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Diagenesis of archaeological bone and tooth

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Abstract

An understanding of the structural complexity of mineralised tissues is fundamental for exploration into the field of diagenesis. Here we review aspects of current and past research on bone and tooth diagenesis using the most comprehensive collection of literature on diagenesis to date. Environmental factors such as soil pH, soil hydrology and ambient temperature, which influence the preservation of skeletal tissues are assessed, while the different diagenetic pathways such as microbial degradation, loss of organics, mineral changes, and DNA degradation are surveyed. Fluctuating water levels in and around the bone are the most harmful for preservation and lead to rapid skeletal destruction. Diagenetic mechanisms are found to work in conjunction with each other, altering the biogenic composition of skeletal material. This illustrates that researchers must examine multiple diagenetic pathways to fully understand the post-mortem interactions of archaeological skeletal material and the burial environment.

Keywords: Bone diagenesis, environment, bioerosion, collagen, hydroxyapatite, ancient DNA

1. Introduction

The survival of biomolecules in archaeological and fossil bone has attracted the attention of a great number of researchers over the past few decades. The post-mortem preservation of bone, however, depends upon a number of complex processes. Thus, some bones survive well, whilst others degrade rapidly. Here we attempt to survey the major ways in which skeletal tissues, specifically those made of mineralised collagen (bone and dentine),

become transformed following deposition in the archaeological or geological record, a process known as diagenesis.

2. Chemistry and structure of bone and tooth dentine

2.1 Skeletal tissues

Bone is a composite material made up of both organic and inorganic components, and water. The fibrous protein, collagen, makes up the vast majority of the organic fraction, and carbonated hydroxyapatite (or HAp) comprises the inorganic, or mineral, fraction (Currey, 2011; White et al., 2012). Bone is only one of several vertebrate collagenous tissues that are strengthened and hardened *in vivo* by the precipitation of poorly-soluble inorganic minerals - a process that in technical terms is called *biomineralisation*. In mammals the other mineralised tissues include antler, dentine and cementum which all share a common chemistry – collagen, HAp and water in various proportions – but they have different microarchitectures and modes of growth. Consequently they share similar diagenetic histories. Tooth enamel, which differs from the other mineralised tissues in its mode of formation - protein scaffolding is removed and replaced with mineral - has an extremely low porosity and low organic content. It is typically, therefore, much more resistant to diagenesis and does not form part of this review.

The percentage of organic and water by weight of the various mineralized tissues differs depending upon species and skeletal element (Fig. 1). Oven-dried human bone has approximately 26 wt. % organic matter, with 9-10 wt. % water and 64 wt. % mineral, which can be compared to bovine bone, as an example, which has a makeup of 24 wt. %, 8 wt. % and 68 wt. %, respectively (Zioupos et al., 2000). In terms of volume percent, the relative proportions of collagen/water and mineral roughly proportional due to the higher density of hydroxyapatite (bulk density 2.8, He et al., 2010) than either dry collagen (bulk density 1.4, Podrazký and Sedmerová, 1966) or water (bulk density 1). Reptiles and marine mammals are typically less heavily mineralised than terrestrial mammal bone (Rogers and Zioupos, 1999; Zioupos et al., 2000). Although sperm whales and sea lions have a higher mineral content than humans, fin whales have less well mineralised bones and fish considerably less so, with as little as half the mineral content as land mammals (Tont et al., 1977).

Amongst land mammals, the collagen content ranges from approximately 35% for deer antler to only around 21% for wallaby femur. Some highly specialized bony elements such as the bulla tympanum and rostrum of whales are almost totally mineralized with only 11 %, and <1% collagen, respectively (Zioupos et al., 2000). Bird bones are stronger and have higher densities (Dumont, 2010) but it is unclear whether this truly reflects a higher proportion of mineral or simply a lower porosity (Schuhmann et al., 2014). Amongst humans, the highly mineralised petrous portion of the temporal bone is attracting attention due to the often high levels of protein and endogenous DNA that can survive in archaeological specimens compared to non-petrous skeletal elements (Gamba et al., 2014; Jørkov et al., 2009). Different species and different skeletal elements not only have differing mineral contents but exhibit a variety of microarchitectures as revealed by histological examination. These reflect the speed and pattern of growth but also the mechanical loads placed upon the various tissues. These are discussed in greater detail below.

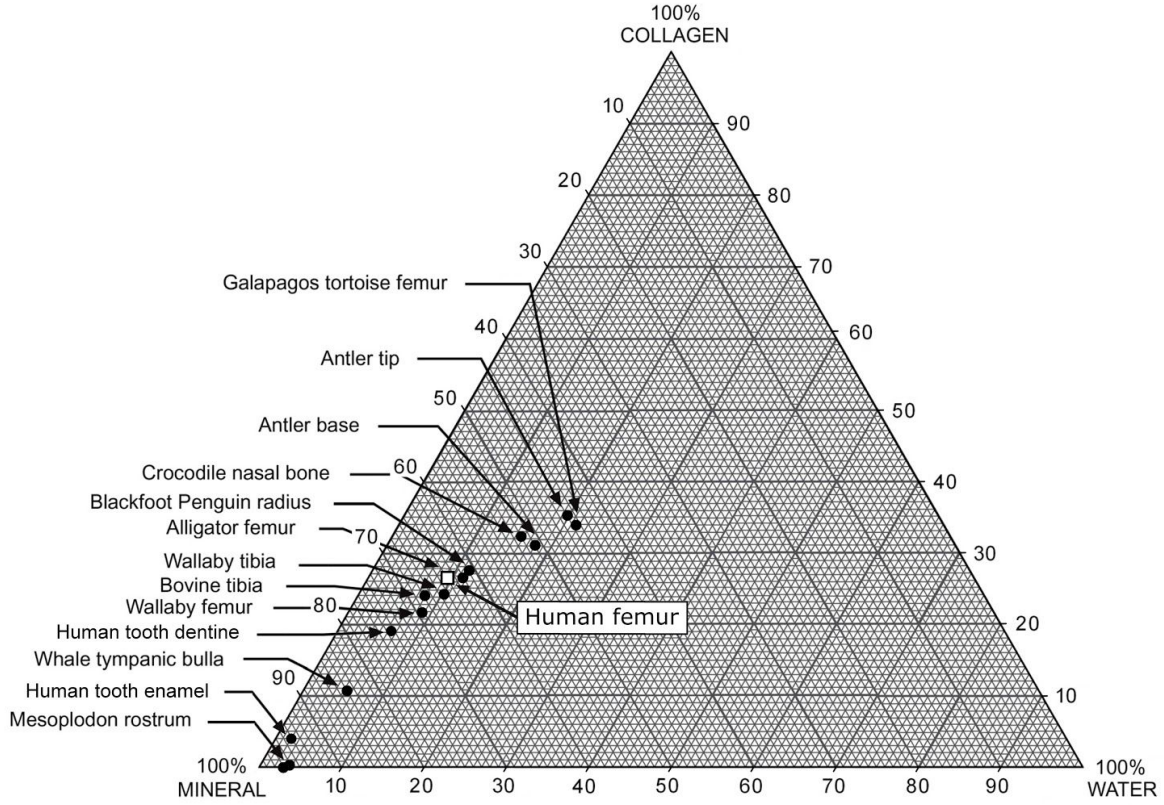

Figure 1 - Relative proportions (as weight %) of collagen, mineral and water for a range of species and tissues. (Diagram adapted from Zioupos et al. 2000).

2.1.1 Collagen

The organic portion of bone is composed largely of Type I collagen, which accounts for approximately 90% of the organic fraction of bone, the remainder being composed of non-collagenous proteins – including bone sialoproteins (Termine, 1988) and osteocalcin (Lian et al., 1978; Price et al., 1976) – various lipoproteins, and mucopolysaccharides (Morgan et al., 2013; Ortner and Turner-Walker, 2003; Tuross, 2003). Collagen is a fibrous protein and the dominant structural protein in most eukaryotes, although (a similar Gly-Xaa-Yaa motif is found in prokaryotes, Yu et al., 2014). The glycine-triad polypeptide chain found in the procollagen molecule, typically depicted as Gly-Xaa-Yaa, is a repetitious sequence in which every third position is glycine (Gly), while the Xaa and Yaa positions are typically found to be the amino acids proline or lysine (Whitford, 2013), which reflect the structural constraints of the polyproline motif. The constituent amino acids confer a structural twist to the peptide bonds, and thus, to the final procollagen molecule. Because each side chain is offset 120° from the preceding residue, every third residue has to be Gly, since the side chain of larger amino acids cannot be accommodated when the side chain is facing in towards the centre of the helix.

Two different polypeptides make up the Type I collagen triple helix, known as tropocollagen. Two of the chains are denoted as α1 (I) the third is the α2 (I) chain (Garnero, 2015; Orgel et al., 2006). A high percentage of the Yaa position prolines are hydroxylated but only a handful of those in the Xaa position (Eyre et al., 2011). These hydroxylation reactions are catalyzed by two different enzymes: prolyl-4-hydroxylase and lysyl-hydroxylase. Although the structural advantage of this hydroxylation is still unclear (Shoulders and Raines,

2009), hydroxylation in the Xaa position does show evidence of stabilization and maintenance of the triple-helix structure (Jenkins et al., 2003; Vitagliano et al., 2001). Furthermore, if the amino acid sequence is changed, for example by a mutation in the relevant gene, or hydroxylation is incomplete as a result of absence of catalytic enzymes or cofactors, then the resulting disruption and destabilisation of the collagen fibrils gives rise to diseases such as osteogenesis imperfecta and scurvy (Bentley and Jackson, 1965).

Collagen also forms cross-links with adjacent collagen molecules. Cross-links are the byproduct of collagenic lysine and hydroxylysine ε-amino group conversion into aldehydes, which through a reaction with lysyl oxidase, form a connection with adjacent collagen molecules (i.e. a cross-link) that are easily reducible (Tanzer, 1973). However, these reducible cross-links diminish and make way for pyridinoline cross-links in matured bone (Eyre et al., 1988; Uchiyama et al., 1981). Pyridinoline cross-links are characterized through a stabilization process brought about after the intermolecular bonds of younger collagen fibrils and cross-links become reduced (Robins et al., 1973). There are two types of pyridinoline cross-links, hydroxylysyl-pyridinoline and lysyl-pyridinoline, where the hydroxyl form is found predominantly in internal connective tissues, and the latter form is typically seen in bone and dentin (Hanson and Eyre, 1996). Most lysyl-pyridinolines are concentrated at the N-telopeptide site of collagen, while only 33% are seen at the C-telopeptide sites (Hanson and Eyre, 1996).

Cross-links not only stabilise the collagen matrix *in vivo*. Post mortem cross-linking of collagenous tissues, either intentionally during the tanning of leather or incidentally as in the formation of bog bodies in sphagnum bogs, increases the shrinkage temperature of the collagen but also renders it less susceptible to fungal and bacterial degradation (Painter, 1991, 1983). This has obvious implications for diagenesis and collagen-humic interactions will be discussed further below.

