Chapter 4
What Doesn't Kill You: Early Life Health and Nutrition in Early Anglo-Saxon East Anglia

Ellen J. Kendall, Andrew Millard, Julia Beaumont, Rebecca Gowland, Marise Gorton, and Andrew Gledhill

Abstract Early life is associated with high vulnerability to morbidity and mortality - risks which can be reduced in infancy and early childhood through strategically high levels of parental or alloparental investment, particularly in the case of maternal breastfeeding. Recent evidence has supported links between early-life health and care patterns and long-term population health. This growing body of research regarding the broader impacts of infant-parent interactions transcends a traditional partitioning of research into discrete life stages. It also highlights implications of childhood data for our understanding of population health and behaviour. Skeletal and environmental data indicate that the 5-7th century cemeteries at Littleport and Edix Hill (Barrington A), Cambridgeshire represent populations of similar material culture but contrasting environments and health. The high prevalence of skeletal stress markers at Littleport indicates a community coping with unusual levels of biological stress, potentially a consequence of endemic malaria present in the marshy Fen environs. In contrast, Edix Hill was an inland site which exhibited lower skeletal stress marker prevalence comparable to wider British data for the early medieval period. Early life patterns relating to diet and physiological stress at Littleport (n=5) and Edix Hill (n=8) were investigated through analyses of carbon and nitrogen stable isotopes from incrementally-sampled deciduous dentine. Meaningful variation in isotopic values within and between populations was observed, and should be a focus of future interdisciplinary archaeological childhood studies.

4.1 Introduction

Population breastfeeding and weaning patterns have been a topic of interest to bioarchaeologists for several decades due to their relevance to demographic factors, such as subsistence, fertility, and child mortality patterns (Sellen and Smay, 2001; Schurr and Powell, 2005). Consequently, biogeochemical analyses (particularly those of carbon ($\delta^{13}C$) and nitrogen ($\delta^{15}N$) stable isotopes) of skeletal tissue have become commonplace in palaeodietary research as a method of discerning infant feeding trends. Additionally, recent epidemiological studies have contributed to a greater understanding of the potential for early life stress and malnutrition to create negative impacts on long-term health. The Developmental Origins of

E. Kendall (✉) • A. Millard • R. Gowland
Department of Archaeology, Durham University
Durham, UK
e.j.kendall@durham.ac.uk

J. Beaumont • M. Gorton • A. Gledhill
School of Archaeological Sciences, University of Bradford
Bradford, UK

S. Halcrow and R. Gowland (eds.), *The Mother-Infant Nexus in Anthropology: Small beginnings, significant outcomes*, DOI [TBA]

Health and Disease (DOHaD), or Barker hypothesis, has identified foetal and infant epigenetic "programming" in response to environmental stress – particularly undernutrition and reduced growth – as an important driver of phenotypic plasticity that significantly increases morbidity risk in later life (Barker *et al.*, 2002; Barker, 2004). The potential negative impacts of such early life stress on future resilience may range from increases in morbidity and mortality arising from inadequate immune response, to the long-term development of chronic metabolic or cardiovascular disease (Wadhwa *et al.*, 2009). Thus, the significance of early life nutrition is not limited to indications of parental resource allocation or the short-term impacts of child mortality, but also creates heavy implications for adult population health. As the Barker hypothesis has received broad acceptance within epidemiological thought, its implications have begun to inform bioarchaeological interpretation (Armelagos *et al.*, 2009; Gowland, 2015; Agarwal, 2016).

Over the past several decades, infant palaeodietary studies have largely exploited a cross-sectional population approach, using bone sampled from women and children of varying ages. Modern studies analysing the hair and fingernails of mother-infant pairs have established that breastfeeding produces a shift of +2-4‰ for $\delta^{15}N$ and a smaller shift of approximately +1‰ for $\delta^{13}C$ in the keratin of exclusively breastfed infants, relative to their mothers (Fuller *et al.*, 2006). These values decrease during the weaning process as the transition from an exclusive diet of human milk to a diet composed wholly of family foods is made. For bone or tooth dentine-derived collagen, the enrichment in isotope values is assumed to be similar in magnitude to those reflected in keratin, with allowances for the tissue offsets between collagen and keratin (cf. O' Connell and Hedges, 1999; O' Connell *et al.*, 2001). As bone collagen is subject to turnover, cross-sectional analyses produce a single, highly time-averaged measurement per individual. For cross-sectional studies, bone collagen values for children are plotted by age against a female mean bone collagen value ($\pm 1\sigma$), and norms for completion of the weaning process are defined as occurring at or around the age where the values of most children fall within the female range.

The assumptions underlying cross-sectional method have been subject to scrutiny and critique in recent years, particularly in terms of the "osteological paradox" (Wood *et al.*, 1992). This has led some researchers to question the suitability of sampling definitively frail juveniles as representative of wider care norms, due to mortality bias, and women of uncertain maternity status as maternal proxies (Jay *et al.*, 2008; Beaumont *et al.*, 2015). In addition to these concerns, the synchronic nature of bone collagen data cannot provide essential temporal context with which to distinguish between anomalous $\delta^{13}C$ or $\delta^{15}N$ values arising from dietary versus physiological shifts. Studies of modern hair and fingernails have demonstrated that while anabolic states experienced during pregnancy produce a slight depression of $\delta^{15}N$ values (Fuller *et al.*, 2004), catabolic states experienced during periods of nutritional stress and undernutrition produce decreases in $\delta^{13}C$ alongside increases in $\delta^{15}N$ values (Fuller *et al.*, 2005; Mekota *et al.*, 2006). The potential for physiologically-altered single isotope measurements, particularly those taken from potentially stressed individuals in cross-sectional studies, to be interpreted as representative of dietary states has thus presented an obstacle to exploring early childhood diet.

In contrast to the interpretive limitations of cross-sectional methodology, the emergence of incremental sampling methods for tooth dentine have allowed for production of high-resolution diachronic data series. The complementary development of longitudinal palaeodietary method and epidemiological theory has allowed increasing numbers of studies to use $\delta^{13}C$ and $\delta^{15}N$ analyses to address the twin issues of diet and health in childhood. In some cases differences in patterning between individuals surviving beyond childhood, and

individuals who did not, have been identified (Henderson *et al.*, 2014; Sandberg *et al.*, 2014; Beaumont *et al.*, 2015; Beaumont and Montgomery, 2016). Primary dentine develops within well-defined timelines regardless of nutritional status, and does not remodel over the course of the lifespan (Beaumont *et al.*, 2013; Elamin and Liversidge, 2013). Sampling this tissue while following the direction of development, therefore, yields an opportunity to interpret isotopic variability in light of individual timelines, rather than as a function of relative values within their community. Within this research context, non-surviving children need not be assumed to reflect a normative range of dietary practices and nutritive states, nor sampled to infer these states in the wider population. Analyses of deciduous dentine, do however, provide a unique opportunity to characterise diet and early life health patterns for the most vulnerable members of past societies, and offer the potential to examine links between early nutritional stress and mortality risk.

The present study investigates the effects of environmental pressures and subsequent health risks on the duration and pattern of early life stress and dietary patterns in non-surviving children through $\delta^{13}C$ and $\delta^{15}N$ analysis of incrementally-sampled deciduous dentine. Two contemporary early Anglo-Saxon (fifth to sixth c. AD) Cambridgeshire cemetery populations sharing geographic proximity (c. 45 km apart) were selected for analysis, representing dissimilar environments and resources.

