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THE LONG-TERM SURVIVAL OF BONE: THE ROLE OF BIOEROSION

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Fossil bones (N = 350) spanning more than 350 million years, and covering a wide range of depositional environments, were studied to compare the distribution of microbial destruction features in fossil bones with previously published data sets of bones of archaeological age. The distribution of bioerosion in fossil bones is very different from that found in bone from archaeological sites. Fossil bones typically show little or no bioerosion. Under normal conditions, if a bone is to survive into the fossil record, then rapid bioerosion must be prevented (or halted). This conclusion suggests that early post mortem processes, such as the mode of death, influence the potential of any bone to survive into deep time.

KEYWORDS: BONE, FOSSILIZATION, MICROBIAL ATTACK, BIOEROSION, MFD, DIAGENESIS, FUNGI, PRESERVATION, TAPHONOMY

INTRODUCTION

The processes leading to bone fossilization are rather complex, but as interest in the geochemistry and biochemistry of fossil bone increases (see, e.g., Barrick and Showers 1995; Collins *et al.* 1995; Schweitzer *et al.* 1997), it is important to understand the how, why and when of bone fossilization. The process of fossilization itself is difficult to define, but for the purpose of this paper, fossilization of bone refers to those processes of change that confer long-term stability to buried bone.

We consider bone fossilization to be a multiphase process involving alteration of the organic fraction of bone matrix, changes to the mineral component at the molecular and/or crystallite level, additional growth of phosphate and infilling of vascular spaces by diagenetic minerals. To understand the processes of bone fossilization, it is useful briefly to consider the nature of bone itself.

The nature of bone

Bone is a composite material, constructed from an intimate association of protein fibres and mineral crystals. The protein phase (principally collagen) provides flexibility, and forms the matrix upon and within which mineral crystals are grown. The mineral phase provides strength, and is composed of small crystals of a carbonated calcium phosphate ($Ca_{10}(PO_4{\cdot}CO_3)_6OH_2$), a

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member of the apatite group. Bone apatite crystals are extremely small, with a correspondingly high surface area of around 200 $m^2 g^{-1}$ (Weiner and Price 1986). Bone crystallites are plate-shaped, of the order of 30 nm in length and breadth, and just a few unit cells thick. This small size means that bone apatite crystals are reactive, and will dissolve in pore waters that are undersaturated with respect to apatite (Nriagu 1983; Karkanas *et al.* 2000). Consequently, once removed from physiological equilibrium conditions, bone apatite crystals are vulnerable to dissolution. However, bone crystals are held within a framework of collagen, and the collagen provides protection for apatite crystallite surfaces, so that fresh bone does not rapidly dissolve in sea water. The collagen matrix therefore provides protection for the apatite crystallites.

Degradation of bone collagen

Collagen is a refractory protein that is insoluble under normal physical and environmental conditions, and rapid collagen hydrolysis requires enzymes collectively known as collagenases (Child 1995). However, the hydrolysis of *unmineralized* collagen can be catalysed by both hydroxyl and hydrogen ions (Collins *et al.* 1995). Collins *et al.* (1995) argue that low-temperature abiological hydrolysis of *mineralized* (i.e., bone) collagen will also occur but, given the relatively high stability of mineralized collagen, this process will be much slower than enzyme-mediated collagen hydrolysis.

Under normal soil conditions, unmineralized collagen is decomposed rapidly. However, mineralized collagen can survive into the archaeological record, indicating that it must be biologically unavailable under some circumstances. Microbially mediated collagen hydrolysis may occur rapidly: however, the intimate association of bone mineral with the organic phase appears to protect the collagen molecule.

Polymers such as collagen are too large to penetrate microbial cell membranes, and so enzymes (collagenases) must be excreted by the microbes, and the polymers broken extracellularly (Child 1995). In dried bone, water molecules can diffuse freely into mineralized collagen along intercrystalline spaces (Child 1995). These spaces are too small to allow the passage of even slightly larger molecules such as ethanol (Lees 1989). Larger molecules such as collagenases could not gain access to fully mineralized bone, and for collagen to be available for microbes, the bone mineral must be put into solution so that enzymes can gain access (Collins 1995). Bone apatite crystallites and collagen fibres thus enjoy a state of mutual protection, affording greater stability to both components in burial environments.

Microbial bone decomposition

Most buried bone does not survive into the fossil record. As suggested above, the principle agent behind the initial degradation of bone is microbial attack. Microbes (bacteria, fungi and protozoa) demineralize the bone, producing one of several types of histological destruction (either tunnels or borings). These microscopical destructive features are easily recognized in thin sections or on polished surfaces of bone, and have received considerable study (see, e.g., Wedl 1864; Roux 1887; Schaffer 1889; Morganthaler and Baud 1956; Martill 1989; Bell 1990; Bell *et al.* 1991, 1996; Davis 1997). Hackett (1981) provided a comprehensive account of bioerosion in bone, and described four readily recognizable forms of bioerosion. Bell (1990), Child (1995) and Hedges *et al.* (1995) use the term ‘microscopical focal destructions’ (MFD), after Hackett (1981), to refer to any microbially produced change in histomorphology. In modern and archaeological bones, Wedl-type tunnels are relatively scarce, and MFD is usually