Tropocollagen molecules, each around 300 nm in length and 1.5 nm in diameter, spontaneously self aggregate extra-cellularly into fibrils with mean diameters of around 50 nm (Tzaphlidou and Berillis, 2005). The intermolecular bonds are such that there is an offset in the alignment between adjacent molecules with the result that there are gaps between the head of one molecule and the tail of the previous one. This permits the creation of fibrils that are much longer than individual tropocollagen molecules. The molecules interdigitate in such a way that there are gap zones (where there is high density of gaps) and overlap zones where the molecules are well aligned and more closely packed. The 40 nm gap zone together and the 27 nm overlap zone give rise to the 67 nm banded appearance of collagen fibrils seen in TEM images, experimentally mineralization of reassemble fibrils is initiated in this gap region (Nudelman et al., 2013/8)

2.1.2 Apatite

The inorganic portion of living bone is a non-stoichiometric, carbonate-substituted form of geologic hydroxyapatite containing traces of F, Na, Mg, Zn and Sr, hereafter referred to as bioapatite or simply HAp (Mathew and Takagi, 2001; Ortner and Turner-Walker, 2003). Bioapatite has different crystal size, composition and atomic disorder than geologic apatites that form hydrothermally (Boskey, 2007, 2003). If basic calcium phosphate is precipitated from a solution containing carbonate or hydrogen carbonate, at room temperature the product is an apatite that invariably contains carbonate up to a few percent and these apatites are of a

similar small size to bone mineral. In fact, strictly carbonate-free solutions and a thorough purging of the laboratory apparatus with nitrogen are required if pure hydroxyapatite is to be precipitated (Posner, 1985). Even in mature bone, the crystallites of bone mineral are exceedingly small, with estimated lengths of 16-50 nm, widths of 8-20 nm and thickness *c.* 2 nm (Eppell et al., 2001; Glimcher, 2006). However, there remains active debate regarding the shape and organization of crystallites in bone (e.g. Georgiadis et al., 2016; Reznikov et al., 2015; Schwarcz, 2015). Their vanishingly small size, combined with the various substitutions and surface adsorbed ions that occur in the non-apatitic hydrated layer, which covers the core of apatite, are responsible for the poor ordering and crystallinity of bioapatite (Figueiredo et al., 2012) and their consequent higher solubility than geologic apatites (Sillen and LeGeros, 1991).

Size, shape and composition of the bioapatite crystals are affected by several intrinsic and extrinsic factors including collagen properties and the distribution of non-collagenous proteins, which are involved in the nucleation and growth of crystals, diet, disease, cell viability and bone turnover (Boskey, 2007, 2003). With reference to crystals' size and orientation in normal living bone, these are controlled by the structure and organization of the collagen fibrils, while size and shape are also regulated by non-collagenous proteins such as phosphorylated proteins, glycoproteins, and proteoglycans (Boskey, 2007, 2003; Duer and Veis, 2013). Changes in crystal size and homogeneity also affect bone mechanical properties, with an increase being either related with an incorporation of ions into the smaller crystals, merging of existing crystals, or secondary nucleation/mineralization (Boskey, 2003).

Early study of crystallite change in vivo show that in rat bone aged two weeks to one year, older bone had an increase in the crystal size and disorder of the crystal lattice (Matsushima and Hikichi, 1989). In mammals, mature bone is both more heavily mineralised and the mineral is more crystalline compared to recently deposited tissues due to carbonate substitutions and a reduction of the hydrated layer that result in a more stoichiometric structure (Figueiredo et al., 2012). Carbonate ions (3-8 wt.%) may either replace OH^- (type A apatite) or PO_4^{3-} (type B apatite) ions in the crystal lattice (Figueiredo et al., 2012; Lee-Thorp and van der Merwe, 1991; Stathopoulou et al., 2008). These ionic substitutions are responsible for the crystal disorder as the replacing ions and molecules may differ in size, configuration, and charge compared to the ions and molecules they are substituting (Yotam Asscher et al., 2011).

Primary sites of mineral deposition are in the gap zones and that subsequent mineralisation takes place between tropocollagen molecules (in the overlap zone) and in intra-fibrillar spaces (Fratzl et al., 1991; Nudelman et al., 2013/8). Full mineralization is accomplished by the replacement of water within and between collagen fibrils by HAp, with small crystallites developing both within and on the surfaces of the collagen, with the bulk of the mineral load being deposited between fibrils and fibre bundles. Thus, as the volume of the mineral in bone tissue increases, the volume of water decreases correspondingly. Collectively, in dry, mature bone, collagen and bioapatite each account for 46% of the volume of bone, while water makes up the remaining 8% (Todoh et al., 2009). Water plays an important role in stabilising the collagen molecule and it has long been known that the mechanical properties of bone are strongly dependent on its hydration state (Duer and Veis, 2013; Nyman et al., 2006). Furthermore, mineralised tissues swell and shrink with the adsorption and desorption of water into their structure, predominantly as a result of water entering the collagen fibrils. Although the bulk of the volumetric changes occur at right

angles to the long axis of the collagen molecules there is evidence that the lengths of fibrils change slightly as the gap zone is filled or emptied (Habelitz et al., 2002).

2.2 Bone tissue organization

As stated previously, the mineralised collagen in vertebrates is found with a variety of tissue types and microstructural organisation. The three-dimensional architecture of these different tissue types reflects the growth, function, vascularity and especially speed of growth of the bone. These variations have been discussed at length by two giants of the study of comparative histopathology, Donald Endelow and Armand de Ricqlès (de Ricqlès, 2011, 2007). There are even instances of bone in certain fishes that is completely acellular, lacking the osteocytes found in the bone of all living tetrapods (Shahar and Dean, 2013).

The two most commonly found types of bone tissue in adult mammals are cortical (compact) and cancellous (spongy) bone (Ortner and Turner-Walker, 2003; Weiner et al., 1999; White et al., 2012). Cancellous bone is much more porous and it is typically found at the jointed ends of long bones, as flat plates in the cranium, sternum, and ilium, and underlying tendon attachments (Weiner et al., 1999; White et al., 2012). Cortical bone is primarily found along the diaphysis of long bones, the petrous pyramid and the mandible, and as a thin outer sheath at the ends of long and flat bones (Ortner and Turner-Walker, 2003; White et al., 2012).

Bone undergoes several stages of development over an organism's lifespan. Initial, rapidly-growing bone, which is typically found in foetal development through early childhood, but also found in healing fractures and in certain bone tumours, takes the form of woven or fibrous bone and it is characterized by being less dense with greater histological disorder compared to more mature tissues (Manilay et al., 2013). It is gradually resorbed and replaced by appositional growth of lamellar bone, where layers of new bone stack over one another with a microarchitecture analogous to plywood, or by Haversian bone where specialised teams of cells replace old bone tissues with new bone (Ortner and Turner-Walker, 2003). This remodelling of bone throughout life is controlled by a number of factors, including hormonal triggers that are themselves influenced by diet, reproductive status, exercise and mechanical loading (Kini and Nandeesh, 2012; Robling et al., 2006). Medical literature commonly states a bone remodelling rate of approximately 10% per year, often without reference (e.g. Lerner, 2016). Despite this medical truism, based upon markers of bone turnover measured in body fluids, is shown to be 4-10 times higher than the directly measured rate of turnover in cortical bone assessed measuring either the steady increase in levels of racemization (Ritz-Timme et al., 1999; Ritz-Timme and Collins, 2002) or the incorporation of bomb pulse ^{14}C (Hedges et al., 2007). Similar slow levels of turnover have also been reported for cartilage using direct bomb pulse ^{14}C measurements (Heinemeier et al., 2016; Libby et al., 1964).

2.3 Dental tissues

Mammalian teeth contain four dental tissues. With the exception of pulp, three of these are mineralised (hard) tissues: enamel, dentine (dentin), and cementum (Hillson, 2005). Compared to enamel, tooth dentine is more similar to bone tissues in terms of organic to inorganic ratios (Hillson, 2005) and protein composition (Alves et al., 2011; Jágr et al., 2012; Park et al., 2009; Salmon et al., 2016). Whereas human enamel contains only 5.9 % organic

matter and 2.0 % water, dentine contains 19.2 and 6.5 %, respectively (Teruel et al., 2015). Like bone, dentine contains trace amounts of non-collagenous organic matter (mucopolysaccharides, lipoproteins, etc.) collectively called ground substance (Hillson, 2005). Ivory is essentially the dentine of any large mammal including elephant, mammoth, sperm whale and walrus tusks from which objects can be carved, but also includes narwhal, hippo and wild pig teeth which are suitable only for smaller items. Cementum is the most variable portion of a tooth coating the entire tooth in some mammals, but in others, it may cover just the root (Hillson, 2005). Generally, the thickness of the cementum layer increases with age of the individual and, histologically, cementum has a structure very similar to bone (Elliott, 2002; Hillson, 2005) and has been shown, like the petrous portion, to be a good source of ancient DNA (Adler et al., 2011; Hansen et al., 2017). Enamel is the hardest, densest and least porous of the mineralised tissues, and consequently, it is the most durable of human tissues. In some environments, the enamel tooth crowns survive where all other traces of the skeleton have been destroyed by chemical or microbial attack (Kimbel and Delezene, 2009; Zanolli, 2013). Nevertheless, tooth enamel does undergo diagenesis over timescales relevant to the study of human origins and palaeontology, especially in the study of trace elements and stable isotope content (Gehler et al., 2011; Pellegrini et al., 2011; Tütken et al., 2008; Tütken and Vennemann, 2011).

3. Porosity of mineralised tissues

The porosity of mineralised tissues influences the speed and nature of post-mortem alterations to bones and teeth. The diameter and interconnectedness of the various pores determines the ease with which water, microorganisms, and dissolved ions enter and leave the tissue (Hedges and Millard, 1995). As the most common mineralised tissue in the archaeological record we should look at bone first. Bone is a living tissue containing blood vessels and nerves that enter the bone via one or more relatively large nutrient foramina where they branch into Haversian canals running the length of the bone and Volkmann's canals that allow centrifugal flow of blood from arteries in the marrow cavity to the periosteal veins (Brookes, 1974; Bryan, 1996; White et al., 2012).

Bone cells, which require connection to the blood system and a sensory apparatus to detect mechanical strain, are found in lacunae (i.e. depressions/cavities) that are connected by a fine network of canaliculi (i.e. channels) (Kollmannsberger et al., 2017; Ortner and Turner-Walker, 2003). The lacunae are shaped like flattened ellipsoids approximately 10 μm x 5 μm and each is connected to its neighbours by 50-70 canaliculi, which pass through the hard bone tissue. The cells are approximately 20 μm apart and the average diameter of the canaliculi is approximately 259 ± 129 nm or a quarter of a micron (You et al., 2004). This lacuno–canalicular system forms a syncytium of interconnected cells and the extracellular space within canaliculi is accessible to blood in peripheral vessels (Nango et al., 2016). Confocal laser scanning microscopy of modern human femur shows that compact bone contains approximately 10 million osteocytes per cubic centimetre, each with a volume of approximately 500 μm^3 and implying a total length of the canaliculi in that volume of approximately five kilometres (Papageorgopoulou et al., 2010). In mammals, the larger the body, the lower the density of osteocyte lacunae, with osteocyte lacunar density being around 17,000/mm^3 for hippopotamus, 20,000/mm^3 for humans and 50,000–66,000/mm^3 in mouse femora (Nango et al., 2016).

