The Recognition, Frequency, and Taxonomic Association of Skeletal Pathology from Selected Plio-Pleistocene-Aged Sites from the Cradle of Humankind, Witwatersrand, South Africa

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DECLARATION

I declare that this thesis is my own, unaided work. It is being submitted for the Degree of Doctor of Philosophy in Palaeoanthropology at the University of the Witwatersrand, Johannesburg, South Africa. It has not been submitted before for any degree or examination in any other University.

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__19th___ day of ___May___ 2010

Dedicated to my parents, James and Althea,
my brother, Warren,
and my beautiful wife, Dee.

ABSTRACT

Skeletal pathology has been largely unexplored from South African Plio-Pleistocene cave contexts. As a result, there is little known about the types of pathology present in these assemblages or the frequencies at which they occur. This study was designed to identify and analyze skeletal pathology from two sites in the Cradle of Humankind, South Africa. Over 7000 postcranial fossils, representing the broad range of macromammalian taxa from the early hominid sites of Cooper's D and Swartkrans (Members 1-3), were examined for evidence of gross skeletal pathology. Frequencies of pathology were recorded at order and family levels and the elements were categorized to skeletal section to identify possible trends in the anatomical location of lesions. Chi-square and randomization tests for goodness-of-fit were conducted at family level and by skeletal section to note any significant disagreement between observed and expected frequencies of pathology. Pathological fossils were described and lesions were identified to broad diagnostic categories. In total, twenty-four pathological fossils were identified from Cooper's D and forty from Swartkrans. Joint disease, trauma and enthesopathy are the most common disease types from both sites. For the Swartkrans fauna there is the additional presence of neoplasia. The frequency of pathology at order level is similar for both sites, with artiodactyls showing the lowest frequency, followed by carnivores and primates. Pathology by family occurs at frequencies of between 0 and 6%. Hominids fall outside of this range, occurring at a frequency of 100% for Cooper's D and 16% for Swartkrans. For both sites there is a significant disagreement between the observed and expected frequencies of pathology for bovids, felids and hominids. This indicates that pathology does not occur in equal proportion throughout the assemblage, but rather occurs at high frequency for felids and hominids and low frequency for bovids, possibly correlated to the trophic roles of the different taxa. It was, however, found that pathology predominantly occurred on the same elements for all families, specifically the vertebrae and distal-limb. Chi-square and randomization tests by skeletal section revealed a non-significant result for Cooper's D (P = 0.07) and a significant result for Swartkrans (P = 0.03). For Swartkrans there is a significant disagreement between the observed and expected

frequencies of pathology for distal-limb and hindlimb elements. This result may be due to the high frequency of pathology on hominid distal elements, explained, in part, by a mechanical stress aetiology involving activity related to hominid grip and grasping.

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List of Abbreviations Used in the Text:

BMU: Basic Multicellular Unit

BPI: Bernard Price Institute

CD: Cooper's D

cf: *confer* compare

cMNI: Comprehensive Minimum Number of Individuals

CPDD: Calcium Pyrophosphate Deposition Disease

DISH: Diffuse Idiopathic Skeletal Hyperostosis

DJD: Degenerative Joint Disease

E*f*p: Expected frequency of pathology

ibid: *Ibidem* in the same place. Referring to the book cited just before

idem: Referring to the book cited just before, same page

Ma.: Megaannum. One million years.

MNI: Minimum Number of Individuals

MNI_{path}: Minimum Number of Pathological Individuals

***n.*:** Sample size

NISP: Number of Identifiable Specimens per Taxon

OA: Osteoarthritis

OCD: Osteochondritis dissecans

O*f*p: Observed frequency of pathology

TVM: Transvaal Museum

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Chapter 1. Introduction and Background

1.1 Introduction

As the etymology of the word would suggest, *disease* (dis – ease) in its literal sense means 'without comfort'. It is more familiar to us in its common usage as "an impairment of the normal state of the living body or one of its parts that interrupts or modifies the performance of the vital functions" (Pease, 1995). In this sense, it is clear that disease is inseparable from life. Palaeontology and palaeoanthropology by their nature are reconstructive sciences, whereby there is an attempt to reconstruct a comprehensive picture of the biological history of fossil taxa. Given the ubiquitous nature of disease, its study from the past (*palaeopathology*) is integral to the completeness of this reconstruction. Disease, senescence and trauma are all real and observable indicators of systemic stress faced by a particular individual and, in some cases, an entire group. To the extent that heritable factors can play a part in resistance to disease, disease conditions exert a tenable influence on the continuing process of evolution (Ortner, 2003).

A study of a fossil accumulation is incomplete if it does not take pathology into account, considering one of the goals of such a study is to provide a reconstruction of the environment at the time the accumulation was being formed (Brain, 1974). In a trophic system, driven by predator-prey relationships, the role of disease cannot be overstated. The value is implicit, in that by its very definition, disease alters structure or function, and in a wild context even minor changes in structure or function could have potentially devastating effects on an individual animal, affecting fitness, fecundity, and ultimately success (in evolutionary terms). Therefore, pathology is part of the bioecology, and as such, ignoring it, or rather dismissing it as too scarce, difficult to interpret, or simply not relevant to the 'big picture' will lead to an entire aspect of the palaeoenvironment being unexplored.

It is in large part due to the obvious rarity of soft tissue in fossil assemblages that palaeontologists and palaeoanthropologist are limited in their understanding of diseases suffered by individuals or species in the distant past. Most of the evidence of disease has deteriorated and decayed long ago with the soft tissue. How is it possible then to observe the crucial interplay between individuals of a species and this aspect of the palaeoecology? Fortunately, this gap in the compendium of palaeontological knowledge can be bridged by the study of certain types of pathology that leave evidence on skeletal remains.

In addition to the natural changes resulting from normal growth, age and a range of other biological factors, bone changes can occur *in vivo* as a result of disease conditions. For instance, infection, degenerative changes and trauma can each leave a certain (and sometimes distinct) impress on the skeletal tissues. These bone deviations from the normal biological template can provide direct and indirect information regarding the individual affected (Baker and Brothwell, 1980; Martin, 1991; Ubelaker, 1994; White, 2000). Then, when the bones and teeth in the dolomitic cave environment undergo the mineral replacement that is diagenesis, they can retain the evidence of pathological insults. The field of palaeopathology hinges on this preservation of evidence of disease from fossilized or otherwise preserved skeletal tissue (or in certain rare instances, soft tissue). It is this preservation that allows a researcher the opportunity to observe and interpret pathological conditions tens of thousands, or even millions of years after the individual's death. Further, it allows for inferences to be made regarding the individual's life history, behaviour and role in a palaeoecological context.

The broad survey, description and analysis of pathology specifically from a Plio-Pleistocene assemblage provide information as to the types of disease or traumas suffered by specific taxa and the assemblage as an aggregate. Many questions can be explored, for instance: what are the natural levels of disease? Do different diseases affect different species? Do certain animals show signs of long-term survival

following disease or trauma to a greater extent than others? If so, what conditions could be responsible for these disparities? If no evidence of pathology is found from a particular fossil site, this in itself would be a significant result, especially since skeletal lesions have been documented as common within modern wild contexts (Greer, *et al.*, 1977), and are well documented from numerous Plio-Pleistocene contexts (Tasnadi-Kubacska, 1962; Moodie, 1967; Brothwell, 1969; Baker and Brothwell, 1981; Van Valkenburgh and Hertel, 1993; Rooney, 1997; Rothschild, 2002; Rothschild and Martin, 2006). Additionally, by studying skeletal pathology from a South African Plio-Pleistocene carnivore accumulation, we may be able to elucidate predator-prey selection preferences and behaviours if the accumulating agent can be identified.

Recent advances have led to a much wider application of palaeopathology in interpreting human and hominid disease history. When dealing with Plio-Pleistocene fossil sites, however, it is important to recognize that hominid fossils are relatively scarce (de Ruiter, 2001). This suggests that hominids were a small part of a much larger faunal complex, and even though unique and subject to different selective pressures, were most likely affected by many of the same environmental stresses affecting other species at the time. Therefore, analysis of pathology in the fossil fauna may provide important clues to reconstructing aspects of hominid biological history. Moreover, in examining the faunal pathology, it might be possible to identify the precursor of a disease (i.e. the animal carrier) before humans contracted the condition (Brothwell, 1969).

There is no question that the analysis of the pathology from the fossil sites is an important step in expanding our database of the Plio-Pleistocene in South Africa. The lack of information about pathology from this geographical area is likely an artifact of analysis, whereby pathology has historically gone un- or under-recognized. The overarching goal of this study is to address this oversight and to contribute to the overall body of knowledge of the hominid-bearing sites of the region.

1.2 Palaeopathology Background

The development and evolution of the science of palaeopathology ran, in many ways, a predictable course. Many early palaeontologists and archaeologists were equipped with knowledge of vertebrate skeletal function and normal anatomy. Realizing early on that not all specimens conformed to that normal baseline required the special examination, description and interpretation of the abnormal material. It was this need to interpret skeletal aberrations that led to the genesis of the field of palaeopathology. While the technology, terminology and methodology have evolved over time, the overarching goal of palaeopathology has remained much the same.

In the late part of the 18th-century medical expertise was first applied to abnormal and ancient skeletal material in an attempt to describe a condition and infer a cause of an observed abnormality. Esper's 1774 work included the description of a deformed femur of a cave bear, *Ursus spelaeus* (Brothwell, 1969). He attributed the deformity to osteosarcoma, a diagnosis that was later proven incorrect, with the lesion being attributed to a fracture and subsequent callus (ibid). Another notable contribution was made by Cuvier in 1822, when he described a hyaena skull displaying a healed lesion on its occipital crest (ibid). Cuvier, although prominent in the field of comparative anatomy, failed to provide a satisfactory discussion of the lesions (Moodie, 1967), with little or no attempt made to explore the biological or pathological significance of what was being described (Ortner, 2003).

The earliest phase of palaeopathology was characterized by a focus on fossil (rather than sub-fossil) animal remains (Roberts and Manchester, 2005). The cave bear, in particular, received a great deal of interest regarding pathological conditions (Moodie, 1967; Brothwell, 1969). These first steps in the field (although not considered a distinct field or given the name palaeopathology until 1913) led to two important theoretical breakthroughs. Firstly, it became clear that disease is not new to animals and man, but has its history in the distant past. Ackerknecht (1955: 3) in his influential text on the history of medicine makes this point, stating "disease is old, far older than

mankind, in fact about as old as life on earth". Secondly, it was discovered that palaeopathology may allow a level of insight into the relative health of an individual animal that died thousands or even millions of years ago.

By the mid 19th century the interest in animal palaeopathology had waned as the prevailing focus shifted to human archaeological material. Warren in 1822 and Gross in 1855 both described the artificial cranial deformation of human skulls (Ortner, 2003). The close of the 1800's brought with it a debate involving the evolution of the treponemal disease, syphilis. This debate continues to this day, and marks an important step in the evolution of the field, as it was one of the first attempts to reach beyond a descriptive goal and shed light on a true biomedical problem (Ortner, 2003).

Sir Marc Armund Ruffer's research on Egyptian mummies began in the early 1900's. Ruffer has been credited with both coining the term and formally establishing the field of palaeopathology (Goldstein, 1969; Moodie, 1967; Zivanovic, 1982).

Moodie's (1923) publication was the first comprehensive text on pathological conditions in fossil material. In it he addressed skeletal lesions occurring on both non-human and human remains (Goldstein, 1969). The publication focused on gross observation and microscopic analysis of disease conditions (Ascenzi, 1969) and was unmatched as the preeminent text in palaeopathology for decades (Jarcho, 1966).

The palaeopathological studies conducted through early 1900's were undeniably crucial to the evolution of the field, but remained generally descriptive in their scope. The most current phase of palaeopathology has been marked by a veering away from the strictly descriptive methodology and an increased recognition of epidemiological and demographic implications of pathological conditions (Roberts and Manchester, 2005). Also, there has been an ongoing interest in establishing standardized methods for collecting data (ibid), and marked endeavours at the synthesis of large amounts of skeletal data on disease (e.g. Bennike, 1985; Roberts and Cox, 2003; Steckel and Rose, 2002).

Current palaeopathology research has confirmed that skeletal lesions of various aetiologies appear throughout taxonomic groups and geological time. Skeletal manifestations of pathological processes similar to those seen today have been documented in primitive whales (Moodie, 1967), Tyrannosaurs (Rothschild *et al.*, 1997), Plesiosaurs (Hopley, 2001), and Pleistocene fauna (Brothwell, 1969; Rooney, 1997). These studies have shown that although disease processes can and do evolve, the skeletal manifestations of disease are constant and recognizable throughout time and across taxonomic groupings. Most current research in palaeopathology has been conducted on human remains from archaeological contexts. These works include the comprehensive and introductory texts, such as Aufderheide and Rodriguez-Martin (1998), Ortner (2003), and Roberts and Manchester (2005).

1.2.1 Human palaeopathology

There are certain pathologies commonly encountered in archaeological human remains. This section provides an overview of common pathological conditions, including how they can help us decipher life in the past, and, in some cases, benefit life today and in the future. An in depth discussion of the pathological processes and skeletal changes that manifest as a result will be presented in Section 2.2.

Joint disease has classically been divided into hypertrophic and atrophic variants (Ortner, 2003). Osteoarthritis, after dental disease, is the most ubiquitous condition in archaeological skeletal samples (Weiss and Jurmain, 2007). The most common sites for osteoarthritis are the distal interphalangeal joints, metacarpophalangeal joints, knees, hips and vertebrae (Cooper, 1998). Osteoarthritis has been used as an indicator of activity-related or occupational stress (Wienker and Wood, 1987). Patterning of arthritis is an important clue in reconstructing specific behaviour or occupation. Diffuse idiopathic skeletal hyperostosis (DISH), ankylosing spondylitis, and rheumatoid arthritis are several of the many other arthropathies identifiable in skeletal samples.

Traumatic lesions occur with frequency in archaeological samples and can provide evidence for accidental injury or intentional interpersonal violence. Parry fractures, fractured hyoid bone and cranial injuries, for instance, have all been used to indicate interpersonal acts of violence. It is important to note that accidental injury is still the most common cause of fracture; for example, not all fractures to the forearm represent parry type injuries (Grauer and Roberts, 1996; Judd, 2008).

Inflammatory bone lesions can represent chronic infection. In archaeological samples inflammatory lesions are most commonly non-specific and therefore described according to the parts of the bone or skeleton that they affect (i.e. periostitis, osteomyelitis, or sinusitis). Infection was the major cause of mortality in antiquity and has been referred to as the "single greatest threat to life" (Ortner, 2003: 180). Infectious bone lesions can indicate specific infections, including tuberculosis, leprosy, yaws and syphilis. The identification of these lesions can be an important step in determining the antiquity and evolution of the diseases. For example, the history and origin of the treponemal disease, venereal syphilis, is heavily debated (Ortner, 2003).

Metabolic disturbance or imbalance can create a myriad of gross alterations to normal skeletal tissue. Most metabolic disturbances indicate a problem in nutrition resulting from either too much or too little of a dietary component (Ortner, 2003). Vitamin C deficiency can lead to scurvy which results in lesions of the skull and postcrania. Vitamin D deficiency can lead to rickets or osteomalacia, both of which can present skeletal lesions and provide evidence for either dietary imbalance or lack of exposure to sunlight. Indicators of arrested growth or development, such as Harris lines, can indicate systemic stress suffered during the maturation of the skeleton. Similarly, enamel hypoplasia can indicate inadequate or inconsistent nutrition during the formative years (Aufderheide and Rodriguez-Martin, 1998).

Bone neoplasms represent essentially uncontrolled new growth of tissue and can be benign or malignant. While in human skeletal remains benign tumours are most common, malignant tumours are perhaps of "the greatest pathological interest"

(Roberts and Manchester, 2005: 257). Osteosarcoma, which occurs during the growing period of the skeleton, is the most common malignant tumour in modern times and antiquity (Aufderheide and Rodriguez-Martin, 1998). If untreated, it often results in severe pain and early mortality (Roberts and Manchester, 2005). The rarity of these malignant tumours from archaeological sites does not diminish from their significance in modern cancer research.

Palaeopathological analyses of human archaeological remains have generated a wealth of information regarding the aforementioned conditions. While the analysis of pathological human skeletons has provided a database of publications too numerous to list, it is worth noting that specific disease conditions have been identified in the archaeological record. These include specific cases of metabolic disturbances (e.g. Ortner and Mays, 1998; Marcsik *et al.,* 2002; Mays, 2008), neoplasia (e.g. Ortner *et al.,* 1991; Marks and Hamilton, 2007), arthritic disorders (e.g. Bourke, 1967; Waldron and Rogers, 1994; Debono *et al.,* 2004), trauma (e.g. Roberts, 1991; Lovell, 1997; Djurić *et al.,* 2005; Owens, 2007), and infection (e.g. Roberts, 2000; Roberts and Buikstra, 2003).

Analyses, specific to southern African human remains have yielded data on various types of pathological conditions present in the archaeological record in southern Africa. Signs of arthritic or degenerative changes have been discussed (Steyn *et al.,* 2002a; L'Abbe, 2003, Mosothwane and Steyn, 2009). Possible cases of metabolic disturbance and infectious disease have also been reported (Steyn *et al.,* 2002b). Disease frequencies have been noted in several instances (Peckmann, 2002; Mosothwane and Steyn, 2009), and in some cases, palaeopathological analyses have also been used to formulate palaeoepidemiological discussions (e.g. Steyn *et al.,* 2002a*;* Steyn, 2003).

Pleistocene hominids, although older than the archaeological material mentioned above, have also shown signs of recognizable and diagnosable conditions (Walker *et al*., 1982; Trinkaus, 1985; Ripamonti, 1988; Walker and Shipman, 1996; Dawson and

Trinkaus, 1997; Fennell and Trinkaus, 1997; Ripamonti *et al*., 1997; Czarnetzki *et al.*, 2003). Some of these conditions have been interpreted as providing evidence of dietary and behavioural traits for both the individual affected, and the group of which the individual was a part. Perhaps the most heavily debated behavioural character attributed to hominids through this research has been the idea of conspecific care (Walker and Shipman, 1996; Lebel *et al.*, 2001). Although controversial (DeGusta, 2002; DeGusta, 2003), studies of this type show the potential for palaeopathology to ascertain group behavioural traits from pathological remains.

1.2.2 Animal Palaeopathology

As discussed above, information pertaining to human remains is vast, while literature on animal palaeopathology is comparatively scarce and mostly limited to domestic animals with each case treated largely for its own intrinsic interest (Hesse and Wapnish, 1985; Thomas *et al.*, 2002; Vann, S., 2008). Further, it appears that rates at which diseases occur in wild animals are historically poorly known (Hesse and Wapnish, 1985). While recent years have seen a renewed interest in animal palaeopathology, the literature is still largely focused on cases of domestic animals and defects associated with domestication (e.g. Higham *et al.*, 1981; Baker and Brothwell, 1980; Baker, 1984; Bartosiewicz *et. al.*, 1994; Cupere *et. al.*, 2000; Bathurst and Barta, 2004; Fabiš, 2004). There are, however, many palaeopathology studies pertaining to fauna that provide an excellent source of reference.

There are several early monographs that reviewed faunal palaeopathology and, therefore, provide a good starting point for discussion. Brothwell and Sandison (1967) condensed and reprinted a 1917 paper by Moodie (1967), in which he documented a broad range of abnormal conditions in prehistoric animal bone. He identified arthropathy and necrosis in dinosaur remains, osteomyelitis in a Pleistocene wolf, and hyperostosis of Paleozoic fish and Mesozoic reptiles. This was important contribution as it clearly demonstrated the broad range of animals affected by bone pathology. Tasnadi-Kubacska (1962) also reviewed and documented pathological conditions affecting a range of vertebrate and invertebrate fossil fauna. Brothwell (1969) in a

similar review of palaeopathology discussed evidence of arthritic conditions, injury and infection in Pleistocene and more recent fauna.

Baker and Brothwell (1980) published, what remains one of the most important contributions to animal palaeopathology. In the first comprehensive text of its type, the authors discussed the range of abnormalities affecting specifically animal bone, including abnormalities of development, traumatic injury, neoplasia, joint disease, inflammation, infection and oral/dental pathology. It was for many years the preeminent text in faunal palaeopathology.

Rothschild published several informative papers regarding joint disease in wild samples. Rothschild *et al.* (2001) document spondyloarthropathy as being the most common form of arthritis from both fossil and extant groups of the Order Perissodactyla. Rothschild (2003) documents erosive arthritis, osteoarthritis, diffuse idiopathic skeletal hyperostosis (DISH), joint eburnation and dental injury from a wild sample. Interestingly, it was suggested that a pathogen may predispose bovids to an erosive arthritic condition. The research also makes behavioural inferences based on pathological evidence which suggest that a lion, *Panthera atrox*, was forced to adopt a scavenging lifestyle due to a joint disease.

An invaluable contribution to animal palaeopathology was made with the establishment of the International Council for Archaeozoology (ICAZ) Animal Palaeopathology Working Group (APWG) in 1999. The APWG provided a forum for the multi-disciplinary discussion of the challenges and advancements made in the field of animal palaeopathology. Davies *et al.* (2005), in a synthesis of sessions presented at the ICAZ conference in 2002, provide a valuable resource regarding domestic animal health and diet from a range of archaeological contexts. In this volume, Murphy (2005), in a survey of animal palaeopathology from disarticulated assemblages from prehistoric and historic Ireland, noted a marked increase in the frequency of lesions from prehistoric (0.3 - 0.7%) to historic period sites (0.16 - 0.81%). The author proposes that the difference in the prevalence of pathology may be explained by

changes in livestock husbandry practices. Also in the same volume, Daugnora and Thomas (2005) in an analysis of horse burials from Lithuania, noted pathology to occur at a frequency of 12.5%. The lesions were focused on the head, vertebral column and distal extremities. Most of the observed lesions involved ossification of metapodial ligaments and were considered likely caused by mechanical strain during use of the animals for traction or riding.

As with human palaeopathology, there has been increasing interest in establishing standardized recording methods for faunal palaeopathology. Bartosiewicz *et al.* (1997) established a method for scoring deformations on the metapodials and phalanges of cattle. Levitan (1985) similarly established a methodology for recording pathology in ungulate mandibles. While valuable, these recording protocols were created to answer specific research questions and address specific pathologies (Vann and Thomas, 2006). Vann and Thomas (2006) stress the importance of establishing a common language and methodology. Efforts to standardize data collection for faunal palaeopathology are ongoing. Vann (2008), for instance, has made an important contribution by creating a standardized recording method for cranial and postcranial faunal pathology which will allow for inter-site comparisons. Amongst other concerns, she addressed the major issue of ambiguous and confusing terminology by suggesting standard basic descriptive terms when first recording pathology.

1.2.3 Southern African faunal analyses

Site specific faunal analyses and faunal population studies provide possibly the best insight into the type and frequency of pathology from past animal populations. Southern African archaeological faunal assemblages have historically been analyzed to ascertain the types of animals present, any information regarding diet and economy of past human populations, environmental conditions present at the time the assemblage was being formed, and any other incidental information that can be extrapolated from the analysis of the faunal material. Several publications mention pathological individuals or conditions, although this is never the main thrust of the research. When mentioned, these analyses give information on the types of faunal skeletal pathology

encountered in domestic and wild assemblages. Many faunal analyses, however, make no mention of pathology, as pathology may either not be present, may not be within the scope of the project, or may simply be overlooked or unidentified (e.g. Plug and Brown, 1982; Voigt and Plug, 1984; Plug and Keyser, 1994; Badenhorst *et al.*, 2002). Several faunal analyses that do mention pathology, conducted on southern African archaeological sites will be discussed below.

The faunal assemblage from Rose Cottage Cave, South Africa, was analyzed by Plug and Engela (1992). The sediments cover a period from recent times to the late Pleistocene. Pathological specimens were noted as being scarce. The phalanx of a bovid showed an exostosis. A phalanx of a springhare showed a healed but misaligned fracture. Both abnormalities were attributed to trauma.

Faunal remains from Nanda, an Early Iron Age site in Natal, were analyzed by Plug (1993a). The sample contained 8000 specimens including domestic and wild species. Pathological changes were noted on the rib of a sheep/goat and on a cattle incisor. It was noted that the rib had been fractured, resulting in abscess formation and encapsulation of the lesion by newly formed reactive bone. The cattle incisor showed very heavy wear, a pathological damage that was noted by Plug (1993a) as being common among aging bovids. Both cases of identified pathology from the sample of 8000 specimens belonged to domestic animals, and no pathology was noted on the wild faunal remains.

The site of Maqonqo Shelter is located in KwaZulu-Natal. The faunal remains were analyzed by Plug (1996). The main occupation phase of the site is dated to between 9000 and 3500 BP. The assemblage is heavily fragmented and although it exceeds 300 000 specimens, only 1.7% were identifiable to species or animal size category. A wide variety of wild species were present. Pathological changes were identified on only one specimen. A proximal phalanx of a small bovid shows an abnormality occurring on the medial facet of the facies proximalis.

Faunal remains from uMgungundlovu (the military headquarters of the Zulu King Dingane) were analyzed by Plug and Roodt (1990). This fauna represents a comparatively recent assemblage dating from 1829-1838. The vast majority of animals present were domestic. Interestingly, exostoses were apparent on 13 cattle phalanges. The authors propose that aging or draught use may account for the frequency of the abnormalities. No pathology was noted on wild faunal remains.

The macrofaunal remains from Abbot's Cave and Lame Sheep Shelter in the Seacow Valley were analyzed by Plug (1993b). Most of the deposits at both sites were dated to between 700 and 300 BP. Pathology is noted on the proximal metatarsus of a medium-size bovid, in the form of an exostosis on the plantar side near the articular surface. The distal radio-ulna of a springbok shows a similar exostosis. A rib of a medium-size bovid shows a healed fracture. The author suggests that the cases of pathology represent trauma rather than illness, possibly attributable to trampling and kicking encountered in herd animals.

The faunal remains from mMatshetshele, a Late Iron Age site in the Rustenburg district, were analyzed by Badenhorst and Plug (2001). Of the faunal assemblage, 60.1% of the bones were attributed to domestic animals. Exostoses were noted on the first phalanx and metacarpal of a cow. Both bones may have belonged to the same individual.

Several other studies demonstrate faunal assemblages with elements that show some evidence of pathological changes (see Plug, 2000, 2004). These studies do shed light on the types of skeletal pathology encountered in wild and domestic animal assemblages from specific sites, however, in most cases the abnormal specimens are mentioned as a subheading under taphonomic changes, and treated as an aside rather than an integral part of the findings.

1.3 Taphonomic Considerations

Taphonomy, as defined by Lyman (1994: 1) is "the study of the transition, in all details, of organics from the biosphere into the lithosphere or geological record." Fossil bones have undergone extensive mineral replacement in order to have survived to the present. They have also undoubtedly been subjected to other taphonomic processes, often complicating their gross appearance. Whereas palaeopathology strives to interpret all antemortem skeletal changes, taphonomy has, as a focus peri- and postmortem phenomena, including pre- and post-burial histories of preserved remains. This requires both sciences to have as a comparative baseline a model of a specific living specimen (Lyman, 1994). As such, each weighs heavily on the other, due to the fact that not all fossilized specimens were 'normal' during their natural life, and not all changes seen in preserved remains are the result of pathological processes. In order to make inferences on the one, one must first have a grasp of the other.

True taphonomic processes begin with the death of the individual (Lyman, 1994), however, perimortem structural disturbances are difficult to distinguish from postmortem changes to fresh or green bone. As such, perimortem events are included in taphonomic results, and more often than not, excluded from pathological studies.

Gross examination of fossils is extremely important in distinguishing between pathological and taphonomic change. This can often prove difficult as certain taphonomic traces, and disease or traumatic processes can appear grossly similar. These include erosive foci, fractures, disturbances of shape, and projections from the bone surface (Baker and Brothwell, 1980). If due to postmortem conditions, these phenomena are referred to as pseudopathologies (Wells, 1967), and are classified by Ortner (2003: 45) as being the result of two basic conditions, "(1) the immediate burial environment and (2) problems during or after excavation." Therefore, in order to confidently assess pathological conditions, the first step is to determine whether the aberration is in fact antemortem.

1.3.1 Taphonomic fracturing of bones

The same basic principal underlies both ante- and postmortem bone breakage. When loading exceeds tensile strength (the property allowing a material to resist rupture under tension), bone fractures. Fresh bone is viscoelastic and, as such, able to withstand great amounts of pressure before failure (Lyman, 1994). Dry bone, on the other hand, behaves more like an inorganic material, and is stronger when subjected to static loading, but more likely to fail under smaller forces during dynamic loading (ibid). Although the type of fracture will rarely identify the agent of that breakage, it can often indicate whether the injury was suffered while the bone was still fresh (at or around the time of death) or dry (sometime after death and decomposition of soft tissue). This is complicated by the fact that fractures suffered at or soon after the time of death appear grossly similar to fractures suffered while the animal was still alive. The appearance of active bone repair at the point of injury remains the primary way of distinguishing between fractures occurring ante- and postmortem.

1.3.2 Taphonomic modification of bone surfaces

Proliferation of bone tissue is always the result of antemortem events. Any evidence of bone deposition resulting in abnormal morphology can be considered pathological. The burial environment can, however, complicate matters. Sedimentary calcretions and mineral deposits, e.g. manganese dioxide, can attach to bone surfaces to give the appearance of projections of abnormal bone tissue. Fossils can become embedded in matrix which can further obscure the anatomical morphology of the specimen and give the appearance of an abnormal shape.

Whereas all bone proliferation is the result of antemortem processes, bone erosion can be caused by a multitude of postmortem agents. Sedimentary abrasion, water erosion, carnivore damage, fungus, plant roots and damage incurred at the time of excavation can all mimic antemortem erosive lesions. Mechanical stresses can also cause postmortem warping or erosion of the cortex (Wells, 1967). Most antemortem lytic lesions have a depositional component, so evidence of active repair and deposition is the best indicator that the insult was suffered *in vivo*.

1.3.3 Quantification of assemblages

It is important to understand that a collected fossil sample is not directly representative of the living animal community in their natural proportions (Klein and Cruz-Uribe, 1984). There is an inevitable loss of information in the transition from the life assemblage to the death assemblage and eventually to the collected sample assemblage (Klein and Cruz-Uribe, 1984, Lyman, 1994). We are studying, as Roberts and Manchester (2005: 12) refer to it "a sample of a sample of a sample … of the original living population". A number of taphonomic variables influence the eventual assemblage. The fossil sites in South Africa are generally thought to represent carnivore accumulations (Brain, 1981; de Ruiter and Berger, 2000; Pickering, 2002), whereby prey selection would have clearly influenced the composition of the fossil sample. The preservation of the individual bones within the assemblage would also be affected by the structural architecture and density of individual elements (Lyman, 1994). Brain's (1969b) pioneering study suggested that the survival rate of bones in carnivore accumulations correlated to the structural density of the elements. It has since been corroborated that bones with greater structural density are better preserved in fossil contexts (Brain, 1981; Schick *et al.,* 1989; Carlson and Pickering, 2003). With these variables in mind, it becomes clear that the quantification of an assemblage is a complicated, yet critical step in any analysis of fossil bones.

The most commonly used units of quantification in the analysis of faunal assemblages are the number of identified specimens (NISP) and the minimum number of individuals (MNI). The most readily obtainable index of species abundance is the NISP (Klein and Cruz-Uribe, 1984; Lyman, 2008). The NISP is an observational unit and can be directly measured (Lyman, 1994), and simply represents a count of specimens identified to element and taxon. Several authors (Klein and Cruz-Uribe, 1984; Lyman, 1994; Lyman, 2008) have discussed the inherent shortcomings of the NISP, including the following:

- The NISP ignores the fact that some species have more skeletal parts than others.

- The NISP ignores specimen interdependence and treats every unit counted as if it comes from a different animal.
- The NISP is not sensitive to bone fragmentation.

The MNI is a determination of the minimum number of individuals necessary to account for all the identified bones (Klein and Cruz-Uribe, 1984). The MNI is traditionally calculated by separating the most abundant element of a taxon into left and right components. Then the side that gives the greatest number is used to calculate the minimum number of individual animals (White, 1953). The calculation, by its nature, overcomes the key issue of specimen interdependence because it avoids counting the same animal twice (Lyman, 2008). The MNI is also not additive, and therefore not affected by moderate bone fragmentation, or by the number of bones in the skeleton. The MNI, however, does have its own shortcomings. Lyman (2008) discusses some of the inherent problems, including the following:

- MNI values will tend to exaggerate importance of rarely represented taxa.
- MNI values will increase as the intensity of bone fragmentation increases.

1.3.4 Taphonomic variables of pathology

Pathological conditions present their own unique taphonomic variables. Diseases that affect bone metabolism, for instance, can result in decreased bone mass and can counteract bone preservation (Bartosiewicz, 2008). Fractures and infections can also lead to decreased bone density and increase the likelihood of bone taphonomic loss. Age is a critical variable which can affect postmortem survival of elements and can influence the rate of expression of bone pathology (ibid). Older animals will tend to exhibit a higher bone mineral content and have a greater chance at postmortem preservation. Also, with increasing age there is an increase in the likelihood of an individual to suffer illness or injury, which can increase the frequency of observed pathology.

1.4 Surveyed Sites

The two sites surveyed for this study are Cooper's D and Swartkrans (Figure 1.1). The sites fall within a large complex of fossil-bearing dolomitic cave deposits critical to unlocking the mystery of hominid evolution in southern Africa (Brain, 1981). There are dozens of sites from this area that are currently undergoing, or have at one time undergone excavation, and many more yet to be surveyed or actively researched. Collectively, these dolomitic cave deposits have been recognized for their scientific value, and the area in which they are located has been designated a World Heritage Site, *The Cradle of Humankind* (Hilton-Barber and Berger, 2002).

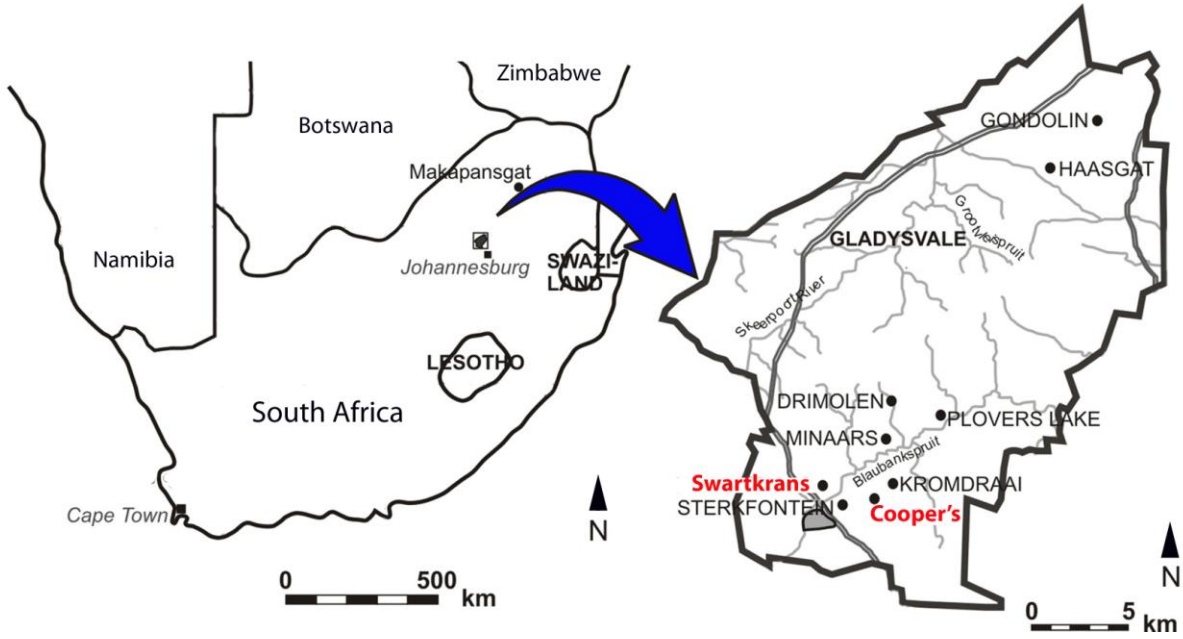


Figure 1.1. Map of the Cradle of Humankind, Witwatersrand, South Africa, showing the sites of Cooper's and Swartkrans. Adapted after Backwell *et al.*, (2009).

1.4.1 Cooper's D

The Cooper's Site is one of the lesser known sites adjacent to Swartkrans, Sterkfontein and Kromdraai. Although only recently recognized for its fossil richness and potential

as a hominid bearing deposit, Cooper's was discovered and first excavated in 1938 (Berger *et. al.,* 2003). The initial surveys yielded a substantial faunal collection and a tooth described as "human" by Middleton Shaw (1939). The specimen, an isolated third molar, was further analyzed by Broom and Schepers in the years that followed, and determined to be attributable to *Australopithecus africanus*. A cast of the specimen was again studied in the 1990's, due to the disappearance of the specimen itself. The researchers further corroborated Broom and Schepers' determination and a taxonomic classification of *Australopithecus* cf. *africanus* was proposed (Berger *et al.,* 1995).

Although broadly considered to be one site, Cooper's is in fact made up of at least three spatially distinct infills, Cooper's A, Cooper's B, and Cooper's D (CA, CB, CD respectively) (Berger *et al.,* 1995). Following the initial excavation in the 1930s, Cooper's B was extensively sampled during the middle part of the 1950s by C.K. Brain. Brain's excavation yielded vastly different fossils from those collected twenty years earlier (Berger *et al.,* 1995). This, and the fact that primates were uncommon in the excavated material, led to the longstanding belief that the hominid material attributed to Cooper's may have in fact originated from another site (ibid). With the Cooper's hominid coming from what some considered dubious provenience, interest in the site waned until further hominid material was discovered in 1989. The specimen, (COA 1, a right central incisor) was found in the faunal collection of the Transvaal Museum. Its morphological signature suggests that the specimen did indeed come from Cooper's and not one of the neighbouring deposits (ibid).

The discovery of this specimen led to the resumption of excavations, and the opening of an entirely new deposit, Cooper's D. Excavation of Cooper's D began in 2001, almost immediately yielding *in situ* hominid remains, confirming Cooper's place amongst the hominid sites of South Africa. Based on the faunal make-up of CD and its similarity to Sterkfontein and Kromdraai, a date of 1.6 to 1.9 Ma has been allocated (Berger *et al.,* 2003). Recent U-Pb testing has adjusted and constrained the dates to 1.526 to 1.413 Ma (de Ruiter *et al.,* 2008).

The initial excavation of Cooper's, that of CA and CB, resulted in the discovery of hominid fossils as well as a "substantial collection of fossil fauna," (Berger *et al.*, 1995). The more recently excavated deposit, Cooper's D, proved richer in faunal material and the collection has been described as "abundant and diverse" (Berger *et al.*, 2003). The CD collection in 2003 numbered in excess of 9000 specimens. The recovered fauna includes numerous primates and hominids as well as animals "not usually common in the Witwatersrand Plio-Pleistocene cave record" (Berger *et al.*, 2003).

CD shows, in particular, an abundance of suids and canids. Cercopithecids, felids, and hyaenids are also well represented. Equids and giraffids are present but rare. As with most sites found within the *Cradle of Humankind*, bovids dominate the faunal assemblage (Berger *et al.*, 2003).

1.4.2 Swartkrans

The University of California conducted an African expedition in 1948, searching for australopithecine fossils in what was then the Transvaal region in South Africa (Brain, 1981). Robert Broom, then of the Transvaal Museum, was approached by the research team and offered the opportunity to excavate a new fossil locality in close proximity to the ongoing excavations at the Sterkfontein site (Brain, 1981). Within one week of beginning excavation at the site of Swartkrans, an australopithecine mandible was discovered. The mandible appeared to belong to a new form of robust australopithecine, named at the time by Broom, *Paranthropus crassidens* (ibid). Many valuable finds would follow; including remains attributed to a new type of hominid referred to as *Telanthropus capensis* by Broom, but later attributed to the genus *Homo* (Robinson, 1961; Clarke *et al.*, 1970).

Excavations continued throughout 1949 until financial constraints curtailed the excavation in November of that same year (Brain, 1981). As the cave was rich in travertine, it was extensively mined and blasted for the next two years. As a result of

the blasting several "spectacular specimens" were recovered (Brain, 1981: 221). Upon Robert Broom's death in 1951, Robinson, who had been working alongside Broom, continued the excavation of Swartkrans until 1957 (idem).

The following years saw the publication of two important Transvaal Museum Memoirs, *The Dentition of the Australopithecine* (Robinson, 1956) and *Transvaal Ape-Man Bearing Cave Deposits* (Brain, 1958).

Excavations resumed in 1965 with the explicit aim to restore order following the destructive and "chaotic" mining episodes during the 1950's (Brain, 1981). In 1968 the University of the Witwatersrand acquired the Swartkrans farm and cave site. During this next chapter in the story of Swartkrans, further significant discoveries were made including evidence for the contemporaneous existence of *Homo* and the robust australopithecines (Brain, 1981). Upon clearing the overburden, the next phase of activity included the excavation of the *in situ* sediments underlying the miner's rubble (Brain, 1993).

Swartkrans has, since the onset of the excavations, become synonymous with palaeoanthropology in South Africa, noted as being among the richest hominid fossil deposits in Africa (Grine, 1993). A staggering amount of work has been conducted on all facets of the site. A monograph published by the Transvaal Museum, devoted to the site of Swartkrans was edited by Brain (1993) and included research on the geology (Brain, 1993a), composition of the faunal assemblage (Watson, 1993), description of hominid crania (Grine, 1993) and postcrania (Susman, 1993), and description of the carnivores (Turner, 1993). These and several other lines of research were addressed in the monograph which was printed a second time (Brain, 2004) with added chapters on the faunal assemblage (de Ruiter, 2004) and new evidence regarding bone tool use (Backwell and d'Errico, 2004). Whereas previous studies had suggested that the bone tools were used by *P. robustus* to dig up tubers (Brain and Shipman, 1993) experimental bone tool utilization and microscopic analysis of wear patterns by

Backwell and d'Errico (2001, 2004) provided evidence for termite foraging by the hominids from Swartkrans.

An important contribution was made by de Ruiter (2001), when a comprehensive study was conducted on the faunal assemblage and taphonomy of Swartkrans. He addressed the long-standing belief that hominids occurred in relatively high abundances, as was proposed by Brain (1981, 1993). It was postulated that a specialized predator of primates operated at the time the cave was being formed or that the cave was used as a shelter for the hominids (Brain, 1981). It was suggested by de Ruiter (2001) that the high relative abundance may have been "an artifact of analysis". A large block of breccia weighing 709kg became detached from the hanging remnant and was prepared, with the anticipation of similar frequencies. The vast majority of the bones were unidentifiable (81.2%) and only 7.4% of bones were identifiable to taxon while 8.9% were identifiable to element alone (de Ruiter, 2001). Primates and hominids were not discovered in the expected relative frequency. After including thousands of specimens not included in previous analyses, de Ruiter (2001) concluded that the assemblages could be attributed mainly to leopard with some involvement of hyaenas.

The site of Swartkrans is divided into 5 Members. These represent contextually and possible temporally discrete deposits. Member 4 is a largely uncalcified deposit rich in Middle Stone Age tools which has not yet been excavated (Brain, 1993a). Member 5 is a lightly calcified deposit dominated by remains of an extinct springbuck form *Antidorcas bondi*, and has been radiocarbon dated to 11000 years B.P. (ibid). The three Members relevant to this study are Members 1, 2 and 3. After a comprehensive study of the fossil bovids, Vrba (1975) suggested an age of between 2 and 1 Ma for the Hanging Remnant of Member 1. This date range was later refined to between 1.8 and 1.5 Ma (Vrba, 1982). The fossil fauna from Members 1, 2 and 3 do not significantly differ and it is possible that all three Members are of similar age (Brain, 1993a; de Ruiter, 2003). The range of 1.8 to 1.6 Ma has been generally accepted for Member 1. Recent U-Pb dating of fossil enamel has, however, yielded dates of 1.83 ± 1.38, 1.36 ± 0.29, and 0.83 ± 0.21 Ma for Members 1, 2 and 3 respectively (Balter *et al.*, 2008).

1.4.3 Site specific relevance

This study will address a relatively unknown facet of the South African Plio-Pleistocene. This research provides a novel approach to the study of fossil accumulation from the area, as it is the first study devoted to identifying and calculating the frequency of skeletal pathology from Plio-Pleistocene sites in the Cradle of Humankind, South Africa. Although pathological fossils have been documented from South African sites (Hendey, 1974; Ripamonti, 1988; Ripamonti *et al.*, 1997), most research has focused on one or two specimens of one taxon (in particular, hominids). While both Cooper's D and Swartkrans are hominid-bearing, both assemblages are dominated by non-hominid fossils. This indicates that an intensive palaeopathological survey of the Cooper's D and Swartkrans deposits would fall short if the focus was solely on hominid pathology. This research, therefore, focuses on macromammal remains across taxonomic groupings.

As the first study of its type focused on the region, this research has the potential to expand our database of the fossil fauna from the Plio-Pleistocene in southern Africa. It can provide information regarding: frequency of pathology at site aggregate level, frequency of pathology by taxa, pathology types, environmental stresses that may have affected the fossil fauna, and possibly individual life history traits.

1.5 Hypotheses

Six main hypotheses were formulated and tested during the course of this research.

1. There is evidence of pathology, in the form of skeletal abnormalities, present in the Cooper's D and Swartkrans fossil assemblages.
2. Different types of pathology occur in the fossil assemblages.
3. Pathology occurs on different skeletal elements at a similar frequency.
4. The frequency of skeletal pathology varies between taxa.
5. The rate of pathology for hominids will be comparable to the rates of pathology for carnivores and other primates.
6. Individual animal life-history traits can be extrapolated by interpreting pathological fossils.

Chapter 2. Bone Biology, Pathology and the Concept of 'Normality'

This chapter will serve to outline key concepts relevant to palaeopathology. The first section will serve as a brief overview of bone biology with a discussion of the cellular activity responsible for normal skeletal remodelling. The next section will provide a discussion of skeletal pathology with emphasis on the aetiology and cellular activities responsible for different types of skeletal lesions. The final section will provide a definition for the concept of 'normality', addressing the fundamental question in any palaeopathological study "what is normal?"

2.1 Basic bone biology

Fundamental to any attempt at studying pathological changes in skeletal material is the basic understanding of the cellular activities of skeletal remodelling. It is this remodelling that gives bone its remarkable reparative ability, that adapts it to changing physical strain, and that, if disturbed, leaves evidence of past diseases or trauma.

Bone tissue is a composite of two materials, collagen the organic component, and hydroxyapatite an inorganic mineral that impregnates the collagen matrix (White, 2000). This combination is what gives bone its hard, rigid, and flexible properties, also giving bone strength greater than either of its two constituents (Smith, 1985). Bone functions to support the body and protect the organs, and it serves as a site of attachments for ligaments and muscles. Bone also plays a crucial role in mineral homeostasis (Hall, 2005).

The three primary cells involved in bone remodelling are osteoblasts, osteoclasts and osteocytes. Osteoblasts are responsible for formation of the unmineralized portion of bone tissue (osteoid). Once embedded within bone matrix, osteoblasts become osteocytes, the most abundant cell in lamellar bone (Ortner, 2003). While osteoblasts

are active in the production of new bone tissue, osteoclasts conversely act to break down old bone tissue (Ortner, 1991; Martin, 1991; White, 2000). Osteoclastic resorption of bone involves both mineral dissolution and enzymatic degradation, firstly acting to lower the pH levels which solubalizes the apatite crystals, exposing the organic component which is further digested by enzymes (Ortner, 2003). The interplay between the osteoblasts and osteoclasts on a cellular level is what gives bone its inherent plasticity. It is a finely orchestrated relationship resulting in the constant change of bone tissue throughout an organism's life (remodelling), responding to loading and stress by new bone being deposited and resorbed to achieve a balance between strength and weight (Wolff, 1986) in what is described today as Wolff's law (Pearson and Lieberman, 2004). It has become clear that a variety of phenomena other than (or in addition to) mechanical stress can play a role in the cycle of bone deposition and resorption (ibid). Bone remodelling is crucial for the maintenance of normal bone structure and is such that the entire adult human skeleton is replaced, on average, in 10 years (Martin and Rodan, 2001).

The abovementioned remodelling occurs throughout the adult skeleton in response to mechanical load and metabolic influences (Sims and Gooi, 2008). The mechanism responsible for this ongoing remodelling of skeletal tissue is known as the basic multicellular unit (BMU) (Parfitt, 2002; Martin, 2002; Ortner, 2003; Parfitt, 2004), which is simply the coordinated activity of osteoblasts, osteoclasts and osteocytes. The principal behind the coupled cellular activity within each BMU is that the amount of bone destroyed by osteoclasts is equal to the amount of bone created by osteoblasts (Sims and Gooi, 2008). Osteocytes play a role in this cycle as well, with increasing evidence suggesting that osteocytes may sense changes in mechanical loading and send chemical or hormonal signals to the other cells (Ortner, 2003). The cycle of resorption and formation has been shown to take between three months and one year (Frost, 1967).

Different bones throughout the skeleton display different rates in turnover, and cancellous and cortical bone also vary in their turnover rates, all of which are the result

of responses to different needs. Cortical bone, for instance, appears to undergo remodelling in response to microdamage with virtually all BMU's being initiated by microcracks (Martin, 2002; Parfitt, 2004). Cancellous bone turnover rates, on the other hand, are largely affected by the bone's role in calcium homeostasis (Parfitt, 2002). It has been suggested that high rates of bone turnover can lead to a greater chance of future bone structural failure in the form of fractures (Parfitt, 2004).

Should the fine cellular balance between the osteoclasts and osteoblasts be disrupted for any reason, the result is either abnormal bone formation or abnormal bone resorption. All abnormal skeletal morphology associated with pathological processes is the result of disturbances in the cell populations between these two cells (Schinz *et al.*, 1951; Ortner, 2003).

2.2 Skeletal pathology

It is important to understand the cellular activity responsible for normal skeletal remodelling and conversely, the processes responsible for skeletal pathology. While veterinary texts can be helpful, they are generally clinical in scope, with a focus on animal productivity. As a result there is more information available regarding the pathogenesis of skeletal pathology common to humans. While it is clear that different specific disease conditions may affect non-human animals, the same broad categories can affect all animals. Therefore, as an analogue, the discussion below will provide examples of the pathogenesis and manifestations of diseases affecting one species, *Homo sapiens*. Humans are not always an ideal analogue group because of their distinct form of locomotion, very specialized behavior and medical intervention in times of illness or injury. The discussion is meant less as a review of specific pathological conditions, and rather, to allow a better understanding of the range of pathological processes and how they can affect skeletal tissues.

2.2.1 Joint disease

There are a number of forms of joint disease, and distinguishing between them proves difficult in dry bone samples (Roberts and Manchester, 2005). The most common form in modern humans is osteoarthritis, and after dental disease, it is the most ubiquitous pathological condition found in human skeletal collections (Weiss and Jurmain, 2007). Osteoarthritis occurs as a result of the gradual breakdown of cartilage between two adjoining bones, allowing the articular surfaces to come into direct contact.

The term, osteoarthr*itis* implies an inflammatory condition, and because inflammation was not considered a primary aspect of the pathogenesis of the condition, other terminologies, such as osteoarthrosis or degenerative joint disease (DJD), have been commonly used (Weiss and Jurmain, 2007). The term osteoarthritis is, however, gaining more acceptance as current research has shown inflammation to be critical to the pathogenesis of the condition (Punzi *et al.,* 2005). The aetiology, although long considered the result of normal aging and 'wear and tear' (Brothwell, 1981; Ubelaker, 1994; Ortner, 2003), is now considered multifactoral with genetics, anatomy, weight and mechanical influences all playing a role in its development (Weiss and Jurmain, 2007).

A common characteristic of osteoarthritis is the buildup of bony projections (osteophytes) around the joint surfaces. These osteophytes represent the body's attempt to spread the load and compensate for stress at the affected joint (Roberts and Manchester, 2005; Waldron, 2009). If the bones make contact for an extended time, the result can be eburnation, a polished appearance of the articular surfaces. Eburnation can be considered pathognomic of osteoarthritis (Waldron, 2009). Areas of bone rarefaction (increase in porosity) can occur beneath the eburnated surfaces (Lipowitz and Newton, 1985). Although osteoarthritis is common, it is often over-diagnosed in archaeological remains, with any bone displaying osteophytes around the periphery of the joint being classified as osteoarthritic. In fact, many conditions can

cause these projections and Baker and Brothwell (1980) suggest that three of the following four criteria be met before making a diagnosis of osteoarthritis:

1) Grooving of the articular surface of the bone

2) Eburnation

3) Extension of the articular surface by new bone formation

4) Exostoses around the periphery of the bone

2.2.2 Enthesopathy

Abnormal osteogenic responses can occur at entheses, sites of ligamentous or tendinous attachment. These are broadly considered enthesopathies and include osteophytic (enthesophytes) and osteolytic expressions (Mariotti *et al.*, 2004). While enthesophytes are considered multifactoral in aetiology, they are also considered to best correlate with senescence. These musculoskeletal markers indicate movement using specific muscles or muscle groups (Roberts and Manchester, 2005), and therefore have been used to interpret *in vivo* muscle activity in human archaeological samples (Kennedy, 1989; Hawkey and Merbs, 1995). There is little evidence linking enthesopathy with specific activities (Jurmain, 1999; Robb, 1994), however, repetitive loading has been shown to result in chronic injury to muscle and tendon tissues (Cutlip *et al.*, 2006). Research has shown that age may impair the ability of skeletal muscle to adapt to this chronic exposure to mechanical loading (Degens and Alway, 2003; Cutlip *et al.*, 2006), suggesting that age and trauma may, in tandem, contribute to the development of enthesial pathology.

2.2.3 Trauma

Trauma is categorized as any physical wound or injury, and can result from either intrinsic or extrinsic forces. This includes various types of fracture as well as dislocation. Bones can break when subjected to abnormal pressures or when weakened by pathological processes. Healing following a fracture is a complex event,

and is typically characterized by four overlapping stages: an initial inflammatory stage, soft callus formation, hard callus formation, and remodelling (Schindeler *et al.*, 2008). The repair process begins immediately after the injury. Local soft tissue integrity and normal vascular function are often compromised as a result of the insult. The blood vessels in the periosteum and Haversian canals are usually ruptured during the fracture (ibid). Blood flows into the fracture zone coagulating into a haematoma (Robbins, 1974). Bone tissue adjacent to the fracture becomes necrotic and fibroblasts activate, forming a fibrous union. This formation of the cartilaginous splint is crucial to the repair process since most fractures result in some level of mechanical instability. This primary callus of fibrous connective tissue is, on a cellular level, dominated by the paired activity of chondrocytes and fibroblasts (Schindeler *et al.*, 2008). Within days blood supply to the fracture develops and fibroblasts transform into osteoblasts and osteoclasts (Roberts and Manchester, 2005). At this stage, the initial fibrous splint is converted to immature or woven bone when calcium salts are deposited within the osteoid (ibid). This woven bone splint is what is known as the primary bony callus. Given sufficient time, the callus of immature bone is converted to lamellar or fully formed mature bone. This process is driven by the coupled phenomena of osteoclastic resorption and osteoblastic formation of lamellar tissue. The time it takes for the fracture to heal completely depends upon various factors, including the bone element involved, the type of fracture, the severity, and the stability during healing (ibid).

Mechanisms of fracture include flexion, shearing, compression, rotation and traction (Aufderheide and Rodriguez-Martin, 1998). Flexion fractures are the most common and occur when force is applied perpendicular to the long axis of the bone (ibid). The maximum stress often occurs at a discrete site and can result in a transverse break (Ortner, 2003). A shearing fracture occurs when two opposing forces are applied to a bone, perpendicular to the long axis of the diaphysis (Aufderheide and Rodriguez-Martin, 1998). Compression fractures occur when force is applied in an axial direction and are the result of sudden excessive impaction (ibid). Rotation or torsion fractures occur when force is applied in a twisting or spiral direction (ibid). Since the force is rotational, the fracture line follows the same natural spiral plane (ibid). Tension

fractures occur when an excessive or violent muscle contraction causes the avulsion of a piece of bone at the site for tendinous insertion (Aufderheide and Rodriguez-Martin, 1998). A sharp injury that breaches the periosteum (or penetrating fracture) can similarly cause a subperiosteal haematoma which can ossify in time, forming a solitary exostosis (Mohanna *et al.*, 2000). All fractures can be classified as either open (compound) or closed (Roberts and Manchester, 2005). In compound injuries there is an open connection between the bone and the skin, which provides an inlet for infectious agents (ibid). Apart from infection, several complications can arise following fracture, including joint degeneration or osteoarthritis, shortening of the limb, neuropathy, poor alignment or non-union, and avascular necrosis (Aufderheide and Rodriguez-Martin, 1998; Roberts and Manchester, 2005). The mechanical induced ischemia as the result of trauma is, in fact, the most common cause for osteonecrosis (Abraham and Malkani, 2004).

The second major category of trauma affecting the skeleton is dislocation. Dislocations occur when a bone is forced out of its normal articulation within a joint. A complete dislocation, or luxation, involves the complete displacement of the articulating joint surfaces (Ortner, 2003). A subluxation is a less severe injury and is characterized by the partial displacement of the articular surfaces (ibid). Dislocations are primarily soft tissue injuries and the only skeletal evidence of their presence are the bone lesions that accompany either a chronic or permanent injury (Roberts and Manchester, 2005). If the dislocation remains unreduced, distinct morphological changes can become present, creating, in some cases, new articular surfaces (Ubelaker, 1994). When a joint is disrupted, the articular cartilage cannot obtain nourishment from the synovial fluid, resulting in accelerated arthritic damage (White, 2000). It has been suggested that post-traumatic arthrosis may be an inevitable consequence of musculoskeletal injury (Browner *et al.*, 2003) whereby the articular cartilage damage occurred as the result of either sudden impact or repetitive trauma.

Occasionally soft tissue trauma can also produce skeletal changes obvious in dry bone. Acute soft tissue damage is similar to bone fracture in that it also creates a haematoma.

The haematoma is usually dissolved in time, but in certain instances the damaged tissue will respond by producing bone directly within the soft tissue. The condition is known as *myositis ossificans traumatica*, and is generally seen as bony projections arising from a site of tendon or ligament origin or insertion (Ortner, 2003; Roberts and Manchester, 2005). These lesions usually present as irregular bone growth, larger in comparison to the more common enthesophytes (or bone spurs).

2.2.4 Infection

Infectious diseases are caused by biological agents ranging from microscopic viruses to large structurally complex parasites (Inhorn and Brown, 1990). Most infectious diseases affect only the soft tissue, leaving no anatomical evidence on the skeleton, and those that do affect the skeleton generally overlap morphologically (White, 2000; Ortner, 2003; Roberts and Manchester, 2005). Infectious agents can enter the bone in three primary ways: through direct exposure due to fracture, through adjacent tissues, or through the circulatory or lymphatic systems (Baker and Brothwell, 1980; Brothwell, 1981; Ubelaker, 1989). Infectious diseases affecting the human skeleton tend to be "subacute, chronic diseases and may not be the immediate cause of death" (Ortner, 2003: 181).

Infection generally promotes an inflammatory response, and terminologies associated with these responses have become broadly used to describe infectious conditions. Osteitis is the general term for an inflammation of compact bone tissue, and is non-specific as to cause (White 2000; Ortner, 2003). Should it be caused by an infectious agent, a term like suppurative osteitis would be most accurate (Ortner, 2003). Periostitis is a similar term, broadly referring to the inflammation of the periosteum. Again, suppurative or infectious periostitis would be used to describe bone response to an infectious disease (ibid). Osteomyelitis refers to an infectious condition that begins in the marrow spaces affecting the endosteal surfaces (ibid), often resulting in an enlarged and deformed bone (Roberts and Manchester, 2005). The most common infective organism is *Staphylococcus aureus* (Ubelaker, 1994; Ortner, 2003). In a severe case, it is possible for an infection to spread to other bones throughout the

skeleton. The result of severe infection is often a disruption in the blood supply resulting in tissue necrosis, whereby the dead bone is removed by pus and discharged through openings known as cloacae (Ubelaker, 1994; White, 2000; Ortner, 2003).

Tuberculosis is an infection of soft or skeletal tissues that can affect human and non-human animals. The Mycobacterium tuberculosis complex (MTBC) includes the following species *M. tuberculosis, M. bovis, M. africanum, M. canetti, M. caprae, M. microti* and *M. pinnipedii* (Stone *et al.,* 2009). All of these can cause human and non-human tuberculosis. The organisms most commonly responsible for tuberculosis in humans are *M. tuberculosis* or *M. bovis.* It has been suggested (Gutierrez *et al.,* 2005) that an *M. tuberculosis* progenitor species and human ancestors began their coevolutionary pathway 2.5 to 3.0 mya. The reservoir of *M. bovis* is animals while *M. tuberculosis* usually begins as a respiratory infection, acquired by inhaling the bacteria laden moisture coughed into the air by an infected human (Aufderheide and Rodriguez-Martin, 1998). Tuberculosis spreads throughout the body via the lymphatic system and bloodstream (Roberts and Manchester, 2005). The tubercle bacilli settle in areas of hemopoietic marrow, which are the areas of cancellous bone. Over 40% of skeletal lesions from tuberculosis affect spinal elements, specifically the lower thoracic and upper lumbar vertebrae (Aufderheide and Rodriguez-Martin, 1998; Roberts and Manchester, 2005). The lesions spread and take hold and lead to local destruction and cavitation in the cancellous tissues. The process in the long bones tends to only affect the metaphyses or epiphyses (Ortner, 2003). The five most common clinical expressions of osteoarticular tuberculosis are spondylitis, peripheral arthritis, osteomyelitis, tenosynovitis, bursitis and Poncet's disease (Sawlani *et al.,* 2003).

The treponematoses are another category of infectious disease that can affect the human skeleton. This category is composed of venereal syphilis and the non-venereal, endemic diseases, yaws, pinta and endemic syphilis. All four diseases are caused by bacteria of the genus *Treponema.* There is some question as to whether the different diseases are caused by different species within the genera or are simply different manifestations of infection by the same species, *Treponema pallidum* (Roberts and

Manchester, 2005). All four diseases share in some basic characteristics, i.e. initial lesions, followed by a disease free latent period, and extensive secondary lesions (Aufderheide and Rodriguez-Martin, 1998; Meheus, 2005). The primary stage of yaws involves initial skin lesions at the site of infection. Osseous involvement can occur early, specifically osteitis and periostitis (Engelkens, 1999). The early stage skin lesions usually spontaneously disappear, and a latent period follows. For the majority of people the latent period is life-long, however, for 10% it is followed by a destructive phase, which results in irreversible lesions of bone, cartilage and soft tissues (ibid). Pinta similarly begins as a primary skin lesion, followed by a latent stage, and late (tertiary) changes resulting in severe and disfiguring skin lesions. Skin appears to be the only organ affected by pinta and the skeletal tissues are spared (ibid). The skeletal expressions of endemic syphilis are almost identical to yaws (Aufderheide and Rodriguez-Martin, 1998). The early stages of endemic syphilis can involve periostitis, often followed by a latent period, and then severe tertiary lesions, including destruction of the nasal area and palate (Roberts and Manchester, 2005). Humans are the only reservoir for venereal syphilis, which is spread by direct contact, through sexual intercourse. It manifests initially as an ulcerative lesion, followed by a secondary rash, and several severe manifestations in its later, tertiary stage (Aufderheide and Rodriguez-Martin, 1998). Bone lesions are thought to occur in only 10 to 20% of infected individuals, and it is probable that the prevalence of the condition in the past may be underestimated by as much as 90% (Roberts and Manchester, 2005). The osseous lesions include osteitis, periostitis and osteomyelitis. The tibiae are the most commonly affected long bones, followed by the fibulae, femora, ulnae and radii. The condition can also lead to destructive joint diseases, specifically affecting the knee (ibid). In long bones, the lesion usually begins as periosteal inflammation at the metaphyses, sometimes creating a thickened and deformed bone appearance (Aufderheide and Rodriguez-Martin, 1998). The most characteristic lesions occur on the plate bones of the cranial vault. These lesions, *caries sicca*, are considered pathognomic of venereal syphilis and are frequently seen as "jagged, radiating, and stellate scars showing depressed centers and rounded borders" (Virchow, 1896, cited in Aufderheide and Rodriguez-Martin, 1998).

Other specific infectious conditions that can affect skeletal tissues are brucellosis (caused by any species of the genus *Brucella*), leprosy (closely related to tuberculosis and caused by *Mycobacterium leprae*), several fungal infections and some viral infections. The specific expressions of the skeletal lesions vary from condition to condition and from host to host depending on the specific pathogen and the individual immune response.

