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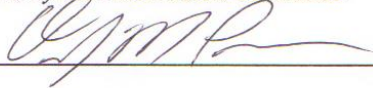
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**THE PREVALENCE OF OSTEOARTHRITIS IN WILD VERSUS
CAPTIVE GREAT APE SKELETONS**

BY

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DISSERTATION

Submitted in Partial Fulfillment of the
Requirements for the Degree of

Doctor of Philosophy

Anthropology

The University of New Mexico
Albuquerque, New Mexico

December, 2009

ACKNOWLEDGEMENTS

I wish to thank all members of my doctoral committee: Dr. Osbjorn Pearson (Chair), Dr. Robert Jurmain, Dr. Jane Buikstra, Dr. Heather Edgar, and Dr. Ed Bedrick. Each contributed greatly to the process and I thank them for their guidance and support.

I also wish to thank three other individuals who provided invaluable support and help. Dr. Debra Komar spent many hours teaching me the various aging techniques used on human skeletons and helping identify aging markers in the great ape skeletons. Dr. Tanya Mueller provided much appreciated advice and encouragement, and Dr. Darcey Mays of Virginia Commonwealth University offered valuable guidance in the area of data analysis. All have been valuable sources of information.

I am also grateful to my family and friends who provided encouragement and cheered me on. In particular, my husband, Edgar, was a constant source of encouragement, pushing me onwards when I lacked confidence in my abilities.

For help in providing access to the skeletal collections, great appreciation is due to staff at the American Museum of Natural History, Arizona State University, California Academy of Sciences, Field Museum, Holloman Primate Facility, Maxwell Museum University of New Mexico, Museum of Comparative Zoology, Museum of Vertebrate Zoology, National Museum of Natural History, British Museum of Natural History, Peabody Museum, Primate Foundation of Arizona, Royal Belgian Institute for Natural Sciences, Royal Museum for Central Africa, Sam Noble Oklahoma Museum of Natural History, University of Arkansas at Little Rock, and the William R. Adams Primate Skeletal Collection Indiana University. Finally, I would like to thank the Leakey Foundation, who helped fund this research.

**THE PREVALENCE OF OSTEOARTHRITIS IN WILD VERSUS
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ABSTRACT OF DISSERTATION

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Degree Title Awarded

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ABSTRACT

This research examined whether the prevalence and skeletal distribution of osteoarthritis (OA) differed between wild and captive great ape skeletons. A secondary, but important, aspect of this research focused on the development of improved aging techniques based on methods commonly used on human osteological samples. Tests were conducted pertaining to the effect that wild versus captive status, sex, and species has on vertebral body lipping, marginal lipping, and eburnation. Age was considered a co-factor.

Of the aging methods examined, use of the basilar suture to distinguish between adult and old adult specimens proved to be very imprecise. The ribs and auricular surface proved to be of limited value in aging the ape skeletons, while the acetabulum demonstrated potential for use as an aging indicator, although it is not recommended for use in isolation. Molar dental wear proved to be the most viable single indicator of age explaining over 78% of the variation seen. However, a model that combined wear of

molars 1 and 2 with certain features of the acetabulum explained over 90% of the variation seen and was the model chosen for aging the apes in this sample.

The effect that wild versus captive status, sex, and species had on vertebral body lipping, eburnation, and marginal lipping was analyzed, with age as a co-factor. It was found that status is a significant predictor of the prevalence of both vertebral body lipping and marginal lipping, but not of eburnation with captive apes suffering significantly more vertebral body lipping than wild apes. Sex is not a significant predictor of disease prevalence for any skeletal marker. Species' differences are evident in vertebral body lipping and marginal lipping, but not in eburnation. In general, chimpanzees are the least frequently affected and gorillas the most frequently affected. Age has an effect, primarily in vertebral body lipping and marginal lipping, with older individuals being more affected than younger individuals.

In summary, while wild versus captive status, species' differences, and age are factors in the development of vertebral body lipping and marginal lipping in many joints, the presence of eburnation is extremely rare in the great apes with very few individuals being affected regardless of status, sex, species, or age. Thus, the results highlight the complex nature of osteoarthritis and enforce the idea that osteoarthritis is markedly multifactorial and that disease prevalence and patterns are not easily understood or interpreted.