4.2 Materials and Methods

Prior to drainage efforts during the 17th to 19th centuries, large portions of the Cambridgeshire landscape consisted of marshland known as the Fens. The Fens were the largest area of resource-rich natural wetland in England, comprising highly biodiverse low-lying areas of saline and freshwater fen dotted by islands of higher elevation. The marshes of eastern England, including the Fens, were known to be afflicted by endemic malaria ("ague") – believed to be temperate *Plasmodium vivax* – from at least the post-medieval period. Fen ague contributed to significantly higher rates of child mortality and population morbidity noted by contemporary chroniclers (Dobson, 1997; Nicholls, 2000; Kendall, 2014). *P. vivax* malaria, unlike the more notoriously virulent tropical *P. falciparum*, produces chronic infection resulting in relapsing disease. *P. vivax* has been demonstrated to lead to chronic malnutrition and episodes of haemolytic anaemia in children (Williams *et al.*, 1997; Douglas *et al.*, 2012; Monteiro *et al.*, 2016). Development of cribra orbitalia, a porous cranial lesion of the superior orbit representing expansion of the marrow cavity, is theorised to result from chronic megaloblastic or haemolytic anaemia (Walker *et al.*, 2009). Increased prevalence of cribra orbitalia in early medieval Fenland skeletal samples, relative to contemporaneous non-Fen sites, may indicate that malaria was present in the Fens during the Anglo-Saxon period as well (Gowland and Western, 2012). Historically, communities from uplands surrounding the Fens enjoyed much better health than their Fen and Fen-edge counterparts. This contributed to a widespread belief of the intrinsically unhealthy nature of the Fenland environment (Dobson, 1997; Rippon, 2009).

Early medieval Littleport was a Fen island community which would have faced significant threats to health from sanitation challenges associated with a high water table, in addition to the likely presence of malaria. Edix Hill (Barrington A) was an upland site located southwest of modern Cambridge which lacked many of the health risks of Littleport, and exhibited a far lower prevalence of skeletal markers considered to indicate stress in early life (Table 4.1). While use of the cemetery at Edix Hill spanned the fifth to seventh centuries, clear phasing during the early period of use allowed for selection of individuals buried during the fifth to sixth centuries. It is hypothesised that variability of breastfeeding duration and incidence of

isotopic shifts associated with stress would have been greater at Littleport than at Edix Hill, in recognition of this elevated health risk and more variable quality of health at the former site.

Dentine was sampled from second deciduous molars (DM2), which begin forming around the 30th week in utero, with apex completion at 3.5 ± 0.5 years of age (Al Qahtani *et al.*, 2010; Beaumont and Montgomery, 2015). Thirteen non-survivor individuals were included in the study, with a single tooth sampled from each. Table 4.2 summarises the study sample. Deciduous tooth data was compared with M1 tooth data (Kendall *et al.*, in prep) from twenty adult individuals to create a measure of comparison between early life isotopic patterning based on survivorship. All selected teeth were free of caries, and surface debris was removed using an abrasive dental burr in advance of enamel removal and longitudinal sectioning. Teeth were prepared using Method 2 of Beaumont *et al.* (2013), with demineralisation taking place in advance of transverse sectioning. Increments were assigned an approximate age using the method of Beaumont and Montgomery (2015), with the first increment of crown dentine known to form during the late foetal period (Wahono, 2017).

Collagen was prepared using a modified Longin method (Longin, 1971). Samples were demineralised in 0.5 M hydrochloric acid at 4°C. The demineralised sections were rinsed with deionised water and placed in sealed microtubes in a pH3 hydrochloric acid solution at 70°C for 24 to 48 hours. First increments were lyophilised without gelatinisation, in order to increase yield, per the method of Beaumont *et al.* (2014), with subsequent increments being gelatinised. The gelatinised samples were centrifuged and decanted for lyophilisation without filtration, to prevent loss of sample, with any insoluble sample discarded. The resulting gelatin was weighed into tin capsules and measured in duplicate at the University of Bradford Stable Light Isotope Laboratory via CFIRMS, using a Thermo Flash EA 1112 coupled to a Finnigan DeltaPlus XL isotope ratio mass spectrometer with a Conflo III interface. Where yields of collagen were insufficient for the minimum sample size of the mass spectrometer, (within smaller samples such as root increments) collagen from neighbouring increments was combined to produce a sufficient sample. For these combined measurements, age estimation was recalculated, resulting in greater time-averaging. Collagen samples were interspersed within each run alongside blanks, internal standards and international standards. International standards measured were: IAEA 600, CH3, CH6, N1, and N2. Internal reference materials (calibrated against the international standards) analysed were: fish gelatine, bovine liver, and methionine. Analytical error for both carbon and nitrogen was ± 0.2‰ or better (1σ).

4.3 Results

C:N ratios obtained from the dentine collagen samples fell within acceptable ranges recommended by Ambrose (1993) and van Klinken (1999). Yields from dentine fell well above the >1% of weight set as a threshold of acceptability by van Klinken (1999), indicating well-preserved collagen. Results for incremental collagen at Littleport and Edix Hill are notated as a delta value (δ) expressed in parts per thousand (per mil, or ‰), relative to international standards.

4.3.1 Littleport

Full results for Littleport dentine collagen data are presented in Table 4.3. Figure 4.1 presents the incremental dentine data profiles for non-survivor individuals at Littleport in graphical form. Both absolute $\delta^{13}C$ and $\delta^{15}N$ values and the pattern of change within profiles varied widely between individuals. Dentine collagen $\delta^{13}C$ values of the sample fell within a range of −21.6 to −19.8‰, while $\delta^{15}N$ values ranged from 9 to 16.2‰. Intra-individual variation in

$\delta^{15}N$ values ranged from 1.5-3.5‰ over the course of tooth formation, while a smaller within-profile variation of ~1‰ was observed for $\delta^{13}C$ data.

4.3.2 Edix Hill

Table 4.4 presents the full results for Edix Hill DM2 dentine collagen data. Incremental dentine data profiles for non-survivor individuals at Edix Hill are presented graphically in Figure 4.2. Variability of $\delta^{13}C$ and $\delta^{15}N$ values was less marked than at Littleport, particularly in terms of intra-individual variability. Dentine collagen $\delta^{13}C$ values of the sample fell within a range of −21.1 to −19.6‰, while $\delta^{15}N$ values ranged from 10.6 to 15.5‰. Intra-individual variation ranged from 0.3-1.1‰ for $\delta^{13}C$ and 0.6-3.1‰ for $\delta^{15}N$ over the course of DM2 tooth development at Edix Hill.

4.3.3 Antenatal values

The $\delta^{13}C$ and $\delta^{15}N$ values of crown dentine providing the first sampled increment in the second deciduous molars are believed to reflect the in utero-environment experienced during foetal life. Thus, they may act as a proxy for maternal diet or metabolic stress experienced by mother or child during pregnancy. Figure 4.3 shows non-survivor DM2 dentine first increment $\delta^{13}C$ and $\delta^{15}N$ values for both sites.

4.3.4 Non-survivors and survivors

In addition to individual-level analyses of isotope variation to observe patterns of dietary change and stress, data was examined at a population-level to identify meaningful patterns among non-survivors. Figure 4.4 compares site variability among non-surviving children sites with data sampled from individuals surviving to adulthood (n=20, adult data from Kendall *et al.*, in prep). Within both non-survivor and survivor cohorts, overall variability was greater at Littleport than at Edix Hill, which exhibited a more constrained range of values. The absolute value of these ranges also differed, with Edix Hill demonstrating broadly higher $\delta^{13}C$ values and lower $\delta^{15}N$ than their counterparts at Littleport. In terms of differences between survivor and non-survivor patterning, non-survivors showed greater variability in values than their surviving counterparts, which was particularly marked in terms of differences in $\delta^{13}C$ variability at Edix Hill. Non-surviving children at Edix Hill were also more likely than their non-surviving counterparts at Littleport to exhibit a flattened $\delta^{15}N$ profile.

