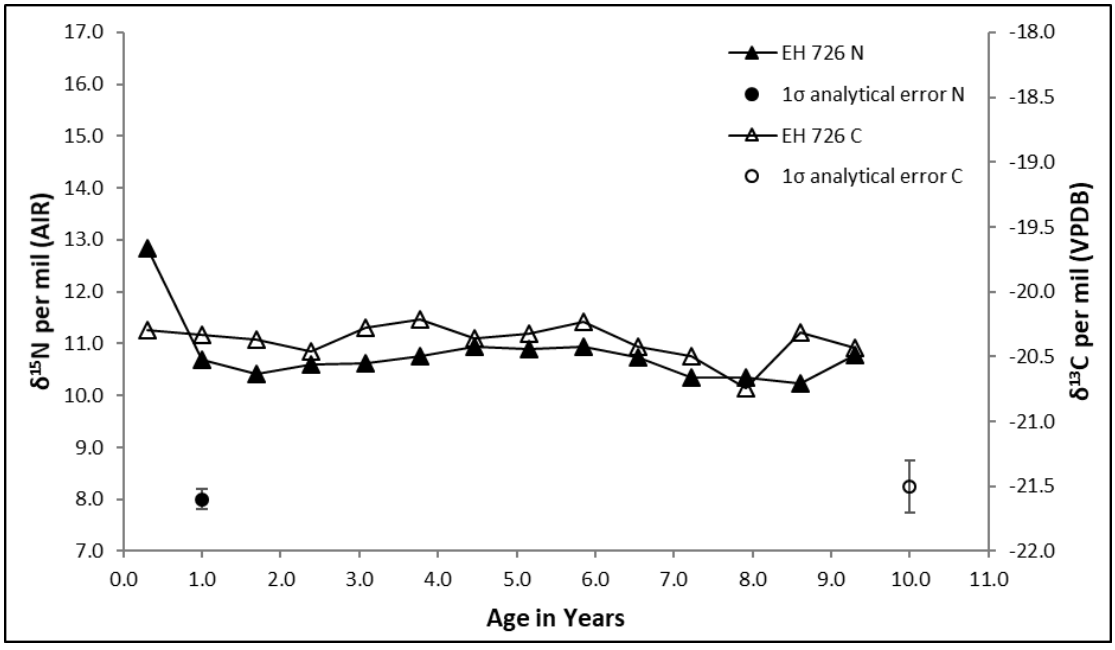

Figure 6.29 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for EH 726

6.2.2 Littleport results

Full results for collagen data for individuals sampled at Littleport appear in Tables A.30-A.59 (Appendix 1) and are shown graphically in Figures 6.30-6.59 in the sections which follow. For the total sample at Littleport, $\delta^{13}C$ values ranged from −22.1 to −19.4

‰ (mean −20.6 ‰ ± 0.4 ‰), while $\delta^{15}N$ values fell between 9.0 and 16.5 ‰ (mean 12.1 ‰ ± 1.4 ‰).

6.2.2.i Deciduous second molar profiles

This section presents data for the six non-survivors sampled at Littleport. while $\delta^{15}N$ values ranged from (mean). Table 6.4 presents summary data for the non-adult sample at Littleport. Incremental dentine data profiles for non-adult individuals will be presented graphically in the subsections below.

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
LP 3311	−21.6 - −20.4‰	−20.8 ± 0.5‰	1.2‰	13.8 - 15.7‰	15.0 ± 0.7‰	1.9‰	OC
LP 3770	−21.1 - −20.0‰	−20.4 ± 0.4‰	1.1‰	10.2 - 11.7‰	11.1 ± 0.5‰	1.5‰	OC
LP 4116	−21.6 - −20.7‰	−21.4 ± 0.3‰	0.9‰	12.6 - 14.6‰	13.5 ± 0.8‰	2.0‰	OC
LP 4144	−21.3 - −20.3‰	−20.8 ± 0.4‰	1.0‰	13.1 - 16.2‰	14.8 ± 1.2‰	3.1‰	OC
LP 4494	−21.2 - −20.2‰	−20.8 ± 0.3‰	1.0‰	12.1 - 15.5‰	13.8 ± 1.3 ‰	3.4‰	OC
LP 4848	−20.8 - −19.8‰	−20.3 ± 0.3‰	1.0‰	9.0 - 12.5‰	9.6 ± 0.8‰	3.5‰	PC
All	**−21.6 - −19.8‰**	**−20.7 ± 0.5‰**	**0.9-1.2‰**	**9.0 - 16.2‰**	**12.4 ± 2.3‰**	**1.5-3.5‰**	**-**

Table 6.4 Summary of incremental data for non-adult dm2 dentine at Littleport (OC= opposing covariance, PC= parallel covariance)

LP 3311

Full data for LP 3311 are found in Table A.30 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3311 appear in Figure 6.30.

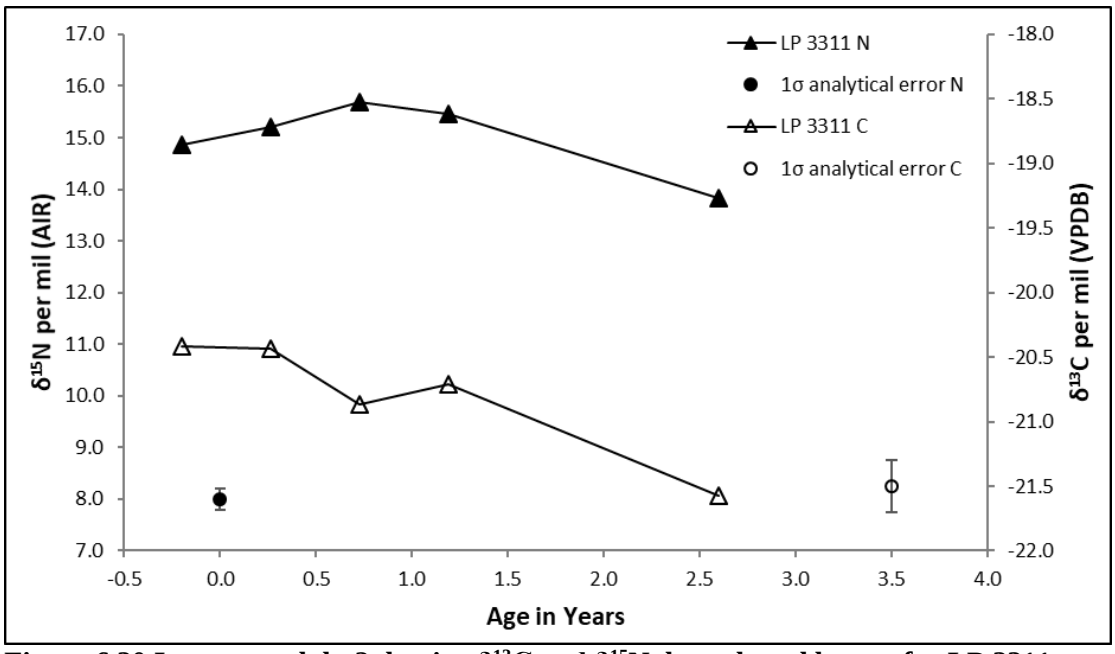

Figure 6.30 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3311

LP 3770

Full data for LP 3770 are found in Table A.31 (Appendix 1). Figure 6.31 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3770.

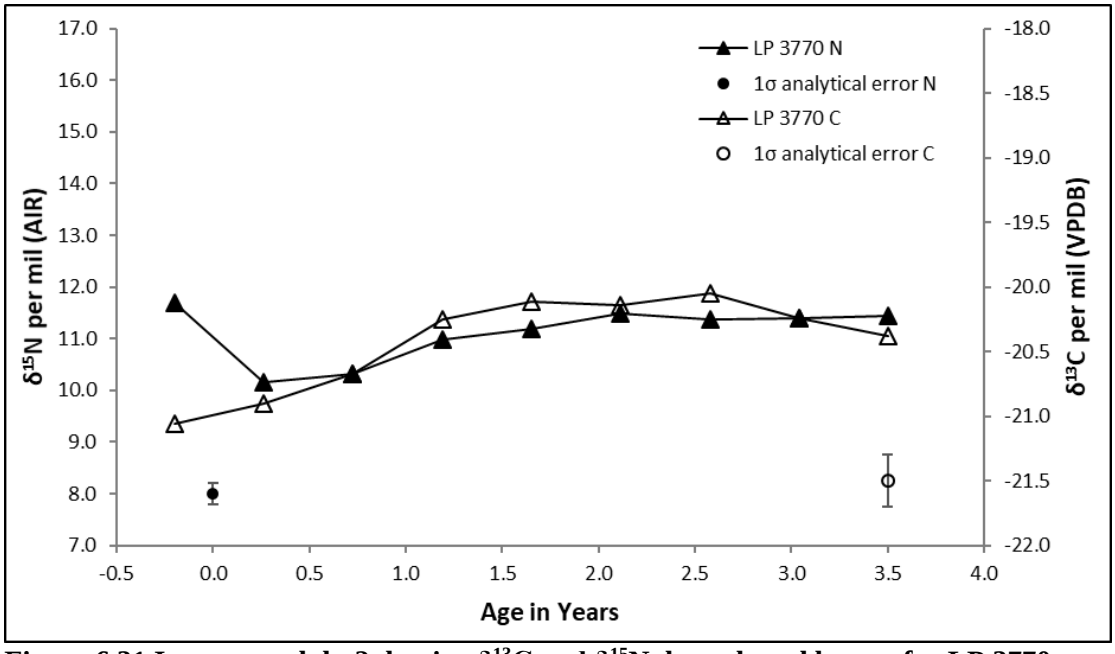

Figure 6.31 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3770

LP 4116

Full data for LP 4116 are found in Table A.32 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4116 appear in Figure 6.32.

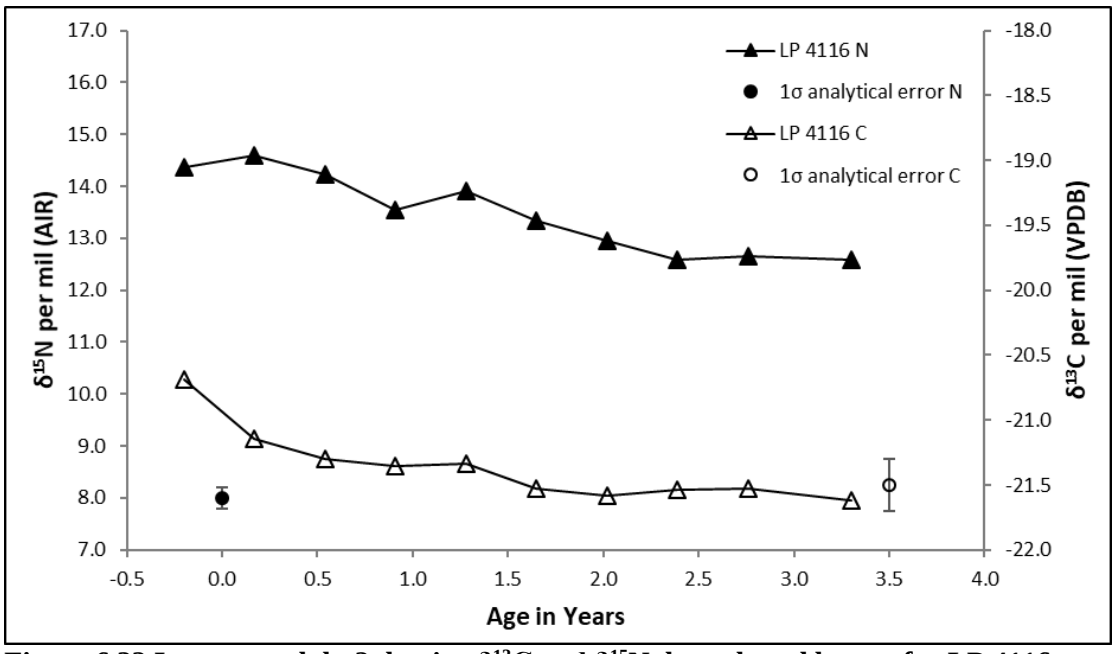

Figure 6.32 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4116

LP 4144

Full data for LP 4144 are found in Table A.33 (Appendix 1). Figure 6.33 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4144.

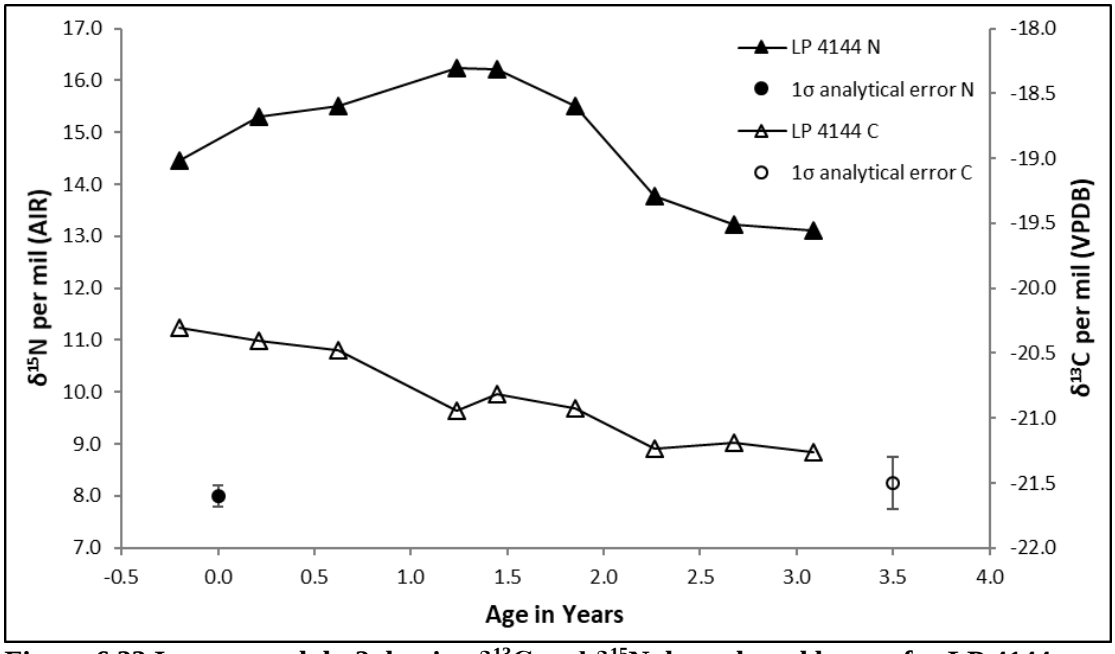

Figure 6.33 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4144

LP 4494

Full data for LP 4494 are found in Table A.34 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4494 appear in Figure 6.34.

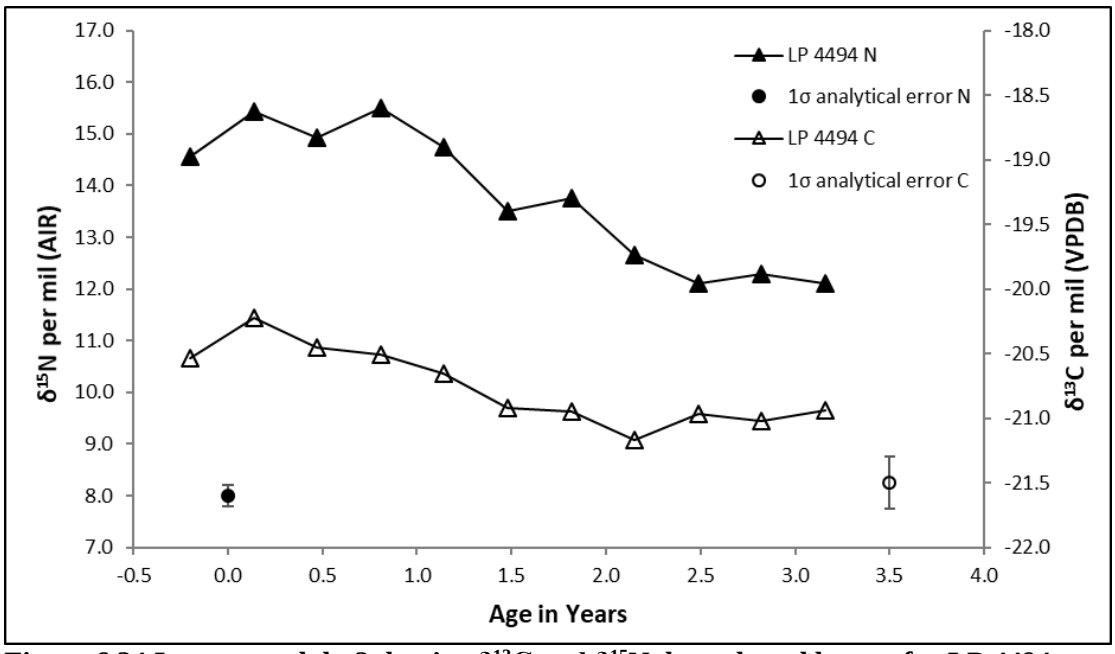

Figure 6.34 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4494

LP 4848

Full data for LP 4848 are found in Table A.35 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4848 appear in Figure 6.35.

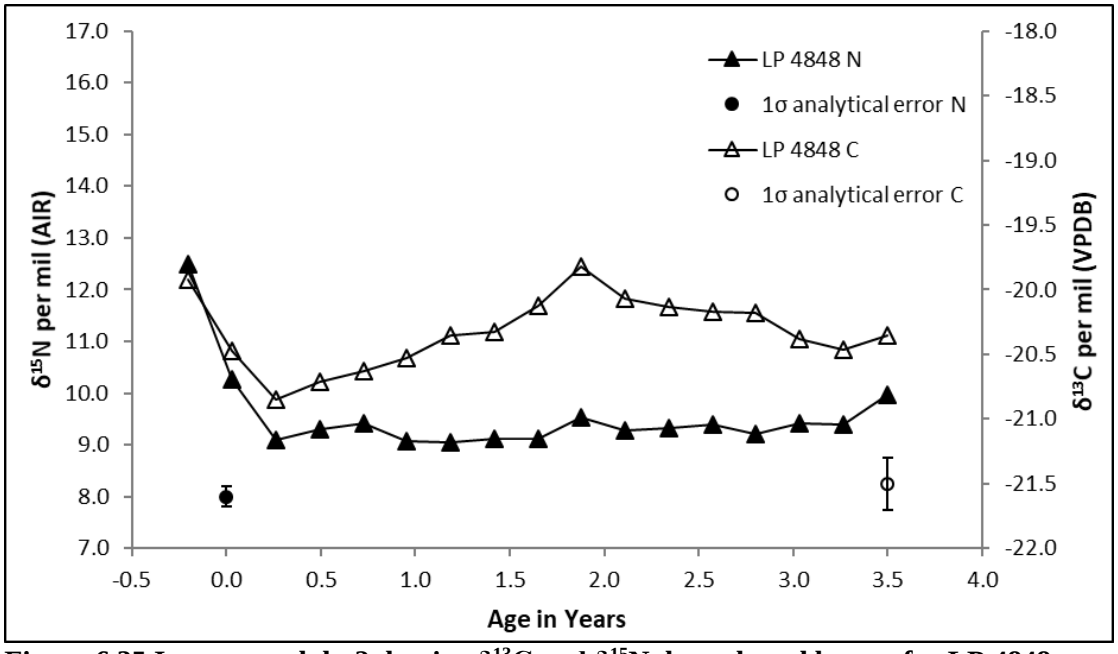

Figure 6.35 Incremental dm2 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4848

6.2.2.ii Permanent first molar profiles

This section presents data for M1 teeth sampled at Littleport. Dentine collagen $\delta^{13}C$ values of the M1 sample fell within a range of −22.1 to −19.4 ‰ (mean −20.6 ‰ ± 0.4 ‰), while $\delta^{15}N$ values ranged from 9.6 to 16.5 ‰ (mean 12.0 ‰ ± 1.2 ‰). Intra-

individual variation ranged from 0.4-1.2 ‰ for $\delta^{13}C$ and 1.3-4.3 ‰ for $\delta^{15}N$ over the course of M1 tooth development in adults sampled at Littleport. Incremental dentine data profiles for adult individuals will be presented graphically in the subsections below.

Males

This section presents M1 data for sampled adult males at Littleport. Table 6.5 summarizes incremental data for this subsample of the Littleport population, which are presented individually in the subsections which follow.

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
LP 3708	−20.7 - −20.1‰	−20.3 ± 0.2‰	0.6‰	10.0 - 12.2‰	10.8 ± 0.6‰	2.2‰	OC
LP 3885	−21.3 - −20.8‰	−21.0 ± 0.1‰	0.5‰	11.6 - 13.2‰	12.1 ± 0.4‰	1.6‰	OC
LP 3893	−21.1 - −20.3‰	−20.7 ± 0.3‰	0.8‰	9.6 - 12.4‰	10.7 ± 0.9‰	2.8‰	OC
LP 4073	−21.0 - −20.0‰	−20.3 ± 0.3‰	1.0‰	12.2 - 13.5‰	12.8 ± 0.3‰	1.3‰	OC
LP 4092	−21.2 - −20.4‰	−20.8 ± 0.2‰	0.8‰	11.5 - 14.2‰	12.3 ± 0.6‰	2.7‰	PC
LP 4095	−22.1 - −21.2‰	−21.5 ± 0.3‰	0.9‰	11.9 - 13.3‰	12.5 ± 0.4‰	1.4‰	OC
LP 4134	−21.3 - −20.6‰	−21.0 ± 0.2‰	0.7‰	11.9 - 13.2‰	12.6 ± 0.4‰	1.3‰	PC
LP 4173	−21.0 - −20.4‰	−20.8 ± 0.2‰	0.6‰	11.1 - 13.3‰	12.0 ± 0.6‰	2.2‰	OC
LP 4178	−20.3 - −19.4‰	−19.7 ± 0.3‰	0.9‰	12.2 - 16.5‰	13.8 ± 1.3‰	4.3‰	OC
LP 4250	−21.2 - −20.2‰	−20.5 ± 0.3‰	1.0‰	10.9 - 12.3‰	11.7 ± 0.5‰	1.4‰	OC
LP 4603	−21.4 - −20.4‰	−20.8 ± 0.3‰	1.0‰	11.2 - 14.4‰	12.3 ± 0.7‰	3.2‰	OC
All	**−22.1 - −19.4‰**	**−20.7 ± 0.5‰**	**0.5-1.0‰**	**9.6 - 16.5‰**	**12.2 ± 1.0‰**	**1.3-3.2‰**	**-**

Table 6.5 Summary of incremental data for adult male M1 dentine at Littleport (OC= opposing covariance, PC= parallel covariance)

LP 3708

Full data for LP 3708 are found in Table A.36 (Appendix 1). Figure 6.36 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3708.

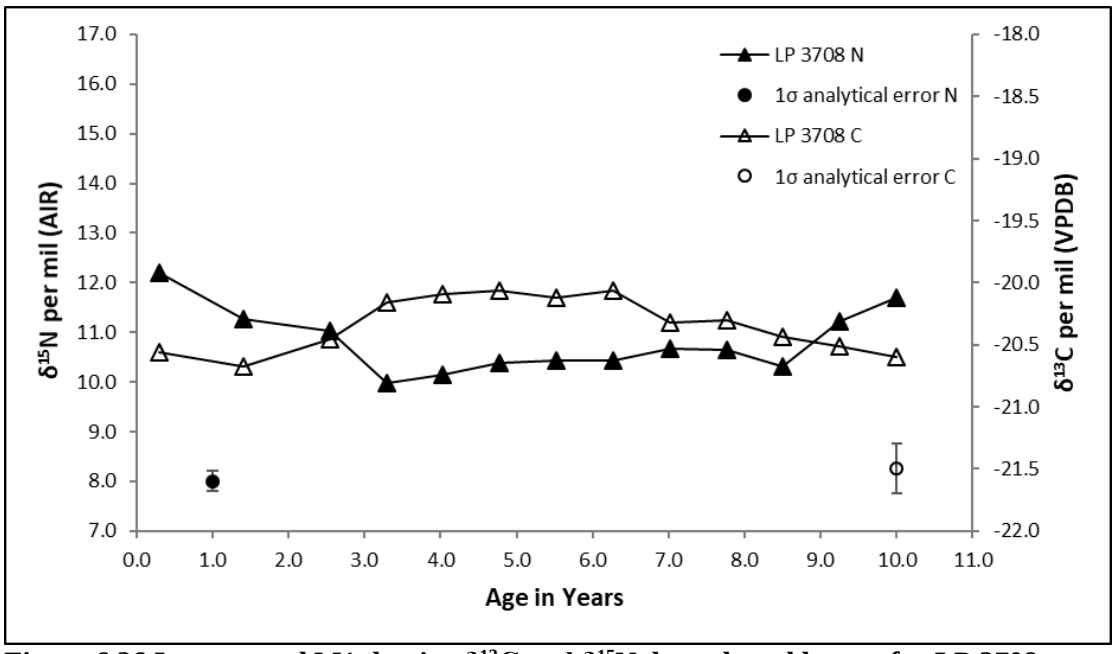

Figure 6.36 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3708

LP 3885

Full data for LP 3885 are found in Table A.37 (Appendix 1$\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3885 appear in Figure 6.37.

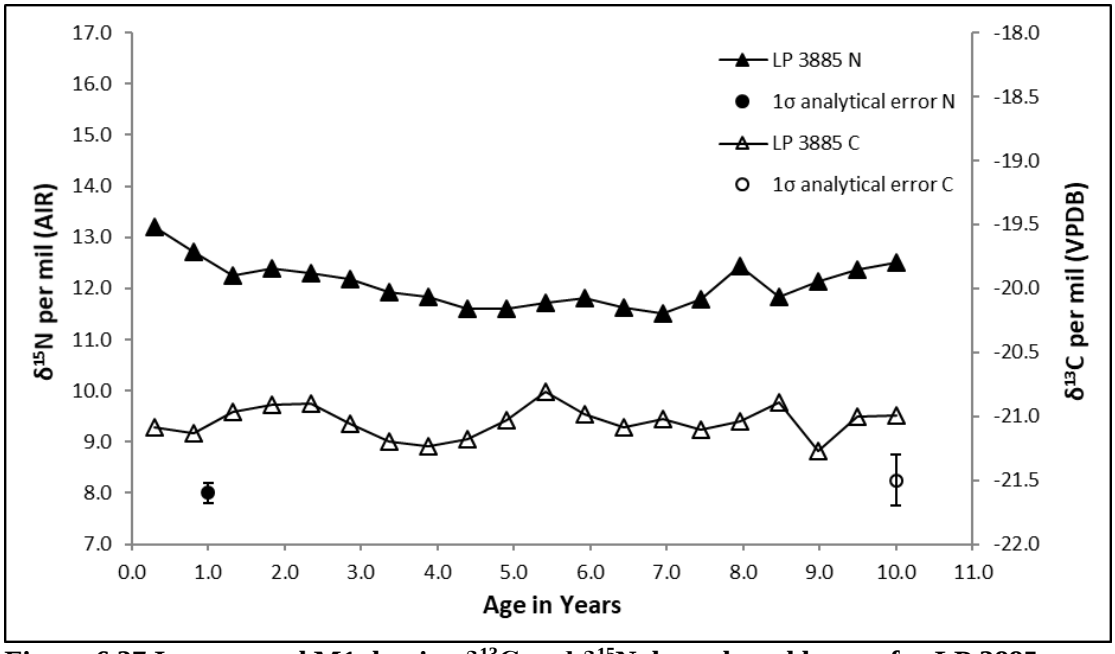

Figure 6.37 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3885

LP 3893

Full data for LP 3893 are found in Table A.38 (Appendix 1). Figure 6.38 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3893.

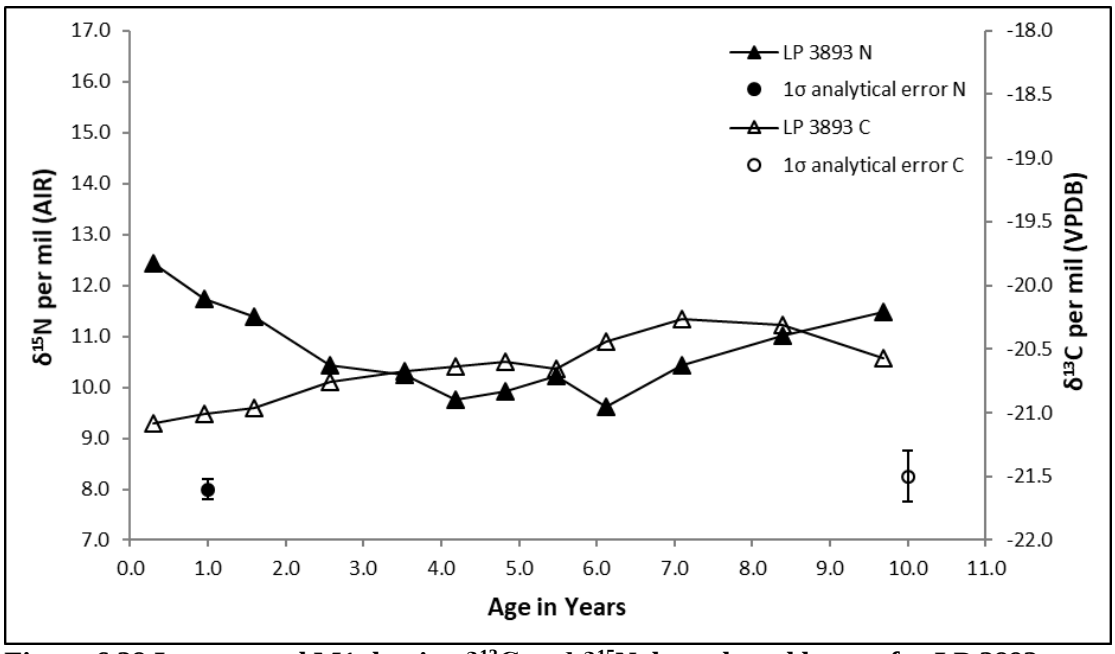

Figure 6.38 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3893

LP 4073

Full data for LP 4073 are found in Table A.39 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4073 appear in Figure 6.39.

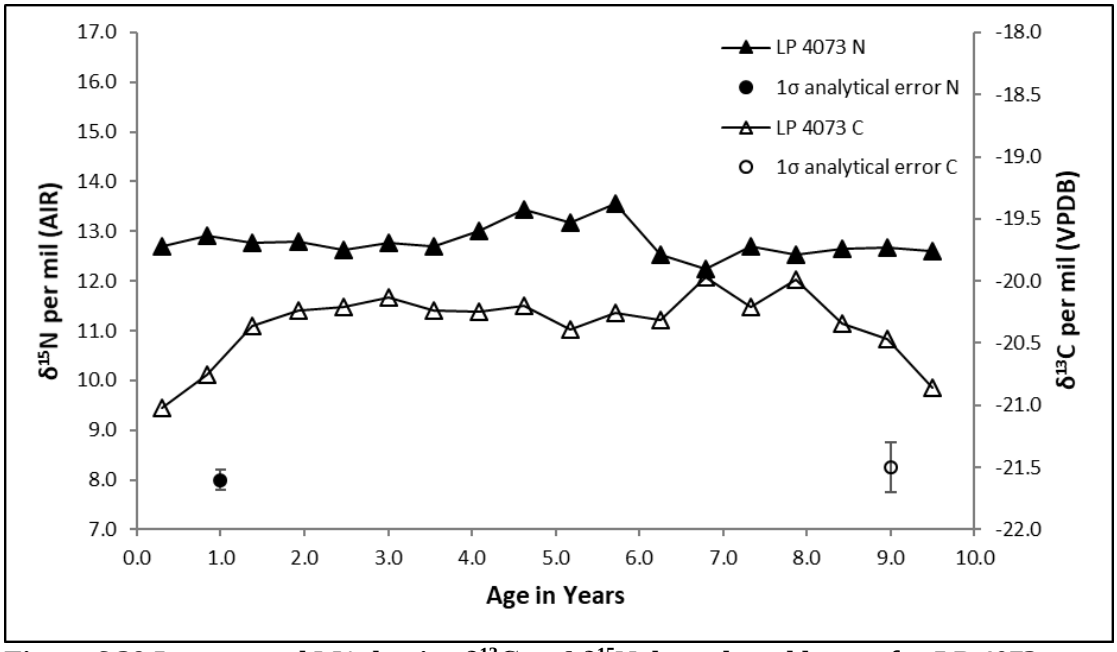

Figure 6.39 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4073

LP 4092

Full data for LP 4092 are found in Table A.40 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4092 appear in Figure 6.40.

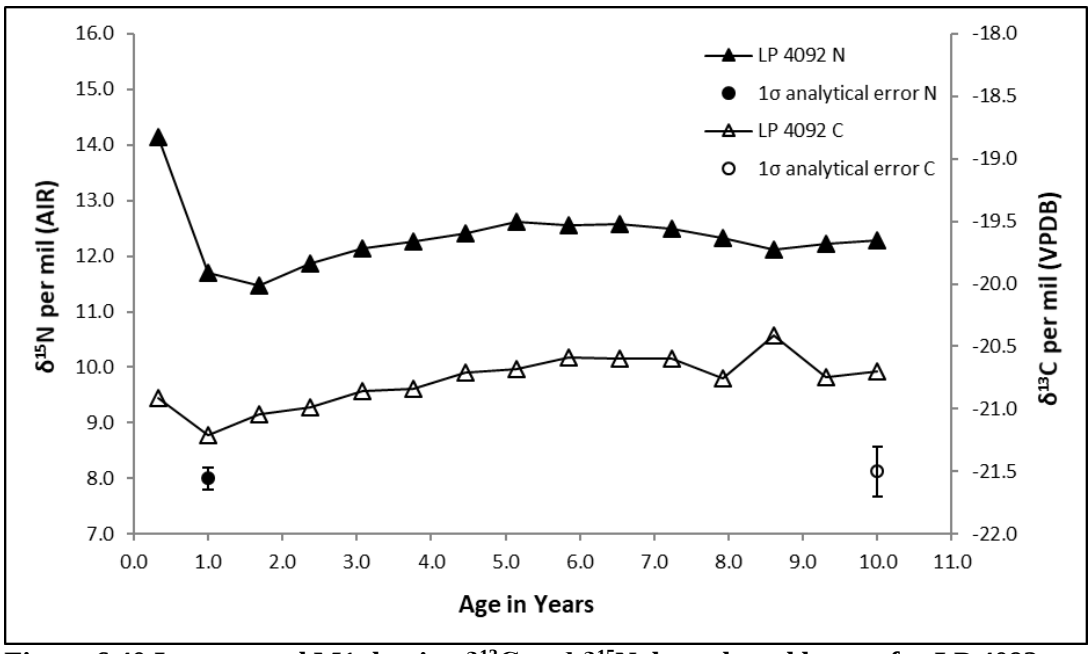

Figure 6.40 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4092

LP 4095

Full data for LP 4095 are found in Table A.41 (Appendix 1). Figure 6.41 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4095.

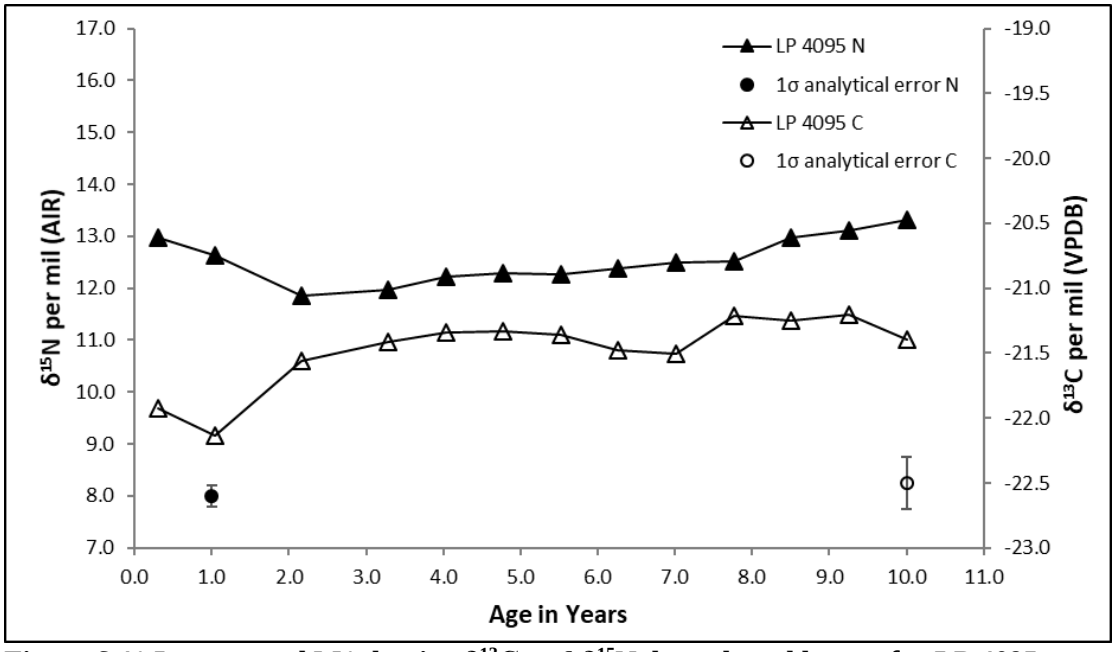

Figure 6.41 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4095

LP 4134

Full data for LP 4134 are found in Table A.42 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4134 appear in Figure 6.42.

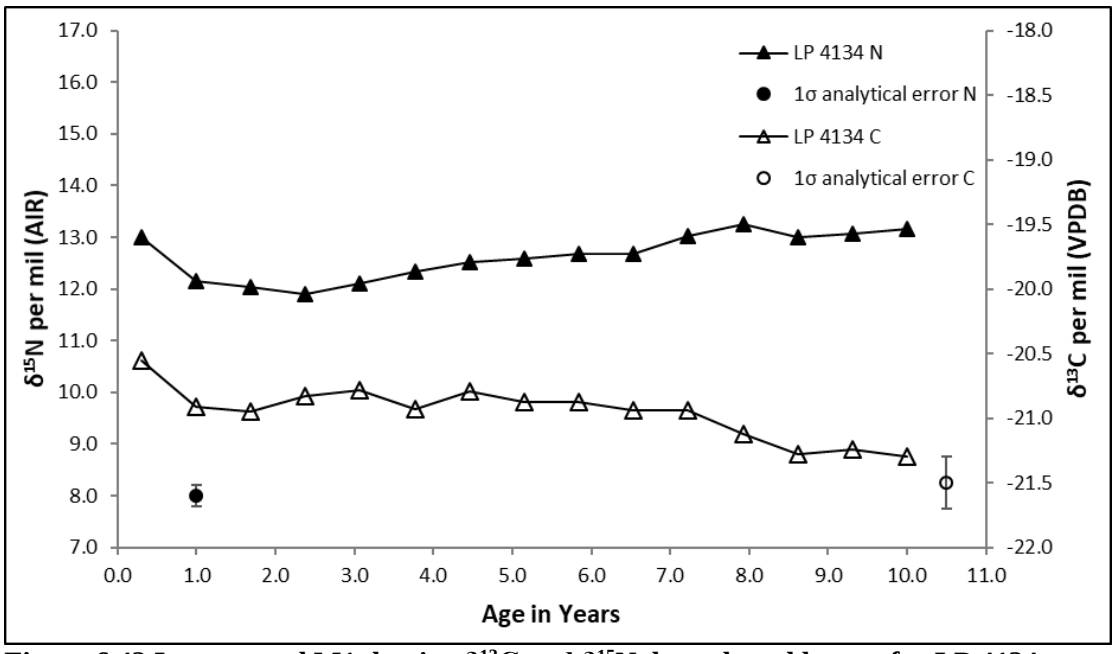

Figure 6.42 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4134

LP 4173

Full data for LP 4173 are found in Table A.43 (Appendix 1). Figure 6.43 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4173.

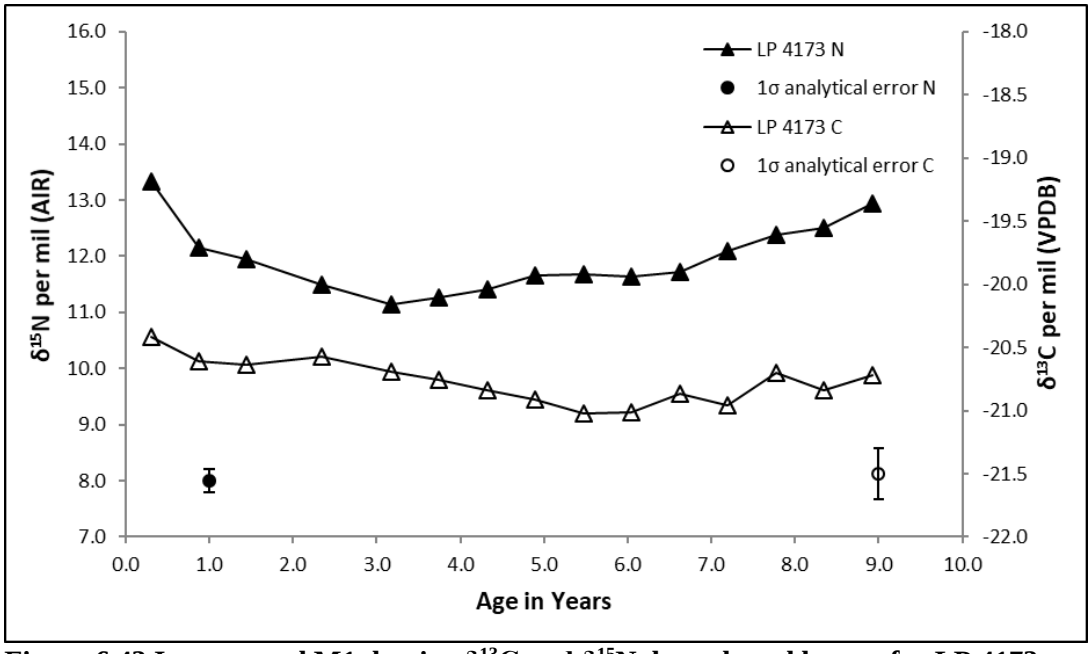

Figure 6.43 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4173

LP 4178

Full data for LP 4178 are found in Table A.44 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4178 appear in Figure 6.44.

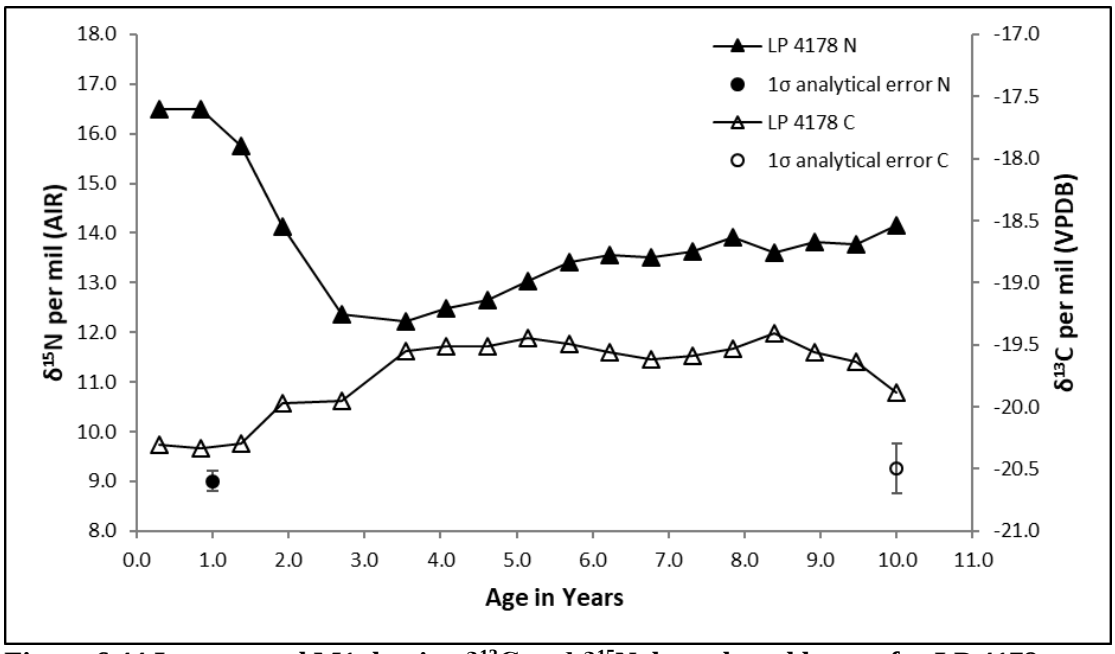

Figure 6.44 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4178

LP 4250

Full data for LP 4250 are found in Table A.45 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4250 appear in Figure 6.45.

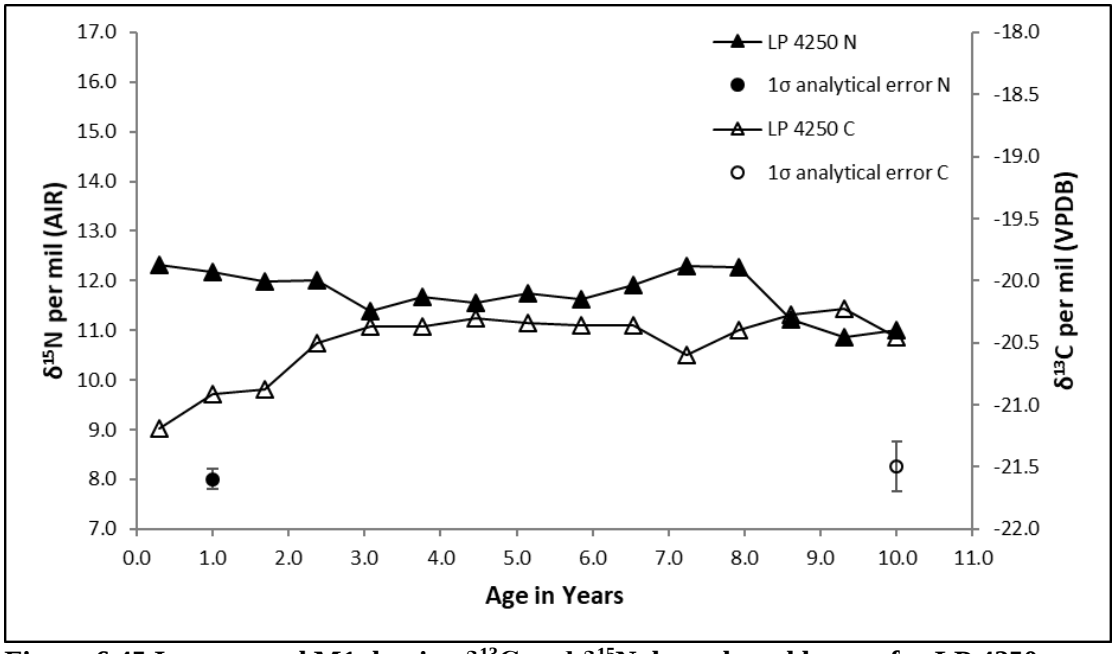

Figure 6.45 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4250

LP 4603

Full data for LP 4603 are found in Table A.46 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4603 appear in Figure 6.46.

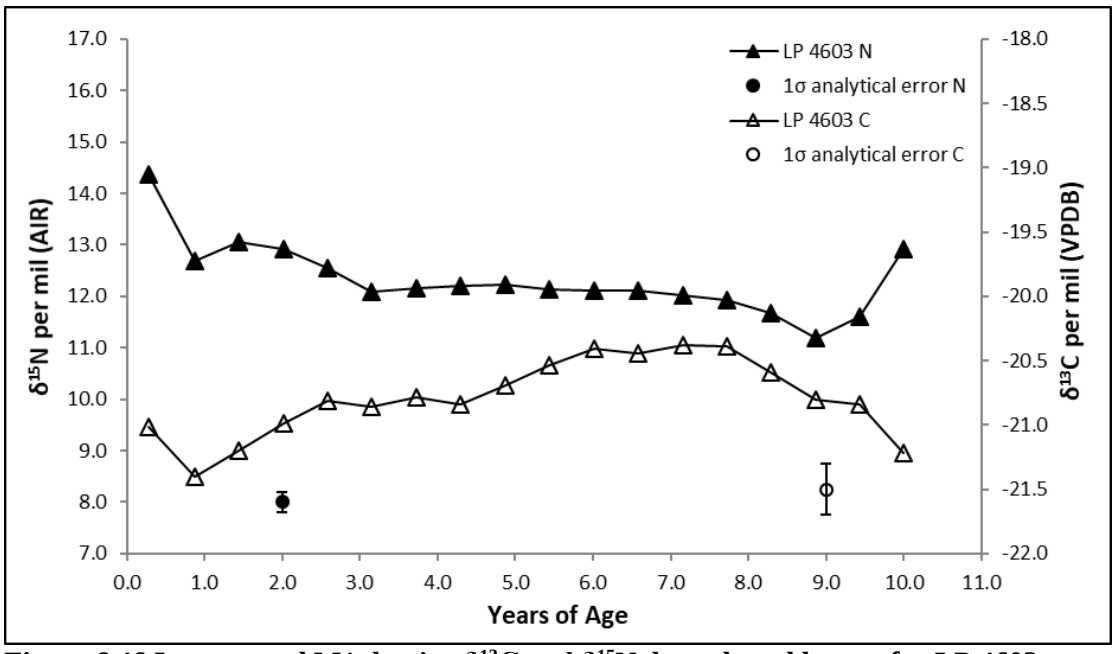

Figure 6.46 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4603

Females

This section presents M1 data for adult females sampled at Littleport. Table 6.6 presents a summary of incremental data for these individuals, which are presented in detailed graphical form in subsections below.

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
LP 3687	−21.0 - −20.0‰	−20.7 ± 0.3‰	1.0‰	12.4 - 14.4‰	13.1 ± 0.7‰	2.0 ‰	OC
LP 3745	−20.8 - −20.0‰	−20.5 ± 0.2‰	0.8‰	11.7 - 16.0‰	12.5 ± 1.1‰	4.3‰	PC
LP 3749	−20.9 - −20.5‰	−20.7 ± 0.1‰	0.4‰	12.0 - 14.1‰	12.7 ± 0.6‰	2.1‰	OC
LP 3819	−20.5 - −20.0‰	−20.3 ± 0.1‰	0.5‰	11.2 - 13.0‰	12.4 ± 0.6‰	1.8‰	-
LP 4035	−21.4 - −20.2‰	−20.5 ± 0.4‰	0.6‰	10.6 - 12.1‰	11.8 ± 0.4‰	2.2‰	PC
LP 4047	−20.8 - −20.1‰	−20.5 ± 0.2‰	0.7‰	10.2 - 12.6‰	10.7 ± 0.7‰	2.4‰	PC
LP 4063	−21.1 - −19.9‰	−20.8 ± 0.3‰	1.2‰	11.5 - 15.4‰	12.6 ± 1.1‰	3.9‰	PC
LP 4067	−20.9 - −20.4‰	−20.6 ± 0.1‰	0.5‰	10.3 - 14.5‰	11.1 ± 1.1‰	4.2‰	Flat
LP 4075	−20.5 - −19.5‰	−20.0 ± 0.2‰	0.7‰	10.3 - 11.9‰	10.6 ± 0.4‰	1.6‰	OC
LP 4139	−21.2 - −20.0‰	−20.4 ± 0.4‰	1.2‰	9.8 - 13.5‰	10.5 ± 1.0‰	3.7‰	OC

Individual	Dentine $\delta^{13}C$			Dentine $\delta^{15}N$			Profile Type < 4 years
	Range	Mean	Max. Var.	Range	Mean	Max. Var.	
LP 4395	−20.6 - −20.1‰	−20.4 ± 0.2‰	0.5‰	11.8 - 15.0‰	12.5 ± 0.9‰	3.2‰	Flat
LP 4585	−21.2 - −20.0‰	−20.5 ± 0.4‰	1.2‰	10.8 - 14.3‰	11.6 ± 0.9‰	3.5‰	OC
All	**−21.4 - −19.8‰**	**−20.5 ± 0.3‰**	**0.4-1.2‰**	**9.8 - 16.0‰**	**11.9 ± 1.2‰**	**1.6-4.3‰**	**-**

Table 6.6 Summary of incremental data for adult female M1 dentine at Littleport (OC= opposing covariance, PC= parallel covariance)

LP 3687

Full data for LP 3687 are found in Table A.47 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3687 appear in Figure 6.47.