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Chapter 1. Introduction

Osteoarthritis (OA) is characterized as one of the most common pathological conditions of the skeleton in modern human populations, occurring worldwide. The disease occurs in many mammalian species, as well as in some birds, and also has been found in fossil species, such as mammoths, dinosaurs, and hominins (Wells, 1973; Sokoloff, 1969; Jurmain, 2000). Animal models used in research on osteoarthritis include studies on guinea pigs, hamsters, rabbits, and dogs, among others (Dequeker et al., 1997; Jurmain, 1999; Otterness et al., 1998). Researchers have also examined the skeletons of many different species of mammals, finding evidence that arthritis is widespread among a variety of captive and wild medium and large-sized mammals (Greer et al., 1977). In restrained animals, osteoarthritis is said to be a well-recognized problem for those housed in captivity (Sokoloff, 1969). However, there have been relatively few studies of OA in non-human primates, even though non-human primates are often used as animal models for human diseases. To date, studies of arthritis have been carried out on some prosimians, a few species of monkeys, as well as gibbons, gorillas, bonobos, and chimpanzees (Duckworth, 1911; Fox, 1939; Bramblett, 1967; Schultz, 1969; Woods, 1986; DeRousseau, 1988; Lovell, 1990; Lim et al., 1996; Carlson et al., 1994 and 1996; Jurmain, 1989 and 2000; Rothschild and Woods, 1992a and 1992b; Nakai, 2003; Rothschild and Ruhli, 2005a and b).

While osteoarthritis is found in non-human primates, it is thought to be relatively uncommon, particularly when compared to the prevalence of the disease in modern humans. Studies in chimpanzees, gorillas, and bonobos (Lovell, 1990; Jurmain, 1989 and 2000) have found that great apes suffer significantly less osteoarthritis when compared to

humans. One study comparing the African apes to data from colony-raised macaques, gibbons, and some human groups, found that for vertebral degenerative disease, African apes are much less affected than the comparable human sample, and that for OA in the four major joints (shoulder, elbow, hip, and knee), African apes demonstrate less involvement than most human groups. This study also concluded that the higher rate of degenerative spinal disease in humans is most likely attributable to the biomechanical adaptations related to bipedality; however, it was also acknowledged that data from Old World monkeys suggest that they likely have a higher frequency of spinal disease than apes; although, this is not yet fully established (Jurmain, 2000). Thus, generalizations relating OA to locomotor adaptations might be difficult to presume given what is currently known.

While the African apes suffer significantly less osteoarthritis than humans, other non-human primates appear to exhibit higher rates of disease involvement. It has been found that in Old World monkeys, rates of disease appear to be higher with one study finding 39.0% of a free-ranging group of savannah baboons affected with degenerative disease (Bramblett, 1967). Rhesus macaques and gibbons also show relatively high prevalence rates at 24.7% and 9.4%, respectively (DeRousseau, 1980). However, the macaque data was from a colony-raised group, which may have an effect on the outcome. In other non-human primate comparisons, one study found low rates of OA (0.9%) in free-ranging animals while captive animals displayed significantly higher rates of involvement (4.8%) (Rothschild and Woods, 1992).

While a commonly held notion is that all captive animals will suffer significantly more osteoarthritis than their wild counterparts, a comparative study of wild and captive

great ape skeletons has not yet been undertaken. Thus, it is currently not known whether captive apes suffer significantly more OA than their wild counterparts in at least some joints. In addition to examining whether captivity might affect disease prevalence, there appears to be a general lack of consistency in scoring criteria. This issue will be discussed in more detail later in this chapter and in chapter 4; however, many researchers have consistently demonstrated variability in scoring criteria that might explain some of the differences in disease prevalence alluded to above.

This study seeks to address whether the prevalence of osteoarthritis and degenerative disease of the spine differs between wild and captive chimpanzee, gorilla, and orangutan skeletal specimens (bonobos are not included). A secondary, but important, aspect of this research is to focus on the development of improved aging techniques based on methods commonly used on human osteological samples. In well-controlled epidemiological and paleoepidemiological studies of osteoarthritis (primarily on humans), age controls are critical because age is known to contribute to OA expression. Current aging methods for the great apes typically utilize categories based on stages of dentition, humeral fusion, and basilar suture fusion (Lovell, 1990). Using these criteria, only very broad age categories are possible and older adults are generally conflated into one category. This research will focus on developing aging criteria based on human standards for rib phase analysis, the auricular surface of the pelvis, and the acetabulum. Dental wear also will be analyzed for its utility in estimating age.