4.4 Discussion

Data from both sites overlap with ranges produced by adult bone collagen analyses in local and time-comparable assemblages. While no previous comparative $\delta^{13}C$ and $\delta^{15}N$ data exists for Littleport, the dentine data produced here compares favourably with palaeodietary analyses at the nearby 7th century site at Westfield Farm, Ely (n=15), which 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). Our data for Edix Hill also broadly overlap the range of bone collagen values previously observed for Anglo-Saxon inland sites in Britain, as well as 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). 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 bone and tooth data and the

effects of breastfeeding. Conversely, these higher values, alongside a greater range of variability in dentine $\delta^{13}C$ values in our data, may implicate malnutrition in some individuals.

4.4.1 Profile variability at Littleport

All isotope profiles at Littleport failed to present 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. Four out of the five individuals presented isotopic profiles suggesting episodes of undernutrition in the first few years of life, with three of these occurring within the timeline represented by the first few increments of dentine. In the profiles of LP 3770, LP 4116, and LP 4144 (Figure 4.1 a,b,c), shifts in $\delta^{13}C$ and $\delta^{15}N$ were non-parallel and discordant, producing "bubbles" of high or increasing $\delta^{15}N$ alongside decreasing $\delta^{13}C$ values. In the data of 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, 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 is suggestive of catabolic states experienced in utero or in infancy, but is not consistent with the expected profile for breastfeeding, based on modern data. In terms of skeletal evidence of stress in these individuals, LP 3770 had no documented skeletal pathology, while LP 4116 exhibited cribra orbitalia and LP 4144 was observed to have new woven bone on the ribs.

Only two individuals at Littleport, neither of whom displayed visible skeletal pathology, produced data suggestive of simple dietary shifts. In the profile of LP 4848 (Figure 4.1 e), $\delta^{13}C$ 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 flattened profile by approximately four months of age indicates that if this individual was breastfed, 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 4.1 d) followed a primarily 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 (Butte, 2000; Fuller *et al.*, 2004).

4.4.2 Isotopic patterning at Edix Hill

In contrast to the erratic profile patterning observed in the Littleport sample, profile variation within the Edix Hill sample was more constrained. With respect to individual-level variability, three primary patterns were observed in the isotope profiles: profiles suggesting dietary change compatible with an interpretation of breastfeeding, "flat" profiles with low levels of variability throughout, and a profile exhibiting episodes of nutritional stress.

Profiles compatible with an interpretation of breastfeeding were identified in three individuals. The first of these, EH 178 (Figure 4.2 c), did not exhibit visible skeletal pathology and following stasis in 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 4.2 e), 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 value-depressing "pregnancy effect" observed for $\delta^{15}N$ 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 4.2 f) 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 over this period.

Four individuals produced data with flattened profiles exhibiting little variability. EH 12 (Figure 4.2 a), an individual exhibiting no skeletal pathology, but showing a discrepancy between estimated skeletal aging and stage of root resorption suggesting skeletal stunting, had a largely flattened profile with little variability overall. However, a drop of 0.3‰ in $\delta^{13}C$ alongside a small rise of 0.1‰ in $\delta^{15}N$ occurring between the penultimate and final increments may suggest that an episode of nutritional stress occurred around the middle of the third year of life. A second individual, EH 133 (Figure 4.2 b) 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 0.6‰ between the approximate ages of birth and one year of age. Individual EH 587 (Figure 4.2 g) displayed no pathology aside from a vertebral anomaly, and had a flattened profile until the approximate age of 18 months, following which the profile suggests two separate stress events of minor isotopic magnitude. EH 679 (Figure 4.2 h) was the final individual with a flattened, low variability in profile. This individual did not display skeletal pathologies, 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, EH 352 (Figure 4.2 d) exhibited non-parallel changes in $\delta^{13}C$ and $\delta^{15}N$ values in later 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. Following this period, $\delta^{15}N$ values began to decline alongside $\delta^{13}C$ values, indicating probable 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.

4.4.3 Inferring maternal health

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 thirteen 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 one (EH 352) 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. Factors such as consumption of freshwater fish may have contributed to this relative shift 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.

Three further individuals from the Littleport sample (LP 4116, LP 4144, LP 4494) produced first incremental dentine data with high $\delta^{15}N$ and low $\delta^{13}C$ values (range 14.4-14.6‰ $\delta^{15}N$ 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 or foetal malnutrition during late pregnancy. It is intriguing that both LP 4116 and LP 4144 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, four out of five antenatal 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 to lead to 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 our 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 endemicity, reinforces the merit of exploring this as a future area of larger-scale investigations.

4.4.4 Survivorship and variability

The relationship expressed in the present dataset between survivorship, physiological stress, and diet appears to require significant levels of nuance to interpret. However, the relationship

between $\delta^{15}N$ and $\delta^{13}C$ profiles provides an indispensable frame of reference for assessing the nature of changes taking place over time in each individual sampled. In terms of infant feeding practices, most children surviving beyond early infancy during the early medieval period were very probably breastfed, due to the risks of artificial feeding at this point in history. However, it is clear from the presence of flattened profiles in both surviving and non-surviving individuals in the present study that a simple interpretation of diet is unconvincing. Reynard and Tuross (2014) questioned the assumption of a "breastfeeding effect" on the scale of full trophic level, arguing that existing evidence suggests that human milk $\delta^{15}N$ and $\delta^{13}C$ may be lower than previously assumed, and well below the value of maternal collagen. If this is the case, then elevation of $\delta^{15}N$ and $\delta^{13}C$ occurring as a result of metabolic fractionation in the breastfed child may be of a smaller magnitude than previously assumed. In the case of a child transitioning onto a solid diet resembling that of the mother, a longitudinally flat isotopic profile may be the result, as the diet-tissue spacing between milk or solid diet and collagen may be similar. While some profiles in the present study display a synchronous harmony of $\delta^{15}N$ and $\delta^{13}C$, which strongly suggests the role of breastfeeding, we cannot assume that flattened non-survivor or survivor profiles indicate the absence of breastfeeding, and thus it is difficult to ascertain the prevalence or patterning of breastfeeding behaviours on a population level. Littleport survivors also present challenges to interpretation, with strong elements of physiological stress exhibited in most profiles. In those Edix Hill individuals who do display profiles consistent with breastfeeding, the complementary phase of feeding appears to have been initiated by the middle of the first year, with the weaning process being completed by two years of age.