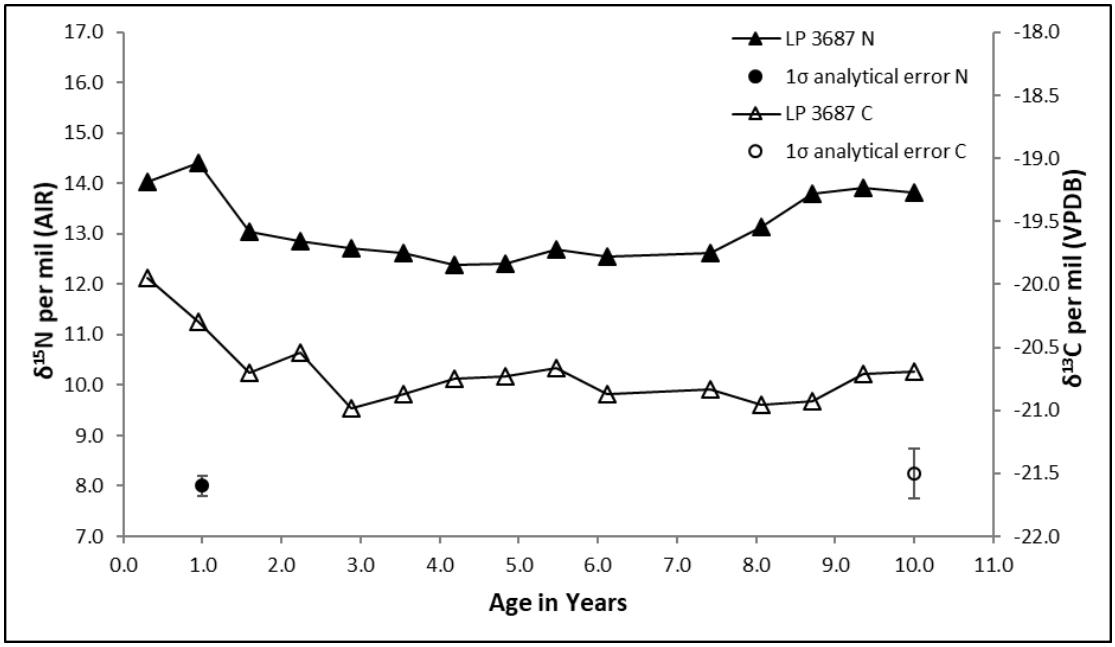

Figure 6.47 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3687

LP 3745

Full data for LP 3745 are found in Table A.48 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3745 appear in Figure 6.48.

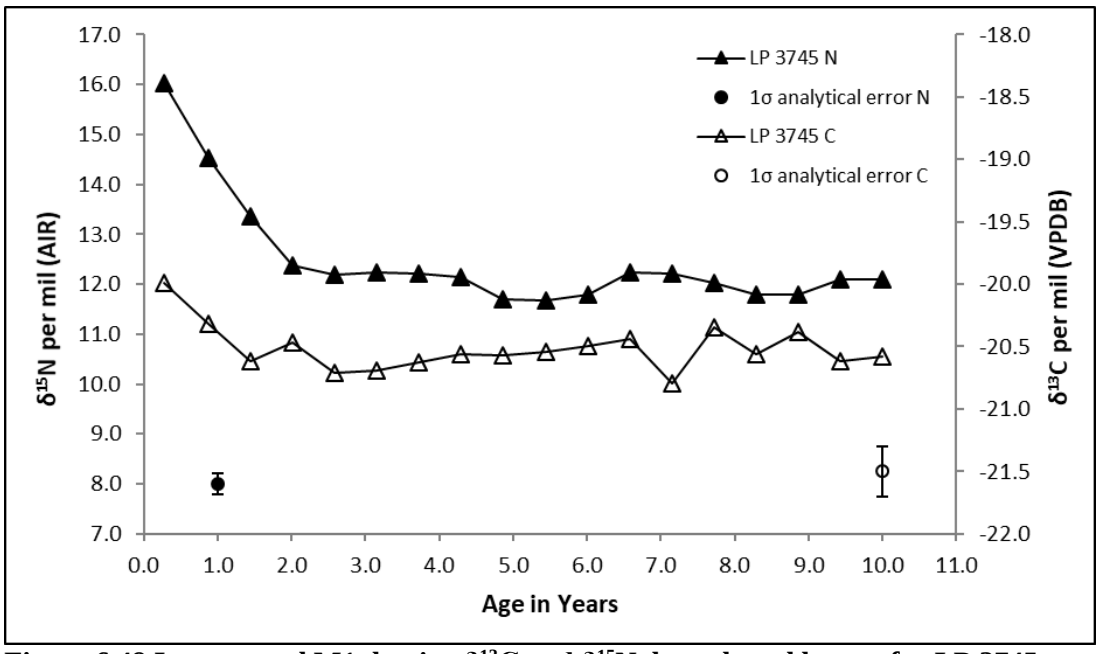

Figure 6.48 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3745

LP 3749

Full data for LP 3749 are found in Table A.49 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3749 appear in Figure 6.49.

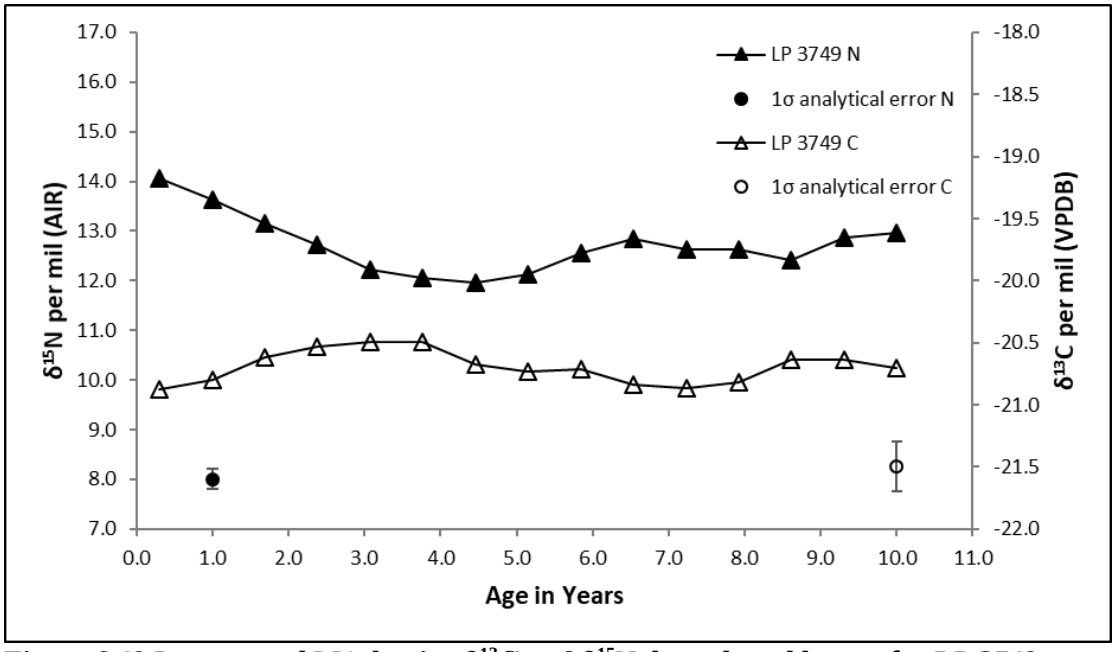

Figure 6.49 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3749

LP 3819

Full data for LP 3819 are found in Table A.50 (Appendix 1). Figure 6.50 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3819.

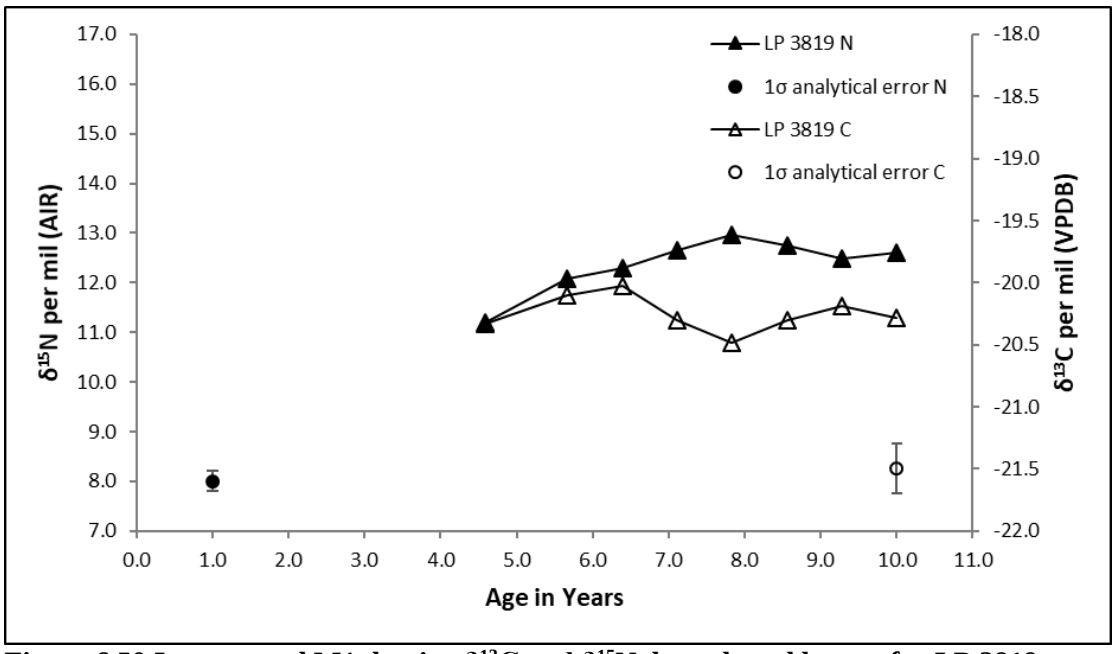

Figure 6.50 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 3819

LP 4035

Full data for LP 4035 are found in Table A.51 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4035 appear in Figure 6.51.

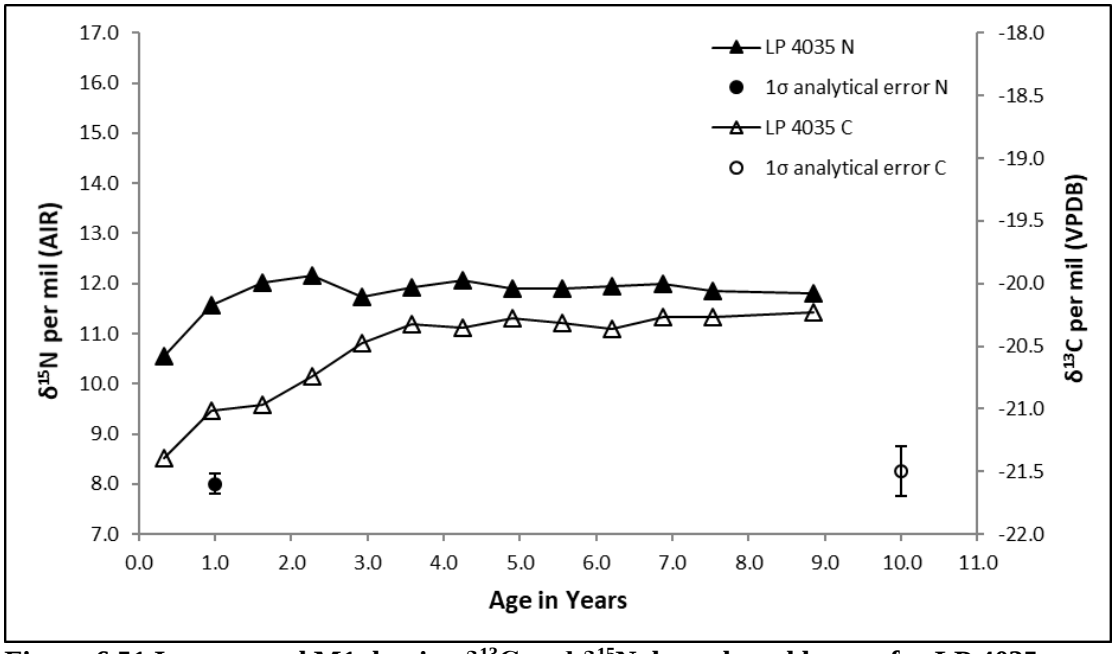

Figure 6.51 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4035

LP 4047

Full data for LP 4047 are found in Table A.52 (Appendix 1). Figure 6.52 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4047.

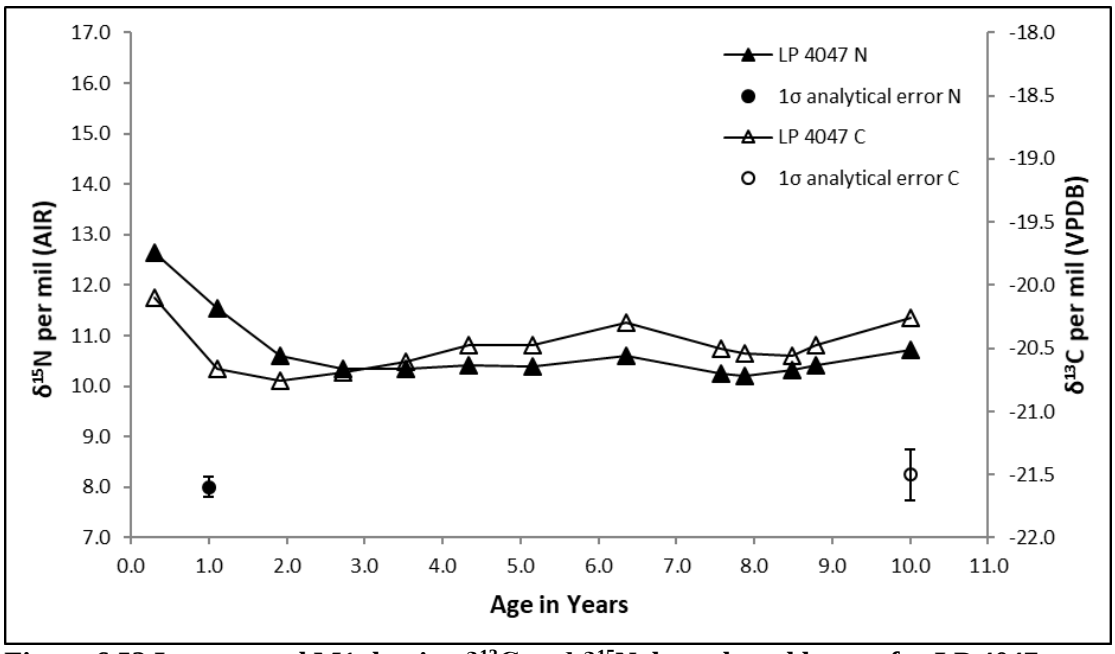

Figure 6.52 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4047

LP 4063

Full data for LP 4063 are found in Table A.53 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4063 appear in Figure 6.53.

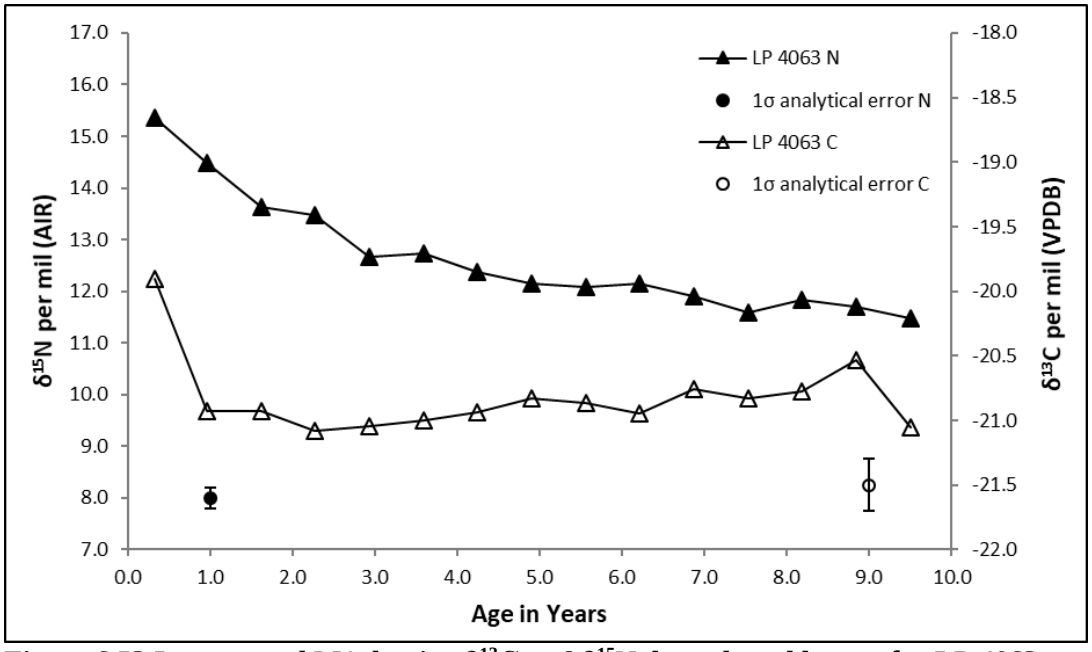

Figure 6.53 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4063

LP 4067

Full data for LP 4067 are found in Table A.54 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4067 appear in Figure 6.54.

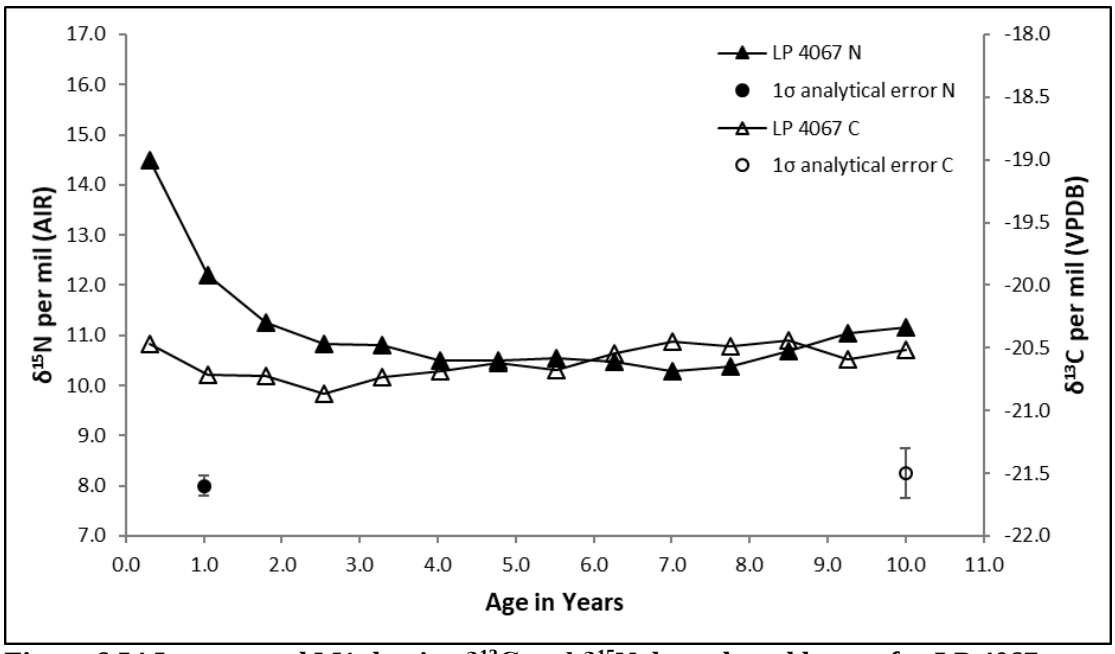

Figure 6.54 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4067

LP 4075

Full data for LP 4075 are found in Table A.55 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4075 appear in Figure 6.55.

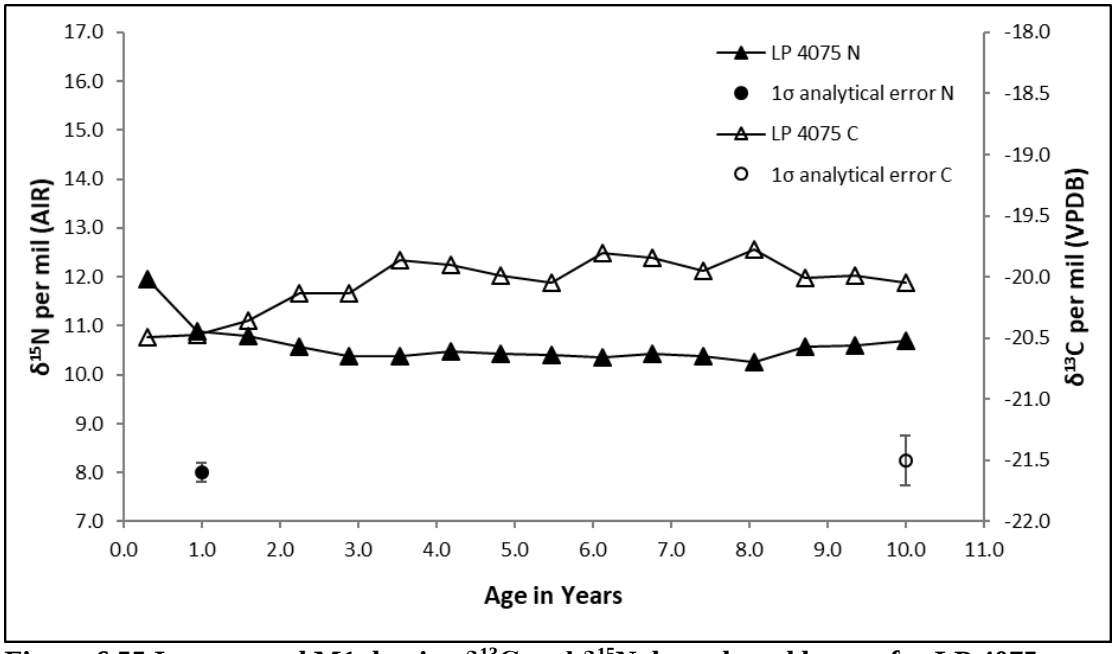

Figure 6.55 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4075

LP 4139

Full data for LP 4139 are found in Table A.56 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4139 appear in Figure 6.56.

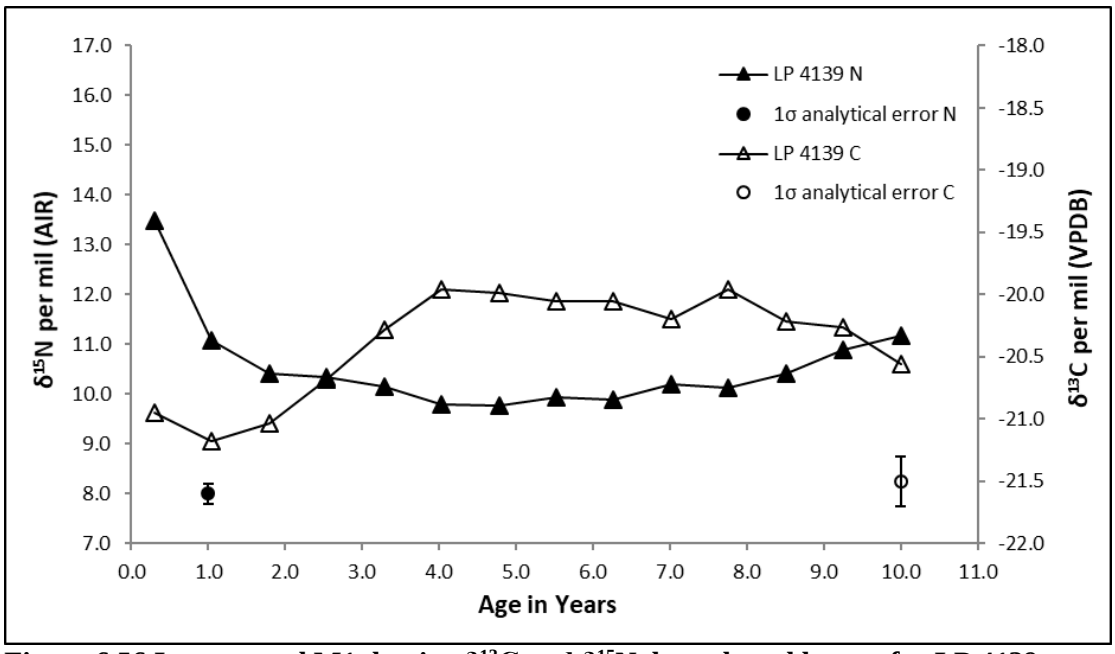

Figure 6.56 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4139

LP 4395

Full data for LP 4395 are found in Table A.57 (Appendix 1). Figure 6.57 shows $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4395.

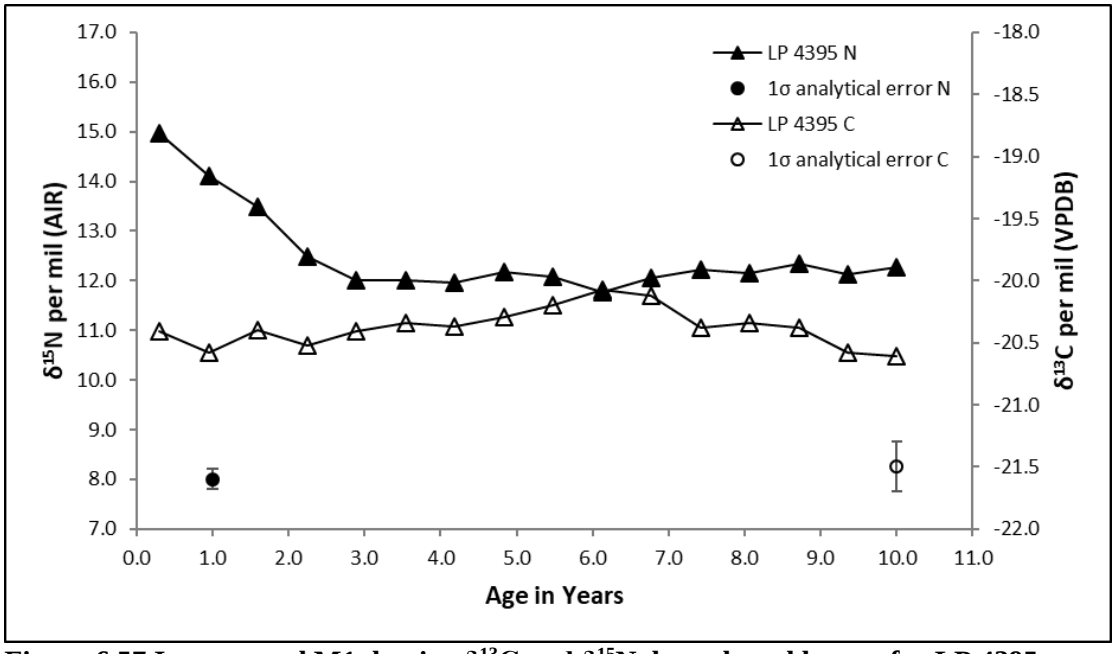

Figure 6.57 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4395

LP 4585

Full data for LP 4585 are found in Table A.58 (Appendix 1). $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4585 appear in Figure 6.58.

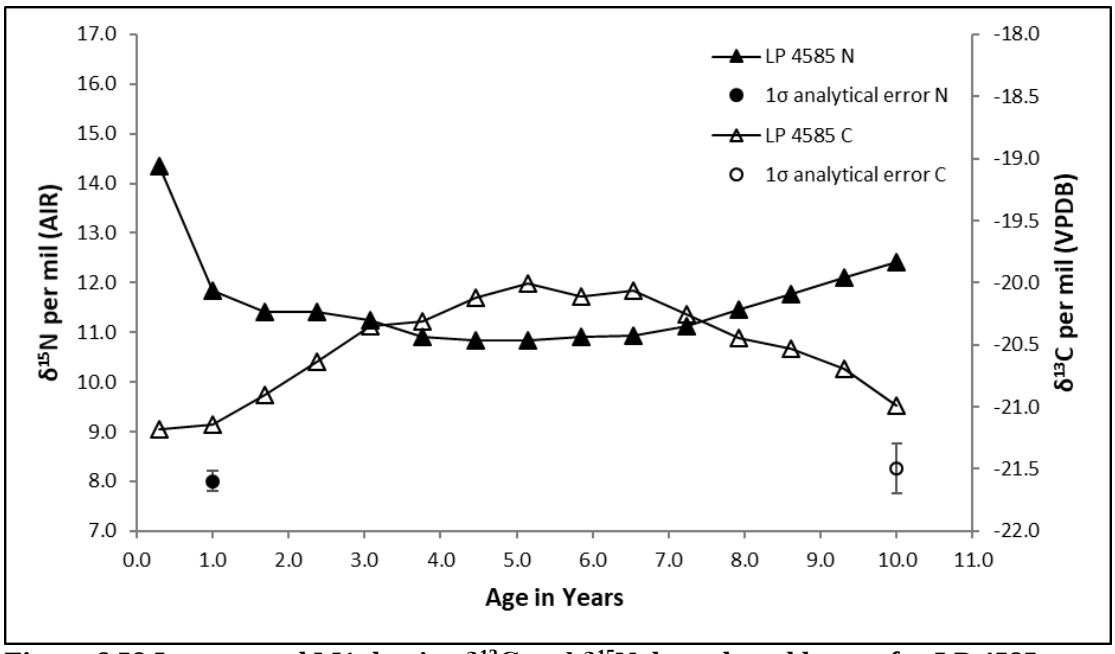

Figure 6.58 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4585

Indeterminate

This section presents M1 data for the sole sampled adult individual of indeterminate sex at Littleport.

LP 4556

Full data for LP 4556 are found in Table A.59 (Appendix 1). Dentine collagen $\delta^{13}C$ values for LP 4556 fell within a range of −20.9 to −20.3 ‰ (mean −20.6 ‰ ± 0.2 ‰), while $\delta^{15}N$ values ranged from 9.7 to 12.8 ‰ (mean 10.9 ‰ ± 0.9 ‰). Overall variability in values of 0.6 ‰ for $\delta^{13}C$ and 3.1 ‰ for $\delta^{15}N$ occurred over the course of M1 increments sampled. $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4556 appear in Figure 6.59.

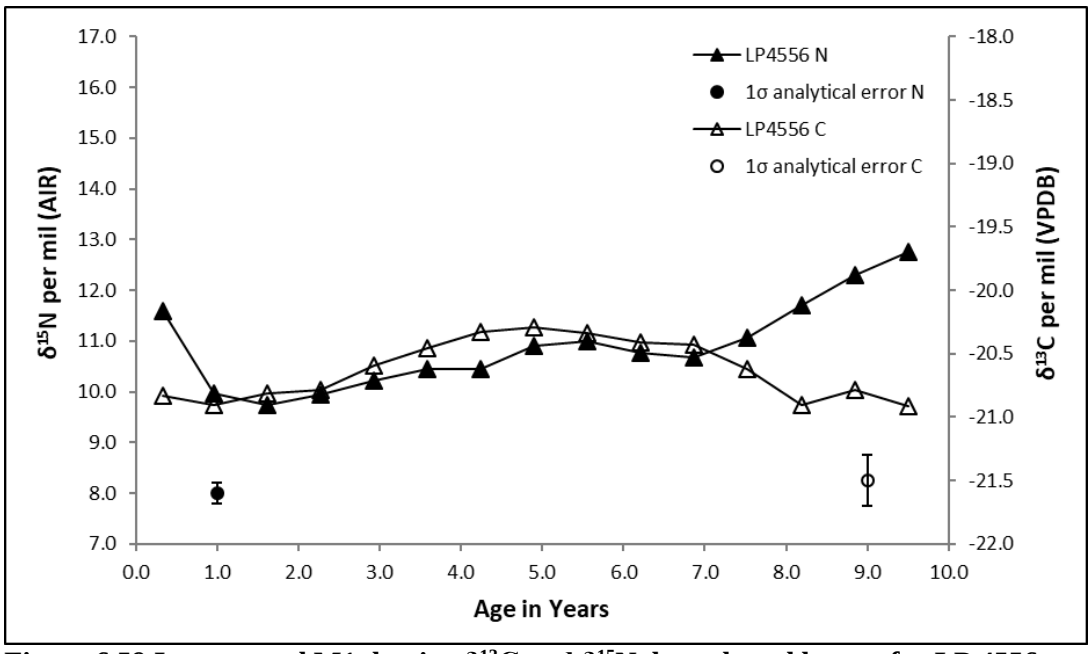

Figure 6.59 Incremental M1 dentine $\delta^{13}C$ and $\delta^{15}N$ data plotted by age for LP 4556

6.3 Grouped Data

The following sections include comparative plots of data grouped by site affiliation, estimated skeletal sex, skeletal pathology, and survivorship.

6.3.1 Edix Hill results

Comparative data for Edix Hill are summarised in Figures 6.60-6.77. Sections 6.3.1.i and 6.3.1.ii present dm2 and M1 tooth data summaries, respectively, while Section 6.3.1.iii presents a comparison of survivor (M1) and non-survivor (dm2) data.

6.3.1.i Deciduous tooth data

Summary plots for dm2 tooth data from sampled non-surviving children at Edix Hill appear in Figures 6.60-6.62.

Figure 6.60 shows grouped $\delta^{15}N$ data for the non-survivor cohort at Edix Hill. Most individuals in the group display a flat longitudinal profile, apart from EH 352 and EH 547B.

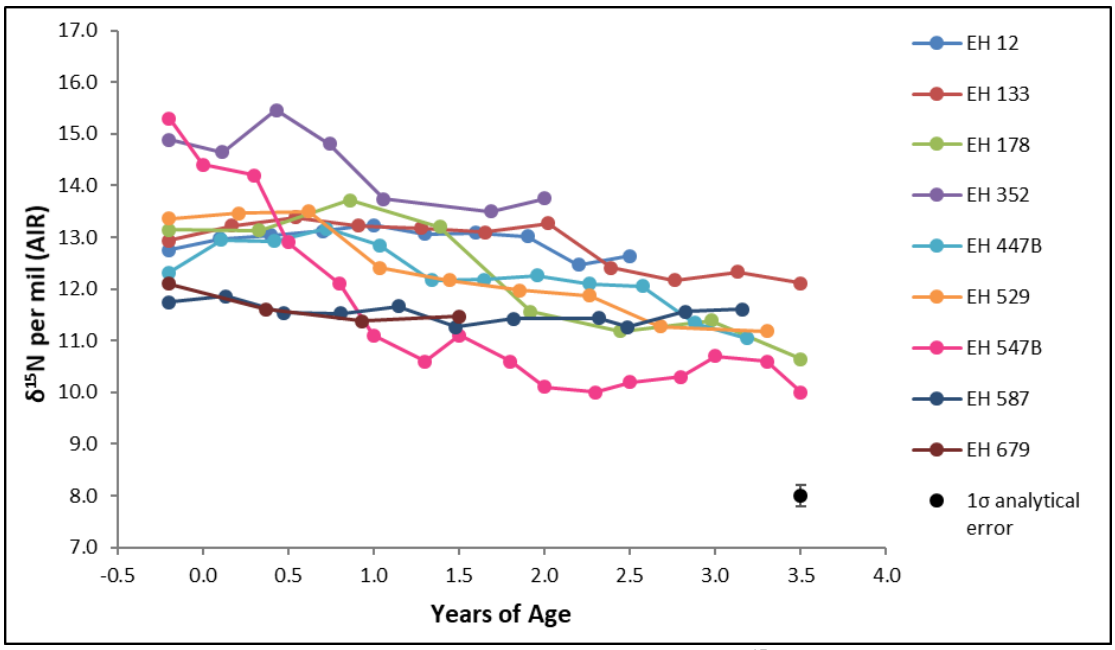

Figure 6.60 Plot of non-survivor dm2 incremental dentine $\delta^{15}N$ data profiles for Edix Hill

Figure 6.61 shows grouped longitudinal $\delta^{13}C$ data series for the sampled non-survivor cohort at Edix Hill. As with the $\delta^{15}N$ data, the predominant observable pattern is flatter, with some individuals showing a gradual decrease in values between the first half of the first year of life, and 1-1.5 years of age.

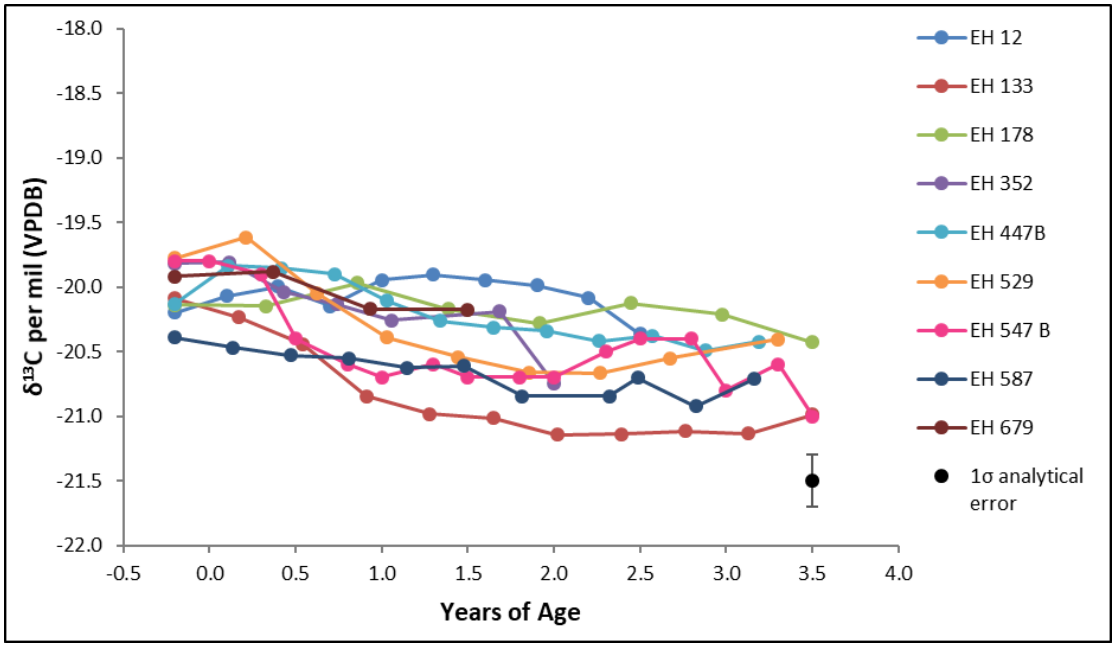

Figure 6.61 Plot of non-survivor dm2 incremental dentine $\delta^{13}C$ data profiles for Edix Hill

Figure 6.62 shows all sampled deciduous dentine increments at Edix Hill plotted individually as $\delta^{13}C$ vs $\delta^{15}N$.

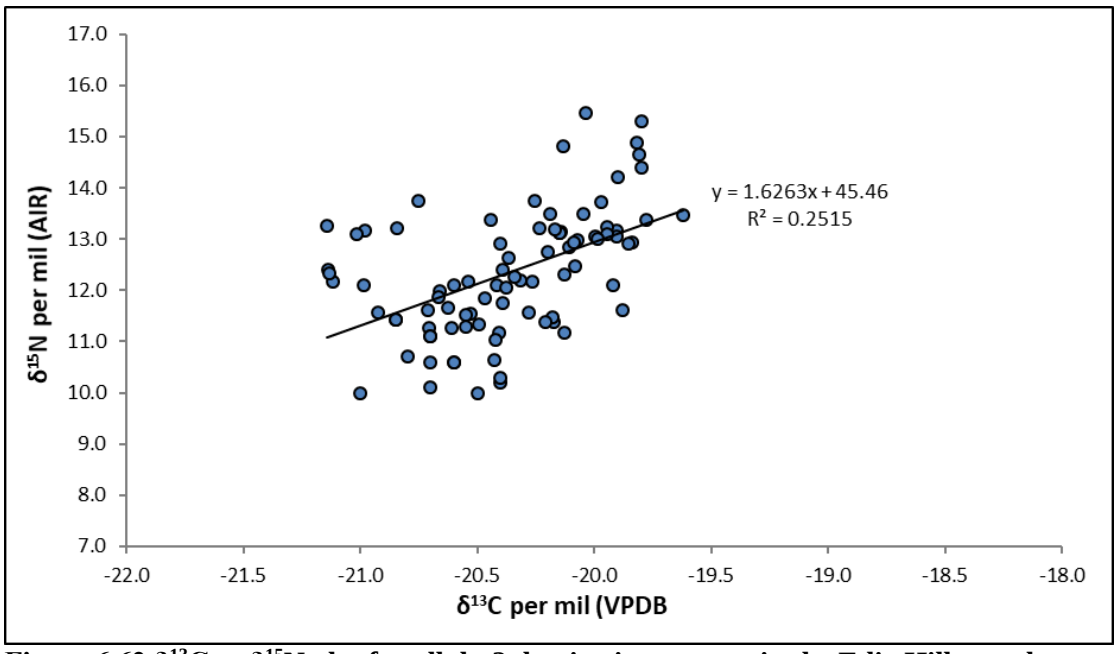

Figure 6.62 $\delta^{13}C$ vs $\delta^{15}N$ plot for all dm2 dentine increments in the Edix Hill sample, showing weak correlation (r^2= 0.25), and a slope of 1.6.

6.3.1.ii Permanent tooth data

This section presents summary plots for M1 tooth data from surviving adults sampled at Edix Hill. These appear in Figures 6.63-6.74, plotted as single-sex or comparative series.

Males

Figures 6.63 and 6.64 present $\delta^{13}C$ and $\delta^{15}N$ data for adult males in the Edix Hill sample.

Longitudinal childhood $\delta^{15}N$ patterns in adult males appear in Figure 6.63. $\delta^{15}N$ values are highly variable before the age of 2.5 years of age (range 9-15.2 ‰). Some display declining values from birth to 2.5, while others exhibit flat profiles with little variance. Following 2.5 years, the sample reflects a more constrained range of approximately 9-12 ‰ and appears less variable overall, with most exhibiting a flat profile after this age.

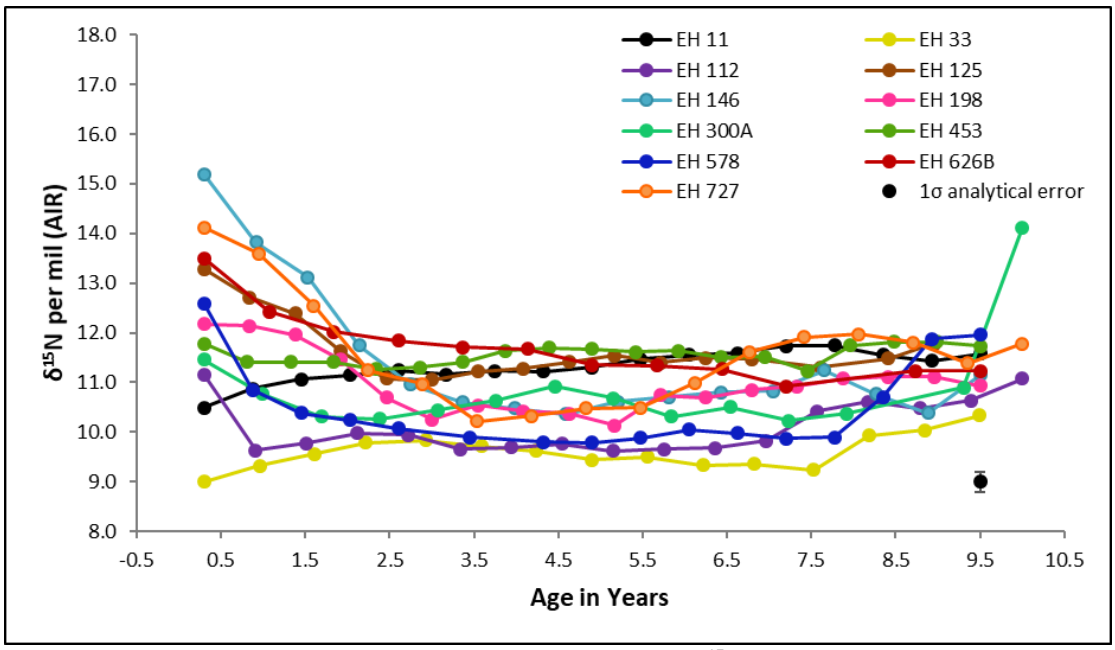

Figure 6.63 Plot of adult male M1 incremental dentine $\delta^{15}N$ data profiles for Edix Hill

Figure 6.64 shows grouped $\delta^{13}C$ profiles for Edix Hill male adults. As with the $\delta^{15}N$ data, the earliest-forming dentine demonstrates the greatest variability in $\delta^{13}C$ values (range −21.1-19.6 ‰), with most variability resolving by two years of age. Following this period, the sample displays a high degree of homogeneity in values, with less than 1 ‰ of variance between the high and low ends of the range in evidence.

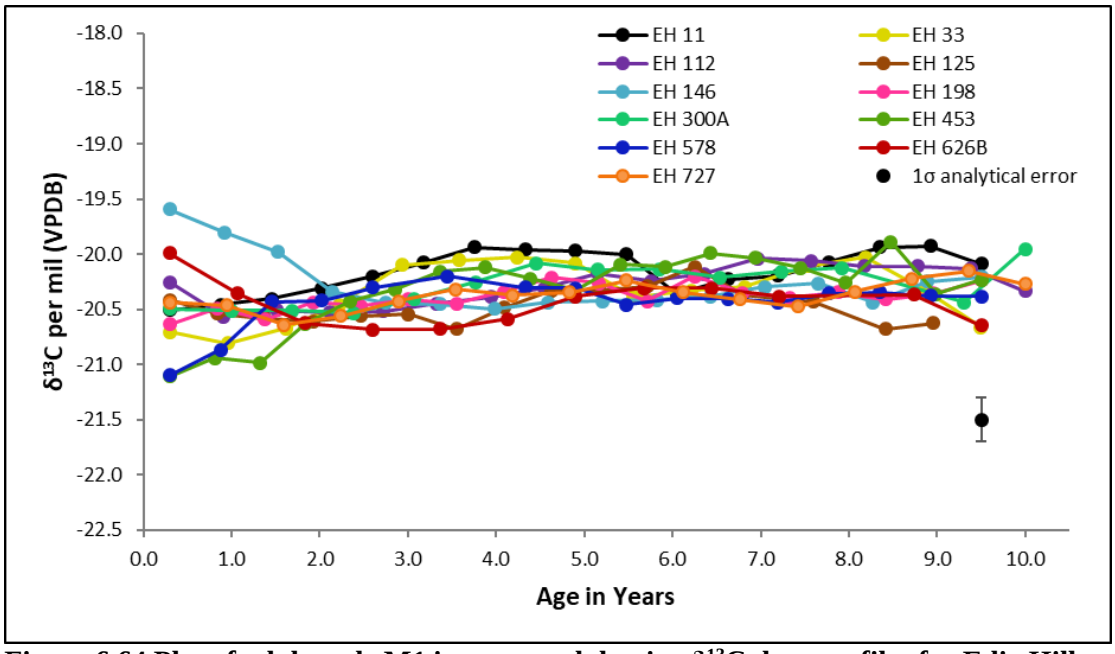

Figure 6.64 Plot of adult male M1 incremental dentine $\delta^{13}C$ data profiles for Edix Hill

Females

Figures 6.65 and 6.65 present $\delta^{13}C$ and $\delta^{15}N$ data for adult females in the Edix Hill sample.

Adult females at Edix Hill display a characteristically different pattern of change to their $\delta^{15}N$ values during childhood (Figure 6.65), when compared to their male counterparts at this site. Edix Hill females predominantly exhibit a decreasing curve over time, with few having a flat profile. In most individuals, this decline reaches a plateau by 2.5 years of age. While variability in females appears lower than that of males in terms of starting values in the first increment, over the longer-term overall variability appears broadly similar (maximum range 8.7-12.7 ‰).

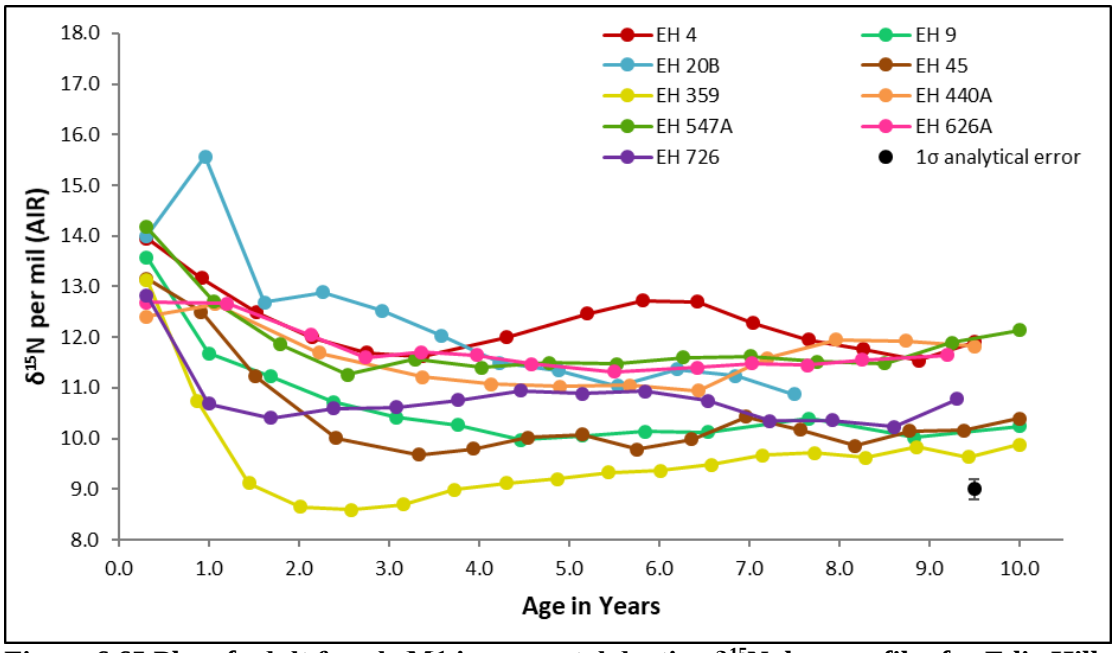

Figure 6.65 Plot of adult female M1 incremental dentine $\delta^{15}N$ data profiles for Edix Hill

Figure 6.66 shows longitudinal $\delta^{13}C$ data for females at Edix Hill. Initial increment $\delta^{13}C$ values, like female $\delta^{15}N$ values, are more constrained than those of Edix Hill males. The overall appearance of the collective female sample series is flat, with any pattern of decrease also subsiding by the age of 2.5 years. As with the male Edix Hill $\delta^{13}C$ data, variability beyond this age is below 1 ‰.

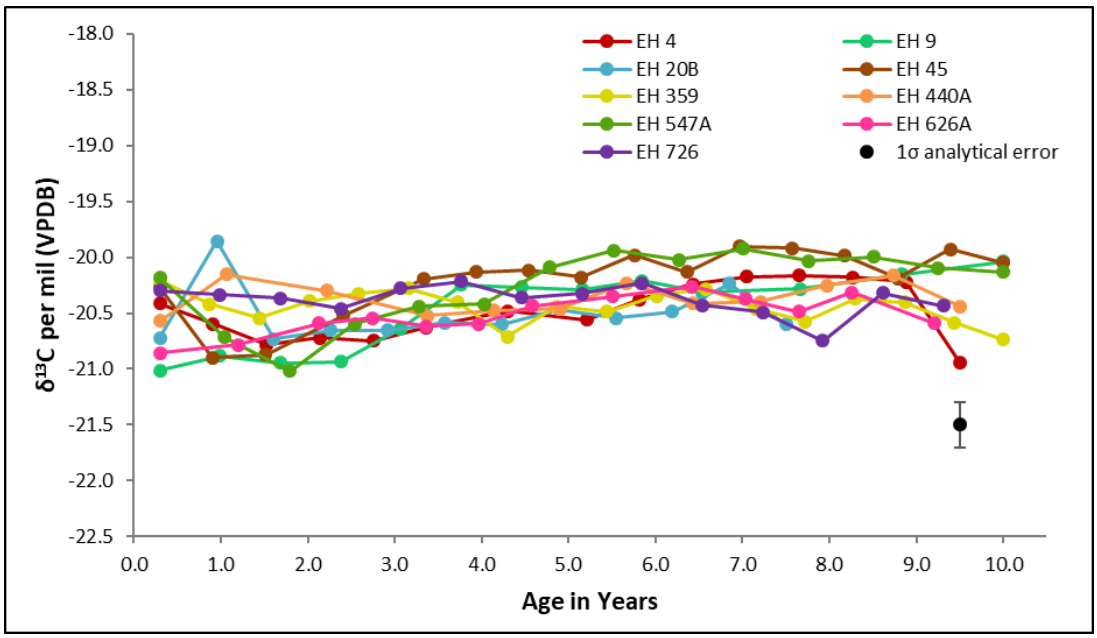

Figure 6.66 Plot of adult female M1 incremental dentine $\delta^{13}C$ data profiles for Edix Hill

Combined

Data were plotted for a combined adult sample by sex, and these appear in Figures 6.67-6.74.