Porosity has been found to vary considerably between different species (Fig. 2). Dry, acellular human, bovine and porcine compact bone specimens have been measured with total porosities of 9.2%, 5.2% and 14.3 % respectively (Figueiredo et al., 2010). These porosities are made up of three different pore size ranges at the micro- and nano-level: a) vascular porosity (VP), with average pore diameters around 50 μm; b) lacunae-canaliculi porosity (LCP) corresponding to an average pore size of 0.1 μm; and c) collagen-apatite porosity (CAP) related to the voids between and within collagen molecules (Figure 3), and HAp crystallites with diameters of only a few nanometers (Figueiredo et al., 2010; Mansilla et al., 2014).

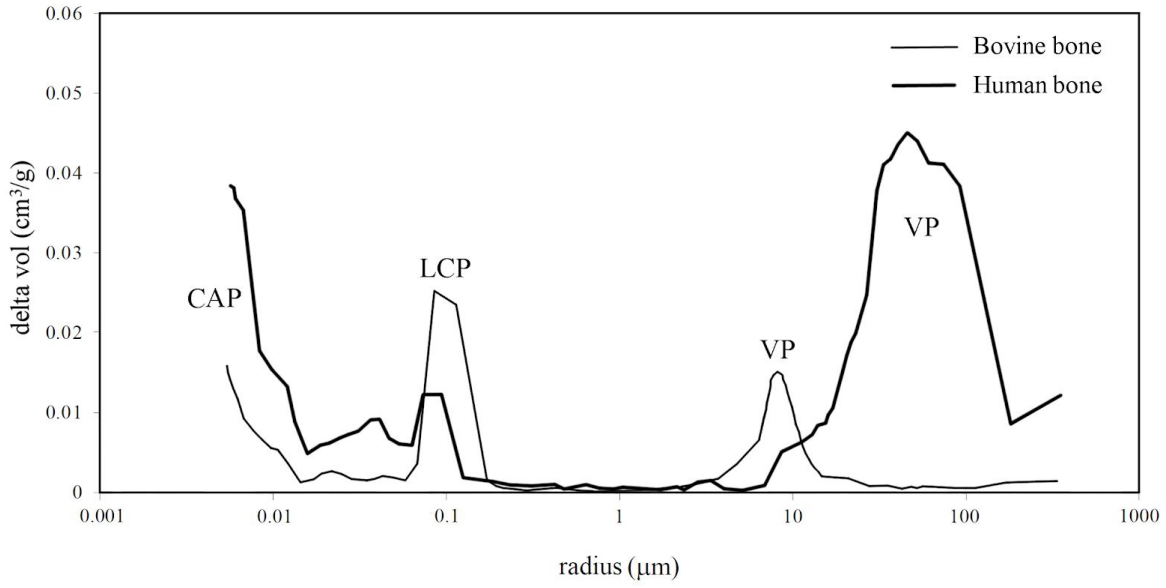

Figure 2 - Differences in porosity of human and bovine compact bone measured by mercury intrusion porosimetry. CAP = collagen-apatite porosity; LCP = lacunae-canaliculi porosity; VP = vascular porosity. (Data adapted from Figueiredo et al., 2010)

A great deal of porosity in dead bone arises from the destruction of soft tissue in the blood vessels and bone cells which are housed in osteocyte lacunae. After loss of soft tissues, the lacuno–canalicular network allows soil water (and associated ions and microorganisms) to infiltrate the bone cortex irrespective of the direction of net flow of water within the sediment. The type and scale of diagenetic alteration influence the porosity of bones post-mortem in such a way that if the porosity decreases the survival prospects of the bone increase with *permineralisation* gradually leading to the fossilization of the specimen. Alternatively, if the porosity increases then the survival prospects of the specimen are severely diminished as water flows increasingly freely through the bone such that it may disappear completely from the archaeological or fossil record. Bones in a free-draining, acid soil, for instance, are particularly prone to this since they suffer both bioerosion and dissolution.

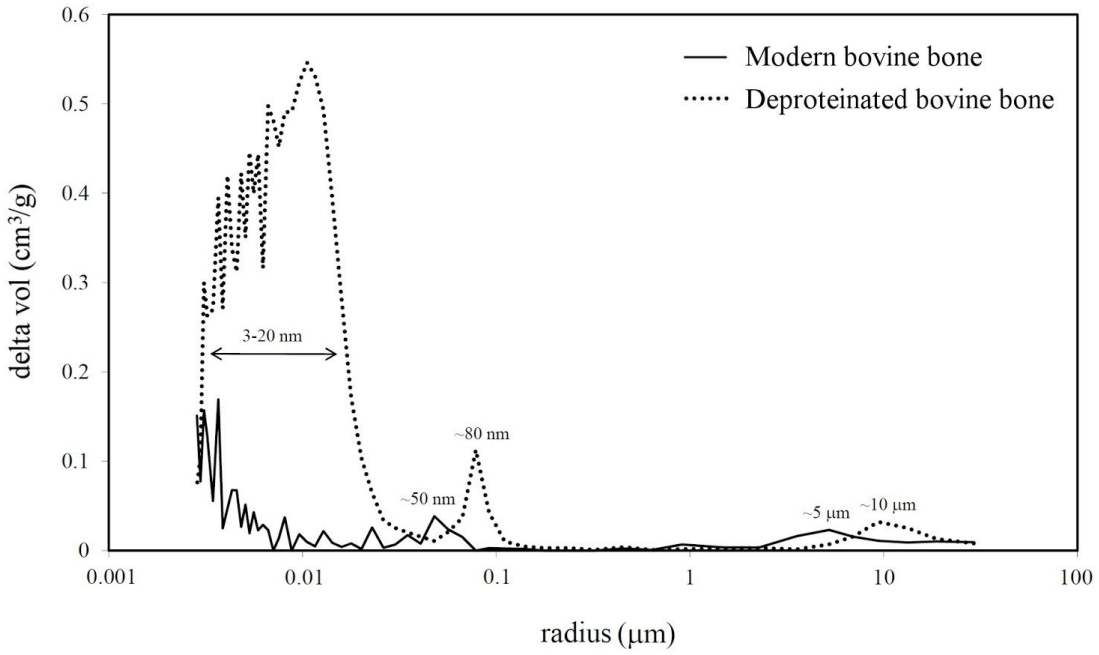

Figure 3 - Changes in porosity of bovine compact bone before and after stripping out the collagen in a non-aqueous medium using hydrazine. Note the large increase in porosity with radii below ~25 nm arising from loss of collagen fibrils. Porosity below 10 nm must reflect pores spaces between HAp crystallites. Note the increase in pore radius and volume associated with the lacunae-canaliculi porosity and vascular porosity revealing a loss of un-mineralised osteoid from their internal surfaces.

The volume of the pore network and its directionality in the other mineralised tissues differ from that of bone. As it might be expected, ivory and tooth dentine is known to survive well in the archaeological and fossil record. The directionality of porosity in different tissues has a very visible influence on patterns of microbial degradation as can be seen in Figure 4 below where, even after considerable bioerosion, the dentine in this tooth root is readily distinguishable from the surrounding cementum. The porosity in dentine has a smaller average diameter than found in bone and does not connect with the exterior of the skeletal element via large Haversian canals (Heckel et al., 2016). The dentinal tubules are not interconnected except via the pulp cavity, which itself is an enclosed volume accessible to soil water predominantly via the apical foramen. This local isolation from sediment pore water may well be reflected in the relatively better preservation of tooth dentine (and associated DNA) compared to bone (Hollund et al., 2014).

The mineralised tissues in antler are more porous, more chaotic in the orientation of blood vessels and have a lower mineral content than the bone found in the axial and appendicular skeleton. One might, therefore, expect it to be more susceptible to diagenesis than human or animal bone. Certainly, antler is less well represented in the archaeological record but this may reflect taphonomic rather than diagenetic factors. Antler is physically weaker and less dense than cortical bone and is more accessible to the predations of scavengers as is often the case with the spongy bone at the jointed ends of long bones that are exposed on the soil surface. By comparison with both antler and bone, ivory is both compact and, being composed of acellular dentine, has a low porosity. Mercury intrusion porosimetry of un-demineralised dentine gives a total pore volume of only 4-5% and a mean pore diameter of ~750 nm (Vennat et al., 2009).

4. Density of mineralised tissues

There is considerable confusion concerning the densities of different skeletal elements arising, in part, from the various different ways of expressing density. Here, a clear distinction should be made between the density values quoted in the osteoarchaeological literature, which is mainly concerned with the survivability and water transport characteristics of different skeletal elements (Boaz and Behrensmeyer, 1976; Lyman, 2014, 1984), and the densities quoted in a clinical context. In the former, density may be expressed as relative density in grams per cubic centimeter (g/cm^{-3}) whereas in the latter, bone mineral densities are measured as grams of mineral per unit area in the projected X-ray shadow of a region of interest (Bonnick, 2000) – although they are more commonly presented in a clinical setting as a comparison with the peak bone mineral density of a healthy 30-year-old adult (T-score) or an age-matched cohort (Z-score).

In bone diagenesis studies, there are two ways to measure density, namely, bulk density in which the measured volume includes the pore space, and the real density (also called material density and/or skeletal density) in which the pore volume is subtracted from the volume used in the calculation (Turner-Walker, 2009; Zioupos et al., 2008). The real density of a skeletal element depends upon the ratios of organic, mineral and water, and as stated above, this may vary considerably from species to species and from one bone element to another (Figueiredo et al., 2010; Zioupos et al., 2000). Regarding faunal measurements of density, an extensive and reasonably up-to-date list of various taxa with density data is available elsewhere (Lyman, 2014). Initially, it was discovered that between deer, sheep, and pronghorn antelope, little density variation was evident between subspecies and species, and larger but nonsignificant variation was seen between genera (Lyman, 1984). Additional studies found similar results and conclude that species of generally similar morphology will have similar bone density values (Lam et al., 1999). Terrestrial faunal bones with higher bulk density values are found to correlate more strongly with survivorship over time (Lyman, 1984), with similar findings in fish (Butler and Chatters, 1994) and aquatic birds (Broughton et al., 2007).

5. Depositional Environment

In vivo, the mineralized tissues experience an environment that is strictly controlled by the body's cellular and metabolic processes that remodel and repair the skeleton, maintain

calcium homeostasis, and limit the action of microorganisms. After death, this protective envelope ceases to function and once skeletonisation has begun the skeleton is exposed to one or more of a wide range of environments that frequently represent an "open system" in terms of physical, chemical, and biological insults to the mineralised tissues. Depending upon the nature of the environment, a skeletal element will undergo a somewhat predictable diagenetic trajectory that may, or may not, result in its preservation in the archaeological or geological record. Of course, if the environment evolves over time, that diagenetic trajectory may change in speed and/or direction such that any excavated bone may exhibit chemical and physical deterioration corresponding to different environments. Nevertheless, it is sometimes possible to untangle these changes in depositional environment with clever use of chemical and histological techniques (Hollund et al., 2012; Pfretzschner and Tütken, 2011; Trueman and Benton, 1997; Turner-Walker and Jans, 2008). In the case of human inhumations, the local environment can change dramatically over short time periods if the decomposing corpse is enclosed within a wooden coffin or stone kist. In the case of a wooden coffin, the skeleton will experience an initially closed system enclosing putrefying soft tissues which becomes progressively more open and free draining once the coffin has begun to decay. The complex chemistry of a decomposing corpse are covered comprehensively in the forensic literature, (e.g. Dent et al., 2004 and references therein). The various factors influencing the survivability of a bone over time and its susceptibility to destructive, rather than preserving diagenetic factors are discussed in more detail below.

