Title: “Captain of all these men of death”: An integrated case study of tuberculosis in nineteenth century Otago, New Zealand.

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Abstract: The South Island of New Zealand saw several major waves of migration in the mid-nineteenth century, predominantly from Europe but also with an ethnically distinct Chinese presence. The rural community of Milton, Otago, was a settler community established primarily by immigrants from the United Kingdom in search of a better quality of life. However, these settlers faced unique challenges related to surviving in an isolated location with very little infrastructure compared to their origin populations. In 2016 excavation was undertaken at St. John's Burial Ground, Milton, with the object of using bioarchaeological methods to elucidate the lived experience of the first organized European settlement of this region, particularly in terms of health and disease. Here we present a case study of Burial 21 (B21), a male individual of known identity and a documented cause of death. We use biochemical and paleopathological methods to ground-truth his written history, which includes a period of invalidism due to tuberculosis, and discuss the implications of our findings for the community, provision of care, and quality of life in rural colonial New Zealand.

"*Yet the captain of all these men of death that came against him to take him away, was the consumption, for it was that that brought him down to the grave*." (Bunyan, 1690)

In the mid-late nineteenth century waves of immigrants travelled to New Zealand in search of new opportunities; most were European but the Chinese also formed a distinctive minority group (Mackay, 1992). Many of these immigrants settled in the Otago region, establishing towns, industries, and farms, and adapting to their new biological and cultural environments (Holland, 2013; Mackay, 1992). While there is a vast literature on some aspects of New Zealand settlement (e.g. goldfields history), historical sources often lack information on the lived experiences of women, children, the sick, and the disenfranchised (White, 2017). Archaeology is often the sole avenue of enquiry regarding these individuals, but here too there are gaps where individual voices and agency are hard to determine (Landon and Tumberg, 1996). Few aspects of colonial health have been examined archaeologically (Smith and Garland, 2012; White, 2017). Bioarchaeological research, the analysis of human skeletal remains from archaeological contexts, allows us to reconstruct individual narratives, something that is lacking through either historical or archaeological enquiry alone (Larsen, 2015).

In the last two decades there has been a movement away from published case-studies in bioarchaeology with an increasing awareness of the benefits of investigating assemblage-level trends in health and disease in the past (DeWitte and Stojanowki, 2015; Waldron, 2007; Yaussy et al., 2016). While case-studies can provide important information on unusual conditions (e.g. Prates et al., 2011; Suzuki, 1987), they do not produce statistically quantifiable data on changes in disease prevalence over time. However, it is important to remember in all bioarchaeological enquiry that each data-point in an assemblage represents an individual whose lived experience is equally as important as quantifiable population-level analyses. The bioarchaeology of care (BoC) model (Tilley and Oxenham, 2011; Tilley, 2015), is one means of exploring the functional impact of disability or disease in a single person and provides more nuanced information about past communities that is not visible through paleoepidemiological analysis alone. This approach integrates osteobiographical information with social theory to characterize the familial and/or community response to debilitating pathologies (Tilley and Oxenham, 2011; Vlok et al., 2017).

Here we explore the social fabric of European colonial communities in New Zealand by examining the life-history of a man of known identity and cause of death from nineteenth century Otago, New Zealand, with both documentary and skeletal evidence of tuberculosis. Tuberculosis, an infection which often has a prolonged disease course, can have a devastating impact on the quality of life of its host for months or years. The clinical impact of this condition means that its sufferers require some form of social support if they are to survive for an extended period of time. The case examined in this paper provides a unique opportunity to apply the BoC model as an interpretative framework. Osteobiographies are generally constructed from macroscopic observations of the bones and teeth of deceased individuals. However, an individual's life-course can be more thoroughly assessed by also examining the tissues that develop during growth and alter again as the body ages (Agarwal, 2016). This paper therefore includes chemical analyses of the diet and origins of this individual to build an integrated case study of this man's life, and death.

1.1 European Settlers in Nineteenth Century Otago

During the nineteenth century numerous diasporas saw millions of men, women and children leave the "Old World" to start fresh lives in the "New World". One small episode in this series of events was the establishment of the settlement of Otago in Southern New Zealand as a joint venture between the Lay Association of the Free Church of Scotland and the New Zealand Company, which purchased 144,600 acres of land in coastal Otago from Ngai Tahu (the local Māori tribe) in 1844. The first two ships carrying settlers, the *John Wickliffe* and the *Philip Laing*, arrived in March and April 1848, with 97 and 247 emigrants aboard,

respectively (Hocken, 1898:94; Olssen,1984:33). The new settlement, centered on Dunedin at the head of the Otago Harbor grew slowly, reaching just 2262 people in 1859. However, the discovery of gold in inland Central Otago sparked a series of gold rushes in 1861-62 accompanied by a massive influx of people to the province. By 1864 the population of Dunedin had reached 15,790 and the previously sparsely inhabited interior was scattered with small towns and villages linked by a rudimentary road and track system (McDonald 1965:44, 51). While most of the miners were men, some brought their wives and families with them, and storekeepers, hotel keepers, packers and barmaids all contributed to the mix. As the gold-rushes waned, some goldfields inhabitants chose to settle permanently as farmers or merchants. This study centers on the colonial settlement of Milton, a largely farming community established in 1850. It is located approximately equidistant between the Tuapeka goldfields (the site of the first major Otago gold rush in 1861) and Dunedin and provided agricultural products (e.g. wool, flour, and oatmeal) to both locations (Sumpter and Lewis, 1949). Milton was also an appealing location for former gold miners looking to settle in the region in the mid -1860s (Sumpter and Lewis, 1949).

Milton in the mid-1800s was still very much a frontier community with little infrastructure (Sumpter and Lewis, 1949). Although the first general medical practitioner arrived in 1856, the nearest hospitals were in Dunedin and Lawrence (from 1860), each of which involved a 50-kilometer journey over rough terrain. Victims of illness or accident were typically cared for by family in their own homes and community support would have been essential for long-term invalid care (Sumpter and Lewis, 1949). This paper aims to create a socially contextualized osteobiography of the life-course of a single individual from St. John's burial ground, Milton (B21).

1.2 Tuberculosis: pathogenesis and functional impact

Tuberculosis (TB) is a chronic bacterial infection caused by a complex of organisms within the genus *Mycobacteria* (MTB complex; Bos et al., 2014; Osoba, 2004; Wilbur and Buikstra, 2006). The transmission of tuberculosis is facilitated by close contact and poor sanitation (WHO, 2009). Although bioarchaeological evidence suggests that tuberculosis was present in New Zealand prior to European colonization, nineteenth century settlers appear to have brought with them a new form of the disease to which the indigenous Māori populations had no previous exposure (Buckley et al., 2010; Woodward and Blakely, 2014). The etiology of tuberculosis in New Zealand is the subject of ongoing and as yet unpublished paleopathological and biomolecular investigation. The question of the antiquity of tuberculosis, changing pathogen virulence, and host adaptation is important for understanding paleo- and historical epidemiology of TB in New Zealand, but is beyond the scope of this paper.

In the modern global context, the usual route of TB transmission is via droplet inhalation with the lungs serving as the site of initial infection and potential dissemination to other organs occurring after a period of latency (Madkour, 2004). The exception to this is *M. bovis*, which is transmitted through ingestion of infected meat or milk and disseminates from the intestines (Abter et al., 1995:78). Primary pulmonary infection generally occurs in childhood and is usually subclinical (Karakousis et al., 2017). The initial pulmonary infection initiates a strong cell-mediated immune response which usually results in the resolution of the disease into latency (Karakousis et al., 2017). Containment of MTB is accomplished by the formation of granulomas: a conglomeration of immune cells (primarily macrophages) that serves to contain an infection or foreign body that cannot be completely cleared through phagocytosis (Davies and Ramakrishnan, 2008; Philips and Ernst, 2012:364). In the majority of cases, MTB will never emerge from this latent state. However, in 5-10% of infected individuals the granulomatous sequestering response becomes compromised by decreased immunocompetence due to advancing age, comorbid infection, or malnutrition, and this results in active secondary infection (Cegielski and McMurray, 2004; Phillips and Ernst, 2012). Alternatively, re-infection from an exogenous source may also result in active secondary tuberculosis, although this is clinically less common (Madkour et al., 2004). The

majority of secondary tuberculosis infections will affect the lungs, resulting in the formation of additional granulomatous tissue with a necrotic center (Akhtar and Mana, 2004; Madkour et al., 2004). The formation of this necrotic center within granulomatous tissue is called "caseation" and is a hallmark of both pulmonary and extra-pulmonary secondary tuberculosis infection (Akhtar and Mana, 2004:154). The tissue necrosis caused by caseating granulomas has devastating effects on the lungs of the host. Secondary pulmonary tuberculosis ("phthisis" or "consumption") is associated with a productive (often bloody) cough, pleural effusion, and progressive respiratory failure (Madkour et al., 2004; Karakousis et al., 2017). Left untreated, disease resolution or death typically occurs within three years (Tiemersma et al., 2011). Without medical intervention mortality from secondary pulmonary infection is likely, with some epidemiological estimates as high as 83% (Tiemersma et al., 2011).

In ~15% of individuals reactivation of latent infection will result in dissemination of MTB from the lungs via the lymphatic or vascular systems (Shah and Chida, 2017). Virtually any organ within the body can be involved; however, because MTB requires an oxygen-rich environment for reproduction, regions plentiful in O_2 such as the trabecular bone of the vertebral bodies of the spine and synovial joints are preferentially affected (Moulding, 1994). Skeletal involvement is quite rare, occurring in only ~3-5% of cases (Jaffe, 1972; Resnick and Niwayama, 1995a). The mechanism of dissemination to osseous tissue is likely hematogenous rather than lymphatic as destructive lesions are most commonly found in highly vascularized regions of the skeleton (Jaffe, 1972; Resnick and Niwayama, 1995a). The spine is the most common site of skeletal lesions, followed by the hip (acetabulum and proximal femur), knee, wrist, and other synovial joints, although any skeletal element including the cranium may be affected (Resnick and Niwayama, 1995a; Tuli, 2016).

Although the individual experience of disease will vary, secondary pulmonary tuberculosis has a profound functional impact on the sufferer. The disease course of secondary tuberculosis can be prolonged, particularly if the infection disseminates outside the lungs. Skeletal tuberculosis in particular can result in slowly progressing destructive lesions within the spine or the joints of the limbs (Resnick and Niwayama, 1995a). Vertebral bodies may collapse, resulting in permanent deformation of the spine, and joint destruction can cause immobility from pain or the pathological fusion of bones (Resnick and Niwayama, 1995a; Tuli, 2016). The systemic pathogenesis of the disease means that the sufferer will be prone to a number of secondary negative effects, including increased susceptibility to other infections, malnutrition, and lethargy (see section 4.4).