Figure 6.67 shows $\delta^{15}N$ data plotted collectively for male and female adults at Edix Hill. Together the full adult sample shows a pattern of decreasing values up to the age of 2.5 years of age, with a stability in values thereafter. Two individuals present as exceptions to this trend, both females (EH 4 and EH 20B), and these continue to exhibit lability of values well outside of the main stratum of data.

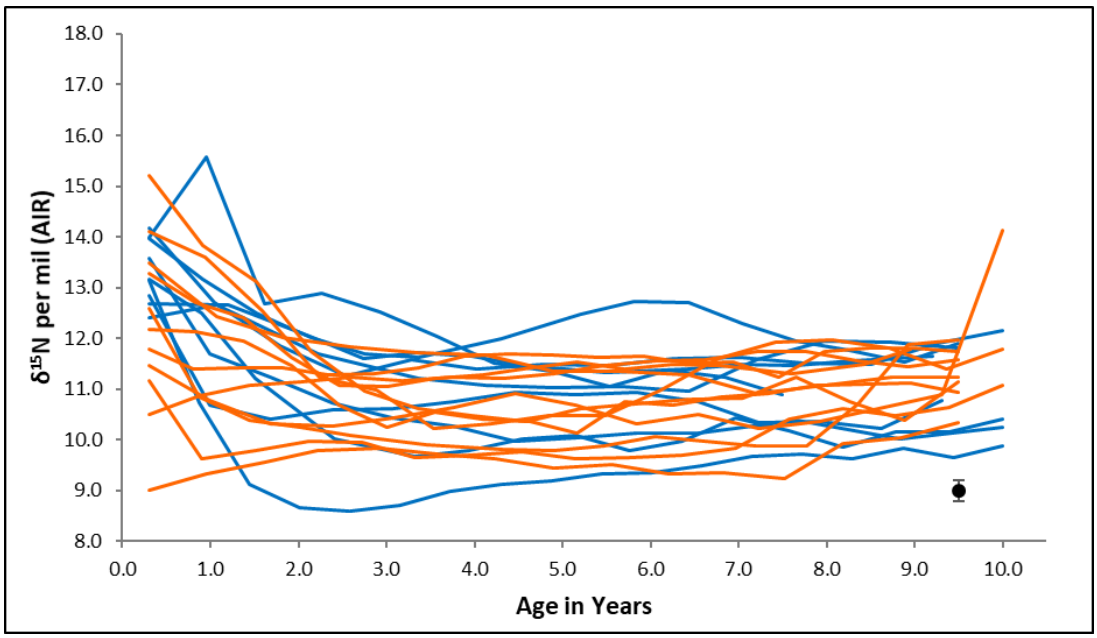

Figure 6.67 Spaghetti plot of M1 incremental dentine $\delta^{15}N$ data profiles at Edix Hill (male individuals shown in orange, females in blue)

Figure 6.68 shows longitudinal $\delta^{13}C$ data plotted for the combined male and female sample. Maximum population variability occurs before the age of 1.5 years of age, with a tightly clustered pattern of linear data in evidence beyond 2.5 years of age. No meaningful differences in pattern between male and female distributions are evident.

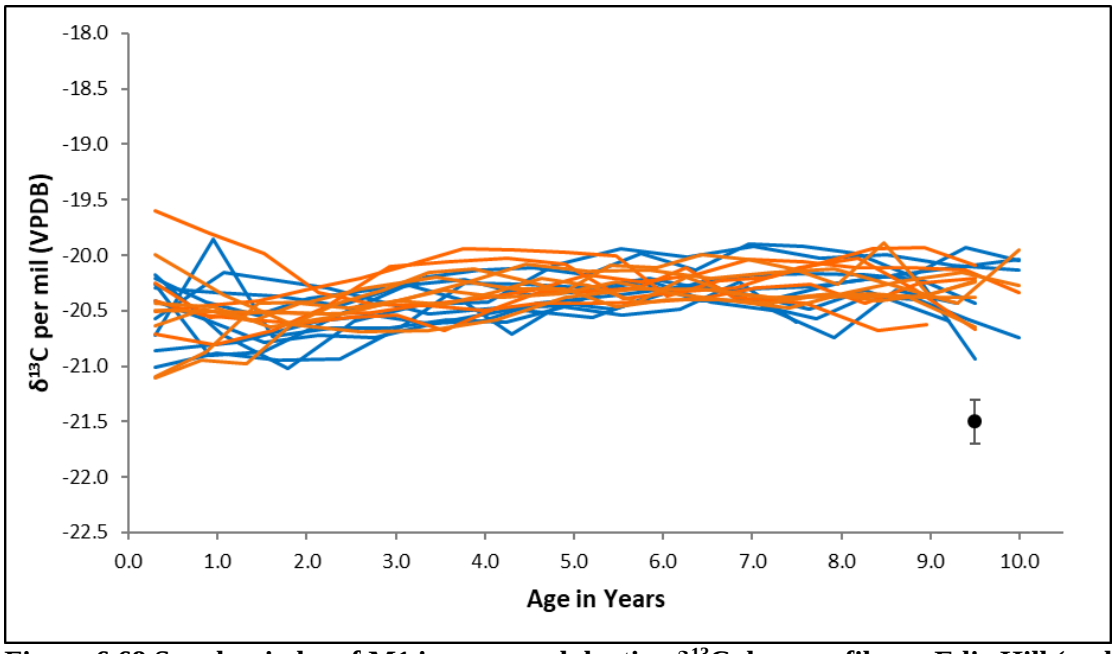

Figure 6.68 Spaghetti plot of M1 incremental dentine $\delta^{13}C$ data profiles at Edix Hill (male individuals shown in orange, females in blue)

Individual increment data is plotted for the entirety of the Edix Hill M1 sample as $\delta^{13}C$ vs $\delta^{15}N$ in Figure 6.69.

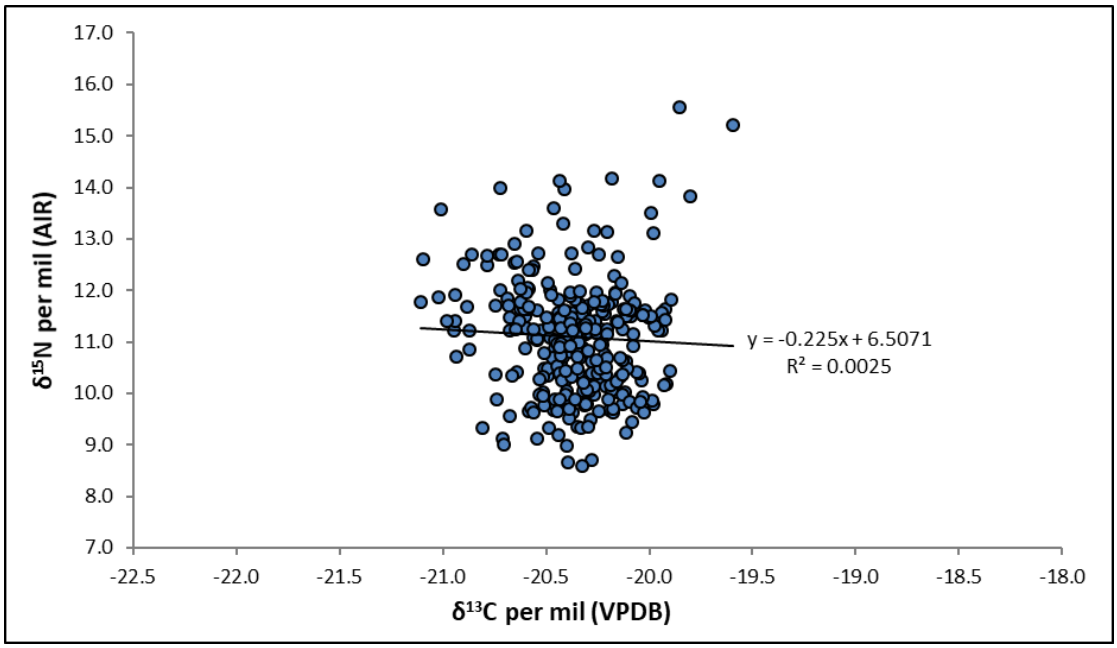

Figure 6.69 $\delta^{13}C$ vs $\delta^{15}N$ plot for all M1 dentine increments in the Edix Hill sample, showing no correlation (r^2= 0.003), and a slope of -0.2

Figure 6.70 shows the total M1 sample as $\delta^{13}C$ vs $\delta^{15}N$, in order to identify differences in patterning based on estimated sex. Male and female incremental data appear to have a similar range and pattern of distribution, with males appearing only marginally more centralised in distribution of data than females.

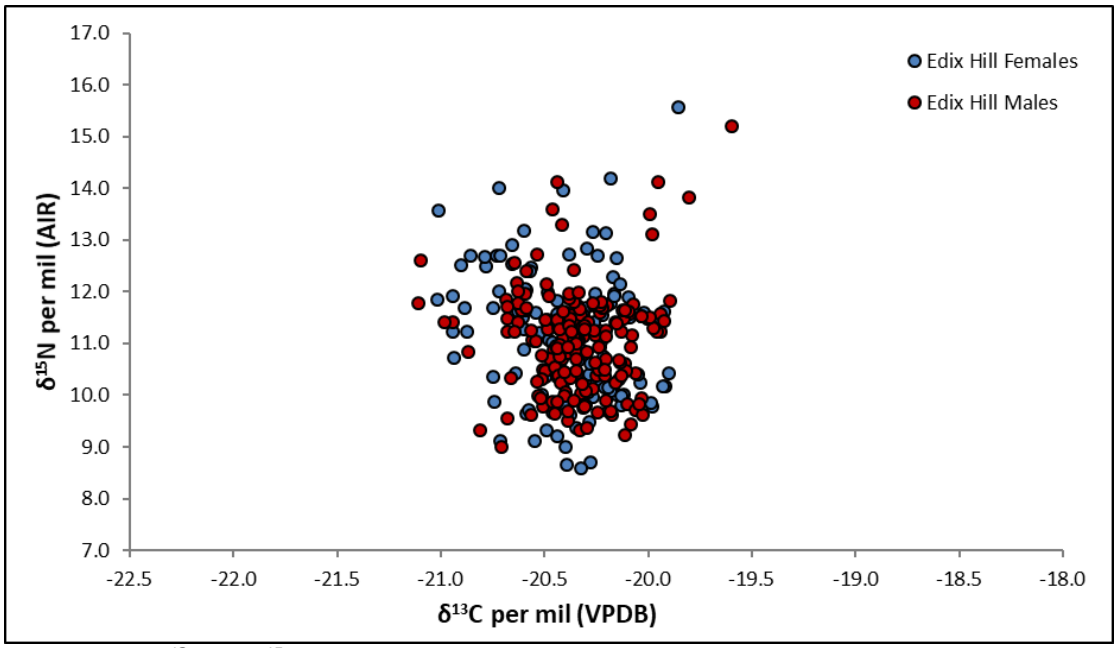

Figure 6.70 $\delta^{13}C$ vs $\delta^{15}N$ plot for all M1 dentine increments in the Edix Hill sample by estimated sex

6.3.1.iii Comparative survivor/non-survivor data

This section will present comparative data for the combined sample of surviving adults and non-surviving children at Edix Hill.

Non-survivor $\delta^{15}N$ data profiles are plotted alongside survivors in Figure 6.71. Most non-survivors fit within the broader range of $\delta^{15}N$ defined by survivors in this population, albeit having values which are at the high end of the range. Two exceptional individuals, EH 352 and EH 547B, fall outside of the main body of data for this population initially, but enter the main range by one year of age.

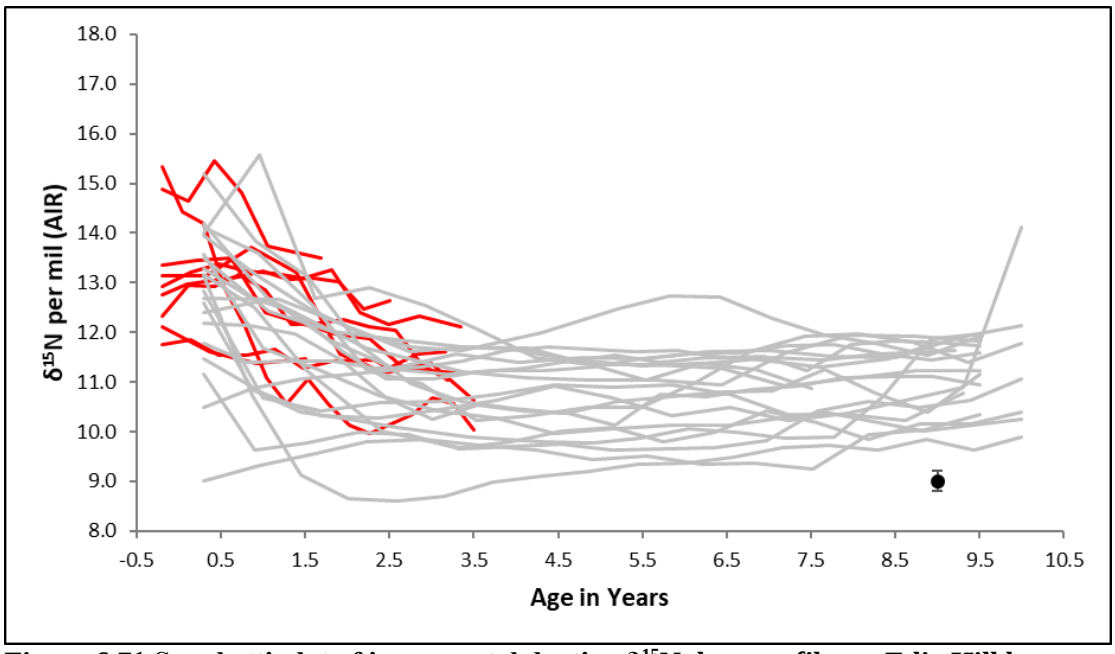

Figure 6.71 Spaghetti plot of incremental dentine $\delta^{15}N$ data profiles at Edix Hill by survivorship (non-surviving juveniles shown in red, surviving adult population shown in grey for reference)

Figure 6.72 shows non-survivor and survivor longitudinal $\delta^{13}C$ data profiles plotted together. Non-survivor $\delta^{13}C$ data also appears at the high end of the surviving population range, but broadly all values conform to the larger population range.

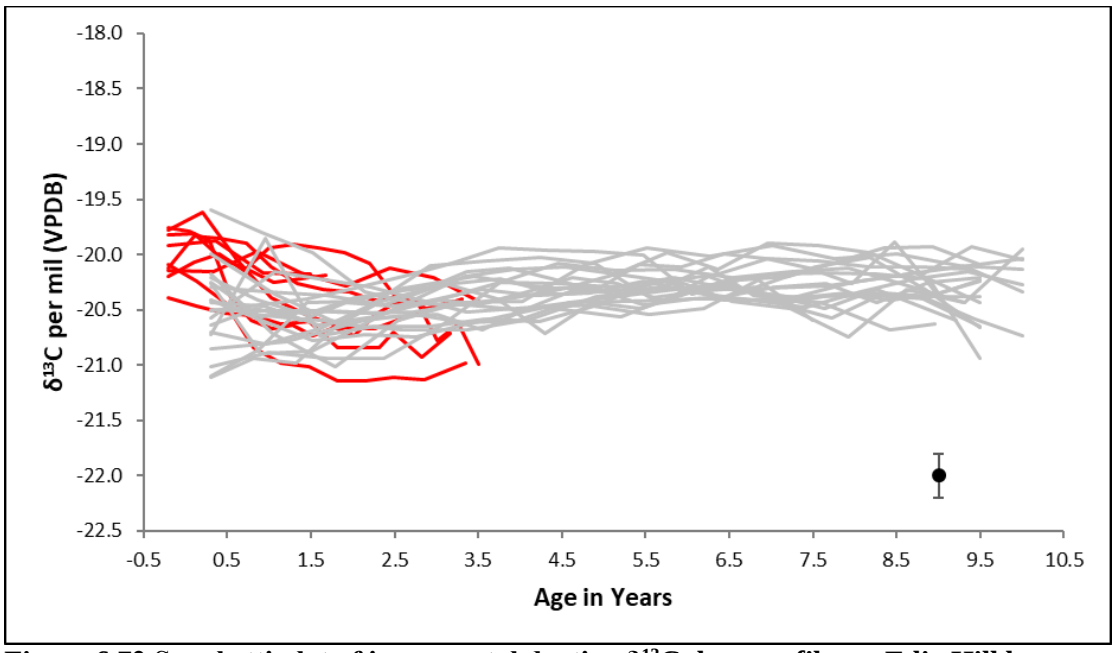

Figure 6.72 Spaghetti plot of incremental dentine $\delta^{13}C$ data profiles at Edix Hill by survivorship (non-surviving juveniles shown in red, surviving adult population shown in grey for reference)

Plotted $\delta^{15}N$ data profiles for individuals with skeletal stress markers in the Edix Hill sample appear in Figure 6.73. No differences between $\delta^{15}N$ values or patterns of change

between individuals with stress markers and other members of the population were observable.

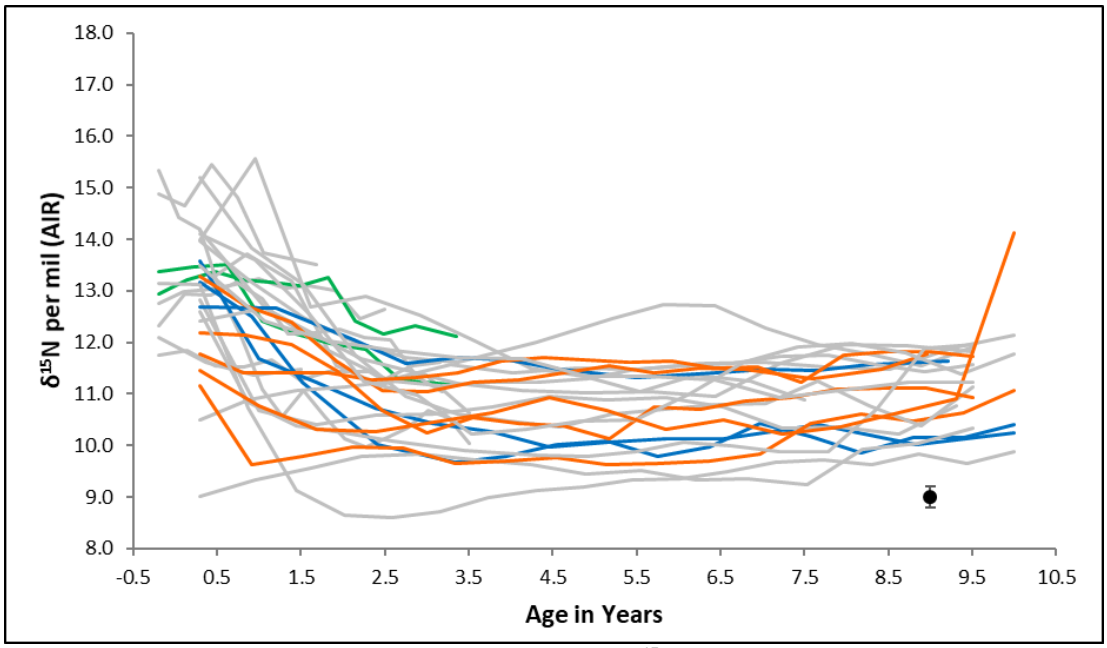

Figure 6.73 Spaghetti plot of incremental dentine $\delta^{15}N$ data profiles for individuals at Edix Hill with skeletal stress markers (male individuals shown in orange, females in blue, non-adults shown in green, with non-stress marker-associated data shown in grey for reference)

Figure 6.74 shows plotted $\delta^{13}C$ data profiles for individuals with skeletal stress makers at Edix Hill. In early-forming dentine, $\delta^{13}C$ sit at the lower end of the population range, but still within this larger range, and values are found within the centre of the range by later childhood. One non-adult individual, EH 133, deviates from this range by 1.5 years of age, demonstrating the lowest values in the sample.

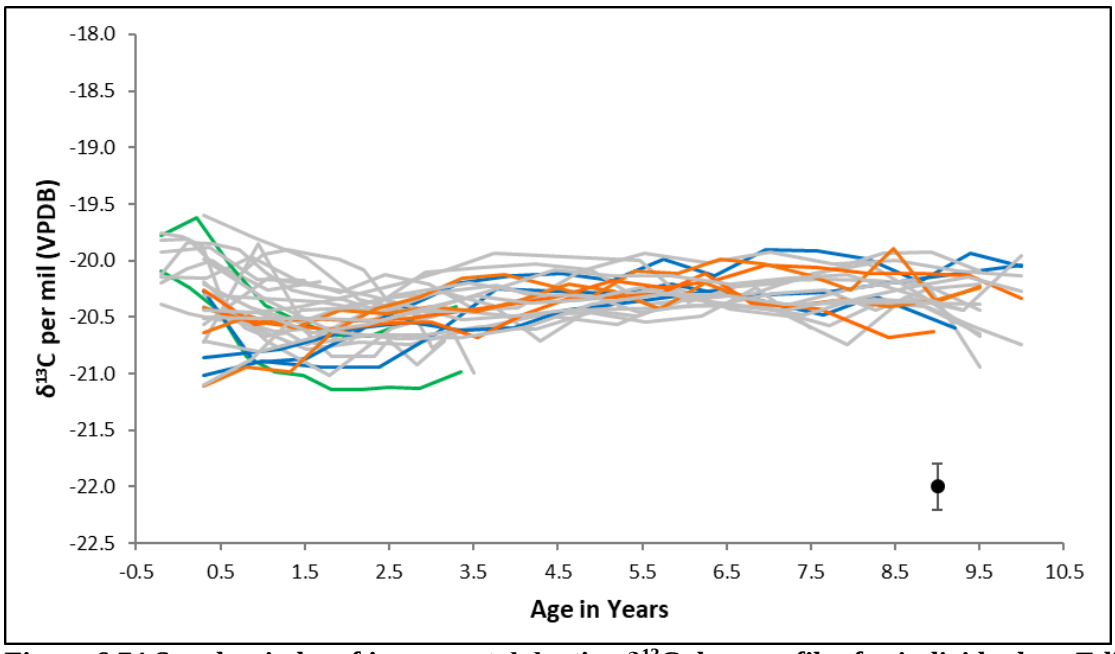

Figure 6.74 Spaghetti plot of incremental dentine $\delta^{13}C$ data profiles for individuals at Edix Hill with skeletal stress markers (male individuals shown in orange, females in blue, non-adults shown in green, with non-stress marker -associated data shown in grey for reference)

Figure 6.75 presents longitudinal $\delta^{15}N$ data for sampled individuals at Edix Hill with the specific skeletal stress marker cribra orbitalia. Only two individuals in the sample (EH 198 and EH 133) had this lesion, and neither of these were exceptional in pattern to the larger sample.

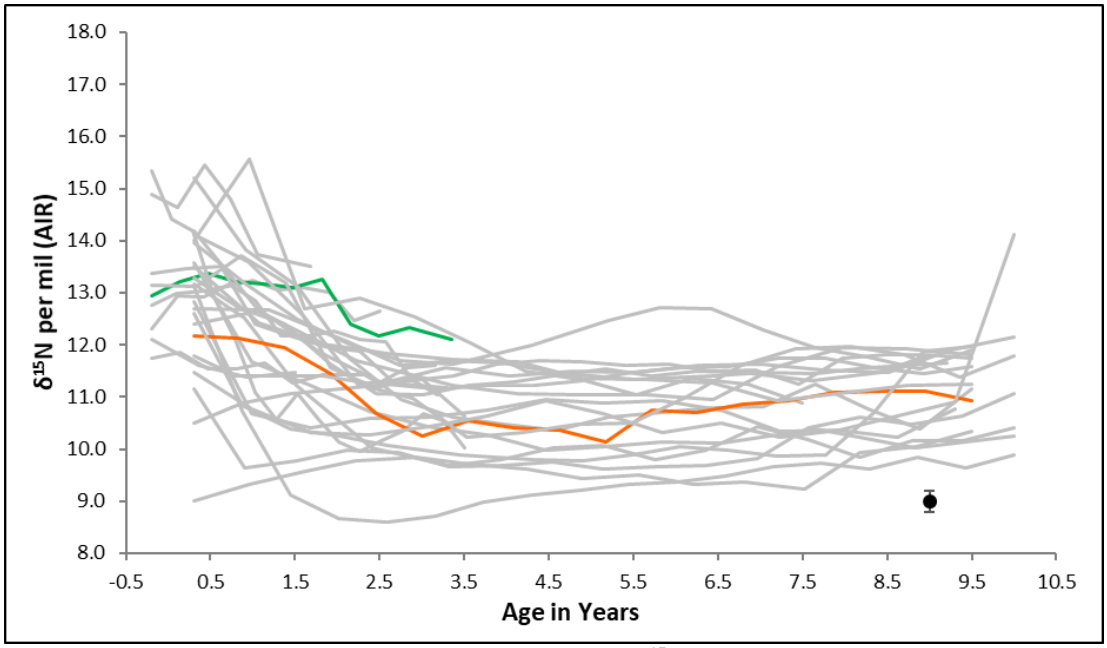

Figure 6.75 Spaghetti plot of incremental dentine $\delta^{15}N$ data profiles for individuals at Edix Hill with cribra orbitalia (male individual EH 198 is shown in orange, non-adult EH 133 shown in green, with non-cribrotic individuals shown in grey for reference)

Figure 6.76 displays the $\delta^{13}C$ profile of individuals at Edix Hill with cribra orbitalia. As with the $\delta^{15}N$, EH 198 plotted centrally with other non-cribrotic individuals. EH 133, however, exhibited a pattern of lower values than the remaining population.

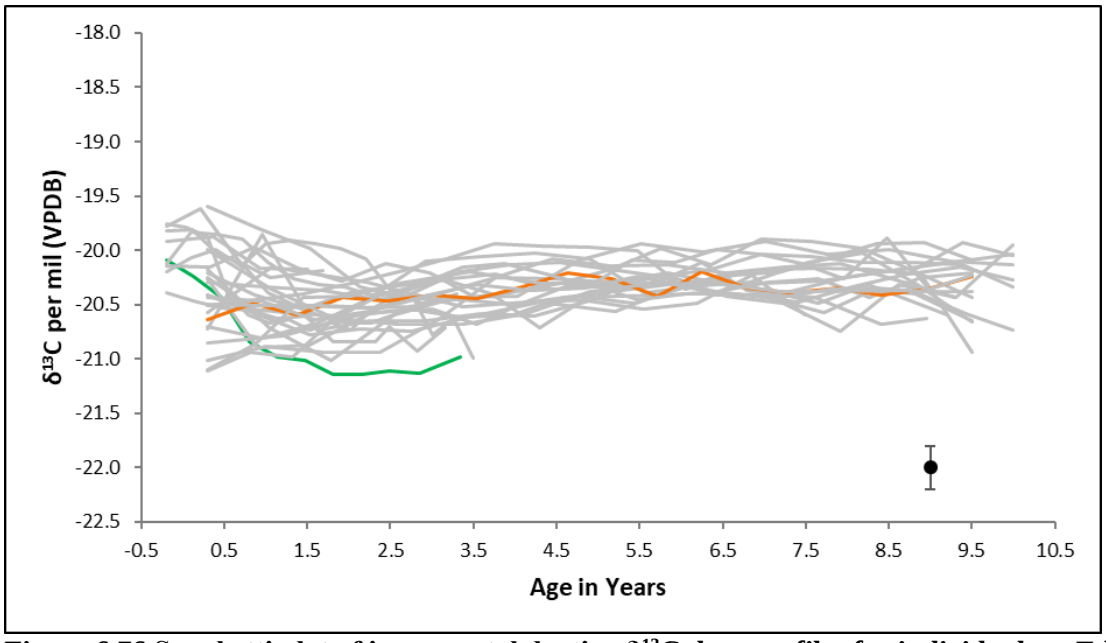

Figure 6.76 Spaghetti plot of incremental dentine $\delta^{13}C$ data profiles for individuals at Edix Hill with cribra orbitalia (male individual EH 198 is shown in orange, non-adult EH 133 shown in green, with non-cribrotic individuals shown in grey for reference)

Figure 6.77 shows the full sample of incremental data for Edix Hill plotted as $\delta^{13}C$ vs. $\delta^{15}N$. Non-survivors exhibit a more linear pattern of distribution with a positive correlation between increasing $\delta^{13}C$ and $\delta^{15}N$, which is not in evidence for surviving adults of either sex.

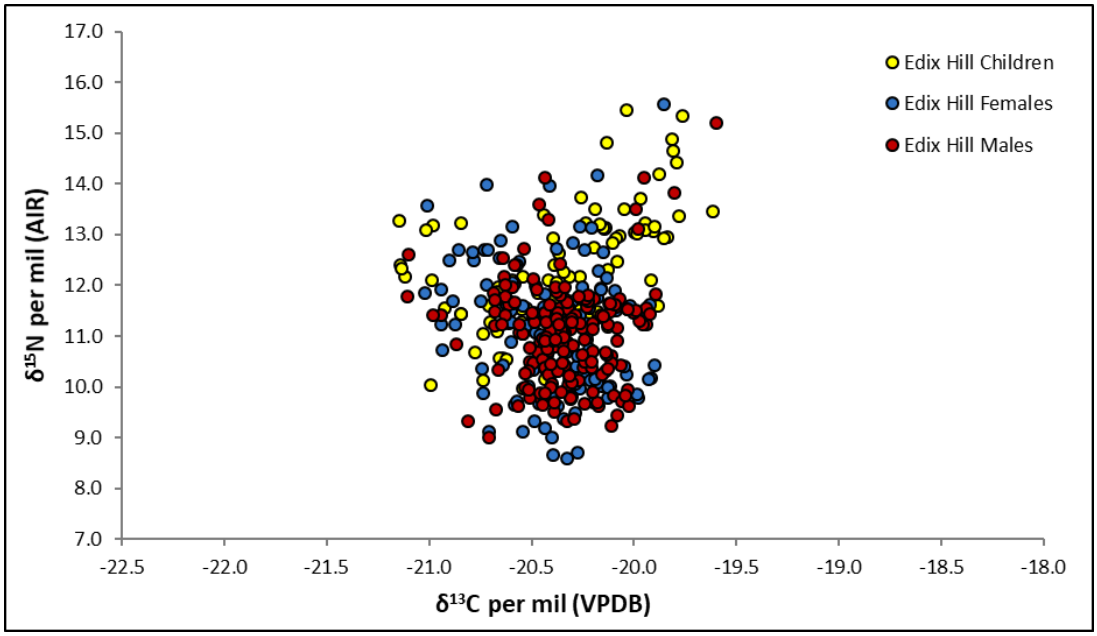

Figure 6.77 $\delta^{13}C$ vs $\delta^{15}N$ plot for all dentine increments in the Edix Hill sample by estimated sex and age-at-death

6.3.2 Littleport results

Comparative data for Littleport are summarised in Figures 6.78-6.95. Sections 6.3.2.i and 6.3.2.ii present dm2 and M1 tooth data summaries, respectively, while Section 6.3.2.iii presents a comparison of survivor (M1) and non-survivor (dm2) data.

6.3.2.i Deciduous tooth data

Summary plots for dm2 tooth data from non-surviving children at Edix Hill appear in Figures 6.78-6.80.

Figure 6.78 shows grouped $\delta^{15}N$ data for the non-survivor cohort at Littleport. Note the lack of flat profiles and the two-tiered grouping of data, with LP 3770 and LP 4848 displaying much lower $\delta^{15}N$ values than the other four non-survivors, from the earliest data. These high and low patterns in the data begin to disappear by around 2 years of age, as the groups converge.

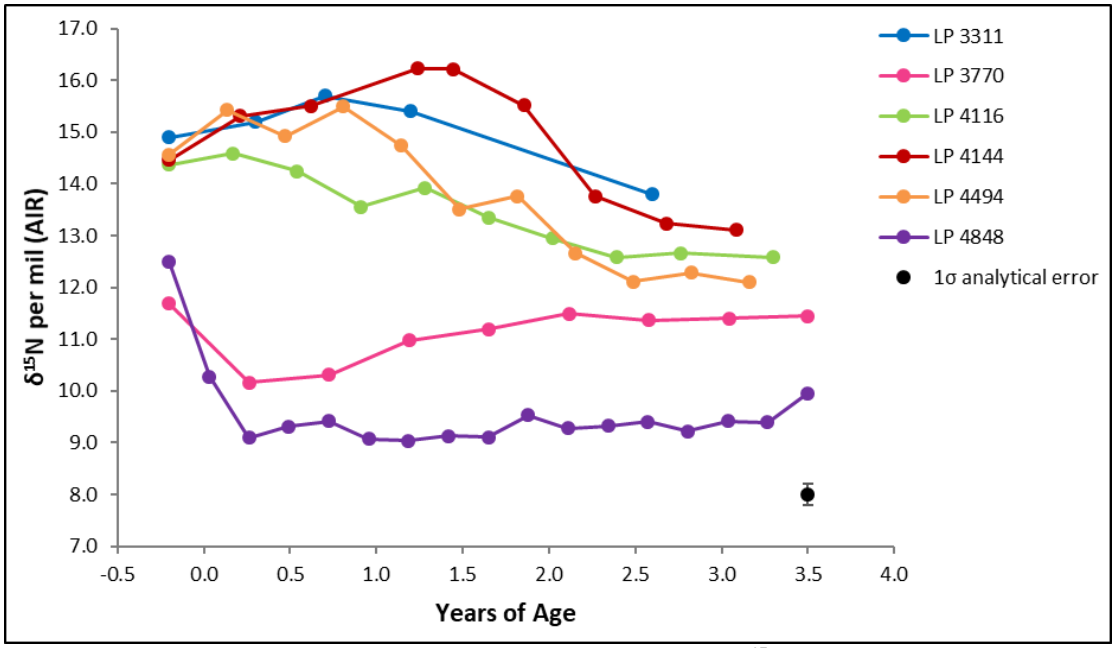

Figure 6.78 Plot of non-survivor dm2 incremental dentine $\delta^{15}N$ data profiles for Littleport

Figure 6.79 shows incremental dentine $\delta^{13}C$ data for the non-survivor sample at Littleport. There is a clustering of antenatal (initial increment) values. Differences in $\delta^{13}C$ trajectory are not apparent in the early postnatal dentine profiles, as they are in the $\delta^{15}N$ data. However, by one year of age, the sample diverges into the same groups in evidence in the $\delta^{15}N$ series data, with LP 3770 and LP 4848 occupying a higher $\delta^{13}C$ range than the other four non-survivors.

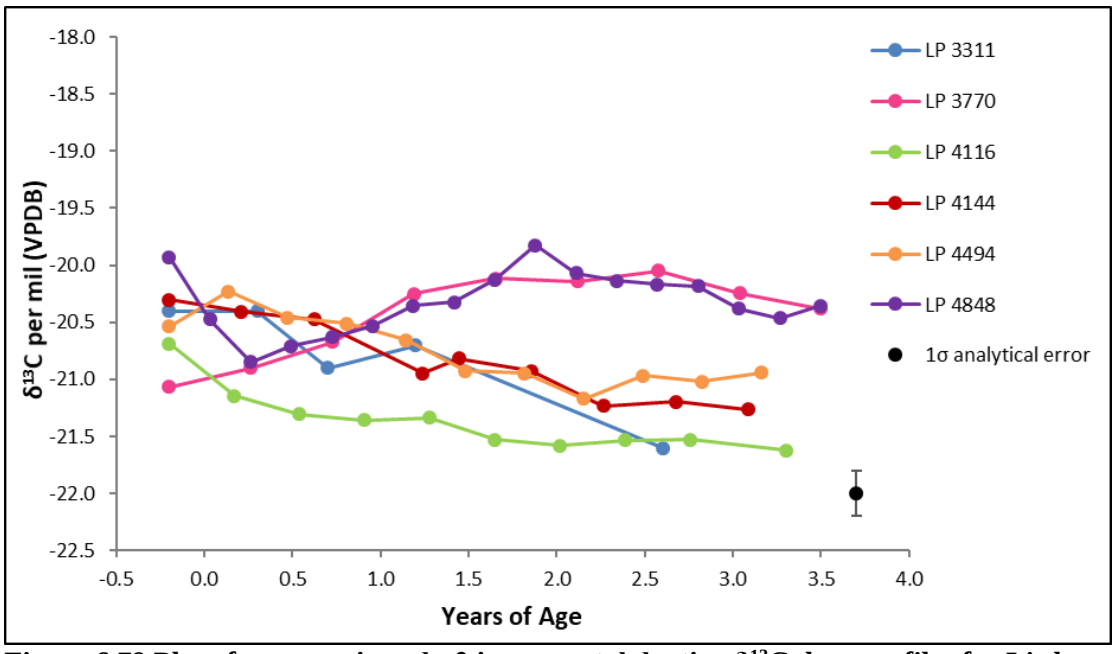

Figure 6.79 Plot of non-survivor dm2 incremental dentine $\delta^{13}C$ data profiles for Littleport

Figure 6.80 shows the Littleport dm2 dentine sample plotted as $\delta^{13}C$ vs. $\delta^{15}N$ and displays a much more scattered pattern than the Edix Hill sample, additionally having a negative slope.

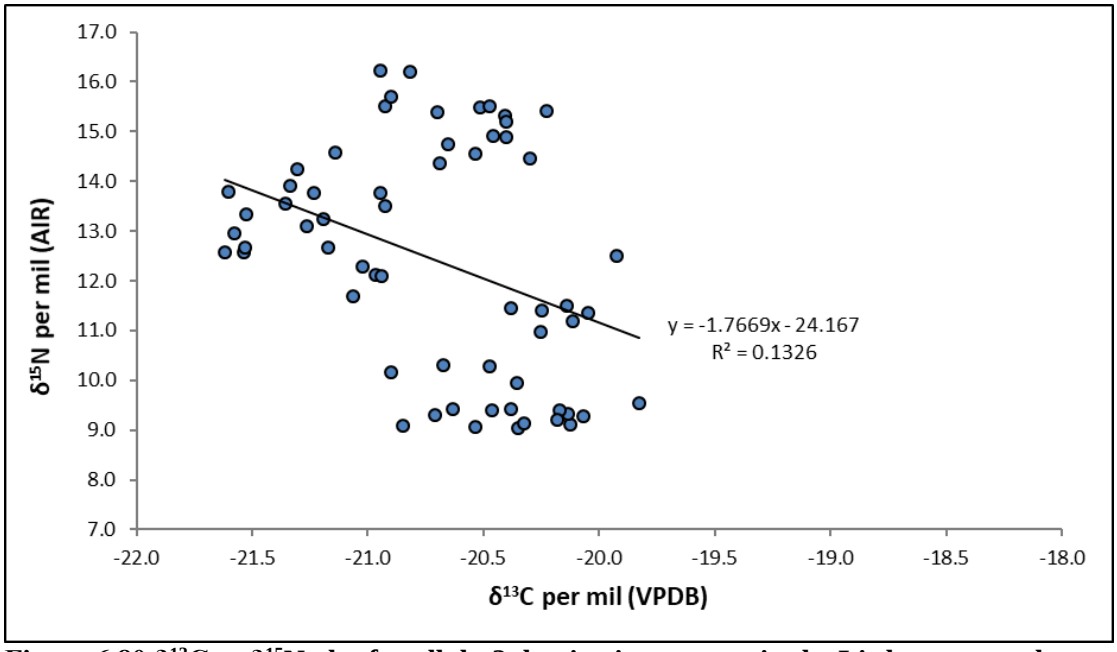

Figure 6.80 $\delta^{13}C$ vs $\delta^{15}N$ plot for all dm2 dentine increments in the Littleport sample, showing no correlation (r^2= 0.13), and a slope of -1.8

6.3.2.ii Permanent tooth data

This section presents summary plots for M1 tooth data from surviving adults at Littleport. These appear in Figures 6.81-6.92, plotted as single-sex or comparative series.

Males

Figures 6.81 and 6.82 present $\delta^{13}C$ and $\delta^{15}N$ data for adult males in the Littleport sample.

Longitudinal childhood $\delta^{15}N$ patterns in adult males at Littleport are shown in Figure 6.81. A high degree of homogeneity is observable among males in this sample, with the exceptions of LP 3708, LP 3893, and LP 4178, who deviate from the predominant range of Littleport males at differing intervals. Most individuals exhibit a decrease from the first increment (midpoint value of 0.3 years) to the second increment (approximately 1 year of age) with relative stasis thereafter, or an overall flat profile. Exceptional individuals, such as LP 4178 or LP 3708 and LP 3893 exhibit dropping $\delta^{15}N$ values over a longer period, extending to 2.5-3 years of age. In the case of the latter two individuals, this results in the occupation of a different range of values than the majority during early childhood.

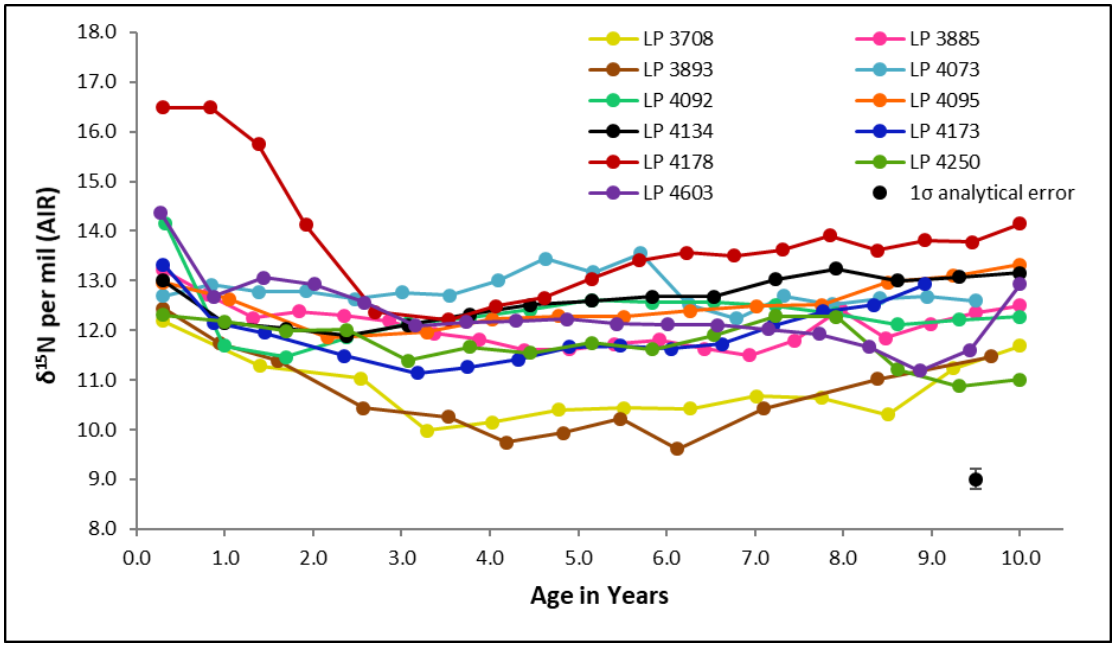

Figure 6.81 Plot of adult male M1 incremental dentine $\delta^{15}N$ data profiles for Littleport

Figure 6.82 shows grouped $\delta^{13}C$ profiles for Littleport male adults. Like the pattern seen in Littleport male $\delta^{15}N$ profiles, a pattern of minor decrease between first and second increments, or a flat appearance throughout, predominated. Overall variability was high, at 2.7 ‰, with most of this variability being accounted for by two individuals, LP 4095 and LP 4178.

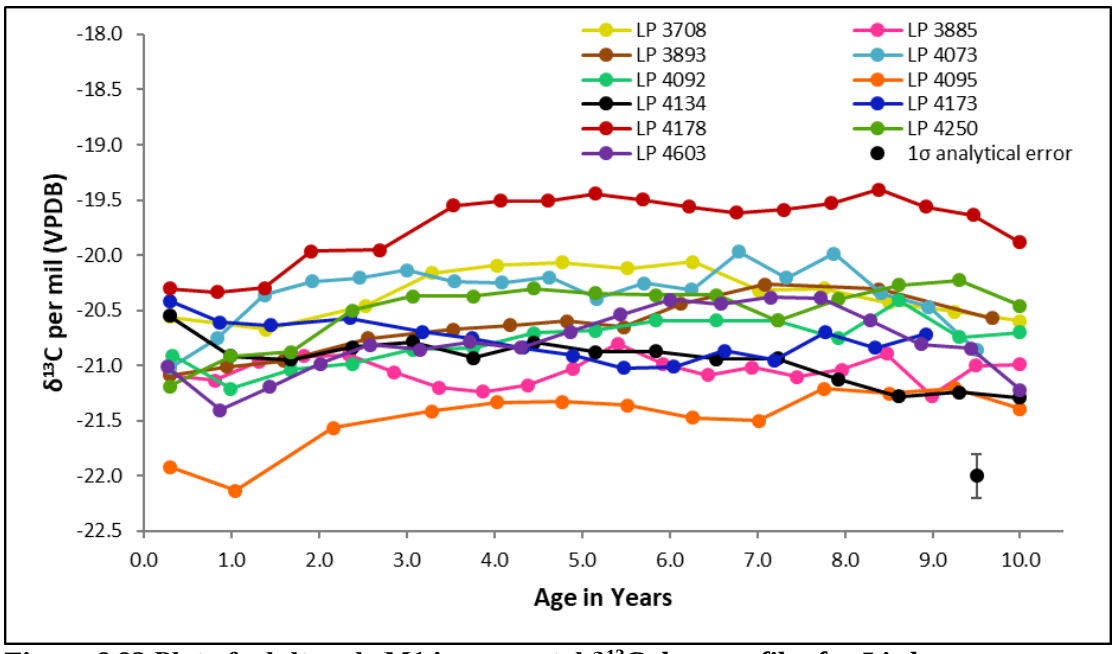

Figure 6.82 Plot of adult male M1 incremental $\delta^{13}C$ data profiles for Littleport

Females

Figures 6.83 and 6.84 show $\delta^{13}C$ and $\delta^{15}N$ data for adult females in the Littleport sample.

Figure 6.83 presents incremental dentine $\delta^{15}N$ data for adult females at Littleport. Nearly all individuals showed a pattern of decreasing values during infancy (with the exception of LP 4035). Within individuals demonstrating this trend, differences in starting value and rate of decrease $\delta^{15}N$ values between the first and second increment demonstrated a two-tiered divergence in range between halves of the sample emerging around a year of age. The lower tier showed stability in values by 1-2.5 years of age, ranging over the longer-term from 9.8-10.9 ‰. The upper tier of $\delta^{15}N$ values declined more gradually, reaching a plateau by 2-3 years of age, and following a range of 12-13.8 ‰. One individual, LP 4585, exhibited a pattern of values within the lower tier up to the age of approximately 6.5 years old. Following this period, her values steadily increased to join the upper range by 8.5 years. Similarly, LP 4063's values appeared to decrease after 8.5 to join the lower range by the end of the $\delta^{15}N$ tooth data series.

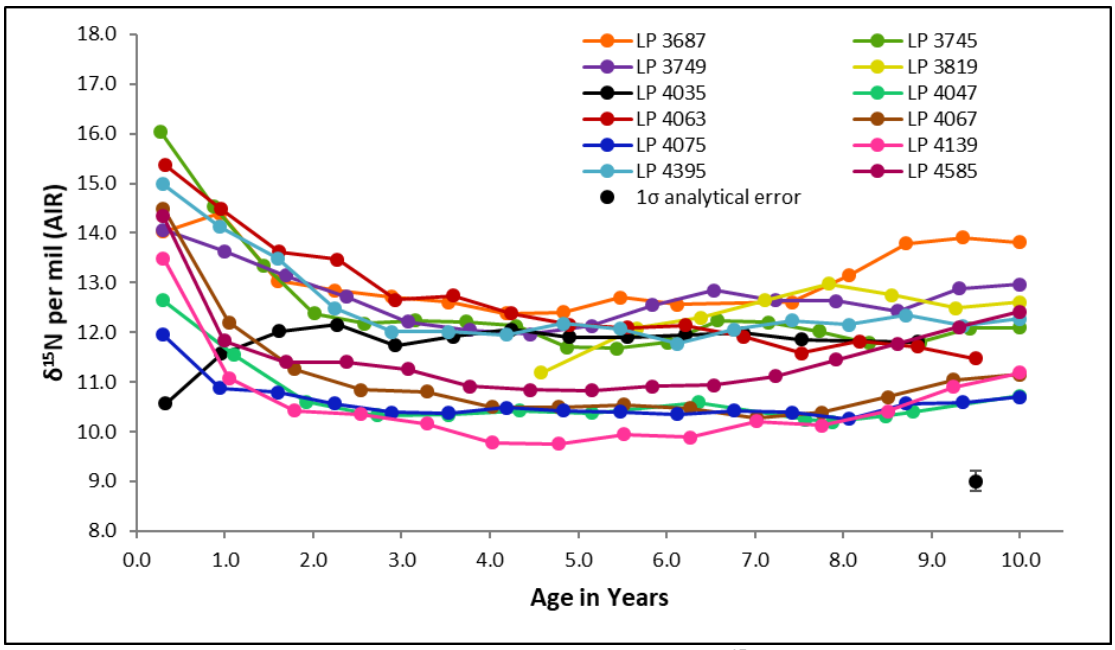

Figure 6.83 Plot of adult female M1 incremental dentine $\delta^{15}N$ data profiles for Littleport

Figure 6.84 shows grouped $\delta^{13}C$ profiles for Littleport female adults. The range of variability in values (1.6 ‰) was much lower than was observed for Littleport male adults. Overall, the appearance of the group data presents as a flat profile. However, when viewed individually in terms of changes to $\delta^{13}C$ during the first years of life, the females in the sample present as a mix of profiles exhibiting increasing or decreasing $\delta^{13}C$ values. Most fluctuation in this early data resolves by 2-3 years of age.

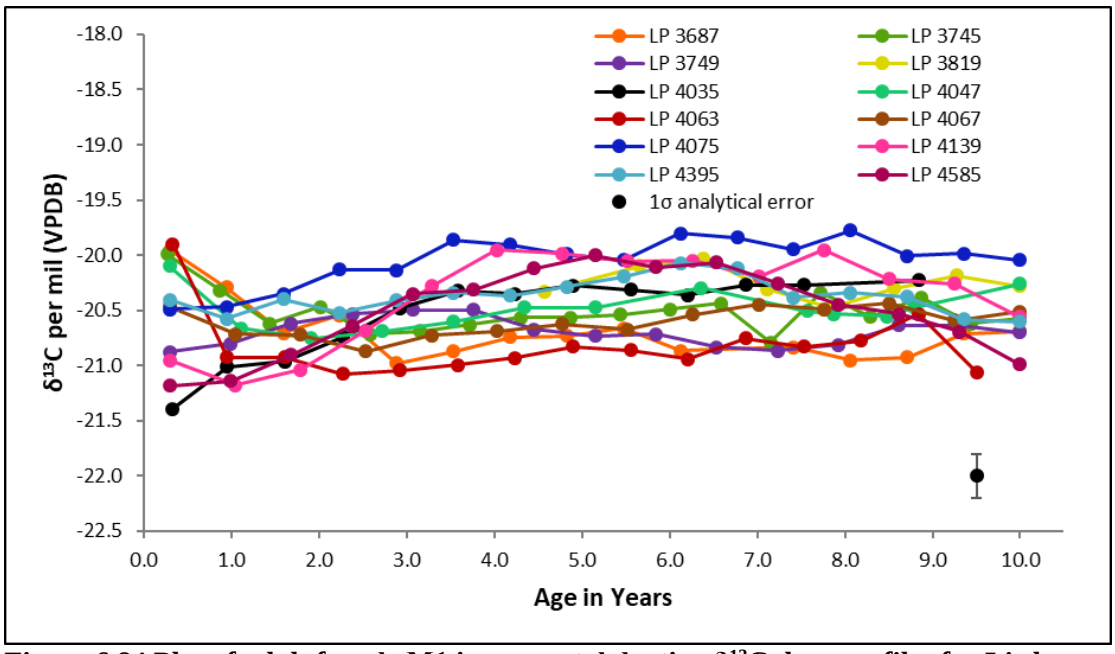

Figure 6.84 Plot of adult female M1 incremental dentine $\delta^{13}C$ data profiles for Littleport

Combined

Data were plotted for a combined adult sample by sex, and these appear in Figures 6.85-6.92.