5.1 Soil pH and Eh

Because of the difficulties in fully reconstructing past soil burial conditions for archaeological bones, field experiments in which the soil pH, hydrologic regime, and temperatures are monitored have been extremely useful in understanding preserving as opposed to destructive environments. Collins et al. (1995, Figure 1) note the greater susceptibility of collagen to alkali rather than acid pH. However, a study of various mammal, bird and fish bones experimentally buried in a range of pH and hydrologic environments found that the acidic (pH 3.5-4.5) soils are more aggressive than alkaline (7.5-8.0) soils (Nicholson, 1996). Similar results have been reported with human bones (Nielsen-Marsh et al., 2007), implying that it is the mineral and not the protein that controls early diagenesis. Increased availability of hydrogen ions leads bioapatite to readily dissolve (Nielsen-Marsh et al., 2000), because bioapatite is found to be most stable at pH 7.8 (Berna et al., 2004), and begins to dissolve in environments below pH 6.0 (White and Hannus, 1983). Decomposing bodies have been shown to change the pH of the localized burial environment, first by dropping pH levels (Gill-King, 1997), followed by more alkaline conditions (Gill-King, 1997; Janaway, 2008). Acidic environments can also arise from the presence of decomposing biomass producing humic and tannic acids, the leaching of base ions such as calcium and potassium, and the formation of carbonic acid by the dissolution of carbon dioxide (CO_2) arising as a decomposition by-product (Pokines and Baker, 2014).

The colour of excavated bones provides clues to the evolution of the burial environment. The soil profile generally exhibits a succession of chemical regimes with oxygen reduction in the uppermost levels; underlain by successive levels of nitrate reduction; Mn^{4+} reduction, Fe^{3+} reduction; sulphate reduction and methane fermentation at the lowest levels. Bones from anoxic layers tend to be stained brown or black from manganese and iron oxides adsorbed onto the pore structure or fixed within the tissues since their solubilities, and hence availability to enter the pore structure of buried remains and interact with either the

collagen or mineral, are dependent upon both pH and Eh (Mielki et al., 2016; Rengel, 2015). Redox and pH values also influence the availability of fulvic and humic acids which stain bone tissues anything from pale brown to dark brown or almost black such as the almost black bones of two women recovered from the Oseberg Ship burial in Norway (Holck, 2007, pers comm).

5.2 Soil hydrology

One of the most important factors for archaeological preservation and/or modification of mineralized tissues is water in and around the archaeological elements. Originally, three important hydrological scenarios had been outlined: a) diffusive; b) recharge; and c) flow regimes (Hedges and Millard, 1995). Diffusive regimes are found where there is little net movement of or change in the amount of water in an environment, recharge regimes are seen where fluctuations and subsequent wetting and drying cycles occur, and lastly, flow regimes occur when water flows through both the mineralized tissue and the soil, such as rainfall in unsaturated or free-draining sandy soils (Hedges and Millard, 1995; Nielsen-Marsh et al., 2000).

Fluctuating environments are the most damaging to mineralized tissues while diffusive environments tended to produce the smallest amount of diagenetic alteration (Fernández-Jalvo et al., 2010; Field et al., 2003; Hedges and Millard, 1995; Nielsen-Marsh et al., 2000; Nielsen-Marsh and Hedges, 2000a; Reiche et al., 2003; Salesse et al., 2014; Smith et al., 2002; Trueman et al., 2004). Cyclical wetting and drying as well as freezing and thawing regimes cause bones to swell and shrink setting up physical strains which result in radial cracking, flaking, and spalling of the bones (Fernández-Jalvo et al., 2010; Pfretzschner and Tütken, 2011; Pokines et al., 2016). This is commonly observed both in bone weathering (i.e. destruction of bone by various physical and chemical processes) at the surface (Behrensmeyer, 1978; Fernández-Jalvo et al., 2010) and those buried in the soil (Fernández-Jalvo et al., 2010; Pfretzschner and Tütken, 2011).

Archaeological bone (i.e. more porous bone) has been shown to adsorb more water, and more quickly, than does fresh bone, and will in turn have a greater desorption rate (Turner-Walker, 1993). The rate at which water moves into and through archaeological material, combined with the speed of both the wetting and drying of the material factors into the preservation or destruction of mineralized tissues in the archaeological record. Water flowing through a bone will leach out mineral, the rate of removal being a function of pH and the volumes of water moving through the bone. Collagenases are too large to penetrate the intimate association of bone and mineral (the collagen-apatite porosity in Figure 2) but the removal of mineral is a prerequisite for collagen decay (Klont et al., 1991). A leaching regimen will expose demineralised collagen to potential microbial enzymolysis, leading to the eventual destruction of the tissue given enough time (Nielsen-Marsh and Hedges, 2000a; Reiche et al., 2003; Trueman et al., 2004; Von Endt and Ortner, 1984). Since well-drained soils also tend to be acidic and well-aerated, both chemical and microbial action can rapidly degrade a skeleton to a few fragments of compact bone (Johns, 2006) or reduce it to no more than a stain in the soil (Bethell and Carver, 1987). Water also substantially alters the element distribution in the bone, which combined with dissolution and recrystallisation, continues to shift the bone mineral composition closer to equilibrium with the burial environment

(Lambert et al., 1985; Müller et al., 2011; Müller and Reiche, 2011; Ina Reiche et al., 2002; Reiche et al., 1999; Trueman et al., 2004; Tuross et al., 1989; Tütken et al., 2008).

5.3 Bone-humic interactions

The breakdown of vegetable matter in the soil is a complex process which lies outside the focus of this review paper. However, because the end results of these processes are often reactive organic molecules that can not only form permanent or semi-permanent cross-links in collagen but also sequester calcium from bone mineral, a short discussion is pertinent.

Humic substances are formed by the modification of plant matter, especially lignin, and the resulting molecules are very resistant to further biodegradation. Although they may derive from various sources, their general properties are very similar and the humic matter in soils may be very diverse. Humic substances may be divided into humic acids, fulvic acids and humins, distinguishable by their different solubilities in water at different pHs. Typically humic substances are large polyphenolic molecules containing phenolic and carboxylic groups. The presence of these carboxylate and phenolate groups gives them the ability to form complexes with ions such as Mg^{2+}, Ca^{2+}, Fe^{2+} and Fe^{3+}. Many humic acids have two or more of these groups arranged in such a way to enable the formation of chelate complexes with divalent and trivalent ions (Evangelou and Marsi, 2001). There is considerable evidence that these humic substances in the soil can form random, non-specific cross-links with bone collagen (van Klinken and Hedges, 1995).

In bones from wet soils (rather than waterlogged), the outer 1-2 mm where the bones tissues have been in direct contact with the soil are partly demineralised and stained a much darker brown than the underlying cortical bone (Turner-Walker, 2008, 1993). This phenomenon is also seen on the fracture edges of broken bones, and thus, does not reflect microstructural differences in the periosteal surface. An extreme example of this demineralisation is seen in the so-called mound people such as the Egtved Girl (Breuning-Madsen and Holst, 1998). These Bronze Age finds are inhumations in oaken log coffins in which the skin, hair, fingernails, and textiles are exceptionally well preserved but the skeleton has vanished completely, leaving only the enamel tooth crowns (Thomsen, 1929). It may be speculated that tannins derived from the oak coffin, combined with waterlogged, anoxic conditions are responsible for both the loss of bone mineral and the spectacular preservation of proteinaceous materials.

Another breakdown product of vegetation that has a bearing on bone diagenesis is found in sphagnum peat bogs and in peat derived from sphagnum. Research has identified a chemical breakdown product of sphagnum mosses, namely sphagnan, a polysaccharide which has powerful tanning and calcium chelating properties (Painter, 1991, 1983). This polysaccharide has been postulated as responsible for the spectacular preservation of the bog bodies found during peat cutting in northern Europe and Scandinavia. Typically, in these finds, the skin, intestines, clothing, and cordage are preserved in a semi-tanned state, whereas, once again, the skeleton has either been completely demineralised or has disappeared entirely. Bog bodies have been found in peat derived mainly or almost exclusively from sphagnum mosses. Field experiments over several years have demonstrated that bones buried in a sphagnum peat bog suffer much more rapid surface demineralisation than those buried in a fenland bog (Turner-Walker and Peacock, 2008).

6. Microbial degradation (bioerosion)

6.1 Patterns of microbial bioerosion

In the bone diagenesis literature, microbiological alterations of bone are generally divided into damage caused either by fungi (Marchiafava et al., 1974), bacteria (Hackett, 1981; Jackes et al., 2001), or in marine and freshwater environments by aquatic microorganisms (Bell et al., 1991; Davis, 1997; Pesquero et al., 2017, 2010; Turner-Walker, 2012). Studies on microbial post-mortem alterations of mineralised tissues have some antiquity. In the 19th century, Wedl was the first to describe microbial activity on human dentine (and later a piece of horse rib) that he had soaked in well water (Wedl, 1864). Wedl assumed the causal organism he saw to be a fungus (Bell, 1995). As such, Wedl was not only the first to report microbial degradation of mineralised collagen but was one of very few researchers to replicate this in the laboratory. Wedl subsequently went on to look at ancient and fossil specimens in which he identified similar tunnelling. Towards the end of the 19th century Roux (1887) and Schaffer (1895) went on to examine fossil bones and teeth with similar results, and similarly concluded that the causal agent was a fungus which Roux named *Mycelites ossifragus*. Rather interestingly, this term has also been used to describe microborings in fossil fish teeth, dinosaur eggs and seashells (Taylor et al., 2014).

Investigations into the tunnelling of ancient skeletal remains resumed in the 1950s (e.g. Sognnaes, 1959, 1956) and have continued up to the present day (e.g. Turner-Walker and Jans, 2008). Hackett (1981) first characterised tunnelling presumably caused by fungi as Wedl microscopical focal destruction (MFD) and tunnels attributed to bacteria as non-Wedl MFD. He described three distinct types of non-Wedl MFD: linear longitudinal, budded, and lamellate (Hackett, 1981). Today, the characterisation of the microorganisms responsible for bone degradation is typically identified by the type of tunnelling present in the bone (Jans, 2008), because success in culturing and identifying the microorganisms has proved elusive.

Marchiafava et al. (1974) are frequently cited as having successfully created tunnels in bones using inoculation with *Mucor* sp. isolated from fresh cadaver vertebrae buried in garden soil. What is less frequently reported is that they autoclaved their bone specimens at 100-200 ºC before inoculating them with fungi. This is hardly a good model for bone degradation in human interments but may well be a good model for domestic bone waste. Furthermore, it is telling that although the fungi grew luxuriantly in the unsterilized soil/bone samples, it only grew sparsely on the sterilised bones – suggesting that the bones may not have been the major source of nutrition for the microorganism. It is also interesting to note that when Piepenbrink inoculated X-ray sterilised bone with a range of fungi he found no tunnelling but did describe rapid and uniform growth of fungi which penetrated the vascular channels (Piepenbrink, 1986). This suggests that heating the bone above the gelatinisation temperature of mineralised collagen can disrupt the protein-mineral bond and leave the bone tissues more susceptible to fungal degradation.