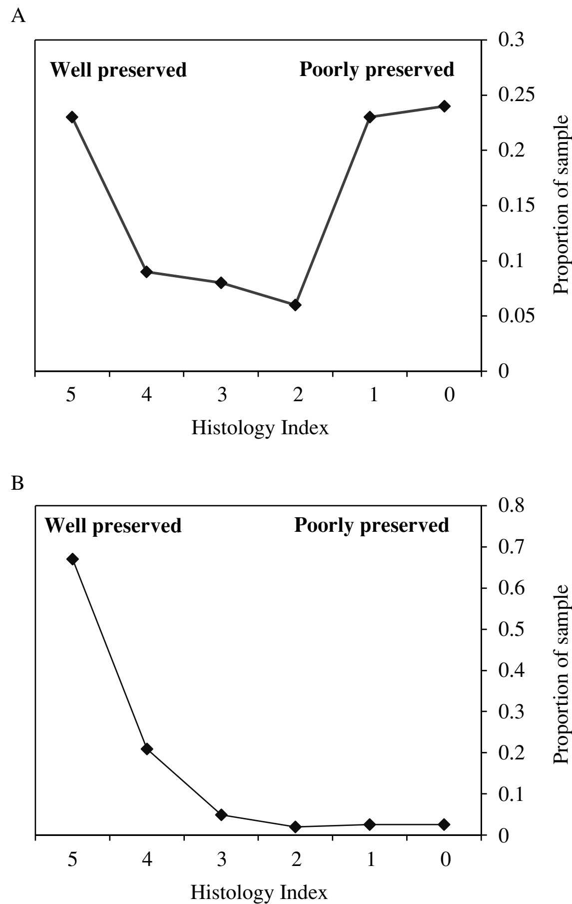

Figure 1 *The distribution of histology index scores in bones of (A) archaeological age (taken from Hedges* et al. *1995) and (B) fossil bones (Silurian–Pleistocene).*

manifested as either small holes or hypermineralized nodules, probably broadly equivalent to the linear longitudinal tunnels described by Hackett (1981).

The extent of bioerosion in a suite of archaeological bones ranging from 100 to 50 000 yr was determined by Hedges *et al.* (1995). They devised a 'histological destruction index' (the Oxford Histology Index, OHI) based on an estimation of the amount of bone affected by bioerosion. The OHI ranges from 5 (<5% affected, bone histology identical to fresh bone) to 0 (no original histological features visible). Hedges *et al.* (1995) found a distinct U-shaped distribution in histological index values that had no relationship to age or burial environment (Fig. 1 (A)). This was interpreted as suggesting that, once started, bioerosion progresses rapidly to complete destruction of the bone. Bones in the advanced stages of degradation (OHI index 0) are weakened structurally, have an increased pore-water flow, and thus would not be expected to survive into the fossil record. If this is true, then only those bones that *never* experience bioerosion will survive into the fossil record, unless this process can be interrupted by rapid physical or chemical changes.

Histology of fossil bone

Despite several records of bioerosion in fossil bone (Roux 1887; Bystrov 1956; Hackett 1981; Martill 1989), few studies investigating fossil bone histology refer to bioerosion and the accompanying loss of histological detail. Indeed, many workers refer to the generally excellent preservation of fossil bone. Fossil bone commonly retains the birefringent pattern seen in fresh, proteinated bone, indicating that the original alignment of apatite crystals (with their *c*-axes parallel to collagen fibres) is retained, despite the loss of the collagen fibres (Hubert *et al.* 1996).

The model developed by Hedges *et al.* (1995) and others predicts that mineralogical changes occurring during bone fossilization should accompany abiological collagen degradation, and therefore fossil bones should show a different distribution of histological index values to archaeological bones. This assumption has never been tested objectively to our knowledge. The aim of this study, then, is to determine the frequency, type and distribution of bioerosion in fossil bones.

METHODS

A suite of 350 thin sections of fossil bone was examined, ranging in age from Silurian to Pleistocene, and covering marine, lacustrine, fluvial and soil environments. All sections were examined under reflected and transmitted light with a petrological microscope, and scored according to the Oxford Histology Index (Hedges *et al.* 1995). Any erosive tunnels were classified according to Hackett (1981). Thin sections were taken from the collections of Bristol University; the National Museum and Gallery of Wales, Cardiff; Portsmouth University; and the British Museum of Natural History, London.

Applying the Oxford Histology Index to fossil remains

The histology index designed by Hedges *et al.* (1995) cannot be directly applied to an investigation of the effect of biological attack on fossil bones, as abiological processes of recrystallization also result in the loss of histology. To avoid this problem, we simply estimated the extent of bone affected by unequivocally microbial alteration. We recognize that this conservative approach may underestimate the true extent of bioerosion.