Figure 6.85 shows $\delta^{15}N$ data plotted collectively for sampled male and female adults at Littleport. Together, the full adult sample shows a pattern of decreasing values up to the age of 2.5 years of age, with values predominantly stable after this age. The divergence of tiers of post-infancy data visible previously in the female sample is repeated in the combined data, and the male sample is heavily biased towards the upper range of data, with only two male individuals and one indeterminate individual appearing in the lower female range.

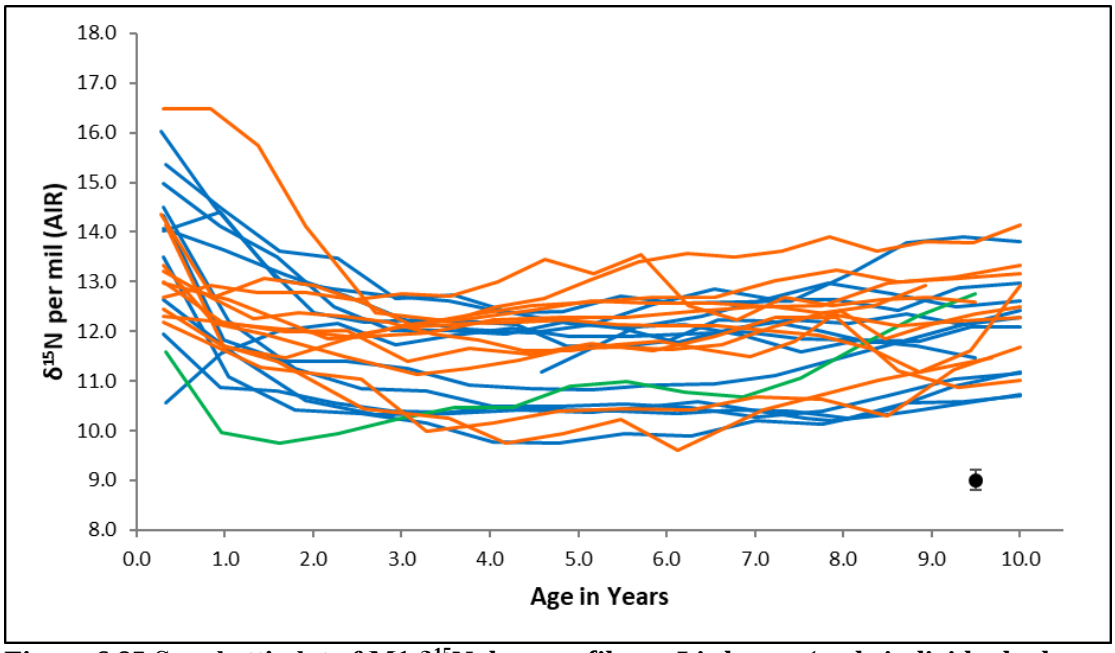

Figure 6.85 Spaghetti plot of M1 $\delta^{15}N$ data profiles at Littleport (male individuals shown in orange, females in blue, indeterminate individual shown in green)

Figure 6.86 shows longitudinal $\delta^{13}C$ data plotted for the combined male and female sample at Littleport. Maximum population variability occurs before the approximate age of 2.5 years, but high levels of variability are visible throughout, with most accounted for by the distribution of male data series, especially the contributions of atypical males.

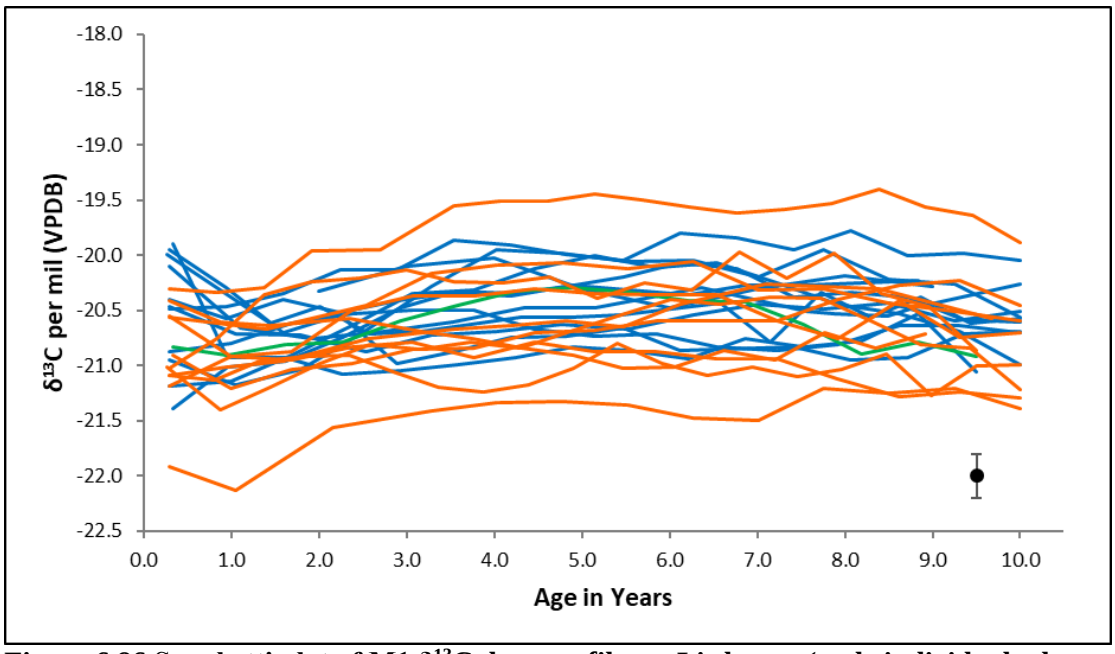

Figure 6.86 Spaghetti plot of M1 $\delta^{13}C$ data profiles at Littleport (male individuals shown in orange, females in blue, indeterminate individual shown in green)

Individual increment data is plotted for the entirety of the Littleport M1 sample as $\delta^{13}C$ vs $\delta^{15}N$ in Figure 6.87.

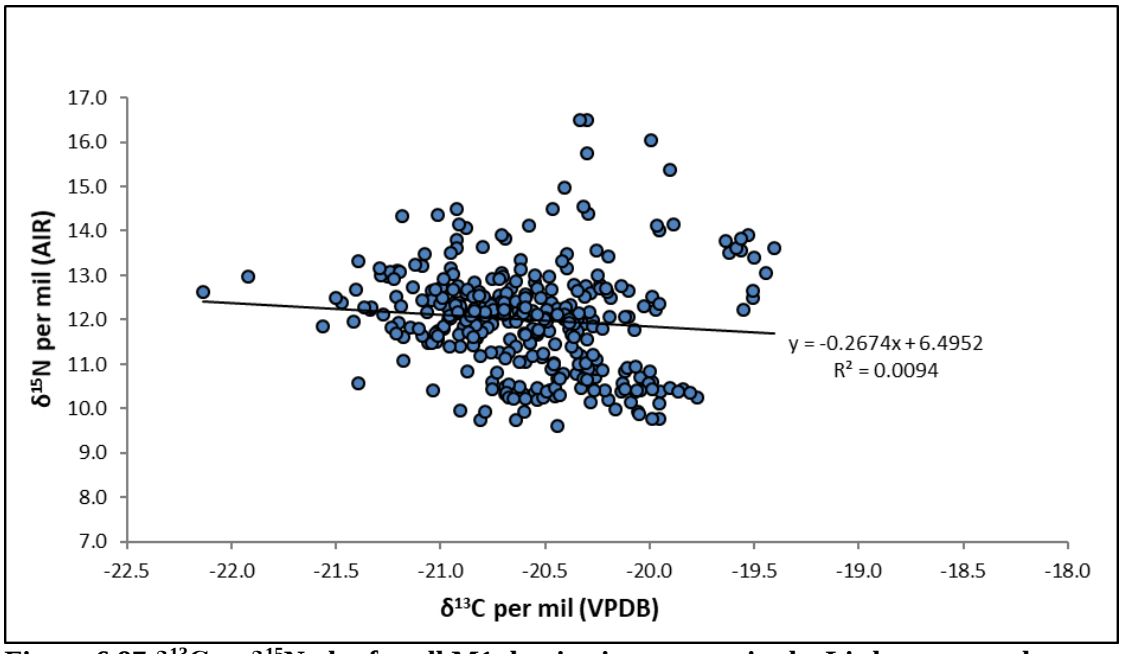

Figure 6.87 $\delta^{13}C$ vs $\delta^{15}N$ plot for all M1 dentine increments in the Littleport sample, showing no correlation (r^2= 0.01), and a slope of -0.3

Figure 6.88 shows the Littleport M1 sample plotted as $\delta^{13}C$ vs $\delta^{15}N$ by estimated sex.

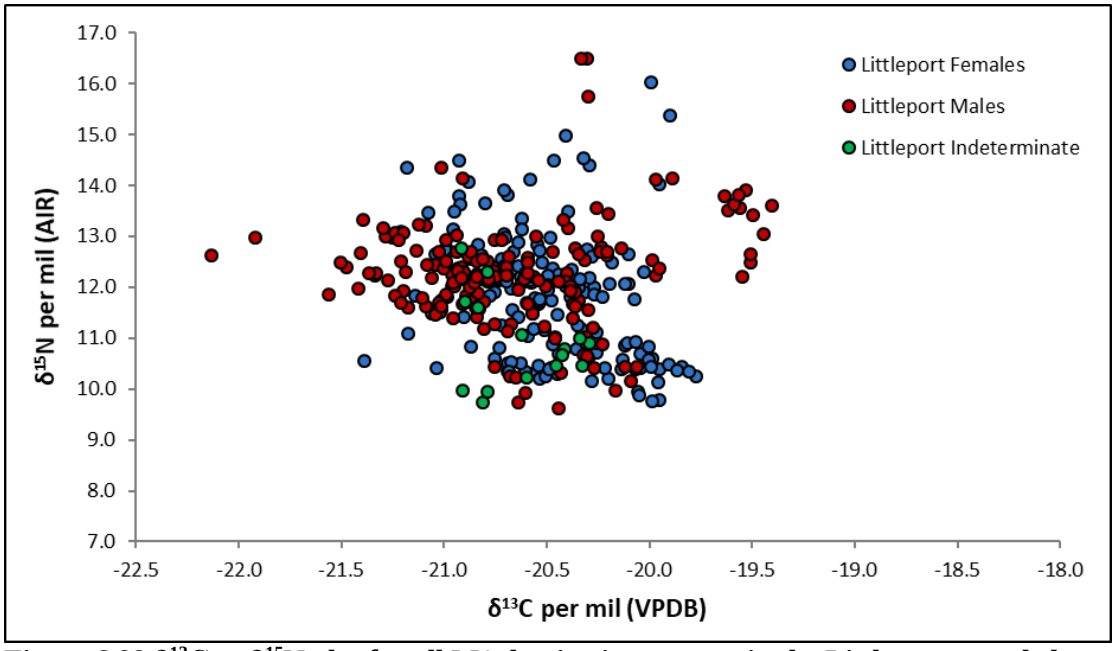

Figure 6.88 $\delta^{13}C$ vs $\delta^{15}N$ plot for all M1 dentine increments in the Littleport sample by estimated sex

6.3.2.iii Comparative survivor/non-survivor data

This section will present comparative data for the combined sample of surviving adults and non-surviving children at Littleport.

Non-survivor $\delta^{15}N$ data profiles are plotted alongside survivors in Figure 6.89. In contrast to Edix Hill, most non-survivors at Littleport do not fall within the most concentrated central distribution of data. Instead, survivors plot at the extreme high and low ends of the sample $\delta^{15}N$ range. Extremes are not exhibited throughout the data series, as non-survivor $\delta^{15}N$ predominantly fall within the main range by 2.5 years.

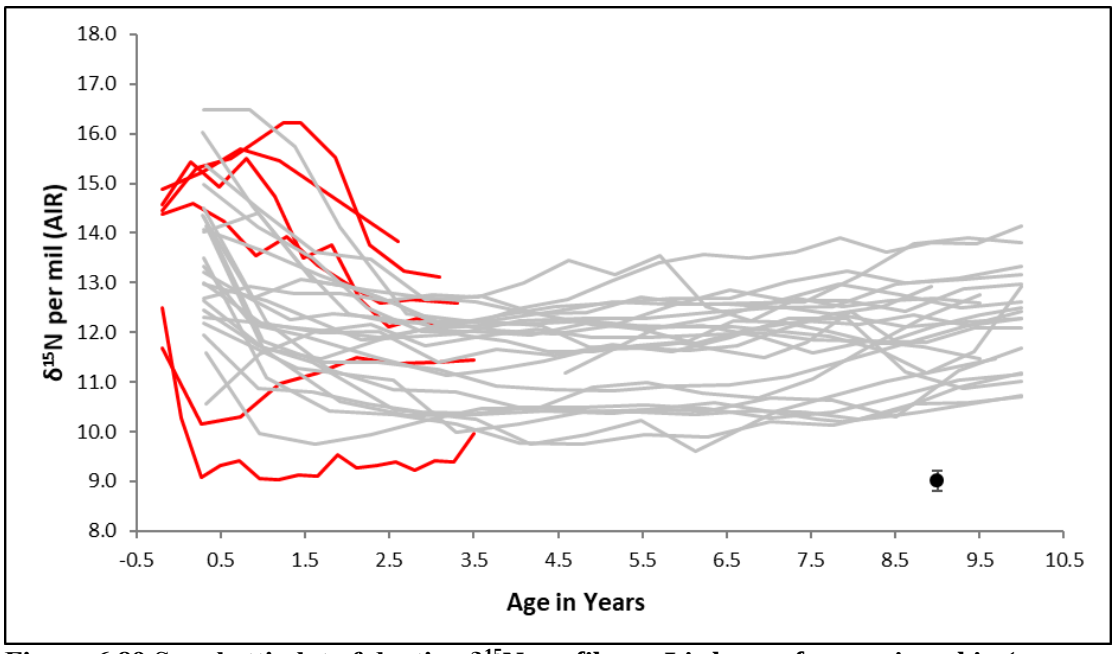

Figure 6.89 Spaghetti plot of dentine $\delta^{15}N$ profiles at Littleport for survivorship (non-surviving juveniles shown in red, surviving adult population shown in grey)

Figure 6.90 shows non-survivor and survivor longitudinal $\delta^{13}C$ data profiles for Littleport plotted together. As with Littleport $\delta^{15}N$ data profiles, non-survivor $\delta^{13}C$ data also appears at both high and low ends of the surviving population range by 2.5 years of age, despite having initial (antenatal) values which fall well within the middle of the survivor range.

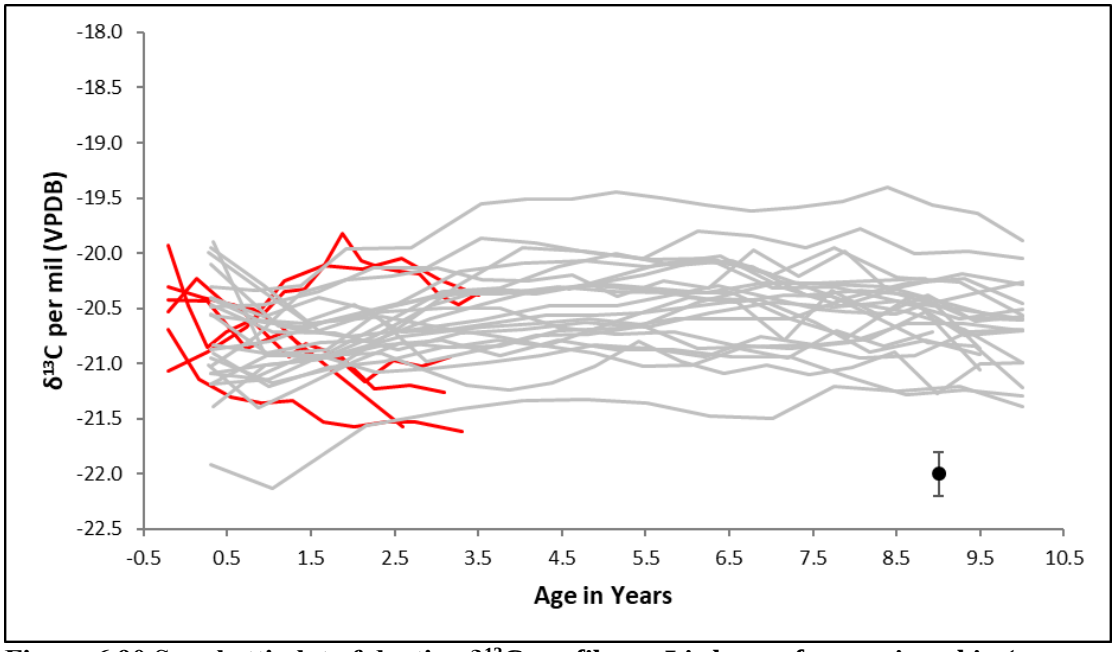

Figure 6.90 Spaghetti plot of dentine $\delta^{13}C$ profiles at Littleport for survivorship (non-surviving juveniles shown in red, surviving adult population shown in grey for reference)

Plotted $\delta^{15}N$ data profiles for individuals with skeletal stress markers in the Littleport sample appear in Figure 6.91. Female adults with stress markers within the sample align with the general trends of the overall site sample. Male adults and non-adults, by contrast, only appear within the upper range of values for $\delta^{15}N$ data profiles, demonstrating a pattern of higher childhood $\delta^{15}N$ values than those of females at the site which emerges independently of starting values. An important difference to note between males, females, and non-adults is the positioning of values at the outset of the data series. While all non-surviving children with stress markers have high initial values which decline over time, and females with stress markers exhibit dropping values between birth and approximately 2.5 years. The starting $\delta^{15}N$ values of male adults with stress markers remain relatively more stable in their position within the population throughout childhood.

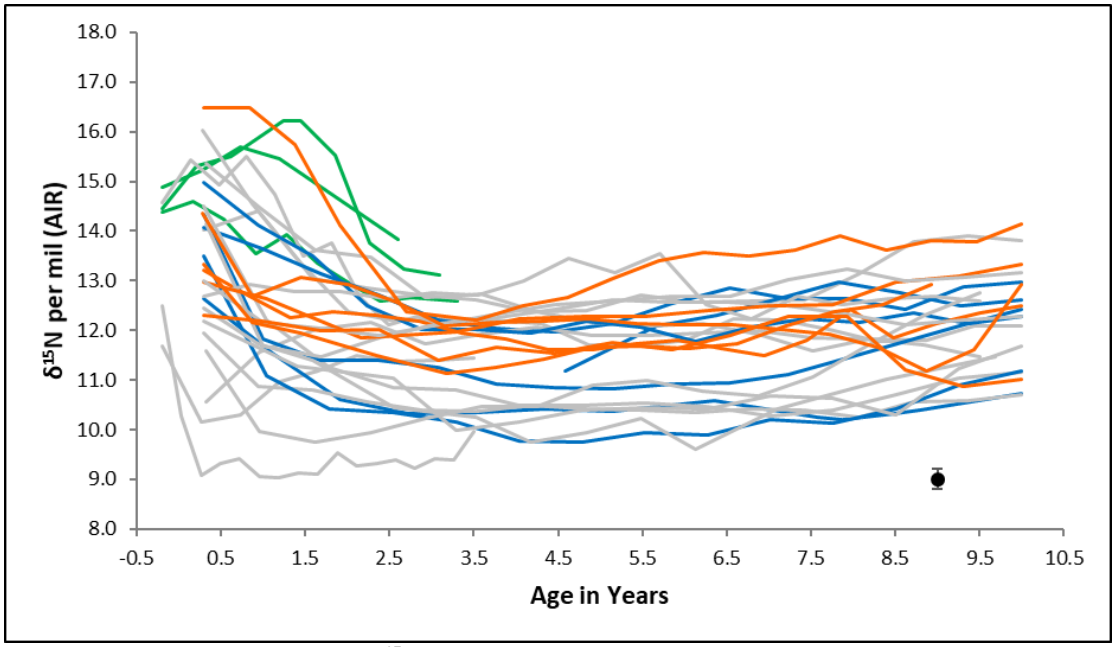

Figure 6.91 Spaghetti plot of $\delta^{15}N$ profiles for individuals at Littleport with skeletal stress markers (male individuals shown in orange, females in blue, non-adults shown in green, with non-stress marker-associated data shown in grey for reference)

Figure 6.92 shows plotted $\delta^{13}C$ data profiles for individuals with skeletal stress makers at Littleport. Female adults with skeletal stress markers sit centrally within the range of $\delta^{13}C$ values defined by the main population sample. Both male adults (−22.1 to −20.2 ‰) and non-surviving children (−21.6 to −20.4 ‰) with skeletal stress markers define lower $\delta^{13}C$ ranges than is characteristic of non-stress marker-associated data profiles in the Littleport sample, or female adults with skeletal stress markers at the site. Contrary to the general shift towards a lower $\delta^{13}C$ range among most males with stress

markers, one adult male in that category (LP 4178) showed $\delta^{13}C$ values which were higher than those of the overall sample. He is therefore exceptional for both the male stress marker cohort, and the population sample generally. In contrast to the differences in starting values for $\delta^{15}N$ among non-surviving children and males with stress markers, for $\delta^{13}C$ this difference is not evident in terms of initial values. However, most males with stress markers exhibit an undulating pattern value of change over time, while non-surviving children with stress markers display a declining $\delta^{13}C$ values over the range of time covered by the dm2.

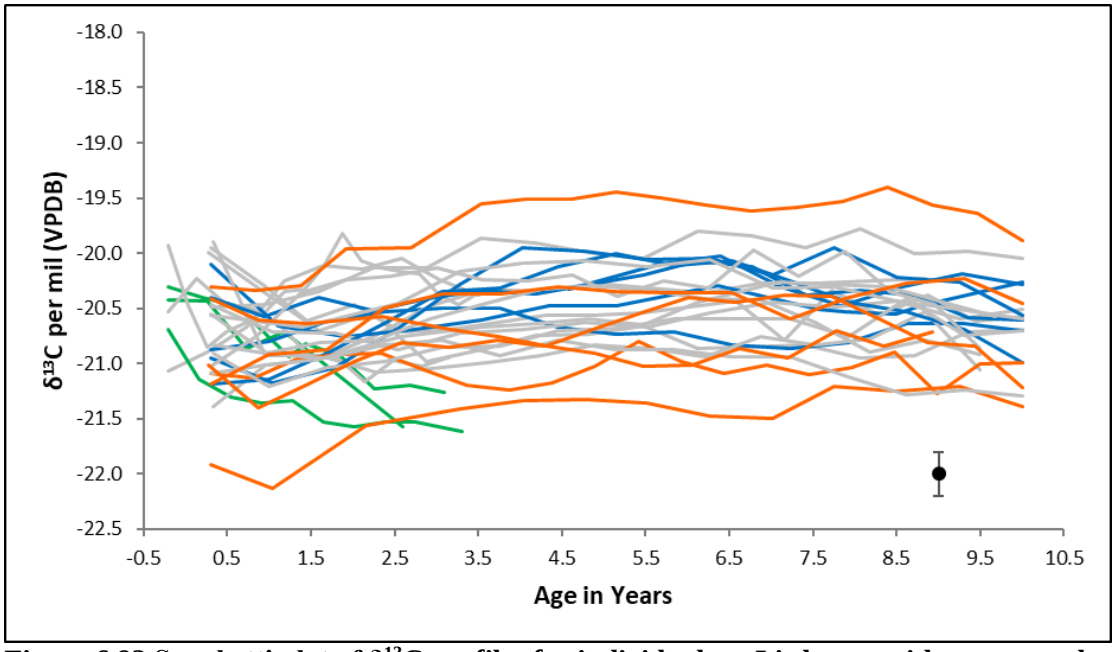

Figure 6.92 Spaghetti plot of $\delta^{13}C$ profiles for individuals at Littleport with stress markers (male individuals shown in orange, females in blue, non-adults shown in green, with non-stress marker-associated data shown in grey for reference)

$\delta^{15}N$ data for individuals at Littleport with cribra orbitalia appear in Figure 6.93. The patterning and distribution range exhibited in these individuals mirrors the pattern and distribution of data seen for individuals exhibiting stress markers more generally. As was the case for the Littleport stress marker cohort, female adults with cribra orbitalia display $\delta^{15}N$ profiles which fall within the parameters of the general sample values, falling within either of the visible tiers of $\delta^{15}N$ values. Similarly, adult males and non-surviving children with cribrotic lesions appear only at the high end of the population range, with and children having characteristically higher $\delta^{15}N$ than the bulk of either male or female adults with cribra orbitalia.

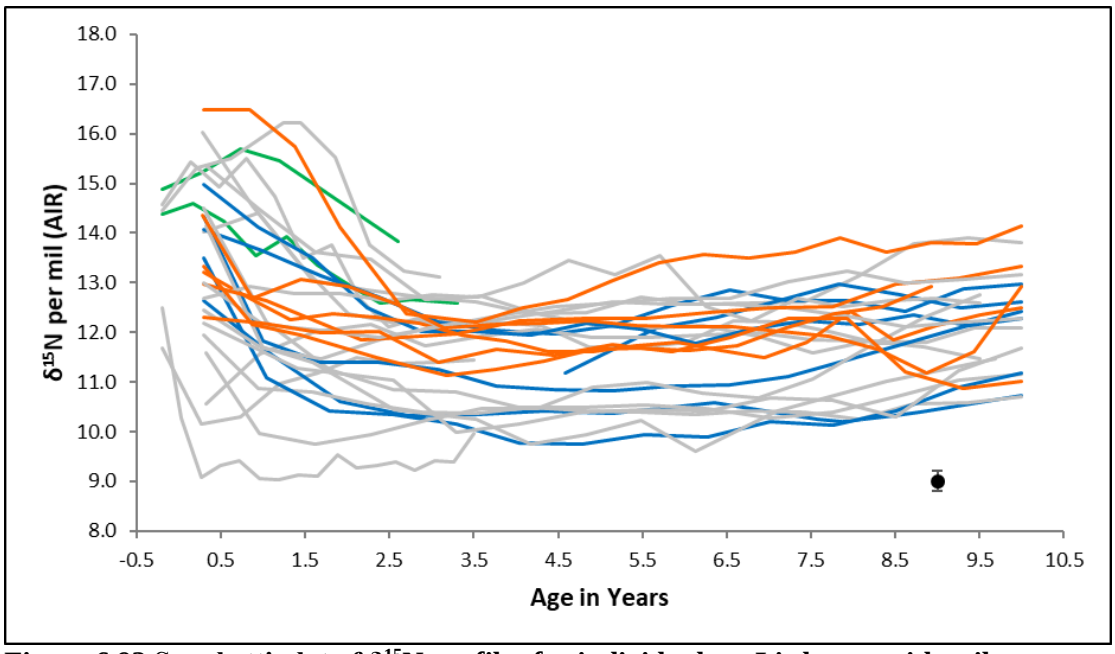

Figure 6.93 Spaghetti plot of $\delta^{15}N$ profiles for individuals at Littleport with cribra orbitalia (male individuals shown in orange, females in blue, non-adults shown in green, with non-cribrotic individuals shown in grey for reference)

Figure 6.94 shows incremental dentine $\delta^{13}C$ data profiles for individuals at Littleport with cribra orbitalia. As was the case with Littleport $\delta^{15}N$ profiles for individuals with cribra orbitalia, the $\delta^{13}C$ data for individuals at Littleport bearing cribrotic lesions strongly resembles the patterns produced by the data individuals under the broader umbrella of skeletal stress markers. As was observed for stress marker cohort $\delta^{13}C$ patterns, adult females with cribra orbitalia are indistinguishable from the general population sample in pattern of change and relative position based on $\delta^{13}C$ values. Similarly, both male adults and non-surviving children with cribra orbitalia plot in a lower range than their female counterparts, with males demonstrating no systematic change over time, and non-surviving children exhibiting a decline in values over time.

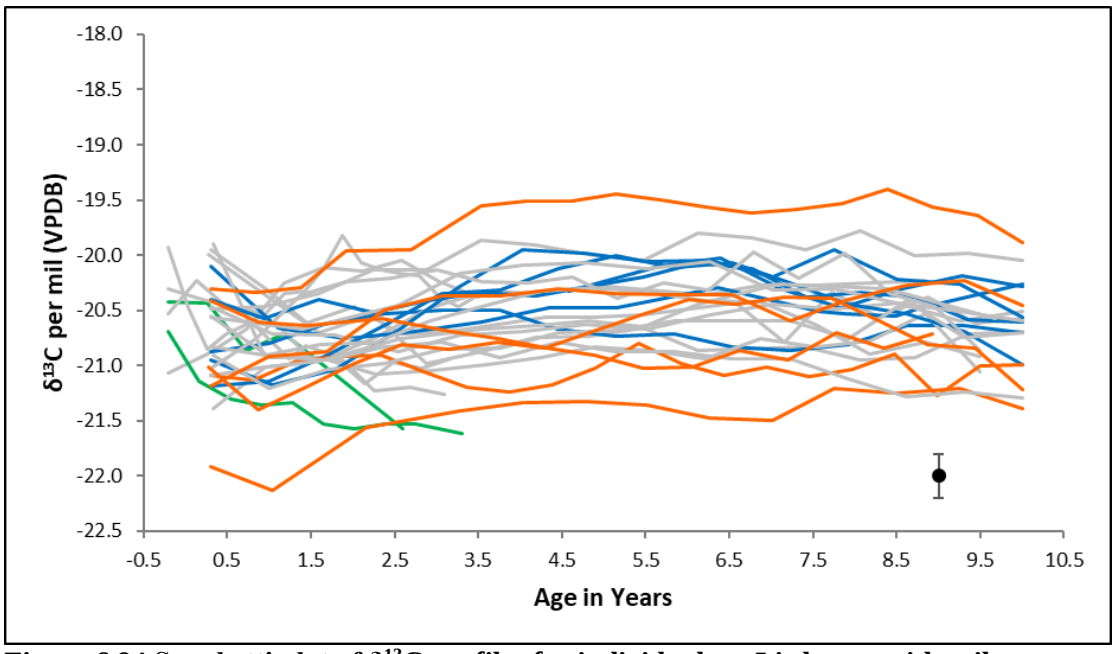

Figure 6.94 Spaghetti plot of $\delta^{13}C$ profiles for individuals at Littleport with cribra orbitalia (male individuals shown in orange, females in blue, non-adults shown in green, with non-cribrotic individuals shown in grey for reference)

Figure 6.95 shows the full sample of incremental data for Littleport plotted as $\delta^{13}C$ vs. $\delta^{15}N$. Non-survivor incremental data do not align with survivor data, instead plotting in a bifurcated pattern of data at the edges of the main cluster.

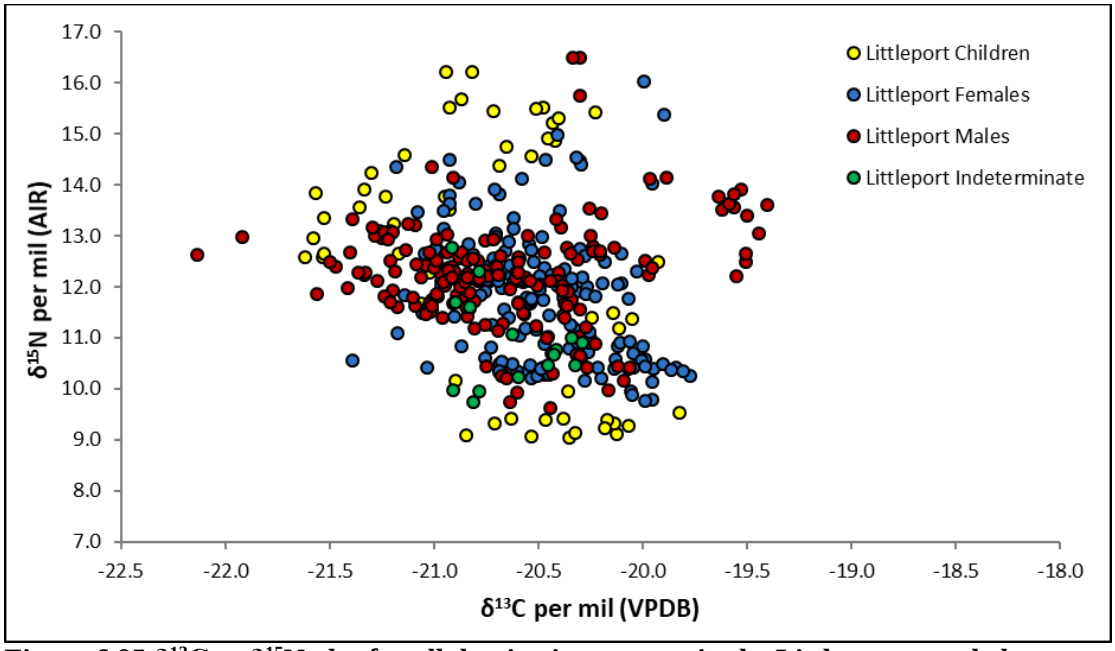

Figure 6.95 $\delta^{13}C$ vs $\delta^{15}N$ plot for all dentine increments in the Littleport sample by estimated sex and age

6.3.3 Intersite comparative data

The following section will compare intersite cohorts of individuals to identify similarities and differences between their members.

6.3.3.i Deciduous tooth data for Edix Hill and Littleport

This subsection provides comparative description of dm2 dentine data from non-survivors at both study sites. Figure 6.96 shows a comparative plot of non-survivor $\delta^{15}N$ data for children at Edix Hill and Littleport. A more marked pattern of flat profiles and moderate $\delta^{15}N$ values is evident for Edix Hill non-survivors than for Littleport.

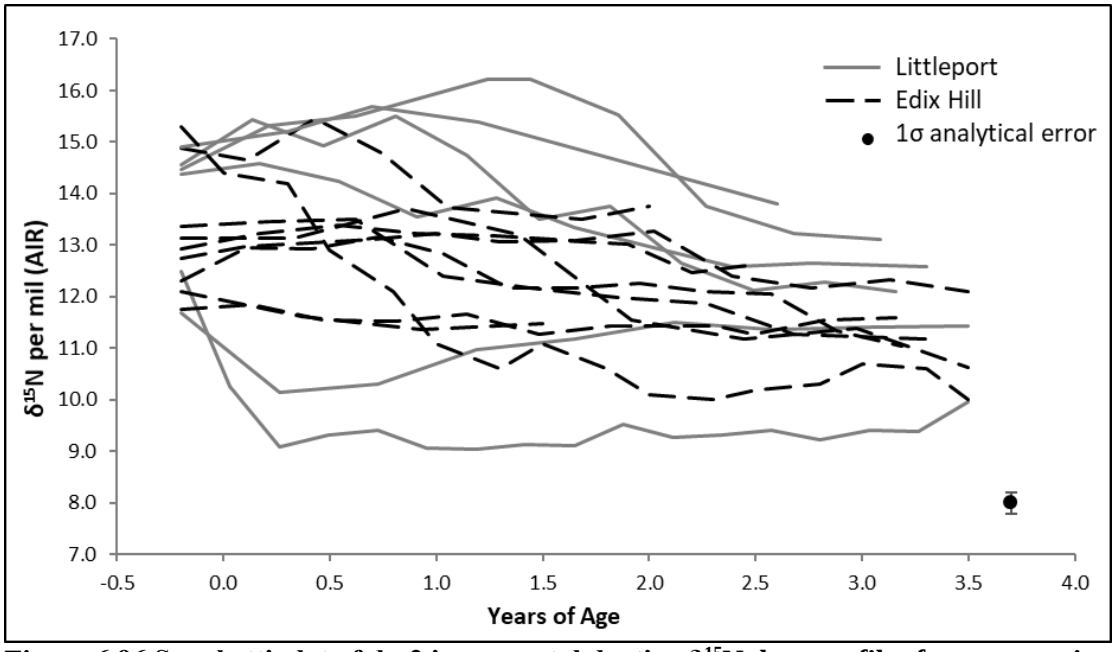

Figure 6.96 Spaghetti plot of dm2 incremental dentine $\delta^{15}N$ data profiles for non-survivors at both sites (Littleport shown in grey, Edix Hill in dashed black)

Figure 6.97 shows a comparative plot of non-survivor $\delta^{13}C$ data for children at Edix Hill and Littleport. Variability in $\delta^{13}C$ among non-surviving children at Littleport is visibly greater than is evident for Edix Hill non-survivors, who exhibit a more constrained range of variation.

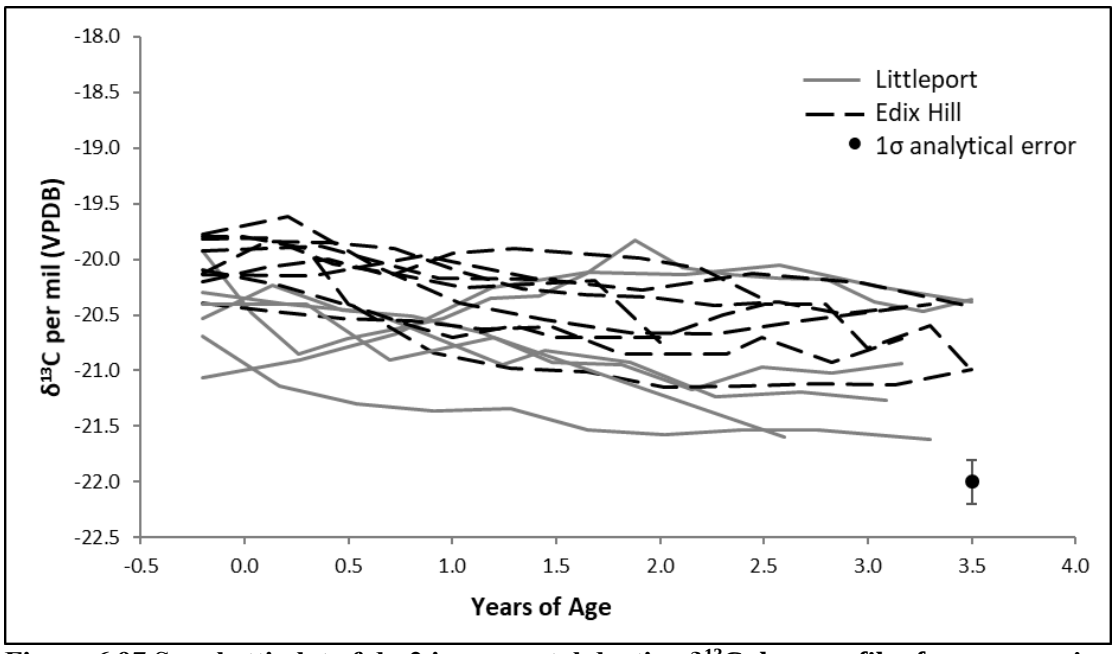

Figure 6.97 Spaghetti plot of dm2 incremental dentine $\delta^{13}C$ data profiles for non-survivors at both sites (Littleport shown in grey, Edix Hill in dashed black)

Carbon and nitrogen stable isotope data for antenatal (first increment) dm2 dentine are plotted as $\delta^{13}C$ vs $\delta^{15}N$, as an indicator of maternal health and diet, in Figure 6.98. Individuals at Edix Hill show a strong similarity of position, with seven individuals forming a loose cluster which is markedly different from other data. Two individuals from Edix Hill, EH 352 and EH 547B, plotted away from the cluster, having much higher $\delta^{15}N$ values alongside $\delta^{13}C$ at the high end of the range defined by the main Edix Hill cluster. One individual from Littleport, LP 4848, demonstrated similarity of values and plotted within the Edix Hill data cluster. However, four of the six Littleport non-survivors demonstrated higher $\delta^{15}N$ and lower $\delta^{13}C$ antenatal values than the Edix Hill cohort, forming a separate cluster. LP 3770 alone stood apart from both clusters in having much lower $\delta^{15}N$ and $\delta^{13}C$ than the majority of either Littleport or Edix Hill non-survivors.

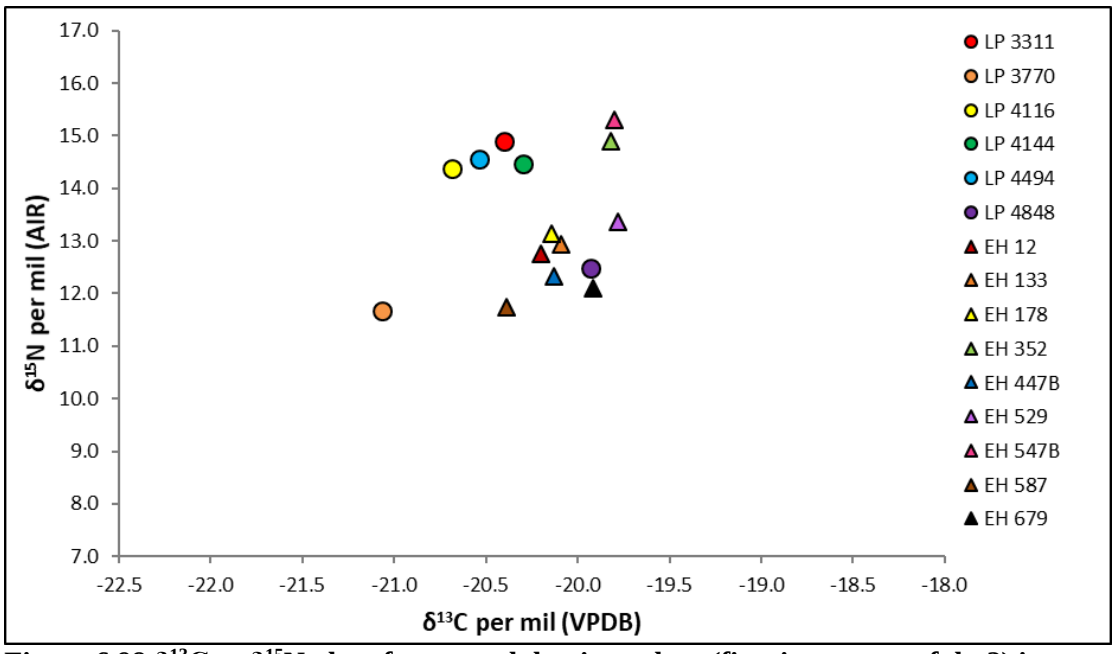

Figure 6.98 $\delta^{13}C$ vs $\delta^{15}N$ plot of antenatal dentine values (first increment of dm2) in non-survivors at both sites

6.3.3.ii Permanent first molar data for Edix Hill and Littleport

This section shows full sample data for adults at both sites, plotted to show similarities and differences in profile and pattern for stable isotope data. Adult M1 $\delta^{15}N$ data for both sites in the study are presented in Figure 6.99. The density of data in the plot makes delineation of trends somewhat difficult. Nonetheless, as in earlier site-based plots, a bifurcated pattern of data is visible for the Littleport sample, as well as a wider variability and overall higher $\delta^{15}N$ range than is apparent at Edix Hill.

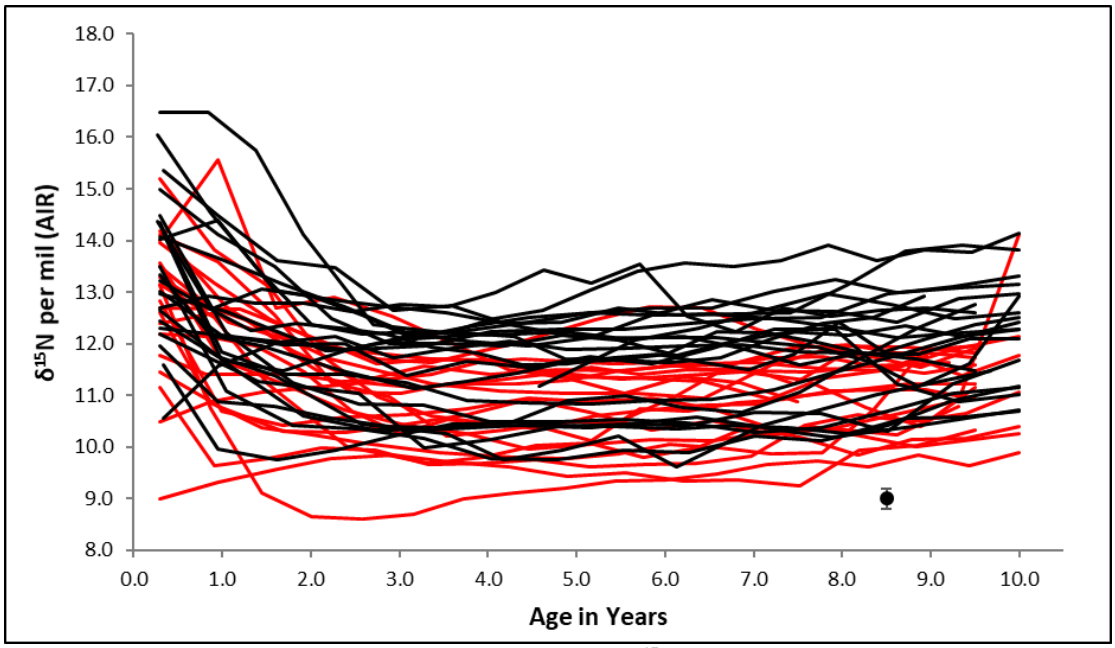

Figure 6.99 Spaghetti plot of incremental dentine $\delta^{15}N$ data profiles for adult individuals at both sites (Littleport shown in black, Edix Hill in red)

Figure 6.100 shows M1 $\delta^{13}C$ data profiles for Edix Hill and Littleport. Notably higher levels of variability are visible in the Littleport sample than in the Edix Hill adult cohort data, which exhibit low variability decreasing further over the course of childhood.

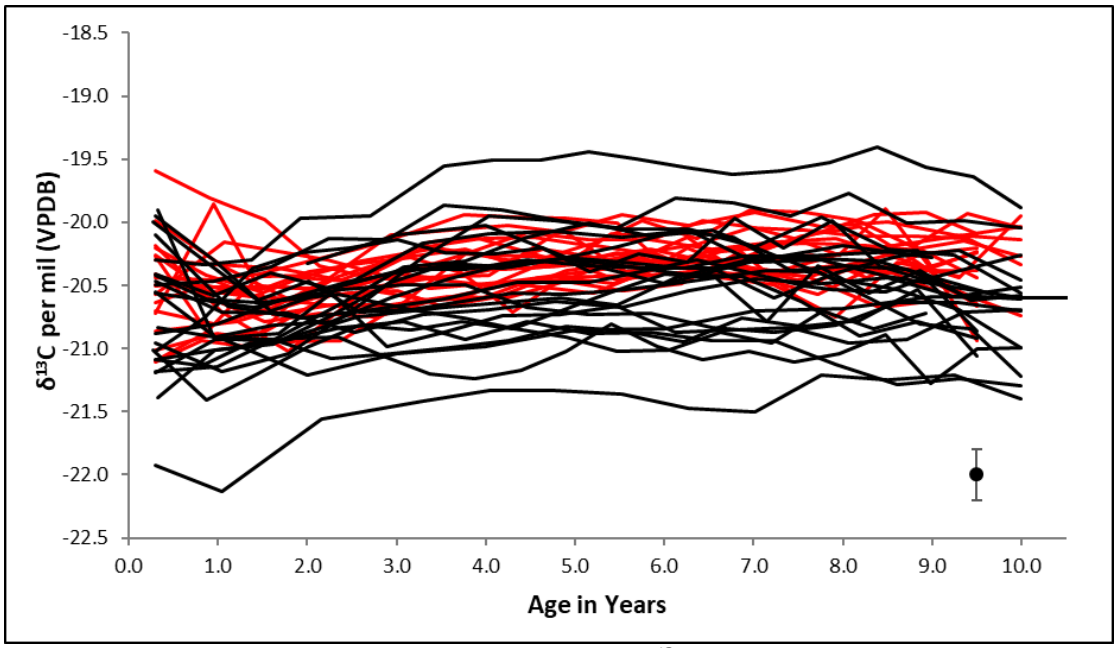

Figure 6.100 Spaghetti plot of incremental dentine $\delta^{13}C$ data profiles for adult individuals at both sites (Littleport shown in black, Edix Hill in red)

First M1 increment (~0.3 years of age) mean data for males and females at each of the sites were plotted as $\delta^{13}C$ vs $\delta^{15}N$ and appear in Figure 6.101. In this early-forming postnatal dentine, females at the two sites appear most alike, while males at both sites plot further away. Males at Edix Hill demonstrate lower mean $\delta^{15}N$ than females at either site, while having similar mean $\delta^{13}C$. Males at Littleport exhibit lower mean $\delta^{13}C$ than any of the other groups, while having similar mean $\delta^{15}N$ to females at Edix Hill in this early-forming dentine.

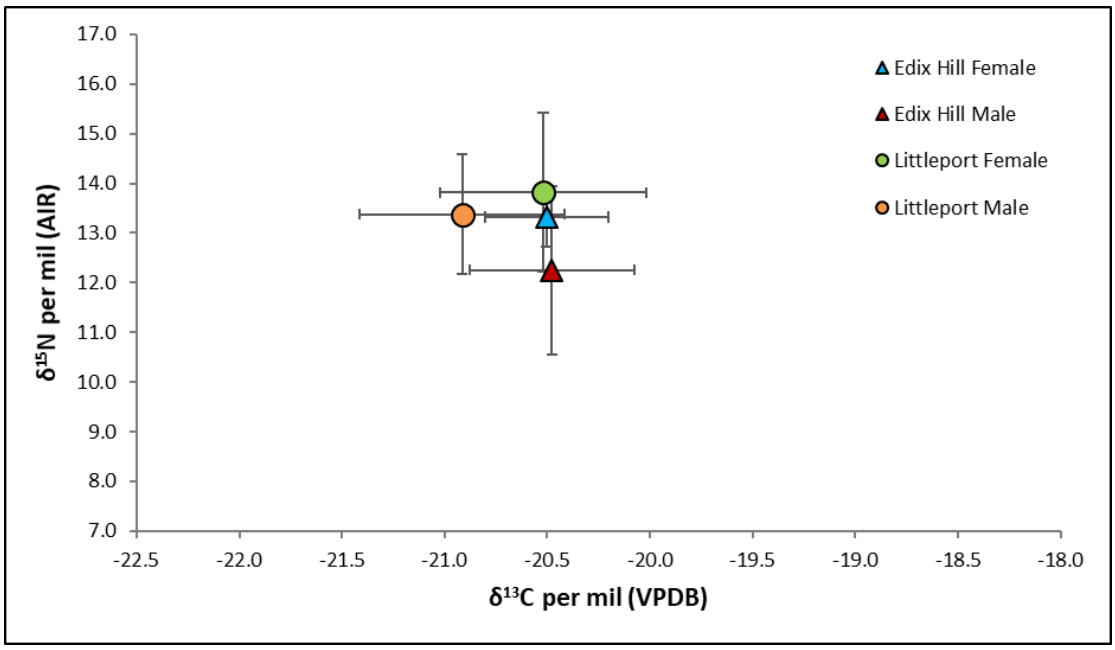

Figure 6.101 $\delta^{13}C$ vs $\delta^{15}N$ plot for mean values of the first M1 dentine increment at both sites by estimated sex (error bars indicate 1σ from the mean)

6.3.3.iii Summary of mean data for total sample at Edix Hill and Littleport

Figure 6.102 shows mean values of M1 and dm2 data for both sites plotted as $\delta^{13}C$ vs $\delta^{15}N$ by estimated sex and tooth type. The pattern evident in this data underlines similarities in pattern previously highlighted in the linear data. Littleport adult males plot most closely with Littleport non-surviving children, with nearly identical mean values, albeit greater variability in $\delta^{15}N$ among non-survivors. Edix Hill male and female adults also show paired $\delta^{13}C$ vs $\delta^{15}N$ plot positioning, demonstrating their similarity of mean values, which have lower $\delta^{15}N$ and higher $\delta^{13}C$ than Littleport males and non-surviving children. Adult females at Littleport show greatest similarity to non-surviving children at Edix Hill. These occupy an intermediate niche, with higher mean $\delta^{15}N$ values resembling those of the Littleport adult male/non-survivor clustering, alongside higher mean $\delta^{13}C$ values which more closely align with those of the Edix Hill adults.