2.2.5 Neoplasia

The term neoplasm literally means 'new growth'. It occurs when there is a growth of localized tissue whose cellular proliferation is no longer affected by normal growth regulating mechanisms (Aufderheide and Rodriguez-Martin, 1998). This unregulated growth of bone, cartilage, fibrous tissue, or blood/marrow can leave a lasting impress on skeletal tissues. Neoplasms are categorized as either benign or malignant. Benign growths refer to a localized mass incapable of destroying surrounding cells or migrating to other parts of the body (ibid). Malignant tumours, on the other hand are characterized by a greater degree of autonomy (Roberts and Manchester, 2005). As a result they create space for their own expansion by destroying the normal surrounding cellular tissues. Malignant cells can also detach from the 'parent clone' and spread from the primary site through the bloodstream or lymphatic channels to distant cells or organs throughout the body (ibid). The tumour is said to have metastasized if it flourishes and forms a new growth at a secondary site (Aufderheide and Rodriguez-Martin, 1998). Although tumours rarely have bone as a primary target there are over 40 different types of tumours that affect the skeleton (Ortner, 2003).

2.2.6 Metabolic disturbances

Bone is susceptible to an array of dietary or metabolic disturbances (Ubelaker, 1994). Most metabolic disturbances indicate either too much or too little of a food component (Ortner, 2003), and as such can indicate systemic stresses faced by an individual during growth. The skeletal response to metabolic imbalance can be varied. Scurvy, (the prolonged inadequate intake of vitamin C), for instance, results in a decrease in

osteoblastic activity and continued osteoclastic activity. The cellular imbalance results in cortical thinning and transverse fractures of the ribs (ibid) and weight-bearing bones. Rickets, which results from inadequate intake of vitamin D, is considered a disease of childhood. The condition generally arises in children between the ages of six months and four years (ibid). Worldwide today, rickets is the most common form of metabolic disease in children (Dimitri and Bishop, 2007). Vitamin D is necessary for the mineralization of osteoid and cartilage. Therefore, in rickets, the skeletal manifestations first occur at the rapidly growing areas of the skeleton and result in the excess formation of unmineralized cartilage and osteoid (Ortner, 2003). Rickets affects the newly formed trabecular and cortical structures and in time the weight-bearing bones of the limbs can become bent or deformed (Roberts and Manchester, 2005). Osteomalacia is caused by a defect in phosphate and/or vitamin D metabolism (Parfitt, 1990). The disease affects adults and leads to a generalized weakening of the skeleton following an accumulation of unmineralized osteoid replacing the bone (Brickley *et al.,* 2005). Pseudofractures (zones of increased radiolucency) are pathognomic of the condition and are possibly the result of stress fractures which have failed to heal (ibid). Other metabolic imbalances that can result in abnormal modification of the skeleton are osteoporosis, flourosis, localized and generalized hyperostosis, hyperparathyroidism, Paget's disease and several hypervitaminoses (Brickley and Ives, 2008).

2.2.7 Congenital and developmental defects

Developmental defects are the result of pathological changes in normal development during intrauterine life (Aufderheide and Rodriguez-Martin, 1998). These conditions can either be hereditary or acquired *in utero*. They are therefore grouped as either genetic or environmental. Sometimes the manifestations of congenital defects are obvious at birth and sometimes only years later. Developmental defects can occur in any body tissue and many affect the skeleton itself. They can result in skull or cranial deformations in the cases of macrocephaly, microcephaly, anencephaly and cleft palate (ibid). The conditions vary in severity, but in the most extreme cases, like anencephaly, are incompatible with independent life (Roberts and Manchester, 2005).

The postcranial axial skeleton can similarly be affected by a range of developmental disorders. Conditions, including cranial or caudal shifts of the lumbar vertebrae, atlas occipitalization, spina bifida occulta, spina bifida aperta manifest differently and range in severity. Spina bifida occulta, for instance, involves the incomplete fusion of the neural arch of sacral segments, without the neural structures protruding through the defect. The condition is common, and has been seen to occur at rates of 22% (Boone *et al.*, 1985) and 23% (Fidas *et al.*, 1987) for studied samples. The condition is usually asymptomatic, and often only diagnosed during a routine radiological examination (Aufderheide and Rodriguez-Martin, 1998). Spina bifida aperta (open) is much more insidious. In most instances the spinal cord and the meninges are extruded through the defect and the condition is often fatal.

2.3 Terminology

The basis for descriptions of skeletal specimens, with regards to anatomical element, osteological landmarks, parts preserved, structures of bones, and directional terms, will always rely on osteological and anatomical terminology. More precise terminology regarding gross appearances of lesions, pathological processes, broad category diagnoses, or specific diagnoses rely on language adopted from medical fields of study. Consequently, the common vocabulary used in palaeopathology literature is comprised largely of medical and specialist terminology. A list of commonly encountered terms is included in Appendix A.

2.4 Defining Normality

> "The normal anatomy, radiology, and histology of bone at all its developmental stages provide the baseline for interpreting abnormal conditions that are encountered in archaeological human skeletal remains" (Ortner, 2003).

> "Generally, one starts with the assumption that even if the true nature of the abnormality is not evident, one can at least separate normality from the rest" (Baker and Brothwell, 1981).

> "Disease, as the term is used in this study, may be defined as any deviation from the healthy or normal state of the body which has left a visible impress upon the fossilized skeleton" (Moodie, 1967).

The most important definition to any study of pathology is unfortunately, also the most ambiguous. I refer to the abovementioned quotes and the words 'normal' or 'normality'. Any study of palaeopathology hinges on the idea of normal morphology inasmuch as all deviations from this anatomical *mode* (in the statistical sense) are abnormal, yet not all gross abnormalities are pathological. If pathology is the study of structural and functional changes due to abnormal conditions, the question arises, what are abnormal conditions? Even though palaeopathology studies structure themselves around this idea of normal vs. abnormal morphology or function, the terms 'normal' and 'abnormal' are seldom elaborated upon. These terms are most often intuited rather than defined. So, the question arises, 'what is normal?'

Normal can be defined as what is statistically prevalent, or what is optimal for health (Murphy, 1972). Both definitions are correct yet sometimes conflict, and in the scope of a survey of pathology it would be assumed that the latter would provide the best fit. Normality is, however, largely contextual and is inherently a consequence of population genetics and the environment. For example, in certain instances (primarily in clinical contexts) things that negatively impact health are so prevalent that they are considered clinically normal. The degenerative bone changes that accompany

senescence are often deemed clinically normal, inasmuch as all individuals surviving to an old age will develop skeletal changes indicative of aging. In a palaeontological context, however, where survival depends on fitness and old age is neither guaranteed nor necessarily expected, age-related changes are contextually abnormal. Another important consideration is that, although age is not a disease, it can and does weaken an individual and allow the ingress of future disease (Moodie, 1967), and with advancing age, immunity is often compromised and nutritional status is often poor (Charlotte Roberts, personal communication, 2008). This is one instance in which the opinion of the clinician and palaeopathologist will inevitably differ.

Conversely, postmortem alterations can appear grossly abnormal and even mimic certain characteristics of skeletal pathology. They must be considered, nonetheless, normal, unless other more obviously antemortem changes are observed. This proves to be complicated when dealing with fossils, as many have undergone severe postmortem structural changes associated with weathering, carnivore accumulation and diagenesis.

Perhaps the best method of defining normality is to straddle the line between the two, sometimes contrary, definitions; firstly by developing a clear idea of prevalence through the examination and scrutiny of an extensive comparative collection, and secondly by considering the impact of a condition on the individual's health, regardless of the commonality of the pathology. Bone production and destruction are normal cellular processes that occur throughout the life of an organism. Bone, as a living tissue, responds to stress in a predictable way. Put simply, bone is deposited and resorbed in response to stress throughout an organism's life. Given this inherent plasticity of the skeleton, a degree of variation is to be expected within each species due to the different stresses and stressors faced by individuals. Only through the examination of a wide-ranging comparative skeletal collection can one approach a comprehension of an acceptable range of inter- and intra-species variation and thus normal skeletal morphology.

With this baseline research done and a mental template of healthy skeletal anatomy established, one is able to determine the characteristics of a normal range of variation, and define the concept of normality for each particular study.

For this study, all fossil material was compared to normal modern analogous elements. Those fossils displaying gross deviations from the otherwise normal comparative skeletons of the same family, genus, or species were selected for further study. Changes that appear to be skeletal responses to locomotory and weight-bearing stresses, such as minor changes in cortical thickness, were regarded as normal, unless similar changes did not appear with frequency in the rest of the fossil sample or the modern comparative analogues.

Traumatic skeletal changes were considered abnormal. This included isolated areas of bone deposition or resorption, and cases in which the bone has adopted an abnormal shape compared to a normal modern analogous bone.

Any other disease condition leaving an impress on the bone was considered abnormal. Manifestations of disease processes include cases of isolated bone formation or loss, cases of diffuse formation or loss, or the adoption of an abnormal shape or abnormal size of the element in comparison to a normal modern analogous bone.

Age-related conditions, in particular, osteoarthritic and degenerative changes were also broadly considered abnormal. Bone development associated with joint structures and sites of enthesial attachment were considered pathological. Any changes to the gross appearance of a joint structure, in comparison to a normal modern analogous element, were included as abnormal.

Any fossil displaying an abnormal gross morphology that could not confidently be ascribed to antemortem processes was considered taphonomic and not pathological. If there were no clear signs of antemortem bone loss or formation, the element was not included in this study.

In sum, any fossil with a gross morphology (determined as having resulted from antemortem processes) that deviated from the normal comparative template was considered abnormal. If a case arose whereby a condition was extremely common (like the case of osteoarthritis in modern human populations) a determination was reached as to whether the condition would have modified or impaired function. If so, regardless of commonality, it was regarded as pathological.

Chapter 3. Materials and Methods

This chapter is divided into three sections. The first section (A) will describe the materials used in the study, including the fossil samples, comparative samples and equipment. The next section (B) will outline the methodology used in identifying, recording and analyzing the pathological fossils. The final section (C) will provide an analogous pilot study conducted on bone accumulations from modern hyaena dens.

A. MATERIALS

3.1 Rationale for sampling

This study is a comprehensive survey of pathological hominid and non-hominid fossils from the sites of Cooper's D and Swartkrans, two Plio-Pleistocene cave sites within the Cradle of Humankind, South Africa. Both collections are extremely large, each containing over 20,000 preliminarily sorted and catalogued specimens. This large quantity required that certain limiting factors be applied in order to better define the project and narrow its scope to allow for time and resource limitations. Nevertheless, all identifiable postcranial macromammal material was examined grossly. Micro-mammalian and small mammal remains, generally considered to be those belonging to mammals less than 5 kg (Andrews, 1990), were not considered in this research. It is understood that craniodental pathology can be valuable in reconstructing animal health and behaviour (e.g. Van Valkenburgh and Hertel, 1993; Van Valkenburgh, 2009). The material studied for this analysis was limited to postcrania due to the large sample size from both sites, and the limited time available in which to conduct meaningful research. It should not be assumed that cranial and dental pathologies are absent in the assemblages. All pathological specimens studied had to be identifiable to skeletal element and family so as to allow comparisons with a normal baseline of that particular bone for that particular taxon.

For Cooper's D the frequency of pathology within different taxa was determined using a reduced sample of 1763 postcranial fossil specimens, comprised of bones preliminarily sorted to family, or more precisely to genus and species. Time constraints prevented the analysis of unidentified fossil specimens in the collection. Considering that the entire collection was examined for potential pathology, while only those already sorted were used in the calculation of disease and pathology frequencies, there was an inherent bias in the intra- and inter-species rates of disease and abnormal conditions. In order to lessen the bias, the contents of each unsorted box yielding a pathological or potentially pathological specimen were identified and added to the identified baseline sample. In view of the fact that each box contained geographically and contextually intermingled specimens, it was reasonable to assume that each box was taxonomically representative of the collection as a whole.

For Swartkrans, the second site examined for this study, the frequency of pathology was calculated using the identified postcranial assemblage of 5336 fossils. The fossil sample was drawn from Members 1, 2 and 3. Swartkrans is one of the larger and most extensively studied hominid-bearing cave sites in South Africa. The fossil sample is extremely large and very well sorted. A large number of the postcrania, the focus of this research, have been previously identified to skeletal element and taxonomic affinity and therefore no attempt was made to survey unidentified elements. Elements affected by taphonomic adhesion of matrix or extensive taphonomic erosion, whereby the gross morphology was obscured, were not included in this study.

3.2 Comparative Collections

Firstly, an extant normal baseline of faunal and human specimens was needed in order to become familiar with normal morphology, and morphological intricacies of various species identical or similar to those from Cooper's D and Swartkrans. The comparative collections housed at the University of the Witwatersrand Bernard Price

Institute (BPI) as well as those from the Transvaal Museum (TVM) were used for this purpose.

Faunal collections containing pathological material were also used for morphological and pathological comparison. These samples were not used for a population comparison, but simply to demonstrate the range of osteological changes associated with different disease or traumatic conditions. The BPI modern faunal comparative collection and the Transvaal Museum collection each contain animal bones exhibiting pathology. These collections are limited and do not contain bones representative of all of the potential bone pathology found in extant species.

In order to become better acquainted with the gross appearance of a wider range of pathological conditions, as well as to serve as a pilot study for this research, a sample of 50 human skeletons was selected at random from a particular population group from the Dart Collection, Department of Anatomical Sciences, University of the Witwatersrand Medical School. The skeletons belong to the 'Zulu' population group with a pre-1960 date of death. The idea being that such a population would have had limited medical intervention in the case of disease or trauma, and would therefore be more likely to display a broad range of skeletal pathology.

3.2.1 Normal modern comparative collections

Non-human fauna

Faunal collections housed at the University of the Witwatersrand (BPI and School of Anatomical Sciences) and Transvaal Museum included a variety of taxonomically diverse skeletal specimens, with little or no obvious signs of pathology. The number of faunal comparatives investigated was extensive due to the high degree of intra- and inter-species variation; what may be mistaken as pathological for one species, may in fact be a normal developmental characteristic of another. Over 200 faunal comparative specimens were available. Table 3.1 shows the number of different families and genera represented in the comparative sample. The complete comparative sample list is given in Table 3.3, Appendix B.

Table 3.1. The number of families and genera represented by the faunal comparative sample.

Order	Families	Genera
Artiodactyla	4	19
Carnivora	6	15
Perissodactyla	2	2
Primates	3	4
Rodentia	2	2
Tubulidentata	1	1
Total	18	43

Modern human

All but one of the human skeletons from the Dart Collection, examined in the pilot study, displayed some signs of pathology ranging from minor osteoarthritic or otherwise age-related changes to more diffuse, severe, or diverse anomalies. For the sake of having a 'normal' comparative analogue, those skeletons displaying evidences of minor solitary or polyarticular osteoarthritis were considered 'normal'. The changes of senescence were noted; however within the context of the sample this pathology was common on otherwise healthy skeletons. These skeletons were used as the modern normal human comparative sample and are listed by accession number, age and sex (see Table 3.4, Appendix B).

3.2.2 Abnormal modern comparative collections

Non-human fauna

The faunal collections housed at the University of the Witwatersrand (BPI) and the Transvaal Museum included a number of abnormal specimens. The abnormalities present were solitary or diffuse. Some bones displaying abnormal features were housed in these collections, not as part of an entire skeleton, but as a solitary skeletal piece. The specimens are listed according to accession number, taxonomic identification, skeletal elements present and nature of the anomaly (see Table 3.5, Appendix B).

Modern human

All modern human skeletons from the Dart Collection displaying conditions different from, or more severe than, a mild degenerative arthropathy were included as the abnormal modern human comparative sample. They are listed by accession number, age, sex and nature of the pathology (see Table 3.6, Appendix B).

3.2.3 Fossil comparative material

Fossil comparatives from other sites within the Cradle of Humankind were also used, not only for familiarization with normal morphology of fossilized bones, but also for familiarization with abnormal morphology of some pathological fossils. Collections containing one or more identified pathological specimens were Makapansgat, Drimolen and Gladysvale. Only seven identified pathological fossils were found in the collections housed at the BPI.

The bones displayed a range of pathology and belonged to different taxonomic groupings. Although the fossils have been identified as abnormal or pathological, none have been further described. They were, however, helpful in familiarization with the gross appearance of pathological fossilized specimens.

3.3 Equipment

- Data from the Cooper's and Swartkrans remains were entered into an Access formatted database, designed specifically for this project.
- A magnifying lamp was used for both normal and abnormal specimens to examine the intricacies of the bone appearance.
- A dissecting microscope was used to further magnify skeletal abnormalities.
- A Phillips Brilliance 16 CT scanner was used for SK 7923.
- Digital pictures of the bones displaying abnormal morphology were taken using a Nikon Coolpix 990 camera.

- All data, including photos, were entered into the aforementioned database and processed on a lap-top (Sony Vaio) computer.

B. METHODS

Interpretation of data in palaeopathology depends on accurate differential diagnosis. While clinicians (e.g. physicians or veterinarians) have at their disposal a multitude of data sources with which to make a diagnosis and structure a treatment, palaeopathologists are at a distinct disadvantage. The palaeopathologist is seldom left with soft tissue material in Holocene deposits, and practically never in more ancient Plio-Pleistocene accumulations. They do, however, in some well preserved archaeological burial contexts have an entire skeleton at their disposal, which facilitates in correct diagnosis. It is beneficial to have a complete skeleton for an accurate differential diagnosis because distribution patterns of lesions are often necessary to accurately diagnosis a condition based on modern clinical criteria (Roberts and Manchester, 2005). Because of preservation factors and methods of analysis, there will always be incongruities between what a clinician and an archaeologist observe on preserved remains (Ortner, 2003).

3.4 Examination of comparative collections

3.4.1 Modern non-human fauna

The first step in a palaeopathology study is the familiarization with a 'normal' morphological baseline for the particular species being investigated. In the case of the fossil assemblages, with their taxonomic diversity, the baseline needed to include normal comparative material for all extant macromammalian groups represented by the Cooper's and Swartkrans fossils. This involved the gross examination of the

comparative material listed in the Materials section as *normal modern comparative collections*. Most fossils from Cooper's D and Swartkrans are categorized to family level, with a low percentage being confidently identified to either genus or species. Given the time and resources available, an exhaustive examination of all extant species found in the fossil assemblages was not possible. In the cases where a species was not represented by modern comparatives, the morphological similarities between mammals of the same family or genus were considered sufficient to identify normal morphology. In the case of extinct species, examination of a modern comparative is impossible, and an extant comparative as similar as possible was utilized. Descriptive literature pertaining to extinct fauna was used in conjunction with the examination of an extant analogue species in order to establish a framework of osteological normality.

Each comparative, normal and abnormal, was examined grossly under harsh incandescent light to highlight topographic detail. Those comparative specimens determined to be pathological were also examined using a magnifying lamp to better see the intricacies of the abnormality.

3.4.2 Modern human material

A database was created specifically for this project for the sample of human material from the Dart Collection. It had as a focus the postcranial skeleton. The database was designed to note skeletal abnormalities by skeletal element and location within that element (e.g. epiphysis, metaphysis, diaphysis). Using human palaeopathology reference literature, in particular Roberts and Manchester (2005), Ortner (2003) and Aufderheide and Rodriguez-Martin (1998) particular attention was paid to the gross descriptions of the lesions. Some of the more interesting pathological bones were photographed in order to use in gross comparison with the fossils. It is clear that findings on a modern human collection cannot be transposed to apply to wild fauna, but the fact that disease, trauma, and degenerative processes affect skeletal tissues similarly in all mammals make it a good analogue. In addition, the gross appearances of conditions within broad descriptive categories (e.g. fracture, infection,

inflammation, neoplasm) are similar even though the specific aetiology may be different.

3.5 Examination of fossil assemblages

3.5.1 Preliminary examination

Having familiarized myself with the normal osteological morphology of a broad range of mammalian taxa, as well as the abnormal morphologies associated with disease conditions, the identifiable postcranial macromammal assemblages from Cooper's D and Swartkrans were examined. Micromammalian, reptilian, avian fossils, as well as unidentifiable bone flakes or fragments as categorized by Watson (1993) were not included in the examination. The identifiable macromammalian fossils were studied under the same harsh incandescent light as the comparative collections. Postmortem or potentially pathological deviations from normal morphology (as determined by the extant sample study) were separated for further study.

The Cooper's D and Swartkrans samples were entered into a database specifically designed for this project. The data fields included taxonomic affiliation, skeletal element and part identification, side information, presence/absence of postmortem modifications, and presence/absence of pathological lesions. This database provided the frequency of pathological skeletal remains within taxonomic categories and by skeletal element.

3.5.2 Bones displaying gross abnormalities

Those bones displaying abnormal morphology were initially examined grossly to separate postmortem processes from ante- or perimortem processes.

Bones displaying abnormal morphology judged as having occurred during the life of the individual constituted the sample relevant to the study. The lesions or

abnormalities having occurred at or around the time of death of the individual, or sometime after the death of the individual, were not included. If a process responsible for abnormal gross appearance of a bone could not be confidently ascribed to an *in vivo* cause that specimen was not included in the sample.

3.5.3 Descriptive criteria

The ultimate goal of the descriptive analysis of a pathological specimen is the identification of the disease or traumatic process responsible. It is important to note, however, that there is often difficulty in arriving at the identification of the specific cause of an abnormality even in modern dry bone specimens. This difficulty is generally compounded in fossil samples due to the fragmentary nature of the bones. The description of the abnormal material has, therefore, become critically important even where diagnoses cannot be reached. An accurate description, where no diagnosis can be discerned, can allow for:

- information on frequencies of cases within specific descriptive categories;
- differential diagnosis;
- future input and contributions.

3.5.4 Identification of skeletal element

A typical pathology description begins with the identification of the location of the abnormality. The abnormal bone in this study was first described to anatomical part after Ortner (2003). The descriptive criteria used here, as set forth in Roberts and Manchester (2005), Ortner (2003), Ubelaker (1994), Aufderheide and Rodriguez-Martin (1998) were intended for human archaeological remains. Modifications to terminology were made to include non-human animal material.

To identify the skeletal part, the bone was first categorized as belonging to either the axial or appendicular skeleton. The specimen was then identified as to specific skeletal element. Each fossil was then identified more precisely as representing either the entire element (complete) or a fragment of the element (e.g. proximal, distal, or

shaft fragment). The fossils were subsequently sided if such a determination was possible.

3.5.5 Determining taxonomic affinity

Once identified to anatomical part, the fossils were compared to the *normal modern comparative collection* to determine taxonomic affinity. Each fossil was examined in conjunction with the same skeletal element for a number of modern species to facilitate a close or exact match. The fossils were then confidently categorized to family, and where possible, to genus or species. Elements belonging to a member of the Family Bovidae were placed into body weight classes following Brain (1981):

- Class I: 4.5 kg – 18 kg of body mass.
- Class II: 18 kg – 84 kg.
- Class III: 84 kg – 223 kg.
- Class IV: over 223 kg.

Elements belonging to Felidae, another family marked by a high degree of variation in intra-family body size, were relatively sized for this particular study as follows:

- Small (African wild cat; *Felis silvestris lybica*);
- Medium (leopard; *Panthera pardus*);
- Large (lion; *Panthera leo*).

The rationale behind such relative sizing was to allow for comparison between individuals of similar size within a particular family.

3.5.6 Determining the nature of the anomaly

Solitary versus diffuse lesions

In practice, once identified as abnormal, lesions are ideally categorized after Ortner (2003) to see if they represent:

- a solitary abnormality with one focus;
- a bilateral and multifocal abnormality with abnormal bone visible in two or more sites within the skeleton;
- a randomly distributed multifocal pathology;
- a diffuse reduction of bone mass throughout the skeleton;

- a local or generalized disturbance in size or shape.

Unfortunately, the fragmentary nature of the fossil assemblages studied here made such a step impossible. Most fossilized bones were fragmentary, with only some of the smaller or sturdier specimens being complete. There were few fossils that readily articulated. Although many pathological bones may have belong to the same individual (based on personal observation of pathological bones from hyaena den accumulations in Mashatu, Botswana) it was impossible to determine whether disease conditions were present on multiple bones of the skeleton, and such assumptions might have lead to a misrepresentation of disease presence. Abnormal fossils categorized to animals of the same taxon and size category, although possibly from the same individual, were treated as solitary lesions unless the bones obviously articulated.

Part of fossil exhibiting pathology

Once the fossil had been identified to skeletal element and taxa, the next step involved the identification of the distribution of the abnormality on that element. Detailed systems have been developed for recording parts of fragmented bones in archaeozoological samples (e.g. Dobney and Reilly, 1988). This analysis, however; used descriptive terminology to identify the fragmented bone. The same methodology used in the pilot study, as adopted from Ortner (2003), was adhered to for the description of the fossils. Long bones were subdivided into:

- proximal joint surface;
- proximal epiphysis;
- proximal metaphysis;
- diaphysis;
- distal metaphysis;
- distal epiphysis;
- distal joint surface.

Other bones were similarly subdivided, and the abnormal part described in relation to osteological landmarks (e.g. vertebral body, articular facet). If the abnormal feature

appeared to be associated with soft tissue attachments, the ligament or muscle involved was identified and named in the description.

Type of abnormality

As mentioned earlier (see Chapter 2), it is the imbalance between the cell populations of osteoblasts and osteoclasts that leads to all abnormal skeletal morphology. Determination of the cellular activity that accounted for the abnormal gross morphology in a specimen can be helpful in reaching a general, or more specific, diagnosis. The first step in such a determination is the typing of the abnormal morphology. For the data in this study, abnormal bones were classed according to Ortner (2003) as representing either:

- abnormal bone size;
- abnormal bone shape;
- abnormal bone formation;
- abnormal bone loss.

Although abnormal bone size and shape are functions of abnormal bone loss and/or formation, they were included as separate categories, as is standard practice. These general categories give insight into the possible cellular imbalances that may have occurred. In cases where fossils display characteristics of more than one category (e.g. a lytic focus with marginal new bone formation – abnormal bone loss and abnormal bone formation), all abnormality types were listed.

Diagnosing or classifying the abnormal morphology

After collecting the aforementioned data, palaeopathology reference literature and modern comparative material were used to broadly classify the diseases and disorders encountered. The accuracy and precision of diagnosis depends heavily on the strength and experience of the individual conducting the study, the comparative material available for use, and the congruity of reference literature with the sample in question. The difficulty in arriving at accurate diagnosis in modern clinical contexts, has led some to recommend the use of broad categories of disease rather than specific diagnoses (Waldron, 1994; Miller *et al*., 1996). This study focused primarily on the

identification and description of abnormalities. Following that, several potential causes of the abnormal skeletal conditions were given. The author understands that even broad category diagnosis has a potential for error. One goal of this project was to determine whether different pathological processes were responsible for the abnormal morphologies encountered in the fossils. Pathological processes are not mutually exclusive, and two or more abnormal processes can be present in one individual and can be evident on one bone. Joint disease, for example, can be the primary pathology, or can appear secondary to trauma. Therefore, an attempt was made, in every case, to classify the primary abnormal process that potentially resulted in the abnormal gross appearance of the fossil. The possible abnormal processes were classed for the project as follows:

- Abnormality of development – Any abnormal size or abnormal shape of a bone resulting from developmental error.
- Joint disease (including spondylosis) – Any bony manifestation of an arthropathy.
- Enthesopathy – Any bone proliferation at an area for ligamentous or tendinous attachment (including the fibrocartilaginous attachment for the joint capsule).
- Infection – Any areas of bone destruction accompanied by sequestration of dead bone and variable bone production.
- Metabolic disorder – Any condition of diminished bone mass or marked mineralization of connective tissues.
- Neoplasm – Any new and abnormal growth of bone or soft tissue resulting in changes to the bone.
- Trauma – Any bone changes that appear to be the result of physical injury.
- Unknown – Any condition that cannot be confidently assigned to a category.

Ideas regarding disease pathogenesis should not be constrained by these broad diagnostic categories. The categories were intentionally broad to avoid misdiagnosis, however, if the lesion conformed to more than one of the abovementioned categories, differential diagnoses were attempted and after consulting clinical reference literature the most likely diagnostic category was suggested, with an explanation as to why.

If a condition closely conformed to a more precise aetiology, mention of that possibility was made. The diagnoses made are not considered definitive; they do, however, provide several possible (in some cases probable) causes for the abnormal conditions based on the author's most sincere efforts.

It was also important to note whether the lesion appeared healed or unhealed. This was done by gross and microscopic examination of the borders of the lesion to see whether the edges were sharp and unremodelled (unhealed) or smooth and rounded (healed) (Roberts and Manchester, 2005). Porous and disorganized bone with sharp borders indicates that the disease process was active at the time of death, and the disease may potentially have played a role in the mortality of the individual.

3.6 Descriptive format for recording pathology

It is important to reiterate that the specific cause of an abnormality cannot always be extrapolated from even the most complete specimens. The recognition and subsequent description of pathological remains becomes increasingly important to any study of fragmentary bones displaying abnormal morphology. Therefore, in the descriptions that follow, particular care was taken in making the analyses as uniform as possible, maintaining the same format throughout. Also attempting, where possible, to principally adhere to a set descriptive terminology. A data form was designed specifically for the descriptive analysis in this research, although it is adaptable for future studies. The format is outlined briefly here in order of appearance.

Taxonomic classification

Provides information on:

1) Order
2) Family
3) *Genus*
4) *species*

5) relative sizing

Skeletal element

Information includes:

1) The anatomical identification of the skeletal element

2) The identification of the part of the bone preserved (e.g. complete, vertebral body fragment, diaphysis)

3) Side preserved (right or left)

Taphonomy

Section includes a brief description of the postmortem changes that are visible during gross inspection. The specific recording of taphonomy includes postmortem fractures, carnivore damage, insect damage, adhesion of matrix, or bone weathering/exfoliation. Elements were not included in the study if the gross morphology was significantly obscured. Fracture types followed those proposed by Marshall (1989: 14).

Abnormal morphology

1) The type of abnormality was categorized as abnormal bone loss, abnormal bone formation, abnormal bone size, or abnormal bone shape. Pathological processes do not always conform solely to one category and can sometimes leave traces that overlap these type categories. Such cases were indicated by listing all the abnormality types present separated by a slash (e.g. destruction / formation).

2) Distribution of abnormality within affected bone was included here adhering principally to the methodology of Ortner (2003). Long bones were separated into parts on a longitudinal axis as follows:

- proximal joint surface;
- proximal epiphysis;
- proximal metaphysis;
- diaphysis;
- distal metaphysis;
- distal epiphysis;

- distal joint surface.

Lesions on irregular bones were described in relation to osteological landmarks.

3) The author's description of the abnormalities discovered. The descriptions relied on terminology consistent with normal anatomical positioning of the specimens. The terminology (see Chapter 2) used was as unambiguous as possible, although certain terms might need clarification.

- The terms hypertrophic or osteoblastic were used interchangeably throughout, and refer to any instance where a disease (or otherwise abnormal condition) resulted in the formation of bone tissue.
- Abnormal projections of bone extending away from the bone surface can be described as spurs or enthesophytes (if occurring at an area of ligament or tendon attachment), exostoses (if non-articular), or osteophytes (if occurring around the joint margin). To standardize the descriptions, the term 'nodule' was used as suggested by Vann (2008).
- The terms osteolytic, erosive, or destructive can be used interchangeably to refer broadly to any case in which the lesion has resulted from the destruction of bone tissue. To standardize the descriptions, the term 'cavity' was used, as suggested by Vann (2008).
- The terms osteoarthritis and degenerative joint disease (DJD) were used interchangeably throughout, while the terms 'joint disease' or 'arthropathy' were used broadly to refer to any condition affecting a joint or joint structures.

Diagnosis

A descriptive paragraph was provided, in which the author attempted to identify the specific cause of the pathology. This included the author's impression of the abnormality using veterinary, anatomy and pathology texts, as well as palaeopathology reference material. Common characteristics of certain abnormalities were listed in the descriptions (e.g. osteophytes as a characteristic of osteoarthritis) without elaboration, more comprehensive discussions of the skeletal abnormalities and the manifestations of each are made in Chapter 2. The reference material provided the basis for the identification of the cause of the anomaly; however, some specimens were also shown

to a veterinary pathologist (Dr. Emily Lane) whose impressions were included as personal communications. Differential diagnoses were made whenever possible. It is important that all possible diagnoses be explored.

Classification

Based on the description and the information derived from the reference literature and personal communications, each abnormality was classified to one of the eight possible diagnostic categories.

Life history interpretation

In each case a comment was included regarding possible life history implications occurring as a result of, or in tandem with, the pathological insult. These comments were based on the gross appearance of the lesion(s), the broad category diagnosis and clinical literature.

Photographs

Photographs are included at the end of Chapters 4 and 5 to highlight, particularly, the pathological or abnormal morphology of each specimen. There are often several photographs of the same specimen shown, either to show different views of the element or to detail the abnormality. The fossils were not necessarily photographed in anatomical position, but rather by the best angle to highlight the pathomorphology. The scale bar is always 1 cm (10 mm) for all figures. The figures appear in the same sequence as the descriptions, and are displayed under the heading of the relevant family.

3.7 Data Analysis

There is an important concept that needs to be understood prior to data analysis, i.e. frequency of pathology will always be affected by sample size. Bartosiewicz (2008), for instance, notes the non-proportionate correlation between the size of the sample

and the likelihood of encountering pathology. This concept presumably holds true in assessment of pathology between sites and between taxonomic groups within a site. Within a carnivore accumulated assemblage, sample sizes of different taxa will always vary due to differential prey selection, differences in bone density and preservation, and the difference in the number of bones in different animals. There is little that can be done to overcome these potential issues of analysis completely; however the methodology outlined below attempted to address these concerns.

The two predominant quantitative units for analysis of faunal assemblages are the NISP and the MNI (Lyman, 2008). Due to intrinsic shortcomings of each quantitative unit in isolation (see Chapter 1.3.4) it was decided that the analysis of the frequency of pathology would take a two-pronged approach. Frequency of pathology was calculated as a percentage of the NISP and the minimum number of pathological individuals was calculated as a percentage of the cMNI by taxon. The rate of pathology by NISP was the principal unit of analysis; however the rate of pathology by cMNI provided a level of perspective to address the inherent problems of the NISP, specifically: the problem of specimen interdependence; the fact that the NISP may exaggerate sample sizes across taxa; and the fact that the NISP is affected by bone preservation/fragmentation. A statistical analysis of goodness-of-fit (outlined below) also served to address the differential sample sizes.

3.7.1 Determining frequency of pathology by NISP

Each pathological specimen was treated as a discrete unit and entered into the database with the faunal aggregate. The sample was then analyzed to determine frequency of pathology by taxon. These frequencies were calculated using the quantitative unit NISP. NISP is defined by Lyman (1994) as the number of identifiable specimens per taxon. Beginning with the broadest category, the number of pathological bones for each assemblage was calculated as a percentage of the total NISP for that assemblage. Similar calculations were conducted for progressively lower taxonomic categories, always calculating the number of pathological bones for a category as a percentage of

the NISP for that category (P/NISP). All percentages were calculated according to the same methodology for each taxonomic category whether it be the entire assemblage, order, family, or relative size within a family.

3.7.2 Expected Frequencies

Expected frequencies of pathology (Ef_p) were determined at family level, assuming a random distribution of pathology across families. This was calculated using the total cases of pathology (P), the NISP for each family ($NISP_f$) and the total NISP for the assemblage (NISP) as follows:

$$\frac{NISPf \times P}{NISP} = Efp$$

As an example: there were 1269 bovid specimens from Cooper's D, there were 24 total cases of pathology, and a total NISP of 1763 for the assemblage. The calculation would be written as follows:

$$\frac{1269 \times 24}{1763} = 17.28 = Efp$$

Given a random distribution of pathology across the assemblage, one would expect 17.28 cases of pathology for bovids, proportionate to the sample size in relation to the total NISP. Actual observed frequency of pathology (Of_p) was analyzed against this calculated expected frequency of pathology for all families:

$$Ofp - Efp$$

Other analyses were performed in order to discern trends with regards to which skeletal elements were represented by pathological bones and frequency of pathology by skeletal part. For this analysis the postcranial skeleton was divided into 5 categories:

1. Vertebral (vertebral column; cervical – caudal)
2. Sternal (ribs and sternebrae)
3. Forelimb (scapula, humerus, radius and ulna)
4. Hindlimb (pelvis, femur, patella, fibula and tibia)
5. Distal-limb (carpals and distal elements and tarsals and distal elements)

All surveyed specimens were entered into a database for this analysis and expected frequencies of pathology (Ef_p) were calculated for the five skeletal categories at family level again assuming a random distribution of pathology for skeletal section. The calculation was done using NISP for the skeletal section ($NISP_s$), the total cases of pathology (P) and the total NISP for the assemblage (NISP) as follows:

$$\frac{NISPs \times P}{NISP} = Efp$$

These analyses were calculated in order to determine if there was a discrepancy between the expected frequency of pathology and the actual observed frequency of pathology. This served to correct for different sample sizes and to highlight trends.

3.7.3 Chi-square test for goodness-of-fit

Goodness-of-fit tests are conducted to evaluate the relationship between two independent variables and to determine whether the observed frequencies deviate from the frequencies expected if a given hypothesis were true (Zar, 1999). A chi-square test of goodness-of-fit was conducted using the expected and observed frequencies of pathology by family and skeletal section for both sites. A significance level of 0.05 was used. The chi-square statistic is expressed by the following formula:

$$x^2 = \sum \left(\frac{(ofp - efp)^2}{efp} \right)$$

A *P*-value less than or equal to the 0.05 significance level would result in the rejection of the null hypothesis (H_0). The following null hypothesis was tested at family level:

- H_0: Pathology occurs at a frequency proportionate to the NISP for each family.

- H_1: Pathology occurs at a frequency not proportionate to the NISP for each family.

The following null hypothesis was tested for skeletal section:

- H_0: Pathology occurs at a frequency proportionate to the NISP for each skeletal section.
- H_1: Pathology occurs at a frequency not proportionate to the NISP for each skeletal section.

3.7.4 Randomization test for goodness-of-fit

Due to limitations of the chi-square analysis resulting from small expected frequencies (Zar, 1998; McDonald, 2008), a randomization test for goodness-of-fit was also conducted. A randomization test begins by calculation of the test statistic, i.e. the chi-square statistic. Using a significance level of 0.05, the randomization test for goodness-of-fit works by running a series of replicate randomizations. If the chi-square of the randomizations is as large as or larger than the chi-square statistic (for the observed data) more than 5% of the time, the null hypothesis is rejected (McDonald, 2008). For this analysis, 10,000 randomizations were run for each test. The proposed null and alternate hypotheses are the same as above.

3.7.5 Frequency of pathology by cMNI

The minimum number of individuals (MNI) is defined as the minimum number of individual animals necessary to account for a specified set of faunal specimens (Lyman, 1994: 100). The MNI is traditionally calculated by separating the most abundant element of a taxon into left and right components. Then the side that gives the greatest number is used to calculate the minimum number of individual animals (White, 1953). For example, if bovid humeri were the most abundant element found in an assemblage and 13 were left and 17 were right, it would mean that a minimum of 17 individual bovids were represented by humeri (MNI=17). The MNI method for calculating relative abundance has been widely criticized (Grayson, 1979) although it

still remains one of the most extensively used methods for quantifying faunal assemblages.

The comprehensive minimum number of individuals (cMNI) method adopts a specimen by specimen comparative approach of all elements for each taxon to compare morphological features. A cMNI, while still calculated using the most abundant element and side, also takes into account factors such as size, dental attrition, developmental age, as well as other morphological features, which in turn works to avoid the skewed estimations of the traditional MNI calculations (de Ruiter, 2004). A cMNI was calculated for the Cooper's D (de Ruiter, personal communication, 2005) and Swartkrans (de Ruiter, 2004) assemblages, using postcranial and craniodental material derived from the same reduced sample surveyed for this study. Teeth were generally the most abundant elements from the site and also most diagnostic for assigning a taxonomic identity, therefore most cMNI calculations were calculated using teeth (de Ruiter, personal communication, 2005).

The cases of pathology were analyzed against the cMNI to highlight the relationship between the minimum number of individuals represented by pathological bones (MNI_{path}) and the minimum number of individuals for each relevant taxonomic grouping. The pathological fossils were subjected to a similar specimen by specimen comparison for each taxonomic grouping, using size and other morphological characteristics to determine whether unmatched skeletal elements for each family, and relative size within a family, could have potentially belonged to the same individual. Comparative faunal material was used to determine taxonomic affinity and allow for specimen by specimen examination and comparison. A calculation was reached in much the same way as an MNI. Two matched skeletal elements (referring to same element and side) for one particular taxon were considered as belonging to a minimum of two individuals. If, however, several fossils could potentially have belonged to one individual (as determined by taxonomic identity, age, size, and morphological similarities) they were, for the purpose of this section, considered as belonging to a minimum of one individual. This particular quantification of the pathological

specimens is subject to many of the same criticisms as an MNI count, and the actual number of individuals represented by the pathological remains could fall anywhere between the calculated minimum number of pathological individuals and the actual number of specimens (Klein and Cruz-Uribe, 1984). The rate of pathology by cMNI was calculated by taxon with the following formula:

$$\frac{MNIpath}{cMNI} = \%MNIpath$$

C. Modern Analogous Assemblage

3.8 Mashatu, Botswana

Various carnivore taxa have been proposed as collecting agents for the sites in the Cradle of Humankind. These include leopard (*Panthera pardus*) (Brain, 1968, 1969a, 1981; de Ruiter and Berger, 2000), hyaenas (Pickering, 2002; Carlson and Pickering, 2003) as well as other large felids (Vrba, 1976; Brain, 1981). Hyaenas have been posited as a possible collecting agent for several sites within The Cradle of Humankind, Swartkrans (Pickering, 2002), and possibly Coopers. Due to the paucity of information regarding rates of pathology from modern southern African carnivore accumulations, a modern analogous assemblage was studied for comparison with the fossil results, and to add a measure of taphonomic perspective.

Over a period of two weeks faunal remains were collected from four hyaena dens in the Mashatu Reservation, Botswana, for a taphonomic study of spotted hyaena (*Crocuta crocuta*) accumulations conducted by Kuhn (2006). The resulting assemblages were surveyed for pathology (Franklin and Kuhn field notes, 2005). All elements identifiable to skeletal part and family were tabulated and entered into a

database for this comparative study. The majority of the assemblage was highly fragmented and unidentifiable. The results were tabulated by site and then as an aggregate (Table 3.2). The purpose of this comparative sample was not the specific identification or description of pathology but the calculation of relative rates of pathology by taxon. The methodology followed was the same as the fossil sample, however, no attempt was made to describe or identify the pathological condition, the simple fact that an element displayed a lesion was sufficient for the study. It is understood that the selective behaviour of hyaenas today may be vastly different from those in the Plio-Pleistocene. The selective behaviours, however, of extant hyaenas provide possibly the best analogue with which to compare the fossil assemblages. The selection of young, sick or old prey by carnivores has long been posited as a fundamental facet of prey selection criteria. Data have been collected for age at death of carnivore prey, but data regarding health of prey animals are difficult to ascertain. It has been observed by Schaller (1972), however, that poor health in hyaena prey can be prevalent, accounting for as much as 30% of their kills.

3.8.1 Site 1

Site 1 had a total of 118 macromammalian postcranial remains identifiable to family. The families present were Bovidae and Equidae, along with the bovid sub-family, Caprinae. Bovids were further classed according to size. A total of 113 bovid elements or identifiable fragments of elements were collected, 41 of which display skeletal lesions. Size classing the bovids revealed five class I, all of which are normal, 28 class II, one of which is pathological, and 80 class III, 40 of which are pathological. Only one caprine bone was found and this element is pathological.

It became clear, during the data collection, that the majority of the pathological elements belonged to a kudu, *Tragelaphus strepsiceros,* with a diffuse pathological condition affecting multiple elements throughout the skeleton. The vertebrae posterior to C-3, the ribs, long bones, and distal-limb bones are affected by the same proliferative condition. At least one class II bovid, unidentifiable to species, displays a skeletal lesion. This shows the potential for a diffuse condition in one individual to

lead to the overestimation of pathology from an assemblage, as high as 50% for class III bovids. It also further highlights the value of palaeopathology in identifying discrete elements sharing a common pathomorphology as likely belonging to one individual, and thus allowing the reconstruction of a skeleton that otherwise would have been impossible.

3.8.2 Site 2

Site 2 had a total of 28 elements from the families Bovidae, Cercopithecidae, Equidae, Hyaenidae and Suidae. No pathology was noted in the remains.

3.8.3 Site 3

Site 3 had a total of 45 elements belonging to the families, Bovidae, Cercopithecidae, Elephantidae and Equidae. Bovids account for the majority of the assemblage, followed by equids, cercopithecids and finally elephantids. One bovid specimen shows evidence of pathology (3%). Bovid size class II and III are the only size classes represented and each accounts for 50% of the bovid sample. Only 1 element from the 15 specimens in bovid size class II is pathological. All other families from this assemblage are free from observable skeletal pathology.

3.8.4 Site 4

A total of 224 elements from Site 4 were identifiable to the following families, Bovidae, Cercopithecidae, Equidae and Suidae. Bovid elements number 185 and account for the majority of the sample. Twelve (6.4%) of the 185 show evidence of skeletal pathology. All four bovid class sizes were represented by the sample.

Table 3.2. Normal and pathological elements from Mashatu spotted hyaena dens.

Site	Family	Normal	Pathological	Total	%path
1	Bovidae	72	41	113	36.28
	Caprinae		1	1	100
	Equidae	4		4	0
2	Bovidae	22		22	0
	Cercopithecidae	2		2	0
	Equidae	1		1	0
	Hyaenidae	1		1	0
	Suidae	2		2	0
3	Bovidae	29	1	30	3.33
	Cercopithecidae	2		2	0
	Elephantidae	1		1	0
	Equidae	12		12	0
4	Bovidae	173	12	185	6.49
	Caprinae	5		5	0
	Cercopithecidae	1		1	0
	Equidae	23		23	0
	Suidae	11		11	0
Total		**361**	**55**	**416**	**13.22**

Chapter 4. Cooper's D Pathology

This chapter serves to present the pathological assemblage from cooper's D. Each fossil is listed by its catalogue accession number (CD) under order and family subheadings. The information given includes the identification, description and classification of each pathological fossil. The format follows the methodology previously described (see Chapter 3.6). Figures highlighting the pathological morphology are provided at the end of the chapter.

4.1. Order: Artiodactyla

Family: Bovidae

4.1.1. CD 16967 (Figure 4.1)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Bovid size class: II

Skeletal element:

Element: Metacarpal

Preservation: Proximal 1/3

Side: Right

Taphonomy:

Postmortem fracture at proximal metaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Proximal joint surface

Description: There is abnormal bone localized to the proximal joint margin extending away from the joint surface. The lesions are seen as small rounded

lamellar nodules primarily on the medial aspect extending medially. The entire joint surface appears to have similar nodules to a lesser degree.

Diagnosis:

The bone nodules are confined to the joint margin. There is no visible abnormal bone growth associated with the entheses. Bone projections arising from joint margins (osteophytes) are noted as being manifestations of several arthropathies. Osteophytes can occur in degenerative joint disease (Baker and Brothwell, 1980), whereby degeneration of the articular cartilage results in abnormal abrasion of the bone and eventually the growth of cartilage and bone around the affected joint structures (Kahn and Line, 2005). The periarticular osteophytes can develop as the body's natural response to counteract joint instability that occurs when the cartilage degenerates and the ligaments become lax (Williams, 2002). While the osteophytes on CD 16967 may represent DJD, there are no signs of eburnation, pitting or sclerosis, making the broad diagnosis of 'joint disease' most fitting.

Classification: *Joint disease*

Life history interpretation: Some pain and possibly an altered function.

4.1.2. CD 7695 (Figure 4.2)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Bovid size class: III

Skeletal element:

Element: Thoracic vertebra

Preservation: 80% complete

Side: N/A

Taphonomy:

Postmortem fracture and possible carnivore damage

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Cranial articular facet joint margins

Description: Normal bovid thoracic vertebrae have a notch at the junction of the transverse process and cranial articular facet. In CD 7695 the space between the left cranial articular facet and the left transverse process (where the anterior notch would be in a healthy analogue) is filled by an area of abnormal bone marked by a sessile, roughly triangular nodule of lamellar bone inferior to the articular facet and projecting inferomedially for 3mm. The right cranial articular facet and transverse process have been sheared off post-depositionally; although the abnormality may be bilateral it is described here as solitary and isolated to the left side. There are also isolated areas of new bone associated with the margins of the left costal facet.

Diagnosis:

The projection of bone appears associated with an enthesis, and is therefore considered an enthesopathy. The basic aetiology for enthesophytes is considered multifactoral. Either age or conformation related changes can be responsible for the proliferation of bone at entheses (Jurmain, 1999). Acute trauma can result in ossification at sites for ligamentous or tendinous attachment to bone. The condition, *myositis ossificans traumatica*, results from a single traumatic event and bone growth is usually extensive (Ortner, 2003). In CD 7695, however, the lesion is a small spur with no other signs of physical injury, suggesting that the lesion may be the result of accumulated micro-trauma resulting from long-term mechanical strain rather than a single event. The marginal osteophytes around the costal facet suggest again that age or chronic mechanical stress may have resulted in the pathology.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

4.1.3. CD 9808 (Figures 4.3 and 4.5)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Bovid size class: II

Skeletal element:

Element: Metacarpal

Preservation: Distal epiphysis

Side: Indeterminate

Taphonomy:

Postmortem fracture at distal metaphysis.

Pathology:

Type: Abnormal bone formation / abnormal bone loss / abnormal shape

Distribution of lesion within affected bone: Distal epiphysis / distal joint surface

Description: A sharp anterior angulation of the element occurs at the distal condyle, at the juncture of the diaphysis and distal epiphysis. There is a relatively large bulbous growth of lamellar bone on the dorsal aspect of the metaphysis. The abnormal bone has a length of 15mm and a breadth of 5mm. The distal joint surface has adopted an unusual shape in comparison to a healthy analogue. The condyle is squared instead of rounded with abnormal angular nodules extending from the inferior border of the joint surface. There is also a roughly oval-shaped subchondral cavity. The subchondral lesion measures 5mm by 4mm and has closed over with a thin layer of bone.

Diagnosis:

The bulbous growth of bone on the dorsal surface occurs at the fibrocartilaginous enthesis of the joint capsule. While enthesophytes tend to be small and discrete, the lesion is large and likely indicative of something other

than age-related or chronic-wear enthesopathy. There are also nodules of bone extending from the joint margin. The extent of the abnormal growth and the sharp abnormal angulation at the metaphysis of the metacarpal suggest a more severe pathology than simply joint disease or enthesopathy, and bring to mind secondary lesions as a sequel to trauma or possibly another pathological condition. As noted in the veterinary literature (Dunn, 1999) severe or repeated trauma to the junction between two bones can create dramatic changes to the bones involved, often indistinguishable from degenerative joint disease (DJD). A sudden trauma to a joint can facilitate or hasten degenerative joint conditions resulting in joint-surface pitting and osteophytes.

The subchondral lesion appears to be *osteochondrosis dissecans* (OCD), which is considered multifactoral in aetiology (Ytrehus *et al.,* 2007), although there is some consensus that trauma is a contributing factor (Ekman and Carlson, 1998; Ortner, 2003). Ekman and Carlson (1998) note that local ischemia is a key factor in the initiation of the lesions. One of the most common sites for *octeochondrosis dissecans* is the fetlock (metacarpophalangeal) joint (Aiello, 2007). O'Connor (2008: 175) notes a similar lesion attributed to OCD on a cow phalanx.

Classification: *Trauma* (with enthesopathy and arthropathy)

Comments: CD9808 articulates with the 1st phalanx (CD10427 & CD10461)

Life history interpretation: Pain and altered gait. Animals with OCD usually present with synovial effusion and varying degrees of lameness (Kahn and Line, 2005). Johnson *et al.* (2008), in their description of *osteochondrosis dissecans* in a juvenile roan antelope, noted swelling and a limited range of motion to the stifle joint.

4.1.4. CD 10427 / CD 10461 (Figures 4.4 and 4.5)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: indeterminate

Species: indeterminate

Bovid size class: II

Skeletal element:

Element: 1st phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Postmortem transverse fracture at mid-diaphysis and weathering at distal epiphysis

Pathology:

Type: Abnormal bone formation / abnormal shape

Distribution of lesion within affected bone: Proximal joint margin / proximal metaphysis / diaphysis

Description: There is a bulbous area of irregular bone on the dorsal surface stretching from the proximal epiphysis to the diaphysis. The lesion measures 7mm by 12mm and has a rough and rutted surface appearance. The proximal articular surface of the phalanx has adopted an exaggerated 'U' shape with areas of marginal osteophytes. The shaft, although obscured by post-depositional fracture, shows signs of abnormal shape. The diaphysis displays two abnormal crests of lamellar bone running the length of the palmaromedial and palmarolateral borders. Most of the distal articular surface has been sheared off taphonomically; although they may be present, no changes to the distal joint surface can be seen.

Diagnosis:

The articulation between CD 9808 (distal metapodial) and CD 10427 and CD 10461 (proximal phalanx) and the severe abnormalities present on both fossils

suggest that the individual suffered either a multifocal or generalized pathology, most likely, a traumatic injury that altered the joint. Abnormal degenerative changes are common at sites of either unreduced or poorly reduced luxation. The thickness of the cartilage depends on the weight being borne by the joint. Thin or eroded cartilage can result from an injury in which a portion of the joint surface is no longer weight bearing (Emily Lane, personal communication, 2005). The pathogenesis of *Osteochondrosis dissecans* is generally thought to be multifactoral, including rapid growth, over-nutrition, mineral imbalance and trauma (Kahn and Line, 2005). The OCD compounded with the distorted shape and angle of the epiphysis of the metacarpal and the secondary arthritic changes suggest trauma as being the principal cause for the observed lesions. The two crests of bone on the palmar surface of the diaphysis represent an enthesopathy. An injury to the metacarpophalangeal joint may have created functional alterations to the animal's gait and changes in biomechanical stress, necessitating structural responses by the associated elements.

Classification: *Trauma* (with enthesopathy and arthropathy)

Comments: Articulation with CD9808

Life history interpretation: Pain and altered gait.

4.1.5. CD 7653 / CD 7532 (Figures 4.6, 4.7 and 4.8)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: cf. *Connochaetes*

Species: Indeterminate

Bovid size class: III

Skeletal element:

Element: Metacarpal

Preservation: Proximal epiphysis

Side: Left

Taphonomy:

Longitudinal fracture to proximal epiphysis and transverse (diagonal) fracture to proximal metaphysis

Abnormal morphology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion within affected bone: Proximal joint surface / proximal epiphysis / proximal metaphysis

Description: There is extensive periosteal bone formation on the dorsomedial surface of the proximal metaphysis extending proximally to, and overlapping, the epiphysis. The lesion begins, at its distal-most extent, as a large ovoid-shaped nodule of abnormal bone (10mm at its thickest) with a rough and rutted surface. As the lesion extends proximally towards the epiphysis it thins to 5mm. Dorsal to this lesion are two small circular lesions, one a nodule and the second a small cavity. Adjacent to these lesions is a sharp crest of lamellar bone running parallel to the long axis of the element for 10mm. The entire remaining epiphysis is covered with a layer of woven and lamellar bone. The woven bone tissue at the periphery of the lesion is porous and has a striated and latticed appearance. There are also signs of subchondral eburnation, seen as shallow parallel grooves and some periosteal reactive bone on the joint surface itself. The subchondral surface displays two small cavities. A longitudinal post-depositional fracture allows for the examination of the trabeculae and cortical structures. There appears to be a sclerosis of the trabeculae underlying the joint surface. The medial facets of the proximal articular surfaces of bovid metacarpals have a small central fovea of varying size for different species. The fovea in CD 7653 is deeper than those noted in the comparative sample for bovids of comparable size. Such a disparity may be attributable to intraspecies variation or aging; however, in the area surrounding the fovea in CD 7653 are clear areas of reactive bone. The epiphysis is also covered in an irregular layer

of raised bone and small marginal nodules extending proximally and tending to 'lip' towards the junction with the articular surface.

Diagnosis:

The eburnation, osteophytes, sclerosis of the trabeculae, and subchondral pitting are commonly noted as being concomitant with joint disease (Baker and Brothwell, 1980; Aufderheide and Rodriguez-Martin, 1998; Ortner, 2003). Extensive bone hypertrophy can also be symptomatic of some tumours as well as metabolic disturbances. The bone formation is unlikely a neoplasm because it is not a discrete lesion, typical of benign tumours or the large and irregular lesion typical of malignant tumours. Infection is unlikely as there are is no evidence of swelling of the bone and no visible cloacae. Trauma is another possibility, with secondary osteoarthritis and enthesopathy following either an injury specifically to the joint or to the surrounding soft tissues. The primary lesions are consistent with joint disease, and as per guidelines established regarding the diagnosis of joint disease (see Baker and Brothwell, 1980; Rogers and Waldron, 1995; Waldron, 2009) CD7653 meets the criteria for the more specific diagnosis of osteoarthritis.

Classification: *Joint disease* (with enthesopathy)

Life history interpretation: Pain and altered gait/function.

4.1.6. CD 036 (Figure 4.9)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Bovid size class: III

Skeletal element:

Element: Lumbar vertebra

Preservation: Laminae and spinous process

Side: N/A

Taphonomy:

Postmortem fracture at the laminae

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Cranial articular facets

Description: CD 036 exhibits nodules on the dorsal margin of the anterior articular facets. The abnormality is present bilaterally and extensive, with several millimeters of abnormal lamellar bone extending over the articulation. Smaller nodules project from the anterior margin of the anterior articular surfaces.

Diagnosis:

The hypertrophic changes, including marginal bone development and osteophytes, on CD 036, appear to indicate a joint disease. Osteoarthritis of the diarthrodial joints of the spine is similar to joint changes associated with other synovial joints of the appendicular skeleton (Ortner, 2003). Osteophyte development can be severe and substantial around the intervertebral articulations. Stress, age and trauma may be responsible for spinal arthritis.

Classification: *Joint disease*

Life history interpretation: Possible joint pain.

4.2. Order: Artiodactyla

Family: *Suidae*

4.2.1. CD 159 (Figure 4.10)

Taxonomic classification:

Order: Artiodactyla

Family: Suidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Metacarpal

Preservation: Proximal 1/3

Side: Left

Taphonomy:

Postmortem fracture and carnivore damage at proximal metaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Proximal joint margin

Description: There is a small nodule extending from the dorsal surface of the proximal joint margin of CD 159. Three millimeters distal to the first lesion is another, slightly larger, rounded nodule. Both appear lamellar although the surfaces of both abnormalities are roughened and irregular. Both lesions have sharply defined borders with the original cortex.

Diagnosis:

The bone nodules are isolated to the joint margin. The presence of abnormal bone outgrowths at the joint periphery (osteophytes) is indicative of joint disease. Marginal osteophytes are commonly correlated with degenerative joint disease, and occur after a breakdown of the articular cartilage allows for irritation of the joint surfaces, stimulating an osteogenic response. The rest of the bone appears unaffected.

Classification: *Joint disease*

Life history interpretation: Possible pain and altered function.

4.2.2. CD 3992 (Figure 4.11)

Taxonomic classification:

Order: Artiodactyla

Family: Suidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Hindlimb 1st phalanx

Preservation: Complete

Side: Left

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Distal epiphysis

Description: CD 3992 displays a rounded sessile nodule, extending from the medial surface at the distal metaphysis. The lamellar projection is triangular in shape with a basal breadth of 4mm and a height from the medial surface of 2mm. The projection is smooth in surface texture and appearance and is rounded at its apex. The borders of the lesion are smooth and continuous with the surrounding cortex, suggesting a condition that was quiescent at the time of death. There are no signs of other bone changes, with the proximal joint surface, distal joint surface, and bone shaft all appearing normal.

Diagnosis:

The lesion seen on CD 3992 is a sessile exostosis; a benign outgrowth of bone arising from the bone surface. One possibility is that this abnormality may be the result of a benign tumour beginning during the growing period of the skeleton. The neoplastic condition, osteochondroma (*osteocartilaginous*

exostosis) begins close to the growth cartilage. The cartilage undergoes endochondral ossification activating the periosteum and creating exostoses which enlarge, but stop when the growth plate terminates its growth (Ortner, 2003). The lesions grow perpendicular to the plane of the diaphysis and frequently contain a rounded cap (Aufderheide and Rodriguez-Martin, 1998).

Although osteochondroma can be considered a true neoplasm (Harper *et al.*, 1998) there are several other osteochondroma-like mimics which can range in aetiology (Richardson, 2005). It has been suggested that solitary osteochondromas, as seen in CD 3992, may likely be caused by trauma to the periphery of the growth plate (ibid).

Classification: *Trauma*

Life history interpretation: Asymptomatic.

4.2.3. CD 7874 (Figure 4.12)

Taxonomic classification:

Order: Artiodactyla

Family: Suidae

Genus: indeterminate

Species: indeterminate

Skeletal element:

Element: 1st phalanx

Preservation: Complete

Side: Right

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Epiphysis

Description: There is a small rounded lamellar nodule of bone extending laterally from the lateral margin of the distal metaphysis of the phalanx. The projection begins at the metaphysis and ends proximal to the distal joint surface. On the medial surface at the same location there is a circumscribed area of periosteal new bone. The lesion appears as a small raised projection of lamellar tissue. A third lesion, a crest of abnormal bone, appears associated with the enthesis on the plantarolateral aspect of the phalanx.

Diagnosis:

The small exostosis is not associated with the joint periphery and therefore, not an osteophyte. The lesion is rounded and not spiculated like a typical enthesophyte; however, the crest of bone does occur at the enthesis and represents an enthesopathy.

The two Suidae first phalanges (CD 7874, CD 3992) display abnormalities associated with the distal articular structures. Both bones belong to adult individuals with complete epiphyseal fusion. Aside from the abnormal exostoses at the growth plate there are few other visible abnormalities. Exostoses may develop as the result of a developmental dysplasia or neoplasia; however, solitary exostoses can result from localized trauma to the bone itself. CD 7874 and CD 3992 display rounded sessile projections arising from the distal metaphyses extending perpendicular to the long axis of the elements. In the case of CD 7874, the enthesopathy is possibly a conformation response to the principal abnormality (possibly the large lateral exostosis), resulting from mechanical stress caused by either an altered gait or function.

Classification: *Trauma* (with enthesopathy)

Life history interpretation: Possible altered function.

4.3. Order: Carnivora

Family: Felidae

4.3.1. CD 10765 (Figures 4.13 and 4.14)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *leo*

Felidae relative sizing: Large (lion sized)

Skeletal element:

Element: Radius

Preservation: Diaphysis 2/3

Side: Left

Taphonomy:

Postmortem fracture at proximal and distal metaphyses / carnivore damage to the lateral surface and at the proximal break

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Diaphysis

Description: A sessile nodule of lamellar bone extends from the medial aspect of the radius. The lesion extends inferomedially for 5mm. The projection is spiculated; being roughly triangular in shape and widest at its base. The bone is continuous with the original cortex. The nodule is small with a basal breadth of 5mm, extending to a height of 2.3mm from the bone surface.

Diagnosis:

The small spur appears to be associated with a site of enthesial attachment. The lesion is an enthesophyte and represents an osteophytic form of enthesopathy. Acute trauma can result in ossification at sites for ligamentous or tendinous attachment to bone. The condition, *myositis ossificans traumatica,* results from a single traumatic event and bone growth is usually extensive

(Ortner, 2003). In CD 10765, however, the lesion is a small spur with no other signs of physical injury. Enthesopathies are thought to have a multifactoral aetiology with age being the prevailing correlate. There is evidence that enthesophytes can reflect *in vivo* muscle use and can be stimulated by chronic trauma or mechanical stress.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

4.3.2. CD 9909 (Figure 4.15)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *leo*

Felidae relative sizing: Large (lion-sized)

Skeletal element:

Element: 3rd metacarpal

Preservation: Proximal 1/2

Side: Right

Taphonomy:

Postmortem fracture at distal metaphysis

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion within affected bone: Diaphysis

Description: There is a 'U' shaped mass of smooth lamellar bone located on the palmaromedial aspect of the metacarpal, distal to the proximal metaphysis. The lesion is thickest at its proximal-most point and trails off distally, becoming both thinner and less raised as it encircles a relatively large lytic depression. The cavity is irregular and rutted in surface texture and appearance although it appears covered over with a thin layer of cortical bone.

Diagnosis:

The lesion involves periosteal bone formation and associated osteolysis. The rest of the bone has a normal gross appearance. The solitary nature of the lesion suggests a response to trauma (Emily Lane, personal communication, 2005). In the case of a penetrating injury to the posterior surface of the foot there would be a proliferative response with callus formation around the injury. The lesion might represent an enthesopathy; however, it appears larger and more irregular than an enthesophyte. A possible ligament avulsion could also cause proliferation of bone at the area of injury. In the case of either a ligament avulsion or penetrating injury, the lesion represents a trauma and appears longstanding and quiescent at the time of death.

Classification: *Trauma*

Life history interpretation: Pain and altered gait.

4.3.3. CD 20657 (Figure 4.16)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: cf. *Dinofelis*

Species: Indeterminate

Felidae relative sizing: Medium (leopard-sized)

Skeletal element:

Element: 5th metatarsal

Preservation: Complete

Side: Right

Taphonomy:

Postmortem transverse break at mid-diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Diaphysis

Description: There is a smooth and rounded abnormal deposition of lamellar bone on the plantaromedial aspect of the diaphysis. The lesion extends from distal of the proximal articulation to distal of the mid-shaft.