A potential problem with working from museum collections is that very poorly preserved bones are unlikely to be collected or sectioned. Thus the sample could be biased in favour of well-preserved, histologically intact samples. However, Hanson and Buikstra (1987) tested the hypothesis that histological preservation is independent of whole bone preservation, and could not reject this hypothesis on the basis of a G-test of independence. They state that 'A thin section displaying histological alteration is just as likely to come from a macroscopically well preserved femur as from a femur that is less well preserved' (Hanson and Buikstra 1987, 553). Consequently, we are confident that our use of museum collections will not bias our results significantly. A more intractable problem arises as our pooled sample is composed mainly of multiple bone samples recovered from a few localities. Diagenesis and bioerosion appear to be strongly site-specific, so that unequal sampling of sites will lead to sampling bias. Consequently, we have not attempted to draw any conclusions regarding environmental specificity of bioerosion from our data set. A brief summary of our sample set is provided in Table 1.

Table 1 *Locality, age and environmental data for thin-section samples of fossil bone analysed for bioerosion*

Locality (localities)	*Environment/lithology*	*Age*	*N*
Ludlow, central UK	Ludlow bone bed, marine	Silurian	1
Northumberland, UK	Main coal shale, swamp	Carboniferous	125
South Wales, UK	Carboniferous limestone, marine	Carboniferous	3
Unspecified, South Africa	Karoo, terrestrial fluvial/soil	Permian	17
Unspecified, Texas	Unspecified terrestrial	Permian	1
Argentina	Neuquen Fm, fluvial/soils	Triassic	2
Unspecified, California	Unspecified terrestrial	Triassic	2
Bessano, Italy	Unspecified marine	Triassic	1
Gloucestershire, UK	Durdham Down fissure fill deposit	Triassic	3
Gloucestershire, Glamorgan, UK	Westbury Fm, marginal marine bone bed	Triassic	55
Lyme Regis, Dorest, UK	Lias, marine	Jurassic	1
Holzmaden, Germany	Lias, anoxic marine	Jurassic	4
Oxfordshire, Gloucestershire, UK	Stonesfield slate, coastal marine	Jurassic	5
Tanzania	Tendaguru, coastal plain?	Jurassic	7
Southern and south-central UK	Oxford and Kimmeridge Clay Fm, open marine	Jurassic	15
Swanage, south-central UK	Durlston Fm, marine carbonate bone bed	Jurassic	11
Southern Tunisia	Chenini Fm, Fluvial coastal plain bone bed	Cretaceous	5
Unspecified, Transylvania	Unspecified terrestrial	Cretaceous	1
Unspecified, Nigeria	Unspecified terrestrial	Cretaceous	3
Southern UK and Isle of Wight	Wealden, coastal plain, lake muds	Cretaceous	46
Western Montana, USA	Two Medicine Fm, fluvial/soil	Cretaceous	16
Dinosaur Provincial Park, Alberta, Canada	Dinosaur Park Fm, fluvial	Cretaceous	10
Southern UK and Isle of Wight	Cambridge Greensand, marginal marine	Cretaceous	2
Southeastern UK	Chalk, marine	Cretaceous	2
Unspecified, USA	Unspecified terrestrial	Cretaceous	4
Unspecified	Terrestrial Mesozoic		12
Eastern UK	London Clay, marginal marine	Eocene	1
Siwalik hills, Pakistan	Siwalik Group, fluvial/soil	Pliocene	1
Unspecified, Guadolupe	Unspecified terrestrial	Pleistocene	1
Unspecified	?	?	1

RESULTS

Morphology of tunnels in fossil bone

Of the bones studied, 32% showed signs of bioerosion, which was in most cases very limited, commonly <5% of the examined area. Of the bioeroded bones, 56% contained Wedl-type tunnels, while 50% of bioeroded bones contained tunnels classified as non-Wedl MFD (linear longitudinal tunnels).

Wedl-type tunnels

Wedl-type tunnels are characterized by interconnected asterorhisal networks of tunnels, extending out from the initial point of entry. In the fossil material examined, two morphologies of tunnel fall into this category.

Type 1 Wedl tunnels

Type 1 Wedl tunnels are by far the most common type of Wedl tunnel, found in 56% of all bioeroded bones. These tunnels are relatively simple, branching, three-dimensional networks commonly 10–15 μm in diameter, and the edges of the tunnels appear dense and mineralized (Fig. 2 (A)). Tunnels appear to cross-cut one another, although these tunnels may in fact be separated in the plane of the thin section.

Type 1 Wedl tunnels may be found as well-organized branching networks, as seemingly random almost reticulated networks or, most often, as single tunnels or simple branched tunnels concentrated at the exterior margins of bones. The development of type 1 Wedl tunnels is not obviously controlled or impaired by the bone microstructure, and type 1 Wedl tunnels readily cross osteonal boundaries.

Type 2 Wedl tunnels

Type 2, or 'small', Wedl tunnels were found in only six bones (5% of all bioeroded bones). These tunnels differ from type 1 tunnels in tunnel diameter, branching frequency and the complexity of the tunnel network. Type 2 Wedl tunnels are relatively fine, generally *c*. 5 μm in diameter. They form complex networks, extending from osteonal canals into adjacent osteonal bone (Fig. 2 (B)). Such networks of type 2 Wedl tunnels may pass into adjacent osteonal systems, and are not evidently impaired by cement lines.