Bones exposed above the sediment (i.e. exposed to light) in a marine or freshwater environment are rapidly tunnelled by aquatic microorganisms which are able to penetrate to a depth of around 500-700 μm below the surface, leaving meandering tunnels 5-10 μm across (Turner-Walker, 2012). These tunnels are tightly-spaced, with typically less than one micron between tunnel walls, and appear not to respect the bone microarchitecture. They branch only

occasionally and some tunnels contain clusters of spherical grains of redeposited HAp. These grains vary in diameter from 1-2 μm (Pesquero et al., 2010) and are slightly enriched in manganese (Turner-Walker, 2012) suggesting that the organisms responsible may belong to the cyanobacteria family. It is interesting to note that in Wedl's original research on tunnelling found in teeth, his samples were immersed in "untreated well water" and that the tunnels that had formed after 10 days did not penetrate deeper into the tissues than 0.2-0.25 mm. After immersion for more than a month the organisms responsible were unable to tunnel further. He also described some "few shining grains appearing in the interior" of the tunnels (Wedl, 1864). This raises the intriguing possibility that Wedl's original tunnelling, so often now attributed to fungi (Hollund, 2013; Jans, 2008; Jans et al., 2004; Müller et al., 2011) may actually have been due to cyanobacteria after all.

Bones excavated from the majority of terrestrial archaeological contexts are unlikely to exhibit tunnelling by cyanobacteria. When Wedl-type bioerosion in exhumed bones is attributed to fungi it is most commonly, if not exclusively, along the periosteal margins of bones, whereas non-Wedl MFD commonly occurs around vascular (Haversian) canals (Lam et al., 2009). The dimensions of the tunnels described by Wedl were around 8 μm in diameter. Other researchers have described varying dimensions for different morphologies of MFDs ranging from 5 μm for Type I Wedl tunnels, 5-10 μm for linear longitudinal and anything up to 60 μm for budded and lamellate tunnelling (Jans, 2008).

It is hazardous, and not supported by available evidence, to assign different causative organisms to these different sizes and morphologies of MFDs. Sognnaes (1955) found a similar range of sizes (2-10 microns; 15-25 microns and 50-100 microns) of focal destruction in ancient teeth from Europe and America. He thought that tunnels of different diameters did not mean that they were caused by different organisms but rather were caused by differences in the tissue type tunnelled. This explanation is supported by data coming from mercury intrusion porosimetry (Jans et al., 2004) which shows that all of the MFD types – linear longitudinal, budded, lamellate and even Wedl tunnels – have very similar porosities, exhibiting the double-peaked trace normally attributed to bacterial tunnelling, which supports the interpretation of Sognnaes (1955). The influence of tissue microarchitecture is dramatically demonstrated by the change in the pattern of diagenetic alteration at the transition from cementum to dentine in Figure 4.

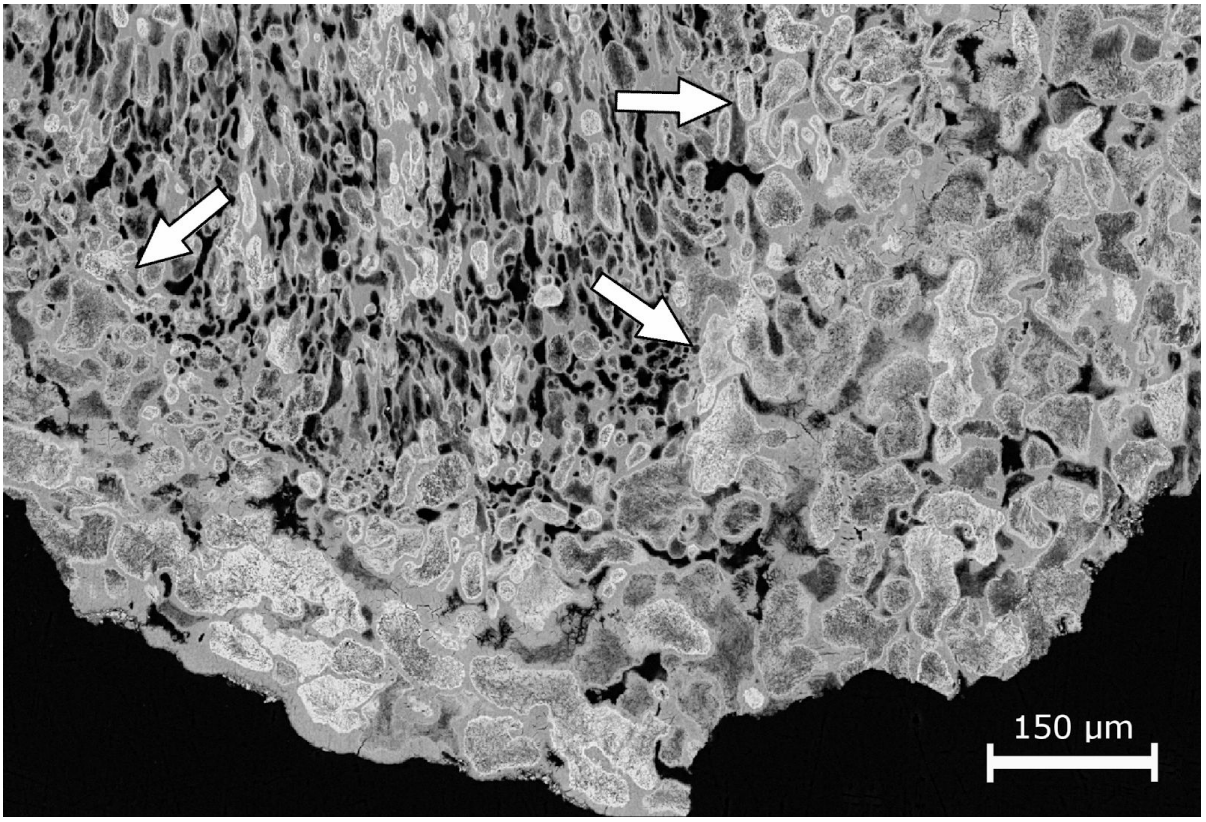

Figure 4 - Differences in the pattern of bacterial degradation between tissue types in the root of a human incisor from the Medieval site of Wharram Percy, UK. Arrows indicate the border between dentine and cementum.

Bacterial tunnelling is associated with a dissolution and redistribution of HAp within the affected zones (Turner-Walker et al., 2002), as was noted by both (Hackett, 1981) and (Bell, 1995) in earlier studies. In fact, Hackett specifically noted the importance of seasonal variations in soil water content and their importance not only for the proliferation of the tunnelling bacteria but also the pattern of distribution (or loss) of the mineral (Hackett, 1981). The most parsimonious explanation for the different morphologies and sizes of MFDs is that one or more closely related species of bacteria are responsible for tunnelling into mineralised collagen and that perceived differences are due to a combination of tissue microarchitecture and local hydrologic flow within and surrounding the bone. Tunnelling that occurs close to the periosteum or in any surface in close contact with the surrounding soil is more likely to be empty because solubilised mineral has a greater likelihood of being swept out of the bone. This may account for some of the Wedl-type porosity attributed to soil fungi penetrating the periosteal surface in some specimens (see Fig. 5A). Bacterial degradation that occurs deeper inside the tissues is more likely to result in any dissolved mineral re-precipitating within the tunnelled space, leading to the observed pattern of often ragged demineralised spaces interspersed with hypermineralised cuffs or infillings which represent HAp re-precipitated within the tissues (Turner-Walker and Syversen, 2002).

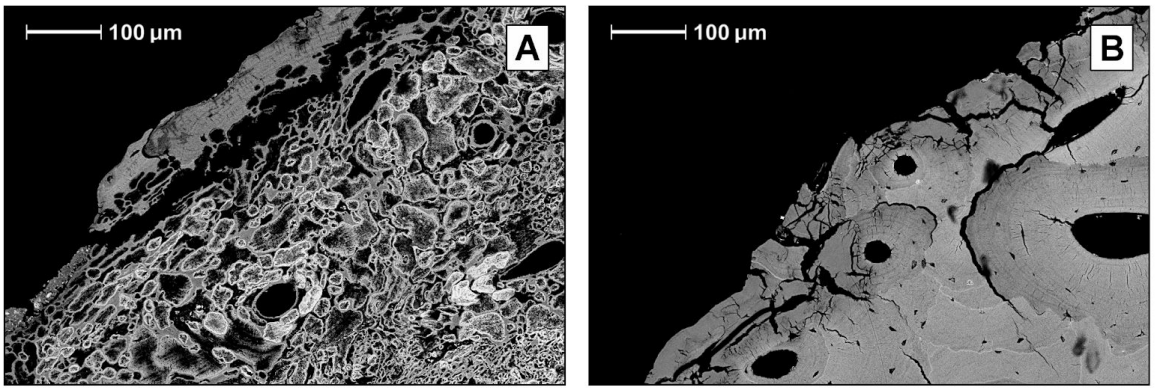

Figure 5 - A: BSEM image of human bone from the Medieval monastery at Cleeve Abbey, UK. This specimen is from the free-draining part of the site and is extensively tunnelled by bacteria. Note that the periosteal bone is characterised by empty pores ~10 µm in diameter, whereas the outer 50 µm where it has been in contact with the soil is un-tunnelled. B: BSEM image of bone from the waterlogged part of the cemetery. Bacterial tunnelling is absent but there is considerable demineralisation and cracking of the periosteal bone. Demineralisation appears to respect the cement lines delineating secondary osteons.

As recognition of the far seeing contribution of Hackett, it is worth quoting him with regard to how he saw future developments in the subject of post-mortem alteration of bone microstructure. "*The application of more sophisticated techniques would contribute to the fuller understanding of these changes and their causes*" (Hackett, 1981). It is somewhat surprising, therefore, that so many researchers persist in applying low-power optical microscopy to the question of diagenetic alterations to bone tissues.

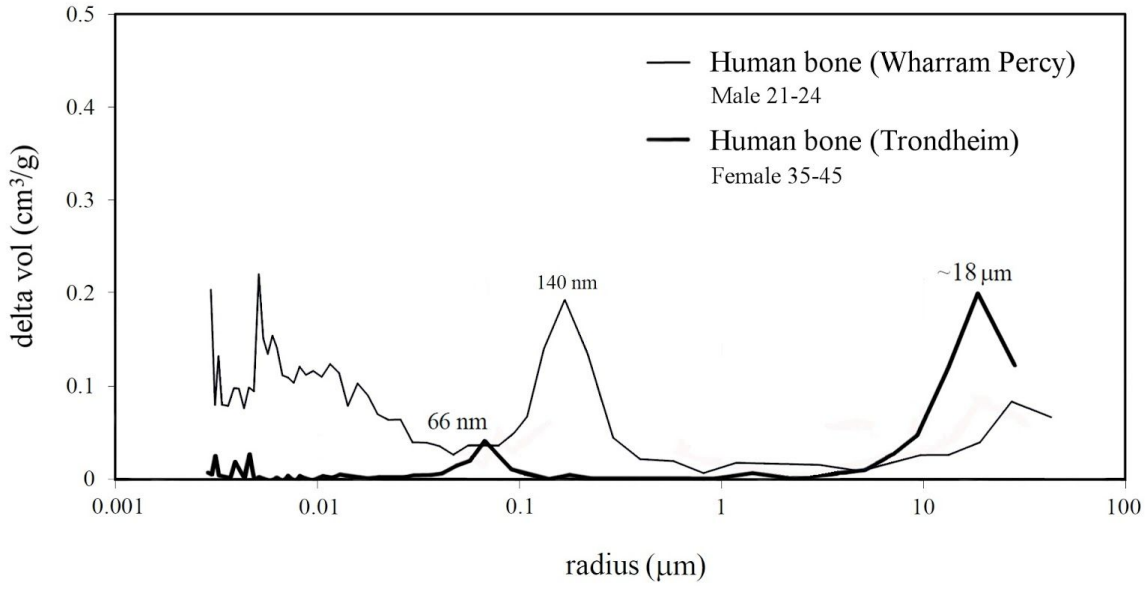

Figure 6 - Differences in porosity for human femora from Mediaeval individuals excavated from two different inhumation environments – aerated soils (Wharram Percy, UK) and waterlogged, anoxic (Trondheim, Norway). Note the increased vascular porosity in the older, female specimen compared to the young male. (unpublished data MJC) as well as the increase in the collagen-apatite porosity in the bone from the warmer, aerated soil.