Diagnosis:

The fossil displays, what is most likely, inflammation of the periosteum. A number of conditions can result in periostitis. Periosteal inflammation can be caused by trauma to the bone itself, resulting in a haematoma which can be ossified in time, or by inflammation of the adjacent soft tissue resulting in proliferation of bone at the area of injury. Baker and Brothwell (1980) show a metacarpal of a rabbit displaying an injury similar in gross appearance to CD 20657, attributing it to an inflammatory response. Periosteal inflammation can also occur in response to a variety of infectious conditions.

The rounded circumscribed area of new bone appears most similar to an ossified haematoma following localized trauma. The fact that the abnormal bone formation appears isolated to the plantaromedial surface might suggest a remodelled chip fracture and associated repair (Emily Lane, personal communication, 2005).

Classification: *Trauma*

Life history interpretation: Pain and altered gait.

4.3.4. CD 6057 (Figure 4.17)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: Indeterminate

Species: Indeterminate

Felidae relative sizing: Medium (leopard-sized)

Skeletal element:

Element: Axis vertebra

Preservation: Odontoid process and cranial articular facets

Side: N/A

Taphonomy:

Postmortem fracture separating cranial surfaces from the vertebral body

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Cranial joint margins

Description: There are two bilateral nodules of compact bone on the ventral surface, lateral to the odontoid process. Almost uniform marginal hypertrophic raised bone encircles both cranial articular surfaces. There is an area of abnormal raised bone on the superior aspect of the odontoid process; as well as a line of raised bone extending from the tip of the odontoid process to its margin with the cranial articular surfaces (present bilaterally but more pronounced on the right side). The lesion is primarily lamellar with areas of woven bone at the periphery and sharply defined borders.

Diagnosis:

CD 6057 shows signs of a periarticular hypertrophic condition. The bone growth is localized to the joint periphery. There are no other obvious lesions. Marginal hypertrophic growths are commonly noted as being one of the manifestations of an arthropathy. Irritation from vertebral contact causes a proliferative response at the joint margins and the development of osteophytes (osteophytosis). The cartilaginous areas would be left unaffected, allowing marginal changes but leaving the interior joint surfaces unchanged (Emily Lane, personal communication, 2005). Vertebral arthritis is present or common in wild animals (Greer *et al*., 1977; Rothschild *et al*., 2001) and man, and appears to be correlated with both longevity and body mass (Greer *et al*., 1977). Degenerative spinal disease is noted as being a frequent and "important problem" in large non-domestic felids (Kolmstetter *et al.,* 2000). Apart from the marginal osteophytes there are no signs of eburnation, pitting, or sclerosis of the underlying bone.

Classification: *Joint disease*

Life history interpretation: Pain and altered function. Clinically, the pain may affect mobility and decrease activity (Kolmstetter *et al.,* 2000).

4.3.5. CD 3843 (Figure 4.18)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: cf. *Dinofelis*

Species: Indeterminate

Felidae relative sizing: Medium (leopard-sized)

Skeletal element:

Element: Patella

Preservation: Complete

Side: Left

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Lateral border, inferior to the articular surface

Description: CD 3843 is a complete left patella of a medium-sized felid, similar in morphology to *Panthera leo* but more similar in size to *Panthera pardus*. In posterior view there is a large triangular projection extending away from the lateral surface of the apex, just below the patellar facet for articulation with the lateral condyle of the femur. The projection is triangular in shape with a basal breadth of 12mm and a height of 11mm from the normal bone surface. It is rough and irregular in both texture and appearance. The lesion itself appears to be lamellar and the borders are continuous with the surrounding cortex suggesting that the condition was longstanding and quiescent at the time of death.

Diagnosis:

CD 3843 shows an enthesial reaction. The stifle joint (knee) is stabilized by numerous ligaments maintaining the alignment of the femur and tibia, as well as providing the joint with both strength and integrity. The knee is, however, particularly susceptible to ligament avulsion injuries caused by running, swift turning, jumping, or any activity resulting in forward compressive or rotational forces on the tibia (Kahn and Line, 2005). There are no signs of arthritic changes and the lesion is solitary and isolated to one border of the patella. This might suggest that the individual suffered acute physical injury, perhaps a partial avulsion of the patellar ligament, which might have caused bleeding into the soft tissues, resulting in the proliferation of bone at the area of injury, i.e. *myositis ossificans traumatica*.

Classification: *Trauma* (with enthesopathy)

Life history interpretation: Pain and altered gait/function. Traumatic dislocation of the patella is often associated with a sprain or sprain-avulsion fracture (Farrow, 2003) which would result in swelling, tenderness and a degree of lameness.

4.3.6. CD 7971 (Figure 4.19)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *pardus*

Felidae relative sizing: Medium (leopard-sized)

Skeletal element:

Element: Two consecutive lumbar vertebrae

Preservation: (cranial) body and lamina / (dorsal) body fragment

Side: N/A

Taphonomy:

Postmortem fracture / carnivore damage, with total obliteration of the spinous and transverse processes of the posterior vertebra

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Vertebral bodies

Description: The fossil, CD 7971, is actually composed of two lumbar vertebrae. The vertebrae have ankylosed, essentially forming one bony unit. The fusion occurs between the two vertebral bodies; visible on the ventral aspect as a sheath of striated hypertrophic bone overlaying both vertebral bodies and bridging the intervertebral space. On the dorsal aspect there appears little evidence that the specimen was ever two separate elements, with the vertebral foramen appearing undisturbed, and the abnormal bone having a smooth appearance and texture. The soft tissue constituents of the intervertebral space appear to have been entirely ossified. The striated bone on the ventral aspect maintains the appearance and position of the ventral ligament. The anterior vertebra at its anterior articulation appears completely normal in comparison with modern leopard (*Panthera pardus*) lumbar vertebrae. It is only at the mid-point of the body that the abnormal bone deposition begins. The lesion appears to be entirely composed of lamellar bone. The cranial and caudal surfaces of the vertebral bodies are normal with no signs of pathology. Also, another specimen, CD 6079, appears to have belonged to the same individual based on spatial proximity and size, and articulates anteriorly with CD 7971. This suggests that the condition, if not isolated, did not continue immediately up or down the spinal column.

Diagnosis:

CD 7971 displays evidence of a spinal arthropathy. The resulting lesions are consistent with a condition that had as a factor enthesopathy, and are visible here as the ossification of the ventral ligament and *annulus fibrosis*. The regularity of the lesions implies an inflammatory response rather than infectious agent. The localization of the lesions and the normal morphology of

the ventral surfaces of CD 7971 suggest perhaps a conformation response rather than an age-related arthropathy.

Congenital fusion (synostosis) of vertebral elements is another possible cause of the abnormal morphology. Diffuse idiopathic skeletal hyperostosis (DISH) might also be considered. DISH is a joint condition in which there is a tendency to create excessive bone at joint margins and entheses (Ortner, 2003), particularly under the anterior longitudinal ligament in modern humans (Roberts and Manchester, 2005). Other spondyloarthropathies, in particular *spondylosis deformans*, results in production of syndesmophytes and fusion of vertebrae (Rothschild, 2005). *Spondylosis deformans* is a non-inflammatory degenerative disease that correlates with senescence (Kahn and Line, 2005). The condition occurs as a result of the gradual breakdown of the outer fibers of the annulus fibrosis and stretching of the longitudinal ligament (ibid).

Classification: *Joint disease* (with enthesopathy)

Life history interpretation: The ankylosis of spinal elements is common in cats, but although the lesions appear dramatic in dry bone they can in fact be clinically insignificant in modern dogs and cats (Couteur and Grandy, 2000; Kahn and Line, 2005). This is especially true in the lumbar vertebrae where the original range of motion is anatomically limited and further reduction in the range of motion may not cause a dramatic change in the life of the animal affected.

4.4. Order: Carnivora

Family: Hyaenidae

4.4.1. CD 6681 (Figure 4.20)

Taxonomic classification:

Order: Carnivora

Family: Hyaenidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Pelvis

Preservation: Acetabulum / partial ischium / partial ilium, missing pubis

Side: Left

Taphonomy:

Postmortem fracture at ischium and ilium

Pathology:

Type: Abnormal bone loss

Distribution of lesion within affected bone: Acetabulum / lunate surface

Description: There is a relatively large lenticular cavity on the lunate surface of the acetabulum at the external margin. The cavity is oval in shape and measures 10mm by 7mm. The inside of the cavity has an irregular roughened texture and rounded borders. An area of isolated periosteal thickening occurs on the periphery of the acetabulum.

Diagnosis:

While pitting within the acetabulum may be associated with arthritic conditions affecting the hip, the lenticular lesion seen in CD 6681 may more likely represent an osteochondrosis or OCD. The pathogenesis of *Osteochondrosis dissecans* is generally thought to be multifactoral, including factors such as rapid growth, over-nutrition, mineral imbalance and trauma (Kahn and Line, 2005). In a wild animal it is unlikely that over-nutrition or rapid growth would

be contributing factors. The most likely cause of the OCD is possibly trauma during the growing period of the skeleton.

Classification: *Trauma*

Life history interpretation: Possible pain and altered gait. Animals with OCD usually present with synovial effusion and varying degrees of lameness (Morgan *et al.*, 2000; Kahn and Line, 2005). Johnson *et al.* (2008), in their description of *osteochondrosis dissecans* in a juvenile roan antelope, noted swelling and a limited range of motion to the stifle joint.

4.4.2. CD 13301 (Figure 4.21 and 4.22)

Taxonomic classification:

Order: Carnivora

Family: Hyaenidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: 2nd phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Proximal joint surface

Description: CD 13301 exhibits hypertrophic bone extending away from the periphery of the proximal interphalangeal joint surface. The nodules flare from the medial, dorsal, and volar borders, creating widened articular facets. The shaft and distal articular surface of the bone appear normal with no evidence of abnormal remodelling.

Diagnosis:

The osteophytes are isolated to the proximal interphalangeal joint periphery. The entheses appear normal. The lesion is indicative of a form of arthropathy. There are several types of arthropathy that can affect animals, including DJD, traumatic arthritis and septic arthritis. During the course of the pilot study conducted for this research, similar lesions were seen on the phalanges of modern faunal comparatives, concomitant with other signs of polyarticular osteoarthritis. Although there is no evidence of grooving or eburnation, the lesions seen on CD 13301 are interpreted as the result of an arthropathy, possibly osteoarthritis.

Classification: *Joint disease*

Life history interpretation: Possible pain.

4.4.3. CD 9985 (Figure 4.23)

Taxonomic classification:

Order: Carnivora

Family: Hyaenidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Terminal phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal formation / abnormal shape

Distribution of lesion within affected bone: Articular surface

Description: CD 9985 is a complete terminal phalanx. The abnormal characteristics appear on the interphalangeal articular surface. There is

extensive bone remodelling on and around the joint surface. Abnormal lamellar bone depositions flare from the lateral, medial, and dorsal margins, resulting in a mediolateral widening of the joint surface. The result of both lytic and blastic processes is the complete obliteration of the normal joint appearance. The rest of the phalanx, however, appears normal.

Diagnosis:

The pathology represents a joint disease, whereby the entire morphology of the joint surface has changed. The severity of the abnormality might suggest trauma rather than chronic mechanical stress. A dislocation (luxation) is the complete and persistent displacement of the articular surfaces of bones within a joint. It occurs with either partial or complete capsular and/or ligament rupture (Aufderheide and Rodriguez-Martin, 1998). Its persistence produces long term changes and accelerated degenerative joint disease. The skeletal alterations depend on survival and are secondary to the bone displacement (ibid). Arthritic degeneration and secondary bone formation in response to shifts in biomechanical loading following such an injury create gross changes similar to those seen in CD 9985. Although the changes to the terminal phalanx are possibly secondary to trauma, the broad classification of 'joint disease' remains the most fitting.

Classification: *Joint disease*

Life history interpretation: Pain and altered gait.

4.5. Order: Primates

Family: Cercopithecidae

4.5.1. CD 7363 (Figure 4.24)

Taxonomic classification:

Order: Primates

Family: Cercopithecidae

Genus: *Papio*

Species: Indeterminate

Skeletal element:

Element: Lumbar vertebra

Preservation: ½ body / pedicle / one transverse process / spinous process / intervertebral articulations on one side / costal facets

Side: N/A

Taphonomy:

Postmortem break through the vertebral body

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Centrum / spinous process

Description: A portion of CD 7363 has been sheared off in such a way that only one costal facet remains. There is marginal osteophyte development circling the costal facet and extending away from it. There is also osteophyte development extending from the costal facet and continuing around the remainder of the vertebral body. The cranial surface of the spinous process is covered in a layer of woven periosteal bone, oriented ventrodorsally, linear in appearance. The caudal surface of the spinous process is covered in the same periosteal linear bone growth.

Diagnosis:

The marginal hypertrophic bone around the vertebral body and costal facets is similar to that seen during the pilot study and in the palaeopathology literature (e.g. Aufderheide and Rodriguez-Martin, 1998; Roberts and Manchester,

2005). Reactive bone formation around the amphiarthrodial joints and associated entheses is commonly correlated with spinal arthropathy (Ortner, 2003). The bone proliferation at the area of attachment for the interspinous ligament on both the cranial and caudal surfaces of the spinous process suggests an arthritic condition with enthesopathy as a contributing factor.

Classification: *Joint disease* (with enthesopathy)

Life history interpretation: Possible pain and altered function.

4.5.2. CD 7267 (Figure 4.25)

Taxonomic classification:

Order: Primates

Family: Cercopithecidae

Genus: cf. *Papio*

Species: Indeterminate

Skeletal element:

Element: 1st ray metacarpal

Preservation: Distal 1/3

Side: Indeterminate

Taphonomy:

Postmortem fracture at proximal metaphysis and carnivore damage

Pathology:

Type: Abnormal bone formation

Distribution of lesion within affected bone: Distal joint surface

Description: The abnormal bone appears on the distal joint surface of the metacarpal. The three condyles on the bone's palmar surface have small roughly circular nodules of bone (no larger than 2mm in diameter), abnormal in comparison with a healthy analogue. The lesions are irregularly shaped lamellar projections occurring at each condyle's proximal border. The osteophytes have a roughened and irregular texture and appearance and protrude outwardly from the palmar surface.

Diagnosis:

Abnormal bone projections extending from the joint surface are commonly noted as corresponding with osteoarthritis. In certain instances, articular cartilage degenerates to such an extent that cells activate to form new cartilage and bone at the joint surfaces. This happens, in particular, at the periphery of the joint surface, producing marginal osteophytes. The osteophytes seen on CD 7267 correspond closely to documented cases of osteophyte development in osteoarthritis of modern human metacarpals examined during the course of the pilot study.

Classification: *Joint disease*

Life history interpretation: Pain and altered function.

4.5.3. CD 3332 (Figure 4.26)

Taxonomic classification:

Order: Primates

Family: Cercopithecidae

Genus: *Papio*

Species: Indeterminate

Skeletal element:

Element: 1st ray metatarsal

Preservation: Complete

Side: Left

Taphonomy:

Minimal

Pathology:

Type: Deposition / abnormal shape

Distribution of lesion within affected bone: Diaphysis

Description: Just proximal to the metatarsophalangeal joint surface, on the metatarsal's anterior aspect, is a bump of abnormally remodelled bone. The bump is 4mm long and runs the width of the diaphysis, 6mm. The periosteal

lesion has a latticed appearance and is composed entirely of woven bone. The margins are sharply defined which indicates that the condition was unhealed at the time of death.

Diagnosis:

The bump of bone is a circumscribed area of inflammation. This could be caused by a localized trauma and breach of the periosteum resulting in the abnormal bone development. The lesion could also result from inflammation of the adjacent soft-tissues (e.g. skin ulcer). There are no cloacae or involucrum to indicate chronic infection. The fact that the lesion is composed of woven bone indicates that the insult was suffered shortly before death, in a time frame long enough to allow for an osteogenic response but not long enough for the bone to heal. A severe infection may have resulted in mortality before more dramatic bone changes, and the small periosteal reaction may be an indicator of a more insidious condition than the lesion would suggest. The lesion is, however, a small and circumscribed area of periostitis on a subcutaneous distal-limb element, and for that reason, is classified here as trauma.

Classification: *Trauma*

Life history interpretation: Initial pain and possibly altered function.

4.6. Order: Primates

Family: Hominidae

4.6.1. CD 5288 (Figures 4.27 and 4.28)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Lumbar vertebra

Preservation: Vertebral body and base of laminae

Side: N/A

Taphonomy:

Postmortem fracture at both pedicles

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion within affected bone: Vertebral body

Description: There is marginal osteophyte development on both the superior and inferior margins of the vertebral body extending away from the body and towards the intervertebral space. On the superior surface the osteophytes extend approximately 1-2mm from the original body, beginning adjacent to one pedicle, running the length of the anterior surface, and ending at the opposite pedicle. Similar osteophyte development is present on the inferior margin of the vertebral body, also running the length of the anterior surface. There is also anterior erosion of both joint surfaces.

Both the superior and inferior endplates are eroded and are marked by a porous appearance. The vertebral body is marked by four large cyst-like erosions (2mm in diameter with a depth of approximately 2mm) on the inferior surface and 3 similar cyst-like erosions on the superior surface. There is a raised

periosteal layer of abnormal bone on the anterior surface of the body running supero-inferiorly at the approximate location of attachment for the anterior longitudinal ligament.

Diagnosis:

The lesions appear similar to osteoarthritic lesions encountered during the pilot study. The changes represent both destructive and hypertrophic processes primarily associated with the amphiarthrodial joints. The fact that both the superior and inferior joint surfaces are affected suggests a polyarticular form of vertebral arthritis with at least several vertebrae involved. Although the pathogenesis is not entirely understood, vertebral osteoarthritis is most likely the result of degenerative or traumatic changes to the intervertebral disk (Ortner, 2003). The fossil also displays erosive areas on the superior and inferior surfaces of the vertebral body, a condition that, if incurred *in vivo*, may be pathognomic of herniated cartilage (Roberts and Manchester, 2005). The erosion may also simply be the result of extensive postmortem erosion. The endplates are eroded to such an extent in CD 5288 that there is difficulty in determining whether the erosions occurred antemortem or postmortem. However, in a case whereby a disk herniates through the end-plate, it results in similar cyst-like lesions (Ortner, 2003) and the possibility should be considered of the erosions representing Schmorl's nodes.

Classification: *Joint disease (spondylosis)*

Life history interpretation: Pain and possibly altered function.

4.6.2. CD 5773 (Figures 4.29 and 4.30)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Thoracic vertebra (T2)

Preservation: Fragment of the vertebral body

Side: N/A

Taphonomy:

Postmortem fracture at both pedicles

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion within affected bone: Costal facet

Description: There is osteophytic development around the right costal facet. The osteophytes extend primarily from the anterior margin, although the entire facet exhibits arthritic changes. The surface of the facet displays several small osteolytic foci and a small nodule of hypertrophic bone. The inferior surface of the vertebral body is preserved and exhibits approximately 2mm of marginal lipping associated with the right posterior surface, adjacent to the right pedicle. The area of lipping displays a small lytic focus measuring 2mm by 1mm. A second localized area of osteophyte development occurs on the margin of the superior surface of the vertebral body 6mm anterior of the costal facet. This localized deposition also shows sign of associated lytic activity. The vertebra is noticeably anteriorly wedged. There appears on the left anterior margin of the inferior aspect of the vertebral body a posteromedial pinching giving the centrum an asymmetrical appearance when viewed anteriorly. Most of the cranial surface of the vertebral body and the majority of the left side of the element are missing, making it difficult to determine the extent of the

abnormality, or whether the arthritic changes to the costal facet are present bilaterally.

Diagnosis:

The vertebra, CD 5773, shows some degenerative changes. Spondylosis is seen, in the form of osteophyte development around the vertebral body. Arthritic changes are apparent on the right costal facet. Osteoarthritis can be responsible for bone proliferation around the costovertebral joints. Osteoarthritic changes can result from both subchondral hypertrophic and destructive processes (Ortner, 2003). The sclerotic area on the costal facet may indicate calcium pyrophosphate deposition disease, CPDD (Bruce Rothschild, personal communication, 2005). CPDD is an arthropathy caused by the deposition of calcium pyrophosphate crystals in and around joints.

The anterior pinching of the centrum appears to have occurred perimortem, with no obvious signs of bone repair at the area of deformity, and only minor osteophyte development associated with the anterior surface of the vertebral body. The abnormalities on CD 5773 appear to be the result of a degenerative joint disease, possibly osteoarthritis, resulting from either age or mechanical use.

Classification: *Joint disease (spondylosis)*

Life history interpretation: Pain and possibly altered function.

Bovidae

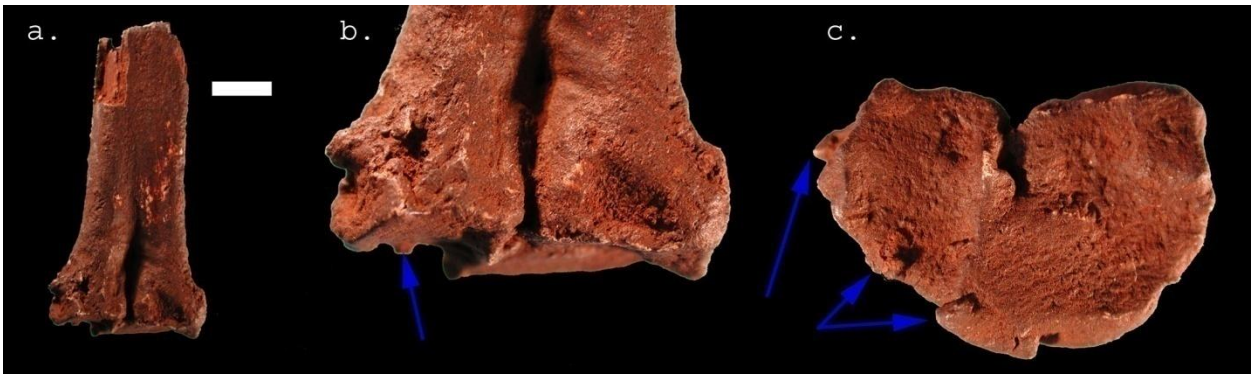


Figure 4.1. ***CD 16967*** **bovid metacarpal**: (a) posterior view showing marginal osteophytes (arrows); (b) detailed view of posterior surface; (c) detailed view of proximal joint surface .

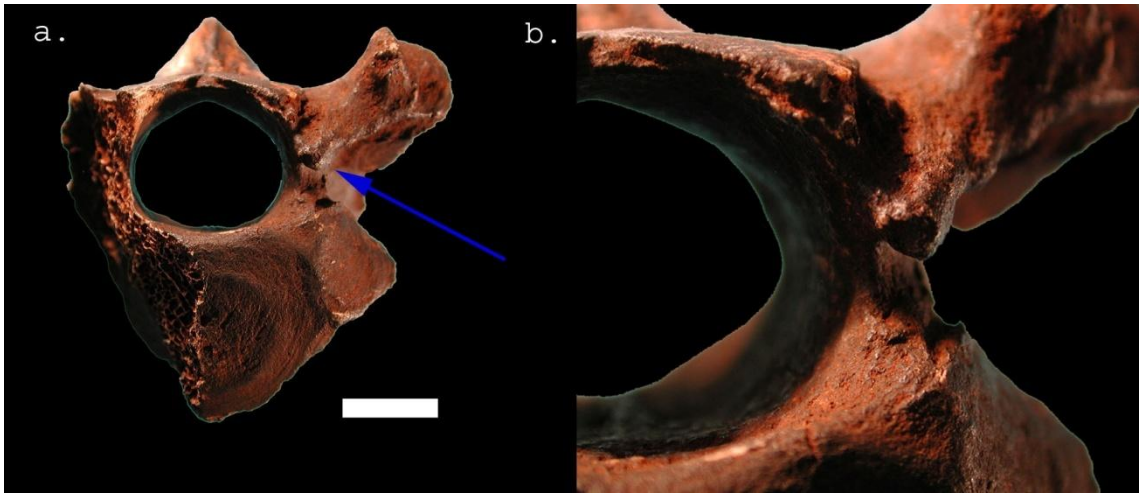


Figure 4.2. ***CD 7695*** **bovid thoracic**: (a) cranial view; (b) detailed view of the lesion, a roughly triangular sessile projection of abnormal bone extending from inferior of the left cranial articular facet (arrow).

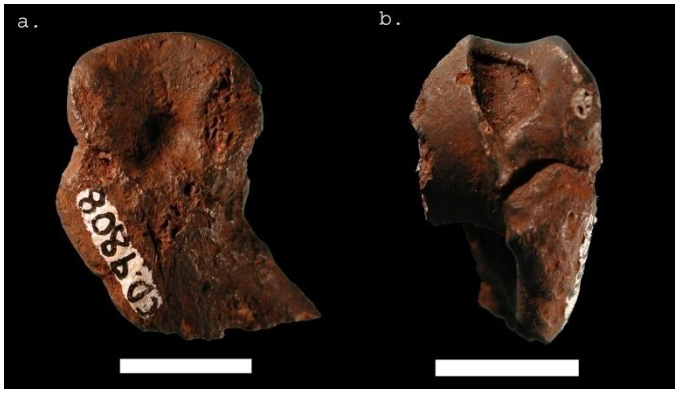


Figure 4.3. ***CD 9808*** **bovid metacarpal**: (a) lateral view; (b) view of distal joint surface showing the subchondral lesion.

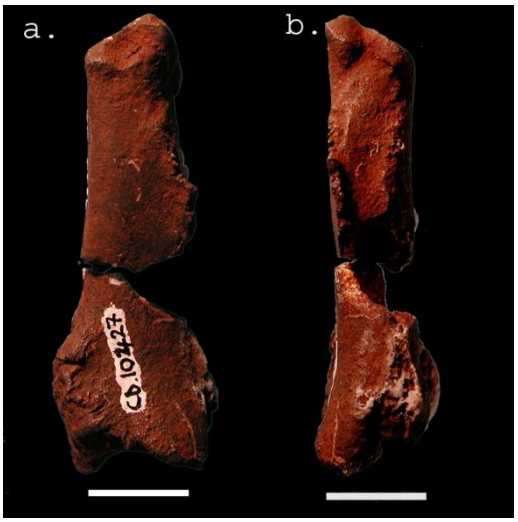


Figure 4.4. ***CD 10427*** **bovid 1st phalanx**: (a) lateral view; (b) volar view showing crests of bone extending from the palmaromedial and palmarolateral borders.

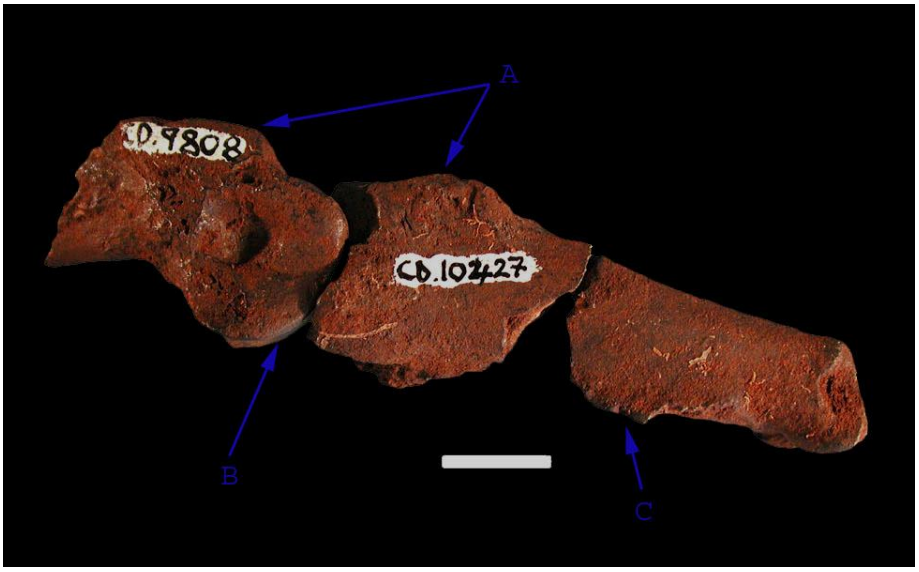


Figure 4.5. ***CD 9808 / CD 10427 / CD 10461*** **articulated bovid distal metacarpal and 1st phalanx**: notice the bulbous growths of irregular bone on the anterior surfaces of both the distal metacarpal and 1st phalanx **(A);** the squared distal condyle of the metacarpal **(B)**; the sharp crest of abnormal bone on the palmar surface of the phalanx **(C)**.

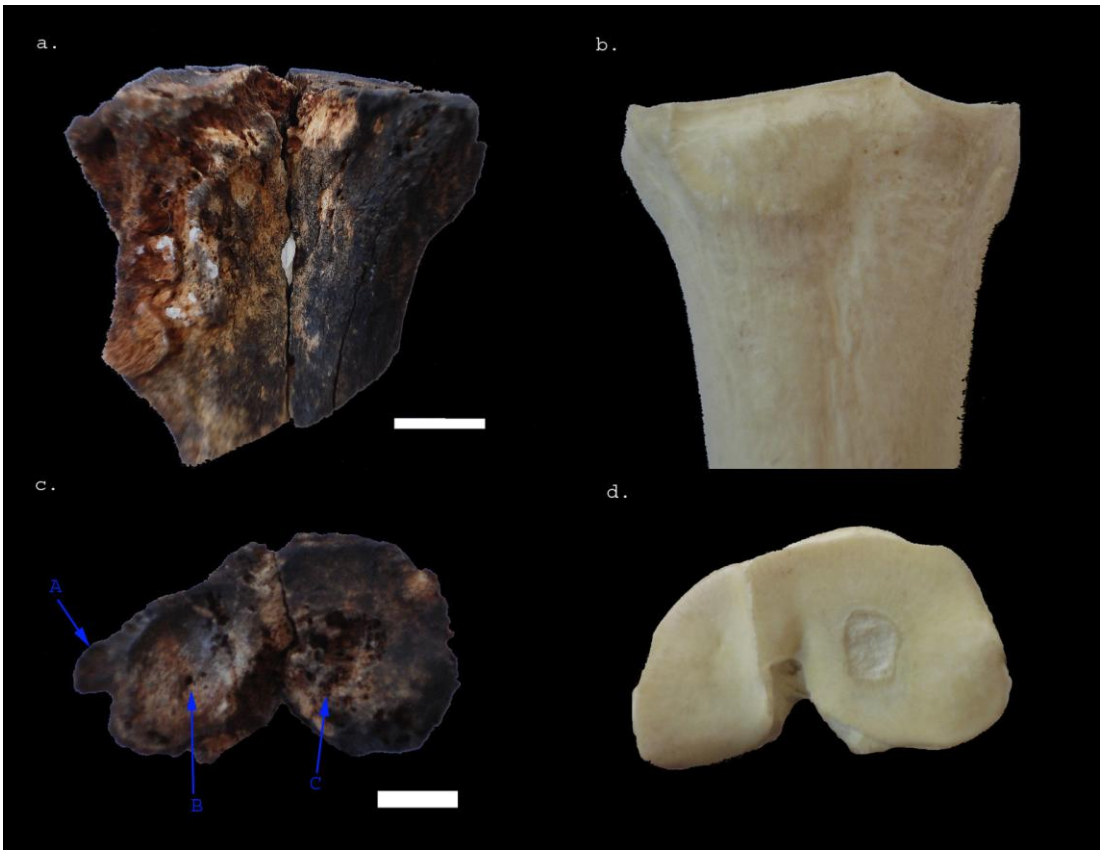


Figure 4.6. *CD 7653* / *CD 7532* proximal metacarpal: (a) dorsal view showing the extensive periosteal reactive bone on the metaphysis and epiphysis compared to a normal bovid analogue specimen (b); (c) proximal view showing the subchondral joint changes including eburnation (**B**) and pitting (**C**) and marginal osteophytes (**A**) compared to the normal bovid analogue specimen (d).

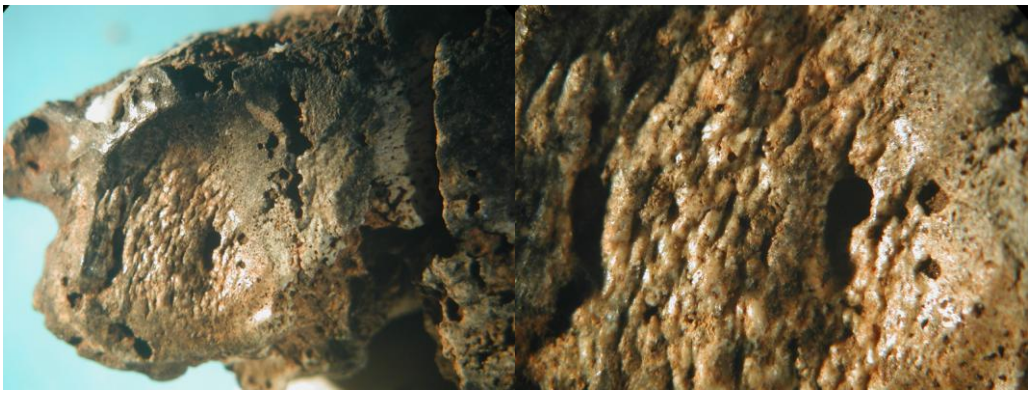


Figure 4.7. *CD 7653* / *CD 7532* proximal metacarpal: (left) detailed proximal view of the subchondral surface showing the lytic pitting and eburnation; (right) detailed view of the subchondral eburnation.

Figure 4.8. ***CD 7653*** **/** ***CD 7532*** **bovid proximal metacarpal:** detailed dorsal view showing the reactive bone on the proximal metaphysis.

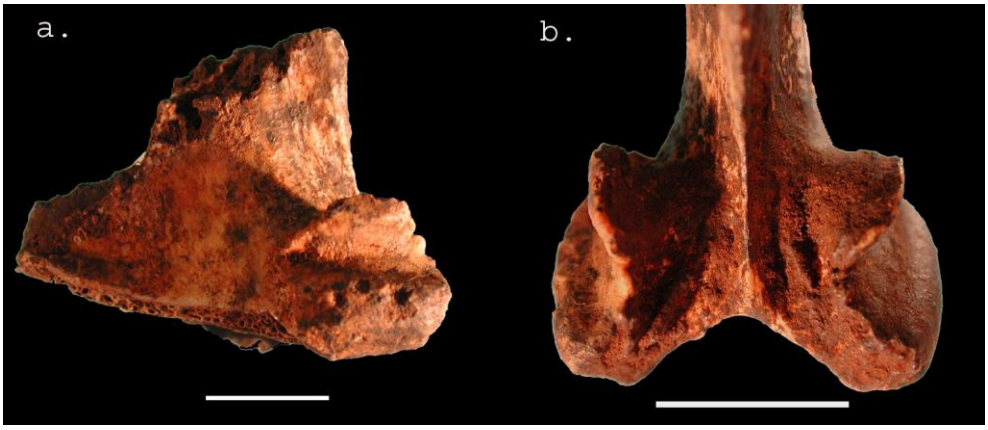


Figure 4.9. ***CD 036*** **bovid lumbar vertebra**: (a) lateral view; (b) anterior view; notice the extensive osteophyte development over the cranial articular facets and marginal osteophytes on the anterior margin of the articular surfaces.

Suidae

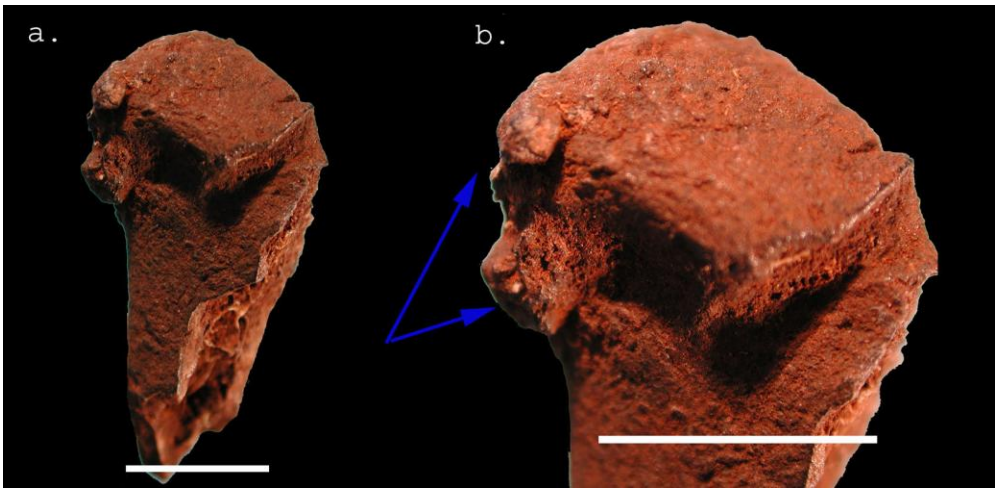


Figure 4.10. ***CD 159*** **suid metacarpal**: (a) medial view of the distal articular surface; (b) detailed medial view; notice the 2 irregular shaped projections of bone associated with the articular margin (arrows).

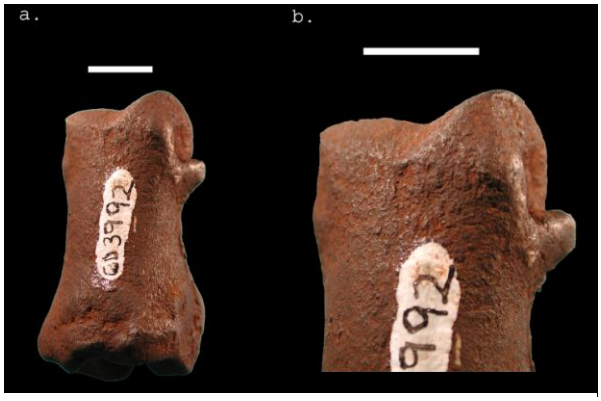


Figure 4.11. ***CD 3992*** **suid 1st phalanx**: (a) dorsal view; (b) detailed dorsal view; notice the large rounded sessile exostosis extending from the medial surface.

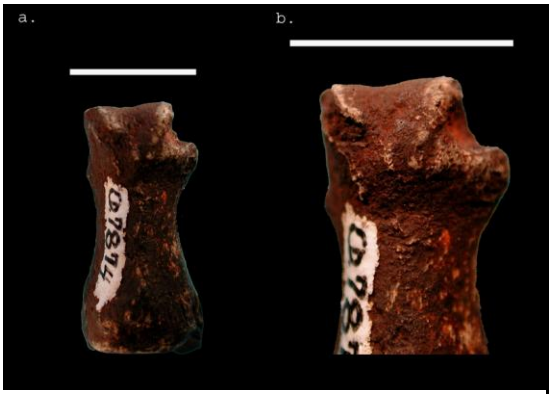


Figure 4.12. ***CD 7874*** **suid 1st phalanx**: (a) dorsal view; (b) detailed dorsal view; notice the sessile exostosis extending from the lateral surface.

Felidae

Figure 4.13. ***CD 10765*** **felid** ***(Panthera leo)*** **left radius**: Posterior view comparing CD 10765 to a normal modern lion analogue specimen (AZ 2443).

Figure 4**.14.** ***CD 10765*** **felid (*Panthera leo*) left radius**: (a) posterior view of the radius showing a small triangular exostosis; (b) detailed view of the lesion; (c) detailed view of the lesion with the bone rotated slightly medially. Notice the smooth appearance of the lamellar projection.

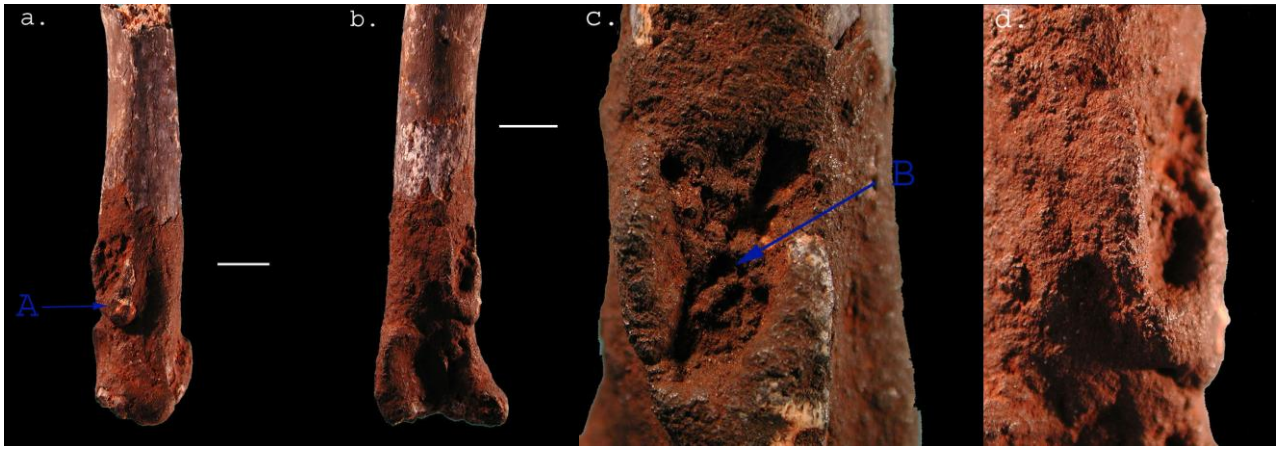


Figure 4**.15.** ***CD 9909 felid (Panthera leo)*** **right 3rd metacarpal**: (a) plantar view showing an area of extensive hypertrophic bone formation **(A)**; (b) lateral view; (c) detailed view of the lesion; note the destructive focus **(B)** surrounded by the area of reactive bone formation; (d) detailed lateral view of the lesion.

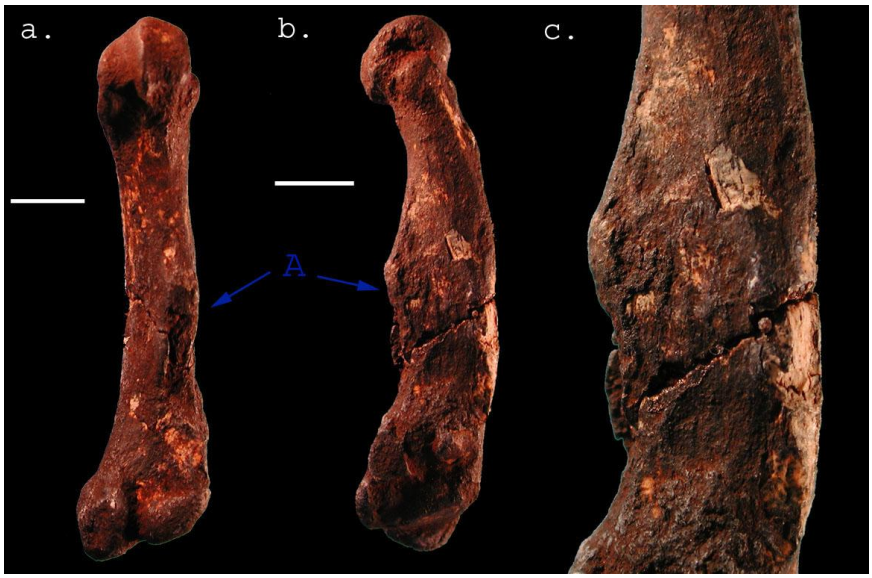


Figure 4**.16.** ***CD 20657*** **felid right 5th metatarsal**: (a) plantar view; (b) medial view; note the raised area of abnormal bone **(A)** on the plantar surface of the diaphysis; (c) detailed medial view of the lesion.

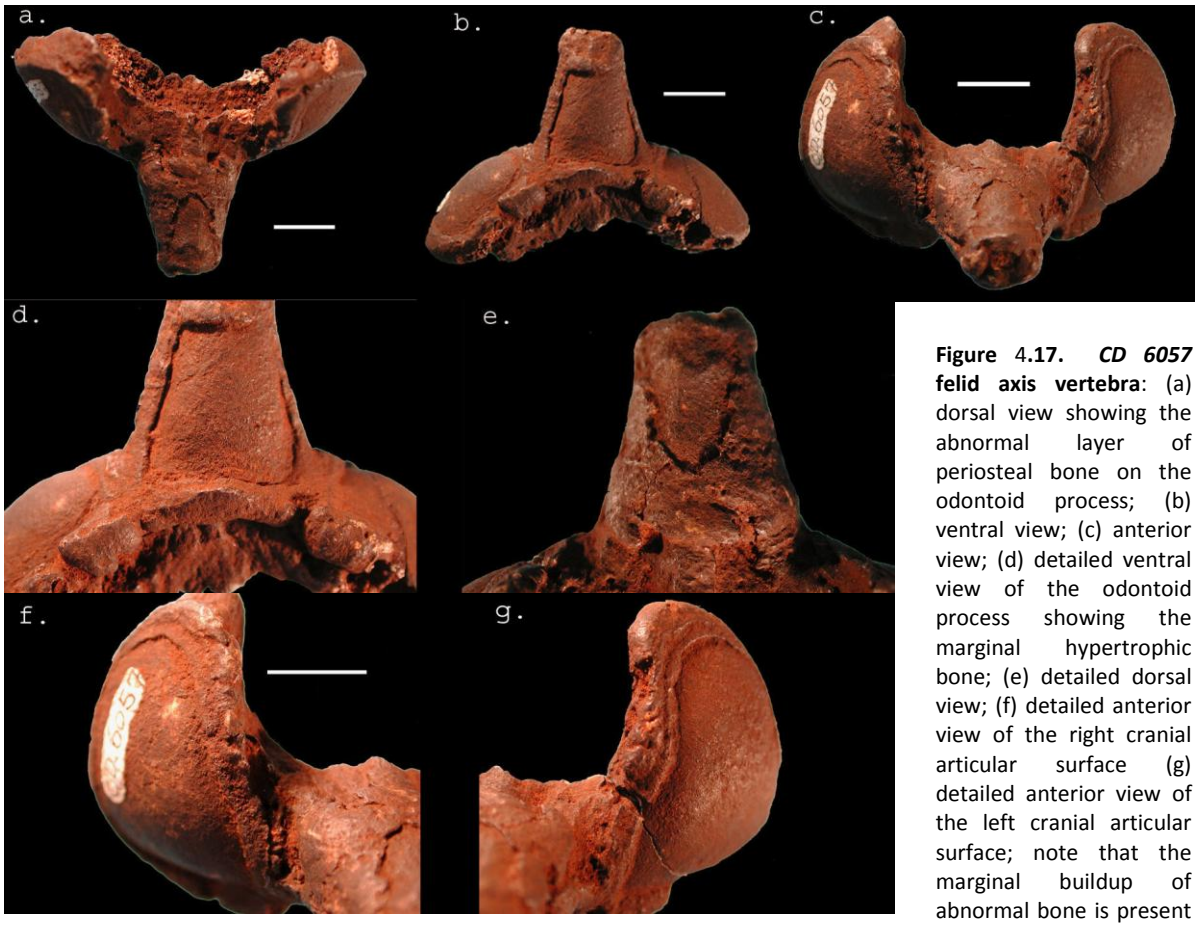


Figure 4**.17.** ***CD 6057*** **felid axis vertebra**: (a) dorsal view showing the abnormal layer of periosteal bone on the odontoid process; (b) ventral view; (c) anterior view; (d) detailed ventral view of the odontoid process showing the marginal hypertrophic bone; (e) detailed dorsal view; (f) detailed anterior view of the right cranial articular surface (g) detailed anterior view of the left cranial articular surface; note that the marginal buildup of abnormal bone is present bilaterally.

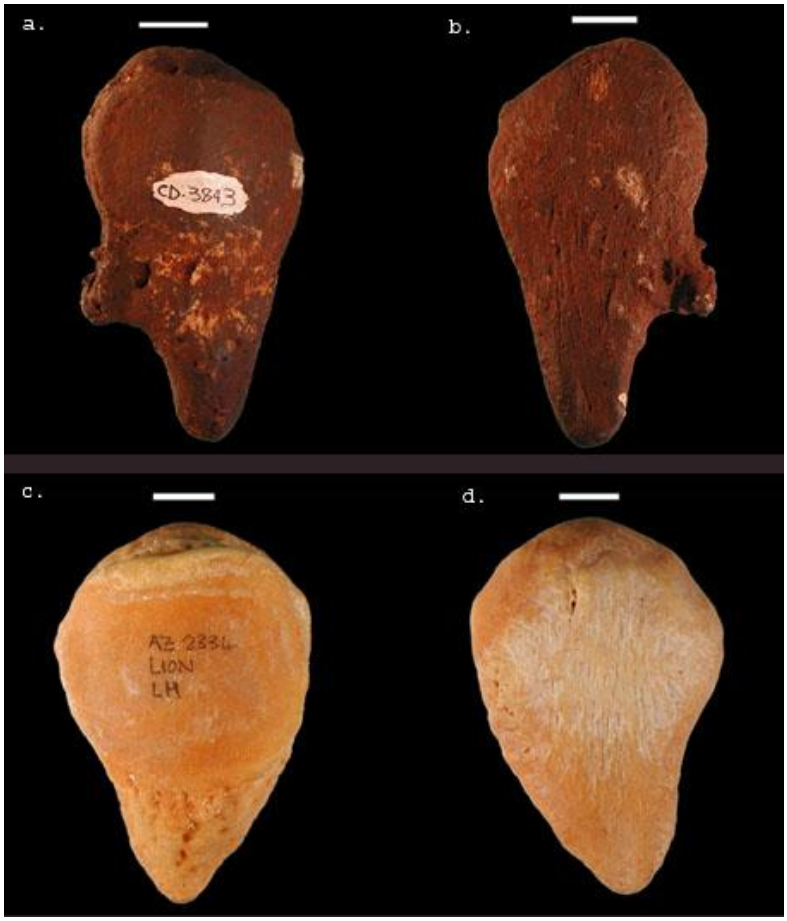


Figure 4**.18.** ***CD 3843*** **felid patella**: (a) posterior view showing a large triangular exostosis extending from the lateral border; (b) The same lesion shown in anterior view; note the roughened and irregular appearance of the projection; (c) posterior view and (d) anterior view of patella from a modern comparative lion (*Panthera leo*) – AZ 2334.

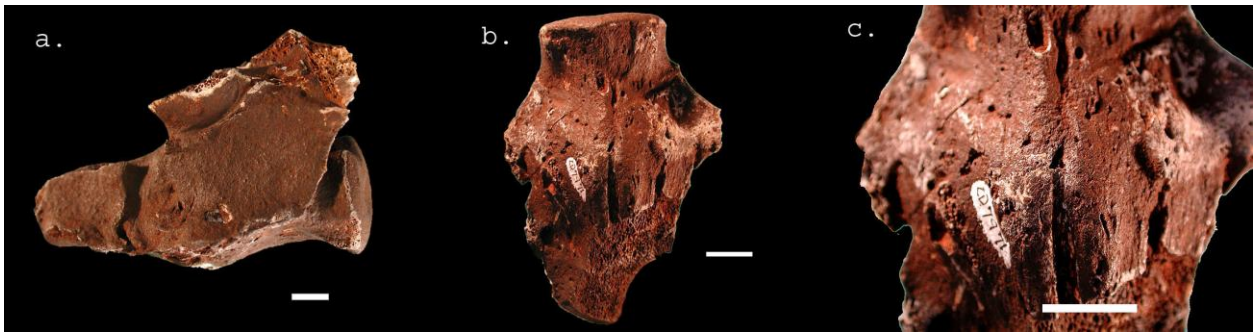


Figure 4**.19.** ***CD 7971*** **felid lumbar vertebrae**: (a) lateral view showing the ankylosed vertebral units; (b) ventral view; (c) detailed ventral view; note the striated bone bridging the intervertebral space appears to maintain the gross appearance of the ventral ligament.

Hyaenidae

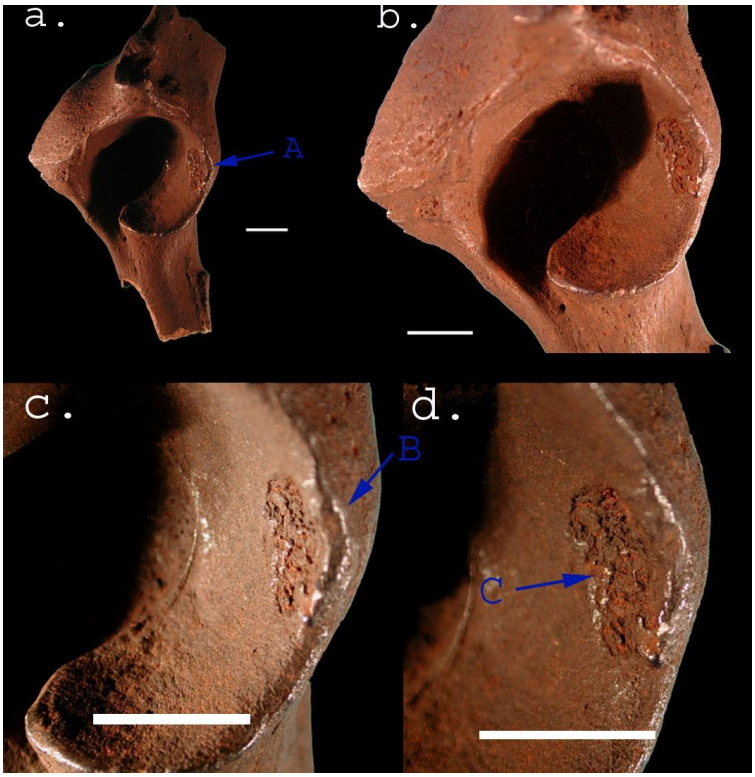


Figure 4**.20.** ***CD 6681*** **hyaenid left pelvis**: (a) lateral view showing the shallow lesion **(A)** on the lunate surface; (b) detailed lateral view; (c) detailed lateral view showing an area of periosteal bone formation **(B)** at the periphery of the acetabulum; (d) detailed lateral view showing the roughened surface inside the depression **(C)**.

Figure 4.21. ***CD 13301*** **hyaenid 2nd phalanx**: proximal view compared to a 2nd phalanx of a normal modern spotted hyaena (*Crocuta crocuta)*; note the marginal osteophytes and signs of bone destructive processes (lytic pitting).

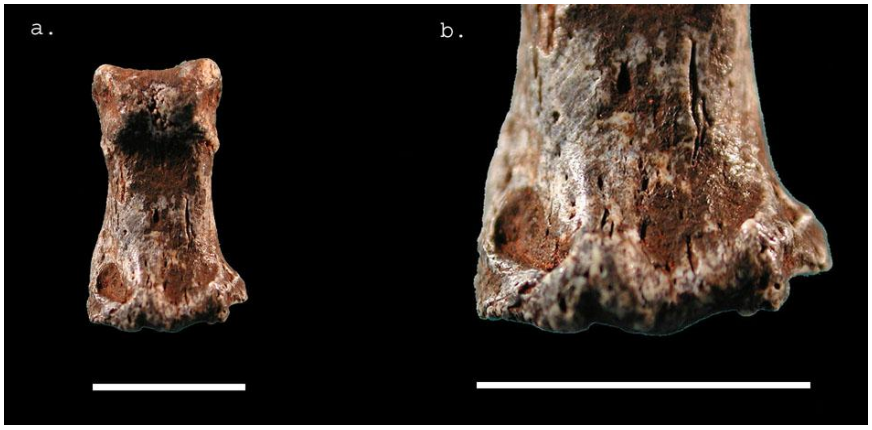


Figure 4**.22.** ***CD 13301*** **hyaenid 2nd phalanx**: (a) volar view; (b) detailed volar view showing osteophytes extending from the joint margin.

Figure 4**.23.** ***CD 9985*** **hyaenid terminal phalanx**: (left) plantar view compared to a 3rd phalanx of a normal modern spotted hyaena (*Crocuta crocuta*); (right) proximal view compared to normal modern spotted hyaena; notice the complete destruction and remodelling of the joint surface with the fossil retaining few of the anatomical characteristics of a normal terminal phalanx.

Cercopithecidae

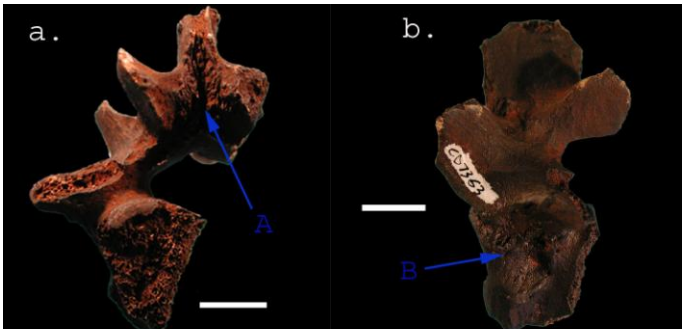


Figure 4.24. ***CD 7363*** **Cercopithecidae thoracic vertebra**: (a) posterior view; note the linear buildup of bone on the spinous process (**A**); (b) lateral view showing osteophyte development around the costal facet (**B**).

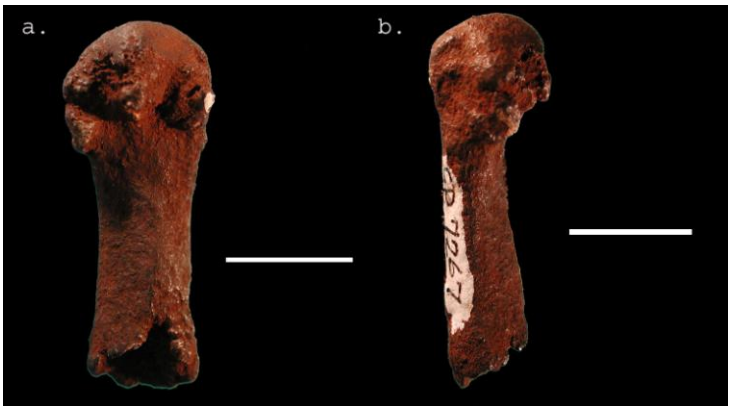


Figure 4.25. ***CD 7267*** **Cercopithecidae 1st ray metacarpal**: (a) palmar view; (b) lateral view; notice the osteophytes extending from the articular margin.

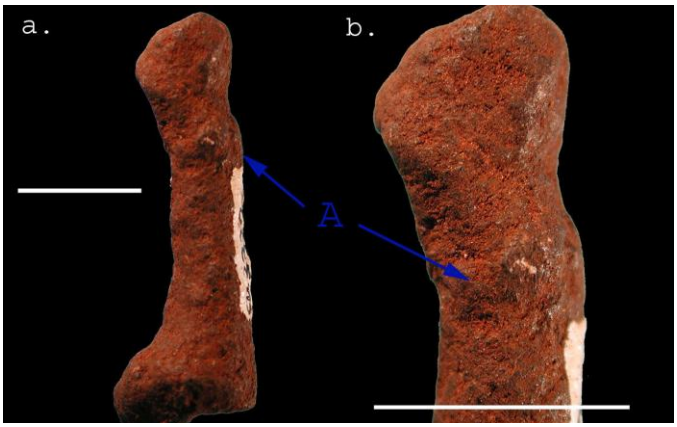


Figure 4.26. ***CD 3332*** **Cercopithecidae 1st ray metatarsal**: (a) medial view; (b) detailed medial view; note the abnormal bump of bone (**A**).

Hominidae

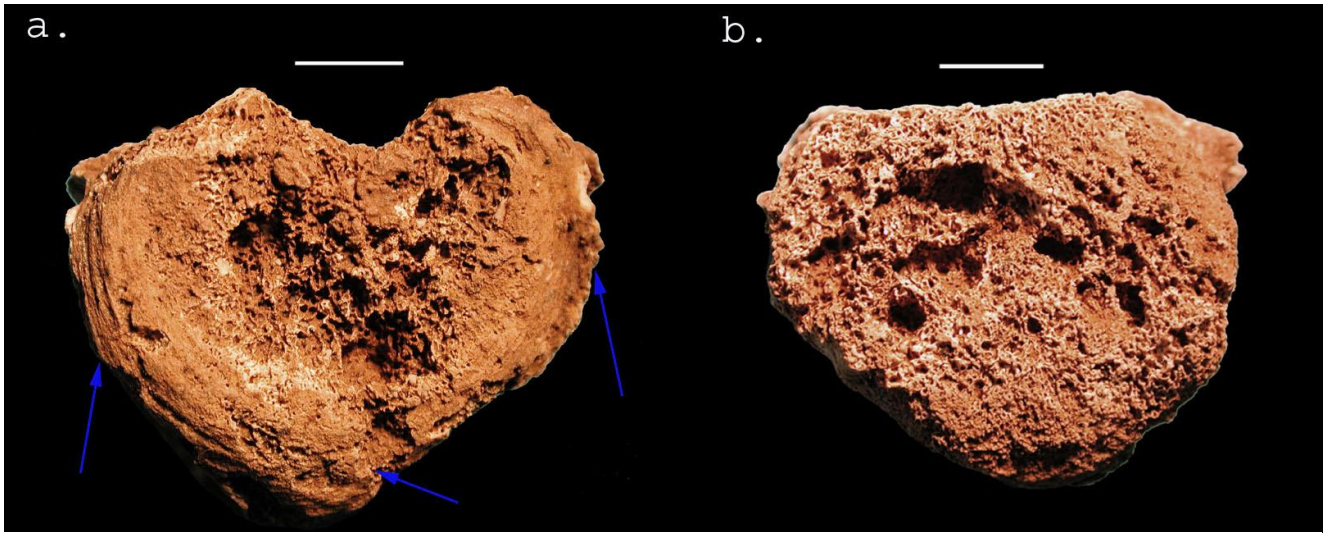


Figure 4.27. *CD 5288* Hominid lumbar vertebra: (a) superior view; notice the marginal osteophytes around the vertebral body (arrows); (b) inferior view; notice the several cyst-like erosions on both the inferior and superior surface of the vertebral body, possibly a result of postmortem weathering or possibly Schmorl's nodes. Notice also, the asymmetrical appearance following osteophyte development and ossification of the anterior longitudinal ligament.

Figure 4.28. *CD 5288* Hominid lumbar vertebra: anterior view; notice the extensive marginal osteophytes on both the superior and inferior aspects and the ossified anterior longitudinal ligament.

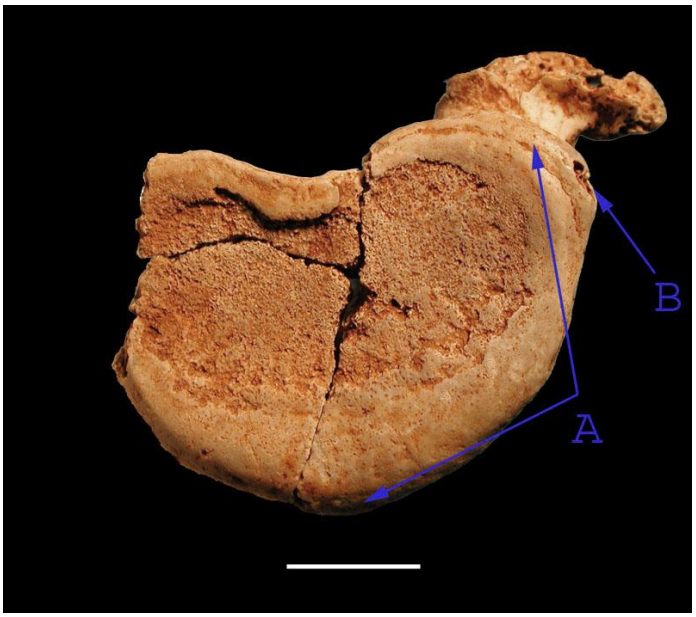


Figure 4.29. ***CD 5773*** **Hominid thoracic vertebra:** inferior view; notice the areas of osteophyte development **(A)** and the lytic focus associated with the marginal new bone **(B).**

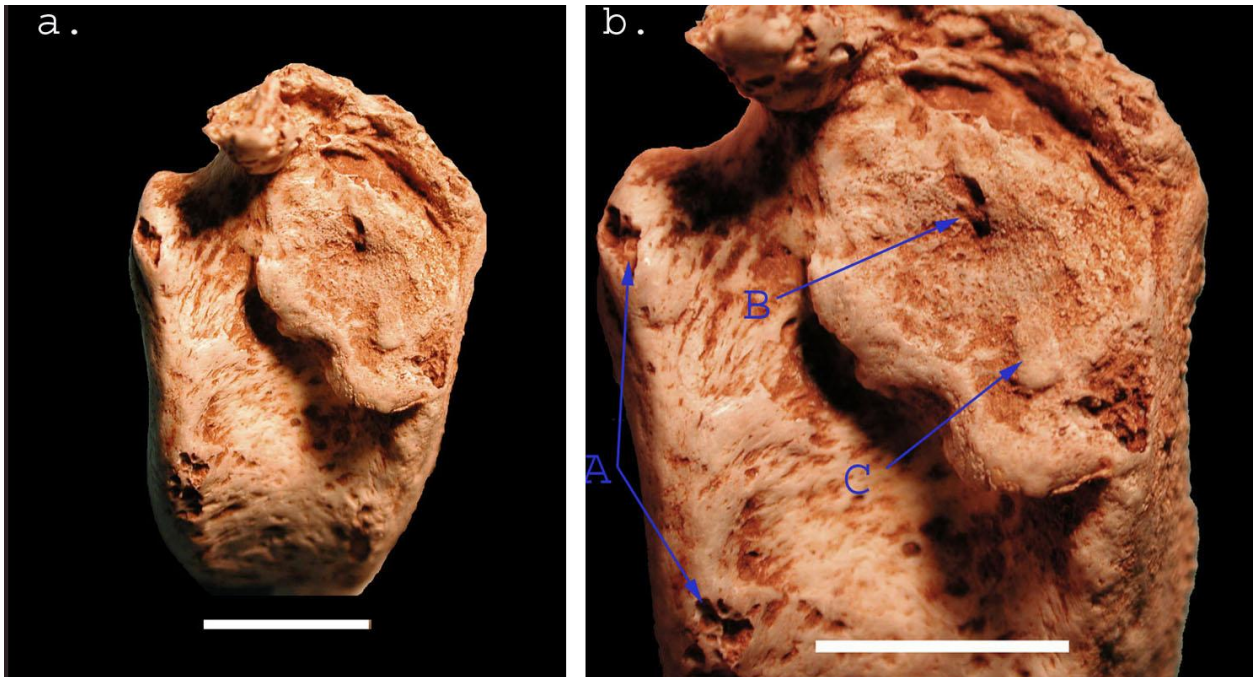


Figure 4.30. ***CD 5773*** **Hominid thoracic vertebra:** (a) lateral view; (b) detailed lateral view; notice the lytic foci **(A)** associated with the marginal new bone, the osteophyte development around the costal facet showing both, localized areas of subchondral bone destruction **(B)** and bone formation **(C)**.