The two forms of Wedl tunnels described above are not found concentrated around post-mineralized fractures, even though these frequently act as conduits for mineral-rich fluids, suggesting that invasion occurred prior to fracturing and permineralization. Type 1 tunnels are concentrated on bone surfaces, generally the perisoteal and endosteal margins, and less commonly at the edges of individual trabeculae, whereas type 2 tunnels are found almost exclusively associated with osteonal bone. While almost a third of all fossil bones examined contained some Wedl-type MFD, the majority of these bones contained very minor, localized type 1 Wedl tunnels, often limited to a single tunnel on the exterior margin of the bone.

Focal destruction

Clear focal destruction is found in 49 cases (14% of the total population of bones, and 44% of all bioeroded bones).

In transverse section, non-Wedl MFD normally appears as small, permineralized holes or destructive foci, often concentrated within osteonal systems. These destructive foci vary in size, but are generally between 10 and 30 μm in diameter. The edges of these holes are sharp and mineralized. Destructive foci are seen most commonly within osteons, adjacent to, but not in contact with, the osteonal canal. Destructive, mineralized foci are seldom seen in isolation: normally, an affected osteon will display at least five or six discrete foci, which may abut one another, but do not appear to cross-cut (Fig. 2 (C)).

Longitudinal sections

In longitudinal section, non-Wedl MFD is seen as longitudinal tunnels around 10 μm in diameter, with dense mineralized edges. In some sections, these tunnels are clearly hollow. They appear to originate at the edges of osteonal canals, and extend through the osteonal bone