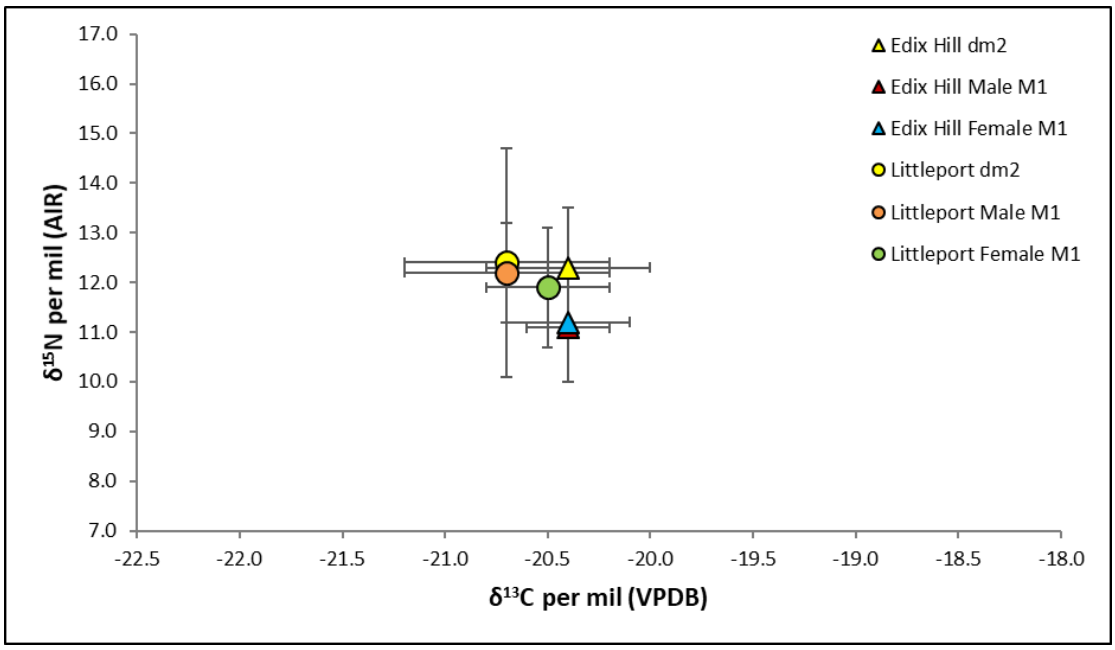

Figure 6.102 $\delta^{13}C$ vs $\delta^{15}N$ plot for mean values of all subgroups at both sites by age and estimated sex (error bars indicate 1σ from the mean)

6.4 Statistical results

Table 6.7 shows the results of statistical analyses of the independence of various categorical variables via Fisher's exact test. The relationship between burial at Littleport and cribra orbitalia was statistically significant (p=0.0045), while the relationship between sex and cribra orbitalia was not (p=1.0000). Regarding the association between isotopic patterning and various demographic factors, significant associations were found between opposing covariance at any age (p=0.0408), opposing covariance before the age of four 0.0252), and opposing covariance and site location (p = 0.0064).

Variables of Interest		Two-tailed Fisher's Exact Test (α=0.05)		
		P-value	Holm-Bonferroni Corrected P-value	Reject H_0?
Site location (Littleport)	Cribra orbitalia	0.0009	0.0045	Yes
Sex	Cribra orbitalia	1.0000	1.0000	No
Isotopic opposing covariance (any)	Site location (Littleport)	0.0016	0.0064	Yes
Isotopic opposing covariance (any)	Cribra orbitalia	0.0204	0.0408	Yes
Isotopic opposing covariance <4 years of age	Cribra orbitalia	0.0084	0.0252	Yes

Table 6.7 Summary of statistical test results on relationships between categorial variables in the study populations. H_0 = null hypothesis

7. Discussion

7.1 Introduction

Beginning with the data found towards the end of the Results chapter, the discussion will begin with a comparison of aggregate mean data placed in regional context. Following this, incremental data are analysed individually and by group cohort, mirroring the reporting in Chapter Six, Results. These cohort data are also compared with each other, and with other data from broadly contemporary (and where possible, regional) data from other early medieval sites in Britain.

These incremental data are analysed as an intentional replacement for cross-sectional methods involving bone; having the advantage of providing a longitudinal dimension to data with which to interpret changes over individual childhoods, and also illuminating patterns of change on an aggregate population level. Following the scholarly contributions of Beaumont *et al.* (2015) and Reynard and Tuross (2014) towards the methodological challenges of interpreting past breastfeeding patterns, the present study recognises the need to integrate interpretive models which account for non-dietary influences on isotopic shifts. Consequently, this chapter will analyse and discuss the evidence for a range of behaviours involving breastfeeding, care, disease, and diet which may be reflected in data from this study.

7.2 Collagen results in regional and temporal context

Data from Edix Hill and Littleport overlap with ranges produced by adult and non-adult bone collagen analyses from local and time-comparable assemblages, and are plotted against these comparative data in Figure 7.1.

There is a general paucity of published stable isotope data for early medieval Fenland sites, which the present study hopes to ameliorate in small part. While no previous comparative $\delta^{13}C$ and $\delta^{15}N$ data exists for Littleport, the dentine data from the present study produces a considerable degree of overlap with palaeodietary analyses of bone at the nearby 7th century site at Westfield Farm, Ely (n=15), a neighbouring island in the peat Fens. Data from Ely yielded $\delta^{13}C$ values falling between −21.0 and −19.7‰, and $\delta^{15}N$ values ranging from 10.5 to 12.7‰ (Lucy *et al.*, 2009). The data ranges for Littleport are broader than those of Ely, as analysis of incremental dentine at Littleport revealed lower $\delta^{13}C$ values (−22.1 to −19.4‰) and higher $\delta^{15}N$ values (9 to 16.5‰) than

were present at Ely. Lucy *et al.* (2009) had observed that the 11.8‰ mean value for $\delta^{15}N$ at Ely was higher than measured for human bone at other East Anglian early medieval sites. A lack of similarly raised $\delta^{13}C$ bone values, relative to the comparative sites, led the authors to interpret higher $\delta^{15}N$ at Ely as possible evidence of freshwater fish consumption in a diet otherwise composed of C_3 plants and terrestrially-sourced proteins.

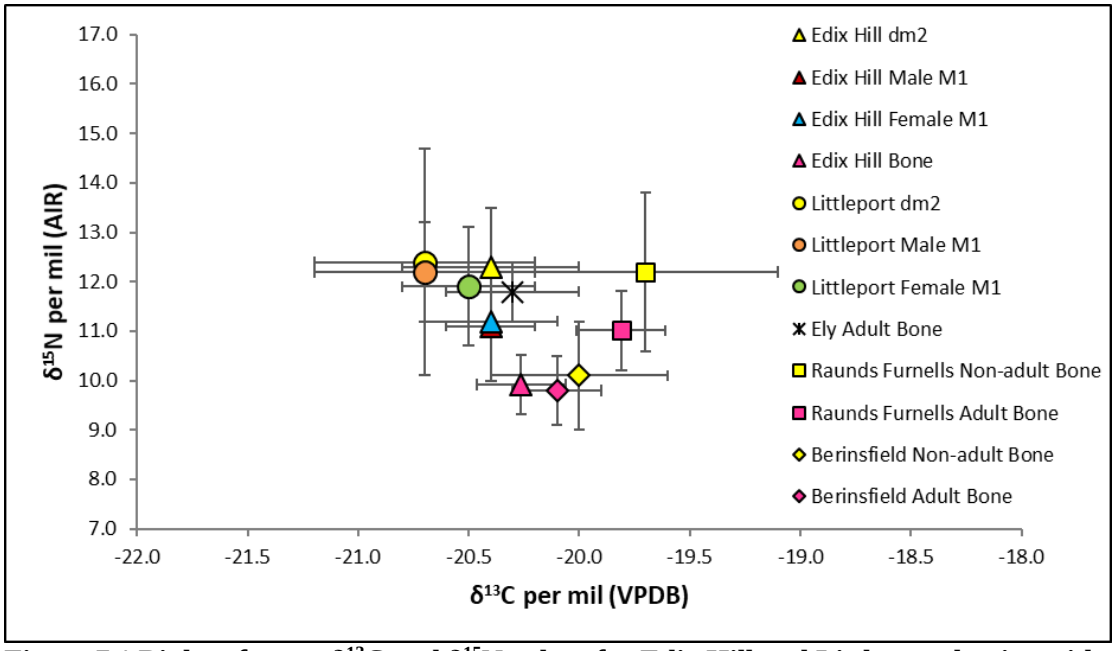

Figure 7.1 Biplot of mean $\delta^{13}C$ and $\delta^{15}N$ values for Edix Hill and Littleport dentine with comparative mean bone data from non-adults and adults from Ely (Lucy *et al.*, 2009), Raunds Furnells (Haydock *et al.*, 2013), and Berinsfield (Privat *et al.*, 2002) (error bars indicate 1σ from the mean).

Freshwater fish would undoubtedly have been consumed on a Fen island as a locally-abundant resource. Dentine $\delta^{15}N$ values at Littleport span a higher range than values recorded for Ely, alongside a $\delta^{13}C$ range which dips into *lower* values, and thus dietary inclusion of freshwater fish protein does not alone present a satsifying explanation for these higher and lower ranges at Littleport. However, Müldner and Richards (2005) analysed a range of fish bone from medieval York, including catadromous or freshwater fish such as eels (*Anguilla anguilla*) and pike (*Esox lucius*), finding values for these predatory species which were typified by high $\delta^{15}N$ and low $\delta^{13}C$. Eel bone (n=6) $\delta^{15}N$ values ranged from 10.6 to 12.8‰, with $\delta^{13}C$ values ranging from −24.9 to −17.6‰, while pike (n=2) produced $\delta^{15}N$ values of 16.7 to 23.4‰, with $\delta^{13}C$ values ranging from −24.5 to −23.4‰. Eels reportedly formed an important food source in medieval England, with Bede claiming that the island of Ely was so named due to the "vast

quantities of eels that are caught in the marshes" (Sherley-Price *et al.*, 1990: 236; Oosthuizen, 2016). Furthermore, environmental archaeologists commonly encounter abundant representation of eel bone within early medieval Fen assemblages (Ballantyne, 2004). While carnivorous fish such as eel and pike almost certainly formed an important resource for Fen islanders, it seems improbable that the proportion of dietary protein which would have had to be derived from this single resource would account for values observed at Littleport. This interpretation seems similarly dubious when aligned with zooarchaeological and environmental evidence supporting a strong engagement with pastoralism, and also the differential isotopic positioning of different survivor and non-survivor subgroups at Littleport.

Salinity, too, could play a part in shifting mean dentine $\delta^{15}N$ to higher values at Littleport. Historically, Littleport sat at the interface of the peat and silt fens. The silt-fen northern aspect of the island would have experienced periodic marine inundation, which would have shifted the salinity of the brackish water, attested to by the present of archaeological salt-making finds on the island (Holt, 2008: 8). Thus halophyte plants, which have higher $\delta^{15}N$ values relative to terrestrial C_3 plants (Britton *et al.*, 2008), may have been present and consumed as a resource by both humans and grazing animals. Ely was not located at such an interface, but securely within the freshwater peat fens, and not likely to have these resources directly at its doorstep. Equally, as the Ely sample does not include individuals of an age likely to be unweaned, it could be argued that mean dentine values at Littleport should be expected to reflect higher mean $\delta^{15}N$ than a sample primarily composed of bone collage from weaned individuals. However, the inclusion of breastfeeding values should produce higher, not lower, $\delta^{13}C$ during infancy and early childhood at Littleport. Furthermore, beyond early infancy (and the period of presumed weaning), the range of $\delta^{15}N$ values remains higher and the range of $\delta^{13}C$ values lower than those observed for similar ages at Edix Hill.

The existence of low $\delta^{13}C$ alongside high $\delta^{15}N$ values at Littleport also presents a similar barrier to the interpretation of these patterns as reflecting simple differences in childhood diet, relative to adult bone collagen (e.g. a higher reliance on freshwater fish protein in childhood than in later life). Recognition of differences in representation of diet between tissues, rather than differencs in diet itself, may provide an alternate explanation for these discrepancies. As bone and dentine collagen represent a similar extract from different tissues, there is not believed to be an offset between the two,

allowing cross-comparability of data (cf. Fuller *et al.*, 2003; Beaumont *et al.*, 2012; Reitsema *et al.*, 2016). However, the time resolution between bone and dentine is vastly different, due to bone collagen turnover. During times of extreme illness or malnutrition bone is also known to experience growth arrest and disturbance to turnover, while dentine continues to form and record these episodes of systemic stress (Weinbrenner *et al.*, 2003; Elamin and Liversidge, 2013). Beaumont and Montgomery (2016: 13) have referred to a characteristic combination of raised $\delta^{15}N$ and low $\delta^{13}C$ values as a "famine pattern" induced by malnutrition (discussed previously at length in Chapter 2, Section 2.4.5). It may be that these overall differences in mean childhood values at Littleport, relative to bone collagen data from Ely, may reflect differences between early childhood stress events and later life stability, as well as differences in the representation of such stress events in the tissues analysed.

More comparative data is available for Edix Hill than for Littleport. Dentine data for Edix Hill in the present study broadly overlap the range of bone collagen values previously observed for Anglo-Saxon inland sites in Britain, as well as those observed for bone collagen at Edix Hill itself. Mays and Beavan (2012) examined diet at coastal, riverine, and inland sites during the Anglo-Saxon period, finding $\delta^{13}C$ and $\delta^{15}N$ ranges of −20.8 to −19.6‰ and 7.9 to 11.4‰, respectively, for bone collagen at inland sites (n=42), including Edix Hill. Respective bone collagen values for $\delta^{13}C$ and $\delta^{15}N$ at Edix Hill (n=8) ranged from −20.6 to −20.1‰ and 8.8 to 10.9‰ within the study (Mays and Beavan, 2012). The higher $\delta^{15}N$ range observed in deciduous dentine included in the present study, relative to adult bone collagen $\delta^{15}N$ values measured by Mays and Beavan, may simply reflect differences in the resolution of and time-range covered by bone and tooth data together with the effects of breastfeeding.

Conversely, these higher values, alongside a greater range of variability in dentine $\delta^{13}C$ values in the data, may imply malnutrition in some individuals, as is suggested for Littleport. The differences seen between mean $\delta^{15}N$ and $\delta^{13}C$, and in the level of variability around the mean, in Figure 7.1 are suggestive of malnourishment. Both survivor and non-survivor dentine data exhibit lower mean $\delta^{13}C$ and higher mean $\delta^{15}N$ than the mean values for Edix Hill bone, while also expressing much higher variability within the sample values. However, while there are no differences in mean $\delta^{13}C$ between survivor and non-survivor dentine at Edix Hill, further differences in mean $\delta^{15}N$ are apparent; non-survivor dentine displays higher $\delta^{15}N$ values than childhood

values for individuals surviving to adulthood. Unless Edix Hill non-survivors were breastfed longer than those surviving childhood (a suggestion not supported by individual-level data from this study, as discussed at greater length in Section 7.3), or were fed a different diet than their peers - one which included a larger proportion of $\delta^{15}N$-increasing foods, such as freshwater fish - these differences may reflect differences in the ability of different tissues to register varying levels of early-life metabolic stress at Edix Hill.

Figure 7.1 also plots comparative mean bone collagen data for two other early-medieval inland sites in Southern Britain, Raunds Furnells and Berinsfield. The large cemetery at Raunds Furnells (n=361) dates to the late Anglo-Saxon period (c. 10^{th}-11^{th} centuries), while Berinsfield, another early Anglo-Saxon site, dates to the 5^{th}-7^{th} century. Mean bone collagen values and range of adults and non-adults at Berinsfield are most similar to mean bone values at Edix Hill, as seen in Figure 7.1, with $\delta^{13}C$ being only slightly offset to a lower mean value at Edix Hill than for the Berinsfield data. The data from Raunds Furnells is quite different, with both higher mean $\delta^{13}C$ and $\delta^{15}N$ apparent in both adults and non-adults, relative to mean bone collagen values at both Edix Hill and Berinsfield. Mean $\delta^{15}N$ for Raunds Furnells adult bone collagen is similar to mean dentine $\delta^{15}N$ for Edix Hill survivors, while non-adult mean bone collagen $\delta^{15}N$ at Raunds Furnells is most closely aligned with dentine values for non-survivors at Edix Hill, or survivors and non-survivors at Littleport. At all four sites (Littleport, Edix Hill, Berinsfield, and Raunds Furnells), non-survivor data shows a consistent pattern of increased variability in range, relative to data from survivor data from bone and dentine collagen at those sites. What distinguishes the data at these comparative sites from Edix Hill is the relationship between adult bone collagen values and non-adult values. Unlike Edix Hill, where adult mean bone collagen exhibits lower $\delta^{15}N$ and higher $\delta^{13}C$ than survivor and non-survivor dentine mean values, non-surviving children at both Berinsfield and Raunds Furnells have mean bone collagen $\delta^{13}C$ and $\delta^{15}N$ values which exceed adult bone values at their respective sites. Whether this illustrates different trends in childhood health and diet between the three sites, differences in tissue representation, or simply differences in analytical variability is not clear. Comparative incremental dentine data from these sites would be a welcome future contribution to interpreting these patterns.

7.3 Individual data

While identification of population-level patterning is a high priority for this study, correct interpretation of variability in carbon and nitrogen stable isotope data requires simultaneous cross-referencing of $\delta^{15}N$ and $\delta^{13}C$ profiles. As this is only possible at the individual level, the following sections will assess and discuss the individual-level patterning seen for individuals at the study sites.

7.3.1 Edix Hill

7.3.1.i Non-survivors

With respect to individual-level variability, three primary patterns were observed in the isotope profiles at Edix Hill: profiles demonstrating change compatible with an interpretation of breastfeeding (33%), "flat" profiles with low levels of variability throughout (45%), and a profile exhibiting episodes of nutritional stress, or opposing covariance (22%).

Profiles compatible with an interpretation of breastfeeding were identified in four individuals. The first of these, EH 178 (Figure 6.3), did not exhibit visible skeletal pathology and maintained static values between the first and second increments. Modest parallel increases in values of 0.2‰ for $\delta^{13}C$ and 0.6‰ for $\delta^{15}N$ were observed between birth and the latter portion of the first year, with a decline in $\delta^{15}N$ of 2.5‰ occurring between the increments representing estimated midpoints of 0.9 years and 2.5 years of age. This patterning is consistent with the introduction of complementary foods around the middle of the first year and continued breastfeeding until at least 2-2.5 years of age. EH 447B (Figure 6.5), like EH 178, exhibited no skeletal pathologies, but displayed increases of 0.3‰ for $\delta^{13}C$ and 0.6‰ for $\delta^{15}N$ between the first and second increments. This potentially signifies recovery from the $\delta^{15}N$-depressing "pregnancy effect" observed in healthy pregnancies (Fuller *et al.*, 2004), alongside increases due to the onset of breastfeeding. Values peaked for both isotopes by the midpoint of the first year, with a decline thereafter of 0.4‰ for $\delta^{13}C$ and 1.0‰ for $\delta^{15}N$ by the middle of the second year, suggesting that breastfeeding had fully ceased by this time. In contrast, EH 529 (Figure 6.6) had pathological characteristics which included dental disease and extra-cortical new bone formation. In this individual, isotope values were largely static until the middle of the first year. A decline in values of 0.4‰ for $\delta^{13}C$ and 1.5‰ for

$\delta^{15}N$ occurred between approximately 7 months and 2 years of age, indicating that the weaning process may have occurred during this period.

The final individual to exhibit parallel decline in $\delta^{13}C$ and $\delta^{15}N$, consistent with an interpretation of breastfeeding and weaning patterns, was EH 547B (Figure 6.7). EH 547B did not evince any skeletal pathology but did show a 0.9‰ drop in $\delta^{15}N$ from their antenatal first increment value to the second, post-natal increment, alongside stable $\delta^{13}C$ values. This drop may signify some *in utero* nutritional stress during the late portion of pregnancy, which resolved after birth, or alternately it may suggest a change in maternal diet during this interval. Following birth, no further changes to $\delta^{13}C$ and $\delta^{15}N$ occurred until between 4-6 months of age. Between the third and fourth increments, drops of 0.5‰ for $\delta^{13}C$ and 1.3‰ for $\delta^{15}N$ were found. $\delta^{13}C$ continued to drop until approximately 10 months of age, being stable from that point until approximately 2 years, while $\delta^{15}N$ values continued to decrease until approximately 1.3 years of age. These patterns would be consistent with breastfeeding from birth, with the onset of complementary feeding occurring between four and six months of age, and final cessation of breastfeeding occurring around 16 months of age. Following this period, EH 547B underwent a series of episodes of opposing covariance in $\delta^{13}C$ and $\delta^{15}N$ which signify either alternating episodes of malnutrition and recovery; or alternately, seasonal dietary changes to reliance on higher-$\delta^{15}N$ (but not higher-$\delta^{13}C$) foods, with a period of significant body-mass increase between the ages of two and three years.

Four individuals produced data with flat profiles exhibiting little variability. EH 12 (Figure 6.1), an individual exhibiting no skeletal pathology, but showing a discrepancy between estimated skeletal aging and stage of root resorption suggestive of skeletal stunting, had a largely flat profile with little variability overall. A second individual, EH 133 (Figure 6.2) exhibited cribra orbitalia, but displayed a largely static $\delta^{15}N$ throughout the course of tooth formation, alongside a drop in $\delta^{13}C$ of 0.8‰ between the approximate ages of birth and one year of age, most of which occurred during the second six months of life. This may indicate the onset of complementary feeding, but with the lack of parallel covariance in $\delta^{15}N$ this suggestion must remain speculative. Individual EH 587 (Figure 6.8) displayed no pathology aside from a vertebral anomaly and had a flat profile throughout the data series which might indicate a lack of dietary shifts. EH 679 (Figure 6.9) was the final individual with a flat, low-variability profile.

This individual had no evidence of skeletal pathology, but died at 18 months of age, with little variability beyond analytical error in evidence during their short life.

Only one individual at Edix Hill possessed a profile clearly reflective of nutritional stress. While relative stasis was observed between the values of first and second dentine increments, demonstrating metabolic homeostasis between the late antenatal and early postnatal periods, EH 352 (Figure 6.4) exhibited opposing covariance in $\delta^{13}C$ and $\delta^{15}N$ values in subsequent increments. A drop in $\delta^{13}C$ of 0.2‰ was in evidence alongside a rise in $\delta^{15}N$ of 0.9‰ between birth and the middle of the first year. While a rise in $\delta^{15}N$ might be expected to occur during this period if the cause were an increase in dietary contribution from breastfeeding, a concurrent decrease in $\delta^{13}C$ is more consistent with catabolic activity than a breastfeeding effect. This may have occurred due to illness, or due to early feeding difficulties in the first months of life. Following this period, $\delta^{15}N$ values began to decline alongside more stable $\delta^{13}C$ values, indicating possible recovery from an episode of nutritional stress. A discrepancy between estimated skeletal age and the stage of root resorption achieved by EH 352, similar to the discrepancy observed for EH 12, was suggestive of stunting, which may be reflective of these episodes of nutritional stress.

7.3.1.ii Survivors

Examination of individual profiles for survivors at Edix Hill produced similar patterns to those observed in the non-survivor cohort. The proportion represented in each of the groups, however, were slightly different in the survivor sample. Adults were classified based on the predominant pattern observed before 4 years of age (approximating the period covered by non-survivor dm2 teeth and allowing for greater uncertainty in the timing of M1s). While most of the non-survivor sample data was evenly divided between individuals exhibiting flat (45%) or parallel covariance (33%) patterns in infancy, with only two individuals (22%) exhibiting a potentially catabolic pattern of change; in the survivor cohort at Edix Hill parallel covariance had a slight majority, with 40% of individuals displaying this pattern in infancy. The remainder of the sample was evenly accounted for by individuals with at least one flat profile (30%), or patterns of opposing covariance in early life data (30%).

Parallel covariance

The largest group, individuals displaying predominantly synchronous changes in $\delta^{13}C$

and $\delta^{15}N$, comprised eight individuals. Of these, three individuals were male (EH 112, EH 146, and EH 626B) and five were female (EH 4, EH 20B, EH 359, EH 440A, and EH 547A). EH 112 (Figure 6.12) exhibited strikingly little variability in values. Between approximately four and eleven months of age, a decrease of 0.3‰ for $\delta^{13}C$ and 1.6‰ for $\delta^{15}N$ occurred, which may suggest that the weaning process was both initiated and completed during this period. Aside from this early decline in values, the profile exhibits low variability, although a gradual and parallel $\delta^{13}C$ and $\delta^{15}N$ increase of 0.4‰ and 1.0‰, respectively, occurred between the second and penultimate increments of the tooth series (representing approximately 0.9 to 9.4 years of age). At the end of the series, between 9.4 and 10 years of age (the completion of tooth development) a 0.2‰ decrease in $\delta^{13}C$ occurred alongside a 0.5‰ increase in $\delta^{15}N$. This may suggest a concurrent episode of nutritional stress. EH 146 (Figure 6.14) showed more dramatic shifts in both $\delta^{13}C$ and $\delta^{15}N$ over a more protracted period. $\delta^{13}C$ values fell by 0.7‰ between 4 months and 2 years of age, reaching a plateau after this time, while $\delta^{15}N$ declined by 4.6‰ between 4 months and 3.4 years of age, with a flatter profile after this point. As both $\delta^{13}C$ and $\delta^{15}N$ are declining between the first (~0.3 years of age) and second (~0.9 years of age) increments, it is likely that the dietary shift and the introduction of a complementary diet occurred in the interim, probably around the middle of the first year, as would be expected. Interpreting the cessation of breastfeeding as occurring with the final decline in $\delta^{15}N$, at 3.4 years, would position the end of the weaning process later than would be expected for Anglo-Saxon populations. As the $\delta^{13}C$ values stabilise much sooner, by two years of age, it is possible that cessation of breastfeeding occurred during this timeframe, and that $\delta^{15}N$ continued to fall after this point due to concurrent changes in the complementary diet. Indeed, the timing of steepest decrease for $\delta^{15}N$ supports this interpretation; while $\delta^{13}C$ falls steadily at a rate of 0.2-0.3‰ per increment of its decline, $\delta^{15}N$ fell most sharply (at 1.4‰ per episode) between 0.3-0.9 and 1.5-2.1 years of age, signifying the importance of these two transitional periods. This pattern of extended decrease in $\delta^{15}N$ was also followed by the final male to show a parallel covariance pattern, EH 626B (Figure 6.19). $\delta^{13}C$ and $\delta^{15}N$ values both fell, by 0.4‰ and 1.1‰ respectively, between the first (~4 months of age) and second (~1.1 years of age) increments of data, indicating the likelihood that the onset of weaning occurred during this period. Values continued to decline for $\delta^{13}C$ until 1.8 years of age, falling 0.6‰ in that time before assuming a flatter profile. Decline in $\delta^{15}N$ values continued until approximately 2.6 years of age,

decreasing by 1.7‰ over that period. Some further change to the profile of EH 626B occurred between the ages of four and five years old, as a minor increase in $\delta^{13}C$ of 0.2‰ and decrease in $\delta^{15}N$ of 0.3‰ are suggestive of a period of increased growth. There is a further decrease of 0.2‰ in $\delta^{13}C$ in the final increment of data whose significance is unclear; this small change is not accompanied by a concurrent change in $\delta^{15}N$, and values appear largely like those of earlier childhood data.

Female survivors at Edix Hill showing parallel covariance were similar in pattern to the males. EH 4 (Figure 6.21) exhibited decreasing $\delta^{13}C$ values between the ages of approximately four months and 1.5 years of age, decreasing by a modest 0.4‰ over that period. $\delta^{15}N$ declined by 2.4‰ over a longer period between approximately four months and 2.8 years of age. While timeframes suggested by either carbon or nitrogen data would fit within suggested parameters for cessation of breastfeeding taking place in Anglo-Saxon populations, the greatest drops in $\delta^{15}N$ parallel those of $\delta^{13}C$ during the first 1.5 years of life, with 1.5‰ of the observed decrease occurring during this period. EH 4 also exhibited a "bubble" of opposing covariance, with a rise in $\delta^{15}N$ of 0.4‰ and a decrease in $\delta^{13}C$ of 0.7‰, suggesting an episode of nutritional stress occurring between the ages of 8.4-9.5 years of age. EH 20B (Figure 6.23) had a profile with sharply dropping $\delta^{13}C$ and $\delta^{15}N$ from one year of age (the earliest available data). $\delta^{13}C$ fell 0.8‰ by approximately 1.5 years of age, with the profile flatter thereafter. $\delta^{15}N$ decreased by 2.9‰ between 1-1.5 years of age, before rising by 0.2‰ between 1.5-2.3 years of age. This reversal of decline in $\delta^{15}N$ suggests that weaning was probably completed by 1.5 years. A further decline of 1.9‰ in $\delta^{15}N$ occurred between approximately 2.3 and 5.5 years of age. As $\delta^{15}N$ in this profile started with an unusually high value of 15.6‰ at one year of age, and as $\delta^{13}C$ rose steadily and gradually over the period covered by $\delta^{15}N$ decline, this may indicate a more complex dynamic than can be accounted for by a simple "breastfeeding effect". A plausible interpretation is that this dyad experienced antenatal and postnatal nutritional stress which resolved over time.

EH 547A (Figure 6.27), a female who did not exhibit skeletal stress markers, presented similar challenges to interpretation. She showed a pattern of parallel decline in $\delta^{13}C$ and $\delta^{15}N$ between four months and 1.8 years of age, dropping 0.8‰ for $\delta^{13}C$ and 2.3‰ for $\delta^{15}N$ during that period. However, $\delta^{15}N$ values for EH 547A continued to drop until the age of 2.5 years, while $\delta^{13}C$ values began to rise from 1.8 years and continued in an upward trend until 4.8 years of age, rising a total 0.9‰ over this period. After 2.5

years of age $\delta^{15}N$ again rose to track the trajectory of $\delta^{13}C$, before both profiles reached a plateau in values at 4.8 years. It is likely that for EH 547A, this brief divergence from parallel covariance between 1.8-2.5 years represents a period of increased growth during the transition from full breastfeeding to full reliance on a solid childhood diet.

The remaining two female individuals showing a pattern of parallel covariance (EH 359 and EH 440A) offered data profiles with fewer interpretive challenges. EH 359 (Figure 6.25) showed a decline in both $\delta^{13}C$ and $\delta^{15}N$ from approximately four months (0.3 years) of age, which continued until a plateau was reached at around 11 months (0.9 years) of age for $\delta^{13}C$ and at approximately two years of age for $\delta^{15}N$. Variation after these ages was minor, indicating no evidence of major age-related changes in diet, and that weaning was probably initiated around the middle of the first year, and completed by two years of age. The profile of EH 440A (Figure 6.26) began at its outset with a rise in $\delta^{13}C$ and $\delta^{15}N$ between the ages of 0.3 and 1.1 years of age, with parallel decline thereafter. This comparatively late peak in values may be explained either as this being the period of peak reliance on human milk, or alternately maternal diet may have included a greater allowance of higher-trophic level protein during this interval. Falling $\delta^{13}C$ values reached a plateau by approximately 1.1 years of age, while $\delta^{15}N$ reached stasis in values by around three years of age, suggesting that the transition from predominant breastfeeding to full reliance on a solid diet occurred during this period.

Flat profiles

Only marginally secondary to individuals exhibiting parallel covariance in their early childhood data were individuals demonstrating a flat trend. This group accounted for six out of twenty adults, or 30% of the adult sample at Edix Hill. In contrast to the non-surviving individuals exhibiting this pattern, most adults did not possess *two* flat and parallel data profiles. Instead, in all cases, either $\delta^{13}C$ or $\delta^{15}N$ data failed to show systematic longitudinal shifts in pattern, while some variation occurred in the other.

Two individuals, both males, exhibited flat $\delta^{15}N$ profiles with accompanying variation in $\delta^{13}C$ values. EH 11 (Figure 6.10) demonstrated a small and incremental 0.6‰ increase in $\delta^{15}N$ from 10.5 to 11.1‰ between the beginning of the tooth series data (0.3 years) and 1.5 years of age. $\delta^{15}N$ values are static beyond this age, with no change beyond the instrument error range. $\delta^{13}C$ values, by contrast, show only a gradual 0.6‰ increase between the beginning of the series and 3.8 years of age, and values remain reasonably invariable thereafter, aside from a 0.4‰ dip occurring between the

approximate ages of five and six years. $\delta^{13}C$ and $\delta^{15}N$ values are both higher at the end of the tooth series data than at the beginning, showing no age-related trend to change, or patterning which might suggest breastfeeding. EH 33 displayed an altogether more variable $\delta^{13}C$ profile (Figure 6.11) alongside a predominantly flat $\delta^{15}N$ profile. Like EH 11, EH 33 did not show the expected elevation in $\delta^{13}C$ and $\delta^{15}N$ during early childhood which might suggest breastfeeding, instead showing $\delta^{13}C$ and $\delta^{15}N$ values at the end of the tooth series which were equal or higher than those at the outset of the series. $\delta^{13}C$ values rose by 0.4‰ between 2.2 and 2.9 years of age, remaining elevated until a decline of 0.3‰ occurred between 4.9 and 5.6 years of age. These values again rose by 7.5 years of age, before dropping 0.7‰ between 8.2 and 9.5 years of age. Variability in $\delta^{15}N$ did not at any time in the series exceed 0.3‰ from one increment to another, failing to demonstrate evidence of substantial and meaningful change in diet during this time.

The reverse trend, flat $\delta^{13}C$ alongside more variable $\delta^{15}N$ profiles, was observed in three males and one female. The first of the males, EH 125 demonstrated declining $\delta^{15}N$ from the outset of the data series at 0.3 years of age (Figure 6.13). A drop of 2.2‰ for $\delta^{15}N$ was observed to occur by 2.5 years, with no concurrent decrease in $\delta^{13}C$ values, which did not exceed variance of 0.3‰ between any sequential increments. EH 198 also exhibited low variance in $\delta^{13}C$ throughout, and a gradual decrease in $\delta^{15}N$ of 1.9‰ between the ages of 0.3 and 3.0 years of age (Figure 6.15). Beyond this age, $\delta^{15}N$ values remained static until the ages of 4.6-6.8 years, when a short-term dip of 0.3‰ was followed by a sharp rise of 0.8‰ and a new plateau of higher values which persisted to the end of the tooth series. Both of these individuals exhibited skeletal stress markers: EH 125 had enamel hypoplasia, while EH 198 was the sole adult at Edix Hill to exhibit cribra orbitalia. It is possible that in both cases, declining $\delta^{15}N$ values were representative of the weaning process, and that $\delta^{13}C$ values simply failed in these cases to reflect the ~1‰ expected increase in value associated by Fuller *et al.* (2006) with exclusive breastfeeding, perhaps due to complementary feeding from birth. If so, this would suggest that the end of the weaning process occurred for these individuals around the ages of 2.5-3 years. However, in consideration of the exhibited stress markers, and the lability of $\delta^{15}N$ values in response to physiological non-dietary processes, simple dietary interpretation should be viewed with some scepticism.

Remaining individuals exhibiting flat $\delta^{13}C$ profiles alongside varying $\delta^{15}N$ did not exhibit observable skeletal stress markers. EH 300A did not produce variance between increments exceeding instrument error in nearly all cases (Figure 6.16). A 1.2‰ decrease was noted between the ages of 0.3 and 1.7 years for $\delta^{15}N$, after which $\delta^{15}N$ variability was minimal. Between the penultimate and final increments of data, a spike of 0.4‰ and 3.2‰, for $\delta^{13}C$ and $\delta^{15}N$ respectively, occurred, signifying a dietary shift of sizeable magnitude. As such a surge in values did not occur in other individuals, it is impossible to tie this shift to anything systematic in terms of age-related dietary change, and it is likely that this increase in values is idiosyncratic to the circumstances of the individual. One female also demonstrated flat $\delta^{13}C$ alongside variable $\delta^{15}N$ values; EH 726 displayed strikingly little variability in $\delta^{13}C$ or $\delta^{15}N$ values over the course of tooth development (Figure 6.29). Between the approximate ages of 0.3 and 1.7 years, $\delta^{15}N$ values declined by 2.4‰, while $\delta^{13}C$ values remained steady. Only two fluctuations to $\delta^{15}N$ profile emerged beyond this early decline: a 0.4‰ decrease occurred from 6.5-7.2 years, and a 0.6‰ increase between 7.9-8.6 years of age.

Although neither of these individuals displayed skeletal stress markers suggestive of physiological stress during tooth formation, it is probable that the presence of adaptive skeletal markers to stress underestimates the number of individuals experiencing these challenges. The physiological, rather than dietary, origin of increases and decreases in $\delta^{15}N$ seems particularly likely, given that dietary factors such as breastfeeding (even when combined with a heavy reliance on freshwater fish as a complementary food) do not provide a satisfactory explanation for such elevated $\delta^{15}N$ early in life, where a similar breastfeeding effect is not observed for $\delta^{13}C$.

Opposing covariance

Six individuals (30% of adults), three males and three females, accounted for the remainder of the adult sample at Edix Hill. These exhibited opposing covariance in $\delta^{13}C$ and $\delta^{15}N$ profiles consistent with an interpretation of nutritional stress.

Three of the six individuals exhibiting opposing covariance also showed skeletal stress markers. EH 453, a male with enamel hypoplasia, displayed a pattern of rising $\delta^{13}C$ and decreasing $\delta^{15}N$ values which spanned from the outset of the data series at 0.3 years to three years of age, when both profiles stabilised (Figure 6.17), suggesting possible recovery from *in utero* nutritional stress. Over that period, $\delta^{13}C$ values

increased by 0.8‰ and $\delta^{15}N$ decreased by a total of 0.6‰. Following the attainment of a plateau in values at three years of age, homeostasis in values was maintained until 8-9 years of age, when $\delta^{13}C$ values rose 0.4‰ over a six-month period before falling 0.5‰ to return to approximate pre-existing values. During this time, $\delta^{15}N$ did not change for EH 453. In contrast, EH 45, a female, presented initially with parallel covariance (Figure 6.24). Within the first two increments of data, representing approximately four to eleven months of age, parallel drops in both $\delta^{13}C$ and $\delta^{15}N$ of 0.7‰ occurred, suggesting a shift from a higher-trophic level diet (probably breastmilk) to greater reliance on a lower-trophic level diet. However, $\delta^{13}C$ values remained constant between eleven months and 1.5 years of age, before rising again 0.7‰ to a plateau by 3.3 years. During this same period $\delta^{15}N$ values continued to drop and also reached a plateau by 3.3 years. While this strongly suggests that dietary transition was completed by 3.3 years, it is less clear what occurred in the interim between 0.9-3.3 years. It is possible that the pattern of opposing covariance characterised by dropping $\delta^{15}N$ and rising $\delta^{13}C$ indicates a substantial increase in body mass, as observed by Mekota *et al.* (2006) in individuals recovering from anorexia nervosa, and perhaps catch-up growth during this period. While there are no signs of catabolic opposing covariance in this individual's tooth series data, EH 45 exhibited both enamel hypoplasia and Harris lines. It is therefore likely that this individual experienced some growth-interrupting stressors during childhood, which may be somewhat masked metabolically by the combined effects of breastfeeding.

EH 9, a female, showed a pattern of successive plateaus as values changed (Figure 6.22). Exhibiting enamel hypoplasia and Harris lines, EH 9 had $\delta^{15}N$ values which decreased by 3.6‰ between 0.3 and 4.5 years of age, with 0.9‰ of the decline taking place in the first year of life. After 4.5 years, $\delta^{15}N$ remained static to the end of the data series. $\delta^{13}C$ values showed a very different pattern, remaining level from approximately 0.3-2.4 years of age. At around 2.4 years, $\delta^{13}C$ values began to rise, and continued to rise a total of 0.7‰ by 3.8 years of age, at which point values plateaued. As $\delta^{15}N$ continued to fall during this time, and $\delta^{13}C$ values were very low (approximately −21‰) for a child whom one would expect to be breastfed, this suggests two possible interpretations. Firstly, that falling $\delta^{15}N$ values represent declining intake of human milk, and that $\delta^{13}C$ remained low and stable because human milk was the primary source of higher-trophic level protein in the diet, with the bulk of total protein coming

from low-$\delta^{13}C$ C_3 plants until around the age of 2.5 years, when a greater allowance of animal-based proteins was introduced. While this is possible, it seems unlikely that increasing protein (and particularly an increase in trophic level of dietary protein) would increase $\delta^{13}C$ but not $\delta^{15}N$ values in otherwise healthy metabolic function. The second possibility is that high $\delta^{15}N$ and low $\delta^{13}C$ values in early infancy reflect pre-existing nutritional stress, which began to resolve in early postnatal life, being fully resolved by the end of the fourth year of life. Indeed, it is likely that aspects of both of these interpretations have merit. The timing of the steepest decrease in $\delta^{15}N$ is aligned with the timescales where solid foods would be expected to replace some milk volume, and early complementary foods would probably have been plant-based. Meanwhile, the concurrent increase in $\delta^{13}C$ and decrease in $\delta^{15}N$ between the ages of 2.4-3.8 years of age is consistent with an interpretation of increase in body mass associated with recovery from malnutrition, possibly due to an increase in the quantity of protein consumed. This would concur with the finding of Mekota *et al.* (2006) that increasing $\delta^{13}C$ was associated with both increases in body mass and increased meat and fat intake. This may represent three separate phases: maternal/foetal malnutrition, breastfeeding with low-protein complementary foods, and the completion of weaning and transition onto a higher-protein diet, with associated catch-up growth.

Unlike EH 453, EH 45 and EH 9, male skeleton EH 578 did not show evidence of skeletal stress markers but did display a wide range of isotopic profile variability over the course of tooth development. A pattern of opposing covariance was observed from the outset of the data profile (Figure 6.18), with $\delta^{13}C$ values rising by 0.7‰ and $\delta^{15}N$ decreasing by 2.2‰ between the ages of 0.3-1.5 years of age. This may indicate some combination of abating early life metabolic stress and dietary transition. Beyond this point, both $\delta^{13}C$ and $\delta^{15}N$ values maintained a plateau lasting until approximately 7.8 years of age. While $\delta^{13}C$ values were sustained, between 7.8-9 years of age $\delta^{15}N$ values rose by 2‰, remaining constant thereafter. Considering the age at which this change occurred, and the lack of concurrent change in $\delta^{13}C$ which might be expected if the shift were due to nutritional stress, it seems feasible that this latter shift may be accounted for by a shift to higher-trophic level protein consumption. EH 626A, a female, also bore no skeletal evidence of childhood stress or growth disruption. Despite this lack of skeletal markers, a "recovery bubble" is evident at the outset of her data series (Figure 6.28). Between the ages of 0.3-2.7 years of age $\delta^{15}N$ values fell by 1.1‰, while $\delta^{13}C$ rose by

0.3‰ during the same period. After this age, variance was low, and profiles essentially flat, signifying that diet and metabolism were likely to have remained relatively stable. EH 727, a young male, also did not exhibit stress markers, and initially demonstrated early dietary patterning. $\delta^{15}N$ values declined from 0.3 to 3.5 years of age, decreasing by 3.9‰ during this time (Figure 6.20). Thereafter, a plateau in values was maintained until the approximate age of six years, when $\delta^{15}N$ values began to rise. However, between approximately 2 and four years of age, $\delta^{13}C$ rose alongside dropping nitrogen, suggesting anabolic covariance. Between the ages of six and eight years, $\delta^{15}N$ rose by 1.5‰, hinting at a catabolic covariance bubble, before stabilising at this new level.

In contrast to the single Edix Hill non-survivor (EH 352) to show isotopic evidence of catabolic activity, none of these surviving individuals show evidence of rising $\delta^{15}N$ and falling $\delta^{13}C$, which would be typical of a starvation data bubble. Instead, these individuals show evidence of the rising $\delta^{13}C$ and falling $\delta^{15}N$ in their earliest data, typical of recovery from malnutrition. As this occurs in the earliest forming data, the absence of evidence for catabolic activity, and the suggestion of anabolic change, may indicate some intrauterine growth restriction which was ameliorated by catch-up growth and a postnatal return to normal metabolism. As antenatally-forming dentine is not present in the adult dentition, and thus generally unavailable in surviving individuals, this reading of the data must remain speculative.

7.3.2 Littleport

7.3.2.i Non-survivors

In contrast to the more constrained variability among Edix Hill non-survivors, variability within the Littleport non-survivor cohort was more erratic. None of the non-survivor isotope profiles at Littleport presented a classic "weaning curve," when the relationship between the trajectories of carbon and nitrogen were considered alongside developmental chronology. Shifts in isotope values which suggested a physiological, rather than dietary, causative origin were most prominently in evidence. Five out of the six individuals (83%) comprising the non-adult sample at Littleport presented isotopic profiles suggesting episodes of undernutrition in the first few years of life, with four of these occurring within the timeline represented by the first few increments of dentine.

In the profiles of LP 3311, LP 3770, LP 4116, and LP 4144, shifts in $\delta^{13}C$ and $\delta^{15}N$ were non-parallel and discordant, producing patterns of opposing covariance

characterised by high or increasing $\delta^{15}N$ alongside decreasing $\delta^{13}C$ values. For LP 3311 (Figure 6.30), the transition from antenatal to early postnatal (~4 months of age) dentine data demonstrated stability of $\delta^{13}C$ values and a modest increase of 0.3‰ for $\delta^{15}N$, which suggests a healthy pregnancy followed by the potential effects of breastfeeding. However, by approximately 8 months of age, LP 3311 experienced a concurrent 0.5‰ drop in $\delta^{13}C$ and 0.5‰ rise in $\delta^{15}N$ values, followed by a 0.2‰ rise in $\delta^{13}C$ and a 0.3‰ fall in $\delta^{15}N$ values during the first half of the second year of life. Taken together, this pattern of change is suggestive of an episode of nutritional stress, followed by a period of recovery and weight gain. For LP 3770, a decrease in $\delta^{15}N$ of 1.5‰ and more modest increase in $\delta^{13}C$ of 0.2‰ was observed between the first and second dentine increments (Figure 6.31), which may suggest undernutrition *in utero*, with improved nutrition during the first year of life. This pattern of early divergent $\delta^{13}C$ and $\delta^{15}N$ profiles followed by a small and gradual increase in values and stable values thereafter is suggestive of recovery following catabolic states experienced *in utero* but is not consistent with the expected profile for breastfeeding, based on modern data from Fuller *et al.* (2006). Neither LP 4116 (Figure 6.32) nor LP 4144 (Figure 6.33) produced $\delta^{13}C$ and $\delta^{15}N$ profiles consistent with an interpretation of breastfeeding: both show a general trend of decline in $\delta^{13}C$ and $\delta^{15}N$ over time, but much of that change in values is associated with episodes of opposing covariance; dietary interpretation is therefore tenuous. In terms of skeletal evidence of stress in these individuals, LP 3311 and LP 4116 had exhibited cribra orbitalia, while LP 4144 was observed to have extracortical woven bone on the pleural surface of the ribs, and LP 3770 had no documented skeletal pathology at all.

Only two non-surviving individuals at Littleport, neither of whom displayed visible skeletal pathology, produced any data suggestive of simple dietary shifts. In the profile of LP 4848 (Figure 6.35), $\delta^{13}C$ and $\delta^{15}N$ values decreased by 0.9‰ and 3.4‰, respectively, between the first and third increments (representing initiation at approximately 30 weeks *in utero* and a midpoint of 0.3 of a year after birth), before finally reaching a plateau around the middle of the first year of life. This parallel trend in isotopic values suggests a true dietary shift, rather than physiological causes, and may indicate that maternal diet was richer in higher trophic level foods during pregnancy than following birth. The continued drop following birth and flatter profile by approximately four months of age indicates that if this individual was breastfed, the

process of weaning was completed at a surprisingly young age for the early medieval period. Similarly, shifts in $\delta^{13}C$ and $\delta^{15}N$ within the profile of LP 4494 (Figure 6.34) followed a broadly parallel trend suggestive of true dietary shifts. A rise in both $\delta^{13}C$ and $\delta^{15}N$ from antenatal to postnatal values is consistent with the known metabolic effects of a healthy pregnancy and the onset of breastfeeding (Butte, 2000; Fuller *et al.*, 2004). The onset of a decline in $\delta^{13}C$ occurred by 6 months of age and continued steadily until values reached a plateau around 2.5 years of age. A similar, if more undulating, pattern of decline was seen for $\delta^{15}N$, with values dropping from approximately six months of age, and levelling out at 2.5 years of age. The undulating peaks occurring during the overall decline in $\delta^{15}N$ during this period may be associated with seasonal changes to complementary diet including higher-$\delta^{15}N$ foods or, more likely, may be expressive of nutritional stress, as $\delta^{13}C$ continued to decline during these sporadic peaks in $\delta^{15}N$ profile. The overall pattern observed for LP 4494 would be consistent with the introduction of complementary foods at around six months of age, with continued breastfeeding up to the age of 2.5 years.

7.3.2.ii Survivors

Unlike Edix Hill, where the distribution of non-survivor profile types paralleled those of the survivor cohort, survivors at Littleport showed substantial differences from the non-survivors. While Littleport non-survivor sample data was heavily skewed towards profiles showing opposing covariance (83%), survivor profiles were distributed more equally. Out of a sample of 24 adults represented, patterns of parallel covariance accounted for seven individuals (29%), two individuals had a flat profile (8%), and patterns of opposing covariance in the first years of life were associated with 15 individuals (63%).

Parallel covariance

A bias in sex was observed among individuals demonstrating predominant patterns of parallel covariance, with four females, two males, and one sexually-indeterminate individual among those represented.

Five individuals presented profiles which appeared to offer straightforward interpretations in their early data. Only one of these bore any evidence of skeletal stress markers associated with childhood development, although most exhibited opposing covariance expressive of nutritional stress in their late-series data. Strikingly, the only

individual in the parallel covariance group to exhibit a skeletal marker of childhood stress also did not show any signs of opposing covariance at any point in the data series, a singular circumstance at Littleport. LP 4047, a female with cribra orbitalia, showed parallel covariance throughout her data series. $\delta^{13}C$ values declined by 0.6‰ between 0.3 and 1.0 year of age, finding a plateau thereafter, while $\delta^{15}N$ values dropped by 2.3‰ between 0.3 and 2.7 years of age, before plateauing at that level (Figure 6.52). This pattern is strongly suggestive of the weaning process commencing during the middle of the first year of life, with complementary feeding continuing until the middle of the third year of life. As most individuals bearing cribrotic lesions also exhibited abnormal patterns of opposing covariance later in isotopic profile, it is intriguing to speculate on the possible reasons for LP 4047's break from this pattern. It is possible that compensatory inputs are masking the underlying physiological trends in this individual, or that sampling resolution is insufficiently fine to capture these changes in this specific instance.