Chapter 5. Swartkrans Pathology

Chapter 5 serves to present the pathological assemblage from Swartkrans. Each fossil is listed by its catalogue accession number (SK, SKX or SKW) under Order and Family subheadings. The information given includes the identification, description and classification of each pathological fossil. The format follows the methodology previously described (see Chapter 3.6). Figures highlighting the pathological morphology are provided at the end of the chapter.

5.1 Order: Artiodactyla

Family: Bovidae

5.1.1 SK 4248 (Figure 5.1)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: 1st phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation.

Distribution of lesion(s) within affected bone: Diaphysis.

Description: There is a marked crest of lamellar bone on the medial margin of the phalanx. The lesion occurs on the anterior surface, distal to the proximal

epiphyseal line. The crest measures approximately 12mm and extends distally, parallel to the long axis of the phalanx. The borders of the lesion are continuous with the surrounding cortex. The joint surface appears normal and no other associated skeletal abnormalities are present.

Diagnosis:

This lesion occurs on the proximal 1/3 of the element isolated on the medial border, forming a flat shelf extending in an anterior direction (up and out) instead of a medial direction (towards the other 1st phalanx). The new bone growth appears to be associated with the enthesis for ligamentous attachment. The bone projection forms a thin ridge, typical of a minor enthesophyte. Acute trauma can result in bone formation at entheses, but the projections tend to be large. This enthesopathy could be a conformation response following chronic trauma or gait change. The bone growth appears well-healed and there are no signs of joint disease, which may be expected given severe or chronic trauma.

Classification: *Enthesopathy*

Life history interpretation: Initial pain and altered gait.

5.1.2 SK 5645 (Figure 5.2)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Metatarsal

Preservation: Proximal

Side: Indeterminate

Taphonomy:

Postmortem transverse fracture at distal diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: There is a localized bump of bone, a subperiosteal thickening, seen as an oval raised area of lamellar bone on the anteromedial surface of the metatarsal. The lesion occurs on the proximal 1/3rd of the element and is oriented parallel to the long axis. The lesion measures 13mm long by 4mm wide and is raised approximately 1mm off of the natural bone surface. The oval bump of bone is smooth in surface texture and appearance and the borders appear continuous with the original cortex.

Diagnosis:

Localized subperiosteal thickening is generally attributed to blunt force trauma which generates a subperiosteal haematoma, which is almost always ossified by the overlying periosteum (Aufderheide and Rodriguez-Martin, 1998). (See 5.1.4 for discussion).

Classification: *Trauma*

Life history interpretation: Initial pain, later asymptomatic.

5.1.3 SK 5855 (Figure 5.3)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Metatarsal

Preservation: Proximal

Side: Right

Taphonomy:

Postmortem fracture at mid diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: Localized subperiosteal thickening seen as an oval bump of lamellar bone on the anteromedial aspect of the metatarsal. The lesion occurs on the proximal 1/3rd of the element and is oriented parallel to the long axis of the element. The lesion measures 19mm long and 9mm wide and is raised approximately 1mm. The bump of bone is smooth in surface texture and the borders appear continuous with the original cortex. The lesion appears quiescent.

Diagnosis:

Localized subperiosteal thickening is generally attributed to blunt force trauma which generates a subperiosteal haematoma, which is almost always ossified by the overlying periosteum (Aufderheide and Rodriguez-Martin, 1998). (See 5.1.4 for discussion)

Classification: *Trauma*

Life history interpretation: Initial pain, later asymptomatic.

5.1.4 SK 7633 (Figure 5.4)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Metatarsal

Preservation: Proximal

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: Localized subperiosteal thickening seen as an oval bump of lamellar bone on the anterior surface located on the medial aspect of the metatarsal. The lesion occurs on the proximal 1/3rd of the element and runs parallel to the long axis of the element. The lesion measures 11mm long by 6mm wide and is raised approximately 1mm.

Diagnosis:

Localized subperiosteal thickening is generally attributed to blunt force trauma which generates a subperiosteal haematoma which is almost always ossified by the overlying periosteum (Aufderheide and Rodriguez-Martin, 1998). Lesions of this type are common in human archaeological samples (Aufderheide and Rodriguez-Martin, 1998) and have been documented in a similar anatomical location on metatarsals of domesticated fauna (Brothwell *et al.*, 2005) and wild fauna (Brothwell *et al.*, 2005: 78). The lesions tend to be discrete and localized which differentiates them from periosteal reaction to infection which tends to be more diffuse. The fact that the lesions seen in the Swartkrans sample occur on the anteromedial surface of the metatarsal diaphyses suggest that the bone, being subcutaneous, with very little protection from external blunt trauma, may predispose the metatarsals of bovids to these minor traumas.

Classification: *Trauma*

Life history interpretation: Initial pain, later asymptomatic.

5.1.5 SK 6484 (Figure 5.5)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Tibia

Preservation: Distal

Side: Right

Taphonomy:

Postmortem fracture at proximal diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis, distal 2/3

Description: Bone formation seen as a rugose line of raised lamellar bone running parallel to the long axis of the tibia at the area of soft tissue attachment. The raised area of bone is smooth, has rounded borders and is continuous with the surrounding cortex. No other bone changes are present to the diaphysis or distal joint surface.

Diagnosis:

The lesion follows the enthesial line and represents a relatively severe muscle-marker scar. Lesions of this type are broadly considered enthesopathies and the general consensus regarding pathogenesis attributes the bone growth to a multifactoral aetiology. They have correlated with repetitive stress or trauma, sex, genetics, and most commonly as a concomitant of aging. In the case of SK 6484, possible injury or repetitive trauma may have caused the lesion. Possibly an altered gait resulting from another injury may have contributed to the development of the abnormal bone growth. The distal joint surface shows

no signs of arthritic changes, suggesting that age may not have been the principal cause of the enthesopathy.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.6 SK 7955 (Figure 5.6)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Thoracic vertebra

Preservation: Fragment

Side: N/A

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Joint margin

Description: Two irregular small nodules of lamellar bone are located around the cranial articular facet of the thoracic vertebra. The bone growths extend cranially towards the intervertebral space. The lesions appear to be isolated to the left side and are relatively small, 3mm by 1mm. The subchondral bone surfaces appear normal.

Diagnosis:

The lesions appear to be osteophytes associated with the cranial articular surface of the vertebra. This occurs commonly when degeneration of the

intervertebral disk allows closer approximation of the vertebral bodies, irritating the periosteum and resulting in the formation of new bony nodules around the joint margin (Aufderheide and Rodriguez-Martin, 1998). The small lesions on SK 7955 appear to be minor changes, without any signs of increased porosity or subchondral eburnation, suggesting that the condition was in its early stages at the time of death. Osteoarthritis commonly results in development of osteophytes at joint margins; however, without the concomitant changes discussed above, the general classification of 'joint disease' is most fitting.

Classification: *Joint disease*

Life history interpretation: Asymptomatic.

5.1.7 SK 9514 (Figure 5.7)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Metacarpal

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture at distal diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal metaphysis

Description: On the palmar surface, there is a small ridge of abnormal lamellar bone between the two condyles distal of the nutrient foramen. The crest runs

parallel to the long axis of the bone and measures 3mm long by 1mm wide, protruding 1mm from the bone surface. The border of the lesion is continual with the surrounding normal cortex.

Diagnosis:

The pathology likely resulted from bone growth into the soft tissues. Enthesophytes can occur when irritation of the periosteum at an area of soft tissue attachment to the bone surface stimulates bone growth. Osteoblasts infiltrate the area and bone proliferates, creating small spurs. They are commonly thought to be multifactoral in aetiology.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.8 SK 5792 (Figure 5.8)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Metacarpal

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture at distal diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal metaphysis

Description: There is a minor projection of lamellar bone extending from the distal epiphyseal line, at the base of the distal condyle. The lesion extends

medially from the medial aspect. It appears as a small sessile nodule, rounded at its apex, 2mm in diameter and reaching a height of 1mm. Some taphonomic changes are present, including a transverse taphonomic fracture to the diaphysis and some taphonomic abrasion to the dorsal surface of the metaphysis. This may obscure any other pathological changes; however, the rest of the fossil appears normal.

Diagnosis:

The lesion is periarticular and occurs at the line for the attachment of the metacarpophalangeal collateral ligament. There are no concomitant signs of joint disease and the lesion is rounded and not flared like common osteophytes. It is probable that the lesion is therefore an enthesopathy resulting from bone growth into the ligament. Enthesial bone growths are considered multifactoral in their aetiology. Trauma, age and genetics play a part in the development of enthesophytes. There is a strong likelihood that chronic or acute mechanical stress to the distal joints, specifically at the interphalangeal joint may have stimulated the bone growth at the entheses.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.9 SKX 2796 (Figure 5.9)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: 1st phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal epiphysis

Description: There is a small sessile nodule extending from the medial border of the distal metaphyseal surface of the phalanx. The nodule extends mediodistally, rising gradually from the proximal border and dropping more sharply at its distal end. The lesion is smooth and continuous with the surrounding cortex, has a diameter of 3mm and height of 2mm. On the same medial surface at the proximal metaphysis there is a second lesion. This is an irregular shaped flat plaque of periosteal reactive bone resting 1mm atop of the original bone surface. The borders are sharp and clearly defined. The lesion itself is smooth in appearance and texture and appears quiescent.

Diagnosis:

Both lesions occur on the medial surface of the phalanx, at the proximal and distal ends. As the lesions are periarticular, they are not evidence of an arthropathy. These lesions likely represent minor enthesophytes at the proximal and distal soft-tissue attachments. The distal lesion appears to be associated with the interphalangeal collateral ligament. The two distinct lesions are possibly the result of a conformation response to a sudden or chronic trauma, which may have altered the gait of the animal. Without any further evidence of trauma, however, the most appropriate classification is 'enthesopathy'.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.10 SKX 5796 (Figure 5.10)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: 1st phalanx

Preservation: Proximal

Side: Indeterminate

Taphonomy:

Postmortem fracture at proximal metaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Proximal metaphysis

Description: There is a small dorsal exostosis on the proximal metaphysis. The sessile lesion has a diameter of 2mm, is continuous with the original cortex of the bone and extends from the bone surface to a height of approximately 1mm. The taphonomic fractures have left only a portion of the proximal end of the phalanx available for analysis. The distal 2/3 of the element is missing entirely. It is impossible to note any other pathological changes to the bone.

Diagnosis:

The anatomical location of the lesion (i.e. close to the joint margin at an area for attachment of collateral ligaments) might indicate the strong possibility of osteoarthritis or enthesopathy. No marginal osteophytes or subchondral changes are apparent. Without any other concomitant joint changes and with the taphonomic loss of 2/3 of the specimen, it is difficult to determine the exact cause of the abnormality. For the purpose of this research the most appropriate classification is 'enthesopathy'.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.11 SKX 37836 (Figure 5.11)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: 1st phalanx

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture at mid diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal epiphysis

Description: There is a minor crest of abnormal bone on the lateral border of the distal epiphysis. The lesion appears as an arching crest of bone following the base of the distal epiphysis. The lesion is smooth and lamellar, rising gradually from the proximal border and dropping off sharply at its peak. The lesion reaches a height of 0.7mm. The distal joint surface and medial aspect of the bone are both normal.

Diagnosis:

The lesion occurs at the attachment of the interphalangeal collateral ligament. Avulsion, wear and tear, or age can result in bone proliferating at an area of ligamentous or tendinous attachment. It is likely that chronic mechanical stress at the distal interphalangeal joint may have stimulated the new bone growth at the entheses.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.12 SKX 37512 (Figures 5.12 and 5.13)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: 2nd phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diffuse

Description: An irregular line of abnormal bone is oriented on a proximodistal axis on the dorsal surface of the 2nd phalanx. The bone growth is not continuous and appears as small irregular nodules roughly located in a line, raised slightly (0.7 - 1.1mm) above the cortex. The borders of the lesion are sharp and not continuous with the surrounding cortex. The abnormal bone growth continues on the medial surface of the proximal joint margin. The lesion here is similar, presenting as discrete nodules of abnormal bone clustered at the proximal epiphysis and metaphysis. In some places the lesions appear smooth and lamellar and in other places as less organized woven bone.

Diagnosis:

The fact that the margins are clearly defined and not continuous with the surrounding bone suggests a condition that was longstanding but not healed at the time of death. The lesion represents an enthesopathy, with bone developing at the attachment for the collateral ligaments. Enthesopathy is considered multifactoral in its aetiology.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.1.13 SKX 35132 (Figure 5.14 and 5.15)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: III

Skeletal element:

Element: Atlas vertebra

Preservation: Fragment

Side: N/A

Taphonomy:

Postmortem breaks

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Lateral, non-articular surface

Description: A small and isolated area of woven bone occurs on the lateral (non-articular) surface of the atlas vertebra. The lesion is flat against the underlying bone cortex. It has clearly defined and sharp borders and is irregular in texture with a woven 'lattice' appearance. The lesion is made up of immature bone and was still active at the time of death.

Diagnosis:

The fact that the lesion is circumscribed suggests trauma as a likely aetiology. Infection is also a possibility for creating isolated areas of new bone. The new bone occurs in a very circumscribed area and is fairly well remodelled which suggests a relatively longstanding condition. The most accurate classification for the abnormality is trauma, resulting in an area of inflammation and periosteal reactive bone, i.e. periostitis.

Classification: *Trauma*

Life history interpretation: Pain.

5.1.14 SKX 5043 (Figure 5.16)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Tarsal indeterminate

Preservation: Fragment

Side: Indeterminate

Taphonomy:

Postmortem fracture exposing a sagittal cross section

Pathology:

Type: Abnormal bone loss

Distribution of lesion(s) within affected bone: Interior cancellous bone

Description: An oval cavity measuring 8mm by 4mm is visible in the cross section of the tarsal at an area of postmortem fracture. The lytic lesion is hollow and uniform with a smooth interior surface and a thin cortex. The cancellous bone on either side of the lesion appears normally latticed and the

smooth erosion is confined to this oval. The fact that the remainder of the cancellous bone is normal, and the presence of the thin interior cortex, suggest a pathological, rather than taphonomic origin.

Diagnosis:

The cavity is smooth and enclosed by a thin cortex. There are no proliferative changes indicative of infection which suggests that the lesion is likely not an infectious cloaca. The lesion may be a cyst, which is characterized *in vivo* by a fluid-filled cavity enclosed by a lining often composed of connective tissue (Ortner, 2003). An enchondroma is a fairly common benign cartilaginous tumour (Redfern *et al.,* 1997) with a predilection to the tubular bones of the hands and feet (Ortner, 2003) in humans. Although uncommon, cases of enchondromas have been noted in carpal or tarsal bones in modern humans (Takigawa, 1971; Minkowitz *et al.,* 1992; Redfern *et al.,* 1997). With such a fragmentary specimen, it is difficult to determine the specific aetiology of the pathology noted in SKX 4053. Based on reference literature, the lesion is possibly a benign tumour, either a cyst or enchondroma, and is characterized for this study as a neoplasm.

Classification: *Neoplasm*

Life history interpretation: Asymptomatic. Enchondromas are generally asymptomatic until cortical erosion or pathological fracture (Gaulke and Suppelna, 2004).

5.1.15 SKX 13569 (Figure 5.17)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Tarsal indeterminate

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion(s) within affected bone: Joint margins

Description: There is a condition of abnormal bone growth covering half of the joint margin of the tarsal. The lesion is irregular in shape and contour. It is predominantly hypertrophic, however, there are clear lytic foci visible. The lesion extends two millimeters from the normal bone surface and the margins are sharp and irregular. The subchondral bone surface is normal.

Diagnosis:

The bone growths extending from the joint margins appear to indicate an arthropathy, whereby degeneration of the joint capsule over time has led to closer approximation of the articulating bones resulting in bone growth at the margins. The changes appear similar to navicular disease in horses (Stashak, 2002). Navicular disease is a degenerative joint condition that involves damage of the flexor surface of the bone and osteophyte development (Kahn and Line, 2005). Ostblom *et al.*, (1982) suggest that the lesions of navicular disease are a consequence of increased activation of bone remodelling caused by altered pressure and an increased load on the caudal part of the foot. This suggests that a change in mechanical loading following a change in gate may have resulted in the pathology.

Classification: *Joint disease*

Life history interpretation: Pain and altered gait.

5.1.16 SKX 6824 (Figure 5.18)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: II

Skeletal element:

Element: Carpal indeterminate

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion(s) within affected bone: Joint margins

Description: There is an area of diffuse bone development covering over half of the articular margin of the carpal. The lesion is predominantly a deposition, irregular in shape and contour, with visible lytic foci. The lesion is smooth and lamellar in structure and appears to be well remodelled. The subchondral surface shows signs of increased porosity.

Diagnosis:

Bone growth isolated at the joint margins is indicative of degenerative joint disease, whereby degeneration of the joint capsule over time results in closer approximation of the adjacent bones, irritating the periosteum and stimulating osteophyte development. The osteophytes seen on SKX 6824 appear to be an indicator of a degenerative joint condition, possibly osteoarthritis. The subchondral porosity is also indicative of OA. The striking similarity in gross appearance of the lesions between SKX 6824 and SKX 13569 might indicate that the bones belong to the same individual, suggesting diffuse or multifocal joint disease.

Classification: *Joint disease*

Life history interpretation: Pain and altered gait.

5.1.17 SKX 9785 (Figure 5.19)

Taxonomic classification:

Order: Artiodactyla

Family: Bovidae

Genus: Indeterminate

Species: Indeterminate

Class size: III

Skeletal element:

Element: Cuneiform

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion(s) within affected bone: Joint margins

Description: There is diffuse osteophyte development encircling the bone, covering the non-articular surface. The lesion is predominantly hypertrophic, irregular in shape and contour, with associated visible lytic foci. The new bone growth extends towards the joint surface. The bone itself is smooth, lamellar and appears to be well remodelled. The subchondral surface shows some minor signs of increased porosity.

Diagnosis:

Bone growth is isolated to the joint margins. The bone growths appear consistent with osteophytes, whereby degeneration of the joint capsule over time results in closer approximation of the adjacent bones, irritating the periosteum and stimulating osteophyte development. The osteophytes seen on SKX 9785 appear similar to those encountered in the pilot study and in the

palaeopathology reference material. The bone changes appear to be a clear indicator of a degenerative joint condition, possibly osteoarthritis. The subchondral porosity is also indicative of OA. The striking similarity in gross appearance of the lesions between SKX 9785 and SKX 6824 and SKX 13569 might indicate that all three elements belong to the same individual, suggesting a diffuse or multifocal joint disease.

Classification: *Joint disease*

Life history interpretation: Pain and altered gait.

5.2 Order: Carnivora

Family: Felidae

5.2.1 SK 1854 (Figure 5.20)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Dinofelis*

Species: sp.

Class size: Medium

Skeletal element:

Element: Metapodial

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture at proximal metaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: On the dorsal surface of the metapodial there occurs a large irregular protrusion of bone extending dorsolaterally over the diaphysis. The lesion appears at midshaft as a lamellar deposition oriented parallel to the long axis of the bone and extending distally for approximately 2cm. The irregular lesion is raised 6mm above the normal bone surface. The bone is taphonomically fractured at midshaft and it is impossible to see the extent of the lesion towards the proximal end. The lesion appears quiescent, with smooth borders, continuous with the original bone cortex.

Diagnosis:

The lesion appears at an area of soft tissue attachment. It is larger and more irregular than an enthesophyte and is likely not a typical enthesopathy. The

lesion appears to be a post-traumatic ossification of the soft tissues on the dorsal surface of the metapodial, a condition referred to as *myositis ossificans traumatica*. The condition occurs following avulsion of a tendinous or muscular attachment to bone. A haematoma is formed following the injury and often, depending upon the proximity of the injury to the periosteum, the haematoma may become calcified or ossified. SK 9541 shows an irregular ossified mass of bone likely following avulsion of one of the extensors of the foot, possibly *m. extensor digitorum brevis*.

Classification: *Trauma* (with enthesopathy)

Life history interpretation: Pain and altered function.

5.2.2 SK 6747 (Figure 5.21)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *leo*

Class size: Large

Skeletal element:

Element: Metapodial

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture at distal diaphysis; adhesion of manganese and matrix

Pathology:

Type: Abnormal bone formation / abnormal shape

Distribution of lesion(s) within affected bone: Distal metaphysis

Description: A ridge of abnormal lamellar bone extends across the volar surface of the metapodial, perpendicular to the long axis of the bone. It occurs at the distal metaphysis 2mm proximal to the articular border. The lesion

measures 9mm in length and is 2.6mm wide, extending to a height of 1.7mm. The lesion forms a 'shelf' rising gradually from the proximal normal cortex and then cutting sharply back towards the metaphysis.

Diagnosis:

The pathomorphology is consistent with bone growth at the entheses, specifically the collateral ligaments. Enthesopathies are generally considered multifactoral in aetiology. Trauma or chronic microtrauma can stimulate the formation of enthesophytes, although many factors may be involved. While the lesion is larger than a typical enthesophyte it is not as extensive as one might expect in a case of *myositis ossificans traumatic.* The most appropriate classification for the pathology is 'enthesopathy'.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.2.3 SKX 4203 (Figure 5.22)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *pardus*

Class size: Medium

Skeletal element:

Element: Patella

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone loss / abnormal bone formation

Distribution of lesion(s) within affected bone: Non-articular surface

Description: There is a longitudinal groove running the proximal 1/3 of the non-articular surface of the patella. The interior of the lesion appears to be fully remodelled, smooth lamellar bone. The borders of the lesion are rounded, not sharp. The groove dips 1mm below the original cortex and is 8mm long in a proximodistal orientation. The articular facets are normal and no marginal bone growth is present.

Diagnosis:

The groove appears to be associated with the soft tissue on the non-articular surface of the patella. The lesion is consistent with an osteolytic enthesopathy. In modern humans the osteolytic type expression of enthesial pathology is typically correlated with young adults and the frequency of the pathology generally decreases with age (Mariotti *et al.*, 2003). It is unclear whether the same correlation occurs in other mammalian taxa.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.2.4 SKX 558 (Figure 5.23)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *pardus*

Class size: Medium

Skeletal element:

Element: Metapodial

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture at mid diaphysis

Pathology:

Type: Abnormal bone formation / abnormal bone shape

Distribution of lesion(s) within affected bone: Distal metaphysis

Description: On the distal metaphysis of the metapodial, there is an area of hypertrophic lamellar bone extending to one side. This, along with apparent extensive remodelling of the dorsal surface, forms a flat shelf of bone, confined to the distal metaphysis. The bone texture is smooth and the borders are continuous with the surrounding cortex. The condition appears to have been quiescent at the time of death. No concomitant changes are present and the distal epiphysis appears normal.

Diagnosis:

The remodelling of the distal metaphysis has resulted in the normally rounded shape of the dorsal surface of the tubular bone to become increasingly flat towards the distal end. The bone growth is fully remodelled suggesting longstanding and well-healed localized periostitis, or ossified haematoma. Periostitis can result from trauma to the periosteum or infection of adjacent soft tissues. The pathology may be attributed to a trauma to the bone itself, to the overlying soft tissue or to the joint structures.

Classification: *Trauma*

Life history interpretation: Initial pain and altered gait, later asymptomatic.

5.2.5 SKX 5471 (Figure 5.24)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *pardus*

Class size: Medium

Skeletal element:

Element: Metatarsal

Preservation: Proximal

Side: Indeterminate

Taphonomy:

Recent break at distal diaphysis.

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: There is an abnormal lamellar crest of bone on the dorsal surface of the metatarsal, beginning midshaft and extending distally. The crest is thin and irregular with borders that are continuous with the surrounding bone surface. The lesion gradually rises from the bone surface at midshaft at the area for attachment of the extensor muscles, and reaches a height of 3mm from the original cortex. There is a small, pin prick sized, perforation through the lesion in the middle of the crest close to the bone surface. There is a recent fracture separating the distal 1/3 of the metatarsal and it is impossible to see if the lesion extends further distally or if there are any concomitant changes to the distal joint. The proximal articular surface is normal.

Diagnosis:

The lesion seen on SKX 5471 appears to be a rather excessive abnormal bone growth confined to the diaphysis of the metatarsal. It appears that the most likely cause of the abnormality is *myositis ossificans traumatica*, traumatic ossification of the soft tissue following injury. An acute trauma to the metapodial or the metapodial-phalangeal joint may have resulted in injury or avulsion of the soft tissues (extensor muscles). The injury would stimulate the periosteum and result in the development of abnormal bone. The lesion appears longstanding and well-healed, suggesting that the individual recovered from the trauma and survived and at the time of death the lesion was quiescent.

Classification: *Trauma* (with enthesopathy)

Life history interpretation: Pain and altered function.

5.2.6 SKX 8484a (Figures 5.25 and 5.26)

Taxonomic classification:

Order: Carnivora

Family: Felidae

Genus: *Panthera*

Species: *leo*

Class size: Large

Skeletal element:

Element: 2nd phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion(s) within affected bone: Diffuse

Description: The lesion appears as a porous and striated woven bone, visibly more marked on the dorsal surface but apparent throughout. Several ovoid cavities occur on the distal articular subchondral surface and extend into the bone's medulla. The remainder of the element is noticeably light and porous compared to a healthy analogue. There is a clear decrease in bone density and mass and the necrotic appearance of the element.

Diagnosis:

Given the extent of the remodelling, the lesion appears to be longstanding; however, the sharp borders and the obvious areas of visible woven bone suggest that the condition was not healed. Generalized formation of bone may have several possible causes, including trauma, infection, and metabolic disturbance. Hypertrophic pulmonary osteoarthropathy may result in proliferative lesions (Ortner, 2003). The marked decrease in bone density and the lytic component to the defect might indicate a chronic infectious disease. The element, however, lacks the 'swollen' appearance typical of osteomyelitis

or osteitis. The lesion is predominantly lytic and suggestive of osteonecrosis. The most common cause of osteonecrosis is trauma (Abraham and Malkani, 2004).

Classification: *Trauma*

Life history interpretation: Pain and altered gait.

5.3 Order: Carnivora

Family: Canidae

5.3.1 **SKX 7994** (Figure 5.27)

Taxonomic classification:

Order: Carnivora

Family: Canidae

Genus: Indeterminate

Species: Indeterminate.

Skeletal element:

Element: 1st phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal metaphysis

Description: There is a small nodule of lamellar bone extending from the metaphysis distally towards the joint margin. The lesion is only slightly raised from the original bone surface, reaching a height at its apex of 1mm. The width of the lesion at the base is 2mm. The abnormal bone is smooth in appearance and texture and is continuous with the original bone surface. The projection has a sharp distal border where it cuts back towards the bone. The distal and proximal joint surfaces and the diaphysis appear normal.

Diagnosis:

The bony projection from the distal metaphysis is likely an enthesopathy resulting from bone proliferation at the interphalangeal collateral ligament. The aetiology of enthesophytes is generally considered multifactoral with age,

wear and tear, and trauma all playing a role in the development of the bone changes.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.3.2 SKX 8200 (Figures 5.28 and 5.29)

Taxonomic classification:

Order: Carnivora

Family: Canidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Metacarpal

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Proximal metaphysis

Description: There is a large circumscribed area of lamellar bone on the lateral surface of the proximal metaphysis of the metacarpal. The lesion is oriented perpendicular to the long axis of the element; it rises gradually from the bone surface and extends towards the proximal joint surface and rounds off at its apex, sloping back towards the metaphysis. The lesion has a width of 3mm and reaches a height of 2mm at its proximal most extent. The abnormal growth does not overlap the proximal joint surface, but extends to the joint margin. The lesion appears isolated, with no other pathological changes visible on the proximal or distal surfaces or diaphysis. The surface of the lesion is smooth and is continuous with the original cortex of the bone.

Diagnosis:

Bone outgrowths at joint margins are commonly correlated with joint disease, particularly osteoarthritis. Osteoarthritis therefore should be considered as a potential cause of the bone pathology. The size of the lesion relative to the overall size of the element, however, suggests another aetiology. The lesion appears to be an enthesopathy. The location might suggest an avulsion injury or chronic low grade trauma to the soft-tissue constituents of the joint. Although the lesion is extensive and possibly traumatic in origin, the most fitting classification is enthesopathy.

Classification: *Enthesopathy*

Life history interpretation: Pain and altered function.

5.3.3 **SKX 30774** (Figure 5.30)

Taxonomic classification:

Order: Carnivora

Family: Canidae

Genus: Indeterminate

Species: Indeterminate.

Skeletal element:

Element: Patella

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion(s) within affected bone: Joint margin

Description: There is slight osteophytic development around the joint margin. The abnormal bone formation occurs on the inferior surface of the bone extending away from the bone surface. Associated with this minor abnormal

bone formation is a marked zone of abnormal bone loss, giving the inferior surface of the patella a very porous appearance. Both, the proliferative lesion and the interior lytic areas have sharp and irregular borders. The actual articular surface appears normal and the opposite non-articular surface of the bone also appears unchanged.

Diagnosis:

The patella, as a sesamoid and as part of the stifle joint, is susceptible to a myriad of traumatic insults, primarily involving damage to the ligaments and tendons (Kahn and Line, 2005). Similarly, the wear and tear on the knee predisposes it to one of the highest rates of osteoarthritis in humans (Ortner, 2003). The marginal osteophytes on the canid patella, SKX 30774, appear grossly similar to osteoarthritic changes observed during the course of this study. The osteophytes appear minor and the bone lacks the eburnation, surface pitting, grooving, or sclerosis necessary to diagnose the pathology as osteoarthritis. 'Joint disease' is the most fitting classification.

Classification: *Joint disease*

Life history interpretation: Pain and altered function.

5.3.4 SKX 39659 (Figure 5.31)

Taxonomic classification:

Order: Carnivora

Family: Canidae

Genus: Indeterminate

Species: Indeterminate.

Skeletal element:

Element: 1st phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Proximal metaphysis

Description: There is a small round sessile nodule on the proximal metaphysis. The lesion occurs as a raised bump of lamellar bone on the lateral surface extending gradually from its distal extent proximally and rounding off at its proximal margin. The area of abnormal growth has a diameter of 2mm. The surface of the lesion is smooth and is continuous with the original cortex of the bone. The lesion reaches a height at its apex of approximately 2mm.

Diagnosis:

A small sessile periarticular lesion is not an osteophyte as it is not located at the joint margin. The lesion does not project perpendicular to the axis of the element as would be expected in an osteochondroma. The bone projection appears to be associated with the enthesis and is likely an enthesophyte. The lesion is rounded and well-remodelled, suggesting that the enthesopathy was long-standing and not active at the time of death. Accumulated microtrauma, acute trauma or age may affect the development of enthesophytes.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.4 Order: Primates

Family: Cercopithecidae

5.4.1 SK 1506 (Figure 5.32)

Taxonomic classification:

Order: Primates

Family: Cercopithecidae

Genus: Indeterminate

Species: Indeterminate.

Skeletal element:

Element: Humerus

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem spiral fracture at distal diaphysis.

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal epiphysis

Description: There is a large irregular nodule of abnormal bone at the margin of the medial epicondyle of the distal humerus. The bone appears as a thin ridge following the posterior border of the epicondyle. The projection extends medially reaching a height of 3mm from the original cortex. At its extent the bone has a scalloped and irregular appearance; however the lesion itself is lamellar and appears longstanding. There is associated minor osteophyte development on the joint margin of the trochlea.

Diagnosis:

The lesions described above have a joint degeneration component. Minor osteophytes on the trochlear margin are most commonly associated with osteoarthritis of the elbow. It should be noted, however, that osteoarthritis of the elbow in modern humans is rare and usually secondary to trauma (including

occupational trauma) (Aufderheide and Rodriguez-Martin, 1998). Clearly different use of the elbow by a non-human primate, including ambulation, may predispose the joint to degenerative changes. The most dramatic lesion on SK 1506, is, however, not associated with the joint margin and instead occurs on a non-articular site for origin of the common flexor tendon, which facilitates in the flexion of the hand and wrist (Agur and Dalley, 2005). This suggests that an enthesopathy is perhaps the principal aetiology and the joint degeneration may be a conformation response following an altered gait or usage pattern.

Classification: *Enthesopathy*

Life history interpretation: Pain and altered function.

5.4.2 SK 1867 (Figure 5.33)

Taxonomic classification:

Order: Primates

Family: Cercopithecidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Radius

Preservation: Proximal

Side: Indeterminate

Taphonomy:

Postmortem transverse fracture at proximal diaphysis.

Pathology:

Type: Abnormal bone loss / abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: A large lytic focus occurs in the centre of the radial tuberosity extending down into the underlying bone. There is marked remodelling around the lytic focus with a clear buttressing of the borders of the lesion. The external borders of the lesion are lamellar and appear smooth and raised

slightly from the bone surface. A linear groove occurs proximal to the lesion and runs perpendicular to the long axis of the element. There appears to be some remodelling of this second lesion, with its borders being smooth and rounded and some evidence of bone formation at its periphery. The proximal joint surface and margins appear normal.

Diagnosis:

The cavity is located at an enthesis. There are no proliferative bone changes, periostitis, or bone swelling to suggest osteomyelitis or infection. The lesion is unlikely to be a cloaca and more likely to be an osteolytic expression of an enthesopathy. This type of lesion appears to reverse correlate with age, with the highest frequencies in modern humans occurring in young adults with a decrease in mature adults (Mariotti *et al.*, 2004). It has been suggested that this type of pathology may reflect the strong remodelling processes accompanying growth (Robb, 1998).

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5 Order: Primates

Family: Hominidae

5.5.1 SKX 3342 (Figure 5.34)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: *Homo*

Species: Indeterminate

Skeletal element:

Element: Thoracic vertebra (T6-T9)

Preservation: Body fragment

Side: N/A

Taphonomy:

Postmortem sagittal fracture through the centrum

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Vertebral body

Description: There is a clear marginal osteophyte development on the centrum of the thoracic vertebra. The osteophytes occur on the superior and inferior margins of the vertebral body. The lesion begins anterior to the costal facet, gradually increasing in height and reaching a height of 4.2mm at the anterior-most extent of the centrum. The osteophytes extend out from the original cortex and towards the intervertebral space. Only the right half of the vertebral body is present and it is impossible to see the extent of the lesion on the left, although it appears to be continuous and bilateral. No observable changes are visible on the subchondral surface of the vertebral body. The postmortem fracture of the element revealed a clear cross section of the bone, which shows minor sclerosis of the underlying trabeculae.

Diagnosis:

Degenerative changes to the vertebrae are extremely common in modern humans. These changes appear to be age-progressive and occur in modern humans as an inevitable concomitant of normal aging with 80-90% involvement after the age of 75 (Aufderheide and Rodriguez-Martin, 1998). In the case of SKX 3342, the lesions appear as osteophytes at the margins of the vertebral body. This occurs when progressive degeneration of the intervertebral disk allows closer approximation of the two articulating vertebrae. Contact at the vertebral margins irritates the periosteum and stimulates new bone growth. Involved locations are those that are most frequently flexed, for thoracic T8-T9 in modern humans (Aufderheide and Rodriguez-Martin, 1998).

Classification: *Joint disease* (*spondylosis*)

Life history interpretation: Possible pain.

5.5.2 SKX 5017 (Figure 5.35)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: *Paranthropus*

Species: *robustus*

Skeletal element:

Element: Metatarsal I

Preservation: Complete

Side: Left

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal metaphysis

Description: There is a pronounced tubercle of bone on the dorsal surface of the metatarsal immediately proximal to the distal joint margin. The tubercle is oriented on a mediolateral axis, covering over ½ the width of the diaphysis. It measures 5mm by 2.3mm (proximodistal axis). The lesion is pointed at its apex and reaches a height of 2mm. The bone is lamellar and continuous with the original surrounding cortex. No pathological changes are seen on the proximal or distal joint surfaces or margins.

Diagnosis:

Small solitary exostoses have been attributed to osteochondromas in the past, in a process whereby an outgrowth of cartilage from the growth plate undergoes endochondral ossification forming a pedunculated or sessile lesion (Richardson, 2005). There is also a possible traumatic aetiology for the solitary lesions, with injury to the peripheral growth plate triggering the formation of the exostosis.

Classification: *Trauma*

Life history interpretation: Initial pain and possible altered function.

5.5.3 SKX 45690 (Figures 5.36 and 5.37)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: *Paranthropus*

Species: *robustus*

Skeletal element:

Element: Hallucial 1st phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: A raised bar of lamellar bone is located on the dorsal surface of the phalanx 3mm distal of the proximal joint surface, at the site for the insertion of the *m. extensor hallucis brevis*. The abnormal raised area of bone is long and relatively narrow, and runs perpendicular to the long axis of the phalanx from the dorsolateral to the dorsomedial border. It is gradually sloped with a gently rounded apex and continuous borders with the original bone cortex. The lesion is 5mm long (on a mediolateral axis) and 2mm wide (on a proximodistal axis). It reaches a height of 1mm. No observable changes occur to the proximal or distal joint surfaces or the other non-articular surfaces.

Diagnosis:

The bar of bone on the dorsal surface of the phalanx may represent an enthesopathy. Chronic micro-trauma involving the *m. extensor hallucis brevis* may have resulted in the development of an enthesophyte. The *m. extensor hallucis brevis* acts to extend the great toe. The enthesophyte may be so long-standing and heavily remodelled that it has adopted the slightly raised and smooth appearance seen in the fossil, rather than a pointed and sharp appearance common to enthesophytes (Charlotte Roberts, personal communication, 2008).

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.4 SKX 5020 (Figure 5.38)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: *Paranthropus*

Species: *robustus*

Skeletal element:

Element: Metacarpal I

Preservation: Complete

Side: Right

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: There is a slightly raised irregular crest of lamellar bone running the lateral border, parallel to the long axis of the metacarpal at the insertion of the *m. oponens pollicis*. The lesion is very irregular and not continuous, appearing as many small discrete nodules running the length of the line, rather than one continuous crest. The lesion begins midshaft and continues distally to a point of postmortem fracture, proximal to the metacarpophalangeal joint. There is no way to see if the lesion continues further distally or if the distal articular surface is abnormal. The proximal joint surface is normal.

Diagnosis:

The lesion appears to be bone formation associated with the enthesis. The ridge of bone growing into the soft tissue at the area for soft tissue attachment to the bone is multifactoral in its aetiology. The *m. oponens pollicis* is responsible for opposing the thumb towards the centre of the palm and rotating it medially (Agur and Dalley, 2005). Accumulated microtrauma, acute trauma or age may affect the development of enthesophytes. There is increasing evidence suggesting a genetic component may play a part in enthesopathy, creating a possible predisposition to bone forming.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.5 SKX 5022 (Figure 5.39)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: *Paranthropus*

Species: *robustus*

Skeletal element:

Element: 2nd phalanx

Preservation: Proximal

Side: Indeterminate

Taphonomy:

Post-depositional erosion of the distal joint surface

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: There is a slight lamellar bone projection on the palmar surface at the insertion for the *m. flexor digitorum superficialis*. The lesion occurs on the lateral side and extends laterally for 2mm. The borders are smooth and continuous with the surrounding cortex.

Diagnosis:

The lesion is an enthesial reaction with bone proliferating at the area for attachment of the *m. flexor digitorum superficialis.* This muscle is responsible for flexion of the middle phalanges of the fingers at the proximal interphalangeal joint, and under continued action flexion of the metacarpophalangeal joint and wrist (Agur and Dalley, 2005). Recent evidence suggests that the aetiology for enthesopathy is multifactoral. Compounded or chronic microtrauma may spark the development of enthesophytes. The fact that the condition is isolated to one side is not unusual, based on observations made during this study. It is possible that trauma resulted in the bone changes. The lesion is relatively small and may be in its

early stages of development, possibly before the bone changes were obvious on the phalanx bilaterally.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.6 SK 7923 (Figures 5.40, 5.41 and 5.42)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Metatarsal

Preservation: Diaphysis

Side: Indeterminate

Taphonomy:

Postmortem fracture at proximal metaphysis and mid-diaphysis

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Proximal metaphysis

Description: A large, bulbous lesion occurs on the proximal metaphysis of the metatarsal. The taphonomic fracturing of the proximal and distal ends of the bone makes the precise identification of the element difficult. The size and the location of the pathological lesion further obscure the normal morphology. It is clear that the mass of bone extends from the proximal metaphysis of the metatarsal, although it is unclear whether it originates from the plantar surface (for 2nd through 4th ray metatarsal) or lateral surface (for 5th ray metatarsal). It appears most similar to a 2nd or 3rd metatarsal. The margins of the lesion are relatively sharply defined and not continuous with the cortex. The external surface of the lesion is irregular and porous. The lesion is roughly circular with

a diameter of 5.2mm and reaches a height of 4.7mm. A Computer Tomography (CT) scan revealed the lesion to be far less dense than the underlying cortical bone. The area of abnormal bone is discrete and confined to the metaphysis. There are no other concomitant changes to the diaphysis. There are no signs of sclerosis or rarefaction of the cortical bone underlying the lesion. The proximal and distal epiphyses are missing taphonomically and it is impossible to see if the epiphyses show signs of any pathological changes.

Diagnosis:

The lesion appears to be a neoplasm. It occurs as a lump of new bone on the metaphysis of the metatarsal that is highly circumscribed. Benign neoplasms generally have a limited growth potential, and the peripheral borders are usually sharply demarcated (Aufderheide and Rodriguez-Martin, 1998) as seen in the fossil. The benign lesions often affect the bone only by compression which can stimulate the bone to produce a fibrous wall enveloping the neoplasm (Aufderheide and Rodriguez-Martin, 1998). The abnormal growth observed in SK 7923 has a thin layer of cortical bone on the surface, encircling the lesion. It also appears relatively non-invasive, with minimal bone changes to the normal underlying cortical structures (as seen on CT scan). The basic features observed in SK 7923, therefore most closely correspond to a benign neoplasm. Malignant forms usually lack a capsule, have a growth rate that is much faster, and have little respect for the surrounding normal cells (Ortner, 2003). In general the aetiology of most cancers is poorly understood. Tumours that affect the tubular bones of the feet are enchondroma and to a lesser extent osteochondroma. Enchondromas are common, but they occur within the medullary cavity of tubular bones. Osteochondroma displays medullary continuity between the lesion and the bone (Kamath *et al.*, 2007), a feature that is absent in the fossil. It is possible that the lesion represents a periosteal chondroma or an osteoid osteoma. Osteoid osteoma accounts for nearly 20% of all benign tumours in the foot or ankle (Foo and Raby, 2005).

Classification: *Neoplasm*

Life history interpretation: The condition can result in tenderness and swelling (Tsang and Wu, 2008), and would certainly have caused pain and affected gait.

5.5.7 SKX 9342 (Figure 5.43)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: 1st phalanx

Preservation: Distal

Side: Indeterminate

Taphonomy:

Postmortem fracture mid diaphysis

Pathology:

Type: Abnormal bone formation / abnormal bone loss

Distribution of lesion(s) within affected bone: Diaphysis

Description: An irregular area of woven bone growth occurs only on one side (left) of the palmar surface of the phalanx at the area of attachment for the annular digital flexor sheath. The projection of bone is irregular, presenting as a jagged sharp crest. The crest of bone runs parallel to the long axis of the element and is broken by a clear area of abnormal bone loss in the centre. The lesion is 5mm long and 2mm wide and reaches a height of 1mm at its apex. The margins of the lesion are continuous with the original surrounding cortex.

Another, dissimilar lesion is seen on the opposite side (right). The lesion is a sessile projection of lamellar bone occurring immediately proximal of the distal interphalangeal joint margin. The exostosis is rounded at its apex and smooth with continuous borders with the original surrounding cortex. It rises gradually

in a distal direction. The lesion has a diameter of 4mm and reaches a height at its apex of 1.5mm.

Diagnosis:

SKX 9342 shows an enthesial reaction with bone proliferation at the attachment of the A2 pulley for the digital flexor sheath. The sheath functions to keep the tendons (*flexor digitorum profundus* and *flexor digitorum superficialis*) next to the bone if they are under tension. It has been suggested that the aetiology of enthesophytes is multifactoral, with age, chronic micro-trauma and genetics all playing a role in their development. The second lesion appears to be a sessile exostosis on the distal metaphysis of the phalanx. It is debatable whether exostoses can be considered true neoplasms, with evidence suggesting that localized trauma can produce solitary exostoses, grossly similar to those seen on SKX 9342. The two conditions seem unrelated and for this study, the enthesophytes are considered the primary lesions.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.8 SKX 13476 (Figure 5.44)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: *Paranthropus*

Species: *robustus*

Skeletal element:

Element: 2nd phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: A slightly raised crest of lamellar bone occurs isolated to one side of the phalanx at the site of insertion for the *m. flexor digitorum superficialis*. The lesion is very slight and irregular. Its borders are not sharply defined and appear continuous with the surrounding cortex. The crest extends on a proximodistal axis 1.7mm and is very narrow on a mediolateral axis measuring less than 1mm. The lesion reaches a height of 1mm at its apex. No visible pathological changes occur to the distal or proximal joint surfaces or the remainder of the non-articular area.

Diagnosis:

The lesion is an enthesial reaction with bone proliferating at the area of soft tissue insertion. The *m. flexor digitorum superficialis* is responsible for flexion of the middle phalanges of the fingers at the proximal interphalangeal joint, and under continued action flexion of the metacarpophalangeal joint and wrist (Agur and Dalley, 2005). Enthesophytes have historically been used in physical anthropology to determine behavioural or occupational bone changes. Recent evidence, however, suggests that the aetiology is multifactoral. Compounded or chronic microtrauma may spark the development of enthesophytes. The fact that the condition is isolated to one side is not unusual, based on observations made during this study. Localized trauma may have possibly initiated the bone development. The lesion is relatively small and may be in its early stages of development before the bone changes were obvious on the phalanx bilaterally.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.9 SKW 14147 (Figure 5.45)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: Metacarpal V

Preservation: Near complete

Side: Left

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: On the medial border, at the insertion of the *m. opponens digiti minimi*, there is a raised crest of lamellar bone. The lesion extends the length of the attachment but reaches its greatest height at the distal 1/3 of the metacarpal. It crests at a height of 2mm. The bone growth seems excessive when compared to modern comparative samples, and is therefore considered for this study, to be pathological.

Diagnosis:

The *m. opponens digiti minimi* acts to draw the 5th metatarsal anteriorly and rotates it to bring the digit into opposition with the thumb (Agur and Dalley, 2005). The bone growth at the line of insertion for the muscle suggests an enthesopathy with bone proliferating into the soft tissue resulting in a line of enthesophytes. The aetiology of enthesophytes is generally considered multifactoral with age, wear and tear, and trauma all playing a role in the development of the bone changes. There is also a possible genetic component, or predisposition, to bone forming.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.10 SKX 35439 (Figure 5.46)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: 2nd phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Diaphysis

Description: On the lateral ridge of the second phalanx, isolated to one side is a lamellar crest of bone extending away from the bone surface in a palmar direction. The hypertrophic bone growth is located at the site for insertion of the *m. flexor digitorum superficialis.* The crest runs parallel to the long axis of the phalanx. It is sharp and irregular, 0.8mm in width and 6mm long. At its greatest height the lesion protrudes 3mm from the bone surface. The bone is smooth in texture and the lesion is continuous with the surrounding cortex. The borders are not clearly defined. The lesion is unilateral with the opposite side appearing normal with no excessive bone growth on the lateral ridge. The proximal and distal joint surfaces are also normal.

Diagnosis:

The changes are associated with the entheses and represent enthesophytes. The lesion is associated with the *m. flexor digitorum superficialis* which is

responsible for flexion of the interphalangeal joint of the medial four digits, and for flexion of the metacarpophalangeal joint of the hand (Agur and Dalley, 2005). Enthesophytes are multifactoral in aetiology. They correlate with sex and increasing age. The growth can be stimulated by acute trauma, chronic trauma or chronic microtrauma. On the phalanx the lesion is unilateral and relatively large.

Classification: *Enthesopathy*

Life history interpretation: Asymptomatic.

5.5.11 SKX 38653 (Figure 5.47)

Taxonomic classification:

Order: Primates

Family: Hominidae

Genus: Indeterminate

Species: Indeterminate

Skeletal element:

Element: 2nd phalanx

Preservation: Complete

Side: Indeterminate

Taphonomy:

Minimal

Pathology:

Type: Abnormal bone formation

Distribution of lesion(s) within affected bone: Distal epiphysis / diaphysis

Description: There is a clear area of marginal osteophyte development on the distal interphalangeal joint margin. The abnormal bone growth occurs on ½ of the palmar surface of the joint margin extending in a proximal and palmar direction 2.3mm. The new bone is smooth and continuous with the original cortex. The borders of the lesion are, however, clearly defined by the marginal line. The excessive bone occurs as flat nodules with an irregular shape.

On the lateral ridge, isolated to one side, is a marked area of hypertrophic bone. The bone occurs as a slightly raised oval bump at the site of insertion for the *m. flexor digitorum superficialis*. There is clear asymmetry as the other side is much less pronounced. This lesion is also smooth and rounded and continuous with the surrounding cortex.

Diagnosis:

Marginal osteophytes on the distal joint margin of the phalanx are a feature of osteoarthritis. Osteoarthritis of the hand most commonly involves the distal interphalangeal joints and is characterized by marginal osteophytes (Ortner, 2003). These osteophytes in modern humans are referred to as Heberden's nodes (Aufderheide and Rodriguez-Martin, 1998). Heberden's nodes like other osteophyte formations occur in the chronic stages of osteoarthritis. As with other forms of osteoarthritis, the aetiology is generally considered multifactoral with age, mechanical stress and genetics playing roles in the development of the condition (Weiss and Jurmain, 2007). Trauma to a joint can also result in degenerative changes that mimic OA. The extent of the osteophyte development suggests that mechanical wear and tear of the joint occurred. The bone growth at the insertion for the *m. flexor digitorum superficialis* can be considered an enthesial reaction. The arthritic changes, however, for this study are considered the primary lesions.

Classification: *Joint disease* (with enthesopathy)

Life history interpretation: Pain and altered function.

Bovidae

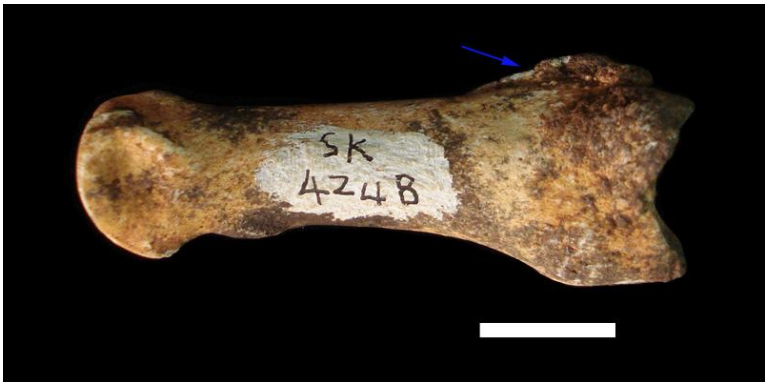


Figure 5.1. *SK 4248* bovid 1st phalanx : lateral view showing the crest of abnormal bone on the medial margin of the proximal metaphysis.

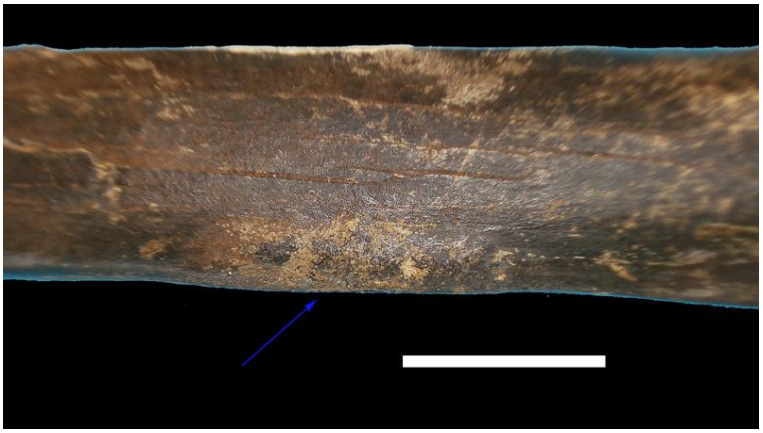


Figure 5.2. *SK 5645* bovid metatarsal: lateral view showing a localized subperiosteal bump of lamellar bone (arrow).

Figure 5.3. *SK 5855* bovid metatarsal: showing a localized subperiosteal bump of bone on the dorsomedial surface (arrow).

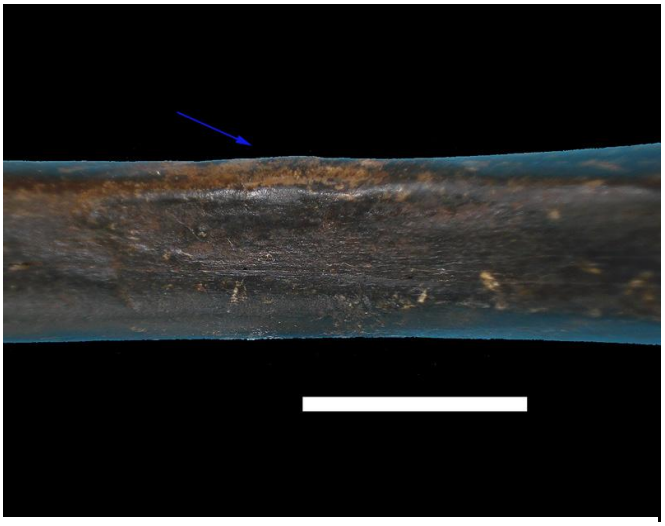


Figure 5.4. *SK 7633* bovid metatarsal: medial view showing a localized subperiosteal bump on the dorsomedial surface of the metatarsal (arrow).

Figure 5.5. *SK 6484* bovid tibia: (A) medial view showing pronounced ridge of bone beginning at the proximal area of taphonomic fracture and narrowing out distally; (B) detailed medial view highlighting the lesion.

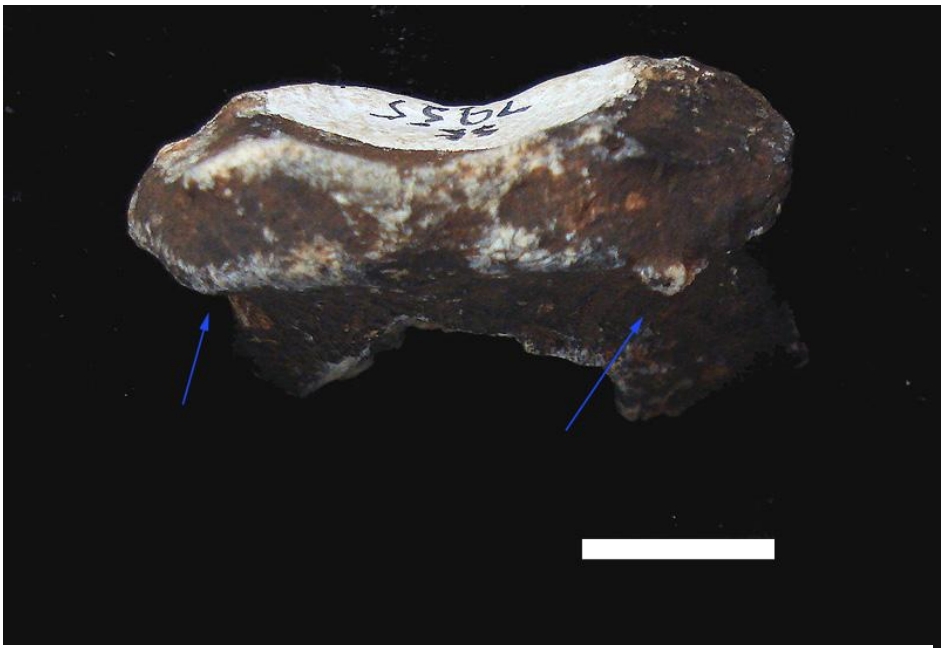


Figure 5.6. *SK 7955* bovid thoracic vertebra: inferior view showing marginal nodules of abnormal bone (arrows).

Figure 5.7. *SK 9514* bovid metacarpal: volar view showing a small bone projection on the distal metaphysis (arrow) in the centre of the two distal condyles.

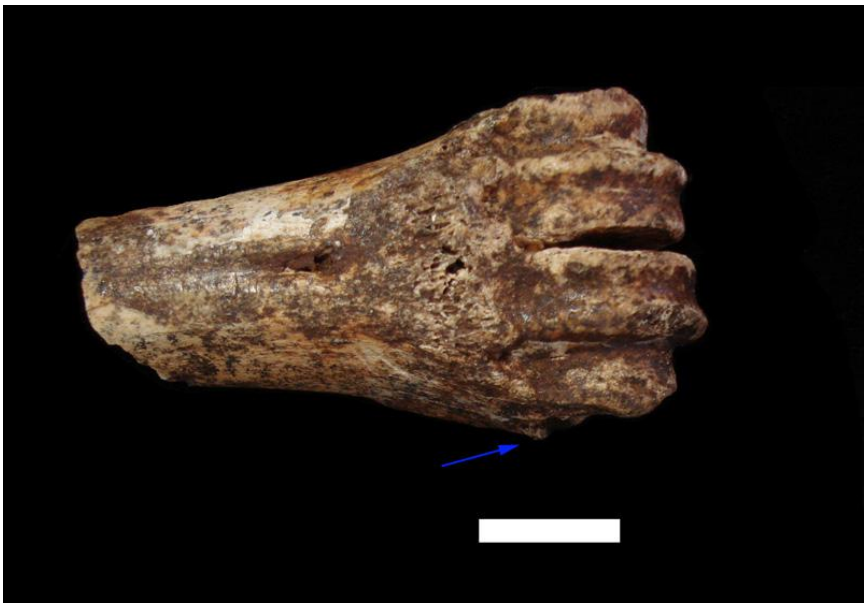


Figure 5.8. *SK 5792* bovid metacarpal: dorsal view showing a small sessile medial exostoses (arrow).

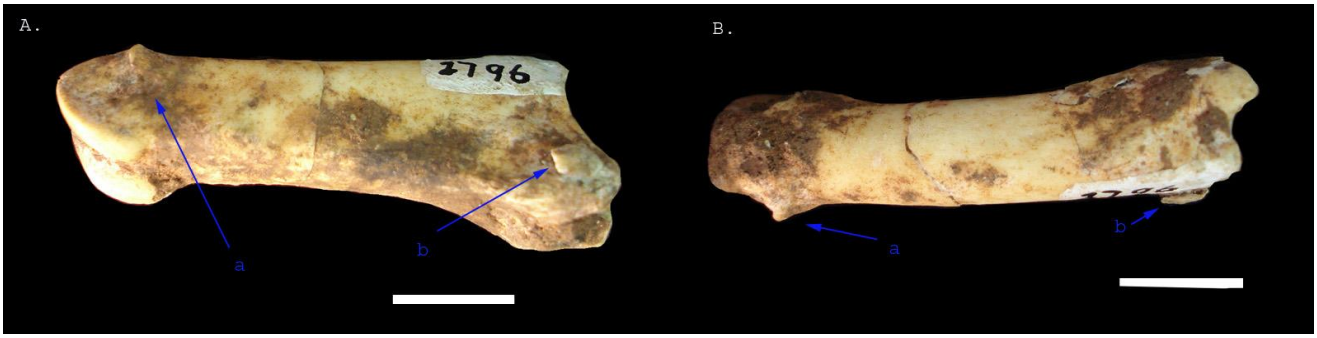


Figure 5.9. *SKX 2796* bovid 1st phalanx: (A.) medial view showing distal exostosis (a) and proximal periarticular lesion (b); (B) dorsal view showing both lesions.

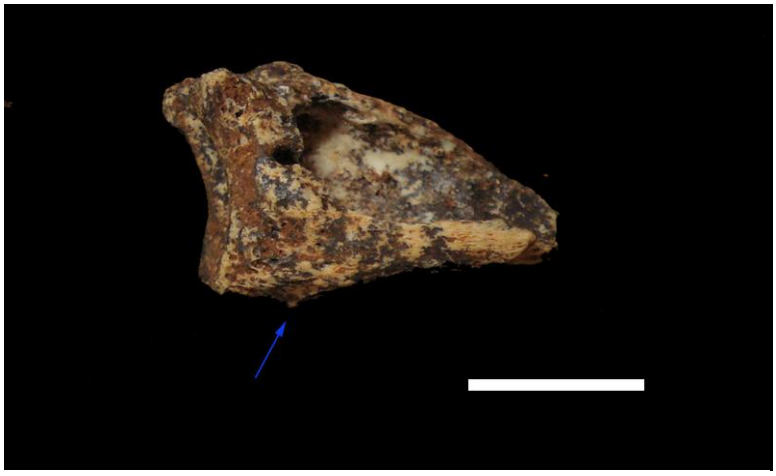


Figure 5.10. *SKX 5796* bovid 1st phalanx: medial view showing small plantar exostosis (arrow).

Figure 5.11. *SKX 37836* bovid 1st phalanx: side view showing enthesophyte development at the site for attachment for the interphalangeal collateral ligament.

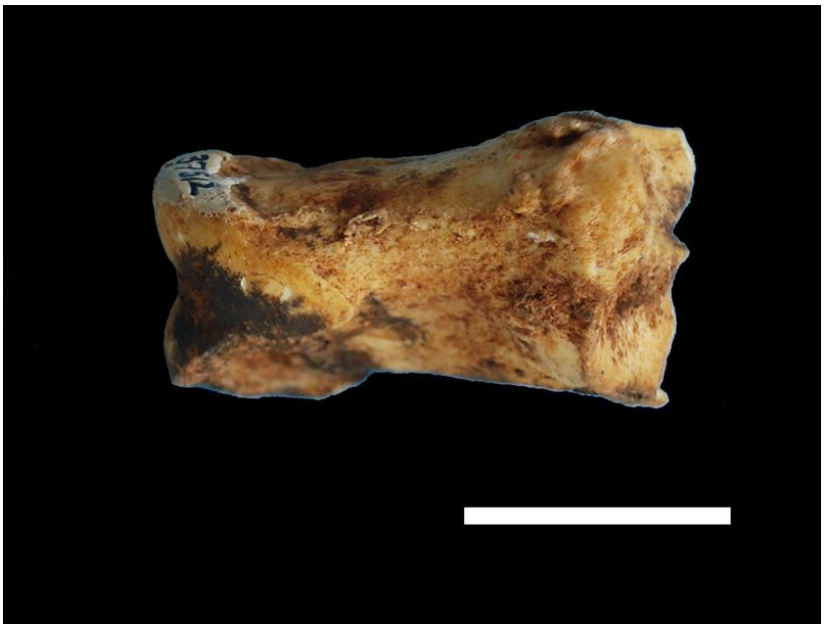


Figure 5.12. *SKX 37512* bovid 2nd phalanx: dorsal view showing nodules of abnormal bone associated with the proximal metaphysis and a similar crest of discrete bone nodules running the length of the dorsal surface and terminating at the distal joint margin.

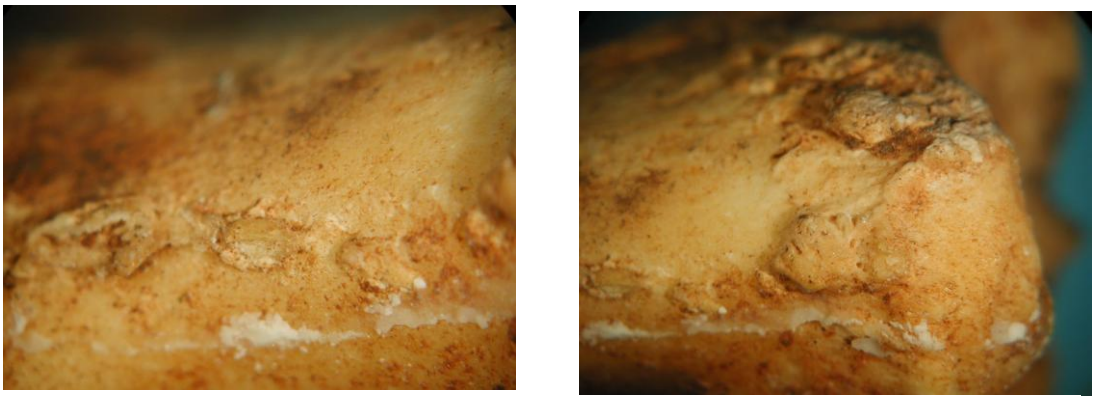


Figure 5.13. *SKX 37512* bovid 2nd phalanx: (left) detailed dorsal view showing a crest of discrete abnormal bone nodules; (right) detailed dorsal view showing bone nodules encircling the proximal metaphysis.