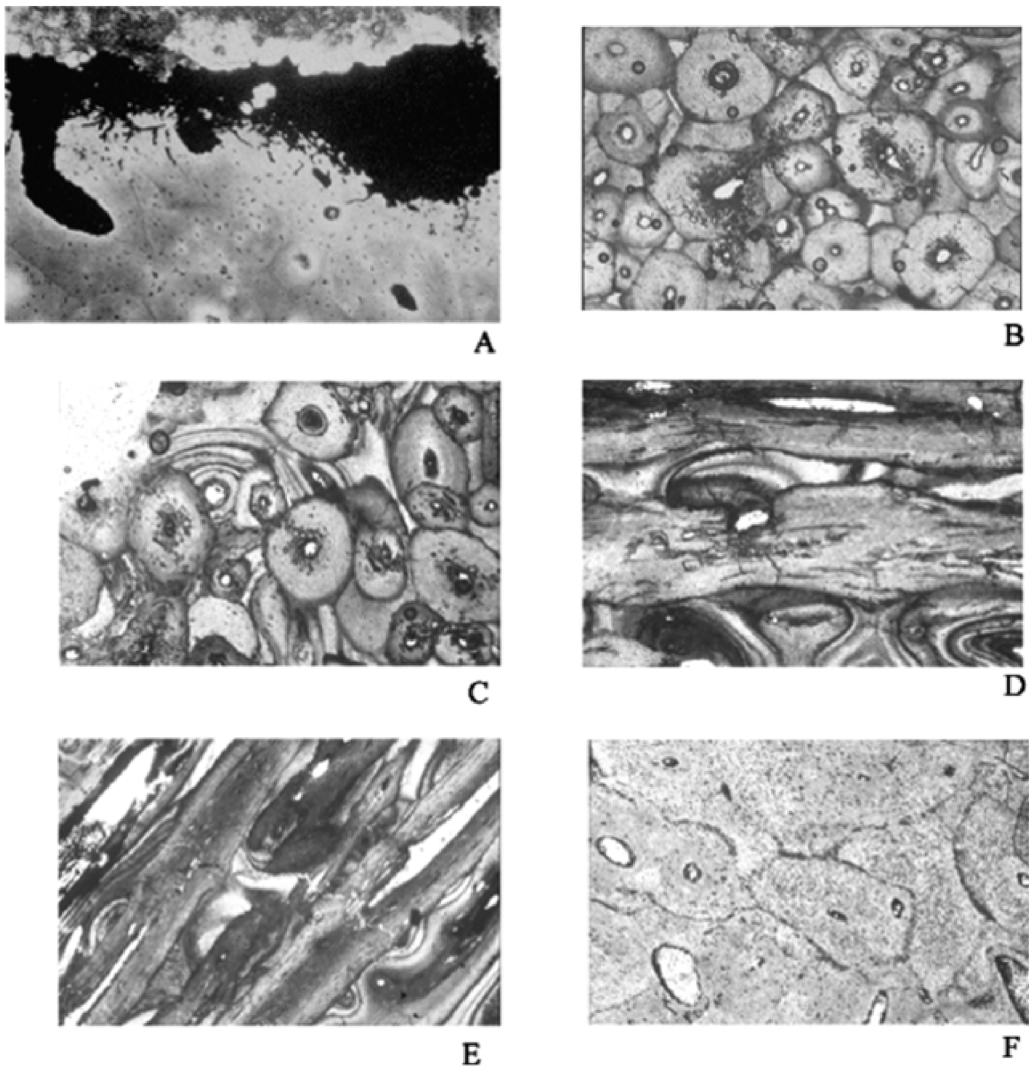

Figure 2 *Histomorphological alteration in fossil bones. (A) Wedl tunnels extending from the perisoteal surface of a fragment of turtle carapace (Lower Cretaceous, Durlston Fm, Dorset, England). Transmitted light. F.O.V. = 2.5 mm. Tunnels are filled with opaque, authigenic iron sulphides. (B) Small (type 2) Wedl tunnels seen in transverse section of an indeterminate bone from the Two Medicine Formation (TMF) (Upper Cretaceous, Montana, USA). Transmitted light. F.O.V. = 1.4 mm. Narrow, branching tunnels extend from the osteonal canal into the body of the osteon. Compare with the large, relatively simple tunnels in Fig. 2 (A). Both types of Wedl tunnel are in close contact with the external environment. The opaque staining is authigenic iron oxide growth. (C) MFD (linear longitudinal tunnels). Transverse section of indeterminate bone from TMF. Transmitted light. F.O.V. = 1.4 mm. Tunnels appear in section as circular to oval holes, with dense mineralization around edges of tunnels. Tunnels are concentrated adjacent to, but not in contact with, the osteonal canal. (D) MFD (linear longitudinal tunnels). Longitudinal section, indeterminate bone, TMF. Transmitted light, F.O.V. = 1.4 mm. The osteonal canal is the sub-linear, light-coloured feature running right to left in the upper third of the image. The canal is surrounded by darker-coloured osteonal bone. Note the branching tunnels extending from the osteonal canal, parallel to the direction of collagen fibres. Note also the growth of opaque iron oxides at the margins of the osteon. (E) MFD and post-mineralization cracking. Same bone as (D). F.O.V. = 2.5 mm. Note the post-mineralization crack conduit for light-coloured authigenic calcite. Calcite also fills tunnels cut by post-mineralization cracking. The tunnelling predates both cracking and permineralization. (F) Diffuse demineralization. Indeterminate bone TMF. Transverse section. F.O.V. = 1.4 mm.*

parallel with the original direction of collagen fibres. Tunnels occasionally branch, producing parallel tunnels, both running parallel to the osteonal canal (Fig. 2 (D)). These tunnels correspond closely to the linear longitudinal tunnels described by Hackett (1981), and are very similar to non-Wedl MFD reported by Hanson and Buikstra (1987), although no longitudinal sections were illustrated.

Occasionally, linear longitudinal tunnels and small Wedl-type branching tunnels are found in association in single osteonal systems. There is no clear temporal relationship between the two sets of bioerosion: however, longitudinal tunnels clearly predate later cracking and permineralization of the bone (Fig. 2 (E)). Most tunnels are found in the early stages of colonization, although a few bones do show almost complete loss of histology due to non-Wedl MFD. The majority of these bones were recovered from bone-beds or palaeosol horisons: however, uneven sampling prevents any analysis of environmental specificity.

Where present, MFD is usually found localized in one or more small areas of a single bone, and thin sections of bone may thus provide a false impression of the extent of bioerosion in a single bone, depending on the chance location of the section. Two processes may also hamper the recognition of focal destruction in thin sections of fossil bone. In many cases, focal destruction and tunnelling is easily recognized as the tunnels have become permineralized, usually by iron-rich minerals (iron oxides), sulphides or carbonates. Where this permineralization has not occurred, it may be more difficult to recognize focal destruction in thin section. Other bones show a loss of histology with no obvious bioerosive features. These bones appear to show recrystallization that does not follow any microstructural features. Such widespread histological loss could result from extensive focal destruction (see, e.g., Hedges *et al.* 1995; Fig. 2). However, in most cases of extreme linear longitudinal type bioerosion, individual destructive foci or tunnels are still visible. Hanson and Buikstra (1987) describe two major categories of post-depositional demineralization, MFD and 'diffuse' demineralization. Diffuse demineralization occurs through abiological leaching of bone mineral with no apparent loss of histology. Recrystallization of bone following such diffuse demineralization could possibly produce the histological loss seen in a few bone sections. In this study, histological loss of uncertain origin was classed as 'other' (Fig. 2 (F)). If diffuse demineralization and recrystallization is the result of an abiological process, then this classification will overestimate the extent of bioerosion. However, only seven bones show such features, and removal of these bones simply emphasizes the lack of bones with low histology index values.

DISCUSSION

Frequency of bioerosion

Given the prevailing assumption that fossil bones are unaffected by bioerosion, it is surprising to find that a third (32%) of all bones examined showed some signs of microbial attack. At first glance, this would seem to suggest that bioerosion does not play a significant role in determining which bones will survive into the fossil record. However, it is immediately apparent that the vast majority of these bioeroded bones in fact show very minor bioerosion. Normally, $<5\%$ of the examined area of any single bone is affected, and often the bioerosion is limited to a single tunnel visible on each thin section. Indeed, when the frequency of histology index values found in fossil bones (Fig. 1 (B)) is compared with the results for archaeological bone given by Hedges *et al.* (1995), a striking difference is obvious. Fossil bone is characterized by a high percentage of OHI = 5 values. Lower OHI values decrease steadily in frequency, rather than

displaying the U-shaped distribution found in archaeological bone. The distribution seen is exactly what may be predicted if the heavily altered bones (OHI = 1–0) are removed from the archaeological sample, and the distribution recalculated. This suggests that bioerosion is indeed a fundamental controlling agent influencing bone fossilization.