2. Materials and Methods

2.1 The Site: St. John's Burial Ground, Milton

"*There is always a damp vapour arising, highly prejudicial to the health of the inmates, and especially the children*" – Milton Medical Officer, Dr. MacBean Stewart (1875)

The burial ground of the Anglican church of St. John (Archaeological Site H45/56) was established in 1860 to serve the growing farming community of Milton (formerly known as Tokomairiro) (Petchey et al., 2017; Figure 1). The majority of burials date to the 1860s and 1870s and the burial ground became disused after 1926. Only a handful of interments took place after 1900 because the focus of the community shifted to the west after the main road was rerouted, and the secular Fairfax Cemetery became the favored interment ground (Petchey et al., 2017). The Tokomairiro Project 60, a community group formed to preserve St. John's burial ground, has collected a substantial amount of historical documentation on the site and the individuals interred there, including a burial register. Death certificates with cause of death for 56 individuals have also been recovered and indicate a high burden of mortality due to tuberculosis (n = 11; 19.6%), accident (n = 6; 10.7%), and obstetric complications (n = 5; 8.9%). Respiratory diseases (e.g. pertussis, asthma, bronchitis,

pulmonary tuberculosis) account for approximately one-third of the recorded deaths and the Milton medical officer reported an overall mortality rate of 60% in 1875 (MacBean Stewart, 1875). The historical epidemiology of the St John's burial ground will be reported in detail in a forthcoming publication. However, as this town was in its infancy the detailed documentation regarding healthcare and general community health is limited.

In December of 2016 an excavation of St. John's Burial Ground was carried out by a joint team from the University of Otago departments of Archaeology and Anatomy (Petchey et al., 2017). The objectives of this excavation were to identify the boundaries of the burial ground and to conduct bioarchaeological analysis of the individuals disinterred. Twenty-five graves containing 27 individuals, including two double infant/child burials, were recovered. Sixteen of these graves were found outside of the existing fenced area. An additional three grave cuts were identified but were not excavated due to time constraints. The preservation of the burials from St John's was highly variable ranging from excellent to a complete absence of bone in most of the infant and young child graves. In the case of these non-adults only teeth and hair were present. Of the adults, only a few had skeletal preservation of a standard where detailed osteological observations of disease could be made. A full report on the bioarchaeology of all the individuals excavated from St John's is forthcoming. However, poor dental health in the form of caries, antemortem tooth loss, and periodontal disease was ubiquitous in the adults at the site. Antemortem trauma in the form of rib and limb fractures was common among the adults and a case of perimortem trauma, probably causing death was also observed. No other gross infectious pathology such as that suffered by B21 was found.

2.2 Burial 21

B21 was in a row of burials facing one of two parallel gravel paths that ran through the graveyard. The burial was within a wooden coffin of the traditional 'single break' style (i.e. wider at the shoulders and tapering to the head and feet). The coffin was 6 feet 5 inches (1.95m) long, of an average 'adult' size. It was wrapped in a black fabric, with embossed lead/tin alloy decorative metal strips around the edges of the lid and coffin sides. There were six plain cast iron coffin handles. These features were typical of many of the excavated burials at Milton. Notable for B21, however, was the presence of a legible coffin plate and evidence of a painted design on the coffin top fabric. Unfortunately, this design was too degraded to identify any detail, other than noting it was extensive and ornate. The coffin had evidently retained its integrity for some time after burial, as it had filled with water causing many skeletal elements to become disarticulated. Only later did the top collapse, stopping any further movement of bones.

B21 was identified from the name, date of death and age at death painted onto his partially preserved coffin plate. While his identity is known we will not be publishing his name. A death certificate, dated July 5th 1873, gives his occupation as "labourer" and his cause of death at the age of 42 years as "*Pneumonic phthisis haemorrhage* [sic]" (Figure 2). Historical records tell us that B21 was born in Mitcham, London in 1830 and initially emigrated to Hobart, Australia in 1856. He travelled to Otago in 1861 following the discovery of gold at Gabriel's Gully, Lawrence. His family followed him in 1862 and they settled in Helensbrook, near Milton (Findlay et al., 2015:69). He remained here until his death and was survived by his wife and their 11 children. B21 was a member of the Court Bruce of the Ancient Order of Foresters (AOF), one of the self-help friendly societies that existed in this period prior to any formal social welfare system. In the last 11 months of his life the AOF helped support him and his family, paid for his funeral, and then raised funds to help his widow pay off a remaining debt on the family house (Bruce Herald 11 February 1873; 10 October 1873). The role of the Ancient Order of Foresters is explored in more detail below.

The skeletal remains of B21 consisted of a well-represented (>75% of elements present) adult individual (Figure 3). Preservation was variable throughout the skeleton; the cortices showed little evidence of erosion and minimal warping. However, the metaphyses of

most long bones were fragmented or absent and the cranial vault had collapsed. All vertebrae were represented but the cervical, upper thoracic, and sacral regions were highly fragmentary. The os coxae were represented by iliae and ischia only.

Methods for assessing the life-course of B21.

2.3 Skeletal and dental analysis methods

Biological identity was estimated using standard methods outlined in Buikstra and Ubelaker (1994). Sex was assessed via cranial sexual dimorphism (Acsádi and Nemeskéri, 1970) and greater sciatic notch morphology (Walker in Buikstra and Ubelaker, 1994). Age was estimated primarily via the degenerative changes to the auricular surface (Lovejoy et al., 1985) with dental attrition (Smith, 1984) employed as a secondary method. Broad age-at-death categories (20-34 years, 35-49 years, and 50+ years) were employed to account for the large margin of error that accompanies adult age estimation methods. Stature was estimated via metric analysis of the left femur (measured in situ prior to lifting) (Totter and Gleser, 1958).

All surfaces of all skeletal elements were examined macroscopically for abnormalities of bony proliferation, destruction, density, size, and shape after Ortner (2003:49). Digital radiographs in antero-posterior and lateral views were taken of any elements that exhibited macroscopic pathology. Because both Otago and the United Kingdom are high-risk environments for vitamin D deficiency, dental radiographs (CDR Dicom, Sirona Dental Inc.) were obtained to screen for abnormalities in pulp-chamber morphology indicative of childhood rickets after D'Ortenzio (2018). Computed tomography (CT) scans in the sagittal and coronal planes (Siemens Somatom Emotion 16 slice scanner) were obtained of the left femur and the os coxae to further assess observed macroscopic lesions. The dentition of B21 was assessed for LEH and other lesions under fluorescent and incandescent lighting using (1.75X) magnification and a dental pick when necessary. All teeth were recorded using the Fédération Dentaire Internationale (FDI) system (Keiser-Nielsen, 1971).

2.4 Isotopic analysis of diet and origins

Isotopic proxies for place of origin and childhood and adult diet were used to both corroborate the historical record and add to our knowledge of B21's life experience. Strontium isotopes in dental enamel were used to establish where B21 lived during childhood (per Bentley, 2006; Montgomery, 2010; Montgomery et al., 2010), while carbon and nitrogen isotopes in B21's dentinal and bone collagen, and hair were studied to establish diet during life (per Ambrose and Norr, 1993; Lee-Thorp, 2008; Makarewicz and Sealy, 2015). Characteristic changes to carbon and nitrogen isotopic ratios associated with episodes of serious metabolic or nutritional stress were also explored (e.g. Beaumont and Montgomery, 2016; Fuller et al., 2005). Both hair and dentinal collagen was sampled incrementally to allow us to examine changes in diet throughout their formation time. This multi-isotope, multi-tissue approach has the potential to give more nuanced insight into the individual's life-course than from single tissues. In this case we were particularly interested in assessing whether his childhood diet differed from his adult diet. The preservation of his hair may also provide information on metabolic state in the months leading up to his death. Tissues sampled in this study and the time period they represent are given in Table 1.

Collagen samples were prepared using a modified Longin (1971) method, with incremental dentine analysis conducted according to Beaumont et al. (2013a), method 2. Both collagen and hair increments were analyzed using a Costech Elemental Analyzer (ECS 4010) connected to a Thermo Delta V Advantage isotope ratio mass spectrometer at the Stable Isotope Biogeochemistry Laboratory (Durham University). Detail on corrections and standards used is reported in Supplementary Table 1.

Strontium isotope analysis was conducted at the Northern Centre for Trace Element Analysis (Durham University) using a ThermoFisher Neptune Multi-Collector Inductively Coupled Plasma Mass Spectrometer (MC-ICP-MS). Prior to analysis dental enamel was mechanically cleaned of dentine and particulates using a dental drill and diamond burr. Strontium was purified prior to analysis using standard column chemistry methods (Charlier et al., 2006) involving sample dissolution in 3N HNO_3 and running of solution through Sr Spec resin. Sr ratios were normalized using repeated measurements of the NBS 987 standard ($^{87}Sr/^{86}Sr$ = 0.710240). Procedural blanks were analyzed alongside the sample to ensure lack of contamination.

Strontium isotope results were interpreted in light of baseline bioavailable/geological isotope work conducted in both New Zealand and the United Kingdom (Duxfield et al., in review; Evans et al., 2010). For visualization purposes hair isotope values were corrected for the known offset between keratin and collagen $\delta^{15}N$ and $\delta^{13}C$. The magnitude of this offset is, unfortunately, not well-understood and does not appear to be systematic. Some studies have shown that hair keratin $\delta^{15}N$ values are depleted by only 1‰ relative to bone collagen (e.g. O'Connell et al., 2001), while other controlled-feeding studies (DeNiro and Epstein, 1981; Tieszen and Fagre, 1993) and archaeological studies (e.g. Jørkov and Gröcke, 2017) estimate it to be between 2-3‰. In this study we use 2‰ as a conservative estimate of $\delta^{15}N$ offset and correct for this to compare bone and hair values. We follow O'Connell et al. (2001) and use an offset of 1.4‰ for $\delta^{13}C$ values.

3. Results

3.1 Skeletal analysis

The sex of B21 was assessed as a probable male and age-at-death was estimated to be middle adult (35-49 years), which is consistent with the death certificate associated with this burial. Stature was estimated to be 163.9cm ± 3.87cm, although it should be noted that as the in situ measurement of the left femur was an estimate only the margin of error is likely larger. Muscle attachment sites were extremely robust, particularly in the upper limb. Skeletal pathology and isotopic findings are outlined below.

3.1.1 Paleopathological analysis

3.1.1a Dental pathology

The dentition for B21 included a complete mandible missing two teeth post-mortem (33 and 47) and a partial maxilla that consisted of the alveolar bone and corresponding teeth from the posterior right (spanning teeth 18-15) quadrant and an almost complete left quadrant (spanning teeth 22-28). Three loose teeth from the anterior maxilla (13, 12, 11) were also recovered. The extent of tooth wear was relatively mild in the posterior dentition (Smith grades 2-3). The heaviest wear was present on the mesial half of the right mandibular canine and the distal half or the right lateral incisor and corresponding wear was observed on the right lateral maxillary incisor. The wear pattern on these teeth is indicative of the repetitive use of a clay pipe resulting in a classic 'pipe facet' (Fig 4a). The relatively mild wear of the posterior dentition for a middle-aged adult indicates B21's diet was reasonably soft and non-abrasive. The heavier wear of anterior teeth may be a result of the preferential use of these teeth for masticating food because of the loss of a number of the mandibular molars and two premolars (25 and 45). Antemortem tooth loss (AMTL) was observed for six teeth (23, 25, 36, 37, 45, 46). The alveolar bone around the sockets of these teeth had completely remodelled indicating the teeth were lost some time before B21's death. There was some mesial shifting of the mandibular third molars and right posterior maxillary teeth, suggesting some of the tooth loss may have occurred earlier in life.

