

**THE PALAEOPATHOLOGY OF WILD MAMMALS
IN ARCHAEOLOGY**
VADON ÉLŐ EMLŐSÁLLATOK BETEGSÉGEI A RÉGÉSZETBEN
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'Hey babe, take a walk on the wild side...'

(Lou Reed)

Abstract

Domestication is known to have increased animal morbidity. Wild animals, however, should not be looked upon romantically like Jean-Jacques Rousseau's "noble savage", untainted by civilisation. Rare pathological lesions found on the bones of wild animals in archaeozoological assemblages, they offer valuable information both from a zoological and a archaeological point of view. In addition to discussing problems of sampling, this paper is a review of major factors such as taphonomy, environment, and heritability that determine the manifestation of disease in wild animals in archaeological assemblages. A simple classification, specifically developed for wild animals, is presented that helps better understand these conditions. Numerous examples from both the author's own work and the broad base of international literature (especially on Europe and the Southwest Asia) are cited to help illustrate how disease is manifested on the bones of wild animals recovered from a variety of archaeological periods. The results of this paper show that although domestication undoubtedly brought about an increase in animal morbidity, depending on the chances of survival of a game species and the functional importance of the body part affected, a variety of pathological lesions regularly occur on the remains of wild animals as well.

Kivonat

A háziasítás köztudottan csökkentette az állatok betegségekkel szembeni ellenálló képességét. Azonban a vadállatokat sem szabad a Rousseau romantikus szellemében a „nemes vadság” állapotában megrekedt, a civilizáció ártalmaitól érintetlen teremtményeknek tekintenünk. Noha a régészeti állattani leletgyűjtemények vadállatcsontjain csak ritkán figyelhetünk meg betegségre utaló elváltozásokat, ezek a tünetek fontos állattani és régészeti ismeretekkel szolgálnak. A mintavételi kérdések áttekintése mellett ez a dolgozat a tafonómiai tényezők, a környezet és az örökletesség hatásaival foglalkozik a kóros csonttani elváltozások régészeti megfigyelhetőségében.

A betegségek egyszerű, vadállatok maradványaira kidolgozott osztályozása segít az ilyen esetek pontosabb megértésében. Az egyes betegségek tüneteinek megjelenését a vadállatok csontjain a szerző saját munkásságából és a szakirodalom áttekintéséből vett, elsősorban európai és közel-keleti példák szemléltetik a különböző régészeti korokban. Az összefoglalás eredményeiből az látszik, hogy az állatbetegségek számának növekedése ugyan részben a háziasítás következménye, de az adott faj túlélési esélyeitől, illetve az érintett testrész működésbeli fontosságától függően különböző tünetek a vadállatok maradványain is rendre megjelennek.

KEYWORDS: ARCHAEOZOOLOGY, PALAEOPATHOLOGY, WILD MAMMALS, TAPHONOMY

KULCSSZAVAK: RÉGÉSZETI ÁLLATTAN, PALEOPATOLÓGIA, VAD EMLŐSÁLLATOK, TAFONÓMIA

Introduction

For a variety of reasons – that have actually inspired the writing of this brief summary – our recent synthesis of animal disease in archaeology (Bartosiewicz 2013; Gál 2013) was dominated by the discussion of domestic animals whose relationships with humans are far more demonstrable archaeologically than those of their wild brethren. It is time to look at the

other side of the coin. What can be said about wild animal morbidity in the past?

According to a widely held view natural selection would eradicate inherited disease in game populations, as wild animals “*afflicted with disease or injury ... soon succumbed to the hostile acts of predatory animals or man. Few survived sufficiently long*

for osseous changes to develop” (Moodie 1923: 141-142). In other words, the paucity of pathological finds from game is to a great extent a consequence of the fact that a host of factors prevent the development of grave pathological conditions in the majority of functionally important skeletal parts of most wild animals. Of these factors, natural selection is unquestionably the strongest. In human palaeopathology, healing is considered the most reliable evidence of *pre-mortem* trauma (Aufderheide & Rodriguez-Martin 1998: 23). However, for a fracture to show signs of recovery at least two weeks of survival are required in humans (Mann & Murphy 1990). In the case of wild animals, even for species of sizes and metabolic rates comparable to that of humans, the chances of survival that long would indeed have been limited to special cases.