Other individuals with clear-cut data patterns did not express skeletal stress markers of any kind. LP 4092, a male, exhibited a parallel decline in $\delta^{13}C$ and $\delta^{15}N$ values of 0.3‰ and 2.5‰, respectively, between the ages of 0.3 and 1.0 year of age (Figure 6.40). Following one year of age, both profiles were flat, suggesting that the weaning process was complete and diet stable after this age. An episodic 0.4‰ increase in $\delta^{13}C$ and a 0.2‰ decrease in $\delta^{15}N$ occurred between the ages of 7.9 and 8.6 years, returning to previous values thereafter. This may be interpreted as a potential pre-pubertal growth spurt. LP 4134, a second male, displayed a similar pattern of early decline and flat profile in early childhood (Figure 6.42). $\delta^{13}C$ values dropped by 0.3‰ between approximately four months and one year of age, while $\delta^{15}N$ values dropped by 1‰ between 0.3-1.7 years. Between 1.7 and 6.5 years of age, values remained similar, suggesting that no further dietary shifts occurred during this time, and that weaning had been completed by 1.7 years of age. Between 7.2 and 7.9 years, a minor episode of opposing covariance occurred, comprising a 0.2‰ drop in $\delta^{13}C$ and rise in $\delta^{15}N$, with values remaining static to the end of the series. LP 3745, a female, evidenced a slightly longer decline (Figure 6.48). Her $\delta^{13}C$ dropped 0.6‰ between 0.3 and 1.4 years of age, while $\delta^{15}N$ fell by 3.6‰ between 0.3 and 2.6 years of age, before reaching respective plateaus in value. Some variability in diet appears to have occurred beyond 6.6 years of age, with changeable $\delta^{13}C$ varying by 0.2-0.5‰ between increments, before a stress

bubble of dropping $\delta^{13}C$ and rising $\delta^{15}N$ emerged between 8.9 and 9.4 years. LP 4556, a very young adult, and the sole individual of sexually-indeterminate status in the sample, showed a minor 0.1‰ drop in $\delta^{13}C$ (not above instrument error) between 0.3 and 1.0 year of age, alongside a 1.9‰ drop in $\delta^{15}N$ between 0.3 and 1.6 years (Figure 6.59). After this period, the profile exhibited parallel covariance as $\delta^{13}C$ and $\delta^{15}N$ rose 0.5‰ and 1.3‰, respectively, by 5.6 years of age. However, after 6.9 years, a pattern of opposing covariance characterised by dropping $\delta^{13}C$ and rising $\delta^{15}N$ is apparent. Between 6.9 and 9.5 years of age, $\delta^{13}C$ fell by 0.5‰ and $\delta^{15}N$ rose by 2.1‰. LP 4556 was chosen for inclusion in the sample due to their differential diagnosis of skeletal tuberculosis, and a desire to identify potential anomalies in profile associated with the onset of active disease. Considering this individual's young age-at-death, and the chronic duration of disease required for skeletal changes, the nutritional stress signified by the emergence of opposing covariance in $\delta^{13}C$ and $\delta^{15}N$ may signal the onset of active disease in this individual. The fact that he or she survived several years beyond the onset of clinical disease also indicates that this individual was likely well cared-for.

The remaining two individuals demonstrating parallel covariance showed more complex patterns. LP 4035, a female, did not exhibit stress markers, but showed a very unusual, and indeed unique, pattern of change in values over time, which probably also indicated an increase in protein intake. The profile expresses low starting $\delta^{13}C$ and $\delta^{15}N$ values at 0.3 years of age, which rise over time, and then find a plateau at a higher level (Figure 6.51). $\delta^{13}C$ values rose 1.1‰ by 3.6 years, before finding a plateau. $\delta^{15}N$ values also rose, from 0.3 years to 2.3 years, increasing by 1.5‰ over that period, before dipping slightly and finally finding a steady plateau at 3.6 years. Values for both $\delta^{13}C$ and $\delta^{15}N$ remained static from this point to the end of the series. This does not fit the expected pattern for breastfeeding and weaning, but also does not exclude the possibility of breastfeeding inputs. Clearly, trophic level inputs in early childhood increased from infancy, and it is possible that this child experienced a change in circumstances which led to a complementary diet which was richer in animal proteins, such as meat and fish, than was the diet of her mother or wet-nurse. Alternately, her mother's diet may have changed during this time to including higher-trophic level proteins.

LP 4063, also a female with no evidence of skeletal stress markers, proffered similar impediments to simple dietary interpretation. $\delta^{13}C$ values dropped 1‰ between 0.3 and

1.0 year of age, forming a flatter profile from that point (Figure 6.53). However, $\delta^{15}N$ patterning is more difficult to parse, as values decrease gradually throughout the series, dropping by 3.9‰ over the course of incremental data. As this decline in $\delta^{15}N$ is not paralleled by a similar decline in $\delta^{13}C$, it is unlikely to represent a long-term dietary trend, particularly as $\delta^{13}C$ values rise subtly over the course of increments. As $\delta^{15}N$ values at the outset of the series are very high, it may be that these were artificially inflated by systemic stress, and the timewise decrease in values is expressive of reducing stress levels. Even within this potential masking of dietary patterning by other physiological processes, it may be possible to infer with some confidence the timing of dietary transition. $\delta^{15}N$ values dropped most steeply from 0.3 to 1.6 years of age, falling 1.8‰, reaching a brief plateau after that age before beginning to again descend. It is therefore likely that this brief plateau in values marks the end of dietary shifts, and that the weaning process was initiated around six months of age, with full cessation of breastfeeding taking place around 1.6 years of age.

Flat profiles

Only two adult individuals (8.3%) at Littleport demonstrated at least one flat profile: LP 4067 and LP 4395. Both individuals were female. LP 4067 displayed no skeletal stress markers and showed an overall flat pattern of $\delta^{13}C$ values (Figure 6.54). A 0.2‰ decrease in $\delta^{13}C$ was observed between 0.3 and 1.0 year of age; however, as similar values were seen in later childhood incremental data, this cannot be seen as a systematic age-related increase in values. $\delta^{15}N$ values declined sharply between 0.3 and 2.5 years of age, falling 3.7‰ in that time, before finding a level position. LP 4395, who had cribra orbitalia, showed a similar pattern of flat $\delta^{13}C$ profile, with no systematic age-related variability in values (Figure 6.57). The profile of $\delta^{15}N$ values for LP 4395 was similar to LP 4067, with a 3‰ drop in values between 0.3 and 2.9 years of age, before an overall plateau was reached. As neither individual exhibited a parallel age-related and systematic decrease in $\delta^{13}C$ and $\delta^{15}N$ values which might suggest a dietary transition (such as the weaning process), it is difficult to confidently argue for the substantial decline in $\delta^{15}N$ exhibited by both individuals as being dietary in origin. A possible explanation for the lack of substantial rise in $\delta^{13}C$ alongside elevated $\delta^{15}N$ values might be an attenuation of the isotopic effects of breastfeeding by maternal nutritional stress.

Opposing covariance

Approximately 37% of adult individuals fell into the combined categories of parallel covariance and flat profiles during infancy. However, a more substantial 15 out of 24 individuals (63%) at Littleport represented a majority displaying isotope profiles in infancy suggestive of opposing covariance. This group comprised nine males and six females. Of these individuals, most (67%) also exhibited cribra orbitalia, with no sex differential: 67% of each sex in the subgroup presented with the lesion.

Six males with cribra orbitalia, despite all displaying patterns of opposing covariance, presented with very different profile patterns. The first of these, LP 3885, showed a largely flat pattern of $\delta^{13}C$ and $\delta^{15}N$ values over time, interspersed with episodes of low-variability opposing covariance (Figure 6.37). The recovery phase of an apparent malnutrition bubble, with rising $\delta^{13}C$ and dropping $\delta^{15}N$, is visible at the outset of the data series, and resolves by 1.8 years. A second episode of probable malnutrition appears between 6.9 and 8.5 years of age, with $\delta^{15}N$ values rising by 0.8‰ between 6.9-8.0 years, and no concurrent rise in $\delta^{13}C$. Between 8.0 and 8.5 years of age, $\delta^{15}N$ decreased by 0.6‰, suggesting some abatement of nutritional stress. However, a further episode of opposing covariance occurred between 8.5-9.0 years, with $\delta^{15}N$ rising by 0.3‰ and $\delta^{13}C$ dropping by 0.4‰. Parallel covariance was restored by 9.5 years of age, as $\delta^{13}C$ and $\delta^{15}N$ values both increased by 0.3‰ from 9-9.5 years. Due to the non-systematic and non-parallel pattern of change within the profile of LP 3885, it was not possible to discern clear dietary transitions; nonetheless, this individual appears to have been subject to intermittent and recurrent episodes of malnutrition.

LP 4095 (Figure 6.41) presented a similar isotopic pattern of recurring episodes of malnutrition alongside cribra orbitalia. Initially, this individual showed parallel drops in $\delta^{13}C$ and $\delta^{15}N$ of 0.2‰ and 0.4‰, respectively, between the ages of 0.3 and 1.0 year. Nevertheless, between 1.0 and 2.2 years of age, this parallel covariance was replaced by opposing covariance, with a rise in $\delta^{13}C$ of 0.5‰ and a further decline in $\delta^{15}N$ of 0.7‰, before both $\delta^{13}C$ and $\delta^{15}N$ profiles plateaued. While the initial drop in $\delta^{13}C$ and $\delta^{15}N$ may be interpreted as the onset of the weaning process, the subsequent emergence of opposing covariance via the rise in $\delta^{13}C$ is difficult to justify as a dietary shift alone, due to the very low value (−22.1) achieved. Interpreting this rise as an increase in normal growth also fails to satisfy, as gradual deceleration of growth velocity characterises the first two years of life, with the highest rates of growth occurring in the first year (Rogol

et al., 2000). Thus, a sudden increase in growth in the second year of life is unlikely, unless this increase is catch-up growth due to an abatement of earlier metabolic stress. It is therefore probable that this pattern reflects the combined effects of decreasing stress and the weaning process. Between the ages of 4.8 and 7.0 years of age, a second episode of covariance occurred, with $\delta^{13}C$ dropping, and $\delta^{15}N$ rising, by 0.2‰. A temporary recovery in $\delta^{13}C$ of 0.3‰ followed between 7-7.8 years of age, before profiles began again to covary. Between 7.8 and 10 years, $\delta^{13}C$ declined by 0.2‰ and $\delta^{15}N$ rose by 0.8‰. The overall profile is suggestive of the weaning process being initiated during the first year, and of major transitions being completed by 2.2 years of age, but due to the frequent appearance of opposing covariance within the profile, LP 4095's childhood data was characterised by episodic illness or deprivation, resulting in malnourishment.

In terms of variability, LP 4178 (Figure 6.44) produced the largest-scale shifts in $\delta^{15}N$ among Littleport adults, and these occurred in the context of opposing covariance during the first few years of life and the presence of cribra orbitalia. Both $\delta^{13}C$ and $\delta^{15}N$ values were static over the first few points of incremental data. $\delta^{15}N$ values began to fall only after 0.8 years of age, dropping by 4.3‰ between 0.8 and 3.5 years. $\delta^{13}C$ values remained static into the second year of life but rose 0.7‰ between 1.4 and 3.5 years of age. A brief period of parallel covariance was observed from 3.5-5.2 years of age, with the remainder of the profile series distinguished by two successive episodes of opposing covariance. These occurred from 5.2 to 8.4 years of age, and 8.4 years to the end of the series at approximately 10 years of age, with a full recovery in values not occurring during this period. The high initial $\delta^{15}N$ values (16.5‰), and sharp decline alongside rising $\delta^{13}C$ values signifies recovery from a severe episode of early life stress, when the attenuation of values due to sampling across multiple developmental layers is taken into consideration (Beaumont and Montgomery, 2016). As early extreme values maintain a plateau through the first few increments, it appears that whether initiated *in utero* or postnatally, systemic stress did not begin to resolve until well into the second year of life. The overall course of childhood health and development for LP 4178 appears to have been mediated by episodic metabolic dysfunction.

The remaining males evidencing cribra orbitalia alongside "stressed" profiles of opposing covariance presented less dramatic shifts in profile. Between 0.3 and 3.1 years of age, LP 4250 (Figure 6.45) showed a 0.8‰ rise in $\delta^{13}C$ and a drop $\delta^{15}N$ values of 0.9‰, before reaching a plateau in both profiles which lasted for several years. This

does not follow the expected pattern for breastfeeding and weaning, where the magnitude of change in $\delta^{13}C$ due to trophic level effects should be much less than the increase in $\delta^{15}N$. Here, the change in $\delta^{13}C$ and $\delta^{15}N$ is nearly equal, and $\delta^{13}C$ values are much lower at the outset of the data series than they are in later data, which would be expected to represent post-weaning childhood diet. Rather than reflecting a classic weaning curve in the first three years of life, LP 4250 appears to present resolution of possible early-life stress. The plateau which followed was interrupted by a series of episodes of opposing covariance, with an initial bubble appearing at approximately 6.5 years of age, and resolving by 9.5 years, and a drop in $\delta^{13}C$ and minor $\delta^{15}N$ rise between 9.5 and 10.0 years signalling the possible beginning of a second stress bubble.

LP 4603 (Figure 6.46) did not present the same pattern of apparent initial stress recovery, instead showing parallel decline in both $\delta^{13}C$ and $\delta^{15}N$, of 0.4‰ and 1.7‰ respectively, between the ages of 0.3 and 0.9 years. However, following this early pattern suggestive of a weaning shift, the profile presents a series of alternating opposing covariance and parallel covariance. The majority of opposing covariance was characterised by increasing $\delta^{13}C$ and falling $\delta^{15}N$ values, interspersed by periods of plateau, which probably represents cyclical periods of growth. These occurred between 1.4-2.6 years and 4.3-7.7 years. However, between 7.7 and 8.9 years of age, a parallel drop in both $\delta^{13}C$ and $\delta^{15}N$ occurred, followed by a drop in $\delta^{13}C$ of 0.4‰ and a rise in $\delta^{15}N$ of 1.7‰ between 8.9 years and the end of the data series at 10 years of age. The parallel drops in $\delta^{13}C$ and $\delta^{15}N$ in advance of the onset of catabolic stress suggests that this individual experienced a reduction in both quality and quantity of dietary protein, which may have contributed to the development of malnutrition. Protein intake may also have mediated the metabolic patterning of LP 4173, a male with cribra orbitalia. He presented $\delta^{13}C$ values which dropped by 0.2‰ between 0.3 and 0.9 years of age, before reaching a plateau (Figure 6.43). $\delta^{15}N$ values also declined in the first few increments, dropping 1.1‰ by 0.9 years, and a full 1.8‰ by 2.4 years of age. Both $\delta^{13}C$ and $\delta^{15}N$ decreased slightly between 2.4 and 3.2 years, but an episode of opposing covariance occurred between 3.2 and 5.5 years of age, resulting in a drop of 0.3‰ for $\delta^{13}C$ and a rise in $\delta^{15}N$ of 0.6‰ during that period. It may be that an overall decrease in dietary protein during this period was a predisposing factor for the development of malnutrition. Beyond 5.5 years, both $\delta^{13}C$ and $\delta^{15}N$ rose, which may signify recovery and an increase in the trophic level of dietary protein.

The four females at Littleport exhibiting both cribra orbitalia and patterns of opposing covariance, like their male counterparts, presented profiles which were strongly marked by alternating catabolic and anabolic activities. In some cases, there were no discernible instances of parallel covariance. The data profile of LP 3749 (Figure 6.49) offered such an example, consisting of a series of oppositional rises and falls in $\delta^{13}C$ and $\delta^{15}N$. From the age of 0.3 to 3.8 years, $\delta^{13}C$ rose by 0.4‰, while $\delta^{15}N$ declined by 2.1‰. Between 3.8 and 6.5 years, the trend reversed as $\delta^{13}C$ dropped by 0.3‰ and $\delta^{15}N$ rose by 0.8‰, before somewhat recovering between 6.5 and 8.6 years of age, as $\delta^{13}C$ values increased by 0.2‰ and $\delta^{15}N$ fell by 0.4‰. Immediately following this catabolic/anabolic bubble, $\delta^{15}N$ again rose by 0.6‰ between 8.6 and 10 years of age, with no corresponding rise in $\delta^{13}C$ values. LP 4585 (Figure 6.58) also showed consistent divergence in patterns of $\delta^{13}C$ and $\delta^{15}N$ change. A large 2.5‰ decrease in $\delta^{15}N$ occurred from 0.3-1.0 year of age, with no accompanying change to $\delta^{13}C$ values. From one year of age to 5.2 years $\delta^{13}C$ steadily increased by 1.1‰, while $\delta^{15}N$ decreased by 1‰ over the same period. From 5.2 to 10 years $\delta^{13}C$ was in decline, falling by 1‰, and $\delta^{15}N$ values increased by 1.6‰. LP 3819 (Figure 6.50), an individual for whom early dietary data was not available (due to tooth damage), did show short-lived parallel covariance. Her data profile showed parallel rises in both $\delta^{13}C$ and $\delta^{15}N$ from 4.6 to 6.4 years. However, from 6.4 to 9.3 years a bubble of opposing covariance was observed; from 6.4 to 7.8 years $\delta^{13}C$ fell by 0.5‰, with $\delta^{15}N$ rising by 0.7‰. Between 7.8 and 9.3 years, the direction of change reversed, with $\delta^{13}C$ rising by 0.3‰ and $\delta^{15}N$ dropping by 0.5‰, and static values at the close of the series. LP 4139 (Figure 6.56) also presented initial parallel covariance, with a drop in $\delta^{13}C$ of 0.2‰ and a larger reduction of 2.4‰ in $\delta^{15}N$ values between 0.3-1.0 year of age. Nevertheless, from one to four years of age, $\delta^{13}C$ began to climb, rising by 1.2‰, while $\delta^{15}N$ values fell by 1.3‰ over this period. Following this phase of anabolic covariance, homeostasis was maintained from four to 6.3 years. From 6.3-10 years of age, a pattern of opposing covariance again resumed: alternating catabolic and anabolic phases occurred from 6.3-7.8 years, and from 7.8 to 10 years $\delta^{13}C$ values declined by 0.6‰ alongside rises in $\delta^{15}N$ totalling 1.1‰.

Only five individuals, representing three males and two females, presented with patterns of opposing covariance and *without* skeletal stress markers. Out of these individuals, LP 4075, a female, presented the least interpretatively challenging data

series (Figure 6.55). From 0.3 to 3.5 years of age, the profile of LP 4075 demonstrated declining $\delta^{15}N$ values and an ascending pattern of $\delta^{13}C$: respective changes consisted of a total $\delta^{15}N$ decrease of 1.5‰ and $\delta^{13}C$ increase of 0.6‰ during that time, with low variability thereafter. As with previous individuals at both sites, this pattern is characteristic of either recovery from antenatal or perinatal nutritional stress, accompanied by an increase of body mass, or increased weight gain associated with a transition to higher caloric intake associated with the normal growth or the weaning process. The former of these hypotheses carries more weight, due to the non-coincidence of these rises with the developmental period of greatest growth acceleration. Between the ages of 0.3 and 3.3 years, LP 3708, a male, also produced a characteristic growth/recovery pattern, with $\delta^{15}N$ values which fell by 2.2‰ and $\delta^{13}C$ which rose by 0.4‰ (Figure 6.36). Following a plateau from 3.3 to 6.3 years, values again began to change. Following a brief episode of opposing covariance between 6.3 and 7.0 years of age, followed by the resumption of homeostasis from 7-7.8 years, $\delta^{13}C$ and $\delta^{15}N$ both declined from 7.8 to 8.5 years. $\delta^{13}C$ continued to fall from 7.8 to 10 years, dropping by 0.3‰, but from 8.5 years, $\delta^{15}N$ began to climb, rising a total of 1.4‰ by 10 years of age. It seems likely that the parallel drop in $\delta^{13}C$ and $\delta^{15}N$ signifies a change in diet linked to the onset of malnutrition. Alternately, the initial drop in $\delta^{15}N$ may reflect a loss of protein in the diet, followed by a rise in values caused by catabolism of body proteins; while $\delta^{13}C$ values fall earlier, reflecting the body's preferential strategy of catabolising body fat prior to muscle mass. LP 3893 (Figure 6.38), also male, offered a similar profile demonstrating increasing $\delta^{13}C$ and declining $\delta^{15}N$ from the outset. From 0.3 to 4.2 years of age, $\delta^{13}C$ rose by 0.5‰ while $\delta^{15}N$ fell by 2.7‰. After a few ensuing months of parallel covariance, from 5.5 years of age the data series demonstrates nearly consistent opposing covariance. Initially, this was manifested in an increase of $\delta^{13}C$ values of 0.3‰ and a decrease in $\delta^{15}N$ of 0.6‰ between 5.5 and 6.1 years. From 6.1 to 9.7 years, $\delta^{15}N$ values rose by 1.1‰, while $\delta^{13}C$ initially remained static before decreasing 0.3‰ by 9.7 years.

The remaining individuals required more nuanced interpretation. The final male in this group, LP 4073 (Figure 6.39), also displayed a pattern of climbing $\delta^{13}C$ values with no parallel change in $\delta^{15}N$ in early data. While it is possible that this individual was never breastfed, both the pervasiveness of flat profiles in this study and the implications of unilateral increase $\delta^{13}C$ should elicit consideration of physiological, rather than

dietary, shifts. An increase of 0.2‰ in $\delta^{15}N$ mirrored a 0.2‰ in $\delta^{13}C$ between 0.3 and 0.8 years of age, but from 0.8-1.9 years $\delta^{15}N$ values remained static, while $\delta^{13}C$ continued to rise, increasing by a total of 0.8‰ between 0.3-1.9 years. From 1.9 to 3.5 years values remained largely fixed on both axes, but beyond 3.5 years values were in a constant state of change. From 3.5 to 4.6, $\delta^{15}N$ rose by 0.7‰ with no corresponding change in $\delta^{13}C$. Opposing covariance was observed throughout the remainder of the series, with a 1.3‰ drop in $\delta^{15}N$ and a 0.3‰ rise in $\delta^{13}C$ between 5.7 and 6.8 years of age, and two further nutritional stress bubbles visible between 6.8-7.9 and 7.9-9.5 years. The latter of these bubbles did not see resolution by the end of the series, with $\delta^{13}C$ dropping by 0.9‰ alongside a 0.2‰ rise in $\delta^{15}N$. This data demonstrates that a lack of metabolic homeostasis characterised not only the early period, expected to reflect dietary shifts (where none are visible here), but also middle and later childhood, which might be expected to reflect more constant values. In contrast to the pattern of rising $\delta^{13}C$, which potentially signals growth and recovery, LP 3687 exhibited early-profile patterning suggestive of active postnatal malnutrition (Figure 6.47). Between 0.3 and 0.9 years of age, $\delta^{15}N$ rose by 0.4‰ and $\delta^{13}C$ dropped by 0.3‰. This is more interpretatively consistent with malnutrition than with the expected parallel increases or decreases of breastfeeding and weaning. A parallel decrease in both $\delta^{15}N$ and $\delta^{13}C$ succeeded this opposing covariance, with decreases of 1.4‰ for $\delta^{15}N$ and 0.4‰ for $\delta^{13}C$ by 1.6 years of age. This may represent a genuine dietary shift, and is probably associated with weaning, based on the developmental timescales. Following this parallel decline, $\delta^{15}N$ and $\delta^{13}C$ again exhibited opposing covariance, with a 0.2‰ rise in $\delta^{13}C$ and a concurrent 0.2‰ decrease in $\delta^{15}N$ between 1.6 and 2.2 years of age, followed by a unilateral drop in $\delta^{13}C$ between 2.2 and 2.9 years of 0.5‰. Only minor variance was observable between the ages of three and seven years. Between 7.4 and 9.4 years, a bubble of opposing covariance was evident, demonstrating a 1.2‰ rise in $\delta^{15}N$, and variance in $\delta^{13}C$ of 0.2‰. Taken as a whole, the pattern of childhood isotopic change for LP 3687 is distinguished by its strong elements of suggested stress, with intermittent periods of dietary change, and it is likely that these factors were connected.

7.3.3 Discussion of individual data patterns

The importance of interpreting individual longitudinal palaeodietary data with simultaneous reference to both carbon and nitrogen stable isotope profiles is underlined by the results of this study, as the narrative suggested by the pattern of $\delta^{15}N$ and $\delta^{13}C$

shifts relative to each other may differ strongly from the narrative suggested by either of the profiles singly. Many of the $\delta^{15}N$ profiles, which in isolation might suggest the weaning process or an increase in protein consumption, when paired with $\delta^{13}C$ data tell a very different tale of childhood nutritional status. Evaluation of these relative relationships improves the likelihood of correctly interpreting shifts in profile as dietary or physiological in nature, and this has been the motivating factor behind the painstaking individual assessment of this large dataset. Ultimately, combining complementary individual and aggregate data (discussed in Section 7.4), can offer insights and identify patterns not evident when analysing data via one method only. A combined approach can promote understanding of the dynamics of individual organisms within the larger populations they help to comprise.

Examination of individual childhood dietary profiles within the context of site, survivorship, and estimated sex highlighted some interesting differences between cohorts. Most striking of the differences which fell along these lines of division were the contrasts which emerged when individuals were compared by other demographic variables. Statistical analysis via Fisher's exact test (Table 6.7) of the pooled sample from both sites revealed relationships between cribra orbitalia and site location ($p=0.0045$) and opposing covariance and site location ($p=0.0064$). No relationship was found between biological sex and opposing covariance ($p=1.0000$). Attempts to directly link cribra orbitalia and opposing covariance found associations between cribra orbitalia and opposing covariance in early childhood ($p=0.0252$), as well as opposing covariance at any age ($p=0.0408$). These associations retained significance after Holm-Bonferroni corrections (corrected values reported here).

The marked association of place with the development of cribra orbitalia and opposing covariance aids in interpreting the pattern of cohort-based biological outcomes. Opposing covariance is known to result from disturbance to homeostasis in protein metabolism (Fuller *et al.*, 2004; Fuller *et al.*, 2005; Mekota *et al.*, 2006), and thus it is likely that development of cribra orbitalia and metabolic disequilibrium share a common cause in this environment. The differences between TPR of cribra orbitalia at the sites (18.3% at Edix Hill vs. 59.5% at Littleport) have been previously discussed (see Chapter 5) and are demonstrated to reach statistical significance in this sample. Location-based disparities between the samples are also evident in the pattern of distribution based on isotopic profile type (Table 7.1). Differences in representation

between survivors and non-survivors at Edix Hill are low, with similar proportions observed in each of the categories. In contrast, greater disparities between survivors and non-survivors at Littleport were evident. Specifically, non-survivors at Littleport possess a much higher proportion of individuals displaying opposing covariance, and lower representation of profiles showing parallel covariance. Isotopic patterning thus supports an interpretation of stress differentials previously suggested by skeletal pathology. Clearly, the levels of childhood stress common at Littleport were not shared by children at Edix Hill. Furthermore, the lower levels of skeletally-manifested stress at Edix Hill also produced fewer differences in isotopic patterning between survivors and non-survivors, while stressors leading to opposing covariance may have been a more decisive factor in survivorship at Littleport.

	Parallel Covariance (%)	**Flat Profile (%)**	**Opposing Covariance (%)**
Edix Hill			
Non-survivors	33	45	22
Survivors	40	30	30
Littleport			
Non-survivors	17	0	83
Survivors	29	8	63

Table 7.1 Distribution of profile pattern types within the study sample

Opposing covariance observed at both sites, but particularly at Littleport, has implications beyond suggesting material differences in the levels of stress negotiated by survivors and non-survivors. The pattern of opposing covariance most commonly observed in individuals at both sites is an early-life anabolic pattern, which is most consistent with the phenomenon of antenatal and/or early postnatal malnutrition, and subsequent catch up growth. Undernutrition *in utero* alongside improved nutrition and rapid growth in early childhood has been linked with decreased longevity and higher risk of obesity, diabetes mellitus, cardiovascular disease, and other manifestations of metabolic dysfunction. This concept is termed the "Barker Hypothesis" or the Developmental Origins of Health and Disease (DOHaD) (Barker *et al.*, 2002; Schulz, 2010). Increased susceptibility to chronic disease is thought to occur as an individual adapts to undernutrition during foetal life by downregulating growth, developing a permanently "thrifty phenotype" which is programmed to utilise limited resources. Where these infants are subsequently introduced into a postnatal environment of nutritional affluence, the abundance of nutrients creates a predisposition to overnutrition

and adiposity. Infants born following intrauterine growth restriction (IUGR) may consequently begin postnatal life with abnormal protein and glucose metabolism and, in addition to having elevated risks of metabolic syndrome in adulthood, are also at higher risk for postnatal growth failure (de Boo and Harding, 2007; Thureen, 2007).

While foetal programming is known to create permanent impacts for lifelong metabolic function, the deleterious effects of foetal growth restriction seem to be somewhat modulated by postnatal diet (Hales and Ozanne, 2003; Dessì *et al.*, 2012). The timing of childhood catch up growth appears to be a critical factor in the level of risk for adult cardiovascular and metabolic disease, with later catch-up growth being associated with better long-term outcomes (Tosh *et al.*, 2010; Wiedmeier *et al.*, 2011). Breastfeeding is thought to be protective against these deleterious sequelae of IUGR, through the low protein content and metabolically-active regulatory hormones of human milk (previously discussed in Chapter 3), which delay catch-up growth beyond the crucial developmental-programming window of early infancy (Agostoni, 2005; Jain and Singhal, 2012). Where anabolic activity in the profiles shown in this study appears as a period of later-than-anticipated growth in the second and third years of life, this may be representative of delayed catch up growth, and a protective mechanism of breastfeeding in these populations. Nonetheless, as higher risks of long-term obesity and chronic disease would have faced these individuals in adulthood, future comprehensive studies on high-stress Fenland populations should attempt to address the longer-term health impacts of early life stress in adult individuals.

7.4 Grouped data

In addition to analysis of individual trajectories, isotopic data were analysed in group and comparative cohorts to better highlight group-level patterning.

7.4.1. Edix Hill

Differences in patterning between cohorts at Edix Hill were subtle. Non-survivor deciduous dentine data exhibited predominantly flat patterning for both $\delta^{15}N$ (Figure 6.60) and $\delta^{13}C$ (Figure 6.61) profiles; with only a few individuals showing substantial changes in value between antenatal, early postnatal, and later measurements. Overall, a subtle reduction in values was observed, with a lower range of values presented at 3.5 years of age than in earlier data. This may represent either a breastfeeding effect or reducing stress during early infancy, but since both $\delta^{15}N$ and $\delta^{13}C$ values both decline

slightly over time, it is likely that dietary inputs are implicated. An interpretation which gives predominant weight to dietary causes for patterning is weakly supported by a positive correlation between $\delta^{15}N$ and $\delta^{13}C$ values (Figure 6.62), although the moderate level of scatter indicates considerable heterogeneity. Due to the predominance of flat profiles, it is not possible to generalise regarding milestone ages for the weaning process for the non-survivor group.

Permanent tooth data presented a broadly similar picture for individuals surviving into adulthood. Males and female $\delta^{15}N$ and $\delta^{13}C$ profiles both included mixed patterning, with both flat and curved declining profiles represented and curved patterns predominating (Figures 6.63 to 6.66). Both males and females had $\delta^{15}N$ and $\delta^{13}C$ patterning consistent at a group level with the weaning process being completed by 2-3 years of age, with reduced variability after that time. However, in terms of $\delta^{15}N$ patterning, females (Figure 6.65) showed overall higher levels of variability than did males at Edix Hill (Figure 6.63), and this difference was maintained throughout the time series. This was not mirrored in $\delta^{13}C$ patterning, which showed low variability in both sexes (Figure 6.64 and 6.66). This sex-based imbalance of isotope-specific variability over the course of childhood indicates a non-dietary cause and, as $\delta^{15}N$ profile variability has been suggested to represent a deleterious loss of homeostasis (Beaumont, 2013), may indicate higher levels of stress for female children at Edix Hill. However, in terms of overall range or pattern, no appreciable differences between male and female $\delta^{15}N$ and $\delta^{13}C$ profiles were apparent (Figures 6.67 and 6.68). Comparison of scatter for all M1 increments also revealed a very weak correlation between $\delta^{15}N$ and $\delta^{13}C$ values (Figure 6.69), and no sex-based differences in distribution (Figure 6.70).

Comparisons between survivors and non-survivors also yielded few marked contrasts. Non-survivor data for both $\delta^{15}N$ and $\delta^{13}C$ plotted within the range defined by survivor dentine (Figure 6.71 and 6.72), as did scatter patterning (Figure 6.77). Data for individuals with stress markers showed that non-survivors had $\delta^{15}N$ profiles at the high end of values (Figure 6.73) and $\delta^{13}C$ profiles which dropped into lower values (Figure 6.74) relative to surviving individuals bearing stress markers, but neither was markedly different from individuals without stress markers. This pattern was also followed for cribra orbitalia; $\delta^{15}N$ for an adult male appeared at the centre of data, while a single non-survivor was positioned within the high end of the main concentration of data (Figure 6.75), and for $\delta^{13}C$ the surviving male also plotted centrally, while the non-

survivor appeared at the low end of the data range (Figure 6.76). The pattern indicated by stress marker data suggests differences in the experience of stress between survivors and non-survivors bearing skeletal stress markers, as reflected in their metabolic activity. However, it is difficult to draw firm conclusions from this data, due to the small numbers of "stressed" individuals represented at Edix Hill. Furthermore, cohort data at Edix Hill shared more similarities than differences, indicating that overall disparities in childhood experience of chronic stressors would have been modest.

7.4.2 Littleport

In contrast to the subtle differences in isotopic patterning between cohorts at Edix Hill, differences between groups at Littleport were more pronounced. Flat profiles were totally absent from the non-survivor group. Instead, individual non-survivor profiles demarcated two groups. For $\delta^{15}N$, these groups converged over time: one group was characterised by high but declining $\delta^{15}N$ values, and a second, lower $\delta^{15}N$ group exhibited an early drop in values, followed by a flat or slightly rising profile (Figure 6.78). These same groupings of individuals were present for $\delta^{13}C$ with a diverging trend following early similarity of values. The group which exhibited high $\delta^{15}N$ with gradual decline demonstrated dropping $\delta^{13}C$ values over time; while the lower-$\delta^{15}N$ value group showed $\delta^{13}C$ values which either dropped early before rising again or rose steadily from antenatal values to the end of the series (Figure 6.79). The discrepancy between the two subgroups in the non-survivor cohort was also reflected in the scatter of increment data (Figure 6.80), which displayed a bipartite clustering pattern. Variability in this group was sufficiently high that, as with the non-survivors at Edix Hill, conclusions regarding the timing of the weaning process based on group-based patterning were not possible. However, the difference between visible patterns of opposing covariance in individual profiles, and the lack of similar aggregate-level patterns, illustrates the limitations of aggregate-based approaches for distinguishing between dietary and non-dietary patterns of change. In most cases among non-survivors at Littleport, metabolic stress is likely to be masking dietary patterning.

Patterns in survivor M1 data were wholly different to those of non-survivors at Littleport. Male survivors exhibited a mixture of flat and curved $\delta^{15}N$ profiles (Figure 6.81), with a tight overall clustering of initial values, and most individuals having similar values at the outset and close of data series. Female $\delta^{15}N$ patterning displayed dissimilarity to the males (Figure 6.83), having a wide variability in initial values, but

more uniformity in shape with nearly all profiles presenting a descending curve. Both male and female groups displaying curves achieved flatter patterning by three years of age, suggesting that the weaning process was completed for most individuals by that age. As with non-survivors, $\delta^{15}N$ data for surviving females showed a two-tiered pattern of high and low value profiles. Contrasts were also seen between males and females in terms of their $\delta^{13}C$ patterning, with males (Figure 6.82) exhibiting higher variability than females (Figure 6.84), and an overall lower range of distribution in $\delta^{13}C$ values. For both $\delta^{15}N$ and $\delta^{13}C$, female data was more uniform in pattern and shape, while male data was more heterogeneous and variable. When plotted together longitudinally, the male data predominantly fell into the upper tier of the female data range for $\delta^{15}N$ (Figure 6.85), while the male $\delta^{13}C$ range was broader than the female range at Littleport (Figure 6.86). This relationship was also visible in the scatter of the adult sample. Overall scatter was diffuse (Figure 6.87), but differences between males and females were visible also, with male $\delta^{13}C$ data occupying a broader range of distribution than female data (Figure 6.88).

Direct comparison of survivor and non-survivor data at Littleport yielded further evidence for differences between subgroups in this population. Variability among non-survivors was higher than for survivors, as non-survivor $\delta^{15}N$ and $\delta^{13}C$ profiles plotted at the high and low extremes of the general population (Figures 6.89 and 6.90). When $\delta^{15}N$ data for individuals with skeletal stress markers (Figure 6.91) or cribra orbitalia (Figure 6.93) were examined, the earlier-observed pattern of males predominantly falling into the high range of the population values was intensified, with all profiles from males bearing stress markers and/or cribra orbitalia falling into the high end of the $\delta^{15}N$ range alongside non-survivors. Non-surviving children and males accounted for the highest $\delta^{15}N$ values among stressed individuals in the sample. Variability in $\delta^{13}C$ patterning was also much higher for males with skeletal stress markers (Figure 6.92) and/or cribra orbitalia (Figure 6.94), than for comparable females in the study. The range of $\delta^{13}C$ data defined by stressed males was also broadly lower in value than the range demarcated by stressed females. Non-surviving children with stress markers and/or cribra orbitalia also showed variable $\delta^{13}C$ profiles, with values decreasing in all cases over time and plotting among the lowest of the values in the population series. The similarity between surviving males and non-survivors was underlined by the scatter

pattern of dentine data (Figure 6.95), which showed both groups at the extremes of sample value ranges.

7.4.3 Intersite comparative data

Comparisons were also made for intersite isotopic patterning, highlighting several important similarities and contrasts between cohorts, based on survivorship and pathology.

7.4.3.i Non-survivors

Direct comparison of $\delta^{13}C$ and $\delta^{15}N$ data profiles for Edix Hill and Littleport non-survivors suggested some potential differences in the experience of early childhood at the two sites. The predominance of flat $\delta^{15}N$ profiles among non-surviving children at Edix Hill, and the total absence of this pattern at Littleport alongside greater profile variability was strongly indicative of differences in either diet or stress among these individuals (Figure 6.96). Profiles for $\delta^{13}C$ also showed greater variability among non-survivors at Littleport, relative to their counterparts at Edix Hill, who displayed a much flatter pattern of data (Figure 6.97).

The implications of a greater bias towards homeostatic patterning at Edix Hill are somewhat clarified by the lower overall prevalence of skeletal stress markers demonstrated, relative to Littleport. While all of the non-survivors at both sites belong to their respective cohorts due to differences in mortality risk relative to survivors at each site, it may be that overall levels of chronic stress and environmental risk – as previously suggested by skeletal stress markers – are also supported by isotopic patterning as being greater at Littleport than at Edix Hill. Thus, while Edix Hill non-survivors were demonstrably vulnerable to accident and acute illness, they may have faced lower risks of early death from chronic health issues than non-survivors at Littleport.

Inferring maternal health at Edix Hill and Littleport

Health inequalities between non-survivors at Littleport and Edix Hill may have begun well before birth, with roots in the late foetal period. In addition to the $\delta^{13}C$ and $\delta^{15}N$ data profiles examined for non-surviving individuals at both sites, the first increment of non-survivor dentine was plotted to evaluate differences in antenatal health for non-survivors (Figure 6.98). Cross-sectional examination of first-increment dentine data

delineated several trends in early life values, including parallels to potential physiological stress suggested by the longitudinal data.

Eight out of the fifteen individuals within the study sample fell within a broad clustering of values within a range between −20.4‰ and −19.8 for $\delta^{13}C$ and 11.7 and 13.4‰ for $\delta^{15}N$ values. Of these individuals, seven belonged to the Edix Hill sample, with only one (LP 4848) individual being from Littleport. Of the five individuals falling outside of this cluster, only two, EH 352 and EH 547B came from Edix Hill. While EH 352's first increment $\delta^{13}C$ value of −19.8‰ was within the range of the larger cluster formed by other first increment samples at Edix Hill, a $\delta^{15}N$ value of 14.9‰ placed this individual 1.5‰ above the top end of the antenatal $\delta^{15}N$ range produced by other Edix Hill samples. EH 547B had an identical $\delta^{13}C$ value of −19.8‰, alongside an even higher $\delta^{15}N$ value of 15.3‰. Factors such as heavier maternal dietary reliance on foods such as freshwater fish may have contributed in these cases to this relative elevation in values. Similarly, diet may have played a role in the first increment dentine values of LP 3770, who produced a $\delta^{15}N$ value of 11.7‰, falling just within the low end of the clustered range, alongside a markedly low first increment $\delta^{13}C$ value of −21.1‰. Both the uncharacteristically low first increment $\delta^{13}C$ value and the concurrent drop in $\delta^{15}N$ and rise in $\delta^{13}C$ observed between first and second increment values for this individual would support an interpretation of maternal metabolic stress. However, the overall low $\delta^{15}N$ and further decline after birth strongly imply that malnutrition may have occurred alongside a lower-trophic level maternal diet, differing from those of their stressed peers, in this case.

Four further individuals from the Littleport sample (LP 3311, LP 4116, LP 4144, LP 4494) produced first incremental dentine data with high $\delta^{15}N$ and low $\delta^{13}C$ values (range 14.4-14.9‰ $\delta^{15}N$ and −20.3 to −20.7‰ $\delta^{13}C$). This combination of overall lower $\delta^{13}C$ and higher $\delta^{15}N$ values, relative to those of their counterparts, may indicate maternal and/or foetal malnutrition during late pregnancy. It is intriguing that LP 3311, LP 4116 and LP 4144 all exhibited skeletal pathology suggestive of metabolic stress, alongside a pattern of divergent $\delta^{13}C$ and $\delta^{15}N$ shifts which continued after birth. When including LP 3770, five out of six antenatal dentine samples from Littleport may be interpreted as indicating metabolic *in utero* stress. This provides a marked contrast between the health of mother-infant pairs at the two sites. These isotopic data illuminate a pattern of disproportionately high levels of systemic stress in mothers and non-

surviving children during the late foetal period and infancy at Littleport, which aligns with existing data on skeletal markers of systemic stress.

The putative endemicity of *P. vivax* malaria in Fenland populations provides a biologically plausible mechanism for triggering such episodes of maternal and offspring stress. Pregnancy is associated with increased maternal susceptibility to all species of malaria, with the greatest risk of infection occurring during the second and third trimesters and the immediate postpartum period (Diagne *et al.*, 2000; Boel *et al.*, 2012). The mechanisms of this increased vulnerability during pregnancy are complex, ranging from greater attraction of the mosquito vector to inhibition of maternal immune response (Whitty *et al.,* 2005). Among the documented sequelae of *P. vivax* infection during pregnancy, maternal anaemia and changes to placental haemodynamics are known causes of low infant birthweight (Anstey *et al.*, 2009). In contrast to outcomes observed for *P. falciparum,* the degree of birthweight reduction in *P. vivax* infections is greater for multiparous mothers than in first-time mothers. This may relate to the relapsing nature of *P. vivax* disease and an increase in Type 1 proinflammatory cytokines in the placenta (Nosten *et al.*, 1999; Price *et al.*, 2007). Inhibition of placental transfer of nutrients to the developing foetus resulting from the increases in inflammatory activity may account for the trend towards high $\delta^{15}N$ and low $\delta^{13}C$ values observed for the late foetal period in Littleport samples. Due to the small sample size, and current lack of biomolecular confirmation for malarial presence in the early medieval Fens, this interpretation must be approached with due caution. However, the plausibility of this interpretive framework, alongside existing bioarchaeological and documentary evidence for *P. vivax* endemicity, reinforces the merit of exploring this as a future area of larger-scale investigations.

7.4.3.ii Survivors

Overall distribution patterns of survivor dentine data at the two sites mirrored the trend towards higher variability at Littleport observed in non-survivor data. $\delta^{15}N$ profiles (Figure 6.99) for Edix Hill occupied a lower range of values, with Littleport producing higher values and greater variability. Profiles for $\delta^{13}C$ were also dramatically different (Figure 6.100), with a tightly-constrained range of variability at Edix Hill alongside highly-variable, diffuse, and lower-value profiles at Littleport.

As an obvious advantage of sampling adult individuals of known sex is the ability to correlate sex or gender to palaeodietary patterns within a larger context, mean values were also examined for site cohorts. Among survivors, mean values for the earliest increment of M1 dentine showed a high level of similarity in the first months of life between females at Edix Hill and Littleport (Figure 6.101). Males in their first months of life differed from females at both sites. Males at Edix Hill had lower $\delta^{15}N$ than females alongside comparable $\delta^{13}C$, while males at Littleport had similar $\delta^{15}N$ values to the females coupled with noticeably lower $\delta^{13}C$ values. When mean values for total dentine were examined for all ages (Figure 6.102), approximating mean values for a bulk sampling approach, distributions were vastly different. High affinity emerged between three pairings: male and female survivors at Edix Hill, non-survivors at Edix Hill and surviving females at Littleport, and non-survivors and surviving males at Littleport. Individuals at Edix Hill, regardless of survivorship, exhibited very similar mean $\delta^{13}C$, while mean $\delta^{15}N$ for Edix Hill non-survivors more closely resembled mean values for groups at Littleport. Conversely, $\delta^{15}N$ for all groups at Littleport was broadly similar, but mean $\delta^{13}C$ values for non-survivors and surviving males was lower than for surviving females.

Differences in distribution of mean values for first-increment data and for total data for adults are indicative of changing inputs and pressures in these groups. The pattern of higher $\delta^{15}N$ and lower $\delta^{13}C$ observed for Littleport males, relative to Edix Hill males in both comparisons, is suggestive of disproportionately high sustained stress among males at Littleport. The interpretation of stress-related contributions to the mean is corroborated by the similarity in values between male survivors and non-survivors, a group expected to differ by levels of either frailty or stress exposure, at Littleport. Indeed, as the male survivor M1 mean reflects greater time-averaging than the non-survivor dm2 mean, there may be some underestimation of early-life stress for male survivors in this evaluation. Similarly, females at Littleport, bearing greatest similarity to non-survivors at Edix Hill, are suggestive of a group which is stressed, but to a lesser extent than their male and non-surviving counterparts at Littleport. Differences between groups at Edix Hill were generally smaller. The position of the male survivor mean at Edix Hill was the most constant between analyses, demonstrating lower levels of stress in data via consistently lower $\delta^{15}N$ and higher $\delta^{13}C$ than other groups in both comparisons. The affinities of Edix Hill females were more changeable over time.

Between the first-increment mean data and whole-dentine mean values, females shifted from early similarity to the Littleport female mean to greater similarity to their male counterparts at Edix Hill. These changes in affinity with other groups over time, together with higher overall variability in incremental profile expressed by Edix Hill females (discussed in section 7.4.1), is expressive of elevated levels of early-life stress which declined over time.

7.5 Synthesis of individual and group-level data

Group-level analysis of data reveals a potential spectrum of chronic stress among cohorts at the two sites, as interpreted through survivorship, isotopic variability, and covariant $\delta^{15}N$ and $\delta^{13}C$ patterning. As expressed in their stable isotope data, these cohorts range from lowest to highest levels of nutritional stress: Edix Hill males, Edix Hill females, Edix Hill non-survivors, Littleport females, Littleport males, and Littleport non-survivors. Individuals buried at Edix Hill, regardless of lifespan, appear to have generally enjoyed not only lower rates of skeletal pathology, but also greater isotopic homeostasis than individuals buried at Littleport.

In some cases, these differences in isotopic patterning between groups were present from the outset of data series, implicating differences in maternal or antenatal inputs which may be associated with poor health, particularly at Littleport. Antenatal data for non-survivors suggests health inequalities between the two sites beginning in foetal life, and some survivor data supports an interpretation of deepening postnatal disparities. While non-survivors, surviving males, and females at Edix Hill showed minimal evidence for chronic nutritional stress in their isotopic patterning, and little disparity from each other, at Littleport substantial and meaningful disparities in isotopic patterning were observed between groups. Surviving males at Littleport as a group maintained high $\delta^{15}N$ over time alongside non-systematically labile $\delta^{13}C$ values, which appear lower than those of their female peers, suggesting a systemic pattern of nutritional stress among males. This isotopic dynamic is mirrored by non-surviving children at Littleport, and especially those displaying skeletal stress markers. The Littleport surviving female pattern presents a stark contrast. Despite exhibiting high initial $\delta^{15}N$ values, and greater group variability in their early data than their male counterparts, females displayed declining values over time and reducing variability; expressive of decreasing group-level stress. What is the basis of this disparity? Antenatal data from Littleport implies that *in utero* stress was a common phenomenon,

although the sex of non-survivors in the small sample is not known. Where sex is known for survivors, females show improvement to health indicators throughout childhood at the group level, while the male group's isotopic patterning suggest a more persistent state of stress present from the outset of data. The parallel between non-surviving children, who are defined by differential frailty, and surviving males at Littleport, makes it likely that a sex-based bias to health was in effect at Littleport.

Consideration should be given to whether social, rather than biological, factors such exogamous mobility among females buried at Littleport (i.e. birth origin outside of the Fens), may account for similarity in first-increment values between Littleport and Edix Hill females. However, such exogamous mobility, described by Sayer (2014) as potentially accounting for bias in the distribution of child burials, does not necessarily apply to small cemeteries such as Littleport. Additionally, this interpretation does not adequately explain the pattern of data. While females buried at Littleport probably did in many cases originate outside of Littleport, mean female values are most like bone values at Ely, which would have shared a similar environment. Finally, there are no differences between the distribution of cribra orbitalia or opposing covariance among the sexes at Littleport, indicating a qualitative difference in disease exposure or dietary sufficiency. Evidence for experiences of early life stress is thus equivocal on the variable of sex, making biological response a more straightforward explanation for differences in patterning than differential exposure or care.

Sex differentials in morbidity and mortality, to the detriment of males, are known to begin during foetal life and to continue throughout the lifespan (Drevenstedt *et al.*, 2008; Muenchhoff and Goulder, 2014; Zarulli *et al.*, 2018). These biological differences in frailty emerge for a combination of reasons (discussed previously in Chapter 3): primarily, these are greater disease resistance conferred to females via X chromosome-inactivation, and also the mediation of the immune response by sex steroids (Muehlenbein and Bribiescas, 2005; Fish, 2008; Klein and Flanagan, 2016). Among the elevated risks to males beginning in early life are intrauterine death and spontaneous early abortion, premature delivery, stillbirth, and foetal distress caused by labour complications (Bekedam *et al.*, 2002; Ingemarsson, 2003; Di Renzo *et al.*, 2007). Beyond the perilous periods of gestation and birth, males also fare worse than their female peers, being less resilient to complications of low birthweight, infectious disease

morbidity and mortality, and undernutrition and impaired growth during childhood (Wamani *et al.*, 2007; Muenchhoff and Goulder, 2014; Simchen *et al.*, 2014).

In terms of the interaction between sex differentials in frailty and the putative endemicity of *P. vivax* in the Fens during the early medieval period, it is difficult to make definitive claims regarding a known dynamic. As previously discussed in Chapter 4, the epidemiology of *P. vivax* has received a low priority in malaria research, with the majority of resources directed to addressing *P. falciparum*. Due to this neglect, evidence regarding sex differentials is sparse. Nonetheless, the limited existing evidence is suggestive. Data from murine models has shown that male mice and female mice treated with testosterone suffer higher mortality and more severe clinical disease from *Plasmodium* infection, while castrated males demonstrate greater resistance (Bernin and Lotter, 2014). Human host-parasite dynamics are known to differ from experimental animal models, and from each other based on species behaviour. Yet, this data provides intriguing, if inconclusive, evidence for the relationship between sex hormones and host defence against *Plasmodium* parasites. In regard to *P. vivax* infection specifically, an epidemiological study from the demilitarised zone in the Republic of Korea found the highest rates among adult males, with the authors suggesting that this was due to differences in vector exposure (Moon and Cho, 2001). However, this incidence data accords with a male sex bias for *P. vivax* among both children and adults found in Indian populations (Pathak *et al.*, 2012; Savargaonkar *et al.*, 2015; Chery *et al.*, 2016). Furthermore, a longitudinal analysis of Honduran *P. vivax*-infected individuals and healthy age-matched controls by Muehlenbein *et al.* (2005) found that for males infected with *P. vivax*, parasitaemia was significantly and positively correlated with serum testosterone levels. The study additionally found that serum testosterone levels were depressed, and cortisol levels elevated, on the day of diagnosis, relative to samples taken approximately eight days following; a systemic response to reduce androgenic immunosuppression and redirect energetic resources from anabolic activity to host defence (Muehlenbein *et al.*, 2005).