Background: Osteoarthritis is one of the most widespread, potentially debilitating, skeletal diseases in modern human populations. The World Health Organization (WHO) states that OA is one of the ten most disabling diseases in

developed countries with a worldwide estimate that 9.6% of men and 18.0% of women over age 60 have symptomatic OA. Of those with OA, 80.0% suffer limitation in movement and 25.0% cannot perform the major daily activities of life (WHO, [online] accessed 2008). In the United States, 46 million people report that a doctor told them that they have arthritis or other rheumatic conditions (including OA), and arthritis is the most common cause of limiting the activities of nearly 19 million American adults (CDC, [online] accessed 2008). The National Arthritis Data Workgroup report that the prevalence of clinical OA in the United States has recently grown to nearly 27 million cases, up from an estimated 21 million in 1995 (Lawrence et al., 2008). Indeed, The National Health and Nutrition Examination Survey I found that 12.1% of the US population between the age of 25-74 had clinically defined OA of some joint (a person is characterized as having clinical OA on the basis of symptoms and physical examination findings) (Lawrence et al., 2008). Nevertheless, estimating prevalence of OA in a population is difficult because structural changes of the disease that occur in bone may not be accompanied by symptoms, and prevalence estimates vary depending on whether mild, moderate, or severe radiographic changes are included (Lawrence et al., 2008).

Terminology: Even though osteoarthritis is a very common disease, there is some disagreement as to what name is most appropriate. The condition has been known by a variety of names including degenerative joint disease, arthrosis, osteoarthrosis, hypertrophic arthritis, and degenerative arthropathy, among others (Sokoloff, 1969). Initially, the term *osteoarthritis* was used to imply an inherently inflammatory process. Later, most clinicians concluded that there was no important inflammatory component and so the term degenerative joint disease was suggested as the most appropriate and

accurate (eg, Comroe, 1944; Hough, 1993). This name also has been criticized because the term *degenerative* is thought to be misleading and not well defined (Dieppe, 1987). More recently, clinical perspectives are shifting with many researchers now regarding inflammation as ‘crucial to the pathogenesis of OA’ (Punzi et al., 2005). While there remains some lack of agreement of the most appropriate terminology, osteoarthritis is once again the most commonly used and preferred term (Weiss and Jurmain, 2007).

Skeletal Location: Among clinical workers, most researchers reserve the term osteoarthritis for the fully movable, diarthrodial joints. Such joints include all the major articulations of the appendages as well as the small interfacetal joints of the spine (Aufderheide and Rodriguez-Martin, 1998). OA is most noticeable in the large joints, such as the hip and knee, but is also found in the interfacetal joints of the spine, the ankle, foot, sacroiliac joint, shoulder, elbow, wrist, hand, and temporomandibular joint (Rogers et al., 1987; Aufderhide and Rodriguez-Martin, 1998). In the disk joints of the spine, the term vertebral osteophytosis (VOP) is usually preferred because non-articular vertebral joints are not true synovial joints; however, the disease processes are similar (Jurmain and Kilgore, 1995).

Disease Definition: Although OA is the most common joint disease, it can be difficult to define (e.g: Radin, 1982 and 1983; Jurmain, 1999; Bailey and Metz, 2001). In clinical settings, OA is often described in one of three ways: *clinically defined OA* (a person is characterized as having clinical OA on the basis of symptoms and physical examination findings), *radiographically defined OA* (radiographs are graded according to the Kellgren/Lawrence scale which defines OA on the basis of the presence of osteophytes), and *symptomatic OA* (a person is considered to have symptomatic OA if

there is frequent pain in a joint and radiographic evidence of OA in the joint) (Lawrence et al., 2008). The apparent difficulty in defining the disease may be because it develops and changes slowly, symptoms may or may not be present, and its heterogeneity results in controversy as to its cause and progression. Indeed, OA pathophysiology still remains poorly described, there are no simple tests (such as blood tests) to detect the disease, and the standard means of diagnosis (radiography) is thought to have limited value (Rogers et al., 2004).