In this paper an attempt is made to systematically review the gross types of pathological lesions visible on wild animal bone finds, looking at some special cases within the context of the relevant literature. This work is based on the macromorphological identification of osteological symptoms, sometimes with limited diagnosis as to the concrete disease that caused the lesion. The actual occurrence of pathologically modified animal bones in archaeozoological assemblages is a product of at least three general factors: taphonomy, environment, and inheritance. These three interact with each other in determining what would be available for palaeopathological studies. They also conspire to reduce the incidence of pathological specimens from wild animals relative to those from domesticates in most find assemblages.

In a broad sense, taphonomy (with special emphasis on the anthropogenic element in biostratinomy), environment, and heritability determine the presence or absence of pathological specimens in archaeozoological assemblages. The classification of disease in palaeopathology, however, is made difficult by the fact that usually only osseous materials are available for study. In addition, living bone has but a limited repertoire of reactions to a variety of disease. Individual symptoms may thus frequently have multiple and/or complex aetiologies. Classifying pathological conditions manifested on bones, therefore, is a complex task far beyond the focus of the narrowly defined topic of this study. Consequently, for purely practical purposes, lesions on the excavated bones of wild animals will be reviewed following a simplified scheme, rather than the complex rules of detailed veterinary nosology. This classification is reduced to the study of pathological specimens as archaeozoological finds, products of the factors reviewed previously (Fig. 1).

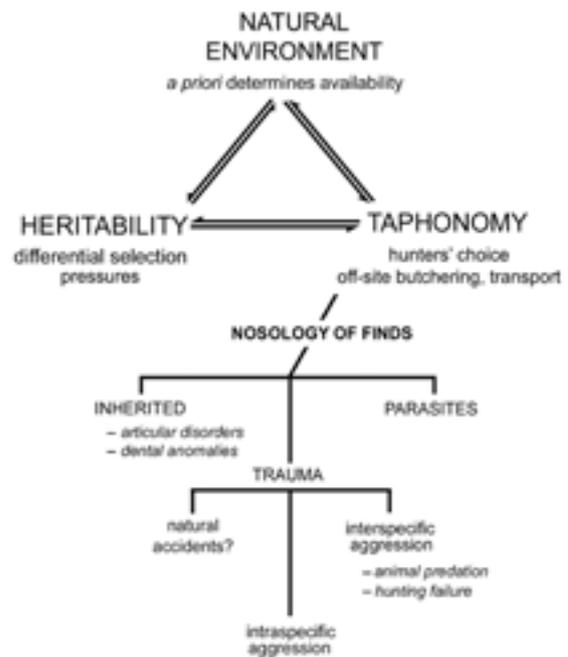


Fig. 1.: Summary of factors influencing the manifestation of pathological lesions on the bones of wild animals in archaeozoological assemblages

1. ábra: A régészeti leletegyüttesek vadállatsontjain megfigyelt tünetek megjelenését befolyásoló tényezők

Sampling and Taphonomy

Archaeozoological assemblages, in general, tend to contain relatively few bone remains showing pathological lesions. From the Neolithic onwards, as farming societies began increasingly relying on domestic animals in meat provisioning, an overwhelming majority of even the few pathologically modified animal bones tend to originate from domestic mammals. The manifestation of these phenomena, to a great extent, depends on the size and richness of the assemblage itself. This may be most easily demonstrated by reviewing the example of bone fractures in the literature that have been reported most consistently. Siegel (1976) reported 19 such cases (0.04%) in her review article based on 47,300 excavated animal bones. In what has become a classic on the topic, Baker and Brothwell (1980: 91) arrived at the same percentage based on the review of 34,926 Holocene animal remains. Statistically speaking, in individual assemblages, the remains of the best represented animal species have the greatest likelihood to display pathological lesions. Fig. 2 shows the occurrence of some individual pathological cases (Bökönyi & Bartosiewicz 1999; During 1986; Graf 1967; König 1993; Krauß 1975; Missel 1987: 81; Noddle et al. 1977; Noe-Nygaard 1989; Prummel 1987; Rauh 1981; Reichstein 1991; Swegat 1976; Teichert 1979)