Bioerosion morphology

Half of the tunnels found in fossil bones are Wedl-type tunnels, characterized by relatively shallow penetration into the mass of the bone from external surfaces (Figs 2 (A) and (B)). These tunnels do not usually follow any particular structure within the bone itself. This contrasts with linear longitudinal tunnels, which are normally developed within osteonal systems, parallel to the osteonal canal (Figs 2 (C)–(E)). Linear longitudinal tunnels (and corresponding focal destruction seen in transverse section) are only seen in 14% of the bones, and the majority of these were recovered from two localities—from a terrestrial bone bed in the Campanian Two Medicine Formation, and from a coastal marine bone bed in the Purbeck Durlston Bay Formation. MFD caused by these linear longitudinal tunnels occasionally reduces bone to OHI stage 1 in localized areas, but bones affected by linear longitudinal MFD more commonly display OHI stages 2–3.

In forensic and archaeological bones, non-Wedl MFD is much more common than Wedl tunnelling. In a survey of 76 bones ranging from *c*. 9000 BC to early 20th century, Garland (1988) found only two bones showing Wedl-type tunnels, these extending from the outer surface into the periosteal cortex, and neither Hanson and Buikstra (1987) nor Bell (1990) report Wedl tunnels in their studies of heavily bioeroded bones. Thus it appears that recent and archaeological bone differs from fossil bone in that it is significantly more bioeroded, and that this bioerosion is mainly in the form of non Wedl-MFD. Indeed, Wedl-type tunnelling seen in fossil bone causes relatively little gross damage to the bone, and may represent and entirely different microbial life strategy (e.g., surface colonization rather than active collagen metabolism).

The tunnelling organism—fungal or bacterial?

Two types of micro-organism may be responsible for the MFD seen in fossil bones. Both fungi and bacteria may metabolize collagen and, in order to do so, both must dissolve the mineral matrix, and thus produce MFD. Previously, MFD has been assigned to fungi (Wedl 1864; Roux 1887; Schaffer 1889; Bystrov 1956; Morganthaler and Baud 1956; Machiafava *et al.* 1974; Hackett 1981; Piepenbrink 1986; Garland 1988; Martill 1989; Child 1995), bacteria (Hackett 1981; Bell 1990; Bell *et al.* 1996), algae (Arnaud *et al.* 1978; Davis 1997) and amoebic protozoa (Ascenzi and Silvestrini 1984). Most authors agree that Wedl-type tunnels are made by fungi (at least in terrestrial environments), as Wedl-type tunnels have been produced by inoculating fresh bone with fungi (see, e.g., Machiafava *et al.* 1974).

The causal agent responsible for non-Wedl MFD is much harder to identify, as these tunnels have not been produced in inoculation experiments. Child (1995) notes that fungi of the genus *Ascomycetes* maintain an acidic environment at the tip of the growing hyphae, which could promote dissolution of apatite, and production of non-Wedl MFD. For Bell (1990), all non-Wedl MFD are produced by bacteria: however, the tunnels associated with non-Wedl MFD are >2 μm in diameter, an order of magnitude larger than most bacterial cells. Fungal hyphae are more than 10 μm in diameter (Plunkett, in Hackett 1981), and appreciably larger than bacteria. This would seem to suggest that bacteria are unlikely causal agents of non-Wedl MFD, but

Hackett (1981) notes the presence of tubules about 300 nm in diameter, visible in archaeological bone under TEM. These he attributes to bacteria, and suggests that they may follow fungal invasions. Recently, Turner-Walker and Syverson (2002) have shown that in archaeological-age bone from West Runton, MFD tunnels several μm in diameter contain a complex internal structure, potentially caused by coalescing smaller invasive structures. In teeth, Bell *et al.* (1991) note the presence of small 'included' tubules within dentine, which represent both the route of microbial attack and morphed dead bacterial consortia, encased within a remineralized larger foci. Finally, Child (1995, 173) notes that 'the micro-organisms responsible for the initial colonization (pioneering microbes) and decomposition of bone may have been long replaced by other organisms (their successors)'. Thus the features seen in archaeological and fossil bone may be the result of more than one microbial process.

What is the significance of bioerosion for bone preservation?

As mentioned above, bone is composed of small reactive crystals of apatite, set within a matrix of collagen. Hackett (1981) states that 'The growth of the invading organism depends on access to the bone collagen. For this to occur, presumably the collagen must be exposed by the removal of the mineral.' Collins *et al.* (1995) add that collagenolytic decay of collagen also requires dissolution of apatite mineral, as otherwise collagenolytic enzymes cannot gain access to the mineralized collagen molecule. On the other hand, as dissolution reactions occur at crystal surfaces, the apatite crystallites are themselves protected from dissolution by their protein coating. Thus, the state of 'mutual protection' alluded to earlier exists between the apatite crystals and the collagen matrix. Once this is broken (presumably by microbial or fungal attack), the positive feedback mechanism of Hedges *et al.* (1995) results in rapid destruction of the bone.