A cementoenamel junction (CEJ) carious lesion (tooth 28), a pit and fissure carious lesion (tooth 38), two massive caries (teeth 24 and 48) and a possible interproximal carious lesion (tooth 12) were observed. Alveolar recession, possibly representing periodontal disease, was observed in the maxilla and the exposure of the tooth root was likely responsible for the observed CEJ caries on tooth 28. Linear enamel hypoplasia was observed on six anterior teeth (13, 12, 11, 43, 42, 32) and all but two of these teeth (12 and 11) displayed at least two hypoplastic lines (Fig 4a). The estimated age of the formation of the LEH was between 2-6 years old (Cares Henriquez and Oxenham, 2019).

The presence of enamel stones in the pulp chambers of the maxillary and mandibular molars (Figure 4b) precluded assessment of vitamin D deficiency-related changes here. Pulp stones are most likely to be found in molars, more often maxillary than mandibular. Etiology is unknown, but a number of different causes are mentioned in reviews, including long-standing irritation of the tooth pulp due to decay or excessive wear (Goga et al., 2008; Jannati et al., 2018; Vibhute et al. 2016), and systemic diseases, in particular cardiovascular disease (Bains et al., 2014; Edds et al., 2005; Khojastepour et al., 2013). There is also a single report linking hypervitaminosis D to pulp stone formation (Giunta, 1998).

3.1.1b Cranial pathology

The endocranial surface of the frontal and parietal bones exhibited several clusters of small, circular, lytic lesions along the sagittal sulcus and concentrated on the left and right margins of the parietals just posterior to the coronal suture. The affected regions also exhibited fine, clustered vascular impressions radiating out from the lytic foci. Radiography revealed multiple radiolucent foci on the endocranial frontal and anterior left and right parietals. Those clustered around the sagittal sulcus have the appearance of arachnoid granulations, a normal anatomical variation. However, there is a cluster of lesions on the right aspect along the meningeal groove which are larger and are more irregular in appearance. Two of these foci have partially coalesced and have blood vessel impressions (appreciated macroscopically) radiating outwards. There is a similar region of radiolucency on the corresponding left side. A single lytic focus with a “tunnel” appearance is visible within the trabeculae in the anterior one-third of the left parietal (Figure 5). This lesion is also macroscopically visible within a post-mortem break.

3.1.1c Post-cranial pathology

All skeletal elements are extremely light and porous, particularly in the axial region. Radiographs of a lower thoracic vertebra and the left and right metacarpals and metatarsals reveal extremely low trabecular density and paper-thin cortices (osteopenia). No lytic or proliferative lesions are present in the vertebral column but the preservation of most vertebral bodies was too poor for macroscopic analysis.

The left os coxa exhibited evidence of a long-standing destructive process in the acetabulum. Well-remodeled lytic activity is present on the superior 2/3rds of the of the margins of the acetabulum. Radiography revealed extensive underlying sclerosis in this region (Figure 6a). CT imaging revealed several lytic foci in the trabeculae of the ischium postero-medial to the acetabulum (Figure 6b). The right os coxa exhibits milder, remodeled lytic activity on the superior 1/4th of the margins of the acetabulum and radiographs revealed some sclerosis in this region but all changes are less extreme than on the left side. CT scans of the right os coxa did not reveal any additional lesions.

The left femur is complete but the distal 1/3rd is too fragmentary to record macroscopic pathology in this region. Multiple unremodeled lytic foci, ranging from ~3-6mm in diameter, are present macroscopically on the greater and lesser trochanters, both of which also exhibit patchy deposition of active subperiosteal new bone on the surfaces (Figure 7a). Radiography revealed that the surface lesions are representative of a much more extensive lytic process affecting the trabeculae of the head and trochanters (Figure

7b). CT imaging characterized these lesions in greater detail as multiple large ovoid foci in the greater and lesser trochanters, the largest of which is ~25x30mm, with extensive but less clearly demarcated lytic activity affecting the trabeculae of the head and neck (Figure 7c). The right femur did not exhibit any obvious pathology but the proximal 1/4th was incomplete and fragmentary.

The hands and feet were macroscopically unremarkable other than extremely thin cortices apparent in regions with post-mortem damage. However, radiographs revealed multiple pin-point radiodense foci within the trabeculae of the left and right carpals and metacarpals. In the right second and third metacarpals these pin-point radiodense foci affect both the trabeculae and the cortex. The first and second proximal phalanges of the right foot each exhibit a single radiodense focus within the trabeculae of the distal metaphyses. These have the appearance of enostoses (cortical bone present within the trabeculae). Enostoses are typically an asymptomatic, incidental clinical finding (Greenspan, 1995).

3.2 Isotopic analysis

Strontium isotope analysis of this individual ($^{87}Sr/^{86}Sr$ =0.70925) shows that they did not spend their childhood in Milton ($^{87}Sr/^{86}Sr$ of 0.70800-0.70900, Duxfield et al., in review). Their enamel strontium ratio is consistent with origins in the greater London area (0.709-0.710), where we know B21 resided prior to emigration to New Zealand (Figure 8).

Carbon and nitrogen isotope results from incrementally sampled dentine, bulk sampled bone and sectioned hair are given in Figure 9 and in full in Supplementary Table 1. Dentinal values gradually decline from the start of tooth formation, but by 5 years of age values have stabilized around 12‰ ($\delta^{15}N$) and -19.5‰ ($\delta^{13}C$). Bone collagen values, broadly-speaking representing adult diet (Fahy et al., 2017), are almost 2‰ lower than dentinal values. Finally, hair isotopic values (corrected for the hair-collagen offset) return to slightly higher/more positive $\delta^{13}C$ and $\delta^{15}N$ values closer to time of death.

4. Discussion

4.1: Differential Diagnosis: reconciling paleopathology with cause of death

B21 exhibits evidence of a systemic and primary osteolytic condition. Remodeling of the acetabuli suggests a long-standing process in play at least several months prior to death. Active lytic lesions in the left proximal femur, interior of the left ischium, and calvarium indicate that this process was ongoing at the time of death. The location of these lesions in trabecular-rich regions and at synovial joints suggests a hematogenous route of dissemination. There are several conditions, both infectious and non-infectious, that could result in a similar suite of skeletal lesions. These are summarized in Table 2 and briefly discussed below in the context of the death certificate and biographical details of this individual.

4.1a: Brucellosis

Brucellosis is a chronic bacterial infection caused by organisms within the genus *Brucella*. The majority of human infections are zoonotic diseases transmitted by livestock and it is endemic in New Zealand (New Zealand Ministry of Health, 2012). Skeletal involvement is variable according to species but the prevalence can be as high as 70% (Ortner, 2003; Jaffe, 1972). Like tuberculosis, brucellosis has a hematogenous route of dissemination and lesions typically occur in highly vascularized skeletal regions such as the vertebral bodies and synovial joints (Ortner, 2003; Jaffe, 1972). However, unlike tuberculosis, the skeletal lesions of brucellosis consist of a small, central and clearly demarcated lytic focus with marginal bony proliferation (Resnick and Niwayama, 1995a). All the lesions recorded in B21 consisted of primary lytic lesions with no marginal proliferative activity other than

sclerosis on the left acetabulum which appears to be an artefact of the remodeling process. A thin layer of subperiosteal new bone was present across the greater trochanter of the left femur but this was not clearly associated with the primary lytic lesions present here. Furthermore, this condition is less aggressive than secondary tuberculosis and very rarely fatal, which is inconsistent with the documentary evidence of this individual's final illness and the active nature of most of the skeletal lesions in B21 (Doganay and Aygen, 2003).

4.1b: Treponematoses

Treponematoses describe a group of diseases caused by subspecies of the bacteria *Treponema pallidum*. Of these, three species (*T. pallidum pallidum, T. pallidum pertenue,* and *T. pallidum endemicum*), can cause diagnostic skeletal lesions in their final (tertiary) stage, which typically occurs many years following infection (Resnick and Niwayama, 1995a). There is considerable difficulty in differentiating the treponematoses by skeletal lesions alone as the diagnostic tertiary manifestations of all three consist of proliferative periostitis surrounding central lytic foci (gummatous lesion), predominantly affecting regions with little overlying soft tissue (e.g. cranial vault, shins, and forearms) (Resnick and Niwayama, 1995a; Buckley and Dias, 2002). However, venereal syphilis (*T. pallidium pallidum*) is the pathogen most likely to have caused treponematosis in colonial New Zealand (Woodward and Blakely, 2014). Given the primary lytic nature of the lesions exhibited by B21, the lack of proliferative periostitis, and absence of ectocranial caries sicca lesions it is unlikely that the skeletal changes exhibited by this individual represent tertiary treponematosis.

4.1c: Malignancy (metastatic cancer, malignant melanoma)

Although most primary bone cancers (e.g. osteosarcoma) are proliferative, there are several malignant conditions which can result in lytic skeletal changes. Metastatic cancer, particularly breast and lung cancer, can result in widespread, circular lytic lesions throughout the axial skeleton and in the proximal metaphyses of the femora and humeri (Ortner, 2003; Resnick and Niwayama, 1995b). Multiple myeloma, a malignancy of plasma cells, results in a similar pattern of numerous, small (5mm-2cm in diameter) destructive lesions throughout the axial skeleton (Grauer, 2019; Giuliani et al., 2006). Both disease processes result in dense and widespread clusters of lesions resulting in a "punched out" or "Swiss cheese" appearance (Ortner, 2003). This is not consistent with the lytic lesions exhibited by B21, several of which were well over 2cm in diameter and restricted to the endocranial skull, hip, and proximal femur.

4.1d: Sarcoidosis

Sarcoidosis, like tuberculosis, is a granulomatous disease (Resnick and Niwayama, 1995c). The etiology is unknown but an autoimmune component has been suspected (Chen and Moller, 2015). Multiple organ systems, including bone and lungs, can be affected. Chronic respiratory failure is not uncommon and hemoptysis can occur (Israel and Ostrow, 1969; Wollschlager and Khan, 1984). However, unlike tuberculosis the granulomas of sarcoidosis do not have a necrotic center and do not result in clearly demarcated osteolytic foci (Resnick and Niwayama, 1995c). Skeletal lesions are rare, occurring in approximately five percent of cases, and are primarily confined to the hands and vertebral column (Resnick and Niwayama, 1995c; Sparks et al., 2014). These consist of diffuse osteolytic activity which gives the trabeculae a characteristic "latticework" appearance (Resnick and Niwayama, 1995c). This condition is chronic, slow progressing, and very rarely associated with fatal complications (Resnick and Niwayama, 1995c). These characteristics are inconsistent with the skeletal lesions present in B21.

4.1e: Tuberculosis

Considering the above, the skeletal lesions manifested by B21 are most consistent with disseminated secondary tuberculosis (section 1.1). These are lytic, of variable size, and originate in the oxygen-rich trabeculae of the axial skeleton. The clearly demarcated, primary lytic foci on the endocranial calvarium, destruction of the left acetabulum, and large, ovoid primary lytic lesions in the proximal left femur are all characteristic features of skeletal tuberculosis (Jaffe, 1972; Resnick and Niwayama, 1995a; Tuli, 2016). This diagnosis is supported by the death certificate and biography of B21 which describe death by hemoptysis after an invalid period of approximately a year. When the documentary and skeletal evidence is considered together, it appears that B21 suffered from active secondary pulmonary tuberculosis as well as disseminated skeletal infection. Other organ systems also may have been affected.