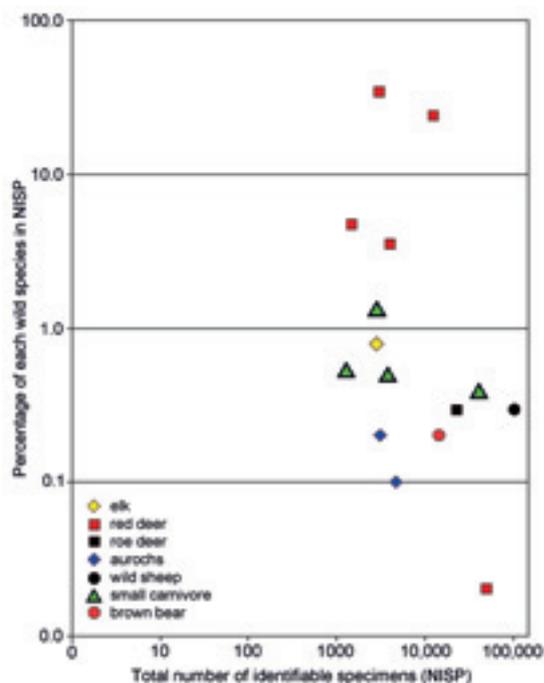


Fig. 2.: The occurrence of some pathological specimens as a function of assemblage size (NISP) and the percentage of bones from the particular animal within that assemblage

2. ábra: A betegségek tüneteit viselő egyes csontok előfordulása a meghatározható állatsontleletek számának (x) és ezen belül az adott faj százalékos arányának (y) tükrében

in light of assemblage size (NISP) and the percentage of bones from the particular wild animal (species or animal group) within that assemblage. The results suggest that fewer than a thousand bones are unlikely to yield pathologically modified remains from game animals. The chances of such finds increase along with assemblage size, but especially with the percentual contribution of the wild animal in question even in relatively small collections. This trend is best shown by the remains of red deer (*Cervus elaphus* Linnaeus, 1758), rather commonly occurring at sites across Eurasia.

Representation, however, is influenced by a host of taphonomic factors. While natural environment and heritability impact on the phenotypic manifestation of pathological lesions on the skeleton *intra-vitam*, the taphonomic process will act as a series of *post mortem* filters that further reduce the chances of recovering the odd cases of diseased bones from wild animals. The generally rare occurrence of pathological remains from wild animals is thus, to some extent, a form of ancient “sampling” bias: hunters’ decisions had a primary impact on how animal disease is represented in find assemblages. The hunter’s choice of its prey is the first step in determining

whether a pathological lesion will be present at the archaeological site or not. Diseased game typically fall victim to animal predation. Even traditional hunters, however, could easily take individuals in prime condition using sophisticated hunting techniques, and would not need to kill only feeble individuals (Kay 1994). Wild animal remains in archaeological assemblages therefore are biased by human behaviour, possibly presenting a lower percentage of remains from diseased prey items in comparison with, for example, wolf kills.

Loss of disease-related information continues with the possibility of off-site primary butchering, a routine procedure in the hunting and processing of large game, where large bones (including those potentially affected by disease) are left behind. Although the ‘*schlepp-effect*’, the selective representation of skeletal parts brought back to the settlement, does not hold as a general law in zooarchaeology, Perkins and Daly (1968, 103) directed attention to the carcasses of large game which were treated differently from those of domesticates of comparable body size. While large game which have fewer predators, have a better chance to survive and develop osteological deformations, it is exactly this group whose pathologically altered bones may be decimated by selective human transport.

At the settlement, the preservation of diseased bone depends rather on the type of pathological condition than on the wild or domestic status of the animal exploited. Pathological changes in bone density determine the rates of bone loss both before deposition (biostratinomy) and thereafter (fossil diagenesis; Bartosiewicz 2008). A curious example of pre-depositional interference was described on the *collum* area of a Mesolithic elk (*Alces alces* Linnaeus, 1758) scapula from Star Carr, which had partially dissolved in a major inflammation. In addition to defleshing marks, a concentration of ‘exploratory’ cuts around the pathological lesion suggests that the diseased tissue was carefully investigated (Legge and Rowley-Conwy 1988, 97).