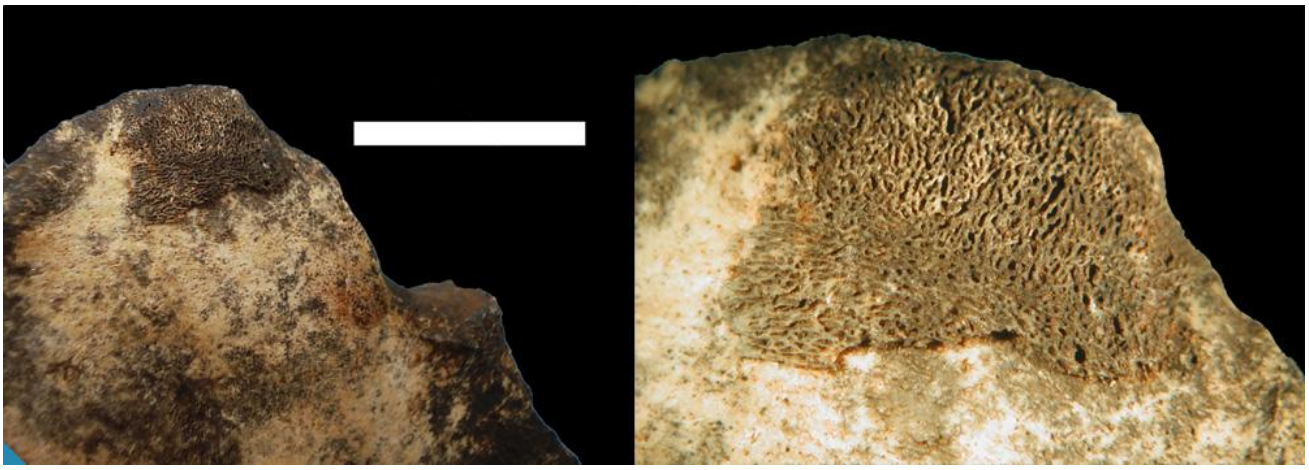


Figure 5.14. *SKX 35132* bovid atlas vertebra: (left) lateral view showing circumscribed area of woven bone on the contra-articular bone surface; (right) detailed lateral view of the lesion; notice the well defined and sharp borders of the reactive bone.

Figure 5.15. *SKX 35132* bovid atlas vertebra: detailed view of the reactive periosteal bone; notice the porous or latticed appearance of the newly formed woven bone.

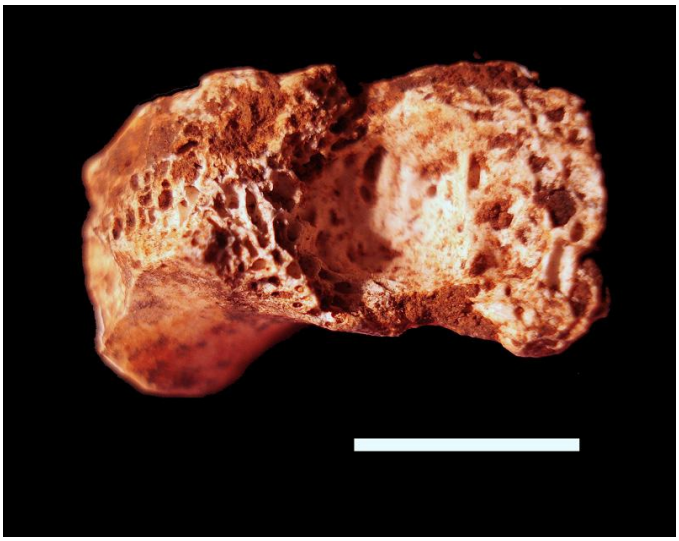


Figure 5.16. *SKX 5043* bovid tarsal indeterminate: cross section at an area of taphonomic fracture showing an oval lytic area, what appears to be an endosteal cyst.

Figure 5.17. *SKX 13569* bovid tarsal indeterminate: view of the articular surface showing the extensive marginal bone growth and small associated lytic foci**.**

Figure 5.18. *SKX 6824* bovid carpal indeterminate: view of the articular surface showing extensive peripheral bone formation covering approximately ½ of the non-articular surface.

Figure 5.19. *SKX 9785* bovid cuneiform: lateral view showing the bone formation associated with the joint periphery; notice the small areas of apparent lytic activity associated with the abnormal bone development**.**

Felidae

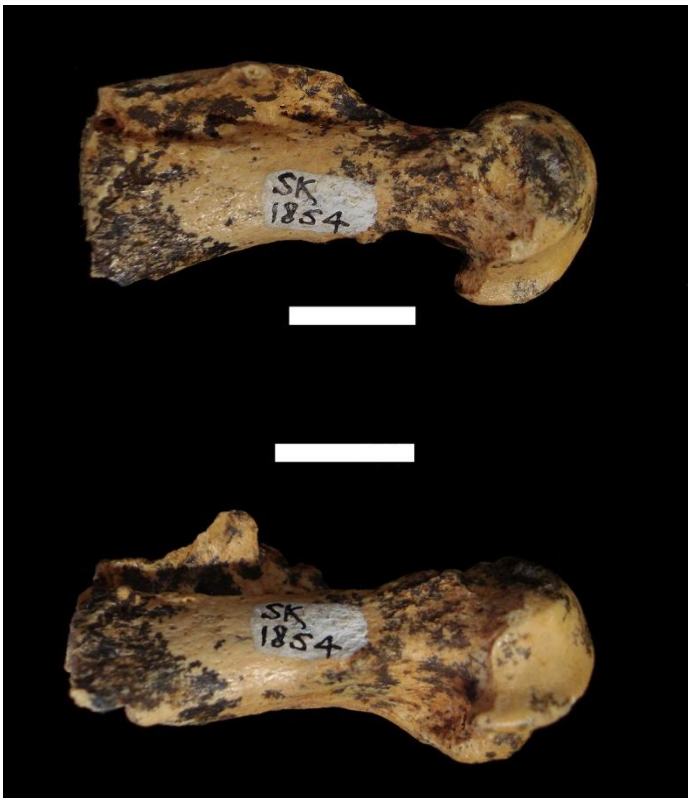


Figure 5.20. *SK 1854* felid metapodial: (top) lateral view showing extensive abnormal bone formation on the dorsal surface extending laterally over the diaphysis; (bottom) lesion seen from the volar view; notice the extent of the abnormal bone growth, 7-8 mm from the natural bone surface.

Figure 5.21. *SK 6747* felid distal metapodial: (left) side view showing abnormal bone formation on the volar surface; (right) volar view showing the area of abnormal bone adjacent to the distal articular margin.

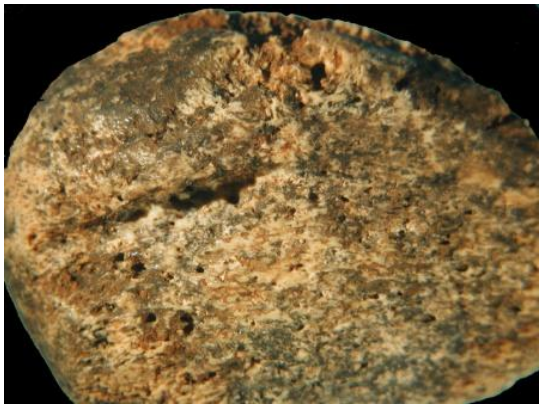


Figure 5.22. *SKX 4203* felid patella: detailed view of the non-articular surface of the patella showing a shallow, well remodelled groove.

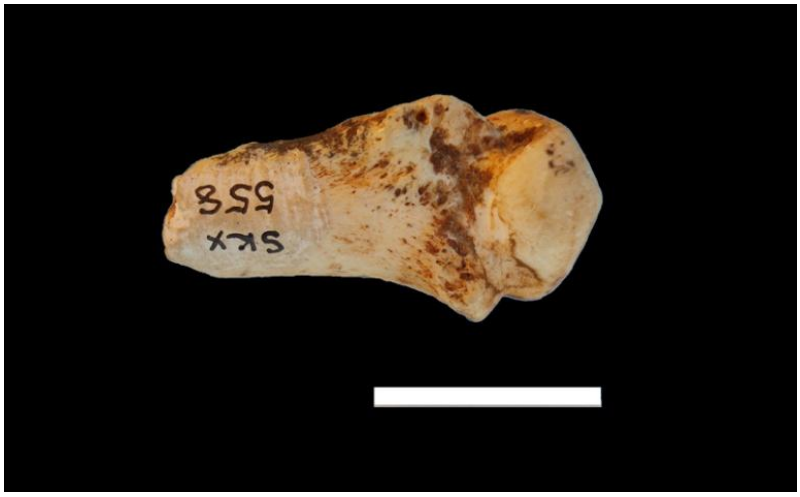


Figure 5.23. *SKX 558* felid distal metapodial: dorsal view showing the remodelling associated with one side of the distal epiphysis; notice that the lesion appears as lamellar bone with smooth margins suggesting a healed condition.

Figure 5.24. *SKX 5471* felid metatarsal: lateral view showing the crest of bone on the plantar surface beginning midshaft and continuing distally to a point of postmortem fracture.

Figure 5.25. *SKX 8484a* felid 2nd phalanx: (left) side view ; (right) volar view showing bone loss and necrosis affecting the entire element.

Figure 5.26. *SKX 8484a* felid 2nd phalanx: (left) dorsal view; (middle) detailed dorsal view; (right) close-up of the lesion highlighting the necrotic appearance of the element.

Canidae

Figure 5.27. *SKX 7994* canid 1st phalanx: dorsal view showing small sessile enthesophyte on distal metaphysis.

Figure 5.28. *SKX 8200* canid metacarpal: plantar view showing a large sessile exostosis on the proximal metaphysis.

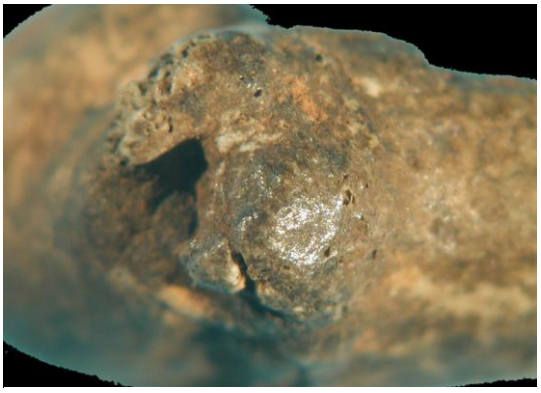


Figure 5.29. *SKX 8200* canid metacarpal: detailed lateral view showing large heavily remodelled exostosis, notice the smooth continuous borders and lamellar bone.

Figure 5.30. *SKX 30774* canid patella: inferior view showing marginal osteophytes.

Figure 5.31. *SKX 39659* canid 1st phalanx: dorsal view showing small sessile exostosis on the proximal metaphysis.

Cercopithecidae

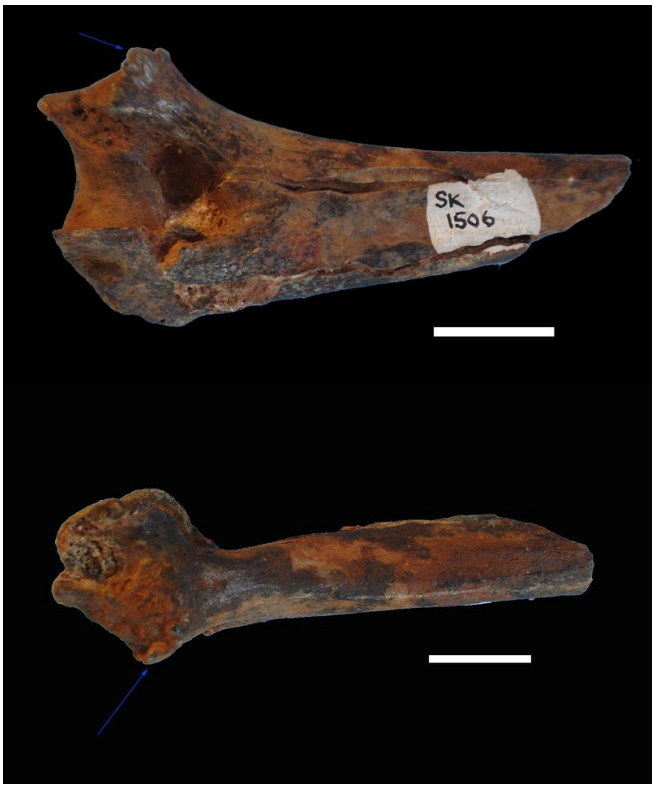


Figure 5.32. *SK 1506* cercopithecid distal humerus: (top) posterior view showing the 'scalloped' abnormal bone formation on the posterior border of the medial epicondyle; (bottom) medial view showing the same lesion; notice the smooth borders and lamellar appearance of the bone growth, indicative of a longstanding condition.

Figure 5.33. *SKX 1867* cercopithecid proximal radius: detailed view of the radial tuberosity; notice the lytic focus in the centre of the tuberosity; the margins of the lesion are smooth and heavily remodelled.

Hominidae

Figure 5.34. *SKX 3342* hominid thoracic vertebra: (left) superior view showing osteophytes on the lateral and anterior margins of the centrum; (above) lateral view showing the extent of the osteophytes on both the superior and inferior joint margins.

Figure 5.35. *SKX 5017* hominid hallucial metatarsal: medial view showing the pointed exostosis on the dorsodistal surface; notice the continuous and smooth borders of the lesion with the surrounding cortex.

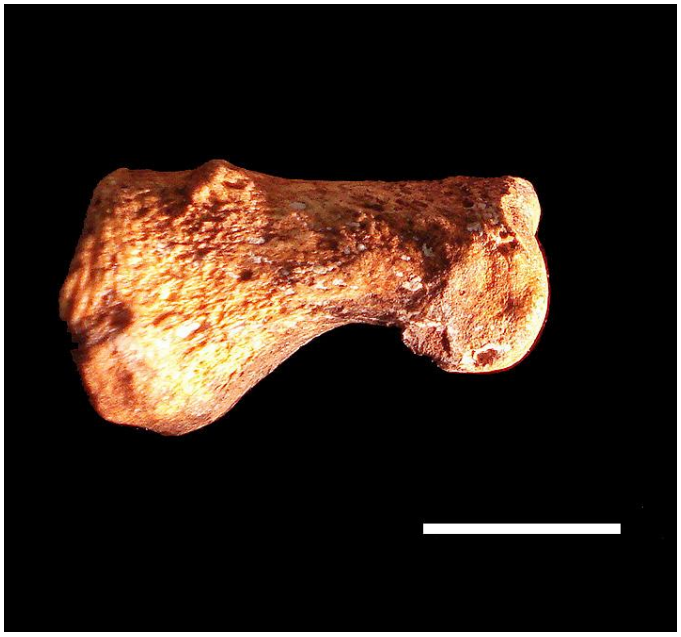


Figure 5.36. *SKX 45690* hominid hallucial 1st phalanx: medial view (rotated slightly) showing a raised bump of bone on the dorsal surface of the phalanx.

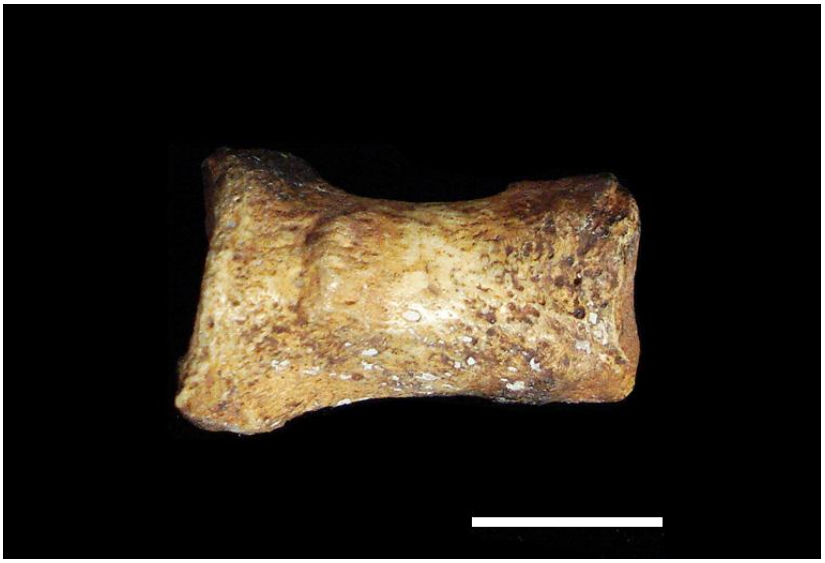


Figure 5.37. *SKX 45690* hominid hallucial 1st phalanx: dorsal view showing the raised bar of bone on the dorsal surface; notice the smooth appearance of the lamellar bone, suggesting a longstanding and well remodelled lesion.

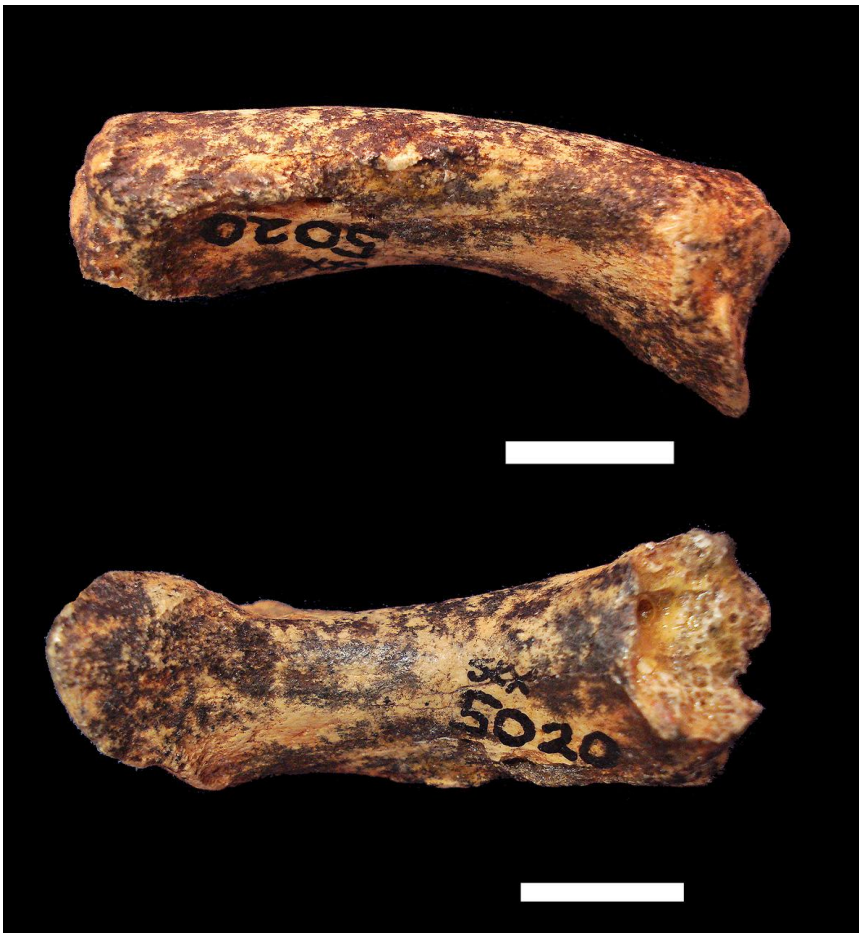


Figure 5.38. *SKX 5020* hominid 1st ray metacarpal: (top) lateral view showing the crest of discrete abnormal bone nodules running the length of the insertion for the *m. oponens pollicis*; (bottom) palmar view showing the same lesion.

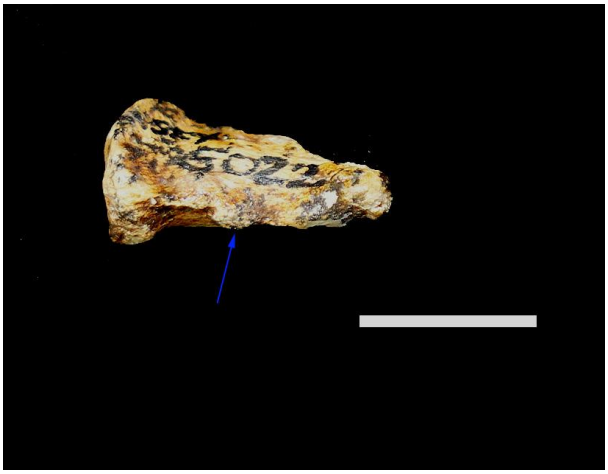


Figure 5.39. *SKX 5022* hominid 2nd phalanx: side view showing a small lamellar bone projection (arrow) at the insertion of the *m. flexor digitorum superficialis*.

Figure 5.40. *SK 7923* hominid metatarsal: (left) volar view showing a large spherical neoplastic growth on the proximal metaphysis; (right) lateral view showing the lesion; notice that the lesion has relatively well defined borders.

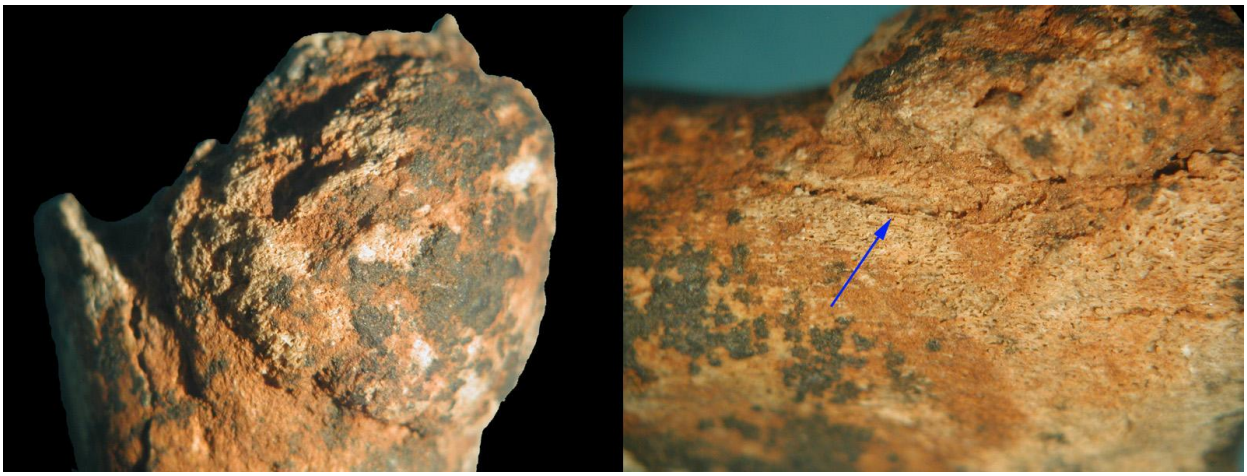


Figure 5.41. *SK 7923* hominid metatarsal: (left) detailed view of the lesion, notice the irregular and rough texture of the surface of the tumour; (right) detailed side view highlighting the reactive bone and clearly defined margins (arrow) at the junction of the lesion and the surrounding cortex.

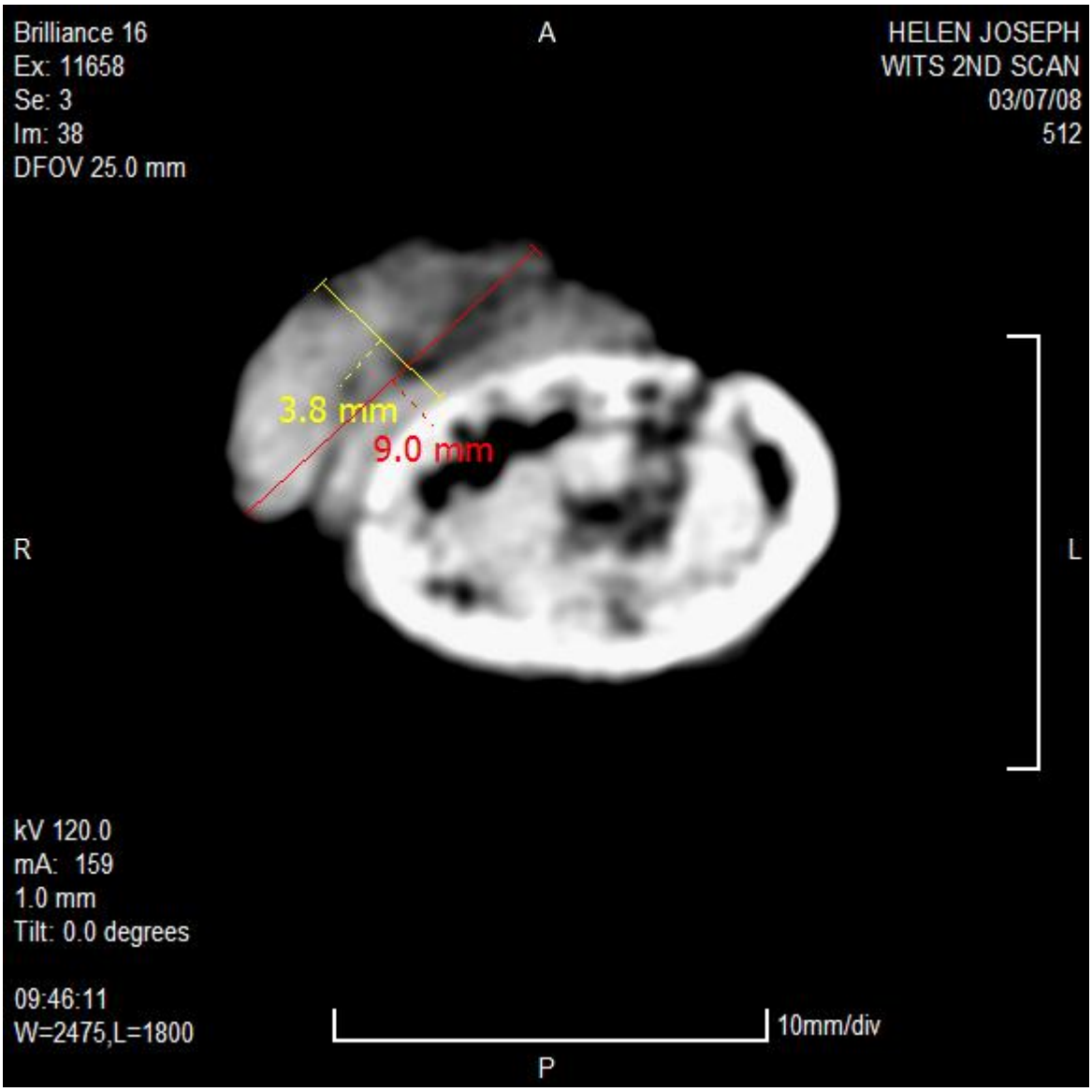


Figure 5.42. CT scan of *SK 7923* hominid metatarsal: notice the density of the lesion, far less dense than the underlying cortical structures. The lesion appears non-invasive and clearly defined, suggesting a benign neoplasm rather than a malignant tumour.

Figure 5.43. *SKX 9342* hominid 1st phalanx: (top) plantar view showing new bone development associated with the insertion for the *fibrous digital sheath,* isolated to one side, on the contralateral distal metaphysis is a small sessile exostosis; (bottom) dorsal view showing the distal exostosis; notice the smooth and continuous borders of the lesion.

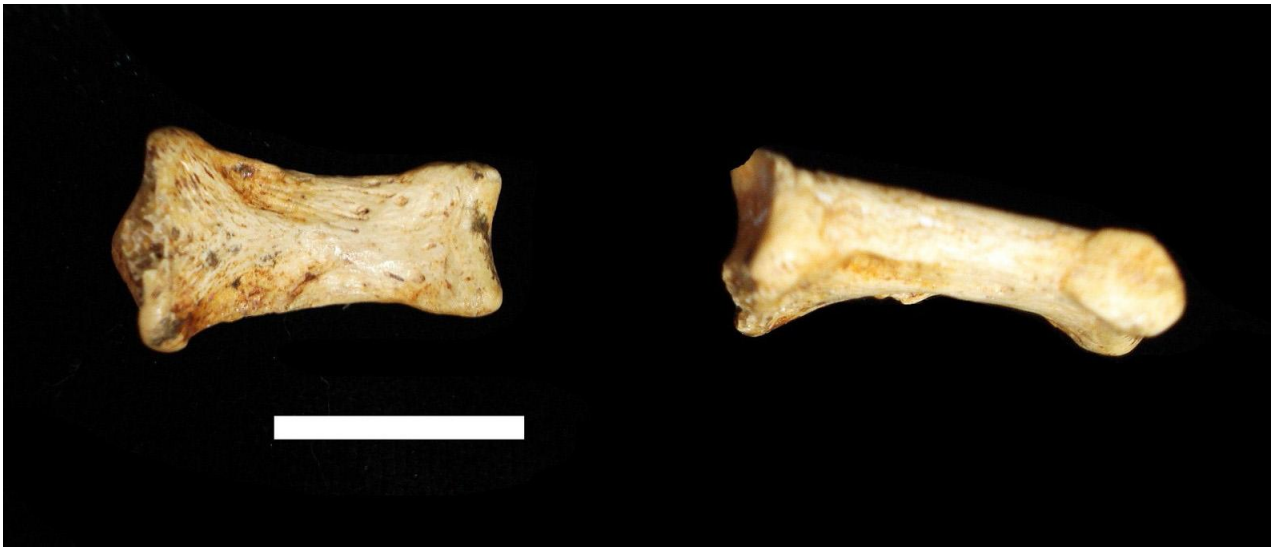


Figure 5.44. *SKX 13476* hominid 2nd phalanx: (left) plantar view; (right) side view showing a small enthesophyte isolated to one side and associated with the insertion for the *m. flexor digitorum superficialis.*

Figure 5.45. *SKX 14147* hominid 5th ray metacarpal: crest of lamellar bone at the area for insertion of the *m. opponens digiti minimi.*

Figure 5.46. *SKX 35439* hominid 2nd phalanx: (left) side view showing a pronounced enthesophyte at the area for insertion of the *m. flexor digitorum superficialis*; (right) plantar view rotated slightly; notice that the lesion is isolated to one side.

Figure 5.47. *SKX 38653* hominid 2nd phalanx: palmar view showing a proliferative bone reaction at the area for insertion of the *m. flexor digitorum superficialis* isolated to one side; also notice the osteophytic lipping on the distal interphalangeal joint surface (Heberden's nodes).

Chapter 6. Results

This chapter serves to describe the results of this research. This chapter will be divided into two parts, A (Cooper's D) and B (Swartkrans). The results will be given for each site independently, firstly regarding classification of the cases of skeletal pathology into broad diagnostic categories: abnormality of development, enthesopathy, infection, inflammation, joint disease, metabolic disorder, neoplasia and trauma.

The next sections present the frequencies of pathology by taxon at order and family level. General comments about the cases of pathology and brief descriptions of pathological specimens are also given under the family subheadings.

Chi-square and randomization test results for goodness-of-fit at family level are given in the sections to follow. The chi-square and *P*-values are given in Tables 6.2 and 6.5. The observed frequency of pathology compared to the expected frequency of pathology at family level is presented graphically at the end of the section.

A minimum number of individuals (MNI_{path}) was calculated for all the pathological fossils at family level, and these results are compared to the comprehensive minimum number of individuals (cMNI) at family level for Cooper's D as calculated by de Ruiter (personal communication, 2005)[1] and for Swartkrans as calculated by de Ruiter (2004).

The final sections present a comparison of the observed and expected frequency of pathology by skeletal section. This is followed by the chi-square and randomization results for goodness-of-fit comparing skeletal sections. The chi-square and *P*-values can be found in Tables 6.4 and 6.7.

[1] Figures have since been adjusted (de Ruiter *et al.*, 2009) following the inclusion of previously unsorted fossils. The survey of pathology did not include the unsorted material; therefore the original data collection is accurate according to the personal communication of 2005. In instances when cMNI figures did not change, the most current reference was used, i.e. (de Ruiter *et al.*, 2009).

A. Cooper's D Results

Following the systematic analysis of the Cooper's D assemblage, 24 fossil specimens were identified as pathological. The pathological specimens were entered into a database with the Cooper's D surveyed sample, totaling 1763 identified specimens. The data were analyzed to determine the frequency of pathology at order and family levels, and the frequency of pathology by relative-size class for bovids and felids. The data were also analyzed to highlight trends regarding pathology by skeletal element (represented graphically in Appendix D). Numbers of pathological specimens are given as a percentage of the NISP at each taxonomic level.

6.1 Classification of the Abnormalities

Following the identification and description of the 24 pathological fossils, the specimens were classed according to pathology type, into broad diagnostic categories. The most fitting classification was determined by assessing the most likely cause for the abnormal morphology. For each specimen, an attempt was made to identify the principal cause for the lesion. For example, if a fossil displayed arthritic changes that appeared to be secondary to trauma (traumatic DJD) the most appropriate classification was trauma. Similarly, if there were minor enthesial changes accompanying a severely arthritic joint structure, joint disease would be the most appropriate classification. This work represents the author's most sincere attempt at classifying pathology in a fragmentary fossil sample, and should serve to highlight that different disease processes are manifest in the fossil assemblage. The categories identified in the Cooper's D fossil sample are: joint disease, enthesopathy and trauma.

Of the 24 pathological fossils, there were 12 cases classified to joint disease. This represents the most common diagnosis of the fossil assemblage and typically involved osteophyte development to the synovial joint periphery. One fossil shows concomitant signs of eburnation, subchondral pitting and sclerosis, which makes it the most

definitive case of osteoarthritis in the sample. One other fossil shows the complete bony ankylosis of two lumbar vertebrae.

Ten fossils show lesions consistent with trauma. These lesions include cases of osteochondrosis/OCD, possible luxation, traumatic ossification following soft tissue injury and solitary exostoses.

Two fossils show signs of enthesopathy. These fossils exhibit minor reactions at entheses in the form of small bony spurs at a site for ligamentous or tendinous attachment. A pie chart was created to present this data graphically and highlight these results (Figure 6.1).

While the 'primary' category diagnosis was used for this study, it is important to note that 7 of the 24 fossils show lesions consistent with multiple categories. Two fossils exhibit arthritis and enthesopathy secondary to trauma, 3 fossils exhibit enthesopathy secondary to trauma, and 2 fossils exhibit enthesopathy secondary to (or in conjunction with) arthropathy.

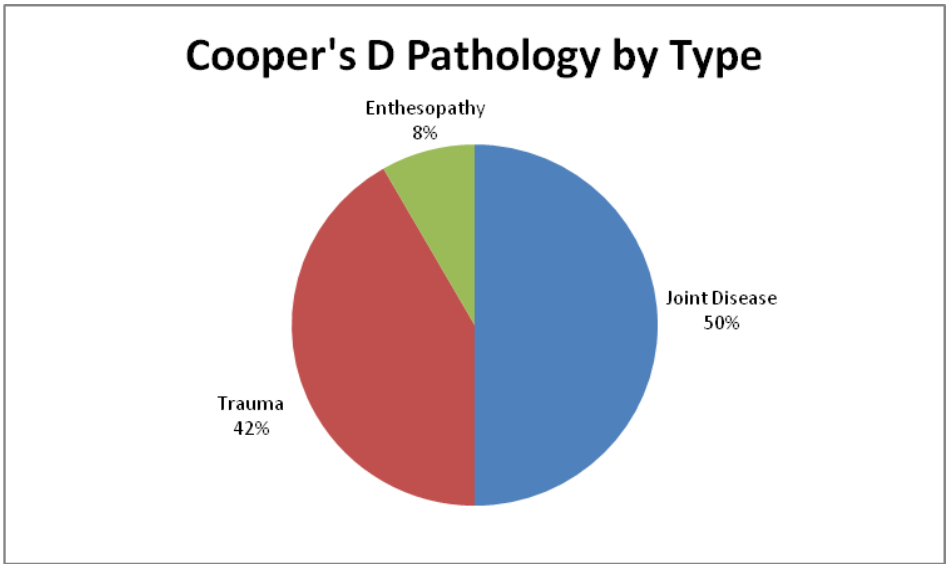


Figure 6.1. Cooper's D graphical representation of pathology by type.

6.2 Frequency of pathology by order

It was determined that there is a clear differential representation of the four orders identified in the Cooper's D assemblage: Perissodactyla, Artiodactyla, Carnivora and Primates (see Table 6.1). Artiodactyls represent the vast majority of the fossil sample, followed by carnivores, primates and perissodactyls. Pathological specimens were found in three of the four orders. Perissodactyl fossils number only 19, accounting for only 1.1% of the total sample. Interestingly, perissodactyls are free of skeletal pathology; however, this may be due to the extremely low representation of perissodactyl postcrania. The remaining orders all include fossils that show gross signs of pathology.

Artiodactyls are, by far the most abundant fossils in the assemblage. Of the 1414 postcranial elements attributed to the Order Artiodactyla, ten (0.7%) were determined to be pathological. Carnivores are the second most abundant order from the Cooper's D assemblage. Of the 223 fossils identified to the Order Carnivora, nine fossils (4.0%) were determined to be pathological. In total, 107 fossils are identified to the Order Primates. Of the 107 fossils, five (4.7%) were determined to be pathological.

Table 6.1. Relative abundance of identifiable skeletal remains from Cooper's D by order.

Order	NISP	Percent of total NISP
Artiodactyla	1414	80
Carnivora	223	13
Perissodactyla	19	1
Primates	107	6
Total	1763	100

6.3 Frequency of pathology by family

6.3.1 Artiodactyls

The Artiodactyla sample consists of bones assigned to animals of the Families Bovidae and Suidae. Cranial and dental material from the Cooper's D sample also includes Giraffidae, however, no postcranial elements attributed to this group have been found to date, and therefore Giraffidae is not included in these results. Bovidae fossils number 1269, accounting for 89.7% of the artiodactyl sample. The remaining 145 bones are attributed to Suidae.

Bovidae

Of the Bovidae sample, only seven specimens (0.6%) were determined to be pathological. Upon analysis of the abnormalities it was noted that all seven fossils show signs of proliferative or degenerative processes associated with joint margins and subchondral bone. Osteophyte development, widening of the articular surfaces or areas of reactive subchondral bone destruction occur to various extents on these seven specimens. All of these lesions are consistent with some form of mild to moderate joint disease, although three of these fossils appear to exhibit osteoarthritic changes that were secondary (sequelae) to trauma. The bovid sample was divided into size-classes following Brain (1981) to allow for an accurate comparison of rates of disease for animals of similar size (see Figure 6.6).

Bovid Class I

Fossils attributed to bovid size Class I number 284. There was no evidence of skeletal pathology.

Bovid Class II

Fossils attributed to bovid size Class II are the most abundant of all bovid fossils, representing 591 of the total bovid sample. Of the 591 specimens, four (0.7%) show signs of skeletal pathology. Three of these fossils, however, appear to articulate and presumably belonged to one individual.

Bovid Class III

Bovid size Class III fossils are the second-most common fossils in the bovid sample. They account for 384 of the total bovid sample, of which three (0.8%) exhibit signs of skeletal pathology.

Bovid Class IV

Bovid size Class IV is the least represented of the bovid sample accounting for only 10 of the bovid fossils. There was no evidence of skeletal pathology.

Suidae

Of the 145 suid fossils, three (2.1%) were determined to be pathological. All three specimens exhibit lesions on extreme distal elements. The lesions are consistent with joint disease, enthesopathy, and trauma.

6.3.2 Carnivores

There are 223 fossils identified to the Order Carnivora. These include specimens of the Felidae, Canidae and Hyaenidae families (Figure 6.7).

Felidae

Felids account for the majority of the Carnivora sample (63.9%). There are 149 specimens ascribed to Felidae. They range in size from large (lion-sized) to small (African wild cat-sized) animals; however only few have been identified further. The designation to family was sufficient for this study, although the specimens were also relatively sized in accordance with the guidelines outlined in Chapter 3. The total Felidae sample contains six specimens (4.0%) with pathology, and includes some of the most striking examples in the pathological sample.

Small felids (Felis lybica; Felis caracal)

There are 33 specimens attributed to small felids. A broad range of different postcranial elements are present, including bones of both juvenile and adult animals. All 33 specimens are free of skeletal pathology.

Medium-sized felids (Panthera pardus)

There are 83 fossil specimens attributed to medium-sized felids. Of the 83 fossils, four (4.8%) were noted as being pathological. There are two cases of vertebral arthropathy, including one case of the complete ankylosis of two lumbar vertebrae. There is one case of traumatic enthesopathy and one case of inflammation as a sequel to trauma.

Large felids (Panthera leo)

Large-sized felids account for 33 of the Felidae sample. Of the 33 fossils, two (6.1%) were noted as pathological. There is one fossil with a small enthesophyte, consistent with a minor prolific enthesopathy. The second fossil appears to indicate trauma, either traumatic avulsion followed by ossification at the enthesis or a penetrating fracture.

Hyaenidae

There are 50 fossils from Cooper's D assigned to the Family Hyaenidae. Several of the fossils have been identified further and attributed to various genera including *Crocuta*, *Proteles*, *Chasmaporthetes* and *Parahyaena* (de Ruiter *et al.*, 2009). This study found the designation to family level to be sufficient. The fossils were included as Hyaenidae and were not relatively sized. Three (6.0%) of the 50 specimens show evidence of pathology. All three fossils exhibit periarticular lesions. Two show changes consistent with arthritic or degenerative joint conditions, ranging from mild to severe. The degenerative changes to CD 9985 have resulted in the complete destruction and remodelling of the articular surface of the terminal phalanx, and appear likely to be a conformation response following trauma, possibly luxation or

subluxation. The depressed pit on the CD 6681, the hyaenid acetabulum, appears to be an osteochondrosis or *osteochondritis dissecans.*

Canidae

Twenty-four postcranial fossils are attributed to the Family Canidae; however, no bones within the Canidae sample show signs of pathology.

6.3.3 Primates

The Order Primates from Cooper's D includes fossils belonging to the Families, Hominidae and Cercopithecidae.

Hominidae

Hominids are primarily represented by craniodental remains and postcranial elements are scarce in the Cooper's D assemblage. Two postcranial elements are, however, assigned to Hominidae indeterminate, and both are pathological. Caution was exercised in the designation of specimens in the assemblage to Hominidae, and some potential hominid specimens are at present simply designated to primate. Including the pathological specimens (Primates cf. Hominidae) and neglecting the others (Primates indeterminate) may increase the risk of overestimation of pathology. This being the case, however, these two elements have been confidently identified to the Family Hominidae, and while each is discussed for its own intrinsic interest, frequencies have been drawn for the Family Hominidae as with all other families.

CD 5288 is a lumbar vertebra with marginal osteophyte development encircling the vertebral body concomitant with the ossification of the anterior ligament. CD 5773 is a thoracic vertebra with osteophyte development around the one preserved costal facet. Both vertebrae display abnormalities typical of spondylosis and vertebral arthritis.

Cercopithecidae

Cercopithecidae are represented by 105 fossils, three (2.9%) display abnormal morphology consistent with pathology. There are two lesions consistent with mild to

moderate arthropathy and one case of circumscribed periosteal inflammation as a sequel to trauma.

6.4 Chi-square and randomization tests for frequencies of pathology at family level.

The chi-square and randomization tests were conducted using a 0.05 significance level. The results yielded an x^2 value of 154, with 7 df, and a subsequent *P*-value of less than 0.0001. The resultant *P*-value is highly significant. Both the chi-square and randomization tests are consistent in these results, which are presented in Table 6.2. There is a significant disagreement between the observed and expected frequencies of pathology for hominids, hyaenids, felids and bovids.

Table 6.2. Chi-square and randomization test results for goodness-of-fit for pathology at family level for Cooper's D.

Family	**Of_p**	**Ef_p**	**diff**	**(Of_p-Ef_p)^2/Ef_p**
Bovidae	7	17.27	-10.27	6.11
Suidae	3	1.97	1.03	0.54
Equidae	0	0.26	-0.26	0.26
Canidae	0	0.33	-0.33	0.33
Felidae	6	2.03	3.97	7.76
Hyaenidae	3	0.68	2.32	7.92
Cercopithecidae	3	1.43	1.57	1.72
Hominidae	2	0.03	1.97	129.36
Total	24	24	0	154

X^2=154, 7 df, P<0.0001
X^2=154, 7 df, P<0.0001*
(*10,000 replicates)

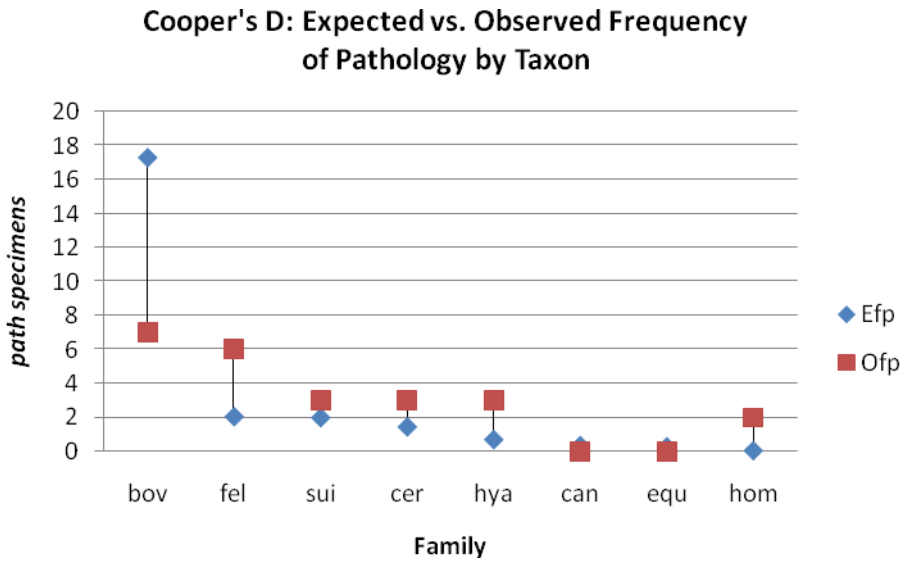


Figure 6.2. Graphical representation of observed frequency of pathology (O*f*p) compared to expected frequency of pathology (E*f*p) by family for Cooper's D.

6.5 Pathology in relation to cMNI

A comprehensive minimum number of individuals (cMNI) was calculated by de Ruiter (personal communication, 2005) for the Cooper's D assemblage using the postcranial and craniodental material derived from the same reduced sample surveyed for this study.

The cases of pathology are given against the cMNI to highlight the relationship between the minimum number of individuals displaying pathology and the minimum number of individuals for each relevant taxonomic grouping. If there was no reason why two elements could not belong to the same individual, they were scored as one individual. This particular quantification of the pathological specimens is subject to many of the same criticisms as an MNI, and the actual number of individuals represented by the pathological remains, could fall anywhere between the calculated minimum number of pathological individuals and the actual number of specimens (Klein and Cruz-Uribe, 1984). For Cooper's D, between 10 and 24 individual animals

are represented by the pathological sample. The cumulative $\%MNI_{path}$ rate is 6.7%. The results are given below (Table 6.3), and these relationships are not definitive as to the actual number of pathological individuals or their relative frequency within the populations.

Table 6.3. The minimum number of pathological individuals (MNI_{path}) compared to the comprehensive minimum number of individuals (cMNI) by family for Cooper's D.

Order	Family	MNI_{path}	cMNI	$\%MNI_{path}$
Artiodactyla	Bovidae (size class I)	0	1	0.0
	Bovidae (size class II)	1	62	1.6
	Bovidae (size class III)	1	20	5.0
	Bovidae (size class IV)	0	5	0.0
	Suidae	2	10	20.0
Carnivora	Felidae (small)	0	5	0.0
	Felidae (medium)	1	7	14.3
	Felidae (large)	1	2	50.0
	Canidae	0	7	0.0
	Hyaenidae	1	7	14.3
Perrisodactyla	Equidae	0	2	0.0
Primates	Cercopithecidae	2	20	10.0
	Hominidae	1	2	50.0
TOTAL		10	150	6.7

6.5.1 Bovids

The pathological bovid elements fall into bovid size classes II and III. Bovid size class II is represented by four specimens. These fossils include a proximal metacarpal, a distal metacarpal, and two fragments of a first phalanx. Several bones clearly

articulate with each other, specifically, the distal metacarpal and the two fragments of the 1st phalanx. The remaining element could not be excluded as belonging to the same individual. Therefore, there is a resultant MNI_{path} of one for pathological specimens falling into bovid size class II.

Bovid size class III is represented by three pathological elements, none of which were matched. These specimens include a thoracic vertebra, a lumbar vertebra, and a proximal metacarpal. There is an MNI_{path} of one for bovid size class III.

6.5.2 Suids

The Suidae pathological specimens include one metacarpal and two first phalanges. Although none of the elements were matched, there is enough of a size discrepancy between the one first phalanx (CD 3992) and the remaining elements, to indicate that the bones came from more than one individual. Therefore an MNI_{path} of two was established. A cMNI of 10 was reached for Suidae, as calculated by de Ruiter (personal communication, 2005), all of which were classified to species as *Metridiochoerus andrewsi*. The extinct pig, *Metridiochoerus andrewsi*, resembled a warthog, but was larger in size, weighing in excess of 120 kg (Brain, 1981).

6.5.3 Felids

Large felids are represented in the sample of pathological remains by a left radius shaft and a right 3rd metacarpal. Both specimens morphologically match modern lion (*Panthera leo*). The specimens are not matched, and may potentially have belonged to the same individual. An MNI_{path} of one was reached for the pathological specimens. The cMNI for *Panthera leo*, as calculated by de Ruiter *et al.*, (2009), was also one. There are, however, two extinct felid genera that have subsequently been found within the Cooper's D assemblage that must be included in this analysis. *Megantereon cultridens* and *Dinofelis* sp. have been identified based on cranial and postcranial elements. Both animals, based on body-size estimations, fall into an intermediate size range, slotting between modern leopards and female lions (Hilton-Barber and Berger,

2002). *Megantereon* (cMNI=1) was included in the large felid cMNI by the author, making a cMNI of two for large felids.

The *Dinofelis* specimens were included as medium-sized by the author after it was noted by de Ruiter (personal communication, 2005) that the elements were 'small' in comparison to the recognized species of *Dinofelis* identified from the Plio-Pleistocene cave sites in the Sterkfontein Valley: *Dinofelis piveteaui* and *Dinofelis barlowi*. The pathological remains attributed to medium-sized felids are a right 5^{th} metatarsal, an axis vertebra, a left patella, and two ankylosed lumbar vertebrae. No elements are matched. An MNI_{path} of one was reached for medium-sized felids. It is important to note that the fossil lumbar vertebrae appeared to morphologically match a modern leopard (*Panthera pardus*), whereas the left patella, axis vertebra, and metatarsal seemed different from *Panthera pardus* or any modern analogue species of similar size, and possibly represent one of the extinct cats. A cMNI of seven was reached for medium-sized felids, including *Panthera pardus*, *Dinofelis* sp., and *Acinonyx jubatus* (de Ruiter *et al.*, 2009).

6.5.4 Hyaenids

The pathological elements attributed to Hyaenidae are a left acetabulum, a second phalanx, and a terminal phalanx. Again, no elements are matched and all may have potentially belonged to one individual. There is a resultant MNI_{path} of one for hyaenids. Elements from the Cooper's D assemblage have been attributed to *Crocuta crocuta*, *Parahyaena brunnea*, *Chasmaporthetes sp.* and *Proteles cristatus*. A cMNI of seven was reached for the family Hyaenidae, as calculated by de Ruiter *et al.*, (2009).

6.5.5 Cercopithecids

The pathological specimens assigned to the Family Cercopithecidae, include a lumbar vertebra, a 1^{st} ray metatarsal, and a 1^{st} ray metacarpal. The 1^{st} ray metacarpal (CD 7267) is relatively large in comparison to the other elements, suggesting that the elements must have belonged to a minimum of two individuals. There is a resultant

MNI_{path} of 2 for cercopithecids. Two species of cercopithecids have been identified from Cooper's D: *Papio hamadryas robinsoni* and *Theropithecus oswaldi*. *Papio hamadryas* still exists today, but is no longer found in southern Africa, even though at one time it was one of the most common baboons found in the Cradle of Humankind (Hilton-Barber and Berger, 2002). *Theropithecus oswaldi* was a large baboon weighing approximately 40 kg (Brain, 1981). A cMNI of 20 was calculated for the Family Cercopithecidae (de Ruiter *et al.*, 2009).

6.5.6 Hominids

Hominids are represented by two pathological elements from Cooper's D. Both, a lumbar and a thoracic vertebra, are similar enough in size not to eliminate the possibility of them representing the same individual. Therefore, an MNI_{path} of one was reached for pathological specimens. A cMNI of two was calculated by de Ruiter (personal communication, 2005) for the species *Paranthropus robustus* (at present, the only species from Cooper's D attributed to the Family Hominidae). Both teeth used in the calculation of the cMNI belonged to juvenile individuals. The vertebrae, however, clearly belonged to one or more, adult individuals. Both vertebrae displayed the complete fusion of the endplates with the vertebral bodies, a condition that occurs in adolescence and young adulthood in modern humans (Steele and Bramblett, 1988). The fossils displayed arthritis and spondylosis, both of which appear to be inevitable concomitants of aging in modern humans. There are early onset, or juvenile, expressions of many forms of joint disease, however vertebral arthritis is more commonly correlated to senescence. Therefore the individual or individuals were presumably adults, old enough to develop degenerative changes to the vertebral column either as the result of long-term mechanical stress or advanced age.

6.6 Pathology by skeletal part

It became apparent that the cases of pathology encountered during the survey of Cooper's primarily occurred on distal-limb elements and vertebrae to the near exclusion of limb long-bones, sternebrae, ribs, scapulae or pelves (see Appendix C). In order to determine whether this represented a real trend and to correct for differential sample sizes, expected frequency of pathology by skeletal section (Ef_p) was calculated assuming pathology to be randomly distributed across the entire sample (see chapter 3). The 14 cases of pathology on the distal-limb elements seemed high, but when calculated against the expected frequency for the distal-limb (Ef_p=13.12), is only slightly higher than expected. The one case of pathology from the forelimb is less than the expected frequency of 2.85. The two cases of pathology from the hindlimb is less than the expected frequency of 3.24. No pathology occurs on the sternal elements which had an expected frequency of 1.62. The seven cases of vertebral pathology is higher than the expected frequency of 3.17. These results are given in Table 6.4.

6.7 Chi-square and randomization tests for frequencies of pathology for skeletal sections.

The chi-square and randomization tests were conducted using the 0.05 significance level. The results yielded an x^2 value of 7.98, with 4 df, and a subsequent *P*-value of 0.09. The *P*-value after the randomization tests was 0.07. The resultant *P*-values were not significant, indicating that there is not a significant difference between the observed and expected frequencies of pathology by skeletal section. These results are presented in Table 6.4.

Table 6.4. Chi-square and randomization test results for goodness-of-fit for pathology by skeletal section.

	Of_p	Ef_p	diff	$(Of_p\text{-}Ef_p)^2/Ef_p$
distal-limb	14	13.12	0.88	0.06
hindlimb	2	3.24	-1.24	0.47
forelimb	1	2.85	-1.85	1.2
sternal	0	1.62	-1.62	1.62
vertebral	7	3.17	3.83	4.63
	24	24	0	7.98

X^2= 7.98, 4 df, P= 0.09
X^2= 7.98, 4 df, P= 0.07*
(10,000 replicates)

B. Swartkrans results

From the Swartkrans assemblage, 40 bones were identified as pathological and formed the basis of this analysis. All pathological fossils were included in the database with the entire Swartkrans assemblage numbering 5384, which was analyzed for frequency of pathology by taxon, and frequency of pathology by relative-size class for bovids and felids. The data were also analyzed to highlight trends regarding pathology by skeletal element (represented graphically in Appendix D).

6.8 Classification of the abnormalities

The 40 elements displaying pathological changes were classified to the following categories: enthesopathy, joint disease, neoplasm and trauma.

Enthesopathy was the most commonly identified type of pathology, with 22 observed cases. Enthesopathy occurs in the bovid, felid, canid, cercopithecid and hominid fossils. It is generally observed as osteophytic expressions (small enthesophytes extending from the bone surface) and less commonly as an osteolytic focus at the enthesis. The frequency of enthesopathy from the Swartkrans assemblage is likely bolstered by the high frequency of the condition among the hominid fossils.

Trauma was the second-most commonly identified type of pathology, with 9 observed cases. Trauma is identified as localized subperiosteal haematomas, traumatic ossification following soft tissue injury, and small solitary exostoses.

There are seven cases of *joint disease*. Joint disease was generally seen as osteophytes at the joint margin, in all cases, lacking any indication of eburnation, subchondral sclerosis or pitting.

Neoplasms (*n.* = 2) are present but rare. One bovid fossil shows signs of what appears to be an endochondral cyst, or cyst-like lesion. The most striking evidence of neoplasm is located on a hominid metatarsal, with what appears to be a poorly organized lump of woven bone at the proximal metaphysis, possibly an osteoid osteoma.

While the 'primary' category diagnosis was used for this study, it is important to note that 3 of the 40 fossils show lesions consistent with multiple categories. Two fossils exhibit enthesopathy secondary to trauma, and one fossil exhibits enthesopathy secondary to arthritis.

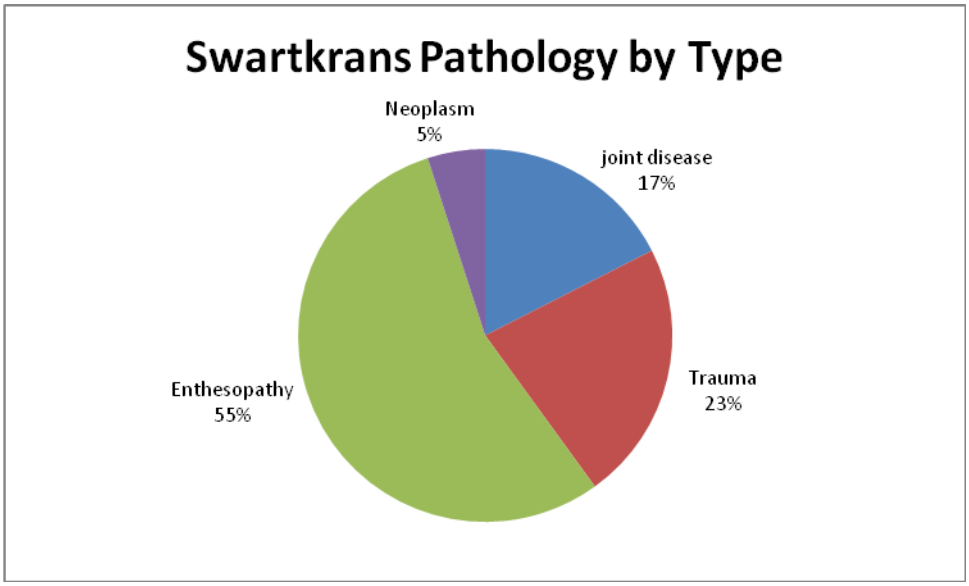


Figure 6.3. Swartkrans graphical representation of pathology by type.

6.9 Frequency of pathology by order

The orders represented by the Swartkrans assemblage include Artiodactyla, Perrisodactyla, Carnivora, Primates and Proboscidea. Artiodactyls dominate the assemblage, accounting for 88.0% of the total NISP. Carnivores account for 7.3% of the assemblage. Perissodactyls account for 0.6%. Primates account for 4.0%. Proboscidea was the least represented of the postcranial faunal assemblage, consisting of only one element and accounting for less than 0.1% of the whole.

A total of 17 (0.4%) of the 4737 artiodactyl fossils display evidence of pathology. Twelve (3.0%) of the 395 carnivore fossils show signs of pathology. Thirteen (6.0%) of the 217 primate fossils show signs of pathology. The remaining two Orders, Proboscidea and Perrisodactyla are free from signs of postcranial pathology, which is possibly due to the overall rarity of the elements, combined accounting for less than 1% of the total analyzed assemblage.

6.10 Frequency of pathology by family

6.10.1 Artiodactyls

Artiodactyls are well represented at the site of Swartkrans. The artiodactyl assemblage is represented by three identified Families: Bovidae, Giraffidae, and Hippopotamidae. Bovidae fossils account for an overwhelming percentage of the artiodactyl specimens, with the remaining two families combined making up less than 0.2% of the total artiodactyl NISP. The subfamily Caprinae is also present but is represented by only one element. It was included with the Family Bovidae. Suidae, although represented by craniodental remains, are not represented in the postcranial assemblage.

Bovidae

Of the 4729 Bovidae specimens sampled for this study, 17 display evidence of skeletal pathology (0.4%). The bovid pathological elements include lesions consistent with arthritis, enthesopathy, trauma and neoplasia. Three pathological tarsals display the same degenerative joint condition. Three metatarsals showed signs of minor trauma in the form subperiosteal haematomas, indicating a possible predisposition for the condition based on the subcutaneous nature of the element. One element shows signs of either an enchondroma or a cyst/cyst-like lesion. The rest of the bovid fossils show lesions representing minor trauma, joint disease or enthesopathy, usually affecting extreme distal-limb elements or vertebrae. The bovid sample was divided into size-classes following Brain (1981) to allow for an accurate comparison of rates of abnormality for animals of similar size.

Bovid Class I

Class I bovids account for 278 of the total Bovidae sample. No evidence of pathology was observed.

Bovid Class II

Class II bovids account for the majority of the bovid sample, numbering 3469. This size class also accounts for 14 of the 17 cases of observed pathology from the artiodactyl sample. Cases of pathology comprise only 0.4% of the Class II NISP.

Bovid Class III

Class III bovids are represented by 906 fossils. Three of the 906 show signs of pathology, establishing a rate of 0.3% of the NISP for this particular size class.

Bovid Class IV

Class IV bovids are the least represented with only 76 fossils assigned to this size class. None of the identified fossils show signs of abnormal morphology or pathology.

6.10.2 Carnivora

The carnivores represented by the Swartkrans assemblage include Canidae, Felidae and Hyaenidae. Canids are the most abundant accounting for 48.4% of the carnivore sample. They are followed by felids, constituting 31.4%, and hyaenids accounting for only 20.2% of the NISP.

Canidae

In total, 191 specimens were assigned to the Canidae family. Most had been previously confidently identified beyond family level to the following genera: *Canis*, *Vulpes* and *Lycaon*. Of the canid sample, four elements (2.1%) show evidence of pathology.

Felidae

The felid specimens number 124. Most were previously identified further to genus, with the following genera represented: *Acinonyx*, *Dinofelis*, *Felis*, *Homotherium*, *Meganterium* and *Panthera*. Six of the 124 fossils show signs of pathology (4.8%). The felid assemblage was further divided into relative size classes and pathology frequency was analyzed in relation to the three established size groups.

Small felids (Felis lybica; Felis caracal)

Small felids are represented by only three specimens. No pathology was observed. This may, however, relate to the overall rarity of the specimens themselves, representing less than 2.4% of the felid NISP.

Medium-sized felids (Panthera pardus)

Medium-sized felids account for the majority of the sample, numbering 104 specimens. This size category also accounts for four of the six cases of observed pathology calculated at 3.8% of the medium size class NISP. Two elements were identified as distal metapodials, one as a proximal metatarsal, and one as a patella. All three of the metapodials show evidence of skeletal remodelling following trauma. A

distal metapodial (SKX 1854) and proximal metatarsal (SKX 5471) show evidence of posttraumatic ossification, *myositis ossificans traumatica*. The lesion typically follows a soft tissue insult whereby avulsion of a soft tissue attachment to a bone creates a haematoma which is ossified over time. SKX 1854 actually shows a very well-healed and relatively long-standing injury with extensive remodelling, suggesting the long-term survival and recovery from the initial injury. The second distal metapodial shows remodelling to the distal metaphysis which has adopted an unusual and flat shape. This pathology appears to be a conformation response following trauma, either partial luxation or subluxation, possibly resulting in an altered gait. The patella (SKX 4203) displays a sunken depression or groove which appears well remodelled and, may represent normal variation rather than a true pathology.

Large felids (Panthera leo)

Large felids are represented by 16 specimens, of which two (12.5%), are pathological. This represents the highest frequency of pathology by felid size category. A distal metapodial and a second phalanx both exhibit evidence of pathology. There is one case of enthesopathy following long-standing and healed trauma. The second lesion occurs on a phalanx (SKX 8484a) and represents one of the most severe cases of pathology encountered during the survey. The phalanx shows diffuse abnormal bone formation and associated bone loss with marked decrease of bone density and possible osteonecrosis. A circulatory disturbance, trauma, or, most likely, an infectious disease resulted in the bone pathology.

Hyaenidae

Hyaenas from the assemblage are identified to genus as follows: *Crocuta*, *Hyaena*, *Parahyaena* and *Proteles*, with the majority being categorized as indeterminate. A total of 80 specimens were surveyed and no signs of skeletal pathology were observed.

6.10.3 Primates

The primate sample is represented by two Families, Cercopithecidae and Hominidae. Cercopithecids comprise the majority of the primate NISP. Hominids, are, nonetheless well represented, accounting for roughly 30.9% of the primate postcranial specimens.