4.2 The Life-Course of B21

The examination of the various tissues from this individual have revealed evidence of his life-course from infancy through to death. Knowing the identity and presumed cause of death of an individual is unusual in bioarchaeology and the preservation of skeletal, dental and hair tissues provides a range of evidence that is unique outside of mummy studies. The following sections offer interpretation of the observations from these tissues to construct an integrated case study of life and death.

4.2a The story from the isotopes:

Strontium isotope evidence is consistent with historical records of B21's place of origin being in the greater London area. We note, however, that were this an unnamed individual we could not identify place of origin with anywhere near this kind of precision. His values align with multiple regions including Somerset, the Midlands, East Anglia, northeast England and Orkney (Evans et al., 2010). Carbon and nitrogen isotope evidence (Figure 9) suggests B21 experienced dietary change through their lifetime. Early life decreases in both nitrogen and carbon isotope values are likely related to the weaning process, with the point at which carbon isotope values stabilize likely representing the completion of the weaning process (at around 20 months of age). However, the individual clearly experienced dietary variation throughout childhood, with nitrogen isotope values continuing to decrease until 5 years of age. During adolescence (around 10-12 years) carbon isotope values increase by around 1‰ while nitrogen isotope values remain stable, perhaps indicating greater C_4 plant input or low trophic level marine foods during this period of their life.

Bone δ^{15}N values around 2‰ lower than dentinal values potentially represent a decrease in dietary intake of meat during adulthood, coinciding with emigration to New Zealand. However, isotopic baseline studies in the UK suggest that nineteenth century London populations in particular had elevated δ^{15}N values relative to other places not because of greater meat intake but because of agricultural practices such as growing on salt marshes and/or centuries of manuring pastoral land (e.g. Treasure et al., 2016; Beaumont et al., 2013b). In reality the difference between bone and dentine values likely reflects a combination of dietary change, and differences in agricultural practices in the new colony.

The hair of this individual has higher δ^{15}N values and more positive δ^{13}C values than bone (once a correction for the hair-collagen offset has been applied). This change could reflect slightly increased meat consumption close to time of death. However, knowing the medical history of this individual, it is possible that these increasing values reflect physiological stress close to time of death (per D'Ortenzio et al., 2015). Catabolism of the body's tissues to meet energy requirements during times of stress can result in further isotopic fractionation as the body effectively consumes its own tissues (Fuller et al., 2005; Mekota et al., 2006). This raises δ^{15}N values and may either raise or lower δ^{13}C values depending on which of the body's tissues are consumed. We acknowledge that the change

in isotopic values close to time of death is not significant, (only 0.5‰), but it may hint at changing conditions close to death.

Controlled feeding experiments (e.g. Warinner and Tuross, 2009) and clinical studies involving patients with anorexia (Mekota et al., 2006) generally agree that wasting or nutritional stress must be severe to affect isotopic values of tissues. If the rise in nitrogen isotopic values in hair is related to disease this may reflect the increasingly debilitating nature of the disease that eventually resulted in B21's death. Alternatively, it may reflect attempts to increase strength close to the end of life, by changing diet. Medical texts of the time recommended over-feeding or use of meat-extract to combat the disease (Barnes, 1995; Riva, 2015; Finlay, 1992).

4.2b His life-course from oral health evidence

The high number of LEH defects in the dentition of B21 indicates that between the ages of two and six years, he experienced periods of physiological stress that interrupted the development of his enamel. Linear hypoplastic enamel defects are considered non-specific lesions as they may develop for a number of reasons including systemic stress (e.g. malnutrition or illness), trauma or exposure to toxins (cf. Kinaston et al., 2019). The earliest estimated age for LEH development in B21's dentition (two years), close to the time of weaning cessation is also identified isotopically. This may indicate that he underwent some type of systemic stress associated with the weaning process, an especially vulnerable time in a child's life (Katzenberg et al., 1996). The multiple LEH lesions observed suggest that B21 experienced subsequent periods of physiological stress during his childhood in England but, importantly, he was resilient enough to survive these early insults and live into adulthood (Wood et al.,1992).

Assessments of tooth wear and oral conditions can provide contextual information regarding diet, oral health, lifestyle factors, physiological stress and fertility (Hillson, 1996). The high number of carious lesions, AMTL of his posterior teeth, and the presence of periodontal disease and a possible alveolar lesion indicate that B21 had poor oral health and would likely have experienced pain, discomfort, and halitosis, potentially from a young age. Diets high in refined carbohydrates are associated with caries formation because they reduce the pH of plaque biofilm, resulting in a proliferation of acid-producing and acid-tolerant bacteria that lead to the demineralization of the tooth enamel (Marsh, 2010; Zaura and Ten Cate, 2015). His apparently soft, non-abrasive diet, as suggested from the mild posterior tooth wear would likely have consisted of flour-based foods (damper or gruel), meat, canned foods such as sardines, and fresh fruits and vegetables when they were available and these observations support those of the isotope analyses (Leach, 2010).

4.3 The final months of life: Ground-truthing the historical documentation with physical evidence

This integrated osteobiography of B21 has shown that physical evidence is essential for ground-truthing historical medical records. Prior to modern diagnostic techniques, "*pneumonic phthisis hemorrhage*" could have been encompassed a number of other respiratory diseases including pulmonary sarcoidosis and lung cancer (section 4.1). The precise etiology of the documented symptoms of B21 could not be known without supporting physical evidence of a specific disease process. In this case, the strong skeletal evidence of extrapulmonary tuberculosis supports the historical documentation of the pulmonary form of the disease. Conversely, the remains in B21 do not provide evidence of pulmonary infection. Pulmonary tuberculosis is notoriously difficult to diagnosis in human skeletal remains and most of the lesions consist of non-specific changes such as islands of subperiosteal new bone on the visceral surface of the ribs (Kelley and Micozzi, 1984; Santos and Roberts, 2006) which were not observed in B21's remains. When the historical documentation is

considered in tandem with the skeletal evidence, a diagnosis of aggressive pulmonary tuberculosis which had disseminated from the lungs is supported.

There are a number of other skeletal features which indirectly support the identification of B21 and are consistent with the recorded history of his life and final illness. Pronounced muscle attachments in the upper limbs, for example, are suggestive of a history of strenuous manual labor although it should be noted that the relationship between entheseal morphology and activity has not yet been experimentally established. Historical evidence suggests that B21 was a goldminer from 1856 to 1862, first in Australia and then in Otago, before working as a laborer in Milton until 1872. However, the apparent osteopenia present throughout the skeleton is consistent with the known period of invalidism lasting a year prior to his death. Reduced bone mineral density is a known consequence of inactivity due to illness or disability and can occur in as little as 17 weeks (Alexandre and Vico, 2011; Takata and Yasui, 2001). Higher δ^{15}N values close to time of death may also support the interpretation of a period of invalidism close to death. It is possible these values relate to a catabolism of muscle tissue prior to death or a period of increased meat-intake as part of palliative care.

4.4: The bioarchaeology of care: what B21 can tell us about Colonial Otago?

The bioarchaeology of care model uses a four-step process to conceptualize the lived experience of an archaeological individual with skeletal evidence of severe pathology ("Index of Care" (IOC); Tilley and Cameron, 2014). This index is extensive and was designed to be used as a primary methodology in biosocial archaeology. However, we have chosen to use the four-step fundamentals of the IOC as a framework in which to discuss our findings, rather than apply the full index of care process outlined in Tilley and Cameron (2014).

The first step in creating an IOC entails identifying the pathological condition(s) the individual suffered from and documentation of the sociocultural and environmental context in which the individual lived ("lifeways context"). As discussed above, the paleopathological findings, combined with the documentary evidence of cause of death, supports a probable diagnosis of disseminated secondary tuberculosis with both pulmonary and skeletal involvement. This individual lived in a relatively isolated rural, frontier environment with few medical resources other than the basic services of a general medical practitioner. Dietary isotopic evidence of the assemblage indicates that these individuals were eating a diet focused on farmed crops and animals, but supplemented extensively with freshwater fish and waterfowl, perhaps due to the scarcity of farmed resources at this early stage of New Zealand pastoralism (King et al., in review).

The second step in the BOC model involves identifying the clinical impact of the disease, its functional consequences, and whether or not the individual would have required care. It is difficult to determine, from skeletal lesions alone, what the functional impact of disease may have been since the individual's experience of disability is tied to both their subjective experience of pain and discomfort and the expectations imposed on them by their society. However, in the case of B21 we know that his experience of his disease was severe enough that he was unable to work for the year prior to his death. Although the skeletal evidence cannot give us direct information on the functional impact of his disease, the extensive lytic lesions in the left femur and hip would have been painful and possibly immobilizing (Tuli, 2016). Clinical literature on secondary tuberculosis indicates that he would have suffered from chronic fevers and increased susceptibility to opportunistic infections (Bezuidenhout and Schneider, 2009; Maher, 2009). Secondary tuberculosis can result in cachexia or "wasting" due to nutrient malabsorption, loss of appetite, and altered metabolism (Schwenk and Macallan, 2000). This in turn induces a chronic catabolic state wherein the body begins to draw on its own tissues to survive. Isotopic analysis of B21's hair suggests that catabolism may have been in effect in the months prior to his death.

The third step involves identifying the care likely to have been required by the individual. Again, it is known from documentary evidence that he was too unwell to work for many months prior to his death which gives us some clues to the extremity of his disease-

related disability. At some stage he would have had required assistance with mobility, hygiene, and feeding, possibly including special preparation of high calorie, easily digested foods. nineteenth century western medicine often prescribed opiates such as laudanum to manage the pertussis from pulmonary TB (Lomax, 1973). These drugs are themselves associated with their own functional impacts such as cognitive impairment and respiratory depression (Brunton et al., 2018). The duration of time for which B21 would have required care cannot be determined with certainty but it is likely that some form of care would have been required once he was unable to work and the intensity of care would have increased progressively until his death in July of 1873.

The final portion of the BOC model considers the individual and social implications of the care provided to the suffering person. In nineteenth century New Zealand there was no formal social welfare or healthcare system, although the 1862 Hospital Ordinance did allow for the establishment of hospitals that were managed by committees of subscribers with each subscriber having the ability to recommend two people for charitable aid (Angus, 1984; Garland, 2012). More common, and in keeping with the nineteenth century ideology of self-help in terms of health care, was the establishment of various 'friendly societies,' which acted both as a source of community security and identity, and as private insurance providers. These societies included the Ancient Order of Foresters, the Manchester Unity Independent Order of Oddfellows and the Good Templars. In Milton, the Court Bruce of the AOF was established in about 1865, and for a regular weekly contribution of one-shilling members were eligible for £1 per week financial support should they be unable to work, while £20 would be paid towards funeral expenses in case of death (Bruce Herald 14 August 1872; Carlyon, 2001). B21 was a long-standing member of Court Bruce of the Ancient Order of Foresters, and as discussed above, was financially supported by them for the last 11 months of his life. The Foresters also paid for his funeral, and then raised funds to help his widow pay off a remaining debt on the family house (Bruce Herald 11 February 1873; 10 October 1873). B21's interment in a coffin that included all of the customary trappings of the day, including ornate detailing, a painted name plate and iron handles, shows that despite his long-term incapacitation he was given the dignity of a traditional burial with full honors through the agency of the AOF (Bruce Herald 11 February 1873).This is not a lone case of the influence of the AOF in the St. John's burial ground, the graves of the surgeon and secretary of the Court Bruce were also both identified during the 2016 excavations (Findlay et al., 2015; Petchey et al., 2017). Petchey et al. (2018: 56) have also raised the possibility of AOF involvement in the care of two individuals from the Cromwell Cemetery. The AOF was the first friendly society to be established at Cromwell, and Forester-funded funerals give us further information about the region-wide extent of this form of community care.