The relationship suggested by these studies between sex hormones and *P. vivax* susceptibility also has implications for differential childhood growth and metabolic activity between the sexes. In addition to IUGR, *P. vivax* infection in childhood is widely acknowledged to compromise nutritional status, leading to chronic underweight status and impaired growth (Holding and Kitsao-Wekulo, 2004; Poespoprodjo *et al.*,

2009; Lee *et al.*, 2012; Monteiro *et al.*, 2016). In a study of over 1500 children from Vanuatu, Williams *et al.* (1997) found that *P. vivax* infection was a strong predictor of acute malnutrition and wasting, and that there was a significant association between stunting, male sex, low birth weight, and *P. vivax* infection. A direct connection was made between the stimulating effect of *P. vivax* on secretion of the cytokine TNF, resulting in anorexia and cachexia. However, the potential relationship between male susceptibility to infection was not further addressed. Similarly, documentary evidence for 19th century American Civil War recruits found that males recruited for the Union Army who had spent their childhood in *vivax* malaria-endemic counties were on average shorter, and more susceptible to infection, than recruits from non-malarial regions of the North (Hong, 2007). It is plausible that androgenic effects on immunity predispose males to higher parasitaemia, and that in children the metabolic effects of TNF place boys at higher risk of growth impairment through nutritional stress. Based on a combination of clinical, documentary, and isotopic evidence, it seems reasonable to interpret the persistent low and variable carbon and high nitrogen value profile patterning among males at Littleport, especially those with cribra orbitalia, as potential confirmation of a sex-based differential in physiological response to malarial infection. While it is possible that another type of infectious disease differentially-affected males at Littleport, or alternately, that they were disproportionately affected by nutritional shortfalls, the hypothesised presence of endemic *P. vivax* at Littleport provides the closest fit with existing isotopic, skeletal, and documentary evidence.

By contrast, at Edix Hill sex-based differences were not substantial, and where differences in profile variability were observable, as with first-increment values, heightened variability affected females, not males. This should, perhaps, cause some consternation regarding the idea of the universal applicability of a theory of differential male frailty. However, a review of infant mortality in 15 developed countries from 1751-2004 by Drevenstedt *et al.* (2008) found that the established biological disadvantage of males was highly dependent on environmental context, and that in low-risk environments the gap between males and females is likely to be less. It is probable that if Edix Hill indeed presented a less heavy epidemiological burden to young children than Littleport, then biological differences between the sexes may have been minimised. The similarity between survivor and non-survivor profiles at Edix Hill also lends weight to the argument that selective pressures were less severe than those in operation at

Littleport during the same period. It is therefore also less clear whether the early postnatal differences between males and females at Edix Hill expressed in the first-increment data stem from issues of biological sex or are attributable to gendered issues of care.

7.5.1 Interpreting individual and group variability

The opening sentence of Leo Tolstoy's classic novel Anna Karenina states: "all happy families are alike; each unhappy family is unhappy in its own way," (Tolstoy, 2003: 1). This assertion forms the basis of the "Anna Karenina principle," which theorises that the parameters of success necessarily define a narrow and homogenous range; whereas maladaptation and failure may be more heterogeneous, each failing in unique ways. This principle is widely applied across disciplines, and has, for example, been utilised in microbiology to explain the greater microbial diversity in dysbiotic animals as a response to stress (Zaneveld *et al.*, 2017). It also provides a coherent interpretive framework for understanding the isotopic variability among differing cohorts at Littleport and Edix Hill, as a function of differing exposure and physiological responses to stress.

The Anna Karenina principle is particularly underscored in the case of non-survivorship. In this study, non-survivors present the greatest group-level isotopic heterogeneity at their respective sites. Moreover, the greater isotopic heterogeneity of individuals at Littleport than at Edix Hill provides support for the claim that greater prevalence of skeletal stress markers at Littleport represents higher levels of metabolic and nutritional stress at that site. Survivors, to a lesser extent, also are consistent with this principle. Survivorship is not a zero-sum game: in regard to stress, the victory of survival may in some cases be pyrrhic. In other words, among survivors, heterogeneity too may be expressive of differential levels of stress exposure. Heterogeneity among survivors is much greater at Littleport than at Edix Hill. Further, as should be expected in keeping with the principle of male immunological frailty, male survivors displayed a more consistent state of heterogeneity throughout childhood than did their female peers, who became progressively more homogeneous over time. These patterns of heterogeneity at Littleport are inconsistent with a dietary source of variability and are more parsimoniously explained as being a function of biological sex, rather than gendered diet or care. Care may nonetheless have been a factor in the low level of

variability during early life at Edix Hill, which presented more generally biologically-robust surviving female children as the more variable sex.

7.5.2 A biocultural model for understanding isotopic variability

While the data discussed in Section 7.4 suggest that biological factors must be considered as a key influencer of data, it is essential to consider both physiological and dietary contributors within a holistic interpretive model. The degree to which isotopic variability in this study, and other recent studies, is not reducible to changes in diet should call into question the confidence with which researchers routinely attribute anomalous values to dietary causes alone. While equifinality has long been acknowledged to hamper interpretation of isotopic data (e.g. Lee-Thorp, 2008; Bogaard and Outram, 2013; Warinner *et al.*, 2013), the primary concern has consistently been to reconcile varying dietary contributors, and to reconstruct synchronic foodwebs. Thus, the role of fluctuating physiological states, such as disease processes, in contributing further to equifinality in archaeological samples, has only recently begun to be addressed via diachronic methods such as incremental dentine analysis (cf. Beaumont *et al.*, 2015; Beaumont and Montgomery, 2016; King *et al.*, 2018a). Due to the novelty of diachronic methods, the extent and causes of intra-individual isotopic variability has not yet been fully explored or understood.

Observed differences in pattern within the current study, representing a mix of dietary inputs and physiological function in varying permutations, may lend some clarity to understanding the general processes which lead to isotopic variability. The size of this dataset, along with the supporting demographic and palaeopathological data, allows for the formation of hypotheses regarding the physiological states underlying observed patterns. Table 7.2 provides an overview of isotopic profile patterns observed in this study, hypothesised causes and associated metabolic states. Evidence for the causes of patterns of parallel covariance and opposing covariance is relatively robust. Studies of modern humans analysing hair have firmly established that parallel changes in $\delta^{15}N$ and $\delta^{13}C$ profile correlate with changes in the isotopic composition of diet under conditions of healthy metabolic function (O' Connell and Hedges, 1999a; Mc Cullagh *et al.*, 2005; Fuller *et al.*, 2006). Similarly, studies of modern humans and animals have tied opposing covariance to catabolic (Hobson *et al.*, 1993; Fuller *et al.*, 2005; Mekota *et al.*, 2006; Warinner and Tuross, 2010) or anabolic (Fuller *et al.*, 2004; Mekota *et al.*, 2006) states associated with undernutrition, disease, pregnancy, or growth. However,

interpretation of changing or static dietary patterns may be complicated by the frequent co-occurrence of changing metabolic state (and vice versa). Thus, in many cases where catabolic or anabolic patterning predominates, it may be difficult to discern the respective proportional contributions of diet and physiology to isotopic values, and within the vulnerable period of early childhood, isotopic breastfeeding and weaning patterns may be entirely obscured.

Profile Pattern	Metabolic State	Cause
Flat $\delta^{13}C$ and $\delta^{15}N$	Homeostasis	Stability of diet + low stress
Flat $\delta^{13}C$, increasing $\delta^{15}N$	Mixed state	Trophic level increase + catabolism
Flat $\delta^{13}C$, decreasing $\delta^{15}N$	Mixed state	Trophic level decrease + anabolism
Parallel covariance of $\delta^{13}C$ and $\delta^{15}N$	Increasing/decreasing trophic level	Breastfeeding/weaning or other change to diet
Increasing $\delta^{13}C$, flat $\delta^{15}N$	Mixed state	Trophic level increase + anabolism
Decreasing $\delta^{13}C$, flat $\delta^{15}N$	Mixed state	Trophic level decrease + catabolism
Decreasing $\delta^{13}C$, increasing $\delta^{15}N$	Catabolism	Body mass loss from undernutrition, injury, or disease
Increasing $\delta^{13}C$, decreasing $\delta^{15}N$	Anabolism	Body mass increase/growth

Table 7.2 Isotopic profile patterns appearing in the study and their proposed interpretive significance

Flat profiles present more of a challenge to interpretation, with hard evidence for the causes being scant. Beaumont (2013) observed in her study of survivors and non-survivors of the Great Irish Famine that all incremental dentine $\delta^{15}N$ profiles for survivors displayed a flat pattern, while non-survivors exhibited a mixture of curved and flat profiles. As it is counterintuitive, understanding the protective immunological and nutritional advantages of breastfeeding, to interpret these patterns as suggesting that survivors were less likely to be breastfed than those who died early in life, Beaumont reasoned that observed flat patterning among survivors is likely to indicate healthy homeostasis, rather than a lack of breastfeeding. Non-survivors with flat profiles were thus interpreted as being otherwise healthy children who died from accidental or acute infectious causes, while curves among non-surviving children were attributed to longer-term patterns of chronic stress. Consequently, expected trophic level effects in $\delta^{15}N$ profile patterning as a straightforward reflection of breastfeeding status were brought

into question, and this was echoed in more recent published work by Beaumont *et al.* (2015).

Though controversial within archaeology, there is ample precedent for the expression of doubts regarding the trophic level paradigm of breastfeeding in both archaeological and ecological research (e.g. Jenkins *et al.*, 2001; Hedges and Reynard, 2007; Martínez del Rio *et al.*, 2009; Reitsema, 2013). These concerns have also become magnified in recent years by increasing evidence that the mother-offspring offset in human tissues (discussed previously in Chapter 2), may not be as systematic, or our understanding of mechanics as complete, as previously assumed (Romek *et al.*, 2013; Tea *et al.*, 2013; Reynard and Tuross, 2014; Herrscher *et al.*, 2017). Considering the ambiguity of evidence for a consistent trophic-level shift between maternal and offspring tissues, interpreting flat profiles in both Beaumont's and the present study's data as being indicative of homeostasis, rather than the absence of breastfeeding, seems reasonable.

However, this interpretation must be constrained by caveats and a critical application of interpretation to context. While the presence of flat patterning in *both* carbon and nitrogen isotope profile data does suggest a state of healthy homeostasis, this cannot be held to be the case when only one profile is displaying a flat trend over time. A single flat isotopic profile, accompanying a more labile and changeable trend in the other isotope, was relatively common in this study, particularly at Edix Hill. This phenomenon presents greater challenges to understanding than the more straightforward patterns of parallel covariance, flat homeostasis, or opposing covariance. Interpretation of the metabolic state underlying the co-occurrence of flat and variable profiles is specific to the pattern of co-occurrence; the probable causes of flat carbon isotope profiles accompanied by variable nitrogen profiles vary based on whether $\delta^{15}N$ is rising or falling and differ again from the causes of static $\delta^{15}N$ values observed alongside rising or falling $\delta^{13}C$ values. Co-occurrence of flat and variant isotopic patterning is likely to arise from a mixed metabolic state, where a change in the trophic level of diet coincides with the onset of anabolic or catabolic activity, to either negate or enhance the directionality of a value shift. Flat profiles may thus result from mixed metabolic effects created by dietary and physiological factors, and it is likely that these interact synergistically.

Regarding the interpretative morass created by the problem of equifinality in archaeological isotope biochemistry, a troubling question may arise: can we confidently

infer past breastfeeding patterns from carbon and nitrogen stable isotope data, as was once assumed? The answer to this question, as to most others concerning levels of uncertainty in archaeological research, is currently unclear, and will remain so, until the parameters of isotopic change in modern populations are studied in more depth, with larger sample sizes, and more attention to the causes of variability. Parallel covariance in a pattern resembling the classic weaning curve is almost certainly indicative of dietary change, and it is possible that this does represent a correlation with breastfeeding behaviours. However, this is far from certain, since it is highly probable that archaeological individuals with parallel flat profiles were also breastfed, and it may be that these decreases in value are reflective of changes to the mother's diet, rather than the child's, during the weaning process, among other possibilities. Thus, we may attempt to interpret breastfeeding behaviours from the co-occurrence of declining $\delta^{15}N$ and $\delta^{13}C$ values, but certain caveats regarding uncertainty must apply. Similarly, in cases where the interpretive complications of opposing covariance, parallel or mixed flat profiles predominate, it cannot be inferred that breastfeeding and weaning did *not* occur during the timescales covered by the data series. It is possible only to conclude that a range of inputs resulting from health, environment, and diet are reflected together in such a way to make discernment of dietary shifts alone difficult. However, a difficulty is not an impossibility. With care towards contextualisation and caution in the interpretative process, together with extensive further observational studies with modern populations, it is to be hoped that future research will be able to more confidently answer questions regarding the mechanics of fractionation within lactation and infant feeding.

7.6 Summary of findings and conclusions

Despite previous caveats regarding the interpretation of patterning, and the expression of concern around the need for due caution when attempting to negotiate the known while avoiding the pitfalls of what Reynard and Tuross (2014) described as the "unknown and the unknowable" of weaning studies, the present study has attempted to view its data in light of its historical and environmental context. In presenting new evidence, interpreted with necessary qualification, it is hoped that some improvements will be made to our current state of knowledge about the health and diet of children in eastern England during the early medieval period.

In terms of what may be inferred regarding breastfeeding practices in the period, if we interpret aggregate data from Edix Hill and Littleport according to the traditional weaning model, the pattern of decline in values is consistent with previous estimations that solid foods were introduced by the second half of the first year of life, and cessation of breastfeeding occurred for nearly all children by two to three years of age. Where conventional dietary patterning is found, the onset of declines in value are universal around the midpoint of the first year. Equally, variability was minimal by two to three years, and nearly all profiles had assumed a flat appearance by that point. As Crawford (1999) has pointed out, the degree of parental investment represented by several years of mother-child proximity and responsiveness required for sustained breastfeeding is suggestive of children being highly valued in early Anglo-Saxon society within eastern England. Introduction of solid foods around the midpoint of the first year, and continued breastfeeding for two to three years is congruent with Soranic and Galenic recommendations for infant feeding, which have been assumed to still be in usage during the medieval period. The cessation of breastfeeding occurring by three years of age also aligns favourably with broad recognition of changes to the Anglo-Saxon burial rite for children at around that age, which are thought to indicate the achievement of a socially-significant age threshold (Härke, 1992; Lucy, 1994; Crawford, 1999; Crawford, 2000; Stoodley, 2000). No differences in apparent weaning age are perceptible between survivors and non-survivors at either site, suggesting a lack of systematic differentials in care which might have been decisive regarding survivorship. Neither are there any perceptible sex-based differences or variations based on funerary rite in the milestone ages of the weaning process, suggestive of socially-mediated differences in the care of infants.

What the study may provide of an innovative nature is not the mere confirmation of previous expectations for average breastfeeding duration, but, rather, a spotlight on the extent of variability in isotopic patterning which may occur in archaeological populations. While not apparent through cross-sectional sampling methods, this variability is likely present in most past populations, and correlates with other meaningful markers of multidimensional difference, such as the intersections of biological sex and gender, or geographical environment and disease risk. These dimensions of difference, and the ambiguity of their intersections do create some difficulties to interpretation. For instance, though the cessation of breastfeeding appears

to have occurred at a maximum of 2-3 years of age at both sites, there are slight indications that individuals at Edix Hill may generally fall on the earlier end of this watershed, while individuals at Littleport are on the later end of this limit. This may imply a longer weaning process at Littleport, as a marker of true differences in parental investment in response to differential disease risk. However, a slight lag in the diminishment of elevated $\delta^{15}N$ values at Littleport may not be due to a longer weaning process, but to extended persistence of early-life stress in childhood. Differentiating between persistently elevated $\delta^{15}N$ values caused by systemic stressors and those caused by continued breastfeeding is likely to prove challenging until better understanding of the parameters and thresholds of each is better understood, particularly since mothers are likely to extend the breastfeeding of sickly children, relative to their healthy siblings. Differences in overall variability of values and the range of estimated weaning age also suggest differences in health. One individual at Littleport appears to have completed the weaning process at the premature age of four months, while other individuals fall at the other extreme, closer to three years. If accurate, the differences in the age at which weaning was completed are potentially indicative of health and readiness to develop independence from the breast. Individuals at Edix Hill vary much less in relative terms. The variability within sites demonstrated by this study has shown that intra-site variability may correlate with environmental hazards such as disease processes, and these indirectly imply a potential demand for differences in care.

7.6.1 Disease processes and isotopic variability

Little is positively known regarding alterations to isotopic profile caused by disease processes which alter metabolic function in archaeological populations. Most of our current state of knowledge is theoretical and inferred from knowledge of pathophysiological effects on body mass, such as the wasting effects of cachexia observed in cancers or tuberculosis (observed in one individual in the present study). Certain knowledge of disease prevalence in past populations is also rare, unless the diseases in question produce pathognomonic lesions of bone. In the case of temperate *P. vivax* malaria, modern populations provide little context with which to better understand isotopic pathophysiology in past populations, as antimalarial chemotherapies are standard and withholding treatment is highly unethical.

The unusual nature of historical Fenland malaria thus presents a unique opportunity to the researcher. As it is unlikely for there to have been any strain present in eastern

England other than the historically-documented *P. vivax*, fewer difficulties of interpretation are present than would be the case in areas with a more complex epidemiological landscape. In general, the Fens are a greatly understudied culture area in bioarchaeological terms. However, the study by Gowland and Western (2012) provided a groundbreaking contribution to the conundrum of endemic Fenland malaria, and its arrival in Britain. The isotopic patterning in the present study, consistent with relapsing malarial episodes and successive catabolic-anabolic cycles, corroborates the previous palaeopathological evidence presented by Gowland and Western for the presence of endemic malaria in the Fens during the early medieval period. It is perhaps overstating matters to describe the present dataset as offering a "smoking gun" for the presence of medieval malaria, as neither *P. vivax* nor its metabolites have been confirmed as being present in these human remains. Furthermore, observational data does not exist to conclusively establish the effects of *P. vivax* on carbon or nitrogen balance in humans. However, in presenting isotopic patterning consistent with the known metabolic effects of *P. vivax* infection during pregnancy and early childhood, the present study may be nonetheless providing the first evidence for the dynamics of endemic malaria in an archaeological population.

The study additionally provides some food for thought regarding characteristic isotopic variability, and the level of variability which should be expected for differing cohorts. The majority of palaeodietary studies have not found sex-based differences in values demonstrative of dietary differences in adults. With respect to children, we should perhaps expect there to be differences not only in diet, but in growth and immune function to be reflected in differences in isotopic patterning. These differences may not be apparent in all settings but should be considered as a possible alternative to a default dietary explanatory model in cases where challenges to immunity are expected to be elevated, as in the present study. In such an environment as this, breastfeeding should be viewed as an essential adaptive activity to protect the health and nutritional status of young children. Consequently, interpretation must integrate a biobehavioural model to understanding differences in patterning, where parity in care, but not physiological responses, are suggested. The differences in $\delta^{13}C$ and $\delta^{15}N$ values and patterning in males and females have suggested a biological sex-differential during childhood at Littleport, rather than gendered differences of diet or care, as these differentials are demonstrated in prenatal and postnatal life equally and align with what

is known about the metabolic demands of immune function. Thus, when studying vulnerable periods of life, future isotopic studies which address environments of high risks to health should engage with the possibility of health differentials in a way that applies known clinical and epidemiological evidence.

8. Conclusion

8.1 Introduction

This chapter reviews the findings discussed in Chapter 7 in light of the original aims and research questions. It will also discuss the impact of these results on the broader discipline as well as addressing inherent limitations in the work. Finally, the chapter will suggest future research avenues which could improve our understanding of biogeochemical dynamics, past infant feeding practices, and Fenland palaeoepidemiology.

8.2 Review of purpose and findings

The primary aim of the study was to establish whether potential differences in health, suggested by skeletal markers of stress between the communities at Edix Hill and Littleport were also reflected in differences in breastfeeding pattern and duration. Identification of carbon and nitrogen isotopic patterning consistent with, or suggestive of, malarial infection during childhood formed a secondary aim. To meet these aims, the study analysed carbon and nitrogen stable isotope ratios in incrementally-sampled dentine from a mixed sample of survivors and non-survivors of childhood at the two sites, comprising a total of 59 individuals. Based on the principle of maternal responsiveness and delayed weaning where risks to infant health are greater, it was hypothesised that cessation of breastfeeding would occur later and variability in isotopic pattern would be higher at Littleport.

The study produced longitudinal data patterns which were broadly suggestive of breastfeeding from birth, followed by a complementary feeding period which began for most children at around six months of age, and was completed by three years. Data patterning was indicative of this process being completed more commonly and uniformly around the age of two at Edix Hill, while cessation of breastfeeding was more variable and carried a higher upper age limit of three at Littleport. In terms of mean values, differences were also seen. Edix Hill showed similarity in adult bone values to those of previous research (Mays and Beavan, 2012) and childhood dentine values (from the present study) to contemporary Anglo-Saxon sites elsewhere; albeit with higher mean $\delta^{15}N$ and lower $\delta^{13}C$ values in the dentine, likely to be expressive of recorded stress. For Littleport, this trend was mirrored, with mean $\delta^{15}N$ values which were similar to local comparative bone values for Ely (Lucy *et al.*, 2009), but with

lower mean $\delta^{13}C$ values. Furthermore, distinct differences in variability were evident at the individual level. Individuals in the Littleport sample more commonly exhibited non-dietary patterns of opposing covariance consistent with cycles of stress and recovery. This patterning was significantly correlated with burial at Littleport in the sample, as burial at Littleport was in turn significantly associated with prevalence of cribra orbitalia, previously associated with a malarial environment by Gowland and Western (2012). These isotopic patterns were not similarly demonstrated at Edix Hill, which had dramatically lower prevalence of skeletal stress markers more in keeping with estimated mean prevalence for early medieval Britain generally.

Differences between subgroups at the sites were also identified. Few differences in isotopic pattern and value were observed between surviving males, females, and non-surviving children at Edix Hill, aside from mildly increased variability among non-survivors. These findings for Edix Hill were interpreted as representing overall lower stress exposure levels for children at that site than at Littleport. Littleport isotopic data indicated more contrasts between subgroups, with surviving males and non-survivors expressing higher mean $\delta^{15}N$ values, lower $\delta^{13}C$, and greater variability than surviving females. Similarities between non-survivors, a group defined by heightened frailty, and surviving male children were read as expressing greater vulnerability to infection in both groups, aligning with clinical evidence for a male immune disadvantage. Together with the palaeopathological-spatial evidence for a link between cribra orbitalia and malaria, and the observed association between cribra orbitalia and opposing covariance, increased variability and non-dietary isotopic patterning was demonstrative of real differences in health between the two sites.

8.3 Contribution to research

The results of the present study in part support the conclusions of previous research, while offering an interpretation integrating additional physiological factors not explicitly addressed in previous work. Very little biogeochemical research on the medieval Fenland diet has been undertaken. Previous carbon and nitrogen stable isotope studies of Anglo-Saxon diet analysing bone have shown that diet varied little across space and environment within Britain, finding a heavy reliance on terrestrial proteins and an agricultural lifestyle (Mays and Beavan, 2012). Edix Hill, acting as a normative standard for the period, mirrors the trends previously observed for Anglo-Saxon Britain. Work from early medieval Ely (Lucy *et al.*, 2009) exhibited overall mean $\delta^{15}N$ values

which were higher than at other early medieval sites, while $\delta^{13}C$ values remained broadly comparable to other Anglo-Saxon sites. Freshwater fish consumption – entirely plausible for a Fen island –was suggested to explain elevated $\delta^{15}N$. The present study, with its similarly elevated $\delta^{15}N$ values in conjunction with lower $\delta^{13}C$ and frequent appearance of opposing covariance patterning, supports the idea of an important piscine contribution to dietary protein, while requiring considerations of pathophysiology to its interpretive model. The incremental and diachronic nature of the data illuminated elements of metabolic function and shifts of magnitude which are not adequately explained by diet alone, as in previous studies of bone. In integrating explanatory considerations such as the known malarial history of the Fens, this study is the first to explicitly identify isotopic patterning consistent with intermittent stress characteristic of chronic malarial infection in childhood. Thus, while foods drawn from the Fen were undoubtedly important, the study highlights the need to address more than dietary inputs in the analysis of palaeodietary data.

Superficially, this study does not present notable or controversial findings in terms of its assessment of breastfeeding duration during the early medieval period. Findings that breastfeeding was likely exclusive until the middle of the first year of life, and that the weaning process was gradual, with a complementary feeding period completed by two to three years of age, are in keeping with expectations based on multiple types of pre-existing evidence. Among these are the recommendations of Classical physicians (still thought to be in use during the medieval period), age-related differences in burial rite, and estimations made by previous palaeodietary studies of Anglo-Saxon children (e.g. Macpherson, 2005; Haydock *et al.*, 2013). However, the present study has identified high levels of variability within these parameters: some individuals appear to wean unexpectedly early, some later than most, and many do not exhibit patterning consistent with the expected trajectory for breastfeeding at all. Consequently, the data make a contribution in terms of underlining the complexity of breastfeeding behaviours and affirming the finding of previous studies that question the adequacy of present knowledge and cross-sectional methodologies to address these frequently-encountered variants from the imagined norm.

8.4 Research limitations

Despite the contribution made by the present study to increasing understanding of isotopic variability in early medieval Britain, and in raising necessary questions about

the validity of current palaeodietary assumptions, significant caveats must be made about the limitations of the data. While the amount of data collected in the study was substantial, due to the incremental sampling method, the number of individuals sampled is relatively small by the standard of cross-sectional studies. This is particularly true of the very small number of non-adult individuals sampled at each site, and this limits the confidence with which this data may be assumed to be representative of non-surviving children in these places at that time. This study prioritised sampling of adult survivors, which were more abundant due to Anglo-Saxon burial practices, and also more likely to represent normative childhood experience. Consequently, differences between survivors and non-survivors of childhood must be approached with some caution, until greater numbers of children are analysed.

Similarly, the scope of the study was narrow in terms of the numbers of sites studied. While both sites were chosen because they typified wider trends in skeletal health for their period and sub-region, each cemetery site should not be assumed to represent more than the living community it served (and that only with due deference to the osteological paradox). In terms of imparting information regarding wider regional trends, these results should therefore be viewed as preliminary. This is particularly the case for Littleport, as the Fens are greatly understudied in terms of addressing their specific health concerns using biochemical and biogeochemical methodologies. It is currently unclear whether health was variable geographically within the Fens; whether culture and status or environment played the largest role, and whether conditions were similar throughout the Fen basin, or varied significantly between silt and peat areas. Despite the fact that Littleport presents a clear architype of what might reasonably be expected for the Fens during the pre-drainage period, more sites should be studied in order to determine whether health at Littleport was typical for the time and place or embodies an extreme for this environment.

Within this vein, a further substantial limitation of this study is the volume of unknown factors, aside from dietary shifts, which may alter isotope values. As alteration to isotopic values is potentially multifactorial, these must be viewed as non-specific signs to some extent. This is particularly the case in regard to the putative presence of malaria in the early medieval Fens. While such an interpretation has biological and historical plausibility, without protocols which can positively identify malarial infection from skeletal tissues and align evidence of infection with anomalous isotopic patterning,

this remains speculative. Exact dating is not possible for the individuals sampled from these cemeteries either, and environmental conditions or cultural practices may change dramatically within the space of a few decades. As with nearly all bioarchaeological research, there are far more unknowns than knowns, necessitating inference rather than objective certainty.

8.5 Recommendations for future research

Future studies should attempt to ameliorate and reduce the level of uncertainties regarding the study of past diet and health. Fenland bioarchaeology, while not the main target of much of this research, would stand to greatly benefit from expansion of research in several key areas.

Firstly, an increase in studies using incremental sampling methods for isotopic research in modern and archaeological populations is necessary. In particular, there is a need for observational studies in modern contexts which include enough participants to grapple with the difficulties of inter-individual variability and allow for correlation of dietary and physiological changes with concurrent shifts in carbon and nitrogen stable isotope values. Past observational studies of weaning in modern populations have suffered from the limitations of small sample sizes and restricted duration, which does not reflect expected behaviours for past populations (cf. Fuller *et al.*, 2006; Herrscher *et al.*, 2017). Studies incorporating larger numbers of individuals, and a wider variety of breastfeeding durations, including individuals breastfeeding well beyond the first year of life, would contribute significantly to understanding of the biogeochemical dynamics involved. In addition to observational studies specifically geared to characterising isotopic shifts associated with infant feeding and growth patterns, studies which document and identify shifts in pattern associated with pathological conditions (such as chronic malarial infection) would be valuable. While in many cases, longitudinal study of untreated disease would provoke ethical issues, analyses of hair of significant length would allow suitable patients to be enrolled in study upon diagnosis, documenting the isotopic impacts of untreated infection, treatment and recovery.

Secondly, an increase in application of incremental methods to archaeological populations would be greatly advantageous. It is clear that biogeochemical processes, and their reflection in skeletal tissues, are far more complex than was previously assumed. Furthermore, these processes may not be equally reflected in all tissues. In light of this, more incremental analyses which produce a dynamic picture of diet and

health in past populations are essential to delineating the full range of challenges to health. Particularly important for the Fens, further engagement with incremental sampling of teeth from both humans and animals would help to identify differences in isotopic value and patterning which might relate to differentials in resource access or environmental heterogeneity. Better spatial and temporal coverage of the region would help to characterise the impacts of meaningful geographic boundaries, such as the differing ecologies of brackish silt or freshwater peat fen, and historical watershed events, such as drainage, on the dietary habits and health of Fenlanders.

Several other emerging advances in archaeological science present opportunities to augment our current understanding of isotopic dynamics by revealing evidence of skeletally invisible biological histories. New methods to conclusively identify malarial infection in archaeological remains are particularly needed. Due to low and fluctuating parasitaemia in chronic *P. vivax* infection, use of ancient DNA methods present a significant risk of underestimating the number of affected individuals. Accordingly, methods which detect lifetime malarial infection, rather than active clinical disease at the time of death, would provide a better measure of disease ecology and impacts. Hemozoin currently presents the most robust target for current biochemical methodology in skeletal materials. This insoluble biocrystal is produced by malarial parasites of all species as a metabolic by-product of haemoglobin consumption, and is not fully cleared by the body, remaining in red bone marrow. The substantial levels of hemozoin persistence in bone over time observed in mouse models (Frita *et al.*, 2012; Lee *et al.*, 2017) suggest that it probably accumulates over time in repetitive or relapsing infection, offering a reliable analytical target even in low-parasitaemia species such as *P. vivax*. Hemozoin is pathognomonic to both malaria and schistosomiasis. However, as the latter does not occur in temperate climates, within Britain the presence of hemozoin may be considered exclusively indicative of malaria, assuming that travel to tropical locations has not occurred. The ability to align anomalous isotopic patterning in wetland contexts with definitive identification of malarial infection would help us to better understand the interplay between environment, culture, and biology occurring in the isotopic landscapes of the human body.

The invisibility of biological sex in immature individuals is another challenge to isotopic interpretation which may be ameliorated in future research. A recent study by Stewart *et al.* (2017) analysed peptides of amelogenin, an enamel protein which has sex-

specific differences in amino acid sequence, finding a very high degree of accuracy for sex estimation in a range of archaeological individuals from varying time periods. Applying this method to reliably correlate biological sex with isotopic data in non-surviving children would allow for questions about the intersection of sex and differential frailty to be addressed. Sex ratios in infant and child mortality in past populations are an extremely important reflector of care practices and biological variability in modern populations. These should be prioritised as an equally important dimension to explore in past populations, which are currently inaccessible to bioarchaeologists. The use of amelogenin analyses in Fenland populations would also help to identify whether the sex bias for stress observed for Littleport was paralleled by a sex bias in the ratio of males represented among non-survivors as well.

The development of other methods for assessing health across the lifecourse should also form an important research strategy with particular relevance to long-term health in Fenland populations. Alignment of childhood biogeochemical data in individuals who survive to adulthood with other biochemical or osteological markers of health and disease would contribute to our understanding of long-term sequelae to early-life diet and health conditions. Currently, no methods exist to detect soft-tissue and metabolic disorders, such as cardiovascular disease or diabetes mellitus. Osteological methods to predict obesity or high body mass, such as measures of cross-sectional geometry in weight-bearing long bone, have also been shown to be unreliable in adults (Pomeroy *et al.*, 2018). However, the development of new methods to assess these important markers of adult health would allow testing of the DOHaD hypothesis in past populations, and specifically enrich our understanding of the impacts of chronic childhood infection on the health of surviving Fenlanders in later life.

Finally, it is important to emphasise the opportunities afforded by these potential future directions for research. Though the present study has highlighted the potential difficulties of interpreting early childhood carbon and nitrogen stable isotope data, and to some extent underlined the vast quantity of unknowns, this should not be viewed negatively. We are in the process of recognising the limits of present knowledge and testing the validity of many precepts thought to be settled wisdom. In so doing, the discipline is moving forward with new recognition of the complexities of a biocultural interface, with more known at present than at any point before. Emerging techniques in bioarchaeological science offer increasing avenues of enquiry to address the unknowns

and gaps in present knowledge. Ultimately, it is an exciting time to be working in the discipline.

Appendix 1: Demographic Tables

This appendix contains tables presenting full demographic data for individuals sampled in the study.

A1.1 Non-adult individuals sampled

Sk.	Age (yrs.)	Tooth	Pathology
Littleport			
3311	8-12	dm2	Cribra orbitalia
3770	4-5	dm2	None
4116	7-10	dm2	Cribra orbitalia
4144	7.5-9	dm2	Woven bone (rib)
4494	8-9	dm2	None
4848	4-5	dm2	None
Edix Hill			
12	4	dm2	None
133	6-7	dm2	Cribra orbitalia
178	3-4	dm2	None
352	3-4	dm2	None
447B	6-7	dm2	None
529	9	dm2	Dental, ECNB
547B	10-11	dm2	None
587	8	dm2	T13 vertebra
679	1.5	dm2	None

Table A1.1 Summary of non-adult individuals sampled. ECNB = extra-cortical new bone

A1.2 Adult individuals sampled

Sk.	Age	Sex	Tooth	Pathology
3687	VYA	Female	M1	None
3708	YA	Male	M1	Spondylolysis
3745	YA	Female	M1	Scoliosis
3749	MA	Female	M1	CO, rib and spinal lesions, periostitis, AMTL
3819	A	Female	M1	CO
3885	VYA	Male	M1	CO, trauma
3893	A	Male	M1	None
4035	VYA	Female	M1	Fused C7 rib
4047	YA	Female	M1	CO, lytic lesion to S1, Os acromiale
4063	YA	Female	M1	None
4067	MA	Female	M1	Toe fracture
4073	MA	Male	M1	Finger fracture, ivory osteoma
4075	YA	Female	M1	Retained deciduous canines
4092	YA	Male	M1	Tibial osteitis
4095	A	Male	M1	CO, Kypho-scoliosis, finger fracture
4134	MA	Male	M1	MO, DJD
4139	A	Female	M1	CO
4173	YA	Male	M1	CO, Ivory osteoma, hypoplastic thoracic
4178	MA	Male	M1	CO
4250	MA	Male	M1	CO
4395	MA	Female	M1	CO, HFI, TMJD, L5 remodelling
4556	YA	Indeterm.	M1	Tuberculosis
4585	MA	Female	M1	CO, Periostitis, DJD, neoplasm of scaphoid, ivory osteoma
4603	MA	Male	M1	CO, DJD multi-joints

Table A1.2 Summary of adult individuals sampled at Littleport. (CO=cribra orbitalia, MO=myositis ossificans, HFI=hyperostosis frontalis interna, TMJD=temporo-mandibular joint dysfunction, DJD=degenerative joint disease)

Sk.	Age	Sex	Tooth	Pathology
4	YA	Female	M1	Dental disease, septal aperture
9	YA	Female	M1	Enamel hypoplasia, Harris lines, T13, endocranial ECNB
11	YA	Male	M1	Dental disease, DJD, squatting facets
20B	YA	Female	M1	DJD, lumbarisation of T12
33	YA	Male	M1	Dental disease, DJD, Os acromiale
45	VYA	Female	M1	DJD, enamel hypoplasia, Harris lines, squatting facets, L6
112	YA	Male	M1	Retained metopic suture, squatting facets, enamel hypoplasia, Harris lines
125	VYA	Male	M1	DJD, dental disease, enamel hypoplasia
146**	MA	Male	M1	Metastatic carcinoma, DJD, retained metopic suture, Os acromiale
198	VYA	Male	M1	DJD, CO, possible PH, enamel hypoplasia, Harris lines
300A	MA	Male	M1	Bilateral ECNB of tibiae, DJD, healed fracture of clavicle, spina bifida occulta and undeveloped sacral segment, squatting facets and platycnemia
359	VYA	Female	M1	DJD
440A	YA	Female	M1	Hyperplatymeric femora, asymmetrical femoral length, unfused sacral arch, bilateral Os acromiale, septal apertures, osteomyelitis of right elbow, scars of "parturition", spinal and extra-spinal DJD, multiple enlarged nutrient foramina
453A	VYA	Male	M1	DJD, enamel hypoplasia, pilasterism
547A	YA	Female	M1	DJD, dental disease
578	VYA	Male	M1	DJD, dental disease, retained metopic suture
626A	VYA	Female	M1	Cranial ECNB (frontal)
626B	VYA	Male	M1	DJD
726	VYA	Female	M1	Retained metopic suture
727	VYA	Male	M1	Os acromiale

Table A1.3 Summary of adult individuals sampled at Edix Hill. (T13=13th thoracic vertebra, ECNB=extra cortical new bone, DJD=degenerative joint disease, L6=6th lumbar vertebra, CO=cribra orbitalia, PH=porotic hyperostosis) **notification was received from Cambridge County Archive at the time of writing that the disarticulated tooth sampled from SK146 was incompatible with that individual and may possibly represent charnel from the same context. In the absence of an appropriate identifier, this individual will continue to be referred to as SK146, with the acknowledgement that the demographic data for that individual does not apply

Appendix 2: Collagen Data Tables

Full data tables for all individuals at the study sites are listed below. Dashed entries within a data column indicate a failed analysis, either due to instrumental fault, or a dearth of yielded collagen. Greyed rows indicate data which are outside of the parameters of acceptability, and which are included in the table, but are not included in data plots or other analyses.

A2.1 Edix Hill

A2.1.1 Deciduous tooth data

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH12-1	-0.2	-20.2	12.8	43.6	16.0	3.2	Rt ¾
EH12-2	0.1	-20.1	13.0	43.3	16.0	3.2	
EH12-3	0.4	-20.0	13.0	42.3	15.7	3.1	
EH12-4	0.7	-20.1	13.1	43.6	16.1	3.2	
EH12-5	1	-19.9	13.2	42.8	16.0	3.1	
EH12-6	1.3	-19.9	13.1	43.3	16.3	3.1	
EH12-7	1.6	-19.9	13.1	43.2	16.1	3.1	
EH12-8	1.9	-20.0	13.0	42.9	16.2	3.1	
EH12-9	2.2	-20.1	12.5	43.3	16.2	3.1	
EH12-10	2.5	-20.4	12.6	43.6	16.3	3.1	

Table A2.1 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH12

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH133-1	-0.2	-20.1	12.9	38.6	14.2	3.2	Complete
EH133-2	0.1	-20.2	13.2	38.8	14.2	3.2	
EH133-3	0.5	-20.4	13.4	38.9	14.3	3.2	
EH133-4	0.8	-20.8	13.2	38.6	14.2	3.2	
EH133-5	1.1	-21.0	13.2	39.0	14.4	3.2	
EH133-6	1.5	-21.0	13.1	39.2	14.4	3.2	
EH133-7	1.8	-21.1	13.3	38.6	14.2	3.2	
EH133-8	2.2	-21.1	12.4	39.1	14.4	3.2	
EH133-9	2.5	-21.1	12.2	38.7	14.3	3.2	
EH133-10	2.9	-21.1	12.3	39.1	14.4	3.2	
EH133-11	3.3	-21.0	12.1	39.5	14.5	3.2	

Table A2.2 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH133

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH178-1	-0.2	-20.1	13.1	40.2	14.9	3.1	Complete
EH178-2	0.3	-20.2	13.1	39.8	14.8	3.1	
EH178-3	0.9	-20.0	13.7	39.7	14.9	3.1	
EH178-4	1.4	-20.2	13.2	39.7	14.8	3.1	
EH178-5	1.9	-20.3	11.6	40.0	14.9	3.1	
EH178-6	2.4	-20.1	11.2	39.2	14.7	3.1	
EH178-7	3.0	-20.2	11.4	39.1	14.7	3.1	
EH178-8	3.5	-20.4	10.6	39.7	14.5	3.2	

Table A2.3 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH178

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH352-1	-0.2	-19.8	14.9	45.7	16.6	3.2	Rt ½
EH352-2	0.1	-19.8	14.6	44.6	16.4	3.2	
EH352-3	0.4	-20.0	15.5	38.9	14.1	3.2	
EH352-4	0.7	-20.1	14.8	45.6	16.6	3.2	
EH352-5	1.1	-20.3	13.7	43.1	15.6	3.2	
EH352-6	1.4	-	-	-	-	-	
EH352-7	1.7	-20.2	13.5	30.5	11.0	3.2	
EH352-8	2.0	-13.7	8.6	27.1	8.6	3.7	

Table A2.4 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH352

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH447B-1	-0.2	-20.1	12.3	39.4	14.3	3.2	Complete
EH447B-2	0.1	-19.8	12.9	39.1	14.3	3.2	
EH447B-3	0.4	-19.9	12.9	39.5	14.4	3.2	
EH447B-4	0.7	-19.9	13.2	39.2	14.4	3.2	
EH447B-5	1.0	-20.1	12.8	38.3	14.0	3.2	
EH447B-6	1.3	-20.3	12.2	37.9	13.9	3.2	
EH447B-7	1.6	-20.3	12.2	38.4	14.1	3.2	
EH447B-8	2.0	-20.3	12.3	38.1	13.9	3.2	
EH447B-9	2.3	-20.4	12.1	37.9	14.0	3.2	
EH447B-10	2.6	-20.4	12.0	38.1	13.9	3.2	
EH447B-11	2.9	-20.5	11.3	37.7	13.7	3.2	
EH447B-12	3.2	-20.4	11.0	38.5	14.0	3.2	
EH447B-13	3.5	-	-	-	-	-	

Table A2.5 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH447B

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH529-1	-0.2	-19.8	13.4	40.1	14.6	3.2	Complete
EH529-2	0.2	-19.6	13.5	39.5	14.7	3.1	
EH529-3	0.6	-20.0	13.5	39.8	14.6	3.2	
EH529-4	1.0	-20.4	12.4	39.7	14.6	3.2	
EH529-5	1.4	-20.5	12.2	39.6	14.6	3.2	
EH529-6	1.9	-20.7	12.0	40.2	14.8	3.2	
EH529-7	2.3	-20.7	11.9	40.0	14.9	3.1	
EH529-8	2.7	-20.6	11.3	40.0	14.7	3.2	
EH529-9/10	3.3	-20.4	11.2	39.8	14.7	3.2	

Table A2.6 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH529

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH547B-1	-0.2	-19.8	15.3	39.4	14.5	3.2	Complete
EH547B-2	0.0	-19.8	14.4	38.9	14.4	3.2	
EH547B-3	0.3	-19.9	14.2	39.5	14.7	3.1	
EH547B-4	0.5	-20.4	12.9	39.8	14.7	3.2	
EH547B-5	0.8	-20.6	12.1	38.8	14.3	3.2	
EH547B-6	1.0	-20.7	11.1	38.3	14.1	3.2	
EH547B-7	1.3	-20.6	10.6	37.6	13.9	3.1	
EH547B-8	1.5	-20.7	11.1	39.5	14.5	3.2	
EH547B-9	1.8	-20.7	10.6	39.4	14.5	3.2	
EH547B-10	2.0	-20.7	10.1	38.5	14.1	3.2	
EH547B-11	2.3	-20.5	10.0	40.4	14.6	3.2	
EH547B-12	2.5	-20.4	10.2	39.2	14.4	3.2	
EH547B-13	2.8	-20.4	10.3	39.5	14.4	3.2	
EH547B-14	3.0	-20.8	10.7	40.4	14.5	3.3	
EH547B-15	3.3	-20.6	10.6	38.9	14.2	3.2	
EH547B-16	3.5	-21.0	10.0	40.1	14.2	3.3	

Table A2.7 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH547B

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH587-1	-0.2	-20.4	11.7	40.5	15.0	3.2	Complete
EH587-2	0.1	-20.5	11.9	40.1	14.9	3.1	
EH587-3	0.5	-20.5	11.5	40.4	15.0	3.1	
EH587-4	0.8	-20.6	11.5	36.7	13.7	3.1	
EH587-5	1.1	-20.6	11.7	40.3	15.1	3.1	
EH587-6	1.5	-20.6	11.3	39.4	14.6	3.1	
EH587-7	1.8	-20.8	11.4	40.5	14.9	3.1	
EH587-8/9	2.3	-20.8	11.4	40.5	14.5	3.1	
EH587-9	2.5	-20.7	11.3	39.7	14.7	3.1	
EH587-10	2.8	-20.9	11.6	40.3	14.8	3.1	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH587-11	3.2	-20.7	11.6	39.8	14.5	3.2	
EH587-12	3.5	-24.0	11.8	53.1	10.1	6.1	

Table A2.8 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH587

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH679-1	-0.2	-19.9	12.1	37.9	13.3	3.3	Cr ¾
EH679-2	0.4	-19.9	11.6	38.1	14.0	3.2	
EH679-3	0.9	-20.2	11.4	39.7	14.5	3.2	
EH679-4	1.5	-20.2	11.5	39.7	14.5	3.2	

Table A2.9 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from EH679

A2.1.2 Permanent M1 data

A2.1.2.i Males

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH11-1	0.3	-20.5	10.5	32.9	12.0	3.2	Complete
EH11-2	0.9	-20.5	10.9	39.8	14.8	3.1	
EH11-3	1.5	-20.4	11.1	40.3	14.9	3.1	
EH11-4	2.0	-20.3	11.1	40.2	14.8	3.1	
EH11-5	2.6	-20.2	11.2	40.4	14.9	3.1	
EH11-6	3.2	-20.1	11.2	40.0	14.8	3.1	
EH11-7	3.8	-19.9	11.2	39.3	14.5	3.2	
EH11-8	4.3	-20.0	11.2	40.3	14.6	3.2	
EH11-9	4.9	-20.0	11.3	40.2	14.7	3.2	
EH11-10	5.5	-20.0	11.5	40.1	14.7	3.2	
EH11-11	6.1	-20.4	11.6	39.8	14.3	3.3	
EH11-12	6.6	-20.2	11.6	40.0	14.7	3.1	
EH11-13	7.2	-20.2	11.7	39.9	14.6	3.2	
EH11-14	7.8	-20.1	11.7	39.2	14.4	3.1	
EH11-15	8.4	-19.9	11.6	40.0	14.6	3.2	
EH11-16	8.9	-19.9	11.4	40.8	14.9	3.2	
EH11-17	9.5	-20.1	11.6	40.1	14.7	3.1	

Table A2.10 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH11

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH33-1	0.3	-20.7	9.0	40.3	14.9	3.1	Complete
EH33-2	1.0	-20.8	9.3	40.1	14.7	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH33-3	1.6	-20.7	9.6	40.3	14.8	3.2	
EH33-4	2.2	-20.5	9.8	40.2	14.7	3.2	
EH33-5	2.9	-20.1	9.8	39.5	14.5	3.2	
EH33-6	3.6	-20.1	9.7	40.0	14.6	3.2	
EH33-7	4.2	-20.0	9.6	39.3	14.4	3.2	
EH33-8	4.9	-20.1	9.4	40.3	14.7	3.2	
EH33-9	5.6	-20.4	9.5	40.2	14.7	3.2	
EH33-10	6.2	-20.3	9.3	38.4	14.0	3.2	
EH33-11	6.8	-20.3	9.4	40.1	14.5	3.2	
EH33-12	7.5	-20.1	9.2	40.0	14.5	3.2	
EH33-13	8.2	-20.0	9.9	39.7	14.5	3.2	
EH33-14	8.8	-20.3	10.0	40.1	14.4	3.2	
EH33-15	9.5	-20.7	10.3	40.1	14.2	3.3	

Table A2.11 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH33

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH112-1	0.3	-20.3	11.2	38.4	13.8	3.2	Complete
EH112-2	0.9	-20.6	9.6	37.7	13.6	3.2	
EH112-3	1.5	-20.5	9.8	37.9	13.7	3.2	
EH112-4	2.1	-20.5	10.0	37.3	13.5	3.2	
EH112-5	2.7	-20.5	9.9	37.0	13.3	3.2	
EH112-6	3.3	-20.4	9.7	37.5	13.5	3.2	
EH112-7	3.9	-20.4	9.7	37.5	13.5	3.2	
EH112-8	4.5	-20.3	9.8	37.5	13.4	3.3	
EH112-9	5.1	-20.2	9.6	38.0	13.6	3.2	
EH112-10	5.8	-20.2	9.7	37.3	13.4	3.2	
EH112-11	6.4	-20.2	9.7	37.7	13.5	3.3	
EH112-12	7.0	-20.0	9.8	36.4	13.2	3.2	
EH112-13	7.6	-20.1	10.4	34.8	12.5	3.2	
EH112-14	8.2	-20.1	10.6	35.7	12.9	3.2	
EH112-15	8.8	-20.1	10.5	37.7	13.6	3.2	
EH112-16	9.4	-20.1	10.6	34.7	12.5	3.2	
EH112-17	10.0	-20.3	11.1	37.8	13.6	3.3	

Table A2.12 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH112

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH125-1	0.3	-20.4	13.3	44.5	16.2	3.2	Complete
EH125-2	0.8	-20.5	12.7	44.5	16.3	3.2	
EH125-3	1.4	-20.6	12.4	45.0	16.4	3.2	
EH125-4	1.9	-20.6	11.6	44.9	16.4	3.2	
EH125-5	2.5	-20.6	11.1	45.6	16.6	3.2	
EH125-6	3.0	-20.5	11.1	46.6	17.0	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH125-7	3.5	-20.7	11.2	45.0	16.4	3.2	
EH125-8	4.1	-20.5	11.3	45.2	16.4	3.2	
EH125-9	4.6	-20.3	11.4	45.0	16.4	3.2	
EH125-10	5.2	-20.3	11.5	45.0	16.5	3.2	
EH125-11	5.7	-20.3	11.4	43.1	15.6	3.2	
EH125-12	6.3	-20.1	11.5	45.5	16.6	3.2	
EH125-13	6.8	-20.4	11.5	45.8	16.7	3.2	
EH125-14	7.6	-20.4	11.3	43.0	15.7	3.2	
EH125-15	8.4	-20.7	11.5	46.3	16.9	3.2	
EH125-16	9.0	-20.6	11.8	46.0	16.7	3.2	
EH125-17	9.5	-	-	-	-	-	

Table A2.13 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH125

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH146-1	0.3	-19.6	15.2	36.9	13.7	3.1	Complete
EH146-2	0.9	-19.8	13.8	37.0	13.6	3.2	
EH146-3	1.5	-20.0	13.1	36.1	13.3	3.2	
EH146-4	2.1	-20.3	11.7	31.7	11.8	3.1	
EH146-5	2.8	-20.4	11.0	37.1	13.8	3.1	
EH146-6	3.4	-20.5	10.6	38.2	14.1	3.2	
EH146-7	4.0	-20.5	10.5	35.4	13.2	3.1	
EH146-8	4.6	-20.4	10.4	36.5	13.5	3.2	
EH146-9	5.2	-20.4	10.6	36.3	13.4	3.2	
EH146-10	5.8	-20.4	10.7	35.7	13.2	3.2	
EH146-11	6.4	-20.4	10.8	37.3	13.8	3.2	
EH146-12	7.0	-20.3	10.8	37.4	13.7	3.2	
EH146-13	7.7	-20.3	11.2	36.6	13.5	3.2	
EH146-14	8.3	-20.4	10.8	37.5	13.8	3.2	
EH146-15	8.9	-20.2	10.4	35.2	12.9	3.2	
EH146-16	9.5	-20.2	11.1	37.4	13.7	3.2	

Table A2.14 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH146

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH198-1	0.3	-20.6	12.2	40.2	14.6	3.2	Complete
EH198-2	0.8	-20.5	12.1	35.8	13.1	3.2	
EH198-3	1.4	-20.6	12.0	36.9	13.1	3.3	
EH198-4	1.9	-20.4	11.5	38.9	14.2	3.2	
EH198-5	2.5	-20.5	10.7	38.4	14.1	3.2	
EH198-6	3.0	-20.4	10.3	38.8	14.1	3.2	
EH198-7	3.5	-20.4	10.5	39.5	14.4	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH198-8	4.1	-20.3	10.4	35.8	13.1	3.2	
EH198-9	4.6	-20.2	10.4	39.7	14.5	3.2	
EH198-10	5.2	-20.3	10.1	39.7	14.6	3.1	
EH198-11	5.7	-20.4	10.7	40.3	14.8	3.2	
EH198-12	6.3	-20.2	10.7	39.7	14.5	3.2	
EH198-13	6.8	-20.4	10.9	39.5	14.5	3.2	
EH198-14	7.3	-20.4	10.9	39.1	14.3	3.2	
EH198-15	7.9	-20.3	11.1	39.5	14.4	3.2	
EH198-16	8.4	-20.4	11.1	40.0	14.6	3.2	
EH198-17	9.0	-20.4	11.1	39.6	14.4	3.2	
EH198-18	9.5	-20.2	10.9	40.0	14.6	3.2	