Several papers have attempted to address the potential for selective mortality to confound attempts to reconstruct early life diet at a population level, through direct comparison of rib collagen and dentine, or more recently, survivor and non-survivor incremental dentine profiles (Fuller *et al.*, 2003; Sandberg *et al.*, 2014; Beaumont *et al.*, 2015). The latter study by Beaumont *et al.* (2015) explicitly attempted to examine survivor/non-survivor differences during the Irish Famine, and address assumptions regarding the purely dietary source of isotopic shifts which implicitly underpin childhood palaeodietary research. Of relevance to the present study, Beaumont *et al.* (2015) identified a pattern of universally flattened profiles among survivors, alongside more variable profiles within the non-surviving cohort. They argued that a simplistic interpretation of non-survivor variability as representing dietary shifts was challenged by the total absence of expected "weaning curves" present within the survivor cohort, and that knowledge of non-dietary physiological processes, particularly their effects on opposing covariance of carbon and nitrogen profiles, must be applied to accurately interpreting data trends. Greater variability observed in the present study between survivor and non-survivor cohorts, as well as between sites exhibiting differential degrees of skeletal evidence for systemic stress processes, mirrors the trend toward non-survivor variability displayed by Beaumont *et al.* (2015)'s Irish famine victims. It is particularly striking that at Littleport, regardless of survivorship, overall variability of range at a population level was higher, suggesting real differences in either dietary variability or physiological stress. Within the survivor cohort, however, conformity to a trend of flattened profiles was by no means universal at either site, with many exhibiting a classic weaning curve with synchronised covariance of both carbon and nitrogen suggestive of bona fide dietary shifts.

Overall, the data strongly reinforce the association between early life stress and isotopic variability during this period. The correlation between non-survivor status and isotopic variability, and particularly between variability and non-survivorship at a site with theorised environmental risks, reinforces the caution with which sampling of non-surviving juveniles for palaeodietary reconstruction should be approached. This relationship between high levels

of early life stress and non-survivorship, as expressed in $\delta^{15}N$ and $\delta^{13}C$, may simply reflect ongoing environmental and selective pressures experienced by all children during this period, expressed at the higher sampling resolution achievable in deciduous teeth. Or, alternately, considering the Barker hypothesis, the increased prevalence of stress exhibited by data derived from early-forming dentine may represent a refutation of the adage that "what doesn't kill you, makes you stronger." Nutritional stress, particularly when experienced in utero, may have predisposed non-surviving children to frailty, reduced immune function, and finally to premature death.

4.5 Conclusion

Our data aligns well with recent isotope research confirming the validity of concerns surrounding the "osteological paradox" and the sampling of non-surviving juveniles for reconstruction of palaeodietary norms. The weight of evidence regarding differences in isotopic patterning between surviving and non-surviving individuals and the importance of understanding the diachronic context of isotopic shifts should suffice to discourage cross-sectional studies of bone for this purpose. Similarly, this diachronic context is necessary to allow researchers to disentangle evidence for parental investment through infant feeding practices from effects of severe or chronic illness, which may skew values and obscure evidence of care. The interpretation of isotopic patterning in children is significantly more complex than has been assumed in past study.

The support provided for the putative association of early life stress with isotopic variability in non-survivors at these sites will hopefully spur further research to explore these themes more fully. The ability of early-forming dentine to act as a proxy for maternal health and diet is promising, as a means of characterising an otherwise ephemeral and predominantly archaeologically-invisible portion of the lifecourse. Future work should additionally revisit the topic of antenatal health as a potential predictor of childhood mortality. Sampling on a more generous scale, using large comparative cohorts of survivors and non-survivors, could be undertaken to illuminate distinctive differences in isotopic patterning which align with bioarchaeological evidence for differences in health and status. While non-survivors are unlikely to be reliably representative of general population health and diet, they may act as the "canary in the mine" within a population, representing the extremes of risk and vulnerability. Finally, in terms of their equal humanity, the non-survivors have a right for their stories to be told, alongside those of their more unexceptional peers.

Acknowledgements

Funding for this study was provided by The Society for the Study of Human Biology, the Durham University Institute of Medieval and Early Modern Studies, and by the Rosemary Cramp Fund, which is gratefully acknowledged. The authors would also like to thank Gaynor Western and Archaeological Project Services, Heckington for access to skeletal material and sampling permissions for Littleport, and the Cambridgeshire Historic Environment Team for access and sampling permissions for Edix Hill. Further thanks are due to Steven Robertson, Beth Upex, Amanda Hayton, Martin West, and Alison George for technical support.

References Cited

Agarwal, S. C. 2016. Bone morphologies and histories: Life course approaches in bioarchaeology. *American Journal of Physical Anthropology,* 159, pp. 130-149.

Al Qahtani, S. J., Hector, M. P. and Liversidge, H. M. 2010. Brief communication: The London atlas of human tooth development and eruption. *American Journal of Physical Anthropology,* 142(3), pp. 481-90.

Ambrose, S. H. 1993. Isotopic Analysis of Paleodiets: Methodological and Interpretive Considerations. *In:* Sandford, M. K. (ed.) *Investigations of Ancient Human Tissue: Chemical Analyses in Anthropology.* Langhorne: Gordon and Breach, pp. 59-130.

Anstey, N. M., Russell, B., Yeo, T. W. and Price, R. N. 2009. The pathophysiology of vivax malaria. *Trends in Parasitology,* 25, pp.

Armelagos, G. J., Goodman, A. H., Harper, K. N. and Blakey, M. L. 2009. Enamel hypoplasia and early mortality: Bioarcheological support for the Barker hypothesis. *Evolutionary Anthropology: Issues, News, and Reviews,* 18(6), pp. 261-271.

Barker, D., Eriksson, J., Forsén, T. and Osmond, C. 2002. Fetal origins of adult disease: strength of effects and biological basis. *International Journal of Epidemiology,* 31(6), pp. 1235-1239.

Barker, D. 2004. Developmental origins of adult health and disease. *Journal of Epidemiology and Community Health,* 58(2), pp. 114-115.

Beaumont, J., Gledhill, A., Lee-Thorp, J. and Montgomery, J. 2013. Childhood Diet: A Closer Examination of the Evidence from Dental Tissues Using Stable Isotope Analysis of Incremental Human Dentine. *Archaeometry,* 55(2), pp. 277-295.

Beaumont, J., Gledhill, A. and Montgomery, J. 2014. Isotope analysis of incremental human dentine: towards higher temporal resolution. *Bulletin of the International Association for Paleodontology,* 8(2), pp. 212-223.

Beaumont, J. and Montgomery, J. 2015. Oral histories: a simple method of assigning chronological age to isotopic values from human dentine collagen. *Annals of Human Biology,* 42(4), pp. 407-414.

Beaumont, J., Montgomery, J., Buckberry, J. and Jay, M. 2015. Infant mortality and isotopic complexity: New approaches to stress, maternal health, and weaning. *American Journal of Physical Anthropology,* 157(3), pp. 441-457.

Beaumont, J. and Montgomery, J. 2016. The Great Irish Famine: Identifying Starvation in the Tissues of Victims Using Stable Isotope Analysis of Bone and Incremental Dentine Collagen. *PLoS ONE,* 11(8), pp. e0160065.

Boel, M., Rijken, M., Brabin, B., Nosten, F. and Mcgready, R. 2012. The epidemiology of postpartum malaria: a systematic review. *Malaria Journal,* 11(1), pp. 114.

Butte, N. F. 2000. Carbohydrate and lipid metabolism in pregnancy: normal compared with gestational diabetes mellitus. *The American Journal of Clinical Nutrition,* 71(5), pp. 1256s-1261s.

Diagne, N., Rogier, C., Sokhna, C. S., Tall, A., Fontenille, D., Roussilhon, C., Spiegel, A. and Trape, J.-F. 2000. Increased Susceptibility to Malaria during the Early Postpartum Period. *New England Journal of Medicine,* 343(9), pp. 598-603.

Dobson, M. J. 1997. *Contours of death and disease in early modern England,* Cambridge: Cambridge University Press.

Douglas, N. M., Anstey, N. M., Buffet, P. A., Poespoprodjo, J. R., Yeo, T. W., White, N. J. and Price, R. N. 2012. The anaemia of Plasmodium vivax malaria. *Malaria Journal,* 11(1), pp. 135.