Cercopithecidae

Cercopithecids are represented by the following genera: *Parapapio*, *Papionini* and *Papio*. However most were simply categorized as genus indeterminate. Two of the 150 elements show evidence of skeletal pathology (1.3%). Both elements exhibit different expressions of enthesopathy. The fact that both lesions are enthesopathies and related to the forelimb may indicate the strong possibility that the elements belong to the same individual. Various metabolic and endocrine conditions can manifest in enthesopathy features (Slobodin *et al.,* 2007). Although enthesophytes are likely manifestations of different etiological processes they seem to positively correlate with advancing age (Kalichman *et al.,* 2007). Microtrauma following biomechanical stress may be an initiating event for enthesopathies and it is reasonable to assume that chronic muscle strain and microtrauma may facilitate the development of the condition.

Hominidae

Hominid postcrania are relatively well represented at the site of Swartkrans. Sixty-seven elements have been assigned to the Family Hominidae and most fall within the genera, *Paranthropus* and *Homo*, with only 19 assigned to genus indeterminate. The observed 11 cases of pathology (16.4% of the NISP) by far represent the highest frequency of pathology at family level.

6.11 Chi-square and randomization tests for frequencies of pathology at family level.

The chi-square and randomization tests were conducted using the 0.05 significance level. The results yielded an x^2 value of 264.19, with 9 df, and a subsequent *P*-value of less than 0.0001. The resultant *P*-value is highly significant. Both the chi-square and randomization tests are consistent in these results which are presented in Table 6.5. There is a significant disagreement between the observed and expected frequencies of pathology for hominids, felids and bovids.

Table 6.5. Chi-square and randomization test results for goodness-of-fit for pathology at family level for Swartkrans.

Family	**Of_p**	**Ef_p**	**diff**	**(Of_p-Ef_p)^2/Ef_p**
Bovidae	17	35.14	-18.14	9.36
Giraffidae	0	0.03	-0.03	0.03
Hippopotamidae	0	0.02	-0.02	0.02
Equidae	0	0.25	-0.25	0.25
Elephantidae	0	0.01	-0.01	0.01
Canidae	4	1.42	2.58	4.69
Felidae	6	0.92	5.08	28.05
Hyaenidae	0	0.59	-0.59	0.59
Cercopithecidae	2	1.12	0.88	0.69
Hominidae	11	0.5	10.5	220.5
Total	40	40	0	264.19

X^2=154, 7 df, P<0.0001
X^2=154, 7 df, P<0.0001*
(*10,000 replicates)

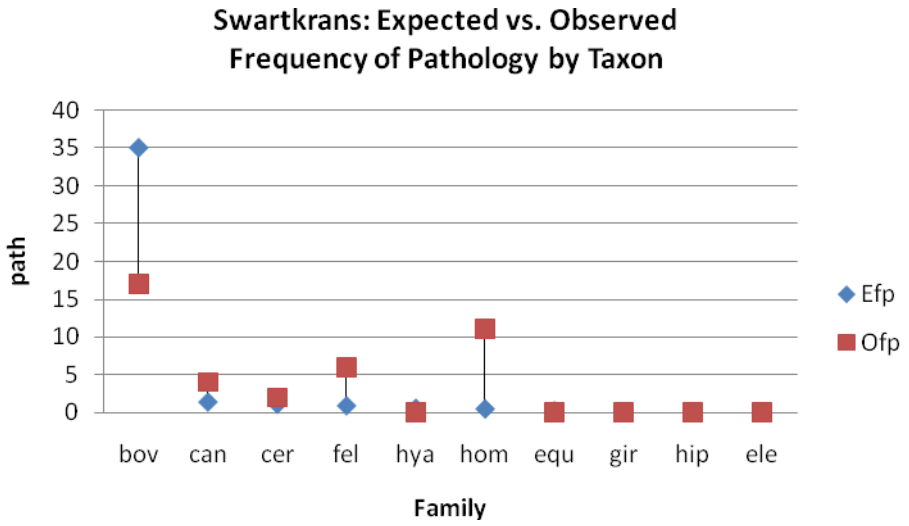


Figure 6.4. Graphical representation of observed frequency of pathology (Of_p) compared to expected frequency of pathology (Ef_p) by family for Swartkrans.

6.12 Pathology in relation to cMNI

A comprehensive minimum number of individuals (cMNI) was calculated by de Ruiter (2004) for the Swartkrans assemblage using the postcranial and craniodental material derived from the same sample surveyed for this study.

As with Coopers D, the cases of pathology are given against the cMNI for the assemblage to highlight the relationship between the minimum number of individuals displaying pathology and the minimum number of individuals for each relevant taxonomic grouping. It should be noted that the actual number of pathological individuals could fall anywhere between the MNI_{path} and the actual number of pathological fossils. For Swartkrans, between 9 and 40 individual animals are represented by the pathological sample. The aggregate $\%MNI_{path}$ rate is 1.9%. In order to make the data more accessible, the results are given in Table 6.6. Further, since Swartkrans is segregated into discrete temporal Members, which probably more

accurately reflect the minimum number of individuals, results regarding MNI_{path} by Member as compared to cMNI by Member are given in Table 6.7.

6.12.1 Bovids

The pathological bovid elements were categorized to size class II and III. For size class II bovids there were four 1st phalanges, a carpal, two tarsals, two distal metacarpals, three proximal metatarsals, a thoracic vertebra and a tibia. The three proximal metatarsals indicate at least two pathological individuals. There is an MNI_{path} of 2 for size class II bovids. All other elements are unmatched and are approximately similar in size. In all likelihood some of the elements belonged to the same individuals due to similar observed pathological lesions. The carpal and tarsals, for instance, displayed the same abnormal bone changes. For bovid size class III, a second phalanx, an atlas vertebra and a cuneiform comprised the pathological sample. All elements are unmatched. There is an MNI_{path} of one for size class III bovids.

When analyzed by Member, the MNI_{path} for size class II bovids increased to four. The MNI_{path} for size class III bovids increased to two. This presents the most accurate MNI_{path} as the Members represent discrete temporal events.

6.12.2 Felids

The extant felid species identified in the cMNI were the leopard (*Panthera pardus*), lion (*Panthera leo*), cheetah (*Acinonyx jubatus*), caracal (*Felis caracal*), serval (*Felis serval*) and African wildcat (*Felis lybica*). The extinct sabre-tooth cat *Megantereon cultridens* also occurs in the assemblage. Other extinct species, specifically *Dinofelis* sp. may be in the assemblage (personal observation) but were not identified in the cMNI. The felids were classified into small, medium and large for the sake of this analysis. None of the small felids show any evidence of gross skeletal pathology. There are four cases of pathology from amongst the medium-sized felid fossils. None of the elements are matched. There is an MNI_{path} of one for medium-sized felids. It is, in fact, probable that one of the metapodials (SK 1854) and the proximal metatarsal (SKX 5471) belong to the same individual, owing to the fact that in both cases the

lesions appear grossly similar, and are considered to be traumatic ossification of soft tissue following trauma. In the large felid size class the pathologies are represented by a distal metapodial and a second phalanx. Neither of these elements are matched and both could possibly come from the same animal. There is an MNI_{path} of one for large felids.

Table 6.6. The minimum number of pathological individuals (MNI_{path}) compared to the comprehensive minimum number of individuals (cMNI) by family for Swartkrans.

Order	Family	MNI_{path}	cMNI	$\%MNI_{path}$
Artiodactyla	Bovidae (size class I)	0	34	0.0
	Bovidae (size class II)	2	154	1.3
	Bovidae (size class III)	1	83	1.2
	Bovidae (size class IV)	0	20	0.0
	Giraffidae	0	3	0.0
	Hippopotamidae	0	3	0.0
Carnivora	Felidae (small)	0	4	0.0
	Felidae (medium)	1	13	7.7
	Felidae (large)	1	3	33.3
	Canidae	1	25	4.0
	Hyaenidae	0	22	0.0
Perrisodactyla	Equidae	0	12	0.0
Primates	Cercopithecidae	1	67	1.5
	Hominidae	2	28	7.1
Proboscidea	Elephantidae	0	3	0.0
TOTAL		9	474	1.9

When analyzed by Member, MNI_{path} for the medium-size felids increased to three, with the Lower Bank, Member 2 and Member 3 each yielding at least one medium-sized felid specimen displaying pathology. The MNI_{path} for large felids remained two as both cases occurred from the Lower Bank (Member 1).

6.12.3 Canids

The two genera included in the cMNI calculation were *Canis* and *Vulpes*. The extant black backed jackal (*Canis mesomelas*) was the predominant canid. The pathological canid fossils include two first phalanges, a metacarpal and a patella. None of the elements are matched. There is an MNI_{path} of one for canids. The pathomorphology of the bones make it difficult to infer whether the fossils belonged to one individual.

When surveyed by Member the MNI_{path} increased from one to two. Pathological canid fossils were identified from both the Lower Bank and Member 3.

6.12.4 Cercopithecids

The cercopithecids included in the cMNI calculation by de Ruiter (2005) were *Cercopithecoides williamsi*, *Papio hamadryas robinsoni*, *Papio (Dinopithecus) ingens* and *Theropithecus oswaldi*. Only two fossils from the family Cercopithecidae display gross skeletal pathology. The two elements are a distal humerus and proximal radius. There is an MNI_{path} of one for cercopithecids. It is in fact likely that the elements come from one individual as they represent the same general area of the forelimb, are of relatively similar size and display a similar pathomorphology, in both instances associated with entheses.

When surveyed by Member, the MNI_{path} of one remained the same. Both pathological elements came from the Lower Bank (Member 1).

6.12.5 Hominids

Two hominid genera have been associated with the Swartkrans Members 1, 2 and 3. The postcranial and craniodental remains have been attributed primarily to the robust

australopithecine, *Paranthropus robustus.* Fossils attributed to the genus *Homo*, without specific assignment, have been identified and quantified as *Homo* sp. A total of 11 postcranial elements attributed to the Family Hominidae display some evidence of gross skeletal pathology and pathological remodelling. A thoracic vertebra, two metatarsals, two metacarpals, one pedal 1st phalanx and five manual 2nd phalanges constitute the pathological sample. The second phalanges were difficult to identify further to side or ray, and even though there are five, they account for an MNI_{path} of one. None of the other elements were matched and on a site aggregate level none can be excluded from belonging to the same individual. All the manual phalanges displayed enthesial reactions and extracortical bone growth associated with the insertion site of the *m. flexor digitorum superficialis*. The vertebra showed signs of vertebral osteophytosis with development of marginal osteophytes surrounding the superior and inferior joint margins of the centrum. The metatarsal and pedal 1st phalanx displayed dorsal proliferative lesions and buttressing of the dorsal surface. All these lesions are indicative of mechanical stress, cumulative microtrauma, advancing age and possibly a genetic predisposition to 'bone forming'. It would be reasonable to conclude that the fossils may have belonged to one individual.

When analyzed by Member the MNI_{path} of one increased to three. The thoracic vertebra (SKX 3342) was found in Member 2 and has been attributed to *Homo* sp. All other elements are from the Lower Bank and Member 3 respectively, and are attributed to *Paranthropus robustus* (*n.* = 5) or Hominidae indeterminate (*n.* = 5). Only two elements are from Member 3. The manual 2nd phalanges (SKX 38653 and SKX 35439) are both Hominidae indeterminate. The remaining eight elements are from the Lower Bank (Member 1). Interestingly, the same manifestation of an enthesopathy, in the form of hypertrophic bone growth at the area for insertion of *m. flexor digitorum superficialis* on the 2nd phalanges is recorded in two temporally discrete Members. This means that at least two hominid individuals (and perhaps as many as five) from the Swartkrans assemblage displayed the same specific pathomorphology.

Table 6.7. The minimum number of pathological individuals (MNI_{path}) compared to the comprehensive minimum number of individuals (cMNI) for Swartkrans by Member.

	Lower Bank: Member 1			Member 2			Member 3		
	MNI_{path}	cMNI	$\%MNI_{path}$	MNI_{path}	cMNI	$\%MNI_{path}$	MNI_{path}	cMNI	$\%MNI_{path}$
Bovidae (size class 1)	0	7	0.0	0	7	0.0	0	20	0.0
Bovidae (size class 2)	1	35	2.9	2	48	4.2	1	71	1.4
Bovidae (size class 3)	1	23	4.4	0	20	0.0	1	40	2.5
Bovidae (size class 4)	0	5	0.0	0	6	0.0	0	9	0.0
Felidae (small)	0	3	0.0	0	0	0.0	0	1	0.0
Felidae (medium)	1	4	25.0	1	2	50.0	1	7	14.3
Felidae (large)	1	2	50.0	0	0	0.0	0	1	0.0
Canidae	1	5	20.0	0	5	0.0	1	15	6.7
Cercopithecidae	1	18	5.6	0	19	0.0	0	30	0.0
Hominidae	1	10	10.0	1	12	8.3	1	6	16.7
Total	7	112	6.3	4	119	3.4	5	200	2.5

6.13 Pathology by skeletal part

In surveying the Swartkrans postcrania it became evident that most pathology occurred on specific elements. In a similar trend as noted from the Coopers sample, distal elements accounted for the overwhelming majority of pathological cases (*n.* = 32). The forelimb (*n.* = 2), hindlimb (*n.* = 3) and vertebrae (*n.* = 3) combined only accounted for 20% of the pathological sample. To correct for differential preservation of the elements, an expected frequency of pathology was calculated assuming a random distribution of pathology across the assemblage. The occurrence of pathology on distal-limb elements (*n.* = 32) is significantly higher than the expected frequency of 22.07. The result of two cases of pathology occurring on forelimb elements is lower than the expected frequency of 8.70. The three cases of pathology occurring on hindlimb elements is lower than the expected 6.31. No cases of pathology are noted on sternal elements, and as this was the least represented of the skeletal sections the expected frequency was only 0.39. The result of three observed cases of pathology on vertebral elements is slightly higher than the expected result of 2.53. These results are included in Table 6.7.

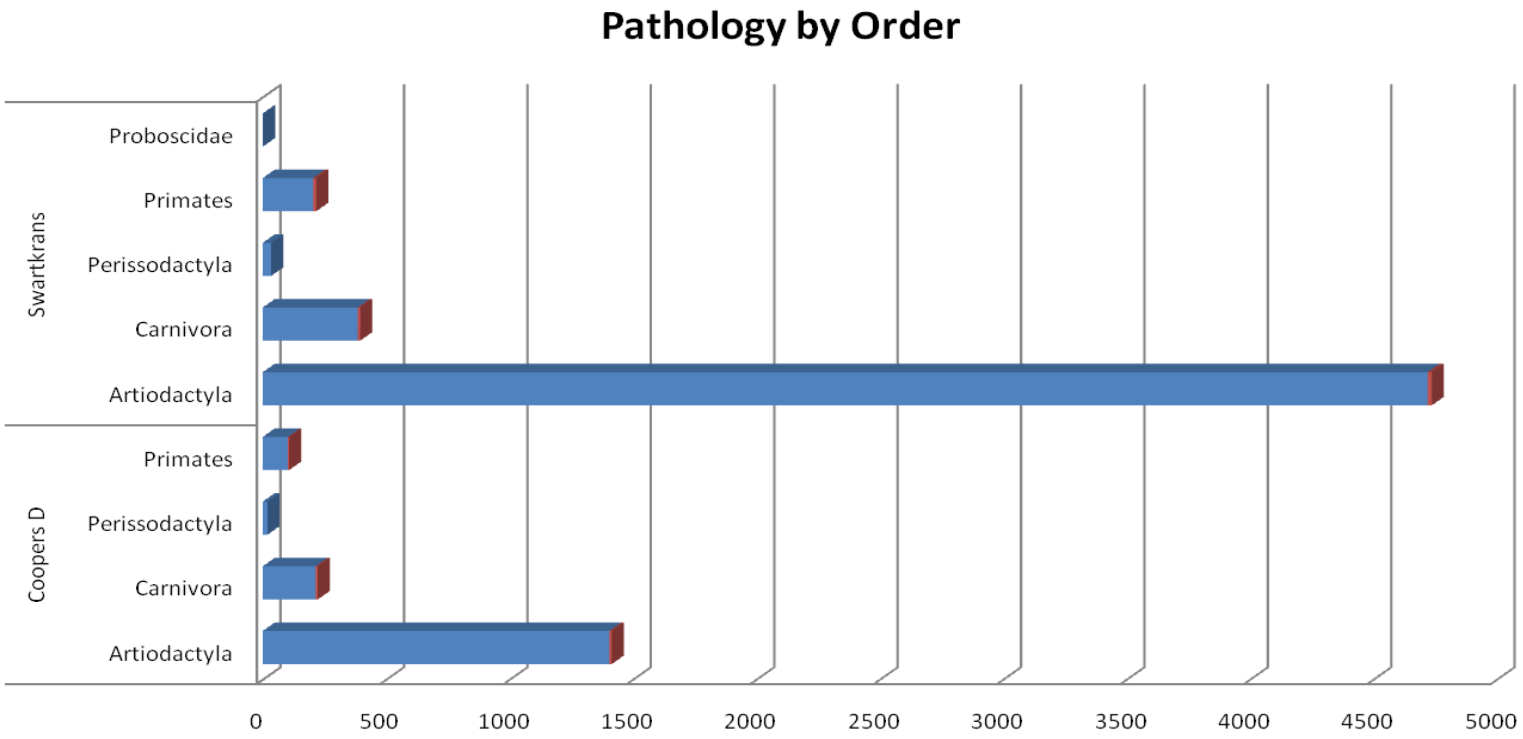
6.14 Chi-square and randomization tests for frequencies of pathology for skeletal sections.

The chi-square and randomization tests were conducted using the 0.05 significance level. The results yielded an x^2 value of 7.98, with 4 df, and a subsequent *P*-value of 0.02. The *P*-value after the randomization tests is 0.03. The resultant *P*-values are significant. These results are presented in Table 6.8.

Table 6.8. Chi-square and randomization test results for goodness-of-fit for pathology by skeletal section.

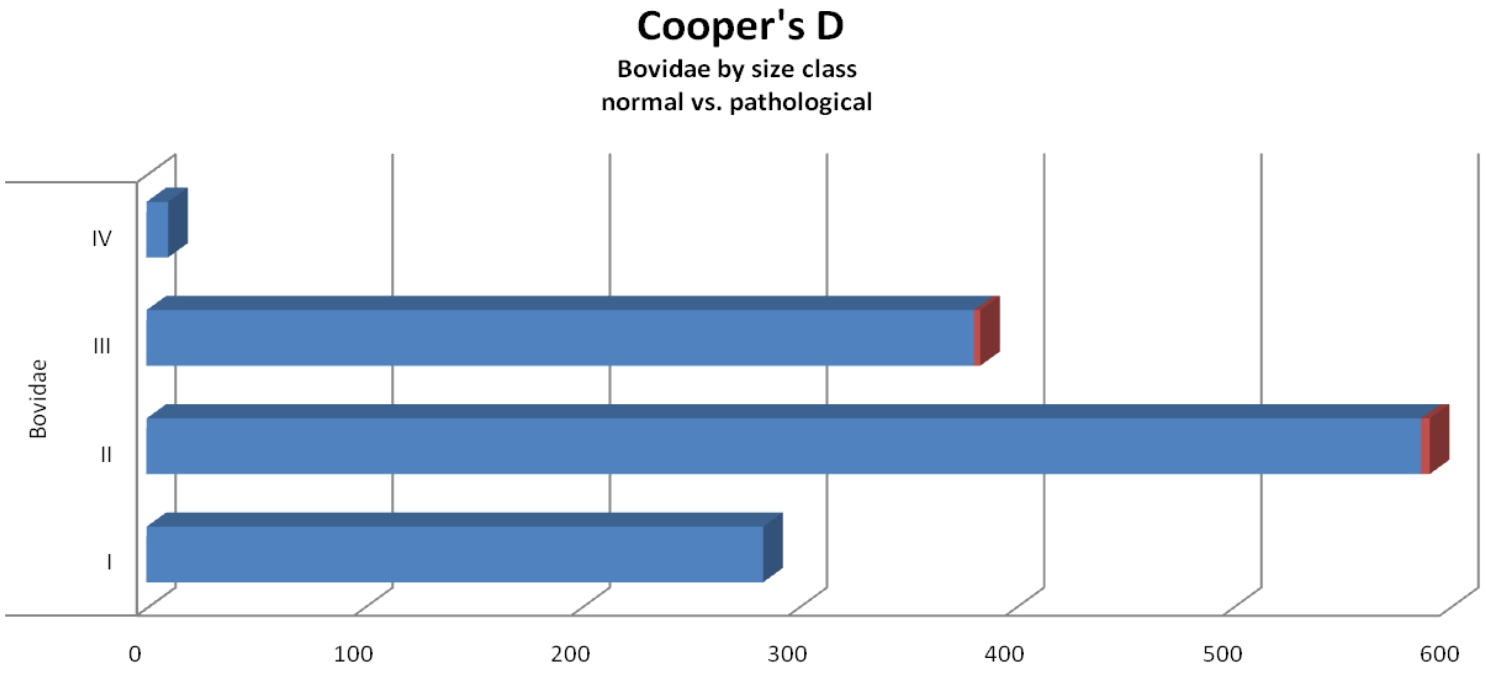
Skeletal section	Of_p	Ef_p	diff	(Of_p-Ef_p)^2/Ef_p
distal-limb	32	22.07	9.95	4.47
hindlimb	2	8.7	-6.69	5.16
forelimb	3	6.31	-3.35	1.74
sternal	0	0.39	-0.38	0.39
vertebral	3	2.53	0.47	0.09
total	40	40	0	11.85

X^2= 11.85, 4 df, P= 0.02
X^2= 11.85, 4 df, P= 0.03*
*(10,000 replicates)



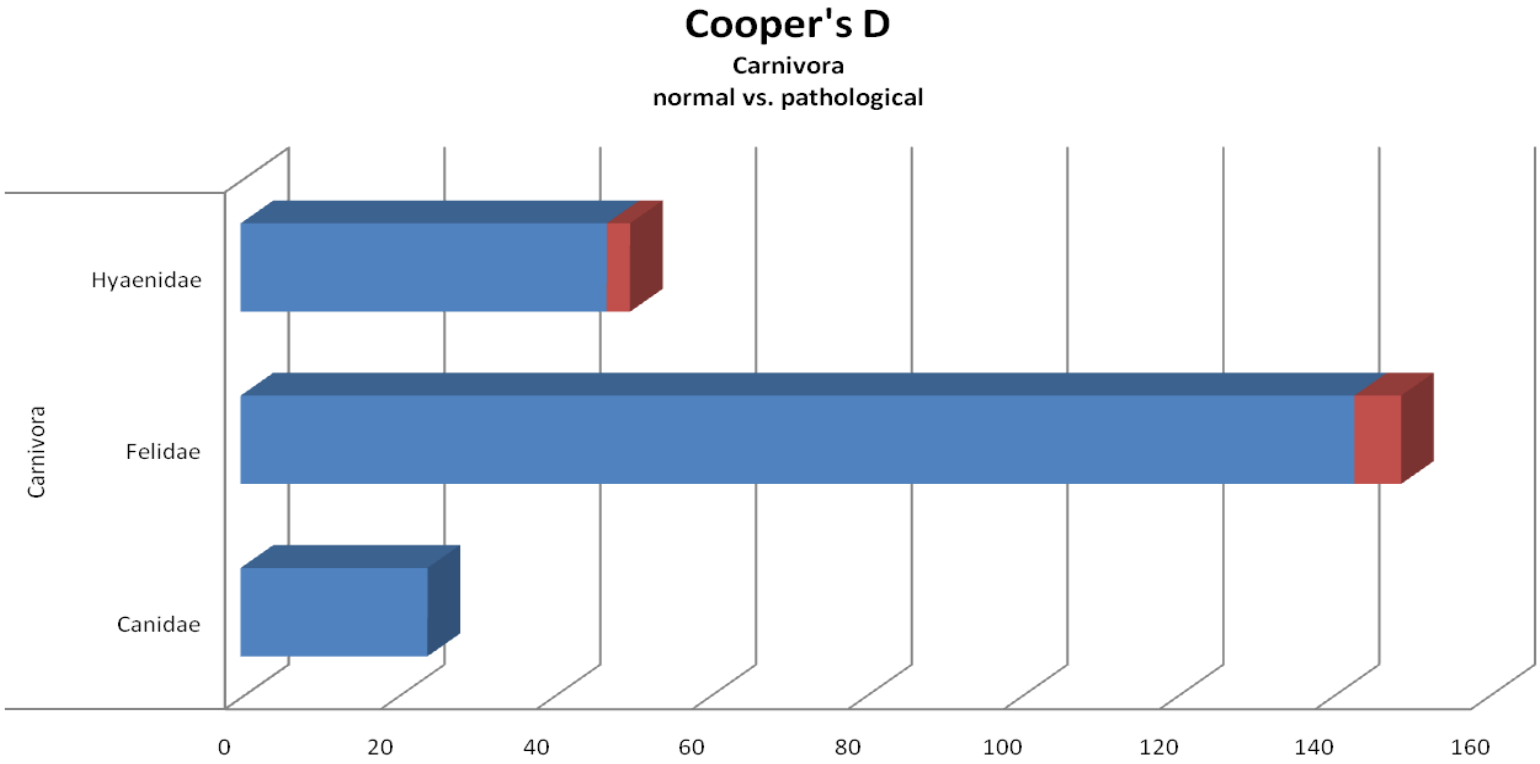
	Coopers D				Swartkrans				
	Artiodactyla	Carnivora	Perissodactyla	Primates	Artiodactyla	Carnivora	Perissodactyla	Primates	Proboscidae
Normal	1404	214	19	102	4720	385	34	204	1
Pathological	10	9		5	17	10		13	

Figure 6.5. NISP by order for Cooper's D and Swartkrans, comparing normal and pathological fossils.



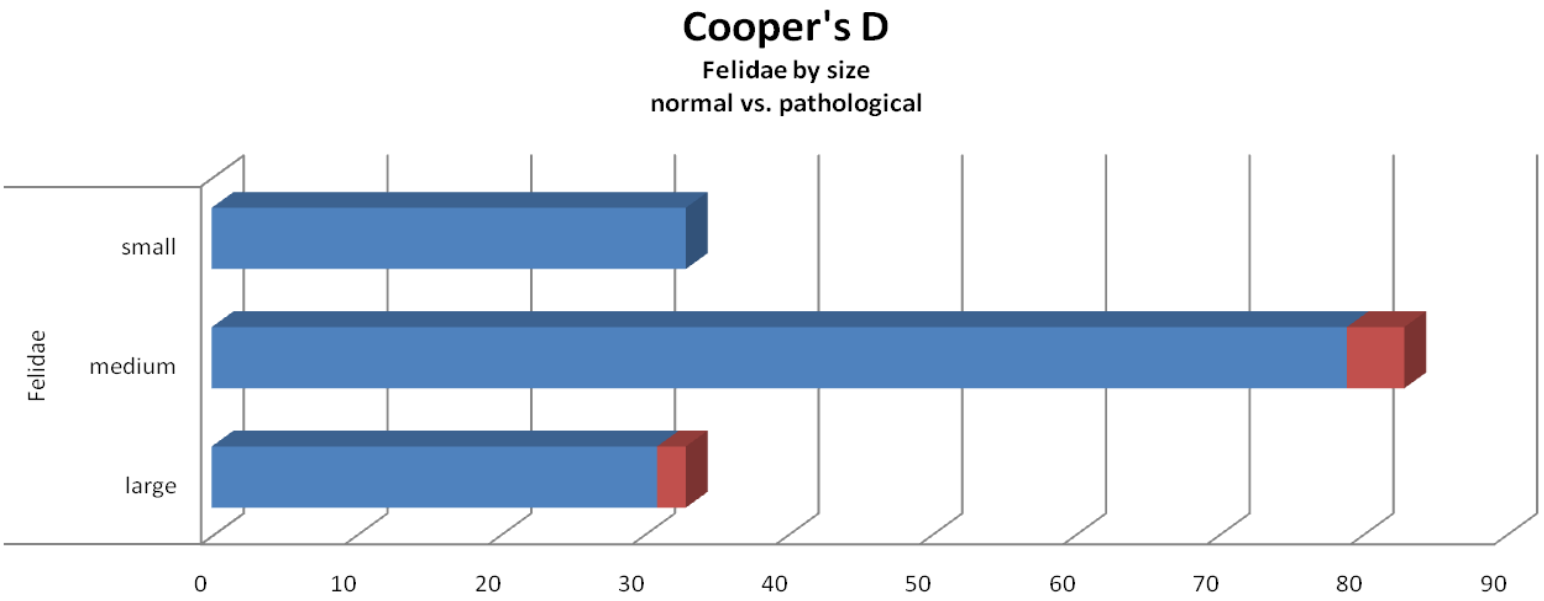
	Bovidae			
	I	II	III	IV
Normal	284	587	381	10
Pathological		4	3	

Figure 6.6. Cooper's D bovid NISP by size class, comparing normal and pathological fossils.



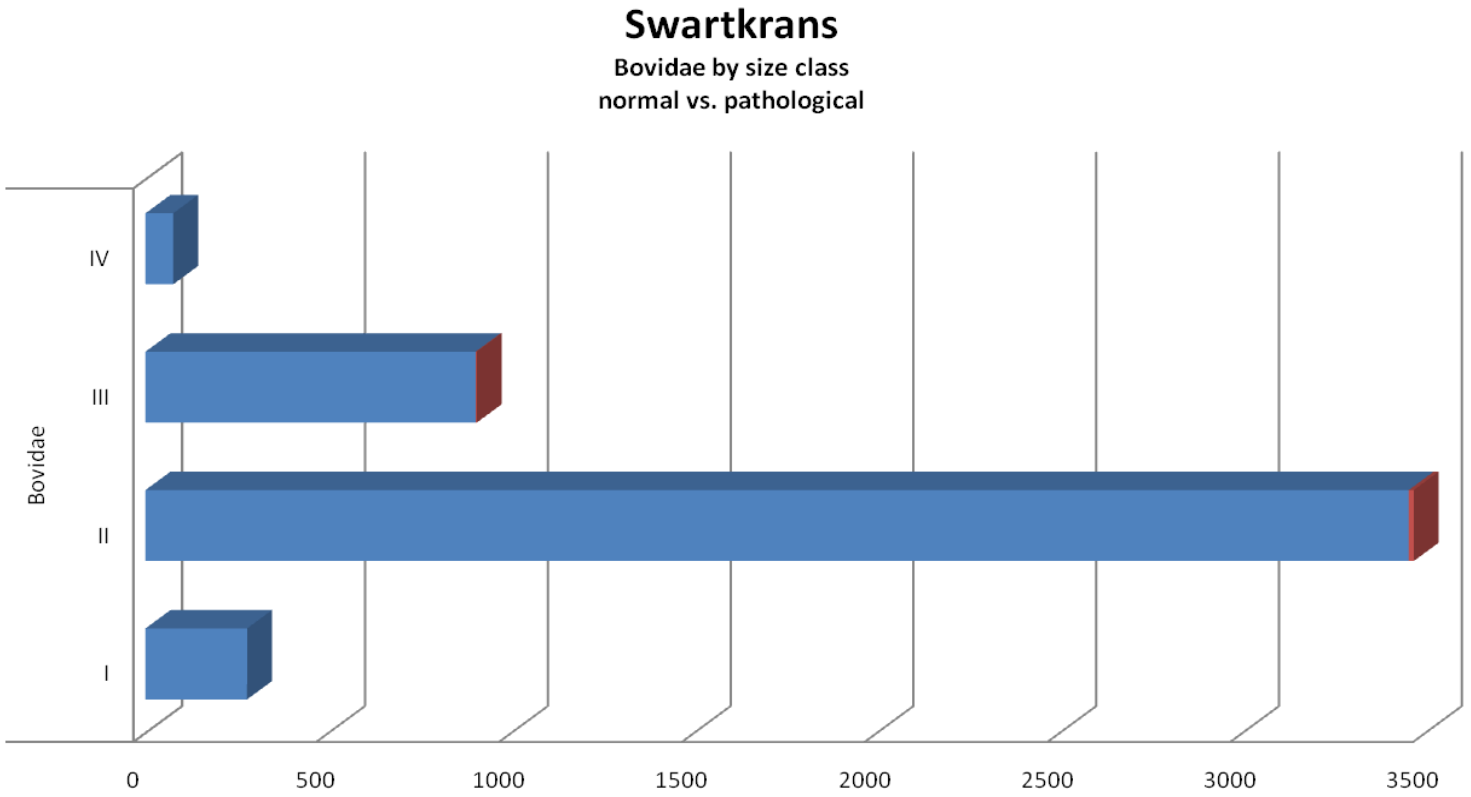
	Carnivora		
	Canidae	Felidae	Hyaenidae
Normal	24	143	47
Pathological		6	3

Figure 6.7. Cooper's D carnivore NISP by family, comparing normal and pathological fossils.



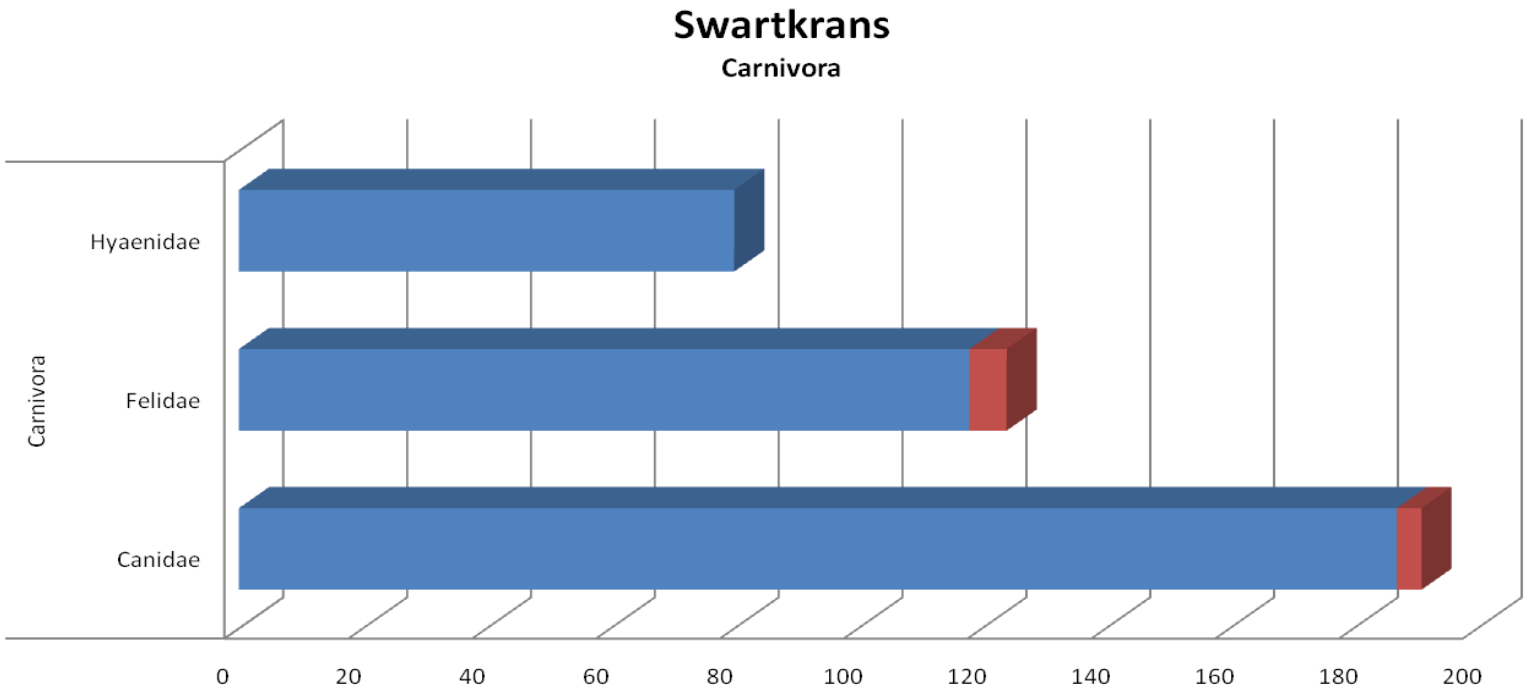
	Felidae		
	large	medium	small
Normal	31	79	33
Pathological	2	4	

Figure 6.8. Cooper's D felid NISP by relative size, comparing normal and pathological fossils.



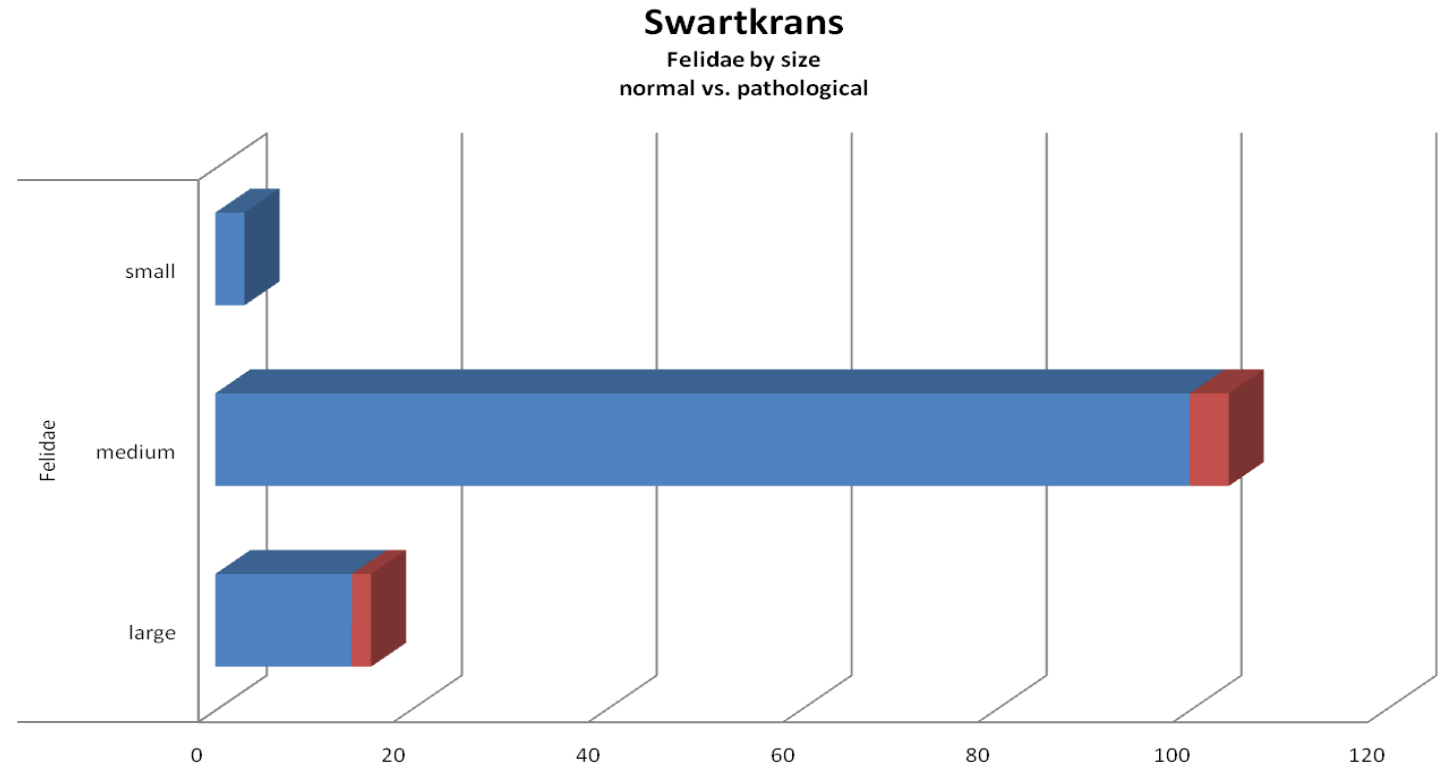
	Bovidae			
	I	II	III	IV
Normal	278	3455	903	76
Pathological		14	3	

Figure 6.9. Swartkrans bovid NISP by size class, comparing normal and pathological fossils.



	Carnivora		
	Canidae	Felidae	Hyaenidae
Normal	187	118	80
Pathological	4	6	

Figure 6.10. Swartkrans carnivore NISP by family, comparing normal and pathological fossils.



	Felidae		
	large	medium	small
Normal	14	100	3
Pathological	2	4	

Figure 6.11. Swartkrans felid NISP by relative size, comparing normal and pathological fossils.

Chapter 7. Discussion

This thesis proposed that there were unrecognized and unidentified pathological fossils from the hominid-bearing sites of Cooper's D and Swartkrans. It set out to test this hypothesis, determine the types of pathology present, and the frequencies with which they occurred by taxon and skeletal element. A baseline and mental template of normal gross morphology was established by extensive examination of modern comparative skeletal collections and utilization of reference literature. A set of criteria was defined for distinguishing abnormal bone from normal dry bone. Through the systematic evaluation of the fossil assemblages, and the analyses of abnormal specimens in the context of a well sorted sample of over 7000 identified specimens, several conclusions were reached, and will be discussed below under the six main hypotheses proposed.

7.1 There is evidence of pathology, in the form of skeletal abnormalities, present in the Cooper's D and Swartkrans fossil assemblages.

Principally, it was confirmed that skeletal pathology was present from both Plio-Pleistocene-aged sites. A total of 24 cases of pathology was noted from the Cooper's D assemblage and 40 cases of pathology from the Swartkrans assemblage. This confirms the original hypothesis that skeletal evidence of disease was present in the fossil samples and fundamentally confirms the proposition that disease, trauma and degenerative changes affected the Plio-Pleistocene faunas from this geographical area.

At the end of the study, it was concluded that at a site aggregate level, pathology represents an expectedly small percentage of both assemblages; 1.4% for Cooper's D and 0.7% for Swartkrans. When analyzed, however, specifically by taxa at order and family levels, the percentage of pathological specimens by taxon reaches frequencies

of as high as 6.0% (Hyaenidae) and 4.8% (Felidae) for Cooper's D and Swartkrans respectively. The Hominidae frequency of pathology is elevated for both sites and will be discussed in section 7.6.

7.2 Different types of pathology occur in the fossil assemblages.

The project hinged on the expectation that fossil pathology was similar enough to modern comparative pathology and archaeological palaeopathology to allow for description and ultimately classification of Plio-Pleistocene-aged abnormalities. A number of taphonomic agents have affected the assemblages, resulting in a largely disarticulated and highly fragmentary sample. These conditions are not ideal for palaeopathological analysis. Fragmentation and differential preservation will tend to bias the type and number of elements preserved, obscure pathological lesions and deprive a researcher of the one thing so crucial to palaeopathological studies, a complete or semi-complete skeleton. From the fossil samples, descriptions and analyses are based upon isolated bones, and it is impossible to see the affect of the pathological condition throughout the skeleton. It is like seeing one small part of the big picture, and is analogous to describing a room by looking through a keyhole. Identification of a pathological condition to a precise or definitive aetiology is very difficult without a complete skeleton. This is especially true when dealing with disease conditions, metabolic disturbances, or congenital defects. Only if the lesion is pathognomic of a specific condition can a more precise diagnosis be attempted.

Ultimately, however, it was concluded that the skeletal manifestations of the fossil pathological conditions are similar enough to those seen in modern comparative faunal collections, modern human reference collections, palaeopathology reference literature, and modern clinical literature to allow the recognition, description and broad classification of the abnormalities.

It was further discovered that clearly different pathomorphologies were present. Through broad category diagnosis it became evident that several different types of pathological disturbance were responsible for the lesions identified in the sample. Therefore the individual fossils show exposure to a range of pathological conditions in the Plio-Pleistocene from this geographical area.

The majority of lesions found from both sites were periarticular, referring to lesions associated with joint structures, whether directly affecting the joint or not. Most of the other lesions occurred at the entheses, sites for muscle or ligament attachment, with few lesions occurring on the diaphyses or non-articular surfaces.

Enthesopathy was the most commonly encountered pathology in the fossil sample (*n.* = 24). Many of the pathological fossils showed signs of bone changes associated with sites of soft tissue attachment. There are two distinct enthesopathies, one involving bone growth (osteophytic) and one involving bone loss (osteolytic) (Mariotti *et al.*, 2004). In both instances, the abnormal changes at the site for ligamentous or tendinous attachment are considered multifactoral in aetiology. Advanced age seems to be the best correlate for the development of enthesophytes, while age appears to reverse correlate with osteolytic enthesopathy, with maximum expression in young adults and a decrease with advancing age (ibid). Both distinct forms of enthesopathy were recognized in the fossil sample and they occurred across taxonomic categories, in high frequency among the hominids from Swartkrans (see 7.5).

There were 19 pathologies categorized to trauma. Many bones showed evidence of primary injury with inflammation and bone callus formation. Post-traumatic ossification (*myositis ossificans traumatica*) was also recognized on several fossils, indicating an indirect trauma, with soft tissue damage resulting in bone proliferation. The classic exostoses, noted in the suids from Cooper's D, are ambiguous in their aetiology. In the past they have been considered a neoplasm with bone growth occurring at the metaphyses forming bony projections. Several authors (e.g. Hwang and Park, 1991; Mintzer *et al.*, 1994; Mohanna *et al.*, 2000; Kontogeorgakos *et al.*,

2007) have made convincing arguments for a traumatic aetiology for solitary exostoses. Exostoses are therefore, increasingly considered as developmental lesions, rather than true neoplasms (Murphey *et al.,* 2000), with a possible traumatic aetiology that can occur in any bone that undergoes endochondral ossification. *Osteochondrosis dissecans* was identified as subchondral cystic lesions in a hyaenid acetabulum and bovid distal metapodial.

There were also 19 pathologies categorized to joint disease. Since the majority of lesions in the sample were associated with joints and joint structures, it was an expected result that many of the fossils had an arthritic component to the abnormal morphology. Joint disease can occur when any condition disrupts the normal metabolism of the joint (Ubelaker, 1994). The most common form of joint disease in archaeological remains is osteoarthritis. Joint use, age, and long term mechanical stress are considered the predominant factors involved in the development of osteoarthritis, although there may also be a genetic component (Solomon, 2001). Only one fossil meets the requirements for classification to the more specific diagnosis of osteoarthritis. Most of the cases of joint disease in the fossil sample consist of marginal osteophyte development. Calcium pyrophosphate deposition disease (CPDD) may be present in the Cooper's hominid thoracic vertebra, CD 5773 (Bruce Rothschild, personal communication, 2005). CPDD is a form of joint disease, resulting from the deposition of calcium pyrophosphate dehydrate in and around joints. Although the pathogenesis of CPDD is not entirely understood, aging or genetic factors may be responsible for the development of the condition (Saadeh, 2004). Spondylosis, although not truly arthritis, is included in this category. Spondylosis occurs in vertebrae, in areas where the annular ligament is stressed, and appears to be an inevitable concomitant of aging.

Neoplastic lesions were recognized, but were exceedingly rare. Only two fossils show evidence of neoplasia. Several of the lesions encountered in the fossil assemblages appear to have an inflammatory component; however, the abnormalities can generally be attributed more confidently to other conditions (e.g. trauma). This was not

unexpected since, skeletal tissue responds to injury, like most other tissues of the body, through inflammation (Anderson and Scotti, 1976).

Clearly, a range of pathological conditions influenced individual fitness during the Plio-Pleistocene from this geographical area. The gross morphology of the abnormalities varied enough to indicate that trauma, degenerative joint changes, enthesopathy as well as several different disease processes were present in the living animal community. This indicates that individuals were subjected to a wide range of pathological conditions. The specific pathogeneses of the lesions could be even more diverse than the broad classifications would suggest; with a multitude of disease conditions potentially having caused the abnormalities encountered in the fossil fauna.

Most of the pathology represents minor degenerative changes or minor trauma, and it is worth noting the conspicuous absence of several conditions. No major long bone fractures were noted. There are no cases of diffuse or severe infectious disease. There are also no obvious developmental or congenital anomalies.

The absence of specific conditions may result from certain *in vivo* potentials for bias. Some pathological processes that can target the skeleton in the chronic stages often end the individual's life while still in the acute phase, leading to no observable skeletal response (Roberts and Manchester, 2005). This would almost certainly lead to the underestimation of the cases of an observable pathological condition. The conditions we do see on dry bone tissue are generally the result of long-term survival of the individual whereby the pathogen had moderate to low virulence, and there was an effective immune response (Ortner, 2003). A pathogen with high virulence, or an individual's weak immune response to an infectious agent, might lead to mortality before the disease reaches the stage of leaving an impress on the skeleton. The same would hold true for severe trauma which may result in the inability to evade predation and almost immediate mortality.

Palaeopathological surveys are subject not only to the *in vivo* potentials for bias discussed above, but taphonomic potentials for bias as well. Bones weakened either post- or perimortem, as well as bones that were thin, were observably more brittle and less likely to survive deposition, diagenesis and excavation unscathed and complete. Differential survivorship of bones has for many years been correlated to bone structural density (Brain, 1969b; Brain, 1981; Lyman, 1994). Certain elements from the fossil sites were rarely found complete, possibly because of their biological architecture and structural density, and preservation trends seemed to correlate with published bone mineral density values (see Lyman, 1994). Presumably the same would hold true for bones, not inherently weak or thin, but rather, weakened or thinned by pathological processes. Diseases that affect bone metabolism, for instance, can result in decreased bone mass and can counteract bone preservation (Bartosiewicz, 2008). If a bone was subject to a pathological disturbance that rarefied it to such an extent as to make it brittle (decreasing its structural density), that element would have had little chance at survival to deposition, and beyond that, to excavation. Therefore the potential arises that osteolysis would be underestimated by palaeopathological surveys of sites that are subject to the rigors of carnivore accumulation, deposition, fossilization and excavation.

It should be noted that the most striking and severe gross expression of pathology from both sites occurred on medium- to large-sized felids. For Cooper's D, the ankylosis of the lumbar vertebrae may indicate long-term survival following a relatively severe physical injury. Similarly, the enthesopathy on the felid patella represents a possible traumatic ossification following an avulsion injury to the knee. This indicates long-term survival following an insult that would have certainly affected the individual's gait. The trauma and osteonecrosis of the 2nd phalanx and traumatic ossification seen on the metapodials also indicate largely longstanding and/or healed pathological insults. Medium and large felids, therefore, represented the most severe and diverse manifestations of pathology from both assemblages. This indicates that of the different taxa, large carnivores are perhaps most likely to survive an initial pathological insult long enough to exhibit an osteogenic response. This suggests

further, that large carnivores may present the most accurate portrayal of the range and severity of pathology suffered by a faunal community during the Plio-Pleistocene.

7.3 Pathology occurs on different skeletal elements at a similar frequency.

The postcranial skeleton was divided into two skeletal groups: the axial and appendicular skeleton, to determine if there were gross trends regarding the location of skeletal abnormalities within the body. It was discovered that pathology was not limited to one particular part of the skeleton, with both axial and appendicular elements showing signs of pathology across taxa at the order level.

The skeleton was further divided into five skeletal sections: vertebral, sternal, hindlimb, forelimb and distal-limb. The chi-square and randomization tests for goodness-of-fit for Cooper's D revealed no significant difference between the expected and observed frequency of pathology by skeletal section at the 0.05 significance level. Interestingly, the results for Swartkrans revealed a significant difference at the 0.05 level. For Swartkrans, there is a significant disagreement between the expected and observed frequency of pathology for the distal- and hindlimb. This can perhaps be explained by the high frequency of distal-limb pathology for the Swartkrans hominids. The specific results will be discussed below.

Distal-limb elements were by far the most common postcranial elements in both assemblages. The relatively high rate of survival of the distal-limb elements is not an uncommon phenomenon (Brain, 1981; Schick *et al.,* 1989; Lyman, 1994) and is likely a result of the high structural density of the distal elements. The distal-limb elements also represented the majority of cases of pathology for both Cooper's D (*n.* = 14) and Swartkrans (*n.* = 32). Bartosiewicz (2008) recognizes that there is a direct and non-proportionate correlation between the frequency of pathology and the size of the assemblage. It would stand to reason that the frequency of pathology in the distal-limb

may be disproportionately high simply due to the frequency of distal-limb elements. Upon analysis it was discovered that in Cooper's D the observed frequency of pathology was not significantly higher than the expected frequency. This result suggests that for Cooper's D, there is not a predilection for pathology of the distal-limb, just elevated numbers proportionate to the sample size of the distal-limb elements. A similar result, however, was not present upon analysis of Swartkrans. The 32 observed cases of pathology on distal-limb elements was significantly higher than the expected frequency of 20. The overwhelming frequency of pathology for distal elements from Swartkrans, therefore, cannot be explained by the vagaries of sampling or chance.

Other appendicular elements, both fore- and hind-limb, were well represented from Cooper's D (*n.* = 447) and Swartkrans (*n.* = 2026), yet accounted for a lower frequency of pathology than expected in both instances, combined accounting for only 10 of the 64 observed cases of pathology.

Vertebral elements accounted for a slightly higher than expected frequency of pathology for both sites. Sternal elements were poorly preserved, and in both cases the expected frequency of pathology was low and no pathological fossils were identified.

In sum, most cases of pathology for both assemblages occurred in the distal-limbs and vertebrae. Differential survivorship of elements can likely explain these results for Cooper's D, however, cannot explain the results for the distal-limb for Swartkrans. Pathology is, therefore, overrepresented in the extreme distal elements and underrepresented in the hindlimb long bones in the Swartkrans assemblage.

It is an interesting, and unexpected result that all animals showed evidence of pathology in a very similar way, with lesions occurring on extreme distal-limbs and/or vertebrae. This suggests that animals filling vastly different niches within the trophic system, adopting different physical forms, and an array of behavioural or physical

attributes tend to display great similarities in the anatomical location of skeletal pathology.

7.4 The frequency of skeletal pathology varies between the different taxa.

7.4.1 Order

Pathology occurred across orders in both assemblages. Only perissodactyls were free of skeletal pathology. The remaining Orders, Artiodactyla, Carnivora, and Primates all exhibited signs of skeletal change consistent with response to pathological stimuli. It is important to note, however, that the inter-order frequencies of pathology varied, as hypothesized.

The results seem remarkably similar at order level for both sites. Artiodactyls comprised the vast majority of specimens within both assemblages, yet these showed the lowest percentage incidence of pathology of all orders (0.7% for Cooper's D and 0.4% for Swartkrans). Carnivores showed a higher yet similar frequency of pathology for Cooper's D (4%) and Swartkrans (3%). In both cases, primates showed the highest frequency at 4.7% and 6% for Cooper's D and Swartkrans, respectively. The primate numbers were bolstered by the high frequency of hominid pathology from both sites, a result that will be discussed further in section 7.5.

The $\%MNI_{path}$ rates for Cooper's D showed a similar result to the %NISP calculations with artiodactyls showing the lowest frequency (3.0%), carnivores showing a slightly higher frequency (10.7%) and primates showing the highest frequency (13.6%). The results from Swartkrans are slightly different. Artiodactyls still show the lowest frequency (1.0%), primates show the next highest frequency (3.2%) and carnivores show the highest frequency (4.5%).

7.4.2 Family

The chi-square and randomization tests for goodness-of-fit revealed a highly significant result at the 0.05 level. Although expected frequencies are low, there is a significant gap between the expected and observed frequency of pathology for specifically the hominids, felids and bovids from both sites. This indicates clearly that pathology does not occur at a frequency proportionate to the NISP for all families, but rather occurs at a much higher frequency for hominids and felids, and a much lower frequency for bovids. It appears that the frequency of bone pathology loosely correlates to the trophic roles of the different taxa. The specific results will be discussed in the paragraphs to follow.

Equids, which are represented by postcrania from both sites, are free from gross skeletal pathology. The sample of equids is low (*n.* = 19 for Cooper's D; *n.* = 34 for Swartkrans) and the absence of pathology might relate to sample size and simple chance. Giraffidae, Caprinae, Hippopotamidae and Elephantidae are all present at Swartkrans, but are free from pathology, possibly due to their extremely low representation. Due to the particularly low sample sizes, the above mentioned families will be excluded from the discussion to follow.

Bovids by far numerically dominate the assemblages. They are, however, responsible for the lowest frequency of pathology in the Cooper's D assemblage (0.6%) and Swartkrans assemblage (0.4%).

These results are interesting when contrasted with those of all remaining families represented in the sample. The primate and carnivore families tend to show a higher frequency of pathology than the ungulates. Families representing Carnivora show a 2.1 to 6.0% frequency of pathology, with the notable exceptions of canids from Cooper's D and hyaenids from Swartkrans, which show a zero incidence of pathology. In contrast, hyaenids from Cooper's D exhibit the highest frequency of pathology for carnivores (6%). Felids from both sites display a 4-5% frequency of pathology. Canids from Swartkrans show a 2.1% frequency of pathology. Cercopithecids, show a

similar occurrence of pathology to the above mentioned families, of 2.9% and 1.3% from Cooper's D and Swartkrans respectively.

Calculation of $\%MNI_{path}$ rates revealed a similar result when pathology was compared between sites for the four families that showed pathology at both Cooper's D and Swartkrans. Rate of pathology was low for bovids (1.0 – 2.3%), and moderately high for felids (10.0% - 14.0%). Pathology fell within this range for cercopithecids from both sites (1.5% - 10.0%) and hominids from Swartkrans (7.1%). Hominids from Cooper's D showed the highest $\%MNI_{path}$ value of 50.0% which is possibly due to the extreme low sample size.

The discrepancies in the frequency of pathology across taxa suggest that behaviour (specifically trophic roles) may influence the rate of observable skeletal pathology. Ungulates, common-prey species, display the lowest overall frequency of pathology at order and family levels. Carnivores display a much higher rate of pathology. This is possibly the result of the comparatively high survival rate of diseased or injured predators and conversely the low survival rate of injured or diseased prey animals. It is a fair assumption that predators (based on their trophic role) will generally live longer than prey (of a similar size) in wild populations. This increased senescence will serve to augment the rate of observed skeletal pathology by increasing the amount degenerative (wear and tear) lesions and by allowing more time for cumulative disease or injury to leave an impress on the skeleton (Bartosiewicz *et al.*, 1997).

Behaviour might also relate to the rate of pathology for primates being higher than the ungulates. Primates possibly utilized the trees to evade predation and survive initial injury or illness. Interestingly, cercopithecids did not show any evidence of long-bone fracture from either site. Previous research (Schultz, 1956) has shown long-bone fracture to be relatively common in primates, occurring at a frequency of between 20 and 30% of individuals per group. Chapman *et al.* (2007) suggest that frequency of trauma becomes higher with an increase in canopy travelling height. The low frequency of pathology supports a similar behaviour of the fossil cercopithecids in the

Plio-Pleistocene to the analogous modern groups. It is noted that modern baboons spend the majority of their time on the ground and vervets utilize both the ground and the canopy but tend to spend more time on the ground (Lavigne, 2009). This would explain the absence of long-bone trauma in the fossil cercopithecids.

7.5 The rate of pathology for hominids will be comparable to the rates of pathology for carnivores and other primates.

The hominid rate of pathology for Cooper's D is 100%, as only two postcranial elements are present and both are pathological. It was hypothesized that the frequency of pathology for the larger Swartkrans hominid assemblage would be more similar to the frequency of pathology for the carnivore families and cercopithecids than the ungulates (bovids and equids in particular). The frequency of pathology for the hominids was higher than hypothesized, accounting for 16% of the total hominid NISP. This rate of pathology is several times higher than that of all other families.

Hominids were surveyed not as an outlier from the palaeo-community, but as a member of the larger faunal complex. It was the author's original assertion that at this particular time (1.6 - 1.9 Ma) the behaviour of hominids (*Paranthropus robustus*) would not drastically affect their palaeoecological role and it would be fair to predict that they would suffer the same 'slings and arrows' of other wild fauna at a similar frequency. The two predominant pathologies encountered in the hominid fossils were joint disease and enthesopathy. Since both conditions have at least a partial functional component to the pathogenesis (see chapter 3) it is possible that they represent bone changes resulting from mechanical stresses.

The two proven outlying behaviours of *Paranthropus robustus* and early *Homo* sp. are the bipedal modality of locomotion and the early stages of tool utilization. The first is pathologically problematic from the start, as the function of bipedality, in itself, creates structural problems to the skeleton that normal physiology cannot efficiently mitigate

against. Lovejoy (2005) proposes that one of the earliest physiological adjustments to bipedality was the elongation of the lumbar spine, which facilitated lumbar lordosis. This elongation may have predisposed *Australopithecus* and its descendents to greater rates of vertebral pathology than even modern humans (e.g. scoliosis and spondylolysis) (ibid). Vertebral arthritis is so ubiquitous in fact that it occurs in 80-90% of modern humans after the age of 75 years (Aufderheide and Rodriguez-Martin, 1998). Similarly, the structure of the foot itself is fundamental to bipedalism and may predispose the bones of the foot to an increased frequency of pathology. Zipfel (2006) noted that pathology of the first ray metatarsal was common, occurring at frequencies of between 36 and 68% for recent human populations (e.g. Zulu, Sotho and European) and 35% for a pre-pastoralist archaeological sample.

All three pathological hominid vertebrae show signs of degeneration of the intervertebral disk and associated arthritic changes. The changes manifest in a remarkably similar way to modern human spinal osteoarthritis. The hominid hallucial metatarsal and first phalanx (both attributed to *Paranthropus robustus*) may also demonstrate the affects of a bipedal gait. There is clear bone development and buttressing of the dorsal surfaces of both elements, manifesting as a sessile crest on the distal metaphysis of the metatarsal and a raised ridge on the basodorsal surface of the phalanx. The elements themselves likely belong to one individual. The prominent tubercle may be the result of periosteal irritation resulting from excessive hyperdorsiflexion of the metatarsophalangeal joint. It could also represent an area of localized trauma or an enthesophyte. The lesion on the phalanx occurs at the *m. extensor hallucis brevis*, which acts to extend the 1st toe, and may represent a heavily remodelled enthesophyte.

The rest of the lesions on the hominid fossils involve enthesopathy and/or osteoarthritis to various degrees on manual distal elements. Most of the enthesopathies described occur on manual phalanges; the manual 1st phalanx (SKX 9342) and the manual 2nd phalanges (SKX 5022, SKX 13476, SKX 35439 and SKX 38653). It is important to restate that the aetiology of enthesopathy appears to be multifactoral, and

caution is advised in interpreting musculoskeletal stress markers, like enthesophytes (Waldron, 1994; Jurmain, 1999; Jurmain and Roberts, 2008), as sex, genetics, age, diet, disease and work/activity can all contribute to their development. They have, however, traditionally been used to interpret behavioural, functional and even occupational aspects of archaeological populations. Entheses mark the muscular or ligamentous attachment to bone and are the interface between soft tissue and skeletal tissue, therefore, they reflect on biomechanics and *in vivo* muscle activity. This has inevitably led to the over-simplification and often over-interpretation of their relationship to activity. While hominids make up only 1.2% of the total NISP for the site of Swartkrans, they account for 39% of the cases of enthesopathy. While caution should be exercised in interpreting these often problematic lesions, this relationship cannot be ignored and will be discussed as a point of interest in the paragraphs to follow.

Recent studies have suggested that entheses morphology does provide "information on muscle activity levels, with greater development of entheses associated with more habitual or powerful muscle use" (Drapeau, 2008). This in no way diminishes from the other potential contributors to enthesial bone changes, but rather intimates that bone changes at entheses, while correlated with age, sex and genetics, may have at least a partial functional component due to injury or overuse (Slobodin *et al.*, 2007). In fact, mechanical loading is a known trigger of osteoblastic activity at entheses (Kannus *et al.*, 1996). Although the best correlate for development of enthesophytes is advancing age, it has never been suggested that they just spontaneously develop, but more likely that they represent degenerative processes, much like osteophytes, with some mechanical stresses initiating the bone changes. This becomes significant when interpreting behaviour for an extinct hominid form.

The affected phalanges come from two temporally discrete Members and signify at least two distinct individuals. The Lower Bank (Member 1) fossils are attributed to *Paranthropus robustus* and the Member 3 fossils are attributed to *Homo* sp. In total there are eight second phalanges in the Swartkrans hominid post-cranial assemblage.

Four of these phalanges are pathological, representing at least two individuals. If one individual was present with a diffuse condition like DISH or *ankylosing spondylitis*, it might explain the high frequency of the pathology. However, the fact that two distinct individuals display the same pathomorphology and account for 50% of the sample raises the possibility that something functional might account for the observed changes. While the entheses on the hominid 2nd phalanges from Swartkrans have been described as representing either strong or pronounced markings for the insertion of the *m. flexor digitorum superficialis* (Susman, 1993), there has been little consideration of them as pathological, i.e. the skeletal expression of an enthesopathy.

Entheses are designed to distribute forces, and moderate endurance exercise has been demonstrated to have no affect on entheses (Zumwalt, 2006). It has, however, been suggested that activity beyond a hypothetical threshold, i.e. beyond the bone's "habitual *milieu*" (Judex and Zernicke, 2000), is necessary to create an osteogenic response. This is corroborated by this study, with the infrequent occurrence of enthesophytes for other surveyed species, many of which are heavily muscled, but performing 'normal' endurance activities. The enthesopathies may, therefore, not represent muscular size and strength, but simply muscle activity beyond the bones structural capabilities; suggesting a behaviour to which the entheses are physiologically ill-adapted, resulting in a predisposition to the condition. In modern humans hand bone enthesophytes appear to be a common phenomenon of aging, with their expression increasing after the age of 25, with either mechanical load or genetics thought to be the contributing factors of their development in response to stress (Kalichman *et al.*, 2007).