Inherited anomalies and analogous lesions

Decapitating hereditary disorders of the skeleton can indeed be discounted in wild animals, since natural selection would have prevented their passing from one generation to the other. Among wild herbivores, present day elk (*Alces alces* Linnaeus, 1758) remains from Kenai Peninsula (Alaska) and Isle Royale (Lake Superior, USA) have exhibited ample evidence of skeletal lesions not only related to old age and poor nutritional status but genetic causes as well (Peterson *et al.* 1982). These animals would have had less chance to survive in mainland populations which are regularly predated upon. A curious

case of multiple antlers was documented in a cervid (putatively fallow deer) by Marsigli (1726, **Fig. 3**), although no comparable archaeological evidence is known to me.

Articular disorders

Even the manifestations of lesser genetic anomalies may be limited by the degree of selection pressures on wild populations. A group of 252 present-day foxes (*Vulpes vulpes* Linnaeus, 1758) from the London area are worth referring to here, 34.5% of whom were affected by spondylosis in spite of their young ages (Harris 1977, 185). These urban foxes were, however, commensal animals, not hunted game, scavenging in neighbourhoods densely inhabited by humans. Pathological lesions on the bones of fox from archaeological sites tend to be unusually rare. Two lesions of non-hereditary nature have been published from the tell site of Demircihüyük (north-western Anatolia, Turkey; Rauh 1981, 134). The neurocranium fragment of an adult fox from an Early Bronze Age (c. 2700-2400 BC) context showed a deformation of the right orbita – the maxilla and lachrymal bone were porous and remodelled. The upper P1 tooth was lost *in vivo* as a result of trauma. The trochanter major of a Middle Bronze Age (c. 2000-1500 BC) right femur developed into a nose-like point as a result of muscle strain.

In the wild, bony bridges (*osteophytes*) may occur between the vertebrae of the lower neck and the sacrum in bear, while in Hyaenidae, they may also develop chiefly in the thoracic and lumbar sections (Fox 1939). An exostosis was observed on the great articular surface of a cervical vertebra from otter (*Lutra lutra* Linnaeus, 1758) at the Neolithic site of Hekelingen III in the Netherlands (Prummel 1987, 218). Aside from age, such disorders seem to be related to posture and species specific patterns of locomotion in large-bodied mammals, the most general form of genetic ‘predisposition’. These, however, are minor lesions compared to the dramatic cases of presumably inherited vertebral fusion in domestic animals, especially horses (e.g. Bartosiewicz & Bartosiewicz 2002).

It must be emphasised that by no means are all articular disorders inherited. Arthritis may result, for example, from overworking, i.e. repetitive strain syndrome (RSI) in domesticates, although this tends to rarely affect wild animals. The chronic inflammation of the hock joint (*peri-arthritis et osteoarthritis tarsi circumscripta*; Tamás 1987, 377) culminating in the fusion between the metatarsal and tarsal bones, may be observed in many cases of unknown aetiology. This condition, also known as spavin (*ostitis rarefaciens et condensans*), is caused by a complex of inherited and functional disorders. In horse



Fig 3.: The development of multiple antlers in a cervid from the Lower Danube region (Marsigli 1726)

3. ábra: Több agancsú szarvas az Al-Duna térségéből (Marsigli 1726)

and cattle, it has been – in a rather simplistic manner – attributed to draught exploitation (Blumenfeld 1909; Stillfried 1926, 151; Wamberg & McPhearson 1968; Wells 1972). Interspecific comparisons of this disease are difficult due to differences in size, locomotion, longevity and selection pressures on inherited foot conformation. This is clearly shown by the example of Cervids. The static loading on the hind legs of deer is significantly smaller than in cattle (Berg & Butterfield 1976, 142), although this difference somewhat decreases with age (Bartosiewicz 1987, 445). More importantly, dynamic strain on the hock joint of a leaping deer is greater than in a running cow (Kreutzer 1992, 274). Spavin, however, is still rare in deer. The author observed a modern case of spavin in a fallow deer (*Dama dama* Linnaeus, 1758) from the game park in Gent, Belgium (*Laboratorium voor Paleontologie, Geologisch Instituut* Gent, Inv. No: P.2422). This is less surprising in a game species artificially introduced to game parks in Western Europe (Clutton-Brock 1987, 182) where the lack of selection pressure gave rise to a variety of skeletal disorders (Chaplin 1971, 118, Fig. 17). It must be noted, however, that natural populations of cervids may also show minor pathological lesions. Marín-Arroyo et al. (2015: 49) reported