Duhig, C. 1998. The human skeletal material. *In:* Malim, T. and Hines, J. (eds.) *The Anglo-Saxon Cemetery at Edix Hill (Barrington A), Cambridgeshire: excavations 1989-1991 and a summary catalogue of material from 19th century interventions CBA Research Report 112.* York: Council for British Archaeology, pp. 154-199.

Elamin, F. and Liversidge, H. M. 2013. Malnutrition Has No Effect on the Timing of Human Tooth Formation. *PLoS ONE,* 8(8), pp. e72274.

Fuller, B. T., Richards, M. P. and Mays, S. A. 2003. Stable carbon and nitrogen isotope variations in tooth dentine serial sections from Wharram Percy. *Journal of Archaeological Science,* 30(12), pp. 1673-1684.

Fuller, B. T., Fuller, J. L., Sage, N. E., Harris, D. A., O' Connell, T. C. and Hedges, R. E. M. 2004. Nitrogen balance and $\delta^{15}N$: why you're not what you eat during pregnancy. *Rapid Communications in Mass Spectrometry,* 18(23), pp. 2889-2896.

Fuller, B. T., Fuller, J. L., Sage, N. E., Harris, D. A., O' Connell, T. C. and Hedges, R. E. M. 2005. Nitrogen balance and $\delta^{15}N$: why you're not what you eat during nutritional stress. *Rapid Communications in Mass Spectrometry,* 19(18), pp. 2497-2506.

Fuller, B. T., Fuller, J. L., Harris, D. A. and Hedges, R. E. M. 2006. Detection of breastfeeding and weaning in modern human infants with carbon and nitrogen stable isotope ratios. *American Journal of Physical Anthropology,* 129(2), pp. 279-293.

Gowland, R. L. and Western, A. G. 2012. Morbidity in the marshes: using spatial epidemiology to investigate skeletal evidence for Malaria in Anglo-Saxon England (AD 410-1050). *American Journal of Physical Anthropology,* 147(2), pp. 301-11.

Gowland, R. L. 2015. Entangled lives: Implications of the developmental origins of health and disease hypothesis for bioarchaeology and the life course. *American Journal of Physical Anthropology,* 158(4), pp. 530-540.

Henderson, R. C., Lee-Thorp, J. and Loe, L. 2014. Early life histories of the London poor using $\delta^{13}C$ and $\delta^{15}N$ stable isotope incremental dentine sampling. *American Journal of Physical Anthropology,* 154(4), pp. 585-593.

Jay, M., Fuller, B. T., Richards, M. P., Knüsel, C. J. and King, S. S. 2008. Iron Age breastfeeding practices in Britain: Isotopic evidence from Wetwang Slack, East Yorkshire. *American Journal of Physical Anthropology,* 136(3), pp. 327-337.

Kendall, R. 2014. *Past Endemic Malaria and Adaptive Responses in the Fens and Marshlands of Eastern England.* Ph.D., Durham University.

Longin, R. 1971. New Method of Collagen Extraction for Radiocarbon Dating. *Nature,* 230(5291), pp. 241-242.

Lucy, S., Newman, R., Dodwell, N., Hills, C., Dekker, M., O'connell, T., Riddler, I. and Walton Rogers, P. 2009. The Burial of A Princess? The Later Seventh-Century Cemetery At Westfield Farm, Ely. *The Antiquaries Journal,* 89, pp. 81-141.

Mays, S. and Beavan, N. 2012. An investigation of diet in early Anglo-Saxon England using carbon and nitrogen stable isotope analysis of human bone collagen. *Journal of Archaeological Science,* 39(4), pp. 867-874.

Mekota, A.-M., Grupe, G., Ufer, S. and Cuntz, U. 2006. Serial analysis of stable nitrogen and carbon isotopes in hair: monitoring starvation and recovery phases of patients suffering from anorexia nervosa. *Rapid Communications in Mass Spectrometry,* 20(10), pp. 1604-1610.

Monteiro, W. M., Alexandre, M. A., Siqueira, A., Melo, G., Romero, G. a. S., D'ávila, E., Benzecry, S. G., Leite, H. P. and Lacerda, M. V. G. 2016. Could Plasmodium vivax malaria trigger malnutrition? Revisiting the Bradford Hill criteria to assess a causal relationship between two neglected problems. *Revista da Sociedade Brasileira de Medicina Tropical,* 49(3), pp. 274-278.

Nicholls, A. 2000. Fenland ague in the nineteenth century. *Medical History,* 44(4), pp. 513-530.

Nosten, F., Mcgready, R., Simpson, J. A., Thwai, K. L., Balkan, S., Cho, T., Hkirijaroen, L., Looareesuwan, S. and White, N. J. 1999. Effects of Plasmodium vivax malaria in pregnancy. *Lancet,* 354, pp.

O' Connell, T. C. and Hedges, R. E. M. 1999. Isotopic Comparison of Hair and Bone: Archaeological Analyses. *Journal of Archaeological Science,* 26(6), pp. 661-665.

O' Connell, T. C., Hedges, R. E. M., Healey, M. A. and Simpson, A. H. R. W. 2001. Isotopic Comparison of Hair, Nail and Bone: Modern Analyses. *Journal of Archaeological Science,* 28(11), pp. 1247-1255.

Price, R. N., Tjitra, E., Guerra, C. A., Yeung, S., White, N. J. and Anstey, N. M. 2007. Vivax malaria: neglected and not benign. *American Journal of Tropical Medicine and Hygiene* 77, pp.

Reynard, L. M. and Tuross, N. 2014. The known, the unknown and the unknowable: weaning times from archaeological bones using nitrogen isotope ratios. *Journal of Archaeological Science,* 53, pp. 618-625.

Rippon, S. 2009. 'Uncommonly rich and fertile' or 'not very salubrious'? The Perception and Value of Wetland Landscapes. *Landscapes,* 10(1), pp. 39-60.

Roberts, C. A. and Cox, M. 2003. *Health & disease in Britain : from prehistory to the present day,* Stroud: Sutton.

Sandberg, P. A., Sponheimer, M., Lee-Thorp, J. and Van Gerven, D. 2014. Intra-tooth stable isotope analysis of dentine: A step toward addressing selective mortality in the reconstruction of life history in the archaeological record. *American Journal of Physical Anthropology,* 155(2), pp. 281-293.

Schurr, M. R. and Powell, M. L. 2005. The role of changing childhood diets in the prehistoric evolution of food production: An isotopic assessment. *American Journal of Physical Anthropology,* 126(3), pp. 278-294.

Sellen, D. W. and Smay, D. B. 2001. Relationship between subsistence and age at weaning in "preindustrial" societies. *Human Nature,* 12(1), pp. 47-87.

Van Klinken, G. J. 1999. Bone Collagen Quality Indicators for Palaeodietary and Radiocarbon Measurements. *Journal of Archaeological Science,* 26(6), pp. 687-695.

Wadhwa, P. D., Buss, C., Entringer, S. and Swanson, J. M. 2009. Developmental Origins of Health and Disease: Brief History of the Approach and Current Focus on Epigenetic Mechanisms. *Seminars in Reproductive Medicine,* 27(5), pp. 358-368.

Wahono, N. 2017. *Deciduous teeth as a tool to record early life exposure of zinc nutrition.* PhD, Newcastle University.

Walker, P. L., Bathurst, R. R., Richman, R., Gjerdrum, T. and Andrushko, V. A. 2009. The causes of porotic hyperostosis and cribra orbitalia: A reappraisal of the iron-deficiency-anemia hypothesis. *American Journal of Physical Anthropology,* 139(2), pp. 109-125.