It is possible that there is an age correlation with cumulative stress and micro-trauma causing the enthesopathies in the hominids. It may be equally reasonable that they result from activity beyond a normal/pathological threshold. In either case, when considering frequency of pathology, we have a clear deviation from an animal model (ranging from 0% to 6% by family) and a move towards the much higher frequency of a human model. When compared to the remainder of the assemblage, several

explanations arise; either the hominids are suffering different diseases, are living longer, or are functioning differently.

Increasing evidence is carrying us away from the idea of the lumbering small brained *Paranthropus* to an idea of a hominid physiologically and anatomically capable of precision grasping and tool use. It has been suggested that *Paranthropus* may have been the first maker of stone tools, and may have relied heavily on bone tools (Susman, 1991). Contextual evidence has suggested the use of bone tools by *Paranthropus robustus* from Swartkrans and other sites in the Cradle of Humankind in digging activities (Backwell and d'Errico, 2001; Backwell and d'Errico, 2003; d'Errico and Backwell, 2003; Backwell and d'Errico, 2008). The *m. flexor digitorum superficialis* is responsible for flexion of the proximal interphalangeal joints of the medial four digits, and further flexes the metacarpophalangeal joints and hand (Agur and Dalley, 2005). Could the physiological stress involved in tool manufacture and use have exceeded the pathological threshold for the *m. flexor digitorum superficialis*, particularly at its insertion with the 2nd phalanges? If so, could this account for the human-like expression of hand bone mid-shaft enthesophytes? This could be explored further by studying the frequency of hand bone mid-shaft enthesophytes on humans and non-human primates. Are they a distinctly human expression of enthesopathy or do they occur with similar frequency in non-human primates? Clearly, several other possibilities exist for the explanation of the high frequency of enthesopathy in the Swartkrans fossils, including:

- metabolic disturbances;
- certain disease conditions;
- genetics;
- over-representation due to one or two pathological individuals in a small sample.

7.6 Individual animal life-history traits can be extrapolated by examining pathological fossils.

Although life-history traits are discussed below, it is important to understand some of the difficulties in drawing conclusions from these results. Wood *et al.,* (1992) review several inherent problems with inferring health from skeletal samples. Firstly, most diseases leave no trace on the skeleton. We are essentially blind to their presence or absence and a skeleton can look perfectly 'healthy' when the individual was diseased or injured. Secondly, in a given assemblage, pathological bones are often considered as having belonged to 'unfit' individuals. The potential exists, however, that the individual's immune response was strong enough that the individual survived the disease long enough to develop the skeletal lesions. Others in the population may have suffered such acute stress from the disease that they died before the development of the lesions. In many cases, it is impossible to distinguish between those who never experienced the disease and those who died in the early stages of the disease (Wood *et al.,* 1992). Similarly, the immune response of an individual might be so strong that it could eliminate the pathogen before the skeleton could be affected; in which case no evidence of a disease would be seen, even though the disease was present. While these cautionary notes are true, it is possible to make some inferences based on the observed lesions in the fossil samples.

As pathology represents functional or structural deviations from the normal anatomy of an animal, it is likely that many of these conditions had a negative impact on the individual affected. It was hypothesized that life-history traits could be extrapolated from the pathological remains. The expressions of pathology were similar enough to modern comparatives to allow for the description of the conditions. This allowed for the opportunity to use clinical resources to interpret behavioural traits based on the pathological conditions in the fossils.

Many of the cases of pathology appear to be an expression of joint disease resulting from age, repetitive injury, trauma or a multitude of other factors. In modern

examples, at the very least these joint conditions result in pain and often altered function (Radostits *et al.,* 2000; Kahn and Line, 2005). Lameness is an indication of an impediment to the musculoskeletal system, generally resulting from pain, weakness or deformity. It can be classified as weight-bearing, usually resulting from injuries to the bones, tendons, ligaments, or motor nerves; or as non-weight-bearing, typically resulting from injuries to the joints (Kahn and Line, 2005). Osteoarthritis, for example, in the more chronic stages creates a reduced range of motion due to fibrous thickening of the joint capsule, the results of which range from mild gait change to severe lameness (ibid). If modern clinical findings hold true for the Plio-Pleistocene, there must have been behavioural changes that accompanied the pathologies found in the fossil assemblage. At the very least the cases of joint disease in the bones of the distal-limb must have caused pain and altered gait in the individual animals. Arthritis specific to the axial skeleton also would have caused pain and possibly altered gait.

CD 7971 the fused lumbar vertebrae of a leopard seems striking in gross appearance; although similar injuries can be clinically insignificant in modern dogs and cats and may have resulted in only minor behavioural changes in this case. Attempting more specific behavioural determinations would require analogous modern studies documenting the behaviour of wild animals following similar pathological insults. The comparative diagnostic veterinary material available seems to focus primarily on domesticated animals, making more specific inferences as to the behaviour of wild animals, largely a matter of conjecture.

The exostoses and enthesophytes noted from both sites are generally considered clinically asymptomatic, and would result in very little negative impact on the function of the bones or the animal. They can in certain cases be painful and lead to disorders of gait or posture (Benjamin *et al.,* 2000). The possible osteoid osteoma seen on the hominid metatarsal SK 7923 would certainly cause swelling and tenderness (Tsang and Wu, 2008) and would have resulted in pain and an altered gait.

Chapter 8. Conclusion

This dissertation has described a new method for analyzing the faunal assemblages of the Plio-Pleistocene fossil sites from the Cradle of Humankind, South Africa. In a consumer-resource driven system many critical interactions occur among species (Vandermeer and Goldberg, 2003). While these trophic relationships have been carefully considered when interpreting hominid-bearing sites, one important predator-prey interaction has too often been overlooked. That is the relationship of disease to host; of the parasite to the infected. In essence the parasite attacks the prey to acquire nutrients and energy, and the prey tries to defend against the attacker (Hall *et al.*, 2008), culminating in what Hudson *et al.* (2008: 1) call an "evolutionary arms race where both were fighting for their lives and their fitness". The difference between this relationship and that of a lion to an impala is simply one of scale. This predator influences the host population dynamics reducing host survival (Tompkins *et al.* 2008). In a trophic system, pathology affects individual survival in two primary ways: indirectly, by decreasing fitness and allowing for predation, or directly, by being the ultimate cause of mortality.

This project did not set out to make inferences as to the relative health of the living community during the Plio-Pleistocene. Rather, it set out to test whether skeletal evidence of pathology existed in the fossil assemblages of two Plio-Pleistocene hominid-bearing cave sites within the Cradle of Humankind, South Africa, and the following results were revealed.

It was confirmed that pathological fossils did exist in both fossil assemblages. This result indicates clearly that individual animals displaying evidence of pathology, in the form of skeletal alterations, lived at the time the deposit was being formed and found their way into the fossil assemblages.

The types of lesions suggest that individual animals during the Plio-Pleistocene suffered different disease conditions, including repetitive stress, chronic infection, neoplasia, degenerative changes of senescence, and musculoskeletal injury.

The pathological remains from the assemblages represent a wide range of fauna, and span taxa at both order and family levels. It was discovered that there is no distinction between the skeletal elements affected by pathology at the order level, although distal-limb and vertebral elements have a slightly higher than expected frequency of pathology at site aggregate level. Interestingly, all orders display skeletal pathology in the same general areas of the body, i.e. vertebrae and distal-limb. This is intriguing when one considers that animal taxa, adapted to vastly different niches in the environment manifest skeletal pathology in a very similar way.

The frequencies of pathology vary between orders and between families. The ungulates showed the lowest frequency of pathology possibly due to the fact that an injured or sick antelope would likely fall prey to a carnivore before an osteogenic response to disease or trauma. Large carnivores showed a progressively higher frequency of pathology, possibly due to modifying their dietary behaviour (e.g. changing preferred prey species, or scavenging instead of hunting). Primates (cercopithecids) have a varying frequency of pathology, but appear higher than ungulates and similar to carnivores, possibly due to an ability to escape from predators to the safety of trees. Hominids fall outside of the range of frequency of pathology for all other families. They appear to deviate from the animal model and tend towards a more human-like model of frequency of pathology.

This was the first effort at the recognition and analysis of skeletal pathology from the fossil sites of the Cradle of Humankind. The questions posed were basic to the problem of un- or under-recognized pathologies in the samples. The results are, by no means an end point, but rather, a jumping-off point. Future research will be critical in understanding the levels and types of disease in the fossil assemblages of South Africa. This research would be enhanced by:

1. The inclusion of craniodental pathology from both sites. This will certainly expand the database on the types of pathologies affecting the fauna, and can also allow for important behavioural assertions.
2. Future analyses using archaeozoological methodologies to more readily allow for comparisons of data. The use of anatomical zones after Dobney and Reilly (1988) and newly designed recording protocols (e.g. Vann, 2008) will make the results from future studies more accessible for inter-site comparisons.
3. Future study of the rate of skeletal pathology in modern wild faunal communities. At present there is a stark discrepancy between what is known about types and frequency of skeletal pathology affecting wild fauna versus domesticated fauna or humans.
4. Monitoring behavioural changes that accompany skeletal manifestations of disease or trauma in wild animals.
5. Surveys specifically focused on pathology in hominid fossils compared to a non-hominid ape sample (with regards to frequency and anatomical location).

In summary, it is clear that pathology existed in the Plio-Pleistocene in southern Africa, and that animals were subjected to abnormal conditions that may have weakened them and increased their susceptibility to predation, and assuming a carnivore accumulating agent, ultimately their inclusion in the fossil record. Whether the principal cause of death or not, pathology is apparent across orders and remains one of our few windows into the stresses and stressors experienced by individual fossil animals. Disease must be considered as one of the potential causes of mortality (either directly or indirectly). Therefore, this study provides some insight into conditions that may have played a critical role in individual survival, fecundity, mortality and ultimately evolution.

APPENDIX A – List of terms

All definitions were taken from the Oxford Concise Medical Dictionary (Martin, 2003), unless otherwise cited.

Abscess – A localized collection of pus anywhere in the body surrounded and walled off by damaged and inflamed tissues.

Anastomosis – A communication between two blood vessels without any intervening capillary network.

Ankylosis – The pathological fusion of bones across a joint space. A complication of prolonged joint inflammation.

Anomaly – Any deviation from the normal, especially a congenital or developmental defect.

Anthropozoonosis – A disease that is transmissible from an animal to a human, or vice versa, under natural conditions.

Apophysis – A protuberance of bone to which a tendon is attached. It ossifies separately from the rest of the bone and fuses with it at maturity.

Apoptosis – Programmed cell death, which results in the ordered removal of cells and occurs naturally as part of the normal development, maintenance, and renewal of cells, tissues, and organs.

Arthrosis – Degenerative disease of a joint (Newton and Nunamaker, 1985).

Asymptomatic – Not showing any symptoms of disease whether disease is present or not.

Avulsion – The tearing or forcible separation of part of a structure.

Bacillus – Any rod-shaped bacterium.

Callus – The bony material that bridges fractured bone fragments (Newton and Nunamaker, 1985).

Consanguinity – Relationship by blood; the sharing of a common ancestor within a few generations.

Cortex – The outer part of an organ, situated immediately beneath its capsule or outer membrane.

Disease – An impairment of the normal state of the living body or one of its parts that interrupts or modifies the performance of the vital functions (Pease Jr., 1995)

Dysplasia – Abnormality of development (Newton and Nunamaker, 1985).

Ectopia – The misplacement, due to either congenital defect or injury, of a bodily part. The occurrence of something in an unnatural location.

Enthesis – The site of insertion of tendons or ligaments into bones.

Enthesopathy – Any disease or trauma resulting in inflammation of entheses.

Exostosis – A benign outgrowth of bone with a cap of cartilage, arising from the surface of a bone.

Fistula – An abnormal communication between two hollow organs or between a hollow organ and the exterior.

Forme fruste – An atypical form of a disease in which the usual symptoms fail to appear and its progress is stopped at an earlier stage than would ordinarily be expected.

Fracture – The breakage of bone, either complete or incomplete.

Haematoma – An accumulation of blood within the tissues that clots to form a solid swelling. Injury, disease of the blood vessels, or a clotting disorder of the blood are the usual causative factors.

Inflammation – A localized non-specific response to defend, repair, or alter body components (Gregg, 1987)

Ischaemia – An inadequate flow of blood to a part of the body, caused by constriction or blockage of the blood vessels supplying it.

Kyphosis – Excessive outward curvature of the spine causing hunching of the back.

Lordosis – Inward curvature of the spinal column.

Osteolysis – Dissolution of bone through disease, commonly by infection or loss of the blood supply (*ischaemia*) to the bone.

Periosteum – A layer of dense connective tissue that covers the surface of a bone except at the articular surfaces. The periosteum provides attachments for muscles, tendons, and ligaments.

Polyarticular – Pertaining to two or more joints (Newton and Nunamaker, 1985).

Rarefaction – Thinning of bony tissue.

Reduction – The restoration of a displaced part of the body to its normal position. A dislocated joint is reduced to its normal seating.

Sclerosis – Hardening of tissue, usually due to scarring (*fibrosis*) after inflammation or ageing.

Sequestrum – A portion of dead bone formed in an infected bone in chronic osteomyelitis. It is surrounded by an envelope (*involucrum*) of sclerotic bone and fibrous tissue.

Spondylosis – A spinal condition resulting from degeneration of the intervertebral disks in the cervical, thoracic, or lumbar regions.

Subcutaneous – Beneath the skin.

Subluxation – Partial dislocation of a joint so that the bone ends are misaligned but still in contact.

Synostosis – The union of adjacent bones by means of osseous union of bones that are normally separate (Newton and Nunamaker, 1985).

Trauma – A physical wound or injury, such as a fracture or blow.

Viscera – The organs within the body cavities, especially the organs of the abdominal cavities.

Zoonosis – Diseases that can be transmitted naturally between vertebrate animals and man (Brothwell, 1991).

APPENDIX B – Comparative collections

Normal Modern Comparative Collections

Table 3.3. Non-human normal comparative sample. University of the Witwatersrand, Bernard Price Institute (BPI) and Department of Anatomical Sciences.

Catalogue		Order	Family	Genus	Species	Common name	Material
ZA	932	Artiodactyla	Bovidae	Cephalophus		Red Duiker	Crania, postcrania
ZA	764	Artiodactyla	Bovidae	Redunca		Reed Buck	Crania, postcrania
ZA	1034	Artiodactyla	Bovidae	Redunca		Reedbuck	Crania, postcrania
BP/4	593	Artiodactyla	Bovidae	Aepyceros	*melampus*	Impala	Postcrania
BP/4	101	Artiodactyla	Bovidae	Aepyceros	*melampus*	Impala	Crania, postcrania
BP/4	102	Artiodactyla	Bovidae	Aepyceros	*melampus*	Impala	Crania, postcrania
BP/4	105	Artiodactyla	Bovidae	Aepyceros	*melampus*	Impala	Crania, postcrania
BP/4	104	Artiodactyla	Bovidae	Aepyceros	*melampus*	Impala	Crania, postcrania
ZA	472	Artiodactyla	Bovidae	Antidorcas	*marsupialis*	Springbok	Crania, postcrania
ZA	477	Artiodactyla	Bovidae	Antidorcas	*marsupialis*	Springbok	Crania, postcrania
ZA	738	Artiodactyla	Bovidae	Antidorcas	*marsupialis*	Springbok	Crania, postcrania
BP/4	687	Artiodactyla	Bovidae	Antidorcas	*marsupialis*	Springbuck	Postcrania
BP/4	688	Artiodactyla	Bovidae	Bos	*domesticus*	Ox	Postcrania
BP/4	689	Artiodactyla	Bovidae	Bos	*domesticus*	Ox	Postcrania
ZA	1024	Artiodactyla	Bovidae	Cephalophus		Grey Duiker	Crania, postcrania
BP/4	279	Artiodactyla	Bovidae	Connochaetes	*taurinus*	Wildebeest	Postcrania
BP/4	280	Artiodactyla	Bovidae	Connochaetes	*taurinus*	Wildebeest	Postcrania
BP/4	87	Artiodactyla	Bovidae	Connochaetes	*taurinus*	Wildebeest	Crania, postcrania
BP/4	88	Artiodactyla	Bovidae	Connochaetes	*taurinus*	Wildebeest	Crania, postcrania
ZA	721	Artiodactyla	Bovidae	Damaliscus	*dorcus*	Blesbok	Crania, postcrania

ZA	722	Artiodactyla	Bovidae	Damaliscus	*dorcus*	Blesbok	Crania, postcrania
BP/4	100	Artiodactyla	Bovidae	Damaliscus	*lunatus*	Tssesebe	Crania, postcrania
ZA	655	Artiodactyla	Bovidae	Kobus	*leche*	Lechwe	Crania, postcrania
BP/4	52	Artiodactyla	Bovidae	Kobus	*ellipsiprymnus*	Waterbuck	Crania, postcrania
BP/4	608	Artiodactyla	Bovidae	Pelea	*capreolus*	Vaal rhebuck	Crania, postcrania
BP/4	890	Artiodactyla	Bovidae	Raphicerus	*campestris*	Steenbok	Crania, postcrania
BP/4/	1289	Artiodactyla	Bovidae	Raphicerus	*campestris*	Steenbok	Crania, postcrania
BP/4	632	Artiodactyla	Bovidae	Redunca	*fulvorufula*	Mt Reedbuck	Postcrania
BP/4	337	Artiodactyla	Bovidae	Redunca	*arundinum*	Southern Reedbuck	Crania, postcrania
ZA	637	Artiodactyla	Bovidae	Syncerus	*Caffer*	Buffalo	Crania, postcrania
ZA	662	Artiodactyla	Bovidae	Syncerus	*Caffer*	Water Buffalo	Crania, postcrania
ZA	718	Artiodactyla	Bovidae	Tragelaphus	*scriptus*	Bushbuck	Crania, postcrania
ZA	915	Artiodactyla	Bovidae	Tragelaphus	*strepsiceros*	Kudu	Crania, postcrania
ZA	1072	Artiodactyla	Bovidae	Tragelaphus	*strepsiceros*	Kudu	Crania, postcrania
BP/4	38	Artiodactyla	Bovidae	Tragelaphus	*scriptus*	Bushbuck	Crania, postcrania
BP/4	283	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	282	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	276	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	619	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	532	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	533	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	534	Artiodactyla	Bovidae			Bovid	Postcrania
BP/4	30	Artiodactyla	Giraffidae	Giraffa	*camelopardalis*	Giraffe	Postcrania
BP/4	284	Artiodactyla	Giraffidae	Giraffa	*camelopardalis*	Giraffe	Postcrania
BP/4	683	Artiodactyla	Giraffidae	Giraffa	*camelopardalis*	Giraffe	Postcrania
BP/4	617	Artiodactyla	Hippopotamidae	Hippopotamus	*amphibius*	Hippopotamus	Crania, postcrania
BP/4	261	Artiodactyla	Suidae	Phacochoerus	*aethiopicus*	Warthog	Crania, postcrania
BP/4	12	Artiodactyla	Suidae	Phacochoerus	*aethiopicus*	Warthog	Crania, postcrania

BP/4	13	Artiodactyla	Suidae	Phacochoerus	*aethiopicus*	Warthog	Crania, postcrania
BP/4	333	Artiodactyla	Suidae	Phacochoerus	*aethiopicus*	Warthog	Crania, postcrania
BP/4	898	Artiodactyla	Suidae	Potamochoerus	*porcus*	Bushpig	Crania, postcrania
ZA	478	Artiodactyla	Suidae	Potamochoerus	*porcus*	Bush-pig	Crania, postcrania
BP/4	685	Artiodactyla	Suidae	Sus	*domesticus (?scrofa)*	Domestic pig	Postcrania
BP/4	273	Carnivora	Canidae	Canis	*mesomelas*	Black-backed jackal	Crania, postcrania
ZA	56	Carnivora	Canidae	Canis	*familiaris*	Domestic Dog	Crania, postcrania
ZA	1036	Carnivora	Canidae	Canis		Jackal	Crania, postcrania
BP/4	224	Carnivora	Canidae	Lycaon	*pictus*	Wild dog/ Cape hunting dog	Postcrania
BP/4	223	Carnivora	Canidae	Lycaon	*pictus*	Wild dog/ Cape hunting dog	Crania, postcrania
BP/4	222	Carnivora	Canidae	Lycaon	*pictus*	Wild dog/Cape hunting dog	Crania, postcrania
BP/4	285	Carnivora	Canidae	Otocyon	*megalotis*	Bat eared fox	Crania, postcrania
BP/4	584	Carnivora	Canidae	Otocyon	*megalotis*	Bat eared fox	Crania, postcrania
BP/4	620	Carnivora	Canidae	Proteles	*cristatus*	Aardwolf	Crania, postcrania
BP/4	621	Carnivora	Canidae	Vulpes	*chama*	Cape fox	Postcrania
BP/4	575	Carnivora	Canidae	Vulpes	*chama*	Cape fox	Crania, postcrania
BP/4	580	Carnivora	Canidae	Vulpes	*chama*	Cape fox	Crania, postcrania
BP/4	227	Carnivora	Canidae	Vulpes	*chama*	Cape fox	Crania, postcrania
BP/4	201	Carnivora	Felidae	Acinonyx	*jubatus*	Cheetah	Crania, postcrania
BP/4	274	Carnivora	Felidae	Felis	*lybica*	African wildcat	Crania, postcrania
BP/4	253	Carnivora	Felidae	Panthera	*pardus*	Leopard	Crania, postcrania
BP/4	258	Carnivora	Felidae	Panthera	*pardus*	Leopard	Crania, postcrania
ZA	853	Carnivora	Felidae	Panthera	*pardus*	Leopard	Crania, postcrania
BP/4	183	Carnivora	Felidae	Panthera	*leo*	Lion	Crania, postcrania
BP/4	186	Carnivora	Felidae	Panthera	*leo*	Lion	Crania, postcrania
BP/4	252	Carnivora	Felidae	Panthera	*leo*	Lion	Crania, postcrania
BP/4	255	Carnivora	Felidae	Panthera	*leo*	Lion	Crania, postcrania

BP/4	203	Carnivora	Hyaenidae	Crocuta	*crocuta*	Spotted hyena	Crania, postcrania
BP/4	254	Carnivora	Hyaenidae	Crocuta	*crocuta*	Spotted hyena	Crania, postcrania
BP/4	239	Carnivora	Mustelidae	Poecilogale	*albinucha*	African weasel	Crania, postcrania
BP/4	242	Carnivora	Protelidae	Proteles	*cristatus*	Aardwolf	Crania, postcrania
BP/4	614	Carnivora	Viverridae	Genetta	*genetta*	Genet	Postcranials, scapula
BP/4	324	Carnivora	Viverridae	Genetta	*genetta*	Genet	Crania, postcrania
BP/4	325	Carnivora	Viverridae	Genetta	*genetta*	Genet	Crania, postcrania
BP/4	251	Carnivora	Viverridae	Herpestes	*sanguineus*	Mongoose	Crania, postcrania
BP/4	917	Carnivora	Viverridae	Suricata	*suricatta*	Meerkat	Crania, postcrania
BP/4	697	Hyracoidea	Procaviidae	Procavia	*capensis*	Dassie	Postcrania
BP/4	604	Hyracoidea	Procaviidae	Procavia	*capensis*	Dassie	Crania, postcrania
BP/4	911	Perissodactyla	Equidae	Equus	*burchelli*	Burchell's Zebra	Crania, postcrania
BP/4	609	Perissodactyla	Equidae	Equus	*caballus*	Horse	Postcrania, hindlimb
BP/4	154	Perissodactyla	Equidae	Equus	*zebra*	Mountain zebra	Crania, postcrania
BP/4	684	Perissodactyla	Rhinocerotidae	Ceratotherium	*simum*	White rhino	Postcrania
BP/4	289	Perissodactyla	Rhinocerotidae	Ceratotherium	*simum*	White rhino	Crania, postcrania
BP/4/	1290	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet monkey	Crania, postcrania
BP/4	295	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet monkey	Crania, postcrania
BP/4	979	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet monkey	Crania, postcrania
ZA	1240	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet Monkey	Crania, postcrania
ZA	1242	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet Monkey	Crania, postcrania
ZA	1244	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet Monkey	Crania, postcrania
ZA	1257	Primates	Cercopithecidae	Cercopithecus	*aethiops*	Vervet Monkey	Crania, postcrania
BP/4	541	Primates	Cercopithecidae	Papio	*ursinus*	Chacma baboon	Crania, postcrania
BP/4	583	Primates	Cercopithecidae	Papio	*ursinus*	Chacma baboon	Crania, postcrania
BP/4	178	Primates	Cercopithecidae	Papio	*ursinus*	Chacma baboon	Crania, postcrania
ZA	663	Primates	Cercopithecidae	Papio	*ursinus*	Chacma Baboon	Crania, postcrania
ZA	664	Primates	Cercopithecidae	Papio	*ursinus*	Chacma Baboon	Crania, postcrania

ZA	665	Primates	Cercopithecidae	Papio	*ursinus*	Chacma Baboon	Crania, postcrania
ZA	455	Primates	Galagidae			Galago	Crania, postcrania
BP/4	544	Primates	Pongidae	Gorilla	*gorilla*	Gorilla	Crania, postcrania
ZA	1354	Primates	Pongidae	Pan	*troglodytes*	Chimpanzee	Crania, postcrania
ZA	1355	Primates	Pongidae	Pan	*troglodytes*	Chimpanzee	Crania, postcrania
BP/4	670	Rodentia	Cricetidae	Pedetes	*capensis*	Spring hare	Crania, postcrania
BP/4	682	Rodentia	Hystricidae	Hystrix	*africaeaustralis*	Porcupine	Postcrania
BP/4	156	Rodentia	Hystricidae	Hystrix	*africaeaustralis*	Porcupine	Crania, postcrania
BP/4	157	Rodentia	Hystricidae	Hystrix	*africaeaustralis*	Porcupine	Crania, postcrania
BP/4	641	Rodentia	Hystricidae	Hystrix	*africaeaustralis*	Porcupine	Crania, postcrania
ZA	896	Rodentia	Hystricidae	Hystrix	*africaeaustralis*	Porcupine	Crania, postcrania
BP/4	693	Rodentia	Pedetidae	Pedetes	*capensis*	Springhare	Postcrania
BP/4	155	Tubulidentata	Orycteropodidae	Orycteropus	*afer*	Aardvark	Postcrania
BP/4	616	Tubulidentata	Orycteropodidae	Orycteropus	*afer*	Aardvark	Postcrania
BP/4	293	Tubulidentata	Orycteropodidae	Orycteropus	*afer*	Aardvark	Crania, postcrania
BP/4	371	Tubulidentata	Orycteropodidae	Orycteropus	*afer*	Aardvark	Crania, postcrania

Table 3.4. Human normal comparative collection; Pilot study conducted on 50 skeletons from the Dart Collection, Department of Anatomical Sciences, University of the Witwatersrand Medical School.

Accession Number	Age	Sex	Pathology	Comments
1813	?	F	Yes	Mild polyarticular osteoarthritic processes distributed throughout skeleton
1925	50	F	Yes	mild polyarticular osteoarthritis (lipping and porosity of articular surfaces) /
1927	38	M	no	none
1689	28	F	Yes	minor spinal osteoarthritis (marginal hypertrophic growth)
1875	14	F	no	none

Abnormal Modern Comparative Collections

Table 3.5. Non-human abnormal modern comparative collection; Data collected from the University of the Witwatersrand Bernard Price Institute (BPI) and Transvaal Museum (TVM).

Accession number	Taxonomic classification	Skeletal Parts affected	Type of anomaly
BPI			
58/72	Baboon; *Papio ursinus*	scapulae, cervical vertebrae, thoracic vertebrae, lumbar vertebrae	Diffuse and bilateral reactive bone formation and lytic foci. .
74/93	Baboon; *Papio ursinus*	left tibia and fibula	Malaligned healed fracture of the distal diaphysis of both the tibia and fibula resulting in the ankylosis (fusion) at the distal extent of both bones.
33/739	Klipspringer	right metacarpal	Hypertrophic bone development on the distal metaphysis and epiphysis
21/69	Sheep	left metacarpal and 1st phalanx	Ankylosis of the lateral 1st phalanx with the metacarpal.
BPI/C 261	Warthog	post-cranial skeleton	Diffuse, multifocal, and bilateral pathology resulting in proliferative reactive bone.
12/75	Leopard; *Panthera pardus*	left 2nd phalanx	Hypertrophic bone development and secondary joint formation; secondary osteoarthritis.
TVM			
AZ/631	Caracal	pelvis, left femur, and right femur	transverse fracture to the left femur distal of midshaft, with malaligned union
AZ/1855	Grey duiker	left and right calcanii, right metacarpal and 1st phalanges	bilateral abnormal bone proliferation to the distal ends of both calcanii,
AZ/2507	Caracal	right humerus, right radius, and right ulna	luxation at the junction of the humerus radius and ulna, secondary arthritic condition

AZ/613	Leopard; *Panthera pardus*	entire skeleton	Diffuse, multifocal, condition resulting in the warped or bowed appearance of most of the long bones
AZ/259	Tiger; *Panthera tigris*	left fibula	multiple fractures to the diaphysis of the left tibia resulting in extensive callus formation, secondary osteoarthritis, and possibly myositis ossificans traumatica

Table 3.6. Human abnormal modern comparative collection; Pilot study conducted on 50 skeletons from the Dart Collection, Department of Anatomical Sciences, University of the Witwatersrand Medical School.

Accession Number	Age	Sex	Lesion	Comments
			Multifocal or Solitary	
1803	37	M	multifocal	Perforation in sternum / periosteal dep. diaphysis of right fibula / periosteal dep. prox metatarsals / mild arthritic
1873	70	M	multifocal	perforation in sternum / bone projection rt humerus distal metaphysis / fused phalanges / severe arthritis
1918	40	F	multifocal	L5 detached neural arch spinal bifida? / fused thoracic vertebrae / bone projection on proximal phalanx (foot) / mild arthritis
1577	60	F	multifocal	mild arthritis on vertebra, hands, and feet / cervical with split lamina / pronounced bone growth on calcaneus
1932	30	M	multifocal	sacralized L6 / cauliflower bone growths on both taluses /
1904	72	F	multifocal	left radius and ulna fractured distal diaphysis (parry fracture) / ossified growth on sternum / osteoporotic / bone growth on metatarsal shafts
1579	60	M	multifocal	compression fracture of L3 secondary osteoarthritis / fusion of right ilium and sacrum / distal rt tibia and fibula fused proximal of fibular notch
617	19	F	multifocal	Osteogenesis imperfecta (type 1 or 2)/ evidences of multiple fractures of long bones, vertebral column severe angulation of lower extremities / reduction of size (reduction in diameter) and abnormal shape throughout skeleton / numerous wormian bones on skull
1697	35	F	multifocal	*atlas congenital deformity resulting in non-fusion* / *abnormal bone formation T11 inferior articular processes* / Schmorl's node L2 / perforated sternum /
1791	47	F	multifocal	distal rt fibula healed fracture
1906	60	M	multifocal	randomly distributed multifocal pathology / small lytic lesions present in acetabulum, on lesser tubercle of rt humerus, distal rt ulna, phalanges. Healed fracture with angulation of 5th ray proximal phalanx, hypertrophic bone development associated with fracture. Tuberculosis / minor polyarticular osteoarthritis
1576	30	F	multifocal	cartilaginous exostosis of rt tibia medial metaphysis / small cartilaginous exostosis rt fibula posterior metaphysis

1566	39	M	multifocal	spinal osteoarthritis cervical, thoracic, and lumbar; osteophytes present on diarthrodial and amphiarthrodial surfaces as well as costal facets / lower thoracic and lumbar also exhibit Schmorl's nodes. Small hypertrophic lesions on femur heads / healed fracture of left fibula diaphysis / fracture of thumb interphalangeal joint / fracture of pinky (5th ray) metacarpal
1797	65	M	multifocal	vertebral osteoarthritis; lipping, porosity of vertebral body surface, hypertrophic buildup and porosity of articular facets of lumbar vertebrae, Schmorl's nodes / striated periosteal reactive bone formation on the shafts of the long bones and ribs / small lytic lesions present on prox humerus, femur, and within the acetabulum
1771	40	M	multifocal	vertebral osteoarthritis; lipping (hypertrophic growth of margins) osteophytes on intervertebral articulations, osteophytes around costal articular surfaces of ribs / fracture of rt acromion process secondary joint formation for clavicle articulation? / dislocation 1st ray metacarpophalangeal joint
1775	80	M	multifocal	vertebral osteoarthritis; marginal lipping, osteophytes on intervertebral articulations, porosity of vertebral bodies / ossification of costal cartilage on sternum / sacrum fused to left ilium / lytic foci on femoral heads, humeral heads, fibula articular surfaces / reactive bone growth around tibial tuberosity, and scarring of linea aspera on femur / multifocal osteoarthritis
1907	70	M	multifocal	vertebral osteoarthritis / fractured ribs / perforated sternum / porous bone in acetabulum / fracture and infection of left prox femur detached femoral head (perhaps nonunion or subsequent necrotic collapse of femoral head, only minor evidence of remodeling) tunnel approximately 1cm in diameter running from the fovea capitis and exiting through the lateral most extent of new bone of the greater trochanter / fracture of tibia circular tunnel running through anterior crest at fracture zone (trauma; gunshot? infection; cavitation with removed sequestrum?) with angulation and multiple fractures of fibula / Periostosis of rt tibia diaphysis / rt fibula exostoses at attachment of the interosseous membrane / remodeling and hypertrophic bone development at rt fibula proximal articulation / osteoporosis ? Malnutrition / small lytic lesion in right glenoid cavity / polyarticular osteoarthritis (osteophytes, enthesophytes, porosity, etc.)
1687	40	M	multifocal	vertebral osteoarthritis; marginal lipping, osteophytes on intervertebral articulations, Schmorl's nodes / ossification of costal cartilage on sternum / reactive bone dep on margins of glenoid cavity w lytic foci / multifocal osteoarthritis
1790	60	M	multifocal	perforated sternum / compression fracture T10 / vertebral osteoarthritis; lipping of amphiarthrodial and diarthrodial joints / Schmorl's node in sacral body / hypertrophic bone deposition at radial tuberosity and lytic lesion / periosteal reactive bone dep on fibula

1685	36	F	multifocal	striated periosteal deposition (femur; along intertrochanteric line, rt tibia shaft and extensively on rt fibula) saber shin
1947	38	F	multifocal	hypervascular porous appearance of vertebral bodies and transverse processes / extensive reactive bone formation at the articulation of the rt scapula and clavicle / hypertrophic reactive bone rt humeral neck / bilateral lytic foci proximal radii articulations / bilateral reactive periosteal deposition lateral tibial diaphyses / misshapen phalanges / increased porosity (vascularity?) resulting in the post mortem damage to proximal and distal long bones / bilateral lytic foci within acetabula
1971	45	M	multifocal	vertebral osteoarthritis; destructive and hypertrophic lesions intervertebral articulations, costal facets, Schmorl's nodes / bilateral lytic foci on manubrium / bilateral bone growth with erosive cavities on humeri greater tubercles / bilateral hypertrophic bone formation adjacent to fovea capiti / polyarticular osteoarthritic processes
1961	60	F	multifocal	vertebral osteoarthritis; diarthrodial joint porosity, porosity and marginal osteophyte development on vertebral bodies, Schmorl's nodes and extensive lytic lesions on vertebral bodies / senile kyphosis? / porosity of glenoid cavity of left scapula / marginal cyst like erosion of glenoid cavity of rt scapula / bilateral erosive lesions on greater and lesser tubercles / multiple lytic lesions on lunate surfaces of pelvis, deepening of acetabular sockets / marginal osteophytes proximal and distal femur /
1958	19	F	solitary	defect in superior articular facets
1952	45	M	multifocal	porosity of C5 and C6 adjacent articular surfaces / marginal lytic focus rt prox humerus / rt ulna diaphysis healed fracture with slight angulation / rt humerus small cyst like erosion on radial tuberosity / proximal left ulna hypertrophic bone formation on the medial olecranon marginal of trochlear notch / rt femur diaphysis hypertrophic bone mid-shaft extending infero-laterally from the linea aspera
1932	30	M	multifocal	left and right saber tibia more extensive on rt / lytic foci on the anterior surface of several thoracic vertebrae /
1972	48	M	multifocal	small lytic focus C3 left inferior articular process / thoracic marginal lipping, increased porosity around costal and articular facets, hypertrophic bone deposition on inferior surface of laminae / marginal lipping on glenoid cavity, hypertrophic bone deposition on acromion processes / muscle scarring on interosseus crest and soleal line / left foot 1st ray metacarpal periosteal reactive bone deposition / bilateral hypertrophic deposition on patella anterior surface extending superiorly, moderate lipping
1991	34	F	multifocal	cervical clean except for C7 left transverse process hypertrophic dep / thoracic clean except T12 superior surface Schmorl's node / reactive bone and lytic foci anterior sacral surface / bilateral lytic activity medial of infraglenoid tubercles / diffuse periostosis fusiform bone hypertrophy on the diaphyses and metaphyses of both femurs, tibias, fibulas, radii, ulnas / rt ulna diaphysis healed fracture

2114	40	M	multifocal	T2 bifocal lytic lesions on costal facets / left distal fibula metaphyseal exostoses / rt patella anterior exostoses extend superiorly / minor polyarticular osteoarthritis / left foot 1st ray metatarsal head shows remodeling following trauma? subluxation? dislocation? angulation and remodeling of the distal articulation concomitant with changes in the proximal articulation of the proximal phalanx / the head of the right foot 1st metatarsal shows a medial cyst like lytic lesion with similar lesions on the proximal articulation of the left navicular / complete lytic obliteration of the distal articulations of two 1st foot phalanges / right foot 4th metatarsal fracture and healing
2101	60	M	multifocal	weakening of underlying cancellous bone (rarefaction of the spongiosa) / saber shin left tibia and fibula / minor periosteal dep on rt tibia and fibula / left femur exostoses on medial epicondyle heavy muscle scarring / minor periostosis of both humeri / cyst like erosions w/ post mortem damage associated with vertebrae, sacrum, pelvis, and scapulae ??? / polyarticular osteoarthritis, / rt foot 3rd ray metatarsal healed fracture / 1st ray metatarsals hypertrophic bone on distal articulations; rt shows eburnation
2113	89	M	multifocal	polyarticular osteoarthritis cervical diarthrodial and amphiarthrodial joint porosity, thoracic shmorle's nodes, diarthrodial eburnation and porosity, lumbar extensive lipping of diarthrodial joints, Schmorl's nodes, porosity, and marginal lipping / eburnation glenoid cavity humeral heads, rt prox tibia; porosity of joint surfaces and marginal lipping / multiple cyst like lytic lesions on ischium acetabular border /
2087	40	F	multifocal	congenital absence of spinous process of L5 and sacrum / minor polyarticular osteoarthritis / fractured and healed rib
2053	57	M	multifocal	C3 and C4 complete ankylosis / thoracic osteoarthritic processes resulting in marginal lipping and hypertrophic development of diarthrodial joint surfaces, in particular those on the rt side of the body / lumbar lipping, Schmorl's nodes and porosity / perforated sternum / acetabular lipping / eburnation of distal humerus, proximal radii, proximal tibia, / marginal lipping of long bone articulations / broken and healed clavicle / broken and healed rib
2102	31	M	none	some lytic activity on the femoral necks (normal?)
2040	90	M	multifocal	vertebral osteoarthritis; marginal lipping and osteophyte as well as porosity of the intervertebral articulations / fusion of left sacrum with pelvis / ossified hyoid / hypertrophic build up on both greater tubercles / healed fracture distal rt ulna (hairline) /minor bilateral periostosis on distal tibia and diaphyses of both fibulae in the form of small exostoses / minor polyarticular osteoarthritis / healed fractures proximal of distal articulations rt 2nd 3rd 4th and 5th metatarsals concomitant wit changes in proximal phalanges / misshapen 1st ray proximal phalanx (hand) square with marginal exostosis at distal articulation
2112	56	M	multifocal	perforated sternum / bilateral lytic foci on both acetabulum lunate surfaces / otherwise minor polyarticular osteoarthritis
1944	60	F	multifocal	large cyst like depression in rt acetabulum along with other lytic activity / minor periostosis on the metaphyses of both tibiae and fibulae

2142	48	M	multifocal	diffuse osteolytic metastases associated with carcinoma of the prostate / earliest and most massive involvement is the pelvis, sacrum, and lumbar vertebrae; destructive without any reactive bone formation around lytic margins / most lytic lesions on the pelvis are circular with diameters of approximately 1cm / similar lesions appear on the sacrum with destruction of the rt ala / much of the damage might be postmortem, the bone being weakened to the point of making it susceptible to damage / some vertebral bodies almost completely destroyed others with similar circular lytic lesions / complete destruction of the distal articulation of one of the proximal hand phalanges / minor lytic foci associated with carpals and metacarpals /
2018	30	M	multifocal	Schmorl's nodes T3-L3 / perforated sternum
2025	36	F	multifocal	polyarticular osteoarthritis / Schmorl's nodes and marginal lipping of vertebrae / rt radius distal of proximal articulation small exostoses / distal rt ulna healed fracture / minor lytic and hypertrophic lesions in both acetabula / fractured metacarpal resulting in lipping of the distal articulation /
2012	42	F	multifocal	minor polyarticular osteoarthritis / perforated sternum, and congenital incomplete fusion of sternum /
2067	50	F	multifocal	fractured rib / minor vertebral osteoarthritis / minor polyarticular osteoarthritis
2055	45	M	multifocal	periosteal hypertrophic growth on tibia and fibula / marginal lytic foci on humeral heads / Rt femur lytic lesion on lateral epicondyle /
1989	56	M	multifocal	complete ankylosis of rt femur with rt acetabulum (almost DISH-like in appearance; dripped candle wax) / Multiple exostoses arising from multiple ribs (bilateral) / complete ankylosis of T5 and rib heads at costal facets / exostosis on left humeral diaphysis / both tali / phalanges / despite the complete fusion and angulation of femur and acetabulum there are few or no proportionate corresponding changes to either lower limb / minor periostosis on both tibiae / multiple rib fractures / distal rt ulna diaphysis fractured and healed / asymmetry in distal humeri (possibly changes to rt distal humerus resulting from trauma to the limb)

Appendix C – Pathology by skeletal section

Table 6.9. Cooper's D faunal assemblage. Pathology by skeletal section.

	Forelimb	Hindlimb	Distal-limb	sternal	vertebral
Normal	208	236	950	119	226
Pathological	1	2	14	0	7
Total	209	238	964	119	233

Table 6.10. Cooper's D pathology by family and skeletal section.

	Normal	Pathological	Grand Total
Coopers D	**1739**	**24**	**1763**
Bovidae	**1262**	**7**	**1269**
distal-limb	608	5	613
forelimb	161		161
hindlimb	190		190
sternal	112		112
vertebral	191	2	193
Canidae	**24**		**24**
distal-limb	14		14
hindlimb	2		2
vertebral	8		8
Cercopithecidae	**102**	**3**	**105**
distal-limb	66	2	68
forelimb	13		13
hindlimb	12		12
sternal	1		1
vertebral	10	1	11
Equidae	**19**		**19**
distal-limb	8		8
forelimb	1		1
hindlimb	1		1

sternal	3		3
vertebral	6		6
Felidae	**143**	**6**	**149**
distal-limb	91	2	93
forelimb	20	1	21
hindlimb	22	1	23
sternal	3		3
vertebral	7	2	9
Hominidae		**2**	**2**
vertebral		2	2
Hyaenidae	**47**	**3**	**50**
distal-limb	40	2	42
forelimb	5		5
hindlimb	1	1	2
vertebral	1		1
Suidae	**142**	**3**	**145**
distal-limb	123	3	126
forelimb	8		8
hindlimb	8		8
vertebral	3		3

Table 6.11. Swartkrans faunal assemblage. Pathology by skeletal section.

	Forelimb	Hindlimb	Distal-limb	sternal	vertebral
Normal	1169	846	2939	52	338
Pathological	2	3	32	0	3
Total	1171	849	2971	52	341

Table 6.12. Swartkrans pathology by family and skeletal section.

	Normal	Pathological	Grand Total
Swartkrans	**5344**	**40**	**5384**
Bovidae	**4712**	**17**	**4729**
distal-limb	2543	14	2557
forelimb	1024		1024
hindlimb	766	1	767
sternal	52		52
vertebral	327	2	329
Canidae	**187**	**4**	**191**
distal-limb	121	3	124
forelimb	42		42
hindlimb	22	1	23
vertebral	2		2
Caprinae	**1**		**1**
distal-limb	1		1
Cercopithecidae	**148**	**2**	**150**
distal-limb	75		75
forelimb	44	2	46
hindlimb	27		27
vertebral	2		2
Elephantidae	**1**		**1**
forelimb	1		1
Equidae	**34**		**34**
distal-limb	31		31
forelimb	1		1
hindlimb	2		2
Felidae	**118**	**6**	**124**
distal-limb	78	5	83

forelimb	28		28
hindlimb	11	1	12
vertebral	1		1
Giraffidae	**4**		**4**
distal-limb	2		2
forelimb	2		2
Hippopotamidae	**3**		**3**
distal-limb	3		3
Hominidae	**56**	**11**	**67**
distal-limb	30	10	40
forelimb	14		14
hindlimb	8		8
vertebral	4	1	5
Hyaenidae	**80**		**80**
distal-limb	55		55
forelimb	13		13
hindlimb	10		10
vertebral	2		2

APPENDIX D – Pathology by family and element

The following figures represent the NISP by family for the sites of Cooper's D and Swartkrans. They give a graphical representation of the count of the fossils by skeletal element, including normal (blue) and pathological (red) elements.

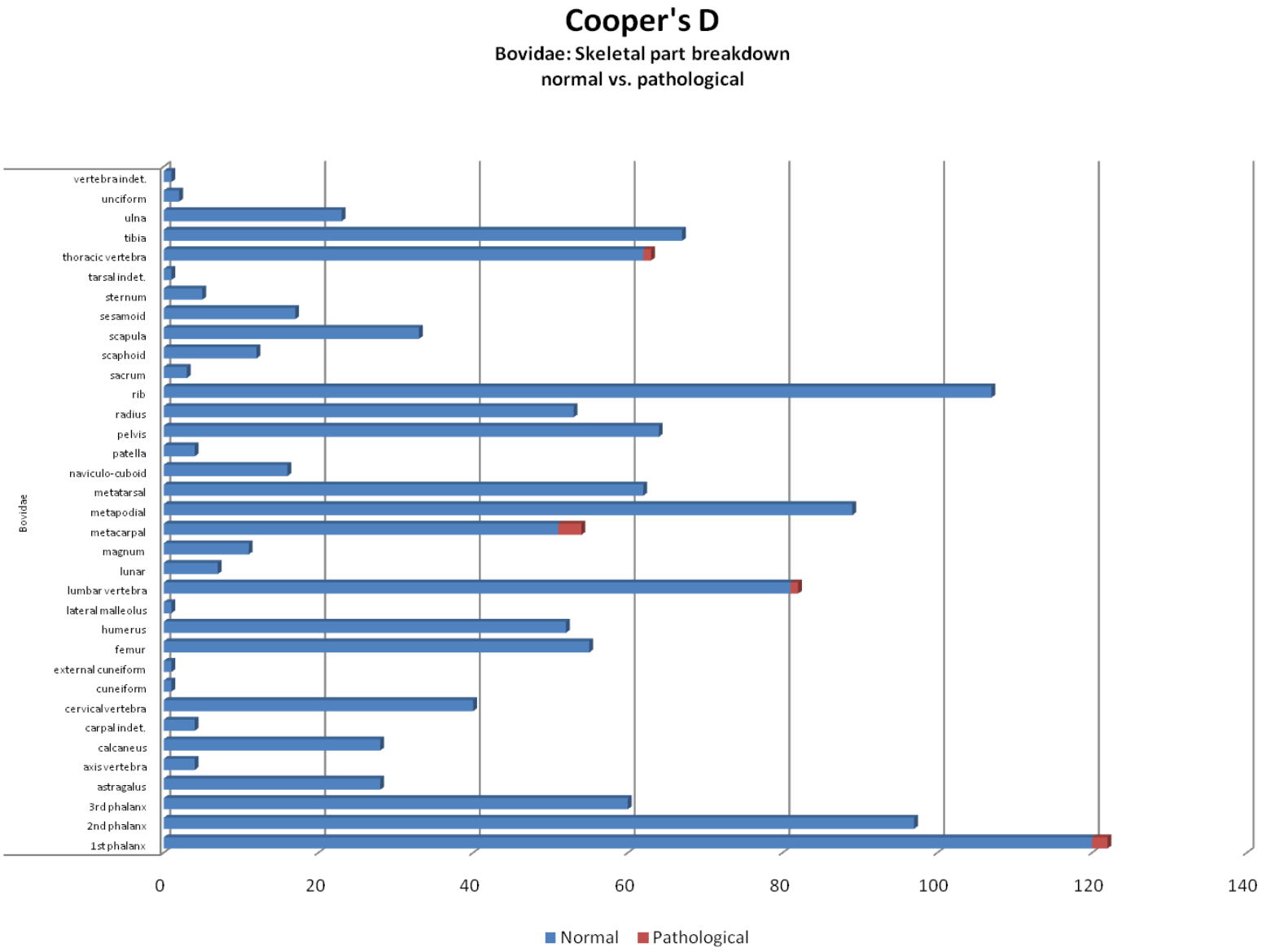


Figure 6.12. Cooper's D bovid NISP by element, comparing normal and pathological fossils.

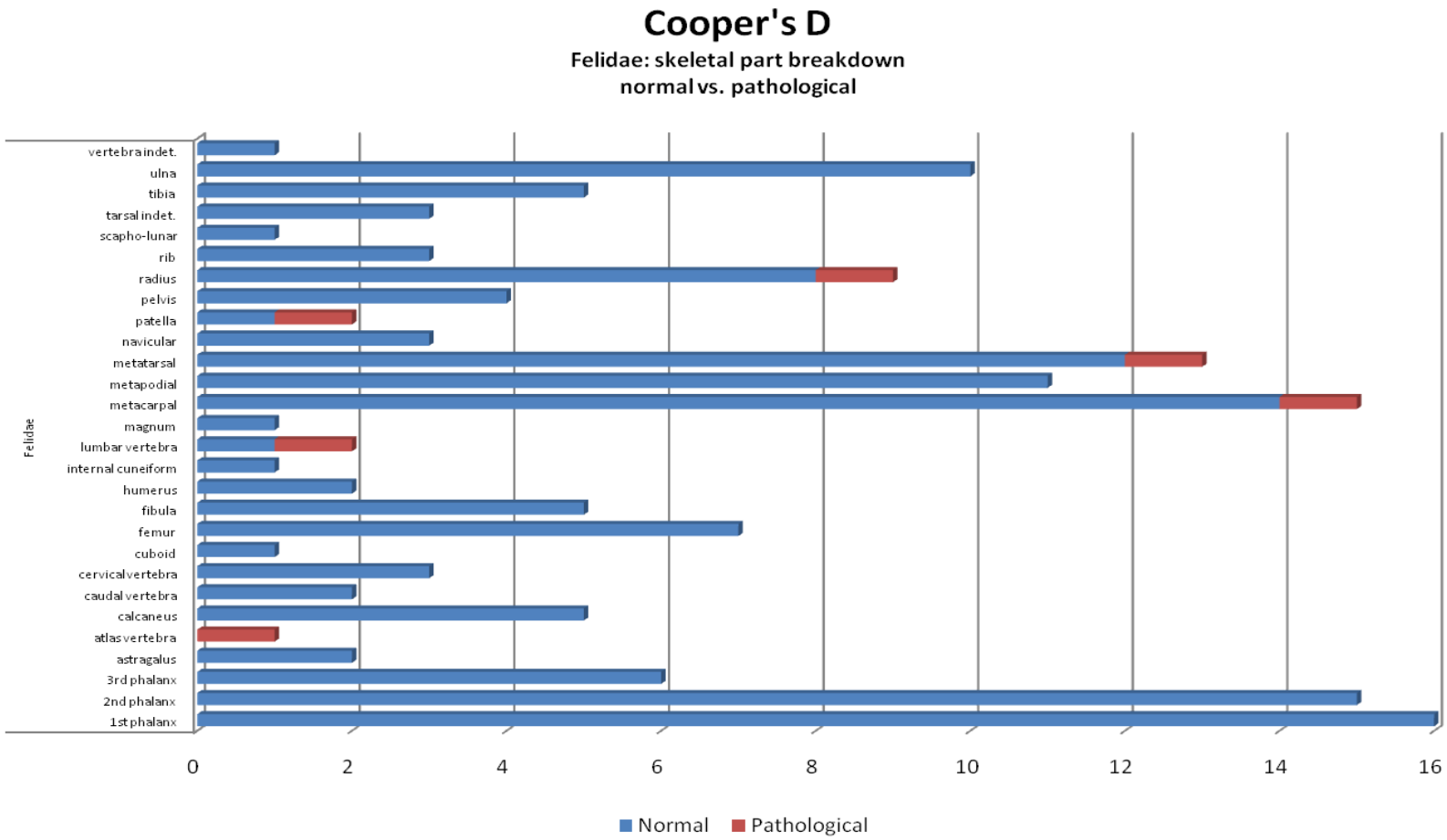


Figure 6.13. Cooper's D felid NISP by element, comparing normal and pathological fossils.

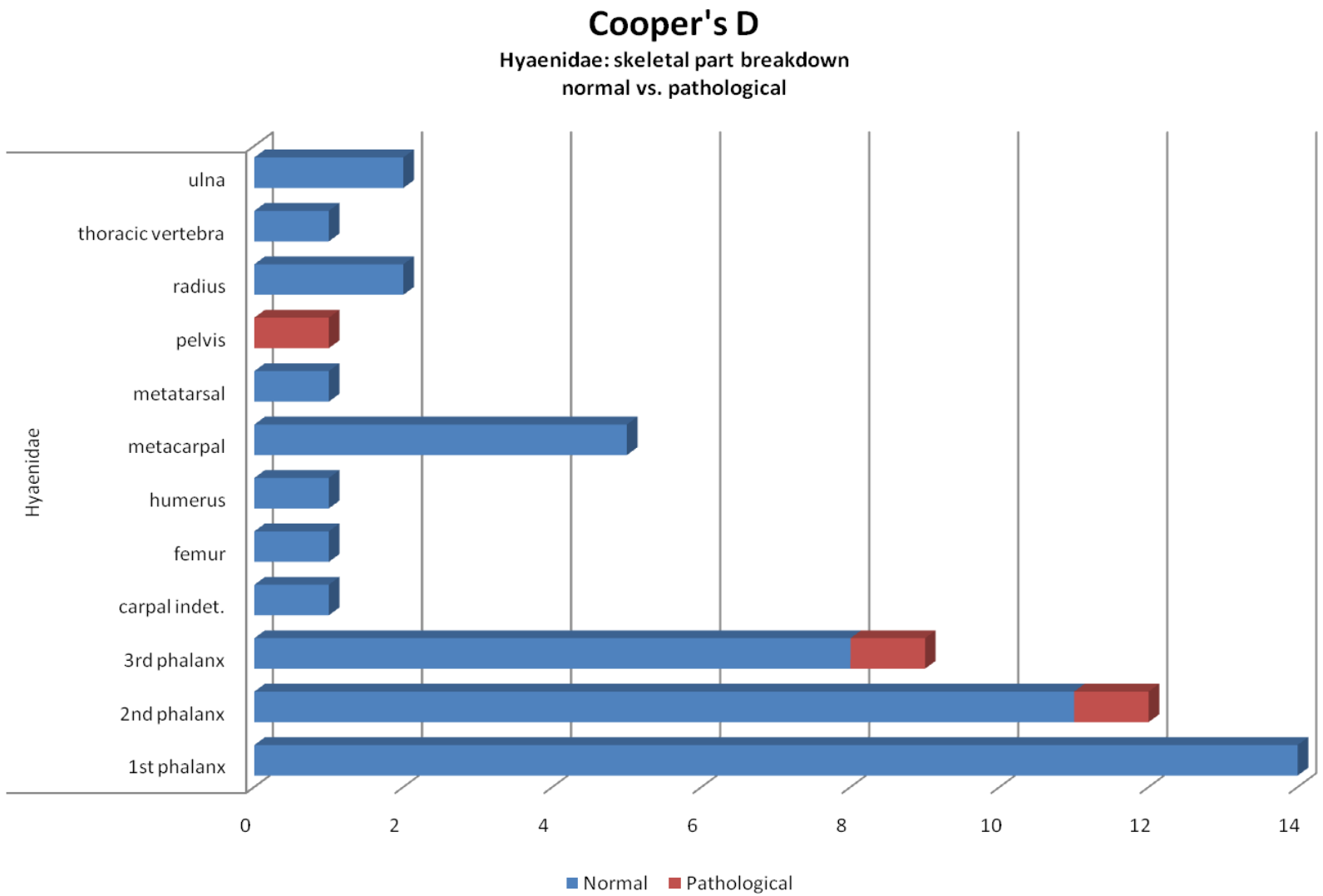


Figure 6.14. Cooper's D hyaenid NISP by element, comparing normal and pathological fossils.

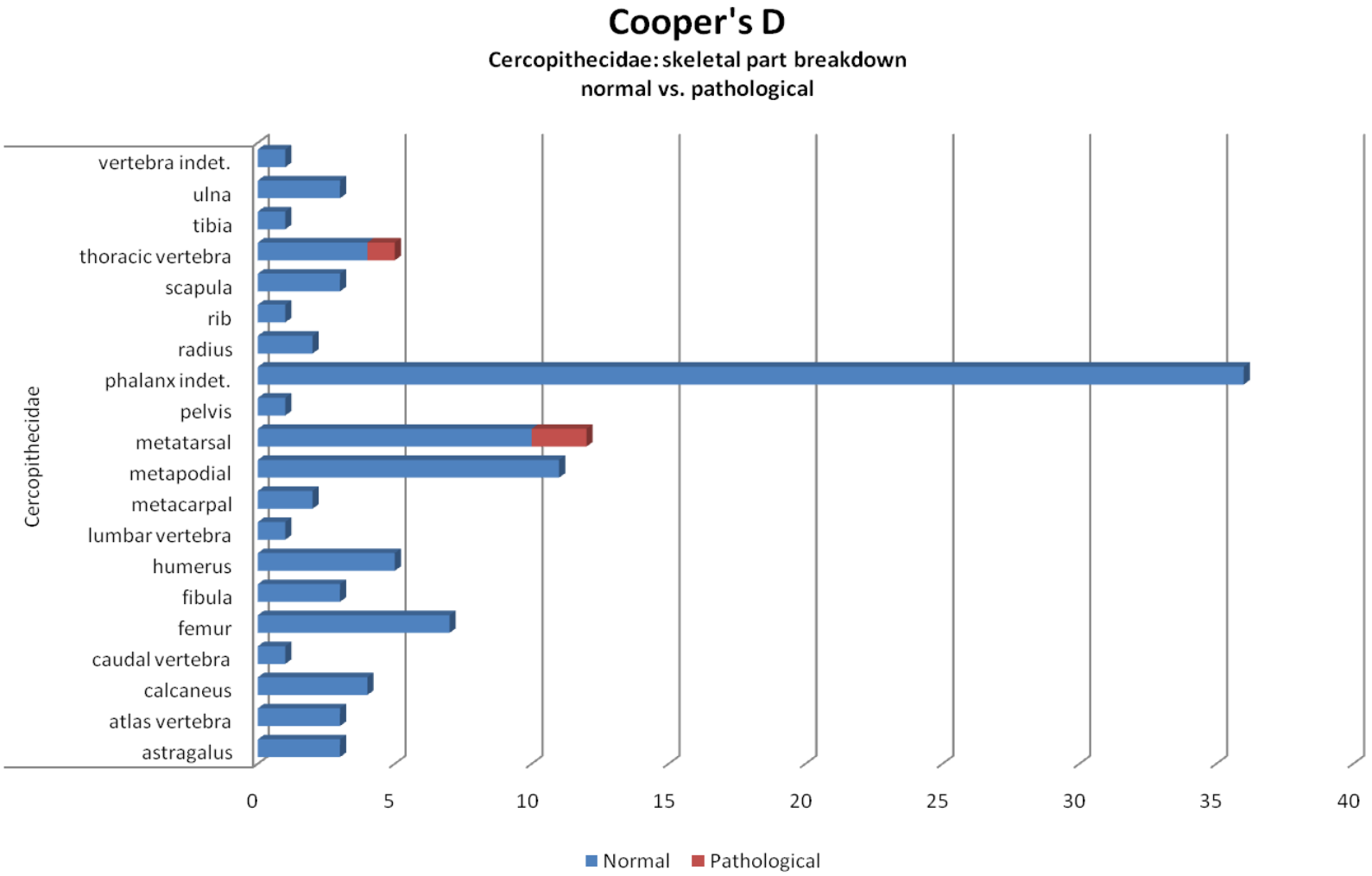


Figure 6.15. Cooper's D cercopithecid NISP by element, comparing normal and pathological fossils.

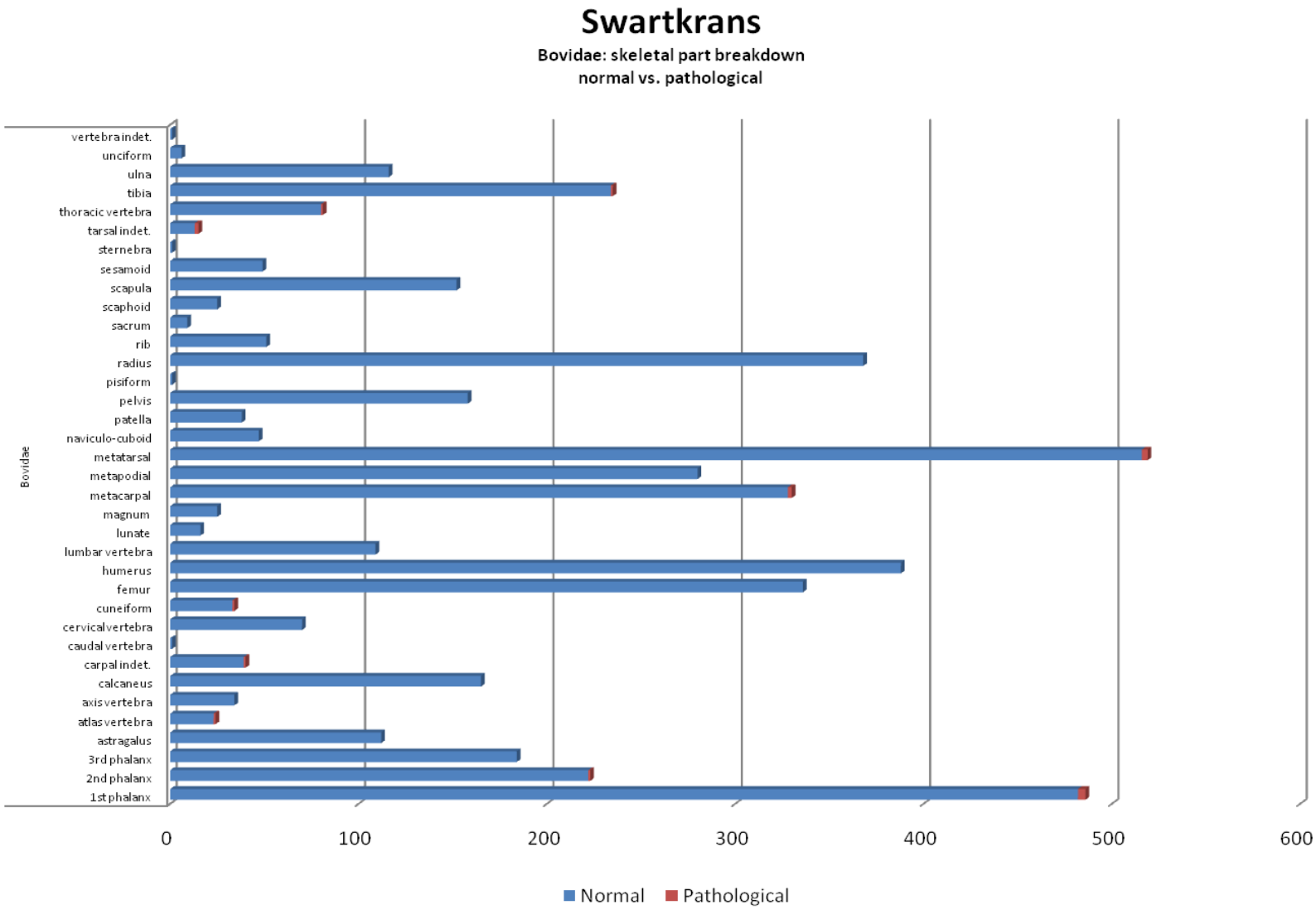


Figure 6.16. Swartkrans bovid NISP by element, comparing normal and pathological fossils.