However, fossil bones do exist, and they rarely contain appreciable quantities of organic molecules. While several studies have demonstrated the persistence of organic molecules in fossil bone (Wyckoff 1972; Ostrom *et al.* 1993), yields of organic molecules are extremely low compared to those in recent bone. Furthermore, no convincing evidence has been provided for the survival of intact type 1 collagen in fossil (post 1 ma) bones. Evidently the collagen and other bone proteins have broken down, but this study shows that there is no evidence for extensive microbial attack in most fossil bones.

Collagen can decay abiologically, through hydrolysis to gelatin, and as this does not require removal or dissolution of apatite crystals it would not result in histological destruction. We believe that the excellent histological preservation found in fossil bones results from abiological decay of collagen (as suggested by Collins *et al.* 1995). If this is true, then it has implications for the rate of bone fossiliation. As exposed bone crystallites are susceptible to dissolution (Nriagu 1983; Karkanas *et al.* 2000), stabilization of these crystallites must keep pace with collagen hydrolysis if the bone is to be preserved (unless the bone remains in an environment that is saturated with respect to calcium phosphate). Thus the rate of collagen hydrolysis may determine the rate of bone recrystallization.

Significance of mode of death

All vertebrates contain a relatively diverse internal bacterial flora. In life, bacteria are prevented from extensive tissue degradation by the action of bacteriophages. After death (or even slightly before death), however, anaerobic gut bacteria are free to invade all body tissues via the vascular system. Bell *et al.* (1996) emphasized the importance of the internal gut flora for rapid

degradation of body tissues, pointing out the speed at which gut bacteria invade all tissues after death. If internal gut bacteria are indeed responsible for the initial *post mortem* colonization and degradation of bone, then unless some process acts to stop bacterial infestation via the vascular network, it would be difficult to prevent rapid microbial attack of bone tissue, and therefore loss of bone to the fossil record. Dismemberment either by butchery or predation will prevent dispersal of bacteria through the vascular system, and may play a critical and largely unrecognized role in determining bone survival. Similarly, burial within soils may expose bone to soil-dwelling micro-organisms that would be inhibited by the wide diurnal fluctuations in temperature, moisture and UV exposure experienced by bones exposed on land surfaces.

Most studies of bioerosion in ancient bone have concentrated on human burials, presumably situations in which the body's vascular systems remain intact, leaving bone more susceptible to rapid attack from the internal gut microflora, and providing a relatively attractive stable habitat for soil micro-organisms. The majority of bones sampled from fossil deposits, however, come from isolated fragmented bones, from animals that probably died either through accident or predation. In most modern terrestrial environments, large carcasses are rapidly scavenged, again lessening the potential for the invasion of tissues by the internal microflora. In terrestrial settings, bone may remain exposed on land surfaces for relatively long periods of time, potentially reducing the availability of bone to microbial attack. It is possible that the mode and location of death is a critical and neglected factor determining the final preservation potential of any single skeleton or bone. If so, mode of death will be a major taphonomic bias in the fossil record.

The arguments above all suggest that bioerosion (with the possible exception of some Wedl-type tunnels) is a very early *post mortem* process, which rapidly proceeds to complete destruction of the bone. However, this study (and others) has shown that fossil bones do occasionally show non-Wedl type MFD, identical to the rapid *post mortem* tunnelling seen in recent and archaeological bone. Evidently bioerosion can be stopped, and bioeroded bone can survive into the fossil record. The most obvious mechanism for halting bioerosion (and indeed preventing bioerosion) is chemical inhibition of bioeroding microbes. In the case of bioeroded fossil bones, a change in chemical conditions must have occurred during the early burial history of the bone.

It appears that if a bone is going to survive into the fossil record, then early microbial degradation of collagen must be avoided. The conditions for hindering early collagen metabolism are unknown, but as bone is found in almost all depositional environments, bioerosion is unlikely to be controlled by a simple single factor. These results have implications for many fields of palaeontology and archaeology, as they suggest maximum ages for bone fossilization, and point towards possible major taphonomic biases.

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REFERENCES

Arnaud, G., Arnaud, S., Ascenzi, A., Bonucci, E., and Graziani, G., 1978, On the problem of the preservation of human bone in sea-water, *Journal of Human Evolution*, **7**, 409–20.

Ascenzi, A., and Silvestrini, G., 1984, Bone-boring micro-organisms: an experimental investigation, *Journal of Human Evolution*, **13**, 531–6.

Barrick, R. E., and Showers, W. J., 1995, Oxygen-isotope variability in juvenile dinosaurs (*Hypacrosaurus*)—evidence for thermoregulation, *Paleobiology*, 552–60.

Bell, L. S., 1990, Palaeopathology and diagenesis: an SEM evaluation of structural changes using backscattered electron imaging, *Journal of Archaeological Science*, **17**, 85–102.