Western, G. 2008. Human Skeletal Remains Assessment Report and Catalogue. *In:* Holt, R. (ed.) *Archaeological Assessment Report on Land at Highfield Farm, Littleport, Cambridgeshire (LHF04) A.P.S. Report No. 120/07.* Heckington: Archaeological Project Services, pp. Appendix 14.

Whitty, C. J. M., Edmonds, S. and Mutabingwa, T. K. 2005. Malaria in pregnancy. *BJOG: An International Journal of Obstetrics & Gynaecology,* 112(9), pp. 1189-1195.

Williams, T. N., Maitland, K., Phelps, L., Bennett, S., Peto, T. E. A., Viji, J., Timothy, R., Clegg, J. B., Weatherall, D. J. and Bowden, D. K. 1997. *Plasmodium vivax*: a cause of malnutrition in young children. *QJM,* 90(12), pp. 751-757.

Wood, J. W., Milner, G. R., Harpending, H. C. and Weiss, K. M. 1992. The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples. *Current Anthropology,* 33(4), pp. 343-370.

Figures:

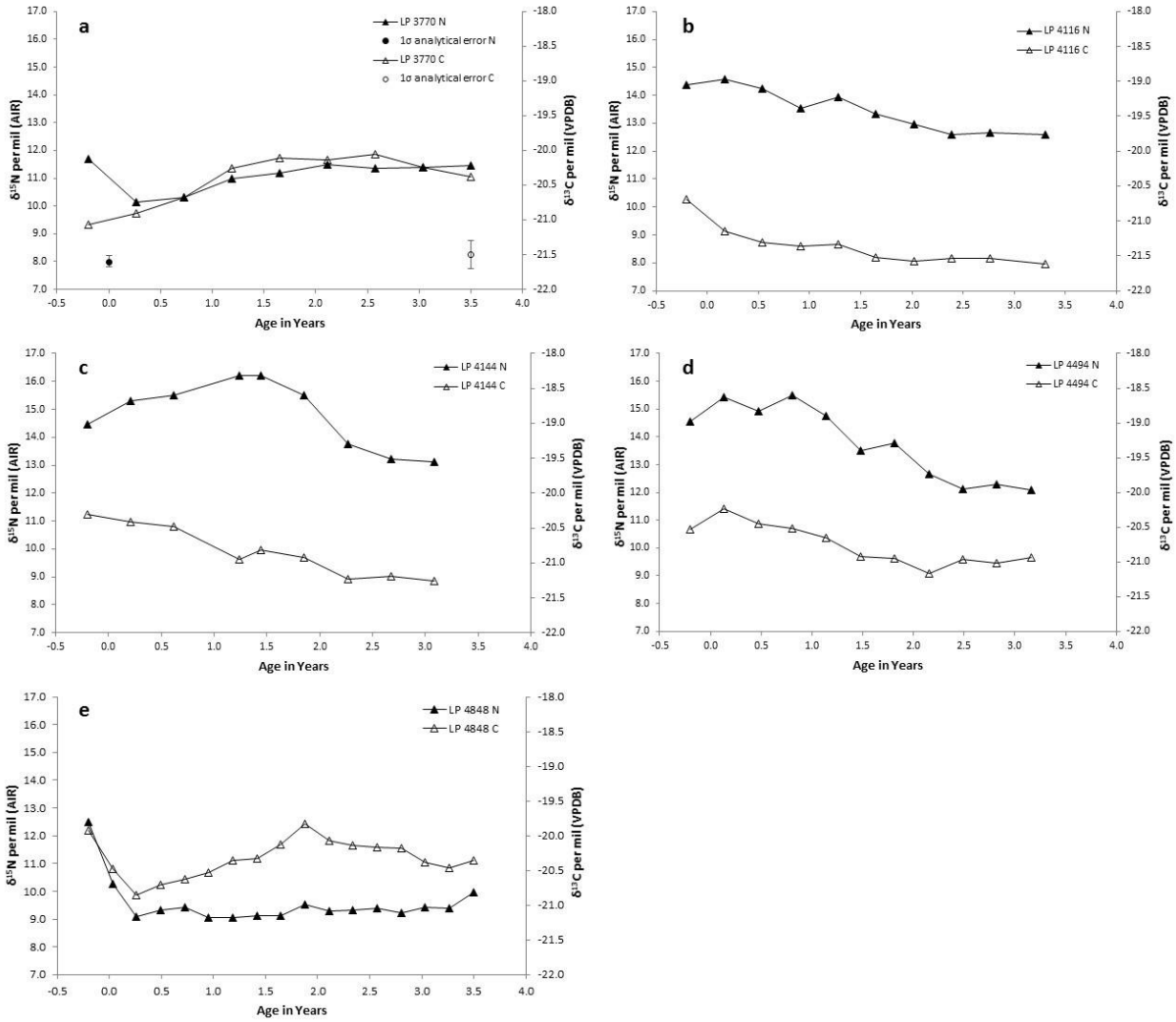

Figure 4.1 $\delta^{13}C$ and $\delta^{15}N$ values of DM2 incremental dentine collagen plotted against age for non-survivor individuals at Littleport. Plots from left to right: (a) LP 3770, (b) LP 4116, (c) LP 4144, (d) LP 4494, (e) LP 4848.