bone resorption on the cortex of the axial surface of the proximal phalanges of Palaeolithic Mesopotamian fallow deer (*Dama mesopotamica* Brooke, 1875) from Level B in Tabun Cave (Israel). They attributed this lesion to an infection originating on the hoof, a possible case of footrot caused by *Fusobacterium necrophorum* or *Dichelobacter nodusus*. Even a typical case of spavin, i.e. the full fusion between the right centrotarsal bone and the proximal end of the metatarsus, was also reported from red deer, found at the seventh–eighth century AD Slavic settlement of Wiesenau (Kreis Eisenhüttenstadt, former GDR; Teichert 1979, Abb. 2/6).

In modern-day draught oxen, the advancement of spavin is in positive correlation with both age and body weight (Bartosiewicz *et al.* 1997). It is less surprising, therefore, that this condition, involving the left *os tarsale secundum et tertium*, occurred in a third – fifth century AD aurochs (*Bos primigenius* Bojanus, 1827) from Hildesheim-Bavenstedt (Misel 1987, 81). The few natural predators and resulting longevity of aurochs make the manifestation of minor lesions possible. For example, extensive exostoses also developed on the dorsal surface of the right metacarpus from a mature aurochs, found in Layer 6 at the site of Sahr-i Sokhta, Sistan (Iran; Bökönyi & Bartosiewicz 2000).

Joints may also be infected as a side effect of compound fractures. Such arthritic deformations have nothing to do with heritability. Healed compound fractures, however, tend to be very rare in wild animals. Interestingly, the first ever description of a palaeopathological lesion by Johann Friederich Esper (1774, 74, Plate XIV/2) was observed on the diaphysis of a cave bear (*Ursus spelaeus* Rosenmüller et Heinroth, 1794) femur. Carl Mayer (1854, 673) identified this as a healed fracture with callus formation and some necrosis, although contradictory diagnoses have appeared ever since partly due to inaccurate quoting (Waldron 2015). Tasnádi Kubacska (1960, 95, Fig. 141) identified a broken cave bear

tibia from Szeleta Cave, Hungary, whose splinters fused into a solid block. As a result, the bone shrank nearly to its half. The development of such grave deformations is attributable to the status of cave bears as top predators, but still subsisting on a vegetarian diet: even when feeble, these large animals would have been threatened by only a few other animals, while they did not need the agility indispensable for carnivores during the hunt. It seems also unlikely that human predation would have exerted a particular pressure on cave bear populations. In later periods game habitats shrunk due to expanding human occupation, the chances of developing such grave symptoms also radically declined. Lesions, therefore, tend to be a lot less pronounced. Lipping and slight exostoses appeared around the proximal articular surface of the right radius from an adult brown bear (*Ursus arctos* Linnaeus, 1758), recovered from a Bronze Age deposit at Arslantepe, Central Anatolia (Turkey; Bartosiewicz 2002). The left radius of another adult brown bear, from the sixth–fifth century BC Celtic settlement of Hundertingen an der Donau, showed callus formation on the middle of its diaphysis resulting from a healed fissure (Graf 1967, Taf. 3/4). The animal must have survived this accident by at least three months.

Dental anomalies

In contrast to some arthritic lesions, dental anomalies are primarily considered congenital phenomena posing relatively little or no disadvantage to the individual animal. While oligodonty is frequently treated a sign of domestication, it is also known to various degrees in wild animals. The statistical analysis of badger skulls (*Meles meles* Linnaeus, 1758) from Twann (Lake Biel, Switzerland; Grundbacher *et al.* 1990, 103) shows no statistically significant difference (P_1 : $\chi^2=2.643$, $p\leq 0.100$, P_1 : $\chi^2=0.046$, $p\leq 0.527$) in the rate of missing first premolar (P_1) teeth between the assemblages of 93 Neolithic and 98 present-day badgers (Table 1).

Table 1.: Comparison of oligodontia between Neolithic and modern badgers

1. táblázat: Az első előzáfogak előfordulása újkőkori és mai borzok esetében

Form	Upper P_1			Lower P_1		
	present	absent	Total	present	Absent	Total
Neolithic	29	21	50	84	9	93
Modern	43	55	98	91	7	98
Total	72	76	148	175	16	191

These small teeth are of no vital importance in the dentition of badgers. These large, omnivorous mustelids can survive even greater forms of tooth loss. The left mandible of an adult badger from the

Middle Neolithic assemblage from Alvästra (Sweden) showed the *in vivo* loss of P_4 - M_2 teeth, possibly as a consequence of inflammation resulting from advanced caries (During 1986, 142, Fig. 23). Although

this lesion had little to do with inherited traits, it illustrates why the lesser degree of congenital oligodontia poses no risk to the species in evolutionary terms.