6.2 Environment and microbial bioerosion

The soil environment is without doubt the dominating factor in whether a buried bone will survive within the archaeological record or whether it degrades rapidly and is lost. Skeletonised remains in the tropics degrade much more rapidly than those in temperate zones, which in turn degrade more rapidly than those close to the arctic circle. Similarly, deeply buried remains tend to survive for longer than those in shallow burial environments. Part of this is because temperature is an obvious factor but so too is access to oxygen. Once again, (Hackett, 1981) was prescient in his understanding of the relationships between post-mortem changes to skeletons and burial environment - "tunnelling in bone does not appear to develop readily in wet, or waterlogged conditions. Moderate soil moisture with periodical, seasonal variation of water table and temperature, e.g. warm summers, are favourable for tunnelling." This conclusion is supported by evidence from the Medieval cemetery at Cleeve Abbey, UK. This cemetery has burials from both free-draining and waterlogged soils and the skeletons show a dramatic difference in microscopic preservation state (Fig. 5 A & B).

The soil bacteria responsible for tunnelling in buried bones are evidently aerobic in their metabolic pathways. Bones that are buried in wet soils with restricted access to oxygen tend to exhibit none of the characteristic diagenetic signatures seen in bones buried in oxygenated soils. Occasionally, bones exhibit limited bacterial tunnelling but show evidence for diagenetic pyrite within the tunnelled tissues that is testament to a transition from an oxygenated to anoxic environment (Turner-Walker and Jans, 2008). Bones from deeply-buried, water-saturated sediments tend to have a higher collagen content and lower porosity (Fig. 6) than similar specimens from free-draining, aerated soils, although because of the deposition of iron sulphides within their pore structure they are prone to rapid deterioration if the environment changes to one with freely available oxygen (Turner-Walker, 2009).

6.3 The origin of the bacteria responsible for tunnelling in bone

It is argued that due to the low frequency of microbially altered bones in the fossil record, microbially degraded bones seldom survive into the geological record (Trueman and Martill, 2002). It is certainly true that archaeological bones and teeth tend to fall into a bimodal distribution on the Oxford histological index (OHI) scale - being either well preserved or poorly preserved (Hedges et al., 1995; Hollund et al., 2014). It has also been demonstrated that bacterial alteration in human bones is more than twice as common as in animal bone (Jans et al., 2004).

These findings have initiated a debate as to whether the way bones have entered the archaeological record (e.g. articulated- vs. disarticulated bodies) could have an influence on whether the bacteria responsible for non-Wedl tunnelling originate from the gut microbiome or from the burial soil post-skeletonisation (Bell, 1995; Damann and Jans, 2017; Jans et al., 2004; White and Booth, 2014). If a link between the bacterial alteration of bone and the presence of gut microbes exists, it can have great implications for the interpretation of the early post mortem period. In whole body decomposition, the bloat event, where the body cavity ruptures, is suggested to be a key point where a shift in the diversity of microbes from anaerobic to aerobic is observed (Bucheli and Lynne, 2016; Metcalf et al., 2013). A link between bone bioerosion and soft tissue decomposition would provide strong evidence that non-Wedl tunnels are produced by an organism's enteric gut microbiota and that these

bacteria migrated from the intestines in the first few days after death, and go on to penetrate the bone microstructure (Booth and Madgwick, 2016; Jans, 2008; Kellerman et al., 1976).

Contrary evidence comes from an experimental burial of whole pig carcass which showed no specific histological attack patterns and no clear relationship between histological preservation and proximity to the abdominal area after 7 years (Kontopoulos et al., 2016). More pertinently, Bell et al. (1996) found no evidence of bacterial degradation of any kind in the skeletons (ribs) of three executed prisoners exhumed after 70-83 years from wooden coffin burials in Canada. These had been isolated from the soil and the coffins were dry when exhumed. We can infer a short interval between death and interment because execution and burial of condemned prisoners under the British Commonwealth followed strict protocols and bodies were buried promptly, without autopsy, soon after the individual was declared dead. Supporting evidence for tunnelling bacteria originating in the burial environment is provided by the identification of this characteristic tunnelling in archaeological ivory artefacts (Large et al., 2011).

Taphonomic setting undoubtedly influences the microstructural decay of bones. In forensic science, the use of genetic analysis to study the microbial diversity in the soil around a body has recently been investigated to estimate time since death (Metcalf et al., 2013; Pechal et al., 2014, 2013). A study by (Metcalf et al., 2015) found that 40% of the microbes responsible for the decomposition of the cadavers are already present in the soil before placement of the body and that other sources, such as insects, may provide a large proportion of the microbial decomposer community. The findings of Metcalf and colleagues are interesting as White and Booth (2014) found in their study of whole pig burials, that there was no bioerosion on the bones of stillborn pigs, which were considered not to have developed microbiota, and therefore concluded that exogenous soil bacteria do not contribute to microbial attack of bones over the first year of decomposition. However, they note that heavy rainfall during the first few month of the experiment could have created anoxic waterlogged conditions that slowed down degradation.

7. Protein degradation

7.1 Collagen

Collagen is linked with the strength of bone (Saito and Marumo, 2010; Schultz, 1997), and loss of collagen is responsible for the weakening of bone structure, potentially causing loss of the skeletal element entirely. Collagen loss happens due to both biological and chemical degradation. Biological loss of collagen, such as from microbial tunneling, assists in the destruction of mineralized collagen due to collagenase properties found on many microbes (Child, 1995a, 1995b; Marchiafava et al., 1974). Due to the size of the microbes and collagenases, which are both too large to fit into the collagen-apatite porosity, bone mineral would first have to be removed to expose the collagen triple helix (Child, 1995a, 1995b; Hedges, 2002; Marchiafava et al., 1974; Nielsen-Marsh et al., 2000; Turner-Walker, 1993) as it is during bone resorption. Microbial tunneling has been described earlier and is clearly a principal mechanism of collagen loss in environments and sites where bone degrading bacteria can flourish (Balzer et al., 1997; Child, 1995a, 1995b; Marchiafava et al.,

1974; Nielsen-Marsh and Hedges, 2000a; Smith et al., 2002; Turban-Just and Schramm, 1998).

Chemical destruction of collagen, however, is influenced by many factors. Collagen is a protein held together by peptide bonds, and peptide bonds can be broken down by hydrolysis (Collins et al., 1995; Smith, 2002). Initial studies demonstrated that collagen degradation was a by product of both temperature and time, but increased collagen loss over shorter amounts of time corresponded with an increase in mean temperature (Ortner et al., 1972; Von Endt and Ortner, 1984). Similar results were also seen with cooked bone, where higher temperatures yielded more efficient collagen loss (Roberts et al., 2002; Solari et al., 2013). Figure 7 depicts collagen loss across a temperature gradient. Soil conditions, such as either extreme acidity or alkalinity can exacerbate the effects of hydrolysis, causing collagen to be lost much more effectively (Collins et al., 1995; Smith et al., 2002). Higher numbers of cross-links (i.e. stronger bone structure) retain collagen better over time than does bone with little, or no cross-linkage (Collins et al., 1995). However, close examination of this model reveals that it is flawed as it demands too large a number of cross-links to explain the stability of chrome-tanned collagen. Subsequently, an alternative hypothesis, the link-lock model, shows it was physical constraint, rather than cross-links, which prevent collagen from melting (Covington et al., 2008).

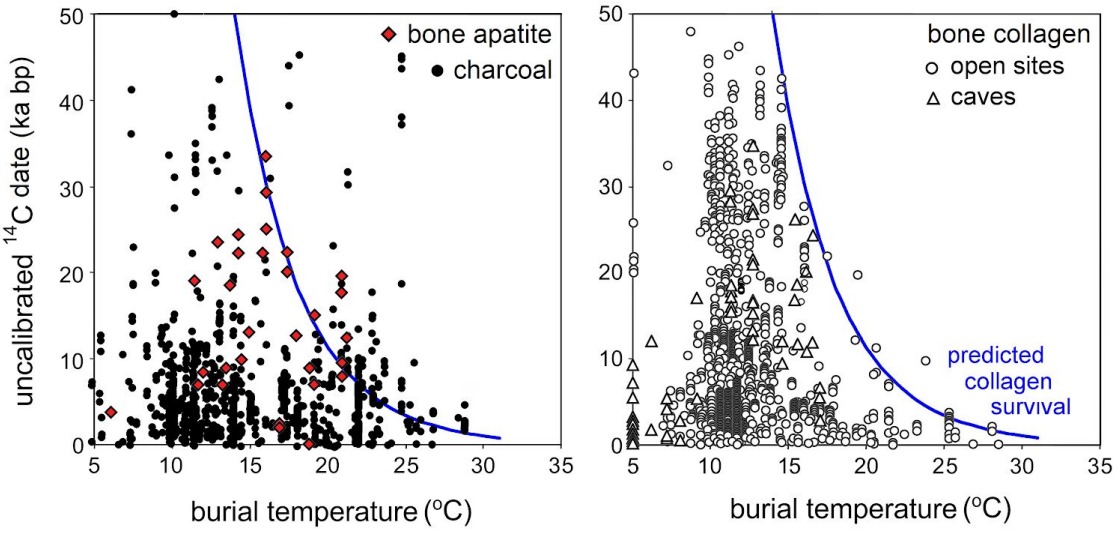

Figure 7 - Plots of radiocarbon dates of charcoal and bone apatite (left), and bone collagen (right) against temperature of burial environment. The blue line indicates the theoretical limit for collagen survival. Drawing adapted from (Buckley et al., 2008b; Smith, 2002).

Therefore, an intricate collection of processes are necessary for the complete removal of the collagen content of an archaeological specimen in less than several thousand years. It has been shown that areas around cracked bone surfaces lose collagen much more rapidly than do non-cracked regions (Boaks et al., 2014; Trueman et al., 2004). These cracks may be due to either taphonomic conditions such as gnawing or weathering, or from density mediated attrition, where less dense bones are more likely to crack over time (Lyman, 2014). If they occur, cracks can then allow exogenous material into the bone, drastically altering the mineral structure (Smith et al., 2007), indicating that protein loss of bone in general is not an independent process, and proceeds in concert with other diagenetic changes such as (Collins

et al., 2002; i.e. microbial attack and deterioration of the inorganic content, Nielsen-Marsh and Hedges, 2000a; Trueman et al., 2004). Bone interaction with water can also increase the speed at which these processes work (Smith, 2002). Essentially, the process of collagen loss can be described as a rapid initial loss of collagen as a byproduct of increasing temperature, followed by a secondary, slower rate of decay of endogenous bone collagen due to chemical damage over extended periods of time (Turner-Walker, 2011).