Milton in the 1870s was a relatively isolated frontier community. However, the osteological and documentary evidence of B21's final illness show that the population was, at least to some extent, characterized by inter-dependence rather than individualism. Support from both within and outside the nuclear family was available to the invalid in the community. Despite his disease state, B21 was effectively cared for both before and after death by a social support network that he belonged to and had been established for just this type of situation. Despite the lack of formal government-funded social support, the society of the time had evolved systems and networks to support those who made the choice (and, importantly, had the means) to belong.

5. Conclusions

In this integrated case study of the life-course of B21 we have demonstrated that this approach, which interprets multi tissue life-course findings and documentary evidence can be particularly powerful when placed within the wider biosocial context of the community and time period. These data have also ground-truthed the documentary evidence of his life and death offering a rare opportunity to gain biological insights into the lived experiences of a single individual living in a time of social and biological change in New Zealand's colonial history which resonates with other similar colonization events throughout the globe.

A colloquial approach for describing the life-course of an archaeological individual has been advocated as a way of making bioarchaeological enquiry more accessible to the general audience (Boutin, 2016). This is attempted here by way of conclusion:

"This man began his life in London. His early life experiences involved periods of stress, both related to weaning and in later childhood. He experienced dietary change as he moved from a well-established agricultural society to the colonies where farmed meat sources were supplemented with wild fish and game. He suffered from prolonged and severe dental health issues that would have likely caused him constant pain and discomfort. Like many of his time, he indulged in the habit of tobacco smoking. He used his body to provide for his wife and large family, working as a manual laborer, first in the pursuit of riches in the goldfields of Australia and Otago and then as laborer after the dream of gold was lost. At some point in the last few years of his life he contracted a disease which ultimately left him debilitated to such an extent that he could not support his family. His descent into death was prolonged, painful and distressing. Fortunately, his good works in the community and foresight of joining the friendly societies meant that he could go to his rest in the knowledge that his family would be provided for after his death."

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Figure and Table Captions for: “Captain of all these men of death”: An integrated case-study of tuberculosis in 19th Century Otago, New Zealand.

Figure 1: Map of the burial ground of the Anglican Church of St. John, Milton.

Figure 2: Death certificate for Burial 21 listing cause of death as “Pneumonic phthisis haemorrhage”

Figure 3: Excavated grave of B21 with skeletal remains *in situ*.

Figure 4: Radiograph of the endocranial surface of the frontal and parietals of B21 showing lytic foci (black circles).

Figure 5a: Mandible of B21 showing wear facets on the distal surface of the right lateral incisor and mesial surface of the right canine (pipe facet), as well as multiple episodes of LEH in the right canine and first premolar.

Figure 5b: *Top*: periapical radiograph of right maxilliary sextant (teeth 15-18). A large central pulp stone is present in tooth 17 (red arrows), numerous small pulp stones in tooth 18 (green arrows), and almost complete stenosis of the pulp chamber in tooth 16 (blue arrows). *Bottom*: left mandibular posterior sextant. Only tooth 38 remains and numerous small pulp stones are evident (green arrows).

Figure 6a: Radiograph of the left ilium and ischium showing sclerosis of the margins of the acetabulum (red arrows).

Figure 6b: CT scan of the left ilium and ischium showing lytic foci (yellow arrows). The large radiolucent focus on the iliac blade (white circle) is an artefact of the CT slice and not pathological.

Figure 7a: Posterior aspect of the proximal left femur showing localised regions of lytic activity along the greater and lesser trochanters (white arrowheads). The region demarcated by the white circle is post-mortem damage but lytic “tunnelling” is also visible within the trabeculae here.

Figure 7b: Radiograph of the posterior aspect of the left femur showing extensive lytic activity in the head and trochanters (blue arrowheads).

Figure 7c: CT scan of the posterior aspect of the left femur showing lytic foci in greater detail.

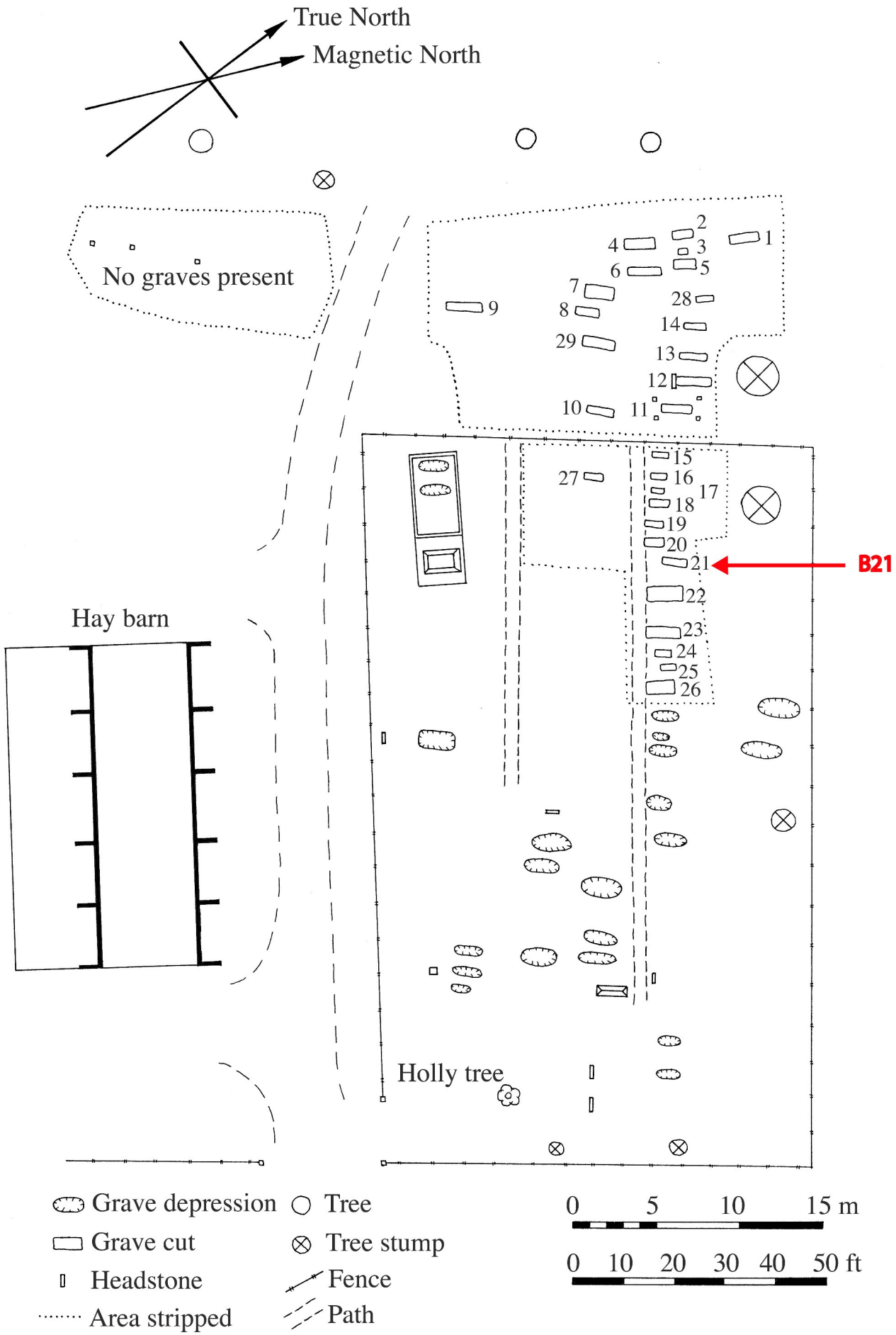
Figure 8: Possible areas of origin for B21 based on strontium isotope results (shaded grey areas). Dark grey circle marks the area of Greater London, where historical evidence suggests B21 actually hailed from. Map adapted from Evans et al. (2010).

Figure 9: Changes in δ^{15}N (primary axis, squares) and δ^{13}C (secondary axis, circles) values within and between B21’s tissues, representing dietary change during life. Here bone

collagen values (long rectangles) are arbitrarily assigned an “adult” age, and do not necessarily represent the exact time period given on the x-axis. Hair values have been corrected to control for the collagen-keratin offset (as described in text).

Table 1: Summary of pathological conditions included in the differential diagnosis for B21

Table 2: Isotopic samples taken from B21.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No graves present
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B21
Hay barn
Holly tree
Grave depression
Grave cut
Headstone
Area stripped
Tree
Tree stump
Fence
Path
0 5 10 15 m
0 10 20 30 40 50 ft

REGISTRAR'S RETURN OF ALL ENTRIES OF DEATHS IN THE REGISTER-BOOK FOR THE DISTRICT OF Dr Monaros DURING THE QUARTER ENDING THE 31st OF March 1873.

Number.	When Died and Where.	Name and Surname.	Sex.	Age.	Rank or Profession.	Cause of Death.	Signature, Description, and Residence of Informant.	When Registered.	Signature of Registrar.
197	5th Feb Dr Monaros	[redacted]	Male	42 Years	Labourer	Medical Certificate Pneumonic Phthisis Remote cause	A. J. Ferguson Informt. Medical Attendant Dr Monaros	15 Feb	Dr Simpson