Comparative studies on a present-day reference collection of 291 wild and 264 domestic silver foxes (Liehn 1952, 8-9), on the other hand, showed a diachronic shortening of the mandible in silver foxes

that resulted in the disappearance of the M_3 tooth (of minor size and no functional significance in canids), while the rest of the cheek tooth row retained its size. My statistical analysis of these data (**Table 2**) showed a highly significant difference ($\chi^2=107.2$, $p\leq 0.000$) between the distributions of oligodonty in the two forms: in spite of comparable sample sizes, the incidence of oligodonty was 3-4 times higher in domestic silver foxes.

Table 2.: Comparison of oligodontia between wild (red) and domestic (silver) foxes

2. táblázat: Az alsó 3. zápfog előfordulása vad (vörös) és háziásított (ezüst) rókákban

Form	both M_3 present	Both M_3 missing	one M_3 missing	Total
red fox	267	14	10	291
silver fox	172	58	34	264
Total	439	72	20	555

Traumatic lesions

The most tangible and best represented palaeopathological phenomenon in wild animals is trauma, encountered in the form of healed wounds and fractures. Some are caused by natural accidents such as the probable reason behinds a zygomatic fracture and concomitant exostosis formation on a massive cave bear skull from Igric Cave, Hungary (**Fig. 4**; Kubacska 1936, Taf. 5/1 and Abb. 2). Other forms of trauma in wild animals may be attributed to lesions caused by intraspecific (e.g. territorial and mating fights or food competition) and interspecific conflict (e.g. predation, including hunting by humans). In some cases these two categories can be clearly distinguished. Healed fractures of the baculum in bear or walrus, for example, could only have been inflicted during mating fights (Capasso 1999; Bartosiewicz 2000, 448, Fig. 1).

Although anomalies of the antler, including traumatic malformation, are well known in extant cervids (e. g. Bubenik 1966; Goss 1983; Jaczewski 1992), excavated specimens rarely exhibit such symptoms. Many archaeological finds actually represent shed antler gathered for the purpose of antler manufacturing; as healed fractures compromised the integrity of antler structure, such specimens may have been left behind during the procurement of this popular raw material. Amorphous primary bone growth prior to osteoclast-mediated remodeling was observed in the microstructure of an antler fragment of a Pleistocene giant deer (*Megaloceros giganteus* Blumenbach, 1799) from the San River near Barycz, south-eastern Poland (Pawłowska et al. 2014, 7, Figures 3.3-4). Histological studies showed no osteons but lamellar bone formation, consistent with a fracture healing and concomitant callus formation in skeletal bone (Grupe & Peters, 2006). The fracture of the developing “fresh” antler covered by velvet did

not lead to the loss of the distal fragment, as it was apparently supported by the skin and periosteum, a mechanism similar to greenstick fractures in skeletal bone within the body. A healed fracture found on the shed antler of an adult roe deer (*Capreolus capreolus* Linnaeus, 1758) buck among the 22,639 animal



Fig 4.: Healed fracture of the left zygomatic arch and a large exostosis (arrow) that developed during healing in a cave bear skull from Igric Cave (*Peștera Igrîța*), Romania (Kubacska 1936; Photo T. Dömök)

4. ábra: A bal járomív gyógyult törése és a vele járó csontkinövés (nyíllal jelezve) a romániai Igric-barlangban talált barlangi medve koponyáján (Kubacska 1936; Fotó: Dömök T.)

bones from the Late Neolithic causewayed enclosure site of Diconche, France, may fall within the same category (Bökönyi & Bartosiewicz 1999).