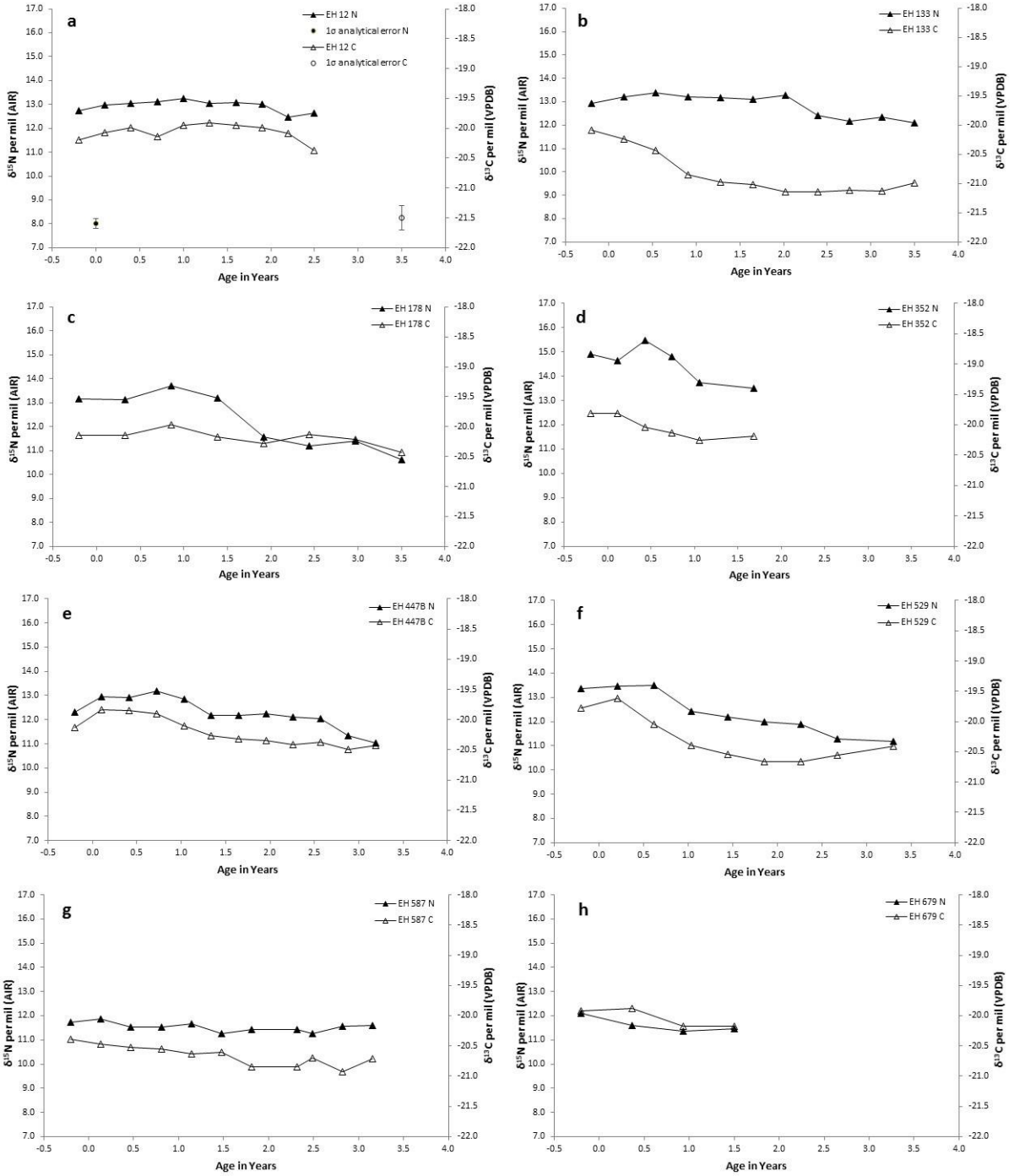

Figure 4.2 $\delta^{13}C$ and $\delta^{15}N$ values of DM2 incremental dentine collagen plotted against age for non-survivor individuals at Edix Hill. Plots from left to right: (a) EH 12, (b) EH 133, (c) EH 178, (d) EH 352, (e) EH 447B, (f) EH 529, (g) EH 587, (h) EH 679.

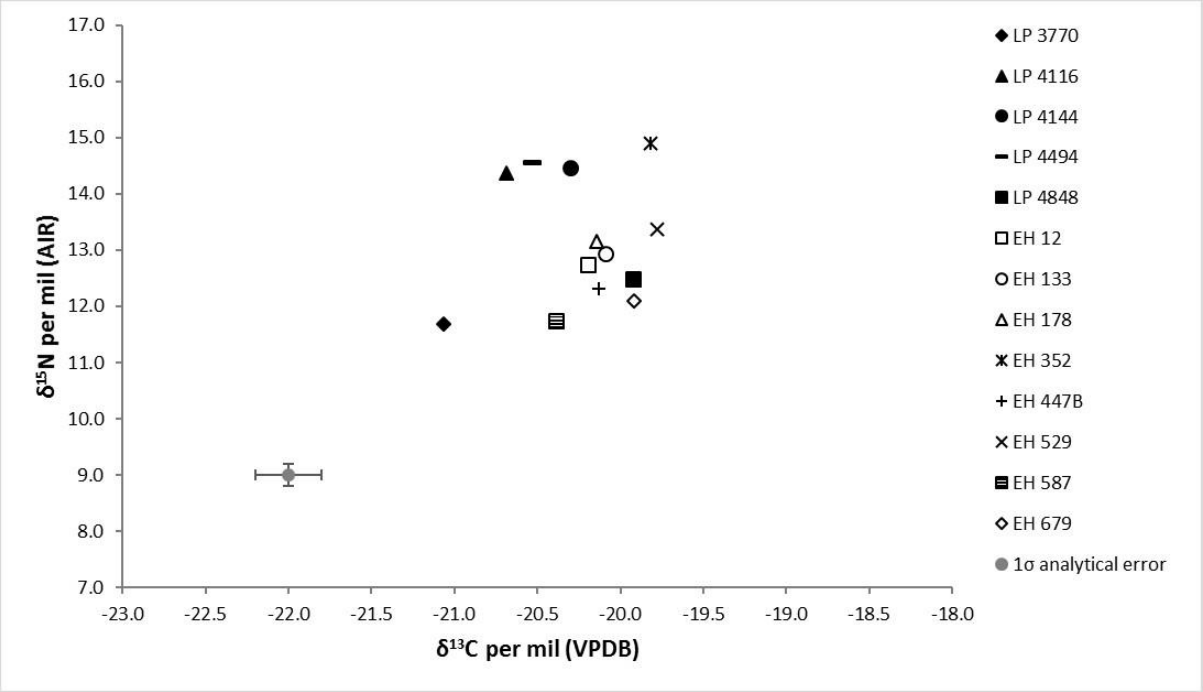

Figure 4.3 Plotted $\delta^{13}C$ and $\delta^{15}N$ values for DM2 dentine first increment data.

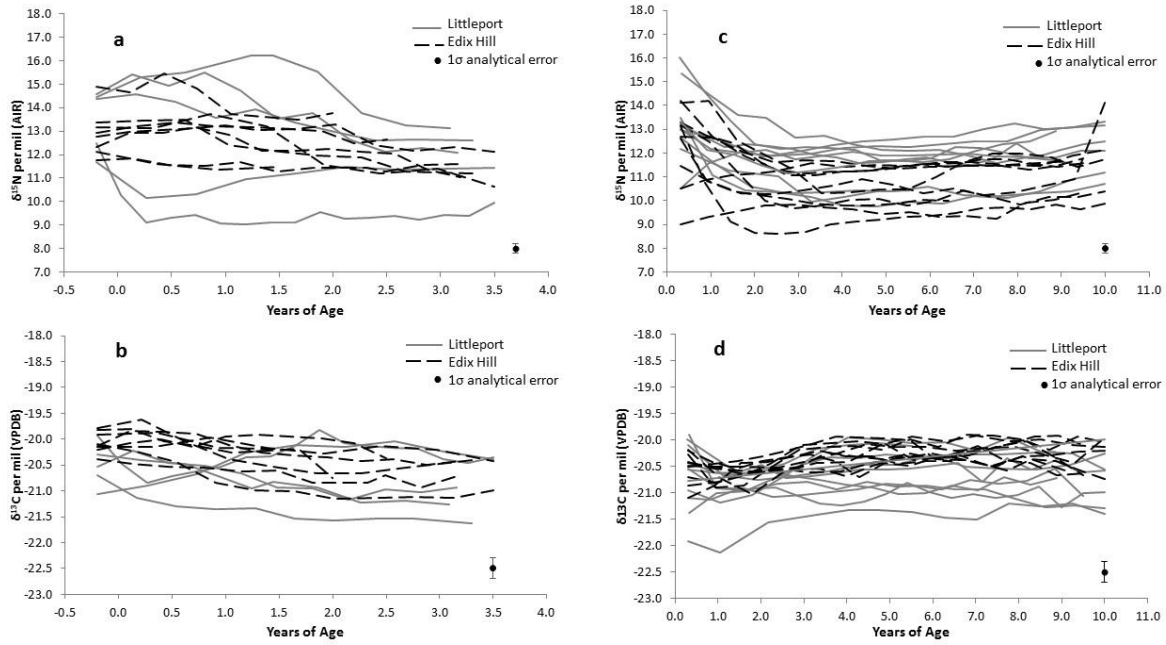

Figure 4.4 Incremental dentine collagen data plotted against age for (a) DM2 non-survivor $\delta^{15}N$, (b) DM2 non-survivor $\delta^{13}C$, (c) M1 survivor $\delta^{15}N$, and (d) M1 survivor $\delta^{13}C$ values (M1 data from Kendall *et al.*, in prep).