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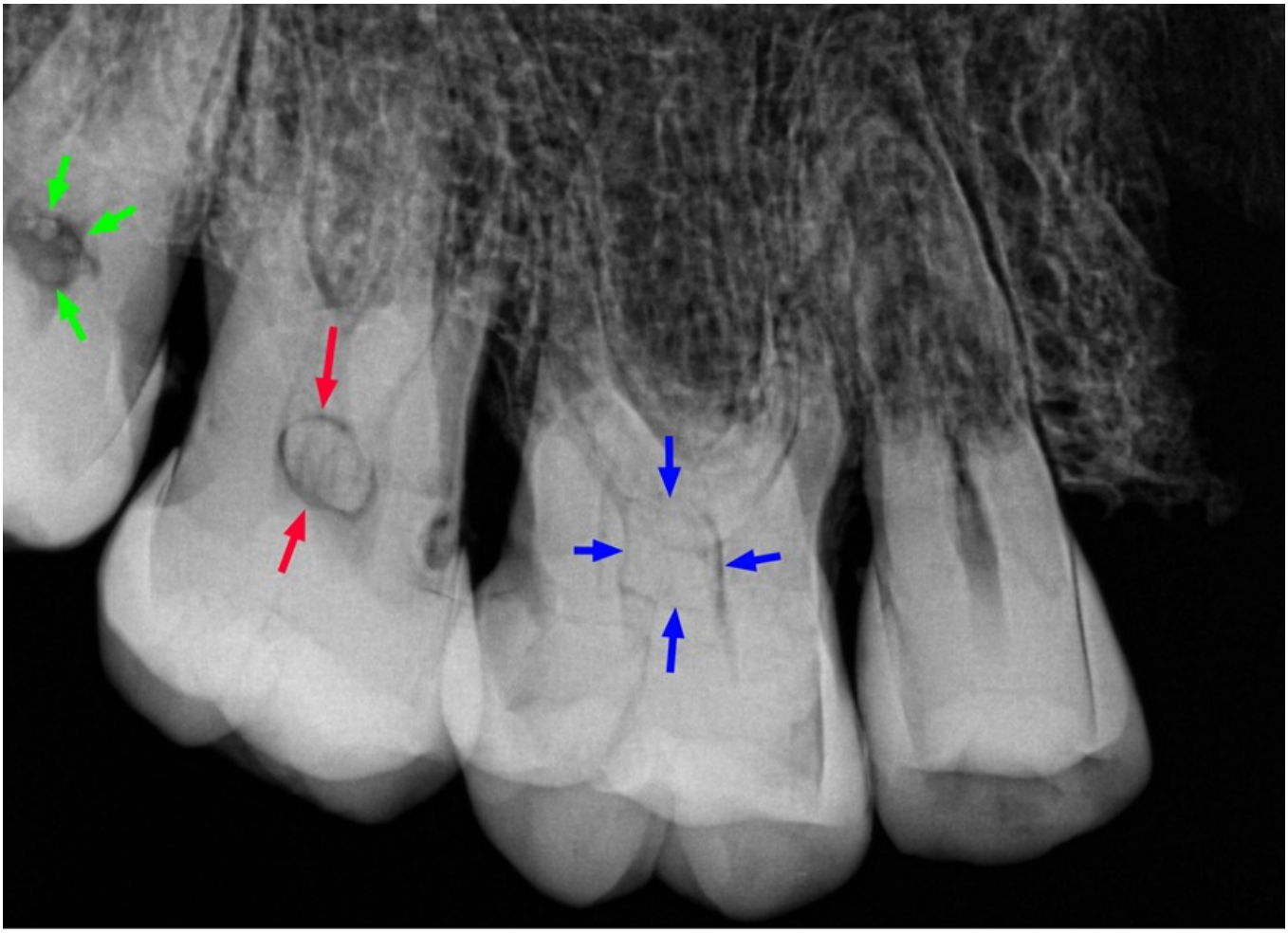

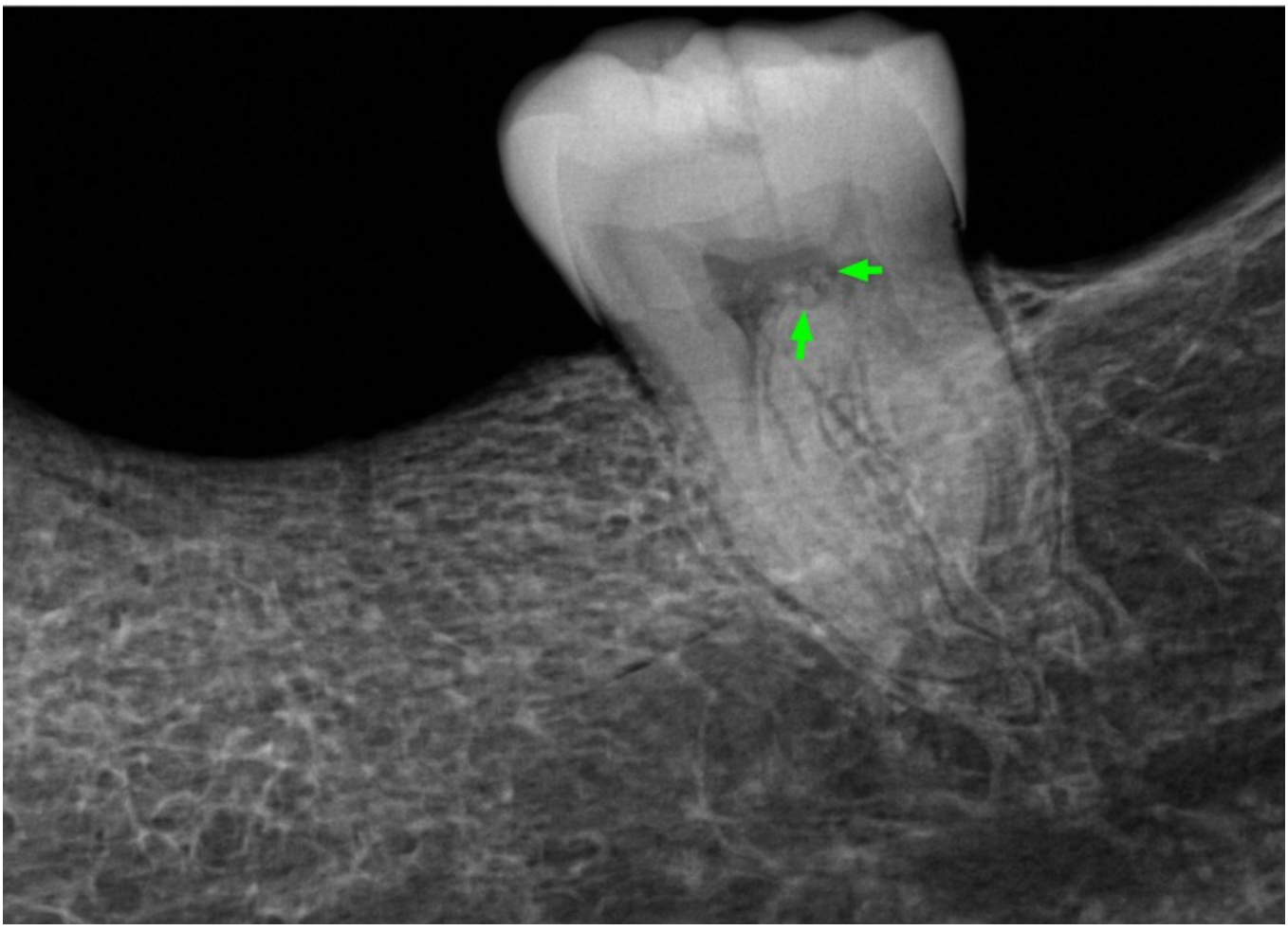

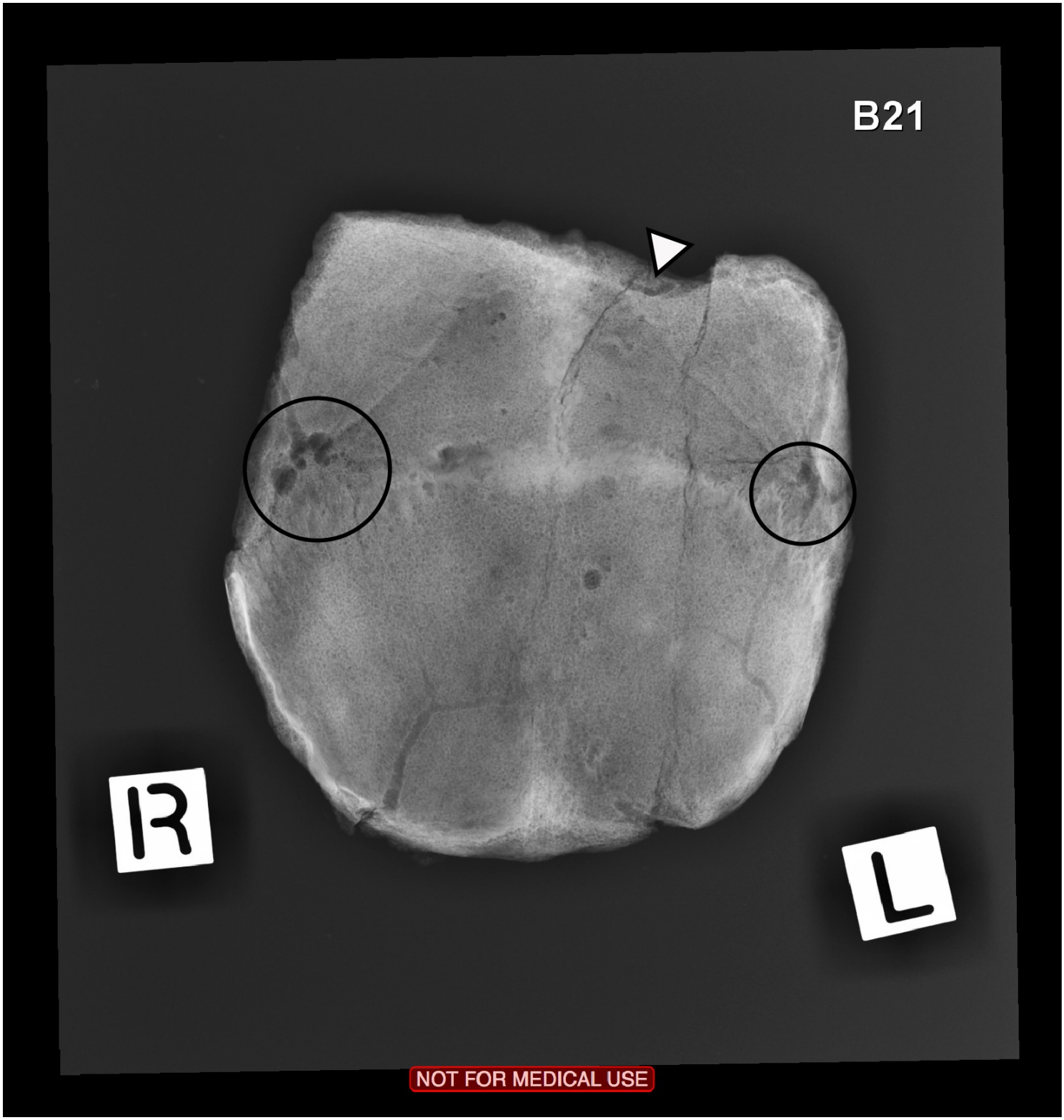
B21
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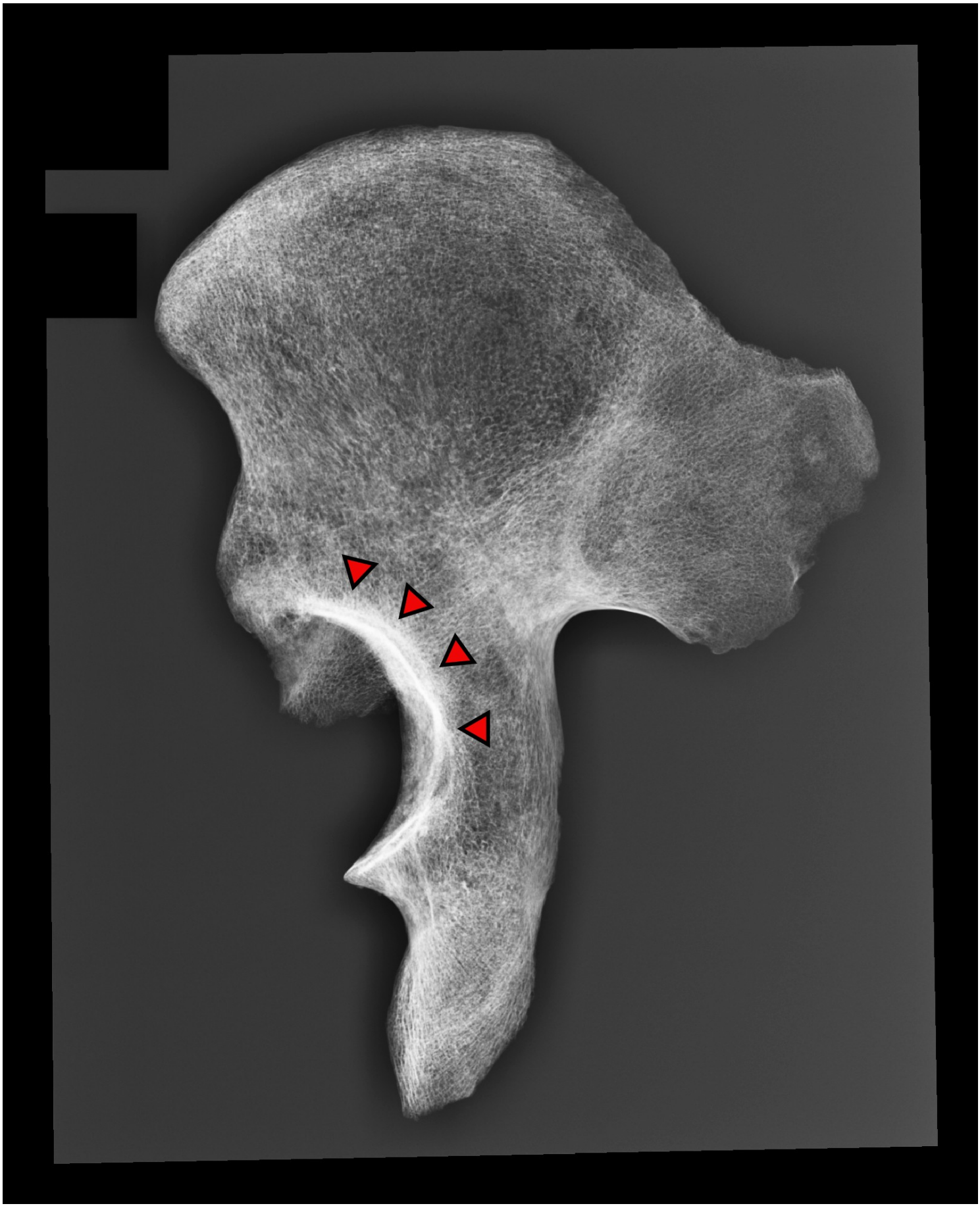
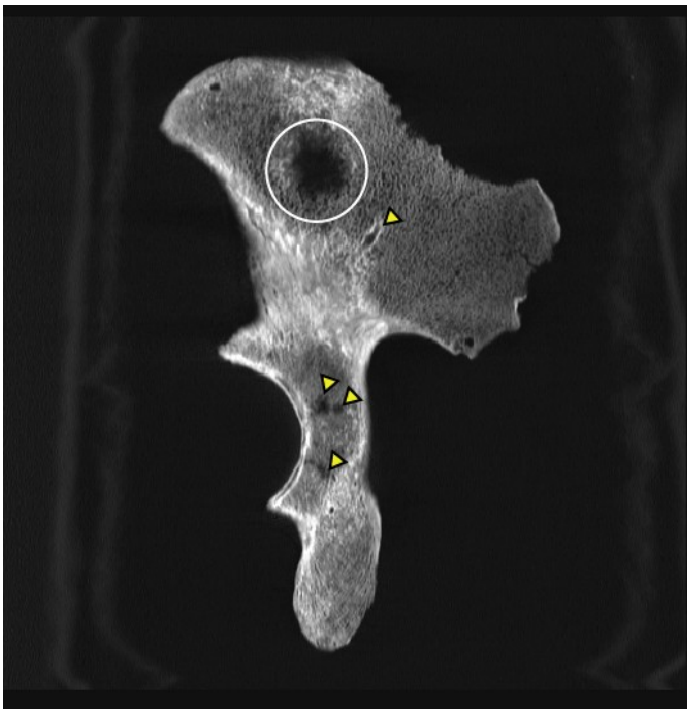
NOT FOR MEDICAL USE