More ambiguous is what looks like a depression fracture that healed with massive new bone formation above the *foramina supraorbitalia* on the forehead of a red deer from the late nineteenth century excavations at Snoldelev, Denmark (Fig. 5), possibly inflicted by a hunter or the antler of a rival stag. Non-traumatic chipping commonly found on the tips of antler tines also bears witness to such violent encounters. Injuries in the rest of the body are even more difficult to link with specific behaviours. For example, Noddle *et al.* (1977, 67) found the healed infection on the *os ilium* of a medieval red deer from Llantrithyd, South Glamorgan in Wales (UK), equally attributable to mating fights or human hunting. In principle, intraspecific trauma should be more common in wild animals than in domestic stock, since herders have a vested interest in suppressing aggression among their animals unless specifically bred for fighting.

Among the lesions caused by interspecific aggression, healed injuries caused by hunting accidents are of greatest interest, since skeletal injuries inflicted by humans are far from random. They depended on the hunters' skill, the weapons used and the type of hunting pursued (Noe-Nygaard 1989, 463). Ancient projectile points have been found embedded in prehistoric animal bones, especially the axial skeleton (skull and vertebrae) of prey animals. Such direct evidence is most common in herbivores that provided the bulk of hunters' prey throughout history. This is a typical bias caused by prey selection, mentioned first among the taphonomic factors (Fig. 1).

Given the limitations of space only a few examples of hunting accidents can be mentioned here. These involved late glacial elk: Hallam *et al.* (1973); Upper Palaeolithic bison (*Bison bonasus* Linnaeus, 1758): Vereshchagin (1967, Fig. 9); Mesolithic red deer: Noe-Nygaard (1989, Fig. 7); Late Neolithic aurochs: Bökönyi (1974, 104, Fig. 4); two Cortaillod Period red deer: Gross *et al.* (1990, 86-87, Abb. 3/1-4). A rare example of carnivore injury was caused by a silex projectile stuck in one of the cervical vertebrae of a brown bear (*Ursus arctos* Linnaeus, 1758) in the Grotte du Bichon (Switzerland; Morel 1993, 114, Fig. 10). This latter, 12,000 year-old deposit, however, also included the remains of the hunter.

Not all injuries have retained material evidence of weapons. Such lesions may be associated with hunting in varying probabilities. Perhaps the best known such lesions were recorded on Mesolithic red deer in Denmark by Nanna Noe-Nygaard (1975; 1989). A dozen of patterned perforations documented



Fig. 5.: Healed forehead injury in a Pollen zone VIII (3000–500 BC) red deer stag from Snoldelev, Denmark (M. Degerbøl)

5. ábra: Homloksérülés a VIII pollenzónába (3000–500 BC) keltezett gímszarvas bika koponyáján Dániából (M. Degerbøl)

in the *corpora scapulae* are consistent with the hypothesis that hunters indeed systematically aimed at the cranial half of the rib cage. Unhealed holes, located in the middle of this bone, ventrally from the *spina scapulae* (*fossa infraspinata*), may be associated with lethal, *perimortem* injuries. Krauß (1975, 205, Taf. 3/Abb. 7) reported a similarly unhealed shot wound in the same anatomical location on the right scapula from an adult wild sheep (*Ovis orientalis* Gmelin, 1774) from the 2200-700 BC settlement of Bassam (north-western Azerbaijan, Iran).

Ancient traps, although probably less efficient, may also have caused injuries that should be reckoned with. However, trapping as a direct cause of trauma would be impossible to identify in archaeozoological assemblages. In the Late Medieval material from Berlin-Köpenick, Müller (1962, 93, Taf. 8, 2) identified the necrotic proximal half of a red deer metatarsus, that he attributed to an injury which had been sustained in the same way. Necrosis (*osteosis*) may be caused by the disturbance of the local blood supply by fracture. The ends of the bone from which the periosteum is separated turn into dead tissue (Jubb *et al.* 1985, 61). Undoubtedly, the cause of this deer injury was a heavy impact on the anterior surface of the bone, which apparently resulted in the loss of the distal portion of the limb (Fig. 6). Milder forms

of trauma were observed on other deer metapodia as well. For example, a metatarsus diaphysis fragment from the Roman provincial site of Künzing-Quintana (Bavaria), showed periostitis ossificans covering most of the plantar side (c. 100 mm), in the form of exostoses (Swegat 1976, 83). This lesion may have equally resulted from chronic tendovaginitis or minor trauma. An even less debilitating fracture showed incomplete healing on a second or fifth rudimentary metacarpus (the so-called ‘*stilleto*’ bone) of a Middle Neolithic adult elk from Alvästra (During 1986, 112, fig. 20). The formation of pseudo-arthritis and callus at the proximal end probably did not put this mighty deer at a particular disadvantage, except for eventual human predation.