Chemical degradation and loss of collagen can be identified as an increase in the smallest porosity in many archaeological bones (Turner-Walker et al., 2002). Comparing Figures 3 and 6 above it is clear that bone deposited in an aerated, free draining soil (Wharram Percy) exhibits an increase in porosity at the collagen-apatite level compared to the bone from the waterlogged site (Trondheim). Figure 8A shows a high resolution BSEM image of bone that has not been bacterially tunnelled. Superficially, it appears that the smallest pore spaces (~30-50 nm) may represent lost collagen fibre bundles. However, a secondary electron image of an EDTA- treated sample of the same bone (Figure 8B) shows that these pores actually represent the inter fibrillar spaces that have suffered mineral loss. This supports the hypothesis that collagen and bone mineral are mutually protective, leading to the long-term survival of mineralised tissues in the burial environment, and that disruption of this intimate association will make bone tissue more susceptible to diagenesis (Koon, 2006; Turner-Walker, 2008).

7.2 Other Proteins

Skeletal material is made up of a series of proteins that are less abundant than collagen. Osteocalcin is one of many non-collagenous proteins found in skeletal material and comprises between 10-20% of the non-collagenous protein of bone (Ajie et al., 1992). Osteocalcin (Bone Gla Protein) is responsible for calcium binding and mineralization regulation (Lian et al., 1978; Price et al., 1976). Matrix Gla protein is similar in construction to osteocalcin, while proteoglycans and sialoprotein are phosphoproteins (Al-Qtaitat and Aldalaen, 2014). Phosphoproteins are likely responsible for calcium binding (Mundy, 1999). Lipids and proteolipids, along with alkaline phosphatase, are attributed to mineralization processes in bone and tooth (George and Veis, 2008). Other proteins in bone include a host of cell-attachment proteins and proteins responsible for regulating growth and cell activity (Al-Qtaitat and Aldalaen, 2014). For the purposes of this review, only osteocalcin diagenesis will be discussed, as a thorough literature search of diagenetic studies on these other proteins yielded no results.

Osteocalcin is typically bound to bone bioapatite (Tuross, 1993) and due to this, similarly to collagen, osteocalcin loss is linked with increased bioapatite crystallinity (Collins et al., 2000; Smith et al., 2005), microbial destruction of bone (Smith et al., 2005), and temperature (Buckley et al., 2008). There is evidence from FTIR studies that 1-2 wt.% of the protein in fresh bone is resistant to hydrazinolysis. Either this may represent osteocalcin or collagen that is tightly bound to, or surrounded by, mineral. Collagen has been found to survive longer than both DNA and osteocalcin in ancient material (Buckley et al., 2008). Therefore, this indicates that at present collagen may be relatively easier to detect and analyze in older mineralised tissues.

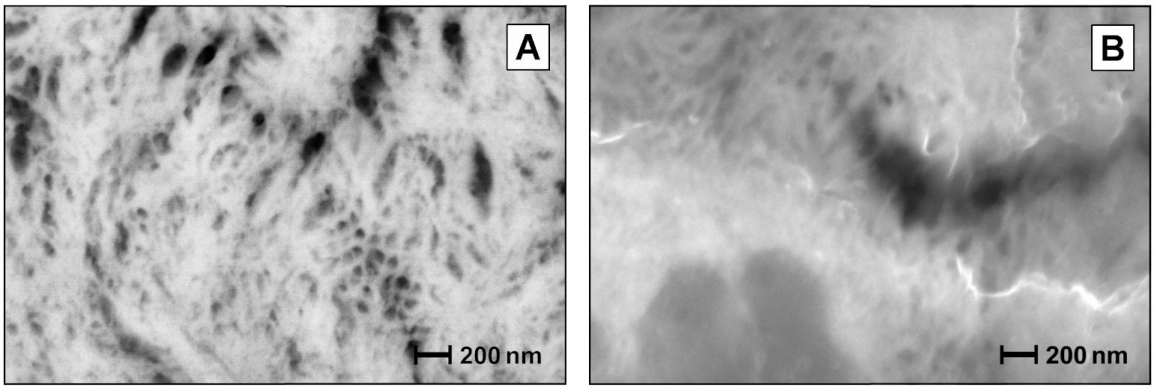

Figure 8 - A: High resolution BSEM image of un-tunnelled cortical bone from Cleeve Abbey. The section was treated with NaOCl for 60 seconds. Larger pores (100-200 nm) are canaliculi. On initial inspection the smaller pores appear to be where collagen has been removed. However, if we look at a secondary electron image of a sample from the same bone treated with 0.1 Mol EDTA for 60 seconds (shown in Figure B) we see that bone mineral from between the fibre bundles has been removed, exposing partly demineralised collagen fibres. (Compare Figure 5D with (Habelitz et al., 2002): Fig. 1)

8. Mineral changes

Crystallinity changes in archaeological bone were discovered when larger bioapatite crystals were noted in both museum specimens of eight ancient humans compared to modern, wild-killed Wildebeest (Tuross et al., 1989). In the modern samples, crystallite size was seen to increase over a period of 10 years, while the ancient samples showed significantly larger crystallite size in comparison to the modern samples (Tuross et al., 1989). As biogenic apatite is a relatively unstable and disordered form of geological mineral, its survival post-mortem depends on reorganization and growth through dissolution (loss of less stable components) and recrystallization (formation of more stable structure) (Nielsen-Marsh et al., 2000; Trueman, 2013). Although early bioapatite diagenetic changes are still unclear (Keenan and Engel, 2017; Tuross et al., 1989), post-mortem alterations of its biogenic chemical signals and composition have long been recognized (Lee–Thorp, 2002; Lee-Thorp and van der Merwe, 1991; Shin and Hedges, 2012; Stiner et al., 2001; Wright and Schwarcz, 1996).

Bone recrystallization increases crystallinity (i.e. crystal size and lattice perfection) and promotes the formation of a more stable, more authigenic mineral phase characterized by lower carbonate content as the initial more disordered crystals are less energetically stable (Asscher et al., 2011; Bartsiokas and Middleton, 1992; Götherström et al., 2002; Gourion-Arsiquaud et al., 2008; King et al., 2011; Nielsen-Marsh and Hedges, 2000b; Person et al., 1995; Reiche et al., 2002; Salesse et al., 2014; Stathopoulou et al., 2008; Surovell and Stiner, 2001; Termine and Posner, 1966; Trueman, 2013; Wright and Schwarcz, 1996). An increase in average and maximum crystal length post-mortem may be due to: i) particle coarsening, a process whereby the larger crystals increase at the expense of the smaller ones (i.e. Ostwald ripening); ii) dissolution of the smaller crystals; iii) or both (Nielsen-Marsh et al., 2000; I. Reiche et al., 2002; Stiner et al., 1995; Trueman, 2013; Trueman et al., 2008, 2004; Tuross et al., 1989; Weiner and Bar-Yosef, 1990; Wright and Schwarcz, 1996).

Increases in crystallinity have been used as one of the parameters to characterise the degree of diagenetic alteration of ancient degraded bones (Asscher et al., 2011; Dal Sasso et

al., 2016; Salesse et al., 2014; Trueman et al., 2004; Tuross et al., 1989; Weiner and Bar-Yosef, 1990) and as a possible screening method for DNA analyses (Grunenwald et al., 2014a). Nevertheless, despite its widespread use as a measure of diagenesis the splitting factor or crystallinity index measured from FTIR spectra is not always a predictor of other measured parameters (Trueman et al., 2008). The degree of recrystallization and change is usually higher in fossil bone comparing to archaeological bone (Berna et al., 2004; Keenan, 2016; Smith et al., 2002). Mineral changes require active hydrology (recharging with fresh water) or a reduction in pH (Hedges, 2002; Hedges and Millard, 1995; High et al., 2015; Nielsen-Marsh et al., 2000; Trueman, 2013). Berna et al. (2004) and Keenan and Engel, (2017) demonstrated that hydroxyapatite is more stable in pH around 7.5-8, displays low solubility (recrystallization) in slightly alkaline to near neutral environments and gives way to dissolution and recrystallization in pH environments below 6 or over 9. This indicates that bone apatite is more soluble than authigenic apatites that form naturally from solution in the soil, meaning that bones lying within this "recrystallisation window" begin to more closely reflect the composition of soluble ions in the burial environment (heteromorphic recrystallization) (Fernández-Jalvo et al., 2016).

Protein loss can also influence crystallinity (Nielsen-Marsh and Hedges, 2000a; Smith et al., 2007; Trueman et al., 2004) as the loss of organic matrix in mineralised tissues may expose some poorly crystalline mineral fractions to water flowing through the bone, allowing leaching and reprecipitation onto existing crystals and into the empty pore spaces. This interaction of the groundwater with the hydrated layer of the apatite crystals allows the incorporation of diverse labile and reactive ions (e.g. HPO_4^{2-}, PO_4^{3-}, CO_3^{2-}, Ca^{2+}, Mg^{2+}), which may in turn substitute other ions into the core domain (Figueiredo et al., 2012; Lee-Thorp and van der Merwe, 1991; Stathopoulou et al., 2008). This ionic exchange is controlled by the pH of the groundwater which can vary from 2.8 to 10 (Nielsen-Marsh et al., 2000; Turner-Walker, 2008). These episodes of dissolution and recrystallization result in a loss of tensile strength (i.e. bone becomes more susceptible to fracture) and mass (High et al., 2015; Turner-Walker, 2011). Changes in the composition of bone apatites also take place, even in neutral to alkaline soils.

Recrystallization can also lead to a distinct drop in microporosity accompanied by increasing macroporosity as seen at a variety of post-Ice Age, European, archaeological sites (Nielsen-Marsh and Hedges, 2000a, 2000b), in human, deer, goat, and sheep bones of between 1 and 55,000 years of age (Berna et al., 2004), fossilized remains of African animals (Trueman et al., 2004), and in assorted, experimentally altered bone (Hiller and Wess, 2006; Turner-Walker, 2011). Crystallite change and porosity alteration, however, is not always due to Ostwald ripening. Microbial tunnelling, in addition to mineral dissolution and recrystallization, also alters crystallinity (Turner-Walker et al., 2002). Recrystallization into authigenic apatite may be assisted by bacterially mediated mineral precipitation (Carpenter, 2005, 1999; Daniel and Chin, 2010). For microbes, this is in good agreement with research showing that microbes would first have to alter the mineral component of bone in order to get to the collagen (Nielsen-Marsh et al., 2000; Turner-Walker, 1993). Microbial activity can, therefore, lead to mineral dissolution, followed by a redistribution of phosphate for the growth of adjacent hydroxyapatite crystals (Trueman et al., 2004).