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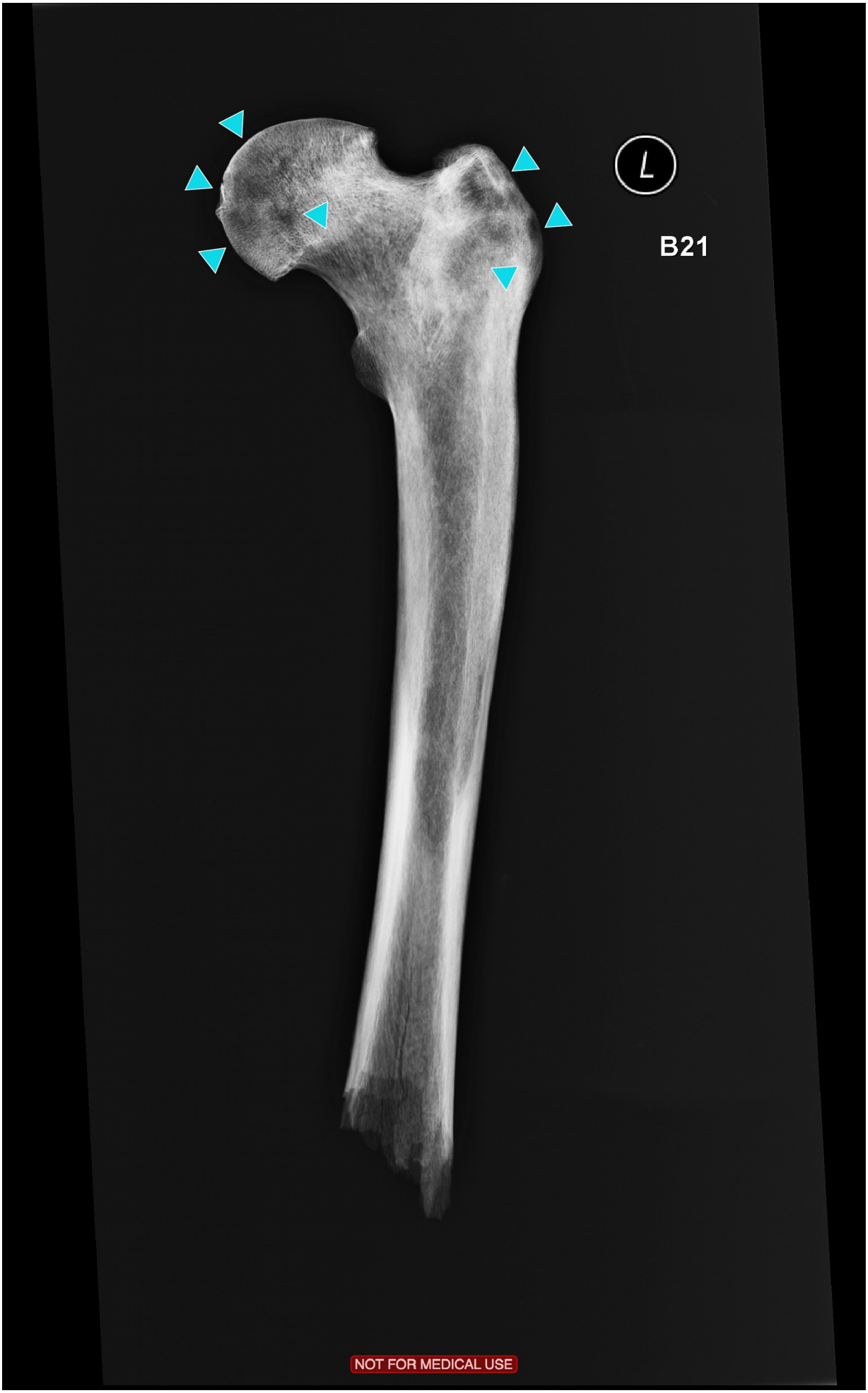
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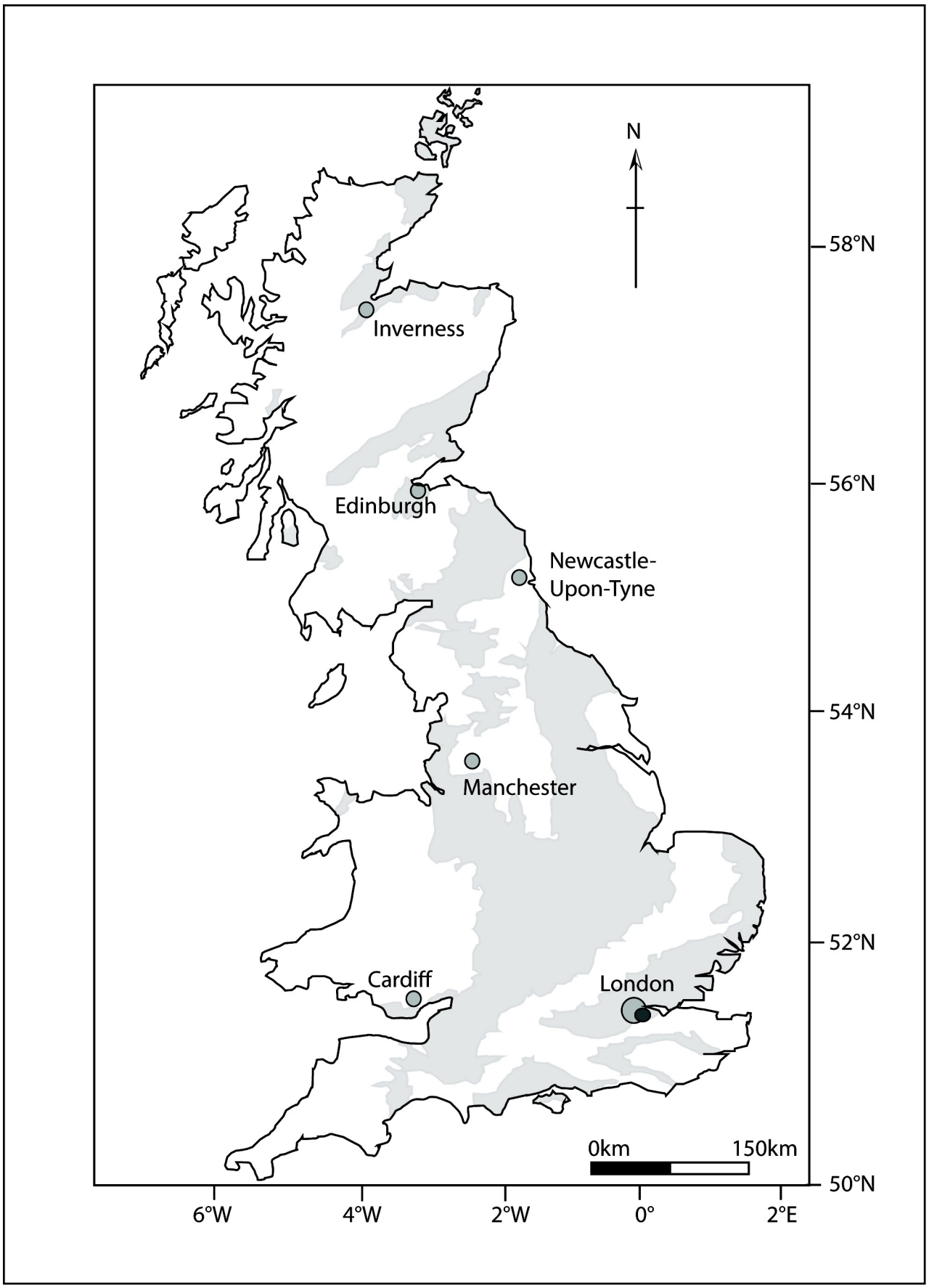
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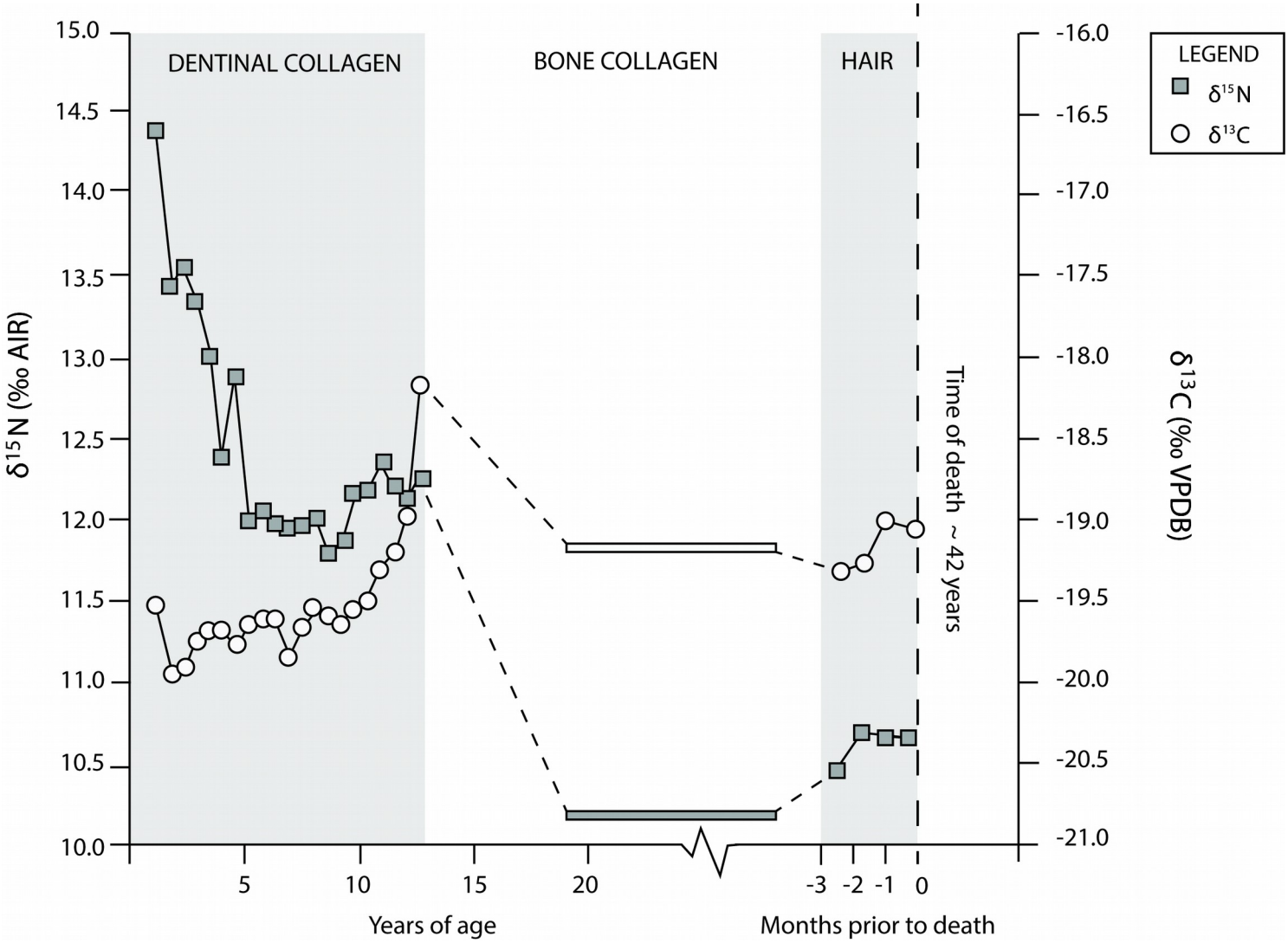
DENTINAL COLLAGEN
BONE COLLAGEN
HAIR
LEGEND
δ15N
δ13C
δ15N (‰ AIR)
δ13C (‰ VPDB)
Time of death ~ 42 years
15.0
14.5
14.0
13.5
13.0
12.5
12.0
11.5
11.0
10.5
10.0
-16.0
-16.5
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-18.5
-19.0
-19.5
-20.0
-20.5
-21.0
5
10
15
20
-3
-2
-1
0
Years of age
Months prior to death