Bone deformations caused by parasites

Macroscopic bone deformations caused by parasites have been relatively often noted in wild animals. A cave bear skull from Doubs (France) had multiple perforations on the frontal sinus interpreted as symptoms of nematode and trematode parasitosis (Koby 1953). These parasites cause conspicuous lesions in the skulls of modern mustelids as well. An archaeological example on the skull of an European polecat was found in a cistern at the Late Antique site of Vranje, Slovenia (Fig. 7). Superficially, the numerous small holes are reminiscent of buckshot wounds. Their edges, however, show bulbous new bone formation characteristic of nematode activity. They look like the diagnostic exit holes left by *Troglorema acutum* Leuckart 1842 (Wetzel & Rick 1962, 78). Another nematode, sinus worm (*Skrjabingylus nasicola* Leuckart, 1842), also attacks mustelids. However, it tends to affect the post-orbital region making it swollen and thinned, often perforated with holes that may grow into large openings, which are visible in clean skulls (Lewis 1967, 562; King & Moody 1982; King 1989). According to van Soest *et al.* (1972), 56% of the viscerocrania in over 200 stoat and weasel skulls showed perforation attributable to this nematode. Similar perforations are caused by several genera in the families of parasite flies (*Tachinidae*) and blow-flies (*Calliphoridae*), whose maggots live in the cranial cavities of mammals including the paranasal sinuses in some ursids (Capasso 1998, 114).



Fig. 6.: Three views of the proximal half of a necrotic Late Medieval red deer metatarsus from Berlin-Köpenick (Müller 1962, Taf. 8, 2)

6. ábra: Gímszarvas elhalt végű lábközépcsontjának proximális fele Berlin-Köpenick késő középkori lelőhelyéről (Müller 1962, Taf. 8, 2)



Fig. 7.: Exit holes left by *Troglorema acutum* on the skull of a Late Antique European polecat from Vranje, Slovenia (Photo: A. M. Choyke)

7. ábra: *Troglorema acutum* kimeneti nyílásai mezei görény koponyáján a szlovéniai Vranje késő antik lelőhelyéről (Fotó: A. M. Choyke)

Conclusions

In contrast to expectations represented by the classic point of Moodie (1923) quoted in the introduction, a whole range of skeletal elements of wild animals found in archaeological deposits show pathological lesions. Natural selection does not affect all species (and all body parts) equally, although it tends to stamp out extreme inherited anomalies. Healed traumatic disorders are most frequently encountered. The larger the species, and the fewer its natural predators, the better the chances of recovery.

Although handicapped carnivores will sooner or later suffer disadvantage in their 'struggle for life', they are still less directly at risk than disabled prey species. Among wild herbivores, large-bodied species, such as aurochs or elk, again less threatened by predators, may survive long enough to develop pathological symptoms in their skeletons. In addition, killing these animals took advanced hunting skills and a degree of luck. Several finds prove that in some instances the animals were able to flee and recover from the minor injuries before they fell victim to another hunting incident.

Aside from taxonomic differences, the anatomical location of lesions also determines the chances of survival. Sub-pathological anomalies, such as oligodontia, are to some extent normal in wild animal

populations. Many pathological phenomena on the postcranial skeleton were also observed in regions less critical from the viewpoint of survival. Some unhealed scapula injuries in red deer and wild sheep, on the other hand, are seen as *perimortal* trauma related to lethal chest wounds.

The relatively great number of lesions found on the metapodia of large wild ungulates raises the question as to whether the under-representation of other bones in palaeopathological assemblages could be related to the fact that many parts of the skeleton were left behind following off-site butchering. Catching large game would also have required cooperative efforts. It is reasonable to assume that old and feeble animals were easier to hunt. On the other hand, those who survived previous attacks by hunters may have learnt to behave increasingly cautiously.

The weakness of diseased animals is more likely to have been taken advantage of by subsistence hunters in early farming societies, who still heavily relied upon the exploitation of wild animals for food. Decisions regarding the conscious culling of disabled game must have depended upon the skills and demands of hunters and may have been made on an opportunistic basis.

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