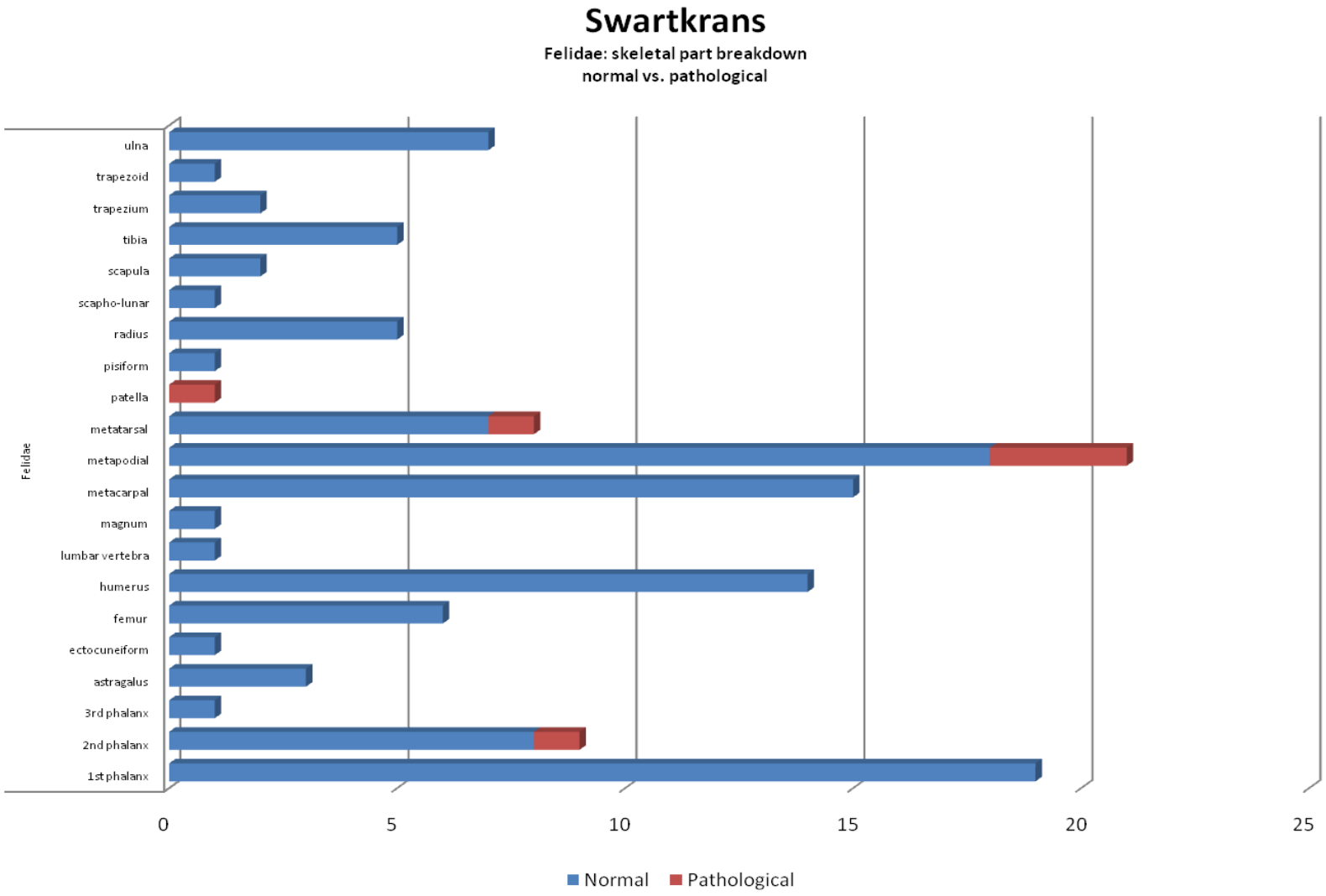


Figure 6.17. Swartkrans felid NISP by element, comparing normal and pathological fossils.

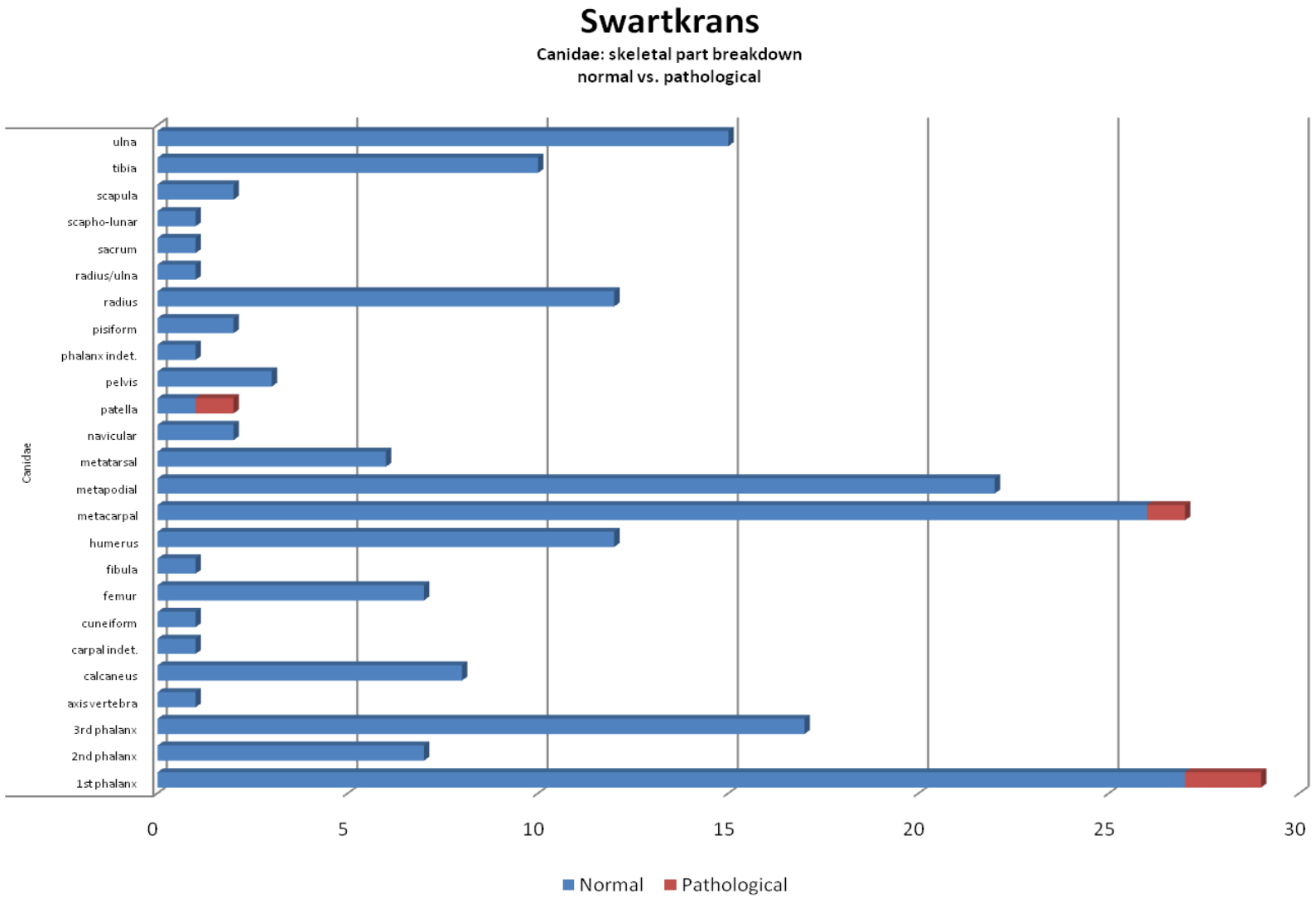


Figure 6.18. Swartkrans canid NISP by element, comparing normal and pathological fossils.

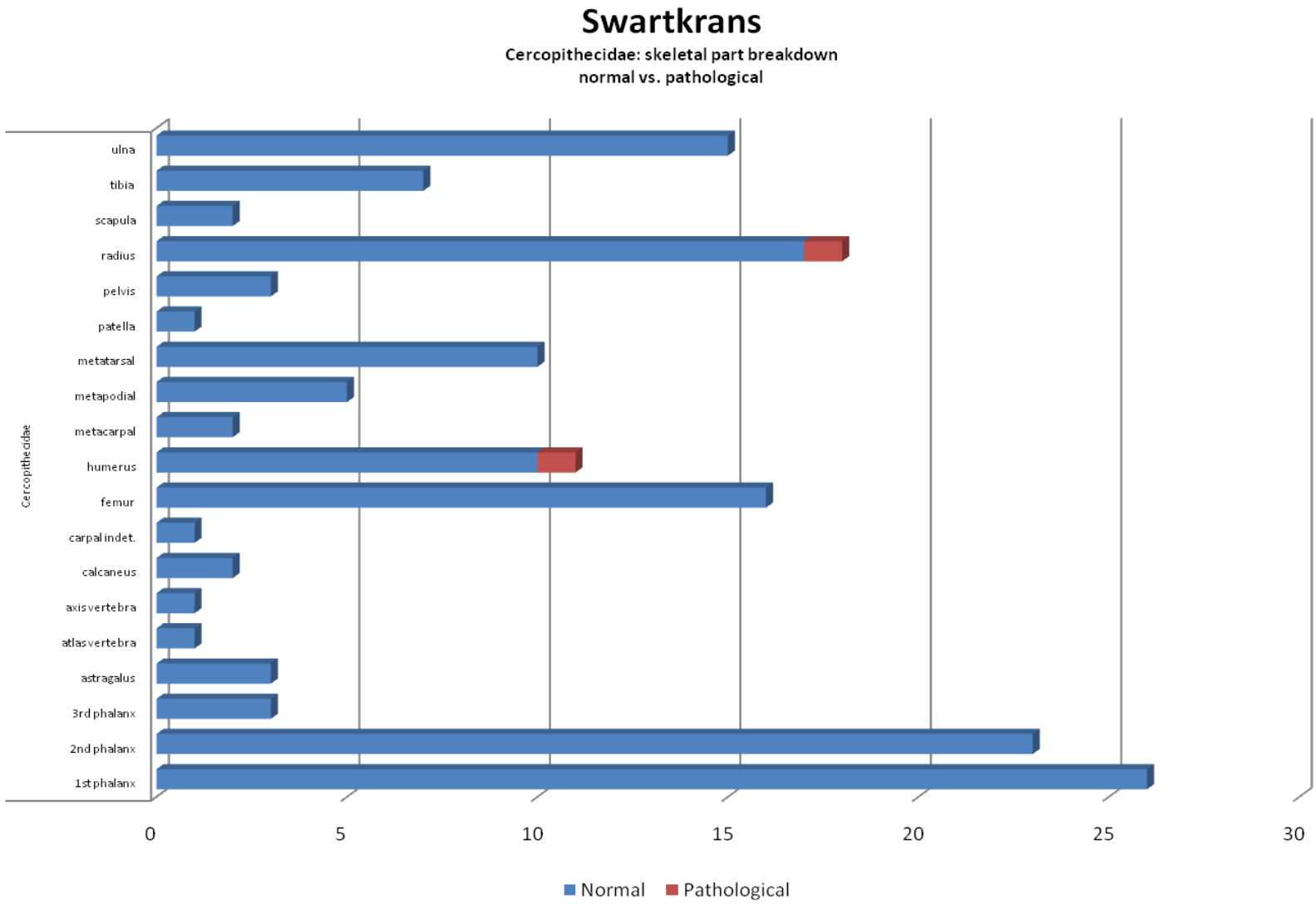


Figure 6.19. Swartkrans cercopithecid NISP by element, comparing normal and pathological fossils.

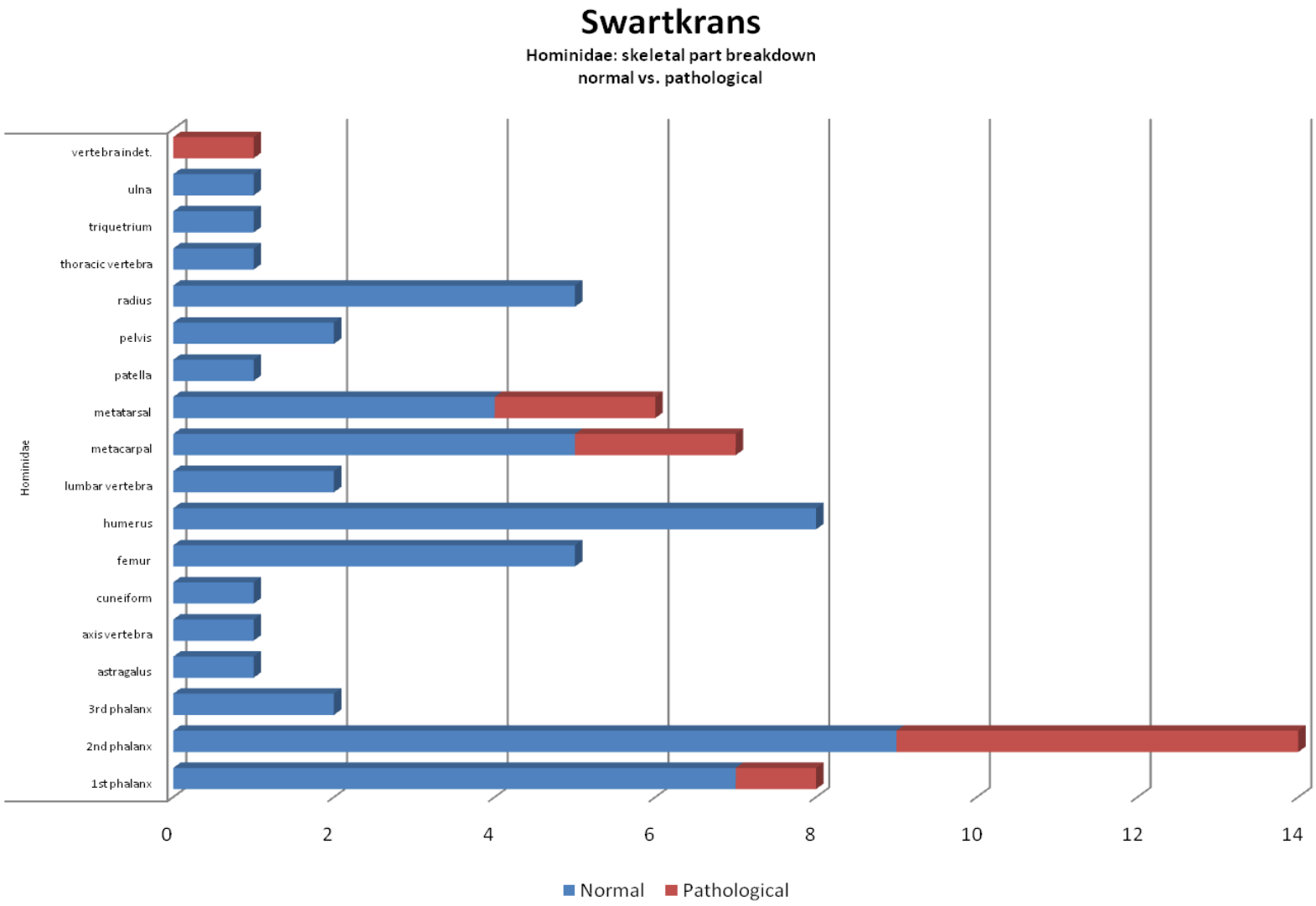


Figure 6.20. Swartkrans hominid NISP by element, comparing normal and pathological fossils.

References

Abraham, R. and Malkani, A.L. (2004) The aetiology and pathogenesis of osteonecrosis. *Seminars in Arthroplasty.* 15(3): 132-139.

Ackerknecht, H. (1955) *A Short History of Medicine.* Ronald Press Company, New York.

Agur, A.M.R. and Dalley, A.F. (2005) *Grant's Atlas of Anatomy, 11th edition.* Lippincott, Williams and Wilkins, Baltimore.

Anderson, W.A. and Scotti, T.M. (1976) *Synopsis of Pathology, 9th edition*. C.V. Mosby Company, Saint Louis.

Andrews, P. (1990) *Owls, Caves and Fossils.* University of Chicago Press, Chicago.

Ascenzi, A. (1969) Microscopy and Prehistoric Bone. *In*: Brothwell, D.R. and Higgs E., ed., *Science in Archaeology: A survey of progress and research*, pp. Thames and Hudson, London.

Aufderheide AC, Rodriguez-Martin C. (1998) *The Cambridge Encyclopaedia of Human Paleopathology,* Cambridge University Press, Cambridge.

Backwell, L.R. and d'Errico, F. (2001) Evidence of termite foraging by Swartkrans early hominids. *Proceedings of the National Academy of Sciences.* 98(4): 1358-1363;

Backwell, L.R. and d'Errico, F. (2003) Additional evidence on the early hominid bone tools from Swartkrans with reference to spatial distribution of lithic and organic artefacts. *South African Journal of Science.* 99: 259-267.

Backwell, L.R. and d'Errico, F. (2004) Additional evidence on early hominid bone tools from Swartkrans. *In*: Brain, C.K. ed., *Swartkrans, a cave's chronicle of early man,* pp. 265-268. Transvaal Museum Monograph No. 8. Pretoria.

Backwell, L.R. and d'Errico, F. (2008) Early hominid bone tools from Drimolen, South Africa. *Journal or Archaeological Science.* 35(11): 2880-2894.

Backwell, L.R., Pickering, R., Brothwell, D., Berger, L.R., Witcomb, M., Martill, D., Penkman, K. and Wilson, A. (2009) Probable human hair found in a fossil hyaena coprolite from Gladysvale Cave, South Africa. *Journal of Archaeological Science.* 36(6): 1269-1276.

Badenhorst, S. and Plug, I. (2001) Appendix. The faunal remains from Mmatshetsele, a Late Iron Age site in the Rustenburg district. *South African Archaeological Bulletin.* 56:55-56.

Badenhorst, S., Plug, I., Pelser, A.J. and Van Vollenhoven, A. (2002) Faunal analysis from Steinaecker's Horse, the northernmost British outpost in the Kruger National Park during the South African War. *Annals of the Transvaal Museum.* 39:57-63.

Baker, J. R. (1984) The study of animal diseases with regard to agricultural practices and man's attitude to his animals. *In:* Clutton-Brock, J. and Grigson, C. eds., *Animals and Archaeology: 4. Husbandry in Europe* (BAR International Series 227). Oxford: British Archaeological Reports.

Baker, J.R. and Brothwell, D. (1980) *Animal Diseases in Archaeology*. Academic Press, New York.

Balter, V., Blichert-Toft, J., Braga, J., Telouk, P., Thackeray, F. and Albarede, F. (2008) U-Pb dating of fosil enamel from the Swartkrans Pleistocene hominid site, South Africa. *Earth and Planetary Science Letters.* 267: 236-246.

Bartosiewicz, L., Van Neer, W. and Lentacker, A. (1994) Metapodial assymetry in draft cattle. *International Journal of Osteoarchaeology* 3: 69-75.

Bartosiewicz, L., Van Neer, W. and Lentacker, A. (1997) *Draught Cattle: Their Osteological Identification and History.* Koninklijk Museum voor Midden-Afrika, Annalen, Zoologische Wetenschappen, Vol. 281.

Bartosiewicz, L. (2008) Taphonomy and palaeopathology in archaeozoology. *Geobios.* 41(1): 69-77.

Bathurst, R.R and Barta, J.L. (2004) Molecular evidence of tuberculosis induced hypertrophic osteopathy in a 16th-century Iroquoian dog. *Journal of Archaeological Science* 31: 917-925.

Benjamin, M., Rufai, A. and Ralphs, J.R. (2000) The mechanism of formation of bony spurs (enthesophytes) in the Achilles tendon. *Arthritis and Rheumatism.* 43(3): 576-583.

Bennike, P. (1985) *Palaeopathology of Danish Skeletons: a comparative study of demography, disease and injury.* Akademisk Forlag, Copenhagen.

Berger, L.R., Pickford, M. and Thackery, J.F. (1995) A Plio-Pleistocene hominid upper central incisor from the Cooper's site, South Africa. *South African Journal of Science* 91: 541-542.

Berger, L.R., de Ruiter, D.J., Steiniger, C.M. and Hancox, J. (2003) Preliminary results of excavations at the newly investigated Coopers D deposit, Gauteng, South Africa. *South African Journal of Science* 99: 276-278.

Bjorkengren, A.G., Sartoris, D.J., Shermis, S. and Resnick, D. (1987) Patterns of paravertebral ossification in the prehistoric saber-toothed cat. *American Journal of Roentgenology.* 148(4): 779-782.

Boone, D., Parsons, D., Lachmann, S.M. and Sherwood, T. (1985) Spina bifida occulta: lesion or anomaly? *Clinical Radiology.* 36(2): 159-161.

Bourke, J.B. (1967) A Review of the Palaeopathology of the Arthritic Diseases. *In*: Brothwell, D.R. and Sandison, A.T., ed., *Diseases in Antiquity*, pp. 352-370. Springfield.

Brain, C.K. (1958) *The Transvaal Ape-Man-Bearing Cave Deposits.* Transvaal Museum Memoir No. 11, Pretoria.

Brain, C.K. (1968) Who killed the Swartkrans ape-men? *South African Museums Association Bulletin.* 9: 127-139.

Brain, C.K. (1969a) The probable role of leopards as predators of the Swartkrans Australopithecines. *South African Archaeological Bulletin.* 24: 170-171.

Brain, C.K. (1969b) The contribution of Namib Desert Hottentots to understanding of Australopithecine bone accumulations. *Scientific Papers of the Namib Desert research Station.* 32: 1-7.

Brain, C.K. (1974) Some suggested procedures in the analysis of bone accumulations from Southern African Quaternary sites. *Annals of the Transvaal Museum* 29(1)

Brain, C.K. (1981) *The Hunters or the Hunted? An Introduction to African Cave Taphonomy.* University of Chicago Press, Chicago.

Brain, C.K. ed. (1993) *Swartkrans: A Cave's Chronicle of Early Man*. Transvaal Museum Monograph, N. 8., Pretoria.

Brain, C.K. (1993a) Structure and stratigraphy of the Swartkrans Cave in light of the new excavations. *In:* Brain, C.K. ed. *Swartkrans: A Cave's Chronicle of Early Man*. Transvaal Museum Monograph, N. 8. Pretoria.

Brain, C.K. ed. (2004) *Swartkrans: A Cave's Chronicle of Early Man.* 2nd ed. Transvaal Museum Monograph, N. 8., Pretoria.

Brain, C.K. and Shipman, P. (1993) The Swartkrans bone tools. *In:* Brain, C.K. ed. *Swartkrans: A Cave's Chronicle of Early Man*. Transvaal Museum Monograph, N. 8. Pretoria.

Brickley, M., Mays, S. and Ives, R. (2005) Skeletal manifestations of vitamin D deficiency osteomalacia in documented historical collections. *International Journal of Osteoarchaeology*. 15(6): 389-403.

Brickley, M. and Ives, R. (2008) *The Bioarchaeology of Metabolic Bone disease.* Academic Press, San Diego.

Brothwell, D.R. (1967) The Evidence of Neoplasms. *In*: Brothwell, D.R. and Sandison, A.T., ed., *Diseases in Antiquity*, pp. 320-345. Springfield.

Brothwell, D.R. (1969) The palaeopathology of Pleistocene and more recent mammals. *In*: Brothwell, D. and Higgs, E., ed., *Science in Archaeology: A survey of progress and research*, pp. Thames and Hudson, London.

Brothwell, D.R. (1981) *Digging up Bones*, 3rd Ed. Cornell University Press, Ithaca.

Brothwell, D.R. (1991) On zoonoses and their relevance to palaeopathology. *In*: Ortner, D.J. and Aufderheide, A.C., eds., *Human Paleopathology: Current Synthesis and Future Options*, pp. 18-23. Smithsonian Institution Press, Washington, D.C.

Brothwell, D.R., Dobney, K. and Jaques, D. (2005) Abnormal sheep metatarsals. *In:* Davies, J., Fabis, M., Mainland, I., Richards, M. and Thomas, R. eds., *Diet and Health in Past Animal Populations: Current Research and Future Directions,* pp. 75-79.Oxbow Books, Oxford.

Brothwell, D. and Sandison, A.T. eds. (1967) *Diseases in Antiquity.* Charles C Thomas, Springfield.

Browner, B.D., Jupiter, J.B., Levine, A.M. and Krettek, C. (2003) *Skeletal Trauma: Basic Science, Management and Reconstruction, Volume 1*. 3rd ed. Saunders, Philadelphia.

Buikstra, J.E. and Cook, D.C. (1980) Palaeopathology: An American Account. *Annual Review of Anthropology.* 9: 433-470.

Carlson, K.J. and Pickering, T.R. (2003) Intrinsic qualities of primate bones as predictors of skeletal element representation in modern and fossil carnivore feeding assemblages. *Journal of Human Evolution.* 44(4): 431-450.

Chapman, C., Legge, S.S. and Johns, S.E. (2007) Canopy height utilization and trauma in three species of Ceropithecoid monkeys. *In*: Robson-Brown, K.A. and Roberts, A.M., eds., BABAO 2004. *Proceedings of the 6th Annual Conference of the British Association for Biological Anthropology and Osteoarchaology*, University of Bristol. Oxford.

Clarke, R.J., Clark Howell, F. and Brain, C.K. (1970) New finds at the Swartkrans Australopithecine Site: more evidence of an advanced hominid at Swartkrans. *Nature.* 225: 1219-1222.

Cooper, C. (1998) Epidemiology of osteoarthritis. *In*: Klippel, J. and Dieppe, P., eds., *Rheumatology*, 2nd ed. Mosby, London.

Couteur, R.A. and Grandy, J.L. (2000) Diseases of the spinal cord. *In*: Ettinger, S.J. and Feldman, E.C., eds. *Textbook of Veterinary Medicine.* W.B. Saunders, Philadelphia.

Cupere, B., de. Lentacker,, A. Van Neer, W., Waelkens, M. and Verslype, L. (2000) Osteological evidence for the draught exploitation of cattle: first applications of a new methodology. *International Journal of Osteoarchaeology* 10: 254-267.

Cutlip, R.G., Baker, B.A., Geronilla, K.B., Mercer, R.R., Kashon, M.L., Miller, G.R., Murlasits, Z. and Always, S.E. (2006) Chronic exposure to stretch-shortening contractions results in skeletal muscle adaptation in young rats and maladaptation in old rats. *Applied Physiology, Nutrition, and Metabolism.* 31(5): 573-587.

Czarnetzki, A., Jakob, T. and Pusch, C.M. (2003) Palaeopathological and variant conditions of the *Homo heidelbergensis* type specimen (Mauer, Germany). *Journal of Human Evolution*, 44: 479-495.

Daugnora L. and Thomas, R. (2005) Horse burials from Middle Lithuania: a palaeopathological investigation. *In*: Davies, J., Fabis, M., Mainland, I., Richards, M. and Thomas, R., eds. *Diet and Health in Past Animal Populations: Current Research and Future Directions.* Oxbow Books, Oxford.

Davies, J., Fabis, M., Mainland, I., Richards, M. and Thomas, R. eds. (2005) *Diet and Health in Past Animal Populations: Current Research and Future Directions.* Oxbow Books, Oxford.

Dawson, J.E. & Trinkaus, E. (1997) Vertebral osteoarthritis of the La Chapelle-aux-Saints 1 Neandertal. *Journal of Archaeological Science*. 24: 1015-1021.

Debono, L., Mafart, B., Jeusel, E., Guipert, G. (2004) Is the incidence of elbow osteoarthritis underestimated? Insights from paleopathology. *Joint, Bone, Spine*. 71(5): 397-400.

Degens, H. and Always, S.E. (2003) Skeletal muscle function and hypertrophy are diminished in old age. *Muscle and Nerve.* 27(3):339-347.

DeGusta, D. (2002) Comparative Skeletal Pathology and the Case for Conspecific Care in Middle Pleistocene Hominids. *Journal of Archaeological Science.* 29: 1435-1438.

DeGusta, D. (2003) Aubesier 11 is not evidence of Neanderthal conspecific care. *Journal of Human Evolution* 45: 831-834.

d'Errico, F. and Backwell, L.R. (2003) Possible evidence of bone tool shaping from the early hominid site of Swartkrans, South Africa. *Journal of Archaeological Science* 30: 1-31.

de Ruiter, D.J. (2001) *A methodological analysis of the relative abundance of hominids and other macromammals from the site of Swartkrans, South Africa.* PhD Thesis, Department of Anatomical Sciences, University of the Witwatersrand, South Africa.

de Ruiter, D.J. (2003) A revised faunal list for the Australopithecine-bearing Members of Swartkrans. *Annals of the Transvaal Museum.* 39: 29-41.

de Ruiter, D.J. (2004) Relative abundance, skeletal part representation and accumulating agents at Swartkrans. *In*: Brain, C.K. ed., *Swartkrans, a cave's chronicle of early man,* pp. 265-268. Transvaal Museum Monograph No. 8. Pretoria.

de Ruiter, D.J. and Berger, L.R. (2000) Leopards as taphonomic agents in dolomitic caves – implications for bone accumulations in the hominid-bearing deposits of South Africa. *Journal of Archaeological Science.* 27(8): 665-684.

de Ruiter, D.J., Pickering, R., Steininger, C.M., Kramers, J.D., Hancox, P.J., Churchill, S.E., Berger, L.R. and Backwell, L.R. (2009) New hominin fossils from Cooper's Cave (Gauteng, South Africa) and the first U-Pb date for *Australopithecus robustus. Journal of Human Evolution.* 56(5): 497-513.

Dimitri, P. and Bishop, N. (2007) Rickets. *Paediatrics and Child Health.* 17(7): 279-287.

Djurić, M.P., Roberts, C.A., Rakočević, Z.B., Djonić, D.D., and Lešić A.R. (2005) Fractures in late medieval skeletal populations from Serbia. *American Journal of Physical Anthropology.* 130(2): 167-178.

Dobney, K. and Rielly, K. (1988) A method for recording archaeological animal bones: the use of diagnostic zones. *Circaea.* 5(2): 79-96.

Drapeau, M.S.M. (2008) Enthesis bilateral asymmetry in humans and African apes. *HOMO.* 59(2): 93-109.

Dunn, J. (1999) *Textbook of Small Animal Medicine.* Saunders, Philadelphia.

Ekman, S. and Carlson, C.S. (1998) The pathophysiology of osteochondrosis. *Veterinary Clinics of North America: small animal practice* 28: 17-32.

Engelkens, H.J.H., Vuzevski, V.D. and Stolz, E. (1999) Nonvenereal treponematoses in tropical countries. *Clinics in Dermatology.* 17(2): 143-152.

Fabiš, M. (2004) Palaeopathology of Findings among Archaeofaunal Remains of Small Seminar Site in Nitra. *Acta Veterinaria Brno* 73 (1): 55-58

Farrow, C.S. (2003) *Veterinary Diagnostic Imaging: the dog and cat.* Mosby, Elsevier Science, St. Louis.

Fennell, K.J. & Trinkaus, E. (1997) Bilateral femoral and tibial periostitis in the La Ferrassie 1 Neandertal. *Journal of Archaeological Science*. 24: 985-995.

Fidas, A., MacDonald, H.L., Elton, R.A., Wild, S.R., Chisholm, G.D. and Scott, R. (1987) Prevalence and patterns of spina bifida occulta in 2707 normal adults. *Clinical Radiology.* 38(5): 537-542.

Foo, L.F. and Raby, N. (2005) Tumours and tumour-like lesions in the foot and ankle. *Clinical Radiology.* 60(3): 308-332.

Frost, H.M. and Wu, K. (1967) Histological measurements of bone formation rates in contemporary archaeological and paleontological compact bone. *In*: Wade, W. ed., *Miscellaneous Papers in Paleopathology.* Museum of Northern Arizona Press, Flagstaff.

Frost, H.M. (1969) Tetracycline-based histological analysis of bone remodeling. *Calcified Tissue Research,* 3: 211-237.

Gaulke, R. and Suppelna, G. (2004) Solitary enchondromas at the hand. *Journal of Hand Surgery: Journal of the British Society for Surgery of the Hand.* 29(1): 64-66.

Goldstein, M. (1969) The Palaeopathology of Human Skeletal Remains. *In*: Brothwell, D and Higgs, E., ed., *Science in Archaeology: A survey of progress and research*, pp. Thames and Hudson, London.

Goodman, A.H. (1993) On the interpretation of health from skeletal remains. *Current Anthropology* 34(3): 281-288.

Grauer, A. and Roberts, C. (1998) Paleoepidemiology, healing, and possible treatment in the medieval cemetery of St. Helen-on-the-Walls, York, England. *American Journal of Physical Anthropology* 100(4): 531-544.

Grayson, D.K. (1979) On the quantification of vertebrate archaeofaunas. *In*: Schiffer, M.B., ed., *Advances in archaeological method and theory* vol. 2, pp 199-237. Academic Press, New York.

Greer, M., Greer, J.K., and Gillingham, J. (1977) Osteoarthritis in selected wild mammals. *Proceedings from the Oklahoma Academy of Science.* 57: 39-43.

Gregg, J.B., and Gregg, P.S. (1987) *Dry Bones: Dakota Territory Reflected.* Sioux Printing, Sioux Falls.

Grine, F.E. (1993) Description and preliminary analysis of new hominid craniodental fossils from the Swartkrans Formation. *In*: Brain, C.K. *Swartkrans: A Cave's Chronicle of Early Man*. Transvaal Museum Monograph, N. 8. Pretoria.

Gutierrez , M.C., Brisse, S., Brosch, R., Fabre, M., Omais, B., Marmiesse, M., Supply, P. and Vincent, V. (2005) Ancient origin and gene mosaicism of the progenitor of *Mycobacterium tuberculosis. PLoS Pathogens.* 1(1):e5.

Hall, B.K. (2005) *Bones and Cartilage: Developmental and Evolutionary Skeletal Biology.* Elsevier Academic Press. San Diego.

Hall, S.R., Lafferty, K.D., Brown, J.H., Caceres, C.E., Chase, J.M., Dobson, A.P., Holt, R.D., Jones, C.G., Randolph, S.E. and Rohani, P. (2008) Is infectious disease just another type of predator-prey interaction? *In*: Ostfeld, R.S., Keesing, F. and Eviner, V.T. eds., *Infectious Disease Ecology: the effects of ecosystems on disease and of disease on ecosystems.*pp. 223-241. Princeton University Press, Princeton.

Harper, G.D., Dicks-Mireaux, C. and Leiper, A.D. (1998) Total body irradiation-induced osteochondromata. *Journal of Pediatric Orthopedics.* 18(3): 356-358.

Hawkey, D. and Merbs, C.F. (1995) Activity induced musculoskeletal stress markers (MSM) and subsistence strategy changes among ancient Hudson Bay Eskimos. *International Journal of Osteoarchaeology.* 5: 324-338.

Hendey, Q.B. (1974) The Late Cenozoic Carnivora of the South Western Cape Province. *Annals of the South African Museum*. 63: 185.

Hesse, P. and Wapnish, B. (1985) *Animal Bone Archaeology*. Taraxacum. Washington, D.C.

Higham, C.F.W., Kijngam, A., Manly, B.F.J. and Moore, S.J.E. (1981) The bovid third phalanx and prehistoric ploughing. *Journal of Archaeological Science* 8, 353-65.

Hopley, P.J. (2001) Pleisiosaur spinal pathology: The first fossil occurrence of Schmorl's Nodes. *Journal of Vertebrate Paleontology*. 21(2) 253-260.

Hudson, P.J., Grenfell, B.T., Hasterbeek, H. and Dobson, A.P. eds. (2008) *The Ecology of Wildlife Diseases*. Oxford University Press, New York.

Hwang, S.K. and Park, B. (1991) Induction of osteochondromas by periosteal resection. *Orthopedics.* 14(7): 809-812.

Inhorn M.C. and Brown, P.J. (1990) The anthropology of infectious disease. *Annual Review of Anthropology.* 19: 89-117.

Işcan, M.Y. and Kennedy, K.A.R. (1989) *Reconstruction of Life from the Skeleton*. Wiley-Liss Inc. New York.

Janssens, P.A. (1970) *Palaeopathology: Diseases and Injuries of Prehistoric Man*. John Baker, London.

Jarcho, S. (1966) *Human Paleopathology*. Yale University Press, New Haven.

Johnson, S., Houser, B.S., Frederick, H. and Patterson, B. (2008) Osteochondrosis dissecans in a juvenile roan antelope. *Journal of Zoo and Wildlife Medicine.* 39(1): 111-114.

Judd, M.A. (2008) The parry problem. *Journal of Archaeological Science.* 35(6): 1658-1666.

Judex, S. and Zernicke, R.F. (2000) High-impact exercise and growing bone: relation between high strain rates and enhanced bone formation. *Journal of Applied Physiology.* 88(6): 2183-2191.

Jurmain, R. (1999) *Stories from the Skeleton: behavioral reconstruction in human osteology*. Gordon and Breach, Amsterdam.

Jurmain, R. and Roberts, C. (2008) Juggling the evidence: the purported 'acrobat' from Tell Brak. *Antiquity.* 82(318)

Kahn, C.M. and Line, S. eds., (2005) *Merck Veterinary Manual.* Merck and Company Inc., Whitehouse Station.

Kalichman, L., Malkin, I. and Kobylianski, E. (2007) Hand bone midshaft enthesophytes: the influence of age, sex, and heritability. *Osteoarthritis and Cartilage.* 15(10): 1113-1119.

Kamath, B., Menezis, R., Binu, S. and Bhardwaj, P. (2007) Solitary osteochondroma of the metacarpal. *Journal of Hand Surgery.* 32(2): 274-276.

Kannus, P., Sievänen, H. and Vuori, I. (1996) Physical loading, exercise, and bone. *Bone.* 18(1): S1-S3.

Kennedy, K.A.R. (1989) Skeletal markers of occupational stress. *In*: Işcan, M.Y. and Kennedy, K.A.R. eds., *Reconstruction of Life from the Skeleton.* pp. 129-160. Wiley-Liss. New York.

Kerley, E.R. and Bass, W.M. (1967) Paleopathology: Meeting Ground for Many Disciplines. *Science* 157: 638-644.

Klein, R.G. and Cruz-Uribe, K. (1984) *The Analysis of Bones from Archaeological Sites.* University of Chicago Press. Chicago.

Kolmstetter, C., Munson, L. and Ramsey, E.C. (2000) Degenerative spinal disease in large felids. *Journal of Zoo and Wildlife Medicine.* 31(1): 15-19.

Kontogeorgakos, V.A., Lykissas, M.G., Mavrondontidis, A., Sioros, V., Papachristou, D., Batsitatou, A. and Beris, A. (2007) Turret exostosis of the hallux. *The Journal of Foot and Ankle Surgery.* 46(2): 130-132.

Kuhn, B.F. (2006) *The Collecting Behaviour and Taphonomic Signatures of Hyaenids.* PhD Thesis, University of the Pretoria, South Africa.

L'Abbe, E. (2003) Uncovering a nineteenth-century typhoid epidemic at the Koffiefontein Mine, South Africa. *World Archaeology.* 35(2): 306-318.

Lavigne, S. (2009) *Monkeying Around: Spatial differences in riverine habitat utilization by Vervets, Sykes, and Baboons*. SIT Tanzania, Wildlife and Ecology Conservation.

Lebel, S., Trinkaus, E., Faure, M., Fernandez, P., Guerin, C., Richter, D., Mercier, N., Valladas, H., Wagner, G.A. (2001) Comparative morphology and paleobiology of Middle Pleistocene human remains from the Bau de l'Aubesier, Vaucluse, France. *Proceedings of the National Academy of Sciences USA.* 98: 11097-11102.

Levitan, B. (1985) A methodology for recording the pathology and other anomalies of ungulate mandibles from archaeological sites. *In*: Fieller, N.R.J., Gilbertson, D.D. and Ralph, N.G.A. eds., *Palaeobiological Investigations: research design, methods and data analysis.* pp 41-56. BAR International Series, Oxford.

Lipowitz, A.J. and Newton, C.D. (1985) Degenerative joint disease and traumatic arthritis. *In*: Newton, C.D. and Nunamaker, D.M. eds., *Textbook of Small Animal Orthopaedics.* International Veterinary Information Service, Ithaca.

Lovejoy, C. (2005) The natural history of human gait and posture: Part 1. Spine and pelvis. *Gait and Posture.* 21:95-112.

Lovell, N.C. (1997) Trauma Analysis in Paleopathology. *Yearbook of Physical Anthropology.* 40: 139-170.

Lyman, R.L. (1994) *Vertebrate Taphonomy.* Cambridge University Press, Cambridge.

Lyman, R.L. (2008) *Quantitative Paleozoology.* Cambridge University Press, Cambridge.

Marcsik, A., Fothi, E., Hegyi, A. (2002) Paleopathological changes in the Carpathian Basin in the 10th and 11th centuries. *Acta Biologica Szegediensis*. 46(1-2): 95-99.

Mariotti, V., Facchini, F., and Belcastro, M.G. (2004) Enthesopathies: proposal of a standardized scoring method and applications. *Collegium Anthropologicum.* 28(1): 145-159.

Marks, M.K. and Hamilton, M.D. (2007) Metastatic carcinoma: palaeopathology and differential diagnosis. *International Journal of Osteoarchaeology.* 17(3): 217-234.

Marshall, L.G. (1989) Bone modification and the laws of burial. *In*: Bonnichsen, R. and Sorg, M.H., ed. *Bone Modification,* pp.7-24. University of Maine Study of the First Americans.

Martin, D.L. (1991) Bone Histology and Paleopathology: Methodological Considerations. *In:* Ortner, D. and Aufderheide, A.C., ed. *Human Paleopathology: Current Synthesis and Future Options,* pp. 55-56. Washington, D.C.

Martin, E.A. ed, (2003) *Oxford Concise Medical Dictionary*, 6th ed. Oxford University Press, Oxford.

Martin, R.B. (2002) Is all cortical bone remodeling initiated by microdamage? *Bone.* 30(1): 8-13.

Martin, T.J. and Rodan, G.A. (2001) Coupling of bone resorption and formation during bone remodeling. *In*: Marcus, R., Feldman, D., and Kelsey, J., eds. *Osteoporosis.* 2nd ed. pp. 361-372. Academic Press. San Diego.

Matsumoto, H., Kawakubo, M., Otani, T. and Fujikawa, K. (1999) Extensive post-traumatic ossification of the patellar tendon. *Journal of Bone and Joint Surgery.* 81: 34-36.

Mays, S.A. (2008) A likely case of scurvy from early Bronze Age Britain. *International Journal of Osteoarchaeology.* 18(2): 178-187.

McDonald, J.H. (2008) *Handbook of Biological Statistics.* Sparky House Publishing, Baltimore.

Meheus, A. (2005) Non-venereal treponematoses. *Medicine.* 33(10): 82-84.

Miller, E., Ragsdale, B., and Ortner, D. (1996) Accuracy in dry bone diagnosis: A comment on palaeopathological methodology. *International Journal of Osteoarchaeology,* 6: 221-229.

Minkowitz, P., Patel, M. and Minkowitz, S. (1992) Scaphoid enchondroma. *Orthopaedic Review.* 21(10): 1241-1245.

Mintzer, C.M., Klein, J.D. and Kasser, J.R. (1994) Post-traumatic osteochondroma formation after a Salter II fracture. *Journal of Orthopaedic Trauma.* 8(5): 437-439.

Mohanna, P.N., Moiemen, N.S. and Frame, J.D. (2000) Turret exostosis of the thumb. *British Journal of Plastic Surgery.* 53(7): 629-631.

Moodie, R.L. (1967) General Considerations of the Evidences of Pathological Conditions Found Among Fossil Animals (1917). *In*: Brothwell, D.R. and Sandison, A.T., eds., *Diseases in Antiquity,* pp. 31-46. Springfield.

Moodie, R.L. (1923) *Paleopathology: An Introduction to the Study of Ancient Evidences of Disease*. University of Illinois Press, Urbana.

Morgan, J.P., Wind, A. and Davidson, A.P. (2000) *Hereditary Bone and Joint Diseases in the Dog: osteochodroses, hip dysplasia, elbow dysplasia.* Schlűtersche GmbH & Co., Hannover.

Mosothwane, M.N. and Steyn, M. (2008) In sickness or in health? Assessment of Early Iron Age human skeletons from Toutswe sites, east central Botswana. *International Journal of Osteoarchaeology.* 19(1): 66-77.

Murphey, M.D., Choi, J.J., Kransdorf, M.J., Flemming, D.J. and Gannon, F.H. (2000) Imaging of osteochondroma: variants and complications with radiologic-pathologic correlation. *Radiographics* 20: 1407-1434.

Murphy, E.J. (1972) The Normal, and the perils of the sylleptic argument. *Perspect. Biol. Med.* 566-582.

Murphy, E. (2005) Animal palaeopathology in prehistoric and historic Ireland: a review of the evidence. *In*: Davies, J., Fabiš, M., Mainland, I., Richards, M. and Thomas, R. eds. *Diet and Health in Past Animal Populations: current research and future directions.* Oxbow, Oxford.

Newton, C.D. and Nunamaker, D.M. eds. (1985) *Textbook of small animal orthopaedics.* International Veterinary Information Service, Ithaca.

O'Connor, T. (1984) *Selected groups of bones from Skeldergate*. Council for British Archaeology, York.

O'Connor, T. (2008) On the differential diagnosis of arthropathy in bovids. *In*: Grupe, G., McGlynn, G. and Peters, J. eds., *Limping Together Through the Ages.* pp. 165-186. Documenta Archaeobiologae 6.

Ortner, D.J. (1991) Theoretical and methodological issues in palaeopathology. *In:* Ortner, D.J. and Aufderheide, A.C. eds. *Human Paleopathology: current synthesis and future options.* pp 5-11. Smithsonian Institution Press, Washington, D.C.

Ortner, D.J. (2003) *Identification of Pathological Conditions in Human Skeletal Remains.* Academic Press, Orlando.

Ortner, D.J. and Mays, S.A. (1998) Dry bone manifestations of rickets in infancy and childhood. *International Journal of Osteoarchaeology.* 8(1): 45-55.

Ortner, D.J., Manchester, K. and Lee, F. (1991) Metastatic carcinoma in a leper skeleton from a medieval cemetery in Chichester, England. *International Journal of Osteoarchaeology.* 1: 91-98.

Ostblom, L., Lund, C. and Melsen, F. (1982) Histological study of navicular bone disease. *Equine Veterinary Journal.* 14(3): 199-202.

Ostfeld, R.S., Keesing, F. and Eviner, V.T. eds. (2008) *Infectious Disease Ecology: the effects of ecosystems on disease and of disease on ecosystems.* Princeton University Press, Princeton.

Oxford Concise Medical Dictionary, 6th ed. (2003) Oxford University Press, Oxford.

Owens, L.S. (2007) Craniofacial trauma in the Prehispanic Canary Islands. *International Journal of Osteoarchaeology.* 17(5): 465-478.

Parfitt, A.M. (1990) Osteomalacia and related disorders. *In*: Avioli, L.V. and Krane, S.M., eds., *Metabolic Bone Disease and Clinically Related Disorders.* 2nd ed. pp. 329-396. W.B. Saunders, Philadelphia.

Parfitt, A.M. (1994) The two faces of growth: benefits and risks to bone integrity. *Osteoporosis International.* 4(6): 382-398.

Parfitt, A.M. (2002) Misconceptions (2): Turnover is always higher in cancellous than in cortical bone. *Bone.* 30(6): 807-809.

Parfitt, A.M. (2004) What is the normal rate of bone remodeling? *Bone.* 35(1): 1-3.

Pearson, O.M. and Lieberman, D.E. (2004) The aging of Wolff's "law": ontogeny and responses to mechanical loading in cortical bone. *American Journal of Physical Anthropology.* 125(S39): 63-99.

Pease Jr., R.W. ed, (1995) *Merriam-Webster's Medical Dictionary.* Merriam-Webster, Springfield.

Peckmann, T.R. (2002) *Dialogues with the dead: an osteological analysis of the palaeodemography and life history of the 19th century northern frontier in South Africa*. Ph.D. Thesis, University of Capetown, South Africa.

Pickering, T.R. (2002) Reconstruction of criteria for differentiating faunal assemblages accumulated by hyaenas and hominids. *International Journal of Osteoarchaeology.* 12: 127-141.

Plug, I. (1993a) The faunal remains from Nanda, an Early Iron Age site in Natal. *Natal Museum Journal of Humanities.*5:99-107.

Plug, I. (1993b) The macrofaunal remains of wild animals from Abbot's Cave and Lame Sheep Shelter, Seacow Valley, Cape. *Koedoe* 36(1):15-26.

Plug, I. (1996) The hunter's choice: faunal remains from Maqonqo Shelter, South Africa, *Natal Museum Journal of Humanities.* 8:41-52.

Plug, I. (2000) Overview of Iron Age fauna from the Limpopo valley. *In:* Leslie, M. and Maggs, T. eds. *African Naissance: The Limpopo valley 1000 years ago. South African Archaeological Society*. Goodwin Series 8:117-126.

Plug, I. (2004) Resource exploitation: animal use during the Middle Stone Age at Sibudu Cave, South Africa. *South African Journal of Science*. 100(3/4): 151-158.

Plug, I. and Brown, A. (1982) Mgoduyanuka faunal remains. *Annals of the Natal Museum.* 25(1):115-121.

Plug, I. and Engela, R. (1992) The macrofaunal remains from recent excavations at Rose Cottage Cave, Orange Free State. *South African Archaeological Bulletin.* 47:16-25.

Plug, I. and Keyser, A.W. (1994) A preliminary report on the bovid species from recent excavations at Gladysvale, South Africa. *South African Journal of Science.* 90(6): 357-359.

Plug, I. and Roodt, F. (1990) The faunal remains from recent excavations at uMgungundlovu. *South African Archaeological Bulletin.* 45:47-52.

Punzi, L., Oliviero, F., and Plebani, M. (2005) New biochemical insights into the pathogenesis of osteoarthritis and the role of laboratory investigations in clinical assessment. *Critical Review of Clinical Laboratory Sciences.* 42(4): 279-309.

Radostits, O.M., Gay, C.C., Blood, D.C., Arundel, J.H. and Hinchcliff, K.W. (2000) *Veterinary Medicine: textbook of the diseases of cattle, sheep, pigs, goats and horses*, 9th ed. Saunders, Philadelphia.

Redfern, D.R.M., Forester, A.J., Evans, M.J. and Sohail, M. (1997) Enchondroma of the scaphoid. *Journal of Hand Surgery.* 22(2)L 235-236.

Richardson, R.R. (2005) Variants of exostosis of the bone in children. *Seminars in Roentgenology.* 40(4): 380-390.

Ripamonti, U. (1988) Paleopathology in *Australopithecus africanus*: A Suggested Case of a 3-Million-Year-Old Prepubertal Periodontitis. *American Journal of Physical Anthropology.* 76: 197-210.

Ripamonti, U., Kirkbride, A.N., Yates, S.C., Thackery, F. (1997) Further evidence of periodontal bone pathology in a juvenile specimen of *Australopithecus africanus* from Sterkfontein, South Africa. *South African Journal of Science*. 93: 177-178.

Robb, J.E. (1994) Skeletal signs of activity in the Italian metal ages: methodological and interpretative notes. *Human Evolution.* 9(3): 215-229.

Robb, J.E. (1998) The interpretation of skeletal muscle sites: a statistical approach. *International Journal of Osteoarchaeology.* 8(5): 363-377.

Robbins, S.L. (1974) *Pathologic basis of disease*. Saunders. Philadelphia.

Roberts, C.A. (1991) Trauma and treatment in the British Isles in the Historic Period: a design for multidisciplinary approach. *In:* Ortner, D. and Aufderheide, A.C., eds. *Human Paleopathology: Current Synthesis and Future Options*, pp. 55-56. Washington, D.C.

Roberts, C.A. (2000) Infectious disease in biocultural perspective: past, present and future work in Britain. *In*: Cox, M and Mays, S., eds., *Human Osteology in Archaeology and Forensic Science*, pp. 145-162. Greenwich Medical Media. London.

Roberts, C.A. and Buikstra, J.E. (2003) *The bioarchaeology of tuberculosis: a global view on a reemerging disease*. University Press of Florida. Gainsville.

Roberts, C.A. and Cox, M. (2003) *Health and Disease in Britain: prehistory to the present day.* Sutton Publishing, Stroud.

Roberts, C.A. and Manchester, K. (2005) *The Archaeology of Disease.* 3rd ed. Sutton Publishing. Stroud.

Robinson, J.T. (1956) *The dentition of the Australopithecinae.* Transvaal Museum Monograph. No. 9.

Robinson, J.T. (1961) The Australopithecines and their bearing on the origin of man and of stone tool making. *South African Journal of Science.* 57: 3-16.

Rogers, J. and Waldron, T. (1995) *A Field Guide to Joint Disease in Archaeology.* John Wiley, New York.

Rooney, J.R. (1997) Equid Paleopathology. *Journal of Equine Veterinary Science*. 17: 430-

Rothschild, B.M. (2002) Contributions of paleorheumatology to understand contemporary disease. *Rheumatismo* 54(3): 272-284.

Rothschild, B.M. (2005) Osseotypes and spondyloarthropathy exposed. *Current Rheumatology Reviews* 1: 57-63.

Rothschild, B.M. and Martin, L.D. (2003) Frequency of pathology in a large natural sample from Natural Trap Cave with special remarks on erosive disease in the Pleistocene. *Reumatismo.* 55(1): 58-65.

Rothschild, B.M. and Martin, L.D. (2006) Did ice-age bovids spread tuberculosis? *Naturwissenschaften.* 93(11): 565-569.

Rothschild, B.M., Prothero, D.R., and Rothschild, C. (2001) Origins of spondyloarthropathy in Perissodactyla. *Clin Exp Rheumatology.* 19(6): 628-632.

Rothschild, B.M. and Rothschild, C. (1999) On pseudoscience and Treponemal Disease in the Western Pacific. *Current Anthropology,* 40(1): 66-71.

Rothschild, B.M., Tanke, D., Carpenter, K. (1997) Tyrannosaurs suffered from gout. *Nature.* 387: 357.

Saadeh, C. (2004) Calcium Pyrophosphate Deposition Disease. Cited in: eMedicine. http://www.emedicine.com/med/topic1938.htm.

Sawlani, V., Chandra, T., Mishra, R.N., Aggarwal, A., Jain, U.K. and Gujral, R.B. (2003) MRI features of tuberculosis of peripheral joints. *Clinical Radiology.* 58(10): 755-762.

Schaller, G.B. (1972) *The Serengeti Lion: A Study of Predator-Prey Relations.* University of Chicago Press, Chicago.

Schick, K.D., Toth, N. and Daeschler, E. (1989) An early paleontological assemblage as an archaeological test case. *In*: Bonnichsen, R. and Sorg, M., eds., *Bone Modification.* pp. 121-137. Center for the Study of the First Americans.

Schindeler, A., McDonald, M.M., Bokko, P. and Little, D.G. (2008) Bone remodeling during fracture repair: the cellular picture. *Seminars in Cell and Developmental Biology.* 19(5): 459-466.

Schinz, H., Baensch, W., Friedl, E., and Uehlinger, E. (1951) *Roentgen Diagnostics: Skeleton, vol. 1 and 2.* Grune and Stratton, New York.

Schultz, A.H. (1956) *The life of Primates.* Weidenfeld and Nicolson, London.

Shaibani, A., Workman, R., and Rothschild, B.M. (1993) The significance of enthesopathy as a skeletal phenomenon. *Clin. Exp. Rheumatology.* 11(4): 399-403.

Shaw, J.C. (1939) Further remains of a Sterkfontein ape. *Nature* 143: 117.

Sims, N.A. and Gooi, J.H. (2008) Bone remodeling: multiple cellular interactions required for coupling of bone formation and resorption. *Seminars in Cell and Developmental Biology.* 19(5): 444-451.

Slobodin, G., Rozenbaum, M., Boulman, N. and Rosner, I. (2007) Varied presentations of enthesopathy. *Seminars in Arthritis and Rheumatism.* 37(2): 119-126.

Smith, G.K. (1985) Biomechanics pertinent to fracture aetiology, reduction, and fixation. *In*: Newton C.D. and Nunamaker D.M. eds. *Textbook of Small Animal Othopaedics.* International Veterinary Information Service, Ithaca.

Solomon, L. (2001) Clinical features of osteoarthritis. *In:* Ruddy, S., Harris Jr., E. and Sledge, C. eds., *Kelley's Textbook of Rheumatology, 6th Edition*. Saunders, pp. 1409-1418. Philadelphia.

Stashak, T.S. (2002) *Adam's Lameness in Horses.* Lippincott Williams and Wilkins, Baltimore.

Steckel, R.H. and Rose, J.C. (2002) The Backbone of History: health and nutrition in the western hemisphere. Cambridge University Press, Cambridge.

Steele D.G., and Bramblett C.A. (1988) *The Anatomy and Biology of the Human Skeleton*. Texas A&M University Press, College station.

Steyn, M. (2003) A comparison between pre- and post-colonial health in the northern parts of South Africa, a preliminary study. *World Archaeology.* 35(2): 276-288.

Steyn, M., Nienaber, W.C., and Meiring, J.H. (2002a) An assessment of the health status and physical characteristics of an early 20th century community at Maroelabult in the North West Province, South Africa. *Homo.* 53(2): 131-145.

Steyn, M., Meiring, J.H., Nienaber, W.C., and Loots, M. (2002b) Large fontanelles in an early 20th century rural population from South Africa. *International Journal of Osteoarchaeology*. 12(4): 291-296.

Stone, A.C., Wilbur, A.K., Buikstra, J.E. and Roberts, C.A. (2009) Tuberculosis and leprosy in perspective. *American Journal of Physical Anthropology.* 140, Suppl. 49: 66-94.

Susman, R.L. (1991) Who made the Oldowan tools? Fossil evidence for tool behavior in Plio-Pleistocene hominids. *Journal of Anthropological Research.* 47(2): 129-151.

Susman, R.L. (1993) Hominid postcranial remains from Swartkrans. *In:* Brain, C.K. ed. *Swartkrans: a cave's chronicle of early man.* Transvaal Museum Monograph. N.8. Pretoria.

Susman, R.L. (1994) Fossil evidence for early hominid tool use. *Science.* 265: 1570-1573

Takigawa, K. (1971) Chondroma of the bones of the hand. *Journal of Bone and Joint Surgery.* 53: 1591-1600.

Tasnadi-Kubacska, A. (1962) *Paläopathologie: Pathologie der Vorzeitlichen Tiere*. Gustav Fischer, Jena.

Thomas, R. (2001) The medieval management of fallow deer: a pathological line of enquiry. *In*: Verghetta, M. and Capasso, L., eds. *Proceedings of the XIIIth European Meeting of the Palaeopathology Association, Chieti, Italy: 18th-23rd September 2000.* Pp.287-293. S.P.A., Teramo.

Thomas, R. and Mainland, I. (2005) Introduction: animal diet and health, current perspectives and future directions. *In*: Davies, J., Fabis, M., Mainland, I., Richards, M. and Thomas, R., eds. *Diet and Health in Past Animal Populations: Current Research and Future Directions.* Oxbow Books, Oxford.

Thomas, R., Davies, J. and Fabiš (2002) Beyond "interesting specimens" palaeopathology and its contribution to the study of animal husbandry. Session Abstract. *Book of Abstracts for the International Council of Archaeozoology, 9th conference.*

Tompkins, D.M., Dobson, A.P., Arneberg, P., Begon, M.E., Cattadori, I.M., Greenman, J.V., Heesterbeek, J.A.P., Hudson, P.J., Newborn, D., Pugliese, A., Rizzoli, A.P., Rosa, R., Rosso, F. and Wilson, K. (2008) Parasites and host population dynamics. *In*: Hudson, P.J., Grenfell, B.T., Hasterbeek, H. and Dobson, A.P. eds., *The Ecology of Wildlife Diseases.* pp. 45-61. Oxford University Press, New York.

Trinkaus, E. (1985) Pathology and the posture of the La Chapelle-aux-Saints Neandertal. *American Journal of Physical Anthropology*. 67: 19-41.

Tsang, D. and Wu, D.Y. (2008) Osteoid osteoma of phalangeal bone. *Journal of Formosan Medical Association.* 107(7): 582-586.

Turner, A. (1993) New fossil carnivore remains from Swartkrans. *In*: Brain, C.K., *Swartkrans, a cave's chronicle of early man.* Transvaal Museum Monograph, N. 8. Pretoria.

Ubelaker, D.H. (1994) *Human Skeletal Remains: Excavation, Analysis, Interpretation.* 2nd ed. Taraxacum. Washington, D.C.

Vandermeer, J.H. and Goldberg, D.E. (2003) *Population Ecology.* Princeton University Press, Princeton.

Vann, S. (2008) *Recording the Facts: A Generic Recording System for Animal Palaeopathology.* Ph.D. Thesis, University of Leicester, United Kingdom.

Vann, S. and Thomas, R. (2006) Humans, other animals and disease: a comparative approach towards the development of a standardized recording protocol for animal palaeopathology. *Internet Archaeology.* 20(5).

Van Valkenburgh B. (2009) Costs of carnivory: tooth fracture in Pleistocene and recent carnivorans. *Biological Journal of the Linnean Society.* 96(1): 68-81.

Van Valkenburgh, b. and Hertel, F. (2003) Tough times at La Brea: tooth breakage in large carnivores of the late Pleistocene. *Science.* 261(5120): 456-459.

Voigt, E. A. and Plug, I. 1984. Happy Rest: the earliest Iron Age fauna from the Soutpansberg. *South African Journal of Science.* 80:221-227.

Vrba, E.S. (1975) Some evidence of chronology and palaeoecology of Sterkfontein, Swartkrans and Kromdraai from the fossil Bovidae. *Nature.* 254: 301-304.

Vrba, E.S. (1976) *The Fossil Bovidae of Sterkfontein, Swartkrans and Kromdraai.* Transvaal Museum Memoir No. 21, Pretoria.

Vrba, E.S. (1982) Biostratigraphy and chronology based particularly on Bovidae of southern hominid-associated assemblages: Makapansgat, Sterkfontein, Taung, Kromdraai, Swartkrans: also Elandsfontein (Saldanha). Broken Hill, and Cave of Hearths. *In*: de Lumley, H. and de Lumley, M.A. eds. *Proceedings of Congress International de Paleontologi Humaine.(Vol. II)* pp. 707-752. Union Internationale des Sciences Prehistoriques et Prohistoriques, Nice.

Waldron, T. (1994) *Counting the dead: The epidemiology of skeletal populations.* Wiley, New York.

Waldron, T. (2009) *Palaeopathology.* Cambridge University Press, Cambridge.

Waldron, T. and Rogers, J. (1994) Rheumatoid arthritis in an English post-Medieval skeleton. *International Journal of Osteoarchaeology.* 4: 165-167.

Walker, A. and Shipman, P. (1996) *The Wisdom of Bones: In search of human origins*. Orion Publishing Group, London.

Walker, A., Zimmerman, M.R., and Leakey, R.E.F. (1982) A possible case of hypervitaminosis A in *Homo erectus*. *Nature* 296: 248-250.

Watson, V. (1993) Composition of the Swartkrans bone accumulations, in terms of the skeletal parts of animals represented. *In*: Brain, C.K., ed. *Swartkrans, a cave's chronicle of early man.* Transvaal Museum Monograph, N. 8. Pretoria.

Wells, C. (1967) Pseudopathology. *In*: Brothwell, D. and Sandison, A.T. eds., *Diseases in Antiquity.* pp. 5-19. Charles C. Thomas, Ft. Lauderdale.

Weiss, E. and Jurmain, R. (2007) Osteoarthritis revisited: a contemporary review of aaetiology. *International Journal of Osteoarchaeology.* 17(5): 437-450.

White, T.E. (1953) A method of calculating the dietary percentage of various food animals utilized by aboriginal peoples. *American Antiquity.* 19: 396-398.

White, T.D. (2000) *Human Osteology*. Academic Press, Orlando.

Wienker, C.W. and Wood, J.E. Osteological individuality indicative of migrant citrus laboring. *Journal of Forensic Science* 33(2): 562-567.

Williams, J. Orthopedic radiography in exotic animal practice. *Veterinary Clinics of North America: Exotic Animal Practice.* 5(1): 1-22.

Wolff, J. translated by: Maquet, P. and Furlong, R. (1986) *The Law of Bone Remodelling.* Springer-Verlag. Berlin, Heidelberg.

Wood, J.W., Milner, G.R., Harpending, H.C. and Weiss, K.M. (1992) The Osteological Paradox: Problems of inferring prehistoric health from skeletal samples. *Current Anthropology,* 33(4): 343-370.

Ytrehus, B., Carlson, C.S. and Ekman, S. (2007) Aetiology and pathogenesis of osteochondrosis. *Veterinary Pathology.* 44(4): 429-448.

Zar, J.H. (1999) *Biostatistical Analysis,* 4th ed. Prentice Hall.

Zipfel, B. (2006) *Morphological Variation in the Metatarsal Bones of Selected Recent and Pre-pastoral Humans from South Africa.* PhD Thesis, Department of Anatomical Sciences, University of the Witwatersrand, South Africa.

Zivanovic, S. (1982) *Ancient Diseases: The Elements of Palaeopathology*. Methuen Co., London.

Zumwalt, A. (2006) The effect of endurance exercise on the morphology of muscle attachment sites. *The Journal of Experimental Biology.* 209: 444-454.