Tissue sampled	Formation time	Isotopes analysed	Aspect of life
Dental Enamel (lLeft mandibular canine)	0.9-5.5 years (AlQahtani et al., 2010)	Sr, C, O	Place of residence, childhood diet
Dentinal collagen (sampled incrementally)	0.9 – 13 years (AlQahtani et al., 2010)	C, N	Dietary change during childhood,- pPossible physiological stress-
Bone collagen (ulna)	Last <10 years of life (Hedges et al., 2007)	C, N	Average adult diet while in NZ
Hair (sampled incrementally). Sample = 4cm	Leading up to time of death 1cm = 1 month (O'Connell et al., 2001)	C, N	Diet close to death, possible physiological stress-

Table 1: Isotopic samples taken from B21.

Disease	Clinical characteristics	Skeletal Lesions?	Consistent with skeletal evidence from B21?	Consistent with documentary evidence?	References
Brucellosis	Chronic bacterial infection with ~~haematogenius~~ hematogenous route of dissemination.	Yes (variable prevalence): small, circular lytic lesions with marginal osteophytes. Vertebral column most commonly affected.	No: lesions are ~~pimary~~primary lytic, vertebral column not affected	No: not associated with ~~haemopytis~~hemoptysis, rarely fatal.	Jaffe, 1972; Ortner, 2003; Resnick and Niwayama, 1995a.
Treponematoseis	Chronic bacterial infection caused by organisms in the genus *Treponema*.	Yes (later stages): mixed osteolytic/osteoblastic lesions, proliferative periostitis	No: no proliferative periostitis, lesions were primary osteolytic.	No: not associated with ~~haemopytsis~~hemoptysis	Buckley and Dias 2002; Resnick and Niwayama, 1995a
Metastatic cancer	Result of vascular dissemination of malignant cells from a primary site (e.g. lungs, prostate, breast)	Yes (common): small, circular primary lytic lesions throughout the axial skeleton; widespread yielding a "swiss cheese" appearance	No: lesions are variable in size (some greater than 5mm across) and regionally confined	Possibly: ~~haemopytis~~hemoptysis can be a complication of lung cancer	Grauer, 2019; Ortner, 2003; Resnick and Niwayama, 1995b
Malignant melanoma	Primary malignancy of plasma cells	Yes (common): as above	No: as above	No: not associated with ~~h~~haemopytsis	Giuliani et al., 2006; Grauer, 2019; Ortner, 2003
Sarcoidosis	Chronic granulomatous disease of unknown ~~aetiology~~etiology	Yes (rare): can result in diffuse osteolytic lesions in the vertebral column, hands, and feet	No: osteolytic lesions are clearly demarcated, vertebral column not affected	Conflicting: ~~haemoptysis~~hemoptysis is a possible complication of pulmonary sarcoidosis but disease is chronic and rarely fatal.	Resnick and Niwayama, 1995c; Sparks et al., 2014; Israel and Ostrow, 1969; Wollschlager and Khan, 1984
Tuberculosis (TB)	Infectious granulomatous disease	Yes (rare): primary osteolytic lesions of variable size in vertebral column, synovial joints, and/or other trabeculae-dense regions	Yes: multiple primary osteolytic lesions in the endocranial skull, hips, and left femur	Yes: ~~haemoptysis~~hemoptysis is a well-documented sign of secondary pulmonary TB; condition is nearly always fatal without antimicrobial therapy but death may take months or years	Jaffe, 1972; Resnick and Niwayama, 1995a; Tuli, 2016

Table 2: Summary of pathological conditions included in the differential diagnosis for B21

Sample Type	Sample Number	Age represented (years)	%N	δ^{15}N	%C	δ^{13}C	C/N atomic
Mandibular canine	B21.1	1.19	15.43	14.39	43.31	-19.53	3.3
	B21.2	1.76	15.33	13.43	43.03	-19.96	3.3
	B21.3	2.34	15.55	13.56	43.11	-19.92	3.2
	B21.4	2.92	15.48	13.34	43.11	-19.76	3.2
	B21.5	3.49	15.44	13.02	43.28	-19.67	3.3
	B21.6	4.07	15.38	12.38	42.79	-19.69	3.2
	B21.7	4.65	15.40	12.88	43.24	-19.78	3.3
	B21.8	5.22	15.40	11.98	42.86	-19.66	3.2
	B21.9	5.80	15.39	12.06	43.16	-19.63	3.3
	B21.10	6.37	15.20	11.98	42.70	-19.61	3.3
	B21.11	6.95	15.60	11.94	44.22	-19.84	3.3
	B21.12	7.53	15.12	11.95	43.09	-19.67	3.3
	B21.13	8.10	15.18	12.01	42.96	-19.56	3.3
	B21.14	8.68	15.35	11.80	44.00	-19.61	3.3
	B21.15	9.25	15.37	11.86	43.82	-19.66	3.3
	B21.16	9.83	15.30	12.16	43.15	-19.56	3.3
	B21.17	10.41	15.15	12.19	43.01	-19.50	3.3
	B21.18	10.98	14.95	12.36	42.27	-19.31	3.3
	B21.19	11.56	15.11	12.20	43.12	-19.20	3.3
	B21.20	12.14	15.01	12.14	43.12	-18.99	3.3
	B21.21	12.71	15.14	12.25	43.72	-18.17	3.4
Bone sample	Bulk B21	Adulthood (~ 30 yrs)	12.70	10.11	36.17	-19.13	3.3
Hair	B21.1(hair)	Time of death	12.07	8.45	40.19	-20.69	3.9
	B21.2(hair)	Time of death – 1 month	12.41	8.72	40.01	-20.64	3.8
	B21.3(hair)	Time of death – 2 months	13.57	8.69	42.20	-20.39	3.6
	B21.4(hair)	Time of death – 3 months	13.49	8.69	41.89	-20.44	3.6

Supplementary Table 1: Results of carbon and nitrogen isotope analysis of dentinal collagen increments, bone collagen and keratin.

Carbon isotope ratios were corrected for ^{17}O contribution. All isotopic measurements are reported in standard delta notation (δ) relative to international standards (VPDB and N_2 Air). Isotopic accuracy was established using repeat measurements of international standards and in-house standards: the latter are calibrated against international standards (e.g., USGS40, USGS24, IAEA-600, IAEA-N-1, IAEA-N-2). Analytical uncertainty in carbon and nitrogen isotope analysis was typically ±0.1‰ for replicate analyses of the international standards and <0.2‰ on replicate sample analysis. Total % organic carbon and nitrogen data were obtained using an in-house standard (Glutamic Acid, 40.82% C, 9.52% N). All samples were considered to have yielded good quality collagen/keratin as they fall within acceptable parameters (C/N =2.9–3.6 *collagen* C/N=2.9–4.0 *keratin)*, weight % C = 30–50, weight % N = 10–16). Technical error of measurement was calculated using repeat analyses of samples as 0.1‰ for both $\delta^{15}N$ and $\delta^{13}C$.