Tables:

Site	Enamel hypoplasia (%)	Cribra orbitalia (%)
Littleport	45.3	29.1

Edix Hill	10.1	10.8
Early Medieval Mean	18.8	7.6

Table 4.1 Prevalence of stress markers (reported as crude prevalence) for Littleport (Western, 2008), Edix Hill (Duhig, 1998), and early medieval mean (Roberts and Cox, 2003).

Skeleton	**Age (years)**	**Tooth**	**Pathology**	**References**
Littleport				Western (2008)
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				Duhig (1998)
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 disease, ECNB	
587	8	dm2	T13 vertebra	
679	1.5	dm2	None	

Table 4.2 Summary of sampled individuals (ECNB=extra cortical new bone, T13=13th thoracic vertebra).

Sample	**Age (years)**	**δ^{13}C ‰**	**δ^{15}N ‰**	**% C**	**% N**	**C:N**
LP 3770-1	−0.2	−21.1	11.7	39.5	13.7	3.4
LP 3770-2	0.3	−20.9	10.2	38.2	13.8	3.2
LP 3770-3	0.7	−20.7	10.3	40.0	14.6	3.2
LP 3770-4	1.2	−20.3	11.0	39.3	14.4	3.2
LP 3770-5	1.7	−20.1	11.2	39.8	14.5	3.2
LP 3770-6	2.1	−20.1	11.5	40.1	14.8	3.2
LP 3770-7	2.6	−20.0	11.4	40.1	14.7	3.2
LP 3770-8	3.0	−20.2	11.4	40.2	14.7	3.2
LP 3770-9	3.5	−20.4	11.4	40.0	14.3	3.3
LP 4116-1	−0.2	−20.7	14.4	38.5	14.2	3.2
LP 4116-2	0.2	−21.1	14.6	38.1	13.8	3.2
LP 4116-3	0.5	−21.3	14.2	38.1	14.0	3.2
LP 4116-4	0.9	−21.4	13.6	37.6	13.9	3.2
LP 4116-5	1.3	−21.3	13.9	34.5	12.8	3.2
LP 4116-6	1.7	−21.5	13.3	38.8	14.2	3.2
LP 4116-7	2.0	−21.6	13.0	38.6	14.1	3.2
LP 4116-8	2.4	−21.5	12.6	38.2	13.8	3.2
LP 4116-9	2.8	−21.5	12.7	38.5	14.0	3.2
LP 4116-10/11	3.3	−21.6	12.6	38.8	13.8	3.3
LP 4144-1	−0.2	−20.3	14.5	39.3	14.5	3.2
LP 4144-2	0.2	−20.4	15.3	40.1	14.8	3.2
LP 4144-3	0.6	−20.5	15.5	40.1	14.8	3.2
LP 4144-4/5	1.2	−20.9	16.2	40.1	14.7	3.2

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N
LP 4144-5	1.4	−20.8	16.2	40.1	14.8	3.2
LP 4144-6	1.9	−20.9	15.5	39.9	14.7	3.2
LP 4144-7	2.3	−21.2	13.8	40.0	14.8	3.2
LP 4144-8	2.7	−21.2	13.2	39.9	14.7	3.2
LP 4144-9	3.1	−21.3	13.1	40.2	14.8	3.2
LP 4494-1	−0.2	−20.5	14.6	45.6	16.8	3.2
LP 4494-2	0.1	−20.2	15.4	45.9	16.7	3.2
LP 4494-3	0.5	−20.5	14.9	44.8	16.4	3.2
LP 4494-4	0.8	−20.5	15.5	44.8	16.3	3.2
LP 4494-5	1.1	−20.7	14.7	44.8	16.4	3.2
LP 4494-6	1.5	−20.9	13.5	45.7	16.7	3.2
LP 4494-7	1.8	−20.9	13.8	45.8	16.8	3.2
LP 4494-8	2.2	−21.2	12.7	44.3	16.1	3.2
LP 4494-9	2.5	−21.0	12.1	44.9	16.4	3.2
LP 4494-10	2.8	−21.0	12.3	46.4	16.8	3.2
LP 4494-11	3.2	−20.9	12.1	48.5	17.6	3.2
LP 4848-1	−0.2	−19.9	12.5	46.7	17.1	3.2
LP 4848-2	0.0	−20.5	10.3	45.3	16.6	3.2
LP 4848-3	0.3	−20.8	9.1	46.1	16.8	3.2
LP 4848-4	0.5	−20.7	9.3	44.7	16.4	3.2
LP 4848-5	0.7	−20.6	9.4	45.7	16.7	3.2
LP 4848-6	1.0	−20.5	9.1	45.5	16.7	3.2
LP 4848-7	1.2	−20.4	9.0	46.1	16.3	3.3
LP 4848-8	1.4	−20.3	9.1	45.6	16.6	3.2
LP 4848-9	1.6	−20.1	9.1	45.8	16.8	3.2
LP 4848-10	1.9	−19.8	9.5	49.5	18.2	3.2
LP 4848-11	2.1	−20.1	9.3	45.9	16.9	3.2
LP 4848-12	2.3	−20.1	9.3	45.6	16.7	3.2
LP 4848-13	2.6	−20.2	9.4	44.4	16.3	3.2
LP 4848-14	2.8	−20.2	9.2	47.1	17.4	3.2
LP 4848-15	3.0	−20.4	9.4	45.4	16.6	3.2
LP 4848-16	3.3	−20.5	9.4	45.4	16.6	3.2
LP 4848-17	3.5	−20.4	10.0	49.5	17.9	3.2

Table 4.3 Incremental dentine collagen data and quality indicators for Littleport DM2 samples in this study.

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N
EH12 - 1	−0.2	−20.2	12.8	43.6	16.0	3.2
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
EH133 - 1	−0.2	−20.1	12.9	38.6	14.2	3.2

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N
EH133 - 2	0.2	−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	0.9	−20.8	13.2	38.6	14.2	3.2
EH133 - 5	1.3	−21.0	13.2	39.0	14.4	3.2
EH133 - 6	1.7	−21.0	13.1	39.2	14.4	3.2
EH133 - 7	2.0	−21.1	13.3	38.6	14.2	3.2
EH133 - 8	2.4	−21.1	12.4	39.1	14.4	3.2
EH133 - 9	2.8	−21.1	12.2	38.7	14.3	3.2
EH133 - 10	3.1	−21.1	12.3	39.1	14.4	3.2
EH133 - 11	3.5	−21.0	12.1	39.5	14.5	3.2
EH178 - 1	−0.2	−20.1	13.1	40.2	14.9	3.1
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
EH352 - 1	−0.2	−19.8	14.9	45.7	16.6	3.2
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 - 7	1.7	−20.2	13.5	30.5	11.0	3.2
EH447B - 1	−0.2	−20.1	12.3	39.4	14.3	3.2
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
EH529 - 1	−0.2	−19.8	13.4	40.1	14.6	3.2
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
EH587 - 1	−0.2	−20.4	11.7	40.5	15.0	3.2
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

Sample	Age (years)	$\delta^{13}C$ ‰	$\delta^{15}N$ ‰	% C	% N	C:N
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
EH587 - 11	3.2	−20.7	11.6	39.8	14.5	3.2
EH679 - 1	−0.2	−19.9	12.1	37.9	13.3	3.3
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 4.4 Incremental dentine collagen data and quality indicators for Edix Hill DM2 samples in this study.