Table A2.15 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH198

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH300A-1	0.3	-20.5	11.5	39.7	14.4	3.2	Complete
EH300A-2	1.0	-20.5	10.8	39.6	14.5	3.2	
EH300A-3	1.7	-20.5	10.3	39.9	14.6	3.2	
EH300A-4	2.4	-20.5	10.3	39.8	14.6	3.2	
EH300A-5	3.1	-20.4	10.4	38.4	14.1	3.2	
EH300A-6	3.8	-20.3	10.6	39.1	14.3	3.2	
EH300A-7	4.5	-20.1	10.9	39.7	14.5	3.2	
EH300A-8	5.2	-20.1	10.7	39.9	14.7	3.2	
EH300A-9	5.8	-20.1	10.3	38.9	14.3	3.2	
EH300A-10	6.5	-20.2	10.5	40.0	14.7	3.2	
EH300A-11	7.2	-20.2	10.2	40.0	14.7	3.2	
EH300A-12	7.9	-20.1	10.4	39.1	14.2	3.2	
EH300A-13	8.6	-	-	-	-	-	
EH300A-14	9.3	-20.4	10.9	39.8	14.5	3.2	
EH300A-15	10.0	-20.0	14.1	35.5	13.1	3.2	

Table A2.16 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH300A

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH453-1	0.3	-21.1	11.8	35.8	12.9	3.2	Complete
EH453-2	0.8	-20.9	11.4	41.0	14.8	3.2	
EH453-3	1.3	-21.0	11.4	32.7	11.8	3.2	
EH453-4	1.8	-20.6	11.4	37.6	13.6	3.2	
EH453-5	2.3	-20.4	11.3	38.1	13.7	3.2	
EH453-6	2.9	-20.3	11.3	38.5	13.9	3.2	
EH453-7	3.4	-20.2	11.4	37.8	13.6	3.3	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH453-8	3.9	-20.1	11.6	37.8	13.5	3.3	
EH453-9	4.4	-20.2	11.7	37.6	13.5	3.2	
EH453-10	4.9	-20.3	11.7	39.3	14.1	3.3	
EH453-11	5.4	-20.1	11.6	37.4	13.4	3.2	
EH453-12	5.9	-20.1	11.6	37.9	13.6	3.2	
EH453-13	6.4	-20.0	11.5	38.8	13.9	3.2	
EH453-14	6.9	-20.0	11.5	39.4	14.1	3.3	
EH453-15	7.5	-20.1	11.2	37.0	13.2	3.3	
EH453-16	8.0	-20.3	11.7	38.3	13.8	3.3	
EH453-17	8.5	-19.9	11.8	38.6	13.9	3.2	
EH453-18	9.0	-20.4	11.8	38.9	13.9	3.3	
EH453-19	9.5	-20.2	11.7	38.8	13.8	3.3	

Table A2.17 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH453

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH578-1	0.3	-21.1	12.6	30.9	11.5	3.1	Complete
EH578-2	0.9	-20.9	10.8	38.2	14.1	3.2	
EH578-3	1.5	-20.4	10.4	38.7	14.3	3.1	
EH578-4	2.0	-20.4	10.2	38.6	14.2	3.2	
EH578-5	2.6	-20.3	10.1	37.6	13.9	3.2	
EH578-6	3.5	-20.2	9.9	38.5	14.2	3.2	
EH578-7	4.3	-20.3	9.8	37.8	13.9	3.2	
EH578-8	4.9	-20.3	9.8	38.2	14.1	3.2	
EH578-9	5.5	-20.5	9.9	38.3	14.1	3.2	
EH578-10	6.1	-20.4	10.1	37.4	13.8	3.2	
EH578-11	6.6	-20.4	10.0	38.4	14.1	3.2	
EH578-12	7.2	-20.4	9.9	38.3	13.7	3.3	
EH578-13	7.8	-20.4	9.9	38.4	13.7	3.3	
EH578-14	8.4	-20.3	10.7	37.3	13.5	3.2	
EH578-15	8.9	-20.4	11.9	38.2	13.7	3.3	
EH578-16	9.5	-20.4	12.0	38.7	13.9	3.3	

Table A2.18 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH578

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH626B-1	0.3	-20.0	13.5	39.7	14.6	3.2	Complete
EH626B-2	1.1	-20.4	12.4	39.3	14.5	3.2	
EH626B-3	1.8	-20.6	12.0	39.8	14.7	3.2	
EH626B-4	2.6	-20.7	11.8	38.8	14.3	3.2	
EH626B-5	3.4	-20.7	11.7	38.8	14.2	3.2	
EH626B-6	4.1	-20.6	11.7	39.0	14.3	3.2	
EH626B-7	4.9	-20.4	11.4	39.4	14.4	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH626B-8	5.7	-20.3	11.3	38.3	14.1	3.2	
EH626B-9	6.4	-20.3	11.3	39.6	14.5	3.2	
EH626B-10	7.2	-20.4	10.9	37.2	13.7	3.2	
EH626B-11	8.0	-	-	-	-	-	
EH626B-12	8.7	-20.4	11.2	38.9	14.2	3.2	
EH626B-13	9.5	-20.6	11.2	39.2	14.2	3.2	

Table A2.19 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH626B

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH727-1	0.3	-20.4	14.1	36.7	13.3	3.2	Complete
EH727-2	0.9	-20.5	13.6	37.7	13.7	3.2	
EH727-3	1.6	-20.6	12.5	39.9	14.5	3.2	
EH727-4	2.2	-20.6	11.2	38.8	14.2	3.2	
EH727-5	2.9	-20.4	11.0	36.0	13.1	3.2	
EH727-6	3.5	-20.3	10.2	37.8	13.7	3.2	
EH727-7	4.2	-20.4	10.3	39.3	14.3	3.2	
EH727-8	4.8	-20.3	10.5	38.9	14.1	3.2	
EH727-9	5.5	-20.2	10.5	40.0	14.5	3.2	
EH727-10	6.1	-20.3	11.0	40.1	14.6	3.2	
EH727-11	6.8	-20.4	11.6	40.1	14.5	3.2	
EH727-12	7.4	-20.5	11.9	39.4	14.3	3.2	
EH727-13	8.1	-20.3	12.0	39.9	14.5	3.2	
EH727-14	8.7	-20.2	11.8	39.4	14.3	3.2	
EH727-15	9.4	-20.2	11.4	39.3	14.3	3.2	
EH727-16	10.0	-20.3	11.8	40.0	14.5	3.2	

Table A2.20 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH727

A2.1.2.ii Females

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH4-1	0.3	-20.4	14.0	39.6	14.6	3.2	Complete
EH4 2	0.9	-20.6	13.2	39.0	14.4	3.2	
EH4-3	1.5	-20.8	12.5	38.4	14.1	3.2	
EH4-4	2.1	-20.7	12.0	37.0	13.6	3.2	
EH4-5	2.8	-20.7	11.7	38.4	14.0	3.2	
EH4-6	3.4	-20.6	11.6	38.4	14.0	3.2	
EH4-7	4.3	-20.5	12.0	32.2	11.8	3.2	
EH4-8	5.2	-20.6	12.5	37.1	13.6	3.2	
EH4-9	5.8	-20.4	12.7	37.2	13.7	3.2	
EH4-10	6.4	-20.2	12.7	35.6	13.1	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH4-11	7.0	-20.2	12.3	39.0	14.3	3.2	
EH4-12	7.7	-20.2	12.0	38.2	14.0	3.2	
EH4-13	8.3	-20.2	11.8	37.5	13.7	3.2	
EH4-14	8.9	-20.2	11.5	35.4	12.8	3.2	
EH4-15	9.5	-20.9	11.9	38.1	13.7	3.3	

Table A2.21 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH4

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH9-1	0.3	-21.0	13.6	40.2	14.8	3.2	Complete
EH9-2	1.0	-20.9	11.7	40.2	14.8	3.2	
EH9-3	1.7	-20.9	11.2	36.3	13.4	3.2	
EH9-4	2.4	-20.9	10.7	40.0	14.8	3.2	
EH9-5	3.1	-20.6	10.4	40.4	14.9	3.2	
EH9-6	3.8	-20.2	10.3	40.2	14.7	3.2	
EH9-7	4.5	-20.3	10.0	38.3	14.0	3.2	
EH9-8	5.2	-20.3	10.1	39.8	14.6	3.2	
EH9-9	5.8	-20.2	10.1	39.7	14.6	3.2	
EH9-10	6.5	-20.3	10.1	40.2	14.8	3.2	
EH9-11	7.7	-20.3	10.4	40.1	14.7	3.2	
EH9-12	8.8	-20.2	10.0	40.0	14.7	3.2	
EH9-13	10.0	-20.0	10.2	40.2	14.7	3.2	

Table A2.22 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH9

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH20B-1	7.5	-20.6	10.9	29.6	10.1	3.4	Rt ¾ (break)
EH20B-2	6.9	-20.2	11.2	41.1	14.4	3.3	
EH20B-3	6.2	-20.5	11.4	43.0	15.3	3.3	
EH20B-4	5.5	-20.5	11.0	41.7	14.6	3.3	
EH20B-5	4.9	-20.5	11.3	43.0	15.2	3.3	
EH20B-6	4.2	-20.6	11.5	41.5	14.6	3.3	
EH20B-7	3.6	-20.6	12.0	39.9	14.1	3.3	
EH20B-8	2.9	-20.7	12.5	41.8	14.7	3.3	
EH20B-9	2.3	-20.7	12.9	46.2	16.5	3.3	
EH20B-10	1.6	-20.7	12.7	28.2	9.7	3.4	
EH20B-11	1.0	-19.9	15.6	42.3	14.9	3.3	
EH20B-12	0.3	-20.7	14.0	20.5	6.6	3.6	

Table A2.23 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH20B

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH45-1	0.3	-20.3	13.2	31.2	11.5	3.2	Complete

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH45-2	0.9	-20.9	12.5	36.1	13.3	3.2	
EH45-3	1.5	-20.9	11.2	37.6	13.9	3.2	
EH45-4	2.4	-20.5	10.0	35.6	13.1	3.2	
EH45-5	3.3	-20.2	9.7	38.5	14.2	3.2	
EH45-6	3.9	-20.1	9.8	39.4	14.4	3.2	
EH45-7	4.5	-20.1	10.0	39.1	14.3	3.2	
EH45-8	5.1	-20.2	10.1	39.3	14.4	3.2	
EH45-9	5.8	-20.0	9.8	33.8	12.4	3.2	
EH45-10	6.4	-20.1	10.0	38.1	14.0	3.2	
EH45-11	7.0	-19.9	10.4	39.5	14.5	3.2	
EH45-12	7.6	-19.9	10.2	36.2	13.3	3.2	
EH45-13	8.2	-20.0	9.9	38.9	14.3	3.2	
EH45-14	8.8	-20.2	10.2	38.2	14.0	3.2	
EH45-15	9.4	-19.9	10.2	38.2	14.0	3.2	
EH45-16	10.0	-20.0	10.4	38.6	14.1	3.2	

Table A2.24 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH45

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH359-1	10.0	-20.7	9.9	32.7	11.5	3.3	Complete
EH359-2	9.4	-20.6	9.6	29.7	10.4	3.3	
EH359-3	8.9	-20.4	9.8	40.0	14.3	3.3	
EH359-4	8.3	-20.4	9.6	42.0	15.2	3.2	
EH359-5	7.7	-20.6	9.7	42.8	15.4	3.2	
EH359-6	7.2	-20.5	9.7	40.3	14.5	3.3	
EH359-7	6.6	-20.3	9.5	43.0	15.6	3.2	
EH359-8	6.0	-20.3	9.4	39.9	14.3	3.2	
EH359-9	5.4	-20.5	9.3	38.8	13.9	3.3	
EH359-10	4.9	-20.4	9.2	44.2	16.0	3.2	
EH359-11	4.3	-20.7	9.1	40.0	14.3	3.3	
EH359-12	3.7	-20.4	9.0	42.7	15.5	3.2	
EH359-13	3.2	-20.3	8.7	43.3	15.7	3.2	
EH359-14	2.6	-20.3	8.6	41.9	15.2	3.2	
EH359-15	2.0	-20.4	8.7	43.4	15.7	3.2	
EH359-16	1.4	-20.5	9.1	42.3	15.3	3.2	
EH359-17	0.9	-20.4	10.7	43.5	15.7	3.2	
EH359-18	0.3	-20.2	13.1	42.2	15.3	3.2	

Table A2.25 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH359

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH440A-1	0.3	-20.6	12.4	40.2	14.7	3.2	Complete
EH440A-2	1.1	-20.2	12.7	35.9	13.3	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH440A-3	2.2	-20.3	11.7	42.3	15.5	3.2	
EH440A-4	3.4	-20.5	11.2	39.8	14.6	3.2	
EH440A-5	4.1	-20.5	11.1	40.6	14.9	3.2	
EH440A-6	4.9	-20.5	11.0	39.6	14.6	3.2	
EH440A-7	5.7	-20.2	11.0	39.9	14.6	3.2	
EH440A-8	6.4	-20.4	10.9	39.2	14.4	3.2	
EH440A-9	7.2	-20.4	11.6	40.9	14.9	3.2	
EH440A-10	8.0	-20.3	12.0	40.2	14.7	3.2	
EH440A-11	8.7	-20.2	11.9	38.9	14.4	3.2	
EH440A-12	9.5	-20.4	11.8	38.1	14.0	3.2	

Table A2.26 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH440A

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH547A-1	0.3	-20.2	14.2	43.9	16.1	3.2	Complete
EH547A-2	1.0	-20.7	12.7	41.6	15.3	3.2	
EH547A-3	1.8	-21.0	11.9	40.8	15.0	3.2	
EH547A-4	2.5	-20.6	11.3	42.8	15.8	3.2	
EH547A-5	3.3	-20.4	11.6	43.0	15.8	3.2	
EH547A-6	4.0	-20.4	11.4	42.1	15.5	3.2	
EH547A-7	4.8	-20.1	11.5	42.9	15.8	3.2	
EH547A-8	5.5	-19.9	11.5	42.0	15.4	3.2	
EH547A-9	6.3	-20.0	11.6	41.4	15.2	3.2	
EH547A-10	7.0	-19.9	11.6	41.8	15.3	3.2	
EH547A-11	7.8	-20.0	11.5	40.1	14.7	3.2	
EH547A-12	8.5	-20.0	11.5	41.0	15.0	3.2	
EH547A-13	9.3	-20.1	11.9	42.2	15.4	3.2	
EH547A-14	10.0	-20.1	12.1	41.4	15.1	3.2	

Table A2.27 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH547A

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH626A-1	0.3	-20.9	12.7	32.6	12.0	3.2	Complete
EH626A-2	1.2	-20.8	12.7	20.2	7.5	3.1	
EH626A-3	2.1	-20.6	12.1	37.7	13.9	3.2	
EH626A-4	2.7	-20.5	11.6	37.9	13.9	3.2	
EH626A-5	3.4	-20.6	11.7	36.9	13.3	3.2	
EH626A-6	4.0	-20.6	11.6	36.7	13.1	3.3	
EH626A-7	4.6	-20.4	11.5	37.8	13.9	3.2	
EH626A-8	5.5	-20.4	11.3	27.2	10.1	3.2	
EH626A-9	6.4	-20.3	11.4	38.2	13.8	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH626A-10	7.0	-20.4	11.5	38.8	14.2	3.2	
EH626A-11	7.6	-20.5	11.5	37.9	13.9	3.2	
EH626A-12	8.3	-20.3	11.6	37.1	13.6	3.2	
EH626A-13/14	9.2	-20.6	11.6	47.6	17.4	3.2	

Table A2.28 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH626A

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
EH726-1	10.0	-21.6	8.0	15.4	4.4	4.1	Complete
EH726-2	9.3	-20.4	10.8	38.1	13.3	3.4	
EH726-3	8.6	-20.3	10.2	37.5	13.1	3.4	
EH726-4	7.9	-20.7	10.4	31.0	10.7	3.4	
EH726-5	7.2	-20.5	10.3	37.6	12.9	3.4	
EH726-6	6.5	-20.4	10.7	39.6	13.8	3.4	
EH726-7	5.8	-20.2	10.9	41.0	14.4	3.3	
EH726-8	5.2	-20.3	10.9	41.1	14.4	3.3	
EH726-9	4.5	-20.4	10.9	42.4	15.0	3.3	
EH726-10	3.8	-20.2	10.8	42.1	14.8	3.3	
EH726-11	3.1	-20.3	10.6	41.8	14.7	3.3	
EH726-12	2.4	-20.5	10.6	41.8	14.8	3.3	
EH726-13	1.7	-20.4	10.4	43.9	15.5	3.3	
EH726-14	1.0	-20.3	10.7	43.5	15.5	3.3	
EH726-15	0.3	-20.3	12.8	41.9	14.7	3.3	

Table A2.29 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from EH726

A2.2 Littleport

A2.2.1 Deciduous tooth data

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3311-1	-0.2	-20.4	14.9	40.2	14.7	3.2	Complete
LP3311-2	0.3	-20.4	15.2	39.8	14.7	3.2	
LP3311-3	0.7	-20.9	15.7	40.2	14.7	3.2	
LP3311-4	1.2	-20.7	15.4	39.2	14.4	3.2	
LP3311-5/6/7/8/9	2.6	-21.6	13.8	41.3	13.8	3.5	

Table A2.30 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from LP3311

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3770-1	-0.2	-21.1	11.7	39.5	13.7	3.4	Complete
LP3770-2	0.3	-20.9	10.2	38.2	13.8	3.2	
LP3770-3	0.7	-20.7	10.3	40.0	14.6	3.2	
LP3770-4	1.2	-20.3	11.0	39.3	14.4	3.2	
LP3770-5	1.7	-20.1	11.2	39.8	14.5	3.2	
LP3770-6	2.1	-20.1	11.5	40.1	14.8	3.2	
LP3770-7	2.6	-20.0	11.4	40.1	14.7	3.2	
LP3770-8	3.0	-20.2	11.4	40.2	14.7	3.2	
LP3770-9	3.5	-20.4	11.4	40.0	14.3	3.3	

Table A2.31 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from LP3770

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4116-1	-0.2	-20.7	14.4	38.5	14.2	3.2	Complete
LP4116-2	0.2	-21.1	14.6	38.1	13.8	3.2	
LP4116-3	0.5	-21.3	14.2	38.1	14.0	3.2	
LP4116-4	0.9	-21.4	13.6	37.6	13.9	3.2	
LP4116-5	1.3	-21.3	13.9	34.5	12.8	3.2	
LP4116-6	1.7	-21.5	13.3	38.8	14.2	3.2	
LP4116-7	2.0	-21.6	13.0	38.6	14.1	3.2	
LP4116-8	2.4	-21.5	12.6	38.2	13.8	3.2	
LP4116-9	2.8	-21.5	12.7	38.5	14.0	3.2	
LP4116-10/11	3.3	-21.6	12.6	38.8	13.8	3.3	

Table A2.32 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from LP4116

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4144-1	-0.2	-20.3	14.5	39.3	14.5	3.2	Complete
LP4144-2	0.2	-20.4	15.3	40.1	14.8	3.2	
LP4144-3	0.6	-20.5	15.5	40.1	14.8	3.2	
LP4144-4/5	1.2	-20.9	16.2	40.1	14.7	3.2	
LP4144-5	1.4	-20.8	16.2	40.1	14.8	3.2	
LP4144-6	1.9	-20.9	15.5	39.9	14.7	3.2	
LP4144-7	2.3	-21.2	13.8	40.0	14.8	3.2	
LP4144-8	2.7	-21.2	13.2	39.9	14.7	3.2	
LP4144-9	3.1	-21.3	13.1	40.2	14.8	3.2	
LP4144-10	3.5	-	-	-	-	-	

Table A2.33 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from LP4144

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4494-1	-0.2	-20.5	14.6	45.6	16.8	3.2	Complete
LP4494-2	0.1	-20.2	15.4	45.9	16.7	3.2	
LP4494-3	0.5	-20.5	14.9	44.8	16.4	3.2	
LP4494-4	0.8	-20.5	15.5	44.8	16.3	3.2	
LP4494-5	1.1	-20.7	14.7	44.8	16.4	3.2	
LP4494-6	1.5	-20.9	13.5	45.7	16.7	3.2	
LP4494-7	1.8	-20.9	13.8	45.8	16.8	3.2	
LP4494-8	2.2	-21.2	12.7	44.3	16.1	3.2	
LP4494-9	2.5	-21.0	12.1	44.9	16.4	3.2	
LP4494-10	2.8	-21.0	12.3	46.4	16.8	3.2	
LP4494-11	3.2	-20.9	12.1	48.5	17.6	3.2	
LP4494-12	3.5	-	-	-	-	-	

Table A2.34 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from LP4494

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4848-1	-0.2	-19.9	12.5	46.7	17.1	3.2	Complete
LP4848-2	0.0	-20.5	10.3	45.3	16.6	3.2	
LP4848-3	0.3	-20.8	9.1	46.1	16.8	3.2	
LP4848-4	0.5	-20.7	9.3	44.7	16.4	3.2	
LP4848-5	0.7	-20.6	9.4	45.7	16.7	3.2	
LP4848-6	1.0	-20.5	9.1	45.5	16.7	3.2	
LP4848-7	1.2	-20.4	9.0	46.1	16.3	3.3	
LP4848-8	1.4	-20.3	9.1	45.6	16.6	3.2	
LP4848-9	1.6	-20.1	9.1	45.8	16.8	3.2	
LP4848-10	1.9	-19.8	9.5	49.5	18.2	3.2	
LP4848-11	2.1	-20.1	9.3	45.9	16.9	3.2	
LP4848-12	2.3	-20.1	9.3	45.6	16.7	3.2	
LP4848-13	2.6	-20.2	9.4	44.4	16.3	3.2	
LP4848-14	2.8	-20.2	9.2	47.1	17.4	3.2	
LP4848-15	3.0	-20.4	9.4	45.4	16.6	3.2	
LP4848-16	3.3	-20.5	9.4	45.4	16.6	3.2	
LP4848-17	3.5	-20.4	10.0	49.5	17.9	3.2	

Table A2.35 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled dm2 dentine from LP4848

A2.2.2 Permanent M1 data

A2.2.2.i Males

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3708-1	0.3	-20.6	12.2	46.0	16.7	3.2	Complete
LP3708-2	1.4	-20.7	11.3	45.1	16.5	3.2	
LP3708-3	2.5	-20.5	11.0	45.1	16.5	3.2	
LP3708-4	3.3	-20.2	10.0	44.1	16.3	3.2	
LP3708-5	4.0	-20.1	10.2	41.5	15.1	3.2	
LP3708-6	4.8	-20.1	10.4	45.2	16.5	3.2	
LP3708-7	5.5	-20.1	10.4	44.9	16.4	3.2	
LP3708-8	6.3	-20.1	10.4	37.1	13.5	3.2	
LP3708-9	7.0	-20.3	10.7	38.5	14.0	3.2	
LP3708-10	7.8	-20.3	10.6	39.5	14.4	3.2	
LP3708-11	8.5	-20.4	10.3	33.8	12.3	3.2	
LP3708-12	9.3	-20.5	11.2	38.8	14.1	3.2	
LP3708-13	10.0	-20.6	11.7	25.3	9.1	3.2	

Table A2.36 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3708

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3885-1	0.3	-21.1	13.2	41.0	15.0	3.2	Complete
LP3885-2	0.8	-21.1	12.7	40.8	15.0	3.2	
LP3885-3	1.3	-21.0	12.3	40.7	14.9	3.2	
LP3885-4	1.8	-20.9	12.4	40.9	15.0	3.2	
LP3885-5	2.3	-20.9	12.3	40.7	14.9	3.2	
LP3885-6	2.9	-21.1	12.2	40.7	14.9	3.2	
LP3885-7	3.4	-21.2	11.9	40.8	14.9	3.2	
LP3885-8	3.9	-21.2	11.8	40.6	14.9	3.2	
LP3885-9	4.4	-21.2	11.6	40.6	14.9	3.2	
LP3885-10	4.9	-21.0	11.6	40.7	14.9	3.2	
LP3885-11	5.4	-20.8	11.7	40.8	14.9	3.2	
LP3885-12	5.9	-21.0	11.8	40.8	14.9	3.2	
LP3885-13	6.4	-21.1	11.6	40.7	14.9	3.2	
LP3885-14	6.9	-21.0	11.5	40.6	14.9	3.2	
LP3885-15	7.5	-21.1	11.8	40.3	14.8	3.2	
LP3885-16	8.0	-21.0	12.4	39.5	14.5	3.2	
LP3885-17	8.5	-20.9	11.8	40.7	14.8	3.2	
LP3885-18	9.0	-21.3	12.1	40.6	14.9	3.2	
LP3885-19	9.5	-21.0	12.4	40.5	14.7	3.2	
LP3885-20	10.0	-21.0	12.5	40.9	14.9	3.2	

Table A2.37 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3885

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3893-1	0.3	-21.1	12.4	39.9	14.0	3.3	Complete
LP3893-2	0.9	-21.0	11.7	39.9	14.1	3.3	
LP3893-3	1.6	-21.0	11.4	40.7	14.4	3.3	
LP3893-4/5	2.6	-20.8	10.4	40.7	14.4	3.3	
LP3893-6	3.5	-20.7	10.3	40.7	14.6	3.2	
LP3893-7	4.2	-20.6	9.7	40.6	14.5	3.3	
LP3893-8	4.8	-20.6	9.9	40.3	14.4	3.3	
LP3893-9	5.5	-20.7	10.2	41.1	14.6	3.3	
LP3893-10	6.1	-20.4	9.6	41.2	14.7	3.3	
LP3893-11/12	7.1	-20.3	10.4	40.3	14.4	3.3	
LP3893-13/14	8.4	-20.3	11.0	41.1	14.8	3.2	
LP3893-15/16	9.7	-20.6	11.5	40.1	13.7	3.4	

Table A2.38 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3893

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4073-1	0.3	-21.0	12.7	40.6	14.6	3.2	Complete
LP4073-2	0.8	-20.8	12.9	36.5	13.4	3.2	
LP4073-3	1.4	-20.4	12.8	36.5	13.5	3.2	
LP4073-4	1.9	-20.2	12.8	38.1	14.0	3.2	
LP4073-5	2.5	-20.2	12.6	38.5	14.1	3.2	
LP4073-6	3.0	-20.1	12.8	39.1	14.3	3.2	
LP4073-7	3.5	-20.2	12.7	38.8	14.3	3.2	
LP4073-8	4.1	-20.2	13.0	34.8	12.8	3.2	
LP4073-9	4.6	-20.2	13.4	38.2	14.0	3.2	
LP4073-10	5.2	-20.4	13.2	37.6	13.7	3.2	
LP4073-11	5.7	-20.3	13.5	39.0	14.2	3.2	
LP4073-12	6.3	-20.3	12.5	39.2	14.2	3.2	
LP4073-13	6.8	-20.0	12.2	36.0	13.1	3.2	
LP4073-14	7.3	-20.2	12.7	32.8	11.9	3.2	
LP4073-15	7.9	-20.0	12.5	37.5	13.6	3.2	
LP4073-16	8.4	-20.3	12.6	37.1	13.4	3.2	
LP4073-17	9.0	-20.5	12.7	33.5	12.3	3.2	
LP4073-18	9.5	-20.9	12.6	37.2	13.4	3.2	

Table A2.39 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4073

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4092-1	0.3	-20.9	14.2	42.7	15.7	3.2	Complete
LP4092-2	1.0	-21.2	11.7	38.3	14.2	3.2	
LP4092-3	1.7	-21.0	11.5	42.3	15.6	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4092-4	2.4	-21.0	11.9	42.6	15.8	3.2	
LP4092-5	3.1	-20.9	12.1	41.8	15.5	3.2	
LP4092-6	3.8	-20.8	12.3	41.9	15.5	3.1	
LP4092-7	4.5	-20.7	12.4	42.6	15.8	3.1	
LP4092-8	5.2	-20.7	12.6	42.0	15.5	3.2	
LP4092-9	5.8	-20.6	12.6	42.7	15.8	3.1	
LP4092-10	6.5	-20.6	12.6	40.5	15.0	3.2	
LP4092-11	7.2	-20.6	12.5	42.0	15.4	3.2	
LP4092-12	7.9	-20.8	12.3	40.7	15.0	3.2	
LP4092-13	8.6	-20.4	12.1	43.0	15.9	3.2	
LP4092-14	9.3	-20.7	12.2	39.8	14.7	3.2	
LP4092-15	10.0	-20.7	12.3	42.1	15.5	3.2	

Table A2.40 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4092

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4095-1	0.3	-21.9	13.0	35.4	12.9	3.2	Complete
LP4095-2	1.0	-22.1	12.6	39.3	14.3	3.2	
LP4095-3	2.2	-21.6	11.9	38.1	13.8	3.2	
LP4095-4	3.3	-21.4	12.0	39.1	14.2	3.2	
LP4095-5	4.0	-21.3	12.2	38.5	14.0	3.2	
LP4095-6	4.8	-21.3	12.3	39.4	14.2	3.2	
LP4095-7	5.5	-21.4	12.3	39.1	14.2	3.2	
LP4095-8	6.3	-21.5	12.4	38.0	13.8	3.2	
LP4095-9	7.0	-21.5	12.5	39.9	14.5	3.2	
LP4095-10	7.8	-21.2	12.5	38.7	14.1	3.2	
LP4095-11	8.5	-21.3	13.0	38.5	14.0	3.2	
LP4095-12	9.3	-21.2	13.1	39.4	14.4	3.2	
LP4095-13	10.0	-21.4	13.3	39.0	14.1	3.2	

Table A2.41 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4095

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4134-1	0.3	-20.6	13.0	39.2	14.2	3.2	Complete
LP4134-2	1.0	-20.9	12.2	38.2	13.9	3.2	
LP4134-3	1.7	-21.0	12.0	37.7	13.7	3.2	
LP4134-4	2.4	-20.8	11.9	36.4	13.3	3.2	
LP4134-5	3.1	-20.8	12.1	36.7	13.4	3.2	
LP4134-6	3.8	-20.9	12.3	39.0	14.2	3.2	
LP4134-7	4.5	-20.8	12.5	35.8	13.0	3.2	
LP4134-8	5.2	-20.9	12.6	39.2	14.2	3.2	
LP4134-9	5.8	-20.9	12.7	38.6	14.0	3.2	
LP4134-10	6.5	-20.9	12.7	39.0	14.1	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4134-11	7.2	-20.9	13.0	36.6	13.3	3.2	
LP4134-12	7.9	-21.1	13.2	33.9	12.3	3.2	
LP4134-13	8.6	-21.3	13.0	38.4	13.9	3.2	
LP4134-14	9.3	-21.2	13.1	35.9	13.0	3.2	
LP4134-15	10.0	-21.3	13.2	37.9	13.7	3.2	

Table A2.42 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4134

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4173-1	0.3	-20.4	13.3	41.4	15.3	3.2	Complete
LP4173-2	0.9	-20.6	12.2	41.7	15.4	3.2	
LP4173-3	1.5	-20.6	11.9	40.6	14.8	3.2	
LP4173-4	2.4	-20.6	11.5	43.7	16.1	3.2	
LP4173-5	3.2	-20.7	11.1	42.0	15.5	3.2	
LP4173-6	3.8	-20.8	11.3	42.1	15.6	3.2	
LP4173-7	4.3	-20.8	11.4	42.5	15.7	3.2	
LP4173-8	4.9	-20.9	11.7	41.9	15.5	3.2	
LP4173-9	5.5	-21.0	11.7	42.0	15.5	3.2	
LP4173-10	6.1	-21.0	11.6	43.1	15.9	3.2	
LP4173-11	6.6	-20.9	11.7	41.1	15.2	3.2	
LP4173-12	7.2	-21.0	12.1	41.4	15.2	3.2	
LP4173-13	7.8	-20.7	12.4	41.9	15.4	3.2	
LP4173-14	8.4	-20.8	12.5	39.8	14.2	3.3	
LP4173-15	8.9	-20.7	12.9	43.5	15.7	3.2	
LP4173-16	9.5	-	-	-	-	-	

Table A2.43 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4173

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4178-1	0.3	-20.3	16.5	36.8	13.6	3.2	Complete
LP4178-2	0.8	-20.3	16.5	39.6	14.6	3.2	
LP4178-3	1.4	-20.3	15.7	39.8	14.7	3.2	
LP4178-4	1.9	-20.0	14.1	39.5	14.5	3.2	
LP4178-5	2.7	-20.0	12.4	40.6	14.8	3.2	
LP4178-6	3.5	-19.6	12.2	39.7	14.7	3.1	
LP4178-7	4.1	-19.5	12.5	39.9	14.8	3.2	
LP4178-8	4.6	-19.5	12.7	40.2	14.8	3.2	
LP4178-9	5.2	-19.4	13.0	39.4	14.5	3.2	
LP4178-10	5.7	-19.5	13.4	40.2	14.8	3.2	
LP4178-11	6.2	-19.6	13.6	38.9	14.4	3.2	
LP4178-12	6.8	-19.6	13.5	39.8	14.6	3.2	
LP4178-13	7.3	-19.6	13.6	38.2	14.0	3.2	
LP4178-14	7.8	-19.5	13.9	40.2	14.7	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4178-15	8.4	-19.4	13.6	41.3	15.1	3.2	
LP4178-16	8.9	-19.6	13.8	40.3	14.8	3.2	
LP4178-17	9.5	-19.6	13.8	39.9	14.6	3.2	
LP4178-18	10.0	-19.9	14.1	38.6	14.1	3.2	

Table A2.44 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4178

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4250-1	0.3	-21.2	12.3	38.9	14.1	3.2	Complete
LP4250-2	1.0	-20.9	12.2	38.1	14.1	3.2	
LP4250-3	1.7	-20.9	12.0	36.7	13.6	3.1	
LP4250-4	2.4	-20.5	12.0	39.0	14.5	3.1	
LP4250-5	3.1	-20.4	11.4	39.2	14.5	3.2	
LP4250-6	3.8	-20.4	11.7	39.0	14.5	3.1	
LP4250-7	4.5	-20.3	11.6	38.6	14.3	3.2	
LP4250-8	5.2	-20.3	11.7	38.6	14.3	3.1	
LP4250-9	5.8	-20.4	11.6	37.8	13.9	3.2	
LP4250-10	6.5	-20.4	11.9	38.5	14.2	3.2	
LP4250-11	7.2	-20.6	12.3	38.4	14.1	3.2	
LP4250-12	7.9	-20.4	12.3	38.2	14.1	3.2	
LP4250-13	8.6	-20.3	11.2	39.2	14.3	3.2	
LP4250-14	9.3	-20.2	10.9	38.0	14.0	3.2	
LP4250-15	10.0	-20.5	11.0	38.7	14.1	3.2	

Table A2.45 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4250

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4603-1	0.3	-21.0	14.4	41.0	15.0	3.2	Complete
LP4603-2	0.9	-21.4	12.7	40.9	15.0	3.2	
LP4603-3	1.4	-21.2	13.1	41.1	15.1	3.2	
LP4603-4	2.0	-21.0	12.9	41.0	15.0	3.2	
LP4603-5	2.6	-20.8	12.6	41.0	15.0	3.2	
LP4603-6	3.2	-20.9	12.1	41.0	15.0	3.2	
LP4603-7	3.7	-20.8	12.2	40.9	14.9	3.2	
LP4603-8	4.3	-20.8	12.2	41.1	14.9	3.2	
LP4603-9	4.9	-20.7	12.2	40.9	14.9	3.2	
LP4603-10	5.4	-20.5	12.1	40.8	14.8	3.2	
LP4603-11	6.0	-20.4	12.1	40.6	14.8	3.2	
LP4603-12	6.6	-20.4	12.1	40.5	14.8	3.2	
LP4603-13	7.2	-20.4	12.0	41.2	15.0	3.2	
LP4603-14	7.7	-20.4	11.9	41.0	14.9	3.2	
LP4603-15	8.3	-20.6	11.7	40.9	14.9	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4603-16	8.9	-20.8	11.2	41.2	15.0	3.2	
LP4603-17	9.4	-20.8	11.6	41.2	14.9	3.2	
LP4603-18	10.0	-21.2	12.9	41.0	14.7	3.2	

Table A2.46 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4603

A.2.2.ii Females

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3687-1	10.0	-20.7	13.8	41.7	14.8	3.3	Complete
LP3687-2	9.4	-20.7	13.9	44.3	15.6	3.3	
LP3687-3	8.7	-20.9	13.8	41.7	14.8	3.3	
LP3687-4	8.1	-21.0	13.1	37.6	13.4	3.3	
LP3687-5	7.4	-20.8	12.6	36.6	12.7	3.4	
LP3687-6	6.8	-	-	-	-	-	
LP3687-7	6.1	-20.9	12.6	39.9	14.0	3.3	
LP3687-8	5.5	-20.7	12.7	42.5	15.1	3.3	
LP3687-9	4.8	-20.7	12.4	39.6	13.9	3.3	
LP3687-10	4.2	-20.7	12.4	43.3	15.3	3.3	
LP3687-11	3.5	-20.9	12.6	42.2	14.9	3.3	
LP3687-12	2.9	-21.0	12.7	41.6	14.7	3.3	
LP3687-13	2.2	-20.5	12.8	41.9	14.8	3.3	
LP3687-14	1.6	-20.7	13.0	43.3	15.4	3.3	
LP3687-15	0.9	-20.3	14.4	40.7	14.2	3.3	
LP3687-16	0.3	-20.0	14.0	33.6	11.7	3.4	

Table A2.47 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3687

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3745-1	0.3	-20.0	16.0	41.4	15.3	3.2	Complete
LP3745-2	0.9	-20.3	14.5	41.9	15.5	3.2	
LP3745-3	1.4	-20.6	13.4	40.6	14.8	3.2	
LP3745-4	2.0	-20.5	12.4	41.4	15.2	3.2	
LP3745-5	2.6	-20.7	12.2	41.5	15.0	3.2	
LP3745-6	3.2	-20.7	12.2	41.7	15.2	3.2	
LP3745-7	3.7	-20.6	12.2	41.8	15.3	3.2	
LP3745-8	4.3	-20.6	12.1	39.9	14.5	3.2	
LP3745-9	4.9	-20.6	11.7	40.1	14.5	3.2	
LP3745-10	5.4	-20.5	11.7	40.1	14.5	3.2	
LP3745-11	6.0	-20.5	11.8	38.4	13.6	3.3	
LP3745-12	6.6	-20.4	12.2	40.1	14.3	3.3	
LP3745-13	7.2	-20.8	12.2	39.9	13.6	3.4	
LP3745-14	7.7	-20.3	12.0	41.4	14.6	3.3	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3745-15	8.3	-20.6	11.8	41.3	14.4	3.3	
LP3745-16	8.9	-20.4	11.8	40.2	14.4	3.2	
LP3745-17	9.4	-20.6	12.1	41.2	15.0	3.2	
LP3745-18	10.0	-20.6	12.1	41.5	15.0	3.2	

Table A2.48 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3745

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3749-1	0.3	-20.9	14.1	35.4	13.1	3.1	Complete
LP3749-2	1.0	-20.8	13.6	39.3	14.5	3.2	
LP3749-3	1.7	-20.6	13.1	38.5	14.2	3.2	
LP3749-4	2.4	-20.5	12.7	39.5	14.6	3.2	
LP3749-5	3.1	-20.5	12.2	38.0	14.0	3.2	
LP3749-6	3.8	-20.5	12.0	39.5	14.5	3.2	
LP3749-7	4.5	-20.7	12.0	39.5	14.6	3.2	
LP3749-8	5.2	-20.7	12.1	39.6	14.6	3.2	
LP3749-9	5.8	-20.7	12.6	37.9	13.9	3.2	
LP3749-10	6.5	-20.8	12.8	38.3	14.1	3.2	
LP3749-11	7.2	-20.9	12.6	37.0	13.7	3.2	
LP3749-12	7.9	-20.8	12.6	37.6	13.9	3.2	
LP3749-13	8.6	-20.6	12.4	40.3	14.8	3.2	
LP3749-14	9.3	-20.6	12.9	39.6	14.5	3.2	
LP3749-15	10.0	-20.7	13.0	39.1	14.5	3.1	

Table A2.49 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3749

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP3819-1	3.5	-	-	-	-	-	Rt only
LP3819-2	4.6	-20.3	11.2	38.3	14.1	3.2	
LP3819-3	5.7	-20.1	12.1	38.9	14.4	3.2	
LP3819-4	6.4	-20.0	12.3	38.0	13.7	3.2	
LP3819-5	7.1	-20.3	12.6	38.5	13.9	3.2	
LP3819-6	7.8	-20.5	13.0	38.5	13.9	3.2	
LP3819-7	8.6	-20.3	12.8	37.7	13.7	3.2	
LP3819-8	9.3	-20.2	12.5	38.9	14.0	3.2	
LP3819-9	10.0	-20.3	12.6	38.6	14.0	3.2	

Table A2.50 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP3819

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4035-1	0.3	-21.4	10.6	40.4	14.5	3.2	Complete
LP4035-2	1.0	-21.0	11.6	40.9	15.0	3.2	
LP4035-3	1.6	-21.0	12.0	41.6	15.1	3.2	
LP4035-4	2.3	-20.7	12.1	40.8	14.9	3.2	
LP4035-5	2.9	-20.5	11.7	41.7	15.1	3.2	
LP4035-6	3.6	-20.3	11.9	41.4	15.0	3.2	
LP4035-7	4.2	-20.4	12.1	41.4	14.4	3.3	
LP4035-8	4.9	-20.3	11.9	41.7	14.6	3.3	
LP4035-9	5.6	-20.3	11.9	41.0	14.8	3.2	
LP4035-10	6.2	-20.4	11.9	37.3	13.6	3.2	
LP4035-11	6.9	-20.3	12.0	40.0	14.6	3.2	
LP4035-12	7.5	-20.3	11.9	40.9	14.9	3.2	
LP4035-13/14/15	8.8	-20.2	11.8	39.1	14.2	3.2	

Table A2.51 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4035

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4047-1	10.0	-20.3	10.7	37.0	13.3	3.3	Complete
LP4047-2	8.8	-20.5	10.4	30.3	10.6	3.3	
LP4047-3	8.5	-20.6	10.3	36.1	13.0	3.2	
LP4047-4	7.9	-20.5	10.2	35.5	12.6	3.3	
LP4047-5	7.6	-20.5	10.2	37.9	13.5	3.3	
LP4047-6	6.4	-20.3	10.6	37.8	13.6	3.2	
LP4047-7	5.1	-20.5	10.4	40.6	14.6	3.2	
LP4047-8	4.3	-20.5	10.4	39.9	14.4	3.2	
LP4047-9	3.5	-20.6	10.3	38.2	13.6	3.3	
LP4047-10	2.7	-20.7	10.3	39.3	14.1	3.2	
LP4047-11	1.9	-20.8	10.6	40.7	14.7	3.2	
LP4047-12	1.1	-20.7	11.5	38.4	13.5	3.3	
LP4047-13	0.3	-20.1	12.6	40.2	14.5	3.2	

Table A2.52 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4047

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4063-1	0.3	-19.9	15.4	42.7	15.5	3.2	Complete
LP4063-2	1.0	-20.9	14.5	42.7	15.5	3.2	
LP4063-3	1.6	-20.9	13.6	42.5	15.5	3.2	
LP4063-4	2.3	-21.1	13.5	43.2	15.8	3.2	
LP4063-5	2.9	-21.0	12.7	41.8	15.3	3.2	
LP4063-6	3.6	-21.0	12.7	42.1	15.4	3.2	
LP4063-7	4.2	-20.9	12.4	41.9	15.4	3.2	
LP4063-8	4.9	-20.8	12.2	42.0	15.4	3.2	
LP4063-9	5.6	-20.9	12.1	42.6	15.7	3.2	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4063-10	6.2	-20.9	12.1	40.6	15.0	3.2	
LP4063-11	6.9	-20.8	11.9	42.5	15.7	3.1	
LP4063-12	7.5	-20.8	11.6	42.2	15.3	3.2	
LP4063-13	8.2	-20.8	11.8	38.8	14.2	3.2	
LP4063-14	8.8	-20.5	11.7	41.2	15.1	3.2	
LP4063-15	9.5	-21.1	11.5	41.0	14.2	3.4	

Table A2.53 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4063

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4067-1	0.3	-20.5	14.5	39.1	14.2	3.2	Complete
LP4067-2	1.0	-20.7	12.2	39.5	14.4	3.2	
LP4067-3	1.8	-20.7	11.3	37.4	13.7	3.2	
LP4067-4	2.5	-20.9	10.8	38.4	14.0	3.2	
LP4067-5	3.3	-20.7	10.8	37.8	13.8	3.2	
LP4067-6	4.0	-20.7	10.5	38.9	14.2	3.2	
LP4067-7	4.8	-20.6	10.5	37.7	13.8	3.2	
LP4067-8	5.5	-20.7	10.5	38.4	14.0	3.2	
LP4067-9	6.3	-20.5	10.5	37.6	13.7	3.2	
LP4067-10	7.0	-20.5	10.3	38.7	14.1	3.2	
LP4067-11	7.8	-20.5	10.4	35.7	13.1	3.2	
LP4067-12	8.5	-20.4	10.7	38.0	13.8	3.2	
LP4067-13	9.3	-20.6	11.0	38.6	14.0	3.2	
LP4067-14	10.0	-20.5	11.2	41.6	15.1	3.2	

Table A2.54 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4067

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4075-1	10.0	-20.0	10.7	38.4	13.4	3.4	Complete
LP4075-2	9.4	-20.0	10.6	42.5	14.9	3.3	
LP4075-3	8.7	-20.0	10.6	41.5	14.5	3.3	
LP4075-4	8.1	-19.8	10.3	40.3	14.1	3.3	
LP4075-5	7.4	-20.0	10.4	40.6	14.4	3.3	
LP4075-6	6.8	-19.8	10.4	41.0	14.4	3.3	
LP4075-7	6.1	-19.8	10.4	41.8	14.7	3.3	
LP4075-8	5.5	-20.0	10.4	42.3	15.0	3.3	
LP4075-9	4.8	-20.0	10.4	41.5	14.7	3.3	
LP4075-10	4.2	-19.9	10.5	41.9	14.7	3.3	
LP4075-11	3.5	-19.9	10.4	41.7	14.8	3.3	
LP4075-12	2.9	-20.1	10.4	42.3	14.9	3.3	
LP4075-13	2.2	-20.1	10.6	41.6	14.7	3.3	
LP4075-14	1.6	-20.4	10.8	42.7	15.1	3.3	

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4075-15	0.9	-20.5	10.9	42.5	15.1	3.3	
LP4075-16	0.3	-20.5	11.9	43.1	15.4	3.3	

Table A2.55 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4075

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4139-1	0.3	-21.0	13.5	40.8	14.8	3.2	Complete
LP4139-2	1.0	-21.2	11.1	40.9	14.9	3.2	
LP4139-3	1.8	-21.0	10.4	40.8	14.9	3.2	
LP4139-4	2.5	-20.7	10.3	40.9	14.9	3.2	
LP4139-5	3.3	-20.3	10.2	40.9	14.9	3.2	
LP4139-6	4.0	-20.0	9.8	41.0	14.9	3.2	
LP4139-7	4.8	-20.0	9.8	40.9	14.8	3.2	
LP4139-8	5.5	-20.1	9.9	40.2	14.5	3.2	
LP4139-9	6.3	-20.0	9.9	36.6	13.1	3.3	
LP4139-10	7.0	-20.2	10.2	39.4	14.0	3.3	
LP4139-11	7.8	-20.0	10.1	41.0	14.6	3.3	
LP4139-12	8.5	-20.2	10.4	40.9	14.7	3.2	
LP4139-13	9.3	-20.3	10.9	40.5	14.5	3.2	
LP4139-14	10.0	-20.6	11.2	40.5	14.6	3.2	

Table A2.56 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4139

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4395-1	0.3	-20.4	15.0	38.5	14.2	3.2	Complete
LP4395-2	0.9	-20.6	14.1	39.1	14.4	3.2	
LP4395-3	1.6	-20.4	13.5	38.5	14.2	3.2	
LP4395-4	2.2	-20.5	12.5	39.1	14.3	3.2	
LP4395-5	2.9	-20.4	12.0	39.2	14.3	3.2	
LP4395-6	3.5	-20.3	12.0	38.8	14.2	3.2	
LP4395-7	4.2	-20.4	12.0	38.5	14.1	3.2	
LP4395-8	4.8	-20.3	12.2	39.3	14.4	3.2	
LP4395-9	5.5	-20.2	12.1	38.7	14.1	3.2	
LP4395-10	6.1	-20.1	11.8	37.9	13.9	3.2	
LP4395-11	6.8	-20.1	12.1	38.4	14.1	3.2	
LP4395-12	7.4	-20.4	12.2	38.8	14.3	3.2	
LP4395-13	8.1	-20.3	12.2	37.2	13.7	3.2	
LP4395-14	8.7	-20.4	12.3	37.7	13.9	3.2	
LP4395-15	9.4	-20.6	12.1	39.5	14.5	3.2	
LP4395-16	10.0	-20.6	12.3	33.8	12.3	3.2	

Table A2.57 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4395

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4585-1	0.3	-21.2	14.3	39.7	14.3	3.2	Complete
LP4585-2	1.0	-21.1	11.8	37.5	13.7	3.2	
LP4585-3	1.7	-20.9	11.4	38.9	14.2	3.2	
LP4585-4	2.4	-20.6	11.4	38.4	14.1	3.2	
LP4585-5	3.1	-20.3	11.3	38.6	14.1	3.2	
LP4585-6	3.8	-20.3	10.9	36.5	13.4	3.2	
LP4585-7	4.5	-20.1	10.8	38.9	14.2	3.2	
LP4585-8	5.2	-20.0	10.8	38.8	14.2	3.2	
LP4585-9	5.8	-20.1	10.9	38.4	14.0	3.2	
LP4585-10	6.5	-20.1	10.9	38.1	13.9	3.2	
LP4585-11	7.2	-20.3	11.1	38.5	14.1	3.2	
LP4585-12	7.9	-20.5	11.5	36.7	13.4	3.2	
LP4585-13	8.6	-20.5	11.8	38.5	14.1	3.2	
LP4585-14	9.3	-20.7	12.1	34.1	12.5	3.2	
LP4585-15	10.0	-21.0	12.4	36.9	13.4	3.2	

Table A2.58 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP 4585

A.2.2.iii Indeterminate

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N	Completion
LP4556-1	0.3	-20.8	11.6	42.4	15.5	3.2	Complete
LP4556-2	1.0	-20.9	10.0	42.9	15.7	3.2	
LP4556-3	1.6	-20.8	9.7	42.4	15.4	3.2	
LP4556-4	2.3	-20.8	9.9	42.4	15.5	3.2	
LP4556-5	2.9	-20.6	10.2	43.1	15.6	3.2	
LP4556-6	3.6	-20.5	10.5	43.5	15.8	3.2	
LP4556-7	4.2	-20.3	10.5	43.3	15.7	3.2	
LP4556-8	4.9	-20.3	10.9	42.5	15.4	3.2	
LP4556-9	5.6	-20.3	11.0	42.5	15.4	3.2	
LP4556-10	6.2	-20.4	10.8	43.1	15.6	3.2	
LP4556-11	6.9	-20.4	10.7	42.7	15.4	3.2	
LP4556-12	7.5	-20.6	11.1	41.9	15.2	3.2	
LP4556-13	8.2	-20.9	11.7	41.6	14.8	3.3	
LP4556-14	8.8	-20.8	12.3	40.8	14.6	3.3	
LP4556-15	9.5	-20.9	12.8	42.3	15.2	3.2	

Table A2.59 Isotope data, collagen quality indicators, and dental development stage for incrementally-sampled M1 dentine from LP4556

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