9. Ancient DNA degradation

Deoxyribonucleic acid (DNA) is a macromolecule responsible for the construction of cellular proteins, which is passed to daughter cells during cell division. DNA is a polymer made up of four nucleotide bases - adenine and guanine, which are double-ringed purines, and cytosine and thymine that are single-ringed pyrimidines - all of which are attached to a deoxyribose sugar and a phosphate group, which make up the structural "sugar-phosphate backbone" of DNA (Jobling et al., 2013). Adenine and thymine pair in double-stranded DNA, as do guanine and cytosine (Jobling et al., 2013). Carbon atoms make up the deoxyribose molecule, where phosphate groups attach at the 3′ and 5′ (3 prime and 5 prime) carbon atoms, where the 3′ and 5′ each have an unattached hydroxyl (-OH) group on these respective carbon atoms (Jobling et al., 2013). A phosphodiester bond is then formed when a 3′ and 5′ carbon from differing sugar molecules conjoin through a covalent bond resulting in the formation of the double-stranded sister bond characteristic of DNA (Watson and Crick, 1953).

Ancient DNA (aDNA) analysis concerns itself with getting DNA out of "old" tissues (Sarkissian et al., 2015), that is, specimens that are typically older than 100 years of age. However, post-mortem, DNA is very susceptible to attack and degradation (Dabney et al., 2013) with quite limited calculated survivorship potential (Allentoft et al., 2012). DNA degradation commences with autolysis, taking place only a few hours after the death of an organism (Burger et al., 1999; Parsons and Walter Weedn, 2006; Pruvost et al., 2007). DNA is rapidly degraded by endonucleases such as lysosomes that cut DNA strands at internal sites (Hofreiter et al., 2001; Pääbo et al., 2004; Parsons and Walter Weedn, 2006). Bacteria and other external microorganisms also proliferate and digest DNA strands with exonucleases from the ends (Pääbo et al., 2004; Parsons and Walter Weedn, 2006; Pruvost et al., 2007).

Initial research believed that bacterial attack was a likely cause for the total loss of endogenous DNA in a sample (Rollo et al., 2002). However, this is unlikely, as many samples commonly produce exogenous bacterial fingerprints while still producing authentic, endogenous DNA (Zaremba-Niedźwiedzka and Andersson, 2013). Also endogenous bacterial attack affecting the cortical bone tissue may preserve endogenous DNA, while environmental conditions may also build reservoirs or niches where DNA is preserved (Geigl, 2002). Hydrolytic breakage of the polynucleotide strands is considered the main long-term reaction in aDNA degradation followed by chain breakage into shorter fragments usually between 100-500 bp in length (Bada et al., 1999; Brown and Brown, 2011; Hofreiter et al., 2001; Lindahl, 1993; Pääbo, 1989). The DNA molecule is particularly prone to this, as water attacks the β-N-glycosidic bonds and/or the phosphodiester bonds (Brown and Brown, 2011; Lindahl, 1993; Parsons and Walter Weedn, 2006). The attack to the phosphodiester bond leads to single-stranded nicks, whilst the attack to the glycosidic bond releases the nucleotides (Pääbo et al., 2004).

Environmental conditions play a key role in the preservation of endogenous DNA (Poinar, 2003). DNA decay is highly temperature-dependent (Allentoft et al., 2012; Lindahl, 1993; Lindahl and Nyberg, 1972). Low temperatures have an advantageous effect on the survival of DNA, whereas high temperatures result in significant loss of DNA (Burger et al., 1999; Poinar, 2003). DNA yields are much higher in tissues from permafrost areas than from moderate temperate environments, while the latter better preserve DNA compared to warmer

areas such as the Mediterranean (Pruvost et al., 2008). A north-south decline in endogenous DNA yields has been reported in the Northern Hemisphere (Bollongino et al., 2008), with a 67% success rate for central European open-air sites compared to only 7% for Near Eastern open-air sites (Bollongino et al., 2008). Caves also undisputedly preserve DNA more effectively as they have stable low temperatures all year-round and the alkaline sediment environment usually protects bone apatite from dissolution (Bollongino et al., 2008). Any variations in long-term DNA fragmentation is assumed to be caused by differences in pH and local hydrology (Allentoft et al., 2012; Burger et al., 1999; Lindahl, 1993; Lindahl and Nyberg, 1972).

Hydroxyapatite crystals are assumed to protect DNA from further degradation as it adsorbs onto the crystal surface (Götherström et al., 2002; Lindahl, 1993; Parsons and Walter Weedn, 2006). Mineral preservation has been linked to the survival of DNA in bone, while its dissolution and recrystallization are related with considerable loss of DNA in archaeological specimens (Allentoft et al., 2012; Götherström et al., 2002). Collagen loss is also assumed to play a crucial role in DNA degradation (Sosa et al., 2013). Experiments modelling the loss of DNA over time have shown that there is a drastic loss of DNA within the first year of burial, which causes a concomitant loss of DNA from the organic fraction of bone as collagen is removed (Campos et al., 2012). As a result, higher proportions of DNA are found more regularly in the bioapatite portion (Campos et al., 2012). This is also in agreement with later studies that found DNA was intimately linked with the maturation, and thus increase in size of bioapatite crystallinity, and the preservation of DNA in bones between 0 and 3,800 years of age (Grunenwald et al., 2014b). A possible explanation of this is that chemical processes, such as hydrolysis and collagenases, are removing proteins, and mineral is then filling in the gap spaces, while other processes are denaturing cells and releasing DNA from the collagen so it then freely binds to the newly formed bioapatite surfaces. This explanation is lent additional support by the observation that in bone artificially aged in water at 60 ºC there are changes in both microporosity and tensile strength associated with dissolution and reprecipitation of mineral as collagen undergoes early degradation (Turner-Walker, 2011). However, the exact mechanisms of DNA preservation in ancient bones remains under debate (Götherström et al., 2002; Ottoni et al., 2009).

Some skeletal elements have been shown to yield larger amounts of DNA than others, meaning that it is easier to obtain authentic DNA from these samples (Gamba et al., 2014; Hansen et al., 2017; Pinhasi et al., 2015; Pruvost et al., 2008). For instance, the petrous bone, one of the inner ear portions of the temporal bone of the skull, has had remarkable success in providing large amounts of endogenous aDNA, which has been attributed to its density, believed to be protecting it from potential destructive attack (Gamba et al., 2014; Pinhasi et al., 2015). However, in other skeletal elements, the unique porosity of human compared to animal bone means contaminating DNA (exogenous DNA) may enter human bone much more easily in an archaeological setting (Gilbert et al., 2005). In teeth, it has been demonstrated that cementum is a much more rich source of aDNA than the typically used dentine, which may be explained by higher mineral content of cementum (Adler et al., 2011; Damgaard et al., 2015; Hansen et al., 2017). It should be noted that in mineralising collagen, the mineral grows at the expense of interstitial water, and if that water contains DNA fragments then increasing mineralisation will result in more DNA becoming bound up in the mineral phase.

10. Conclusions and suggestions for future research

We have outlined a thorough synthesis of all the ways in which mineralized tissues can be altered through the processes of diagenesis. This palimpsest of diagenesis has highlighted the key and influential research from the initial forays into this research field and connected it with more recent research and innovative techniques attempting to explain diagenetic phenomena. We have illustrated the ways in which diagenesis can work to preserve bone, and agree with other researchers that central to this preservation is the interplay between the organic and inorganic constituents and how the environment interacts with the skeletal tissues. We conclude with others that diagenesis works as a collective process, since bone and tooth are made up of intrinsically linked components. Finally, we have argued that while histological preservation is important for samples, this comes at a cost of lowered recoverability of the proteins and endogenous DNA.

Diagenetic processes are all interlinked. A loss of collagen causes the DNA to be lost from the organic fraction. Thus, this causes recoverable DNA to be extracted more readily from bioapatite as time goes on until total loss of DNA is observed (Campos et al., 2012). Some microbes are believed to be assistive in bone survivorship over time (Daniel and Chin, 2010; Smith et al., 2007; Trueman and Martill, 2002), whereas others (those responsible for tunnelling) actually hamper collagen preservation (Child, 1995a, 1995b; Marchiafava et al., 1974), assist in at least some loss of DNA (Rollo et al., 2002), and influence biogenic isotope values through selective feeding (Balzer et al., 1997; Grupe et al., 2002; Turban-Just and Schramm, 1998). Research into exactly how microbes work in the diagenetic framework is key, since they appear to be influential in all aspects of diagenesis of mineralised tissue. They have been shown to remove collagen, alter the inorganic portion of bone, and erode the structure of bone through tunnelling. Further, diverse microorganisms are found in all types of environments and have a hand in both the preservation and destruction of mineralised tissue. Therefore, they are an influential aspect of diagenesis, yet their role remains the least understood.

Limited interaction with water is key for the preservation of the organic and inorganic contents, as well as DNA (Dabney et al., 2013; Smith, 2002). Environments where bone and water interaction remain static preserve bone better than do environments where cyclic wetting and drying occurs (Field et al., 2003; Hedges and Millard, 1995; Nielsen-Marsh et al., 2000; Nielsen-Marsh and Hedges, 2000a). Increased temperatures also affect collagen survival (Ortner et al., 1972; Turner-Walker, 2011; Von Endt and Ortner, 1984) and tend to accelerate loss of DNA (Shapiro, 2015) compared to cooler temperatures, while soil pH has been shown to influence the preservation of skeletal remains (Nielsen-Marsh et al., 2007; Turner-Walker and Peacock, 2008).

Future research could usefully investigate any possible influence of *in vivo* disease on post-mortem diagenetic degradation of bone tissues. One obvious area is the detection of ante- and peri-mortem infections in archaeological skeletons. Staphylococcus DNA has successfully been identified in bones exhibiting osteomyelitic infection in an historic osteological collection (Flux et al., 2017). It remains to be seen if this can be repeated on archaeological specimens. And whether the infectious lesions influence diagenesis. Non-infectious diseases have also been shown to result in microdamage to bone tissues. For example, non-enzymatic cross-linking, called advanced glycation end-products (AGEs),

causes micro-damage to living bone tissue, leading to microcracks while simultaneously halting the repair process by stimulating osteoblast apoptosis (Alikhani et al., 2007; Tang and Vashishth, 2010). Microcracks are seen in the early stages of diagenesis (Hollund et al., 2012; Pfretzschner and Tütken, 2011) and may be expected that AGE compromised tissues may experience accelerate chemical degradation of bone tissue (compared to bone comprised of normal collagen) by increasing both porosity and surface area available to percolating pore waters. However, at the present time we are unaware of any way to explore this hypothesis apart from perhaps using artificially AGE-damaged bone and comparing its diagenetic change to unaltered samples.

Because of recent developments in which the presence, or absence, of bacterial tunneling in human remains has been attributed to specific cultural practices, it is of considerable importance that the question of the origin of the organisms responsible is resolved. Identification based on recovered bacterial DNA is a tantalising prospect but is likely to be definitive. Field and laboratory investigations, combined with histological analyses and DNA studies seem to offer a fruitful area of future research. The soil bacteria versus endogenous gut bacteria hypotheses may prove more difficult to disentangle than it seems, however, since many gut bacteria are commonly found in the soil. Nevertheless, the fact that archaeological ivory is also tunnelled in the same way as bones (Large et al., 2011) suggests that the diagenetic trajectory can be followed in isolation from a decaying corpse. Finally, much of the published research on bone diagenesis to date has focussed on European archaeological sites. Although that material covers terrestrial, freshwater and marine environments from the sub-arctic to the Mediterranean, the range of environments and climates explored should be expanded. That being said, results of field experiments in tropical southeast asia and archaeological bones examined from the Chinese mainland would indicate that differences in diagenesis are one of degree rather than substance.

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