Bell, L. S., Boyde, A., and Jones, S. J., 1991, Diagenetic alteration to teeth *in situ* illustrated by backscattered electron imaging, *Scanning*, **13**, 173–83.

Bell, L. S., Skinner, M. F., and Jones, S. J., 1996, The speed of *post mortem* change to the human skeleton and its taphonomic significance, *Forensic Science International*, **82**, 129–40.

Bystrov, A. P., 1956, O razrushenii skieleitnykh elementov iskopaemikh zhivotnikh gribami [On the damage to the skeleton of fossil animals by fungi], *Vesnik Leningradskogo Universiteta Geologiya & Geografiya*, **11**, 30–46.

Child, A. M., 1995, Towards an understanding of the microbial decomposition of archaeological bone in the burial environment, *Journal of Archaeological Science*, **22**, 165–74.

Collins, M. J., Riley, M. S., Child, A. M., and Turner-Walker, G., 1995, A basic mathematical simulation of the chemical degradation of ancient collagen, *Journal of Archaeological Science*, **22**, 175–83.

Davis, P. G., 1997, The bioerosion of bird bones, *International Journal of Osteoarchaeology*, **7**, 388–401.

Garland, A. N., 1988, A histological study of archaeological bone decomposition, in *Death, decay and reconstruction* (eds. A. Boddington, A. N. Garland and R. C. Janaway), 109–26, Manchester University Press, Manchester.

Hackett, C. J., 1981, Microscopical focal destruction (tunnels) in excavated human bones, *Medicine, Science and the Law*, **21**, 243–65.

Hanson, D. B., and Buikstra, J. E., 1987, Histomorphological alteration in buried human bone from the lower Illinois Valley: implications for palaeodietary research, *Journal of Archaeological Science*, **14**, 549–63.

Hedges, R. E. M., Millard, A. P., and Pike, A. W. G., 1995, Measurements and relationships of diagenetic alteration of bone from three archaeological sites, *Journal of Archaeological Science*, **22**, 201–9.

Hubert, J. F., Parish, P. T., Chure, D. J., and Prostak, K. S., 1996, Chemistry, microstructure, petrology, and diagenetic model of Jurassic dinosaur bones, Dinosaur National Monument, Utah, *Journal of Sedimentary Research*, **66**, 531–47.

Lees, S., 1989, Some characteristics of mineralised collagen, in *Calcified tissue: topics in molecular and structural biology* (ed. D. W. Hukins), 153–73, Macmillan, London.

Karkanas, P., Bar-Josef, O., Goldberg, P., and Weiner, S., 2000, Diagenesis in prehistoric caves: the use of minerals that form *in situ* to assess the completeness of the archaeological record, *Journal of Archaeological Science*, **27**, 915–29.

Machiafava, V., Bonucci, L., and Ascenzi, A., 1974, Fungal osteocalasia: a model of dead bone resorption, *Calcified Tissue Research*, **14**, 195–210.

Martill, D. M., 1989, Fungal borings in neoselacian teeth from the Lower Oxford Clay of Peterborough, *Mercian Geologist*, **12**, 1–4.

Morganthaler, P. W., and Baud, C. A., 1956, Sur une cause d'altèration des structures dans l'os humain fossile, *Actes de la Société Helvétique des Sciences Naturelles*, **136**, 142–3.

Nriagu, J. O., 1983, Rapid decomposition of fish bones in Lake Eerie sediments, *Hydrobiologica*, **106**, 217–22.

Ostrom, P. H., Macko, S. A., and Russell, D. A., 1993, Assessment of the trophic structure of Cretaceous communities based on stable nitrogen isotope analyses, *Geology*, **21**, 491–4.

Piepenbrink, H., 1986, Two examples of biogenous dead bone decomposition and their consequences for taphonomic interpretation, *Journal of Archaeological Science*, **13**, 417–30.

Roux, W., 1887, Uber eine Knochen lebende Gruppe von Faderpilzen (*Mycelites ossifragus*), *Zeitschrift für Wissenschaftliche Zoologie*, **45**, 227–54.

Schaffer, J. R., 1889, Über den feinen Bau Fossilen Knochen, *Akademie der Wissenschafen in Wien, Abteilung III*, **98**, 319–64.

Schweitzer, M. H., Johnson, C., Zoco, T. C., Horner, J. R., and Starkey, J. R., 1997, Preservation of biomolecules in cancellous bones of *Tyrannosaurus rex*, *Journal of Vertebrate Paleontology*, **17**, 349–59.

Turner-Walker, G., and Syversen, U., 2002, Quantifying histological changes in archaeological bones using BSE–SEM image analysis, *Archaeometry*, **44**, 461–8.

Wedl, C., 1864, Uber einen im Zahnbein und Knochen keimenden Pilz, *Akademi der Wissenschafen in Wien. Fitzungsberichte Naturwissenschaftliche Klasse ABI. Mineralogi biologi erdkunde*, **50**(1), 171–93.

Weiner, S., and Price, P. A., 1986, Disaggregation of bone into crystals, *Calcified Tissue International*, **39**, 365–75.

Wyckoff, R. W., 1972, *The biochemistry of animal fossils*, Scientechnica, Bristol.