Skeletal Diagnosis: While the term osteoarthritis was created to describe the swelling and pain associated with the disease, some researchers believe that it is completely appropriate that skeletal studies are used to provide further insights (Rogers, et al., 2004). There is apparent agreement that OA can be divided into two forms, primary and secondary. Primary, or idiopathic, OA is seen in 80% of cases in which no cause is evident, while secondary OA is caused when the joint has been altered by some disease or event (Aufderhide and Rodriguez-Martin, 1998; Jurmain, 1999). However, there is currently no consensus on the various methods used to analyze OA in skeletal material (e.g: Rogers et al., 1987; Duncan, 1979; Rogers and Waldron, 1995; Jurmain, 1999). In skeletal studies, typical features used to diagnose the presence of osteoarthritis include lipping (bony spurs or osteophytes), surface osteophytes (which reflect the addition of compact bone to the joint surface), porosity, and eburnation (bone-on bone polish that develops following degeneration of the cartilage) (Buikstra and Ubelaker, 1994; Aufderhide and Rodriguez-Martin, 1998). There are, however, problems associated with utilizing these criteria and some researchers now recommend diagnosis via the use of the presence of eburnation only (eg: Jurmain, 1999). This is because

eburnation is typically accepted as diagnostic of OA in both clinical and osteological fields, while marginal lipping, surface osteophytes, and porosity are of uncertain diagnostic value. For example, it is now thought that genetic and/or physiological mechanisms that affect the joint margins may be discrete from those mechanisms that affect joint surfaces (Weiss and Jurmain, 2007). Research also indicates that osteophytes develop in relation to biological aging and thus may not be reliable as an indicator of disease severity (Weiss and Jurmain, 2007). Further, porosity, while frequently utilized in OA evaluation, remains poorly described. There are at least three different pathways by which porosity occurs: (1) thinning of the articular plate exposing vascular channels (probably not related to OA); (2) active vascular invasion of calcified cartilage (may be related to OA); and (3) perforation through the articular plate *subsequent* to eburnation. These different pathways produce different types of ‘holes’ that can be difficult to differentiate (Jurmain, 1999). In addition, research now indicates that porosity may be unrelated to osteoarthritis, and may occur independently from eburnation (Woods, 1995; Rothschild, 1997; Weiss and Jurmain, 2007). Indeed, Rothschild (1997) found that “no significant relationship exists between porosity and osteoarthritis” and that “eburnation and porosity are unrelated.” He further recommended that porosity should not be used as an identifier of osteoarthritis. Thus, eburnation remains as the only diagnostic criterion currently universally accepted and is often used as *the* major marker for the presence of OA in skeletal material. Yet, as Rothschild (1997) points out, eburnation identifies the location of total loss of joint cartilage and thus is a sign of severity of arthritis and not the form of arthritis that caused the cartilage loss. Nevertheless, while skeletal diagnosis of OA is not without its challenges, separate and precise recording of marginal and surface

changes is currently recommended (Buikstra and Ubelaker, 1994; Weiss and Jurmain, 2007).

Etiology: The etiology of OA is somewhat contentious, but is certainly multifactorial in nature. Most authors agree that OA shows a strong relationship to age and is more common in women (Comroe, 1944; Sokoloff, 1969; Aufderhide and Rodriguez-Martin, 1998; Jurmain, 1980 and 1999). Some studies show that OA prevalence increases with age and affects the hands and knees of women more frequently than men, especially in those over 50 years of age (Lawrence et al., 2008). However, other studies have found that OA is more common in men or that there is no difference between the sexes (Bridges, 1991). Nevertheless, although OA shows a strong age relationship, it is reportedly not merely an “old age” disease because it may be absent in an individual of 80 and present in an individual of 35 (Comroe, 1944). Recent research indicates that a strong genetic component is evident, particularly in women. Various studies estimate the heritability of OA to be around 40-60% with an overall heritability average estimated at 50% (eg: Williams and Spector, 2006; Zhai et al., 2006). Current opinion also considers OA to be polygenic (Williams and Spector, 2006). Anatomical variances among individuals, such as knee height and acetabular dysplasia, can influence the onset and severity of osteoarthritis, while populational differences in anatomy (eg: knee alignment) are also found. Thus, normal anatomical differences, combined with weight and activity, affect the onset and severity of OA (Weiss and Jurmain, 2007). It also appears that body mass index (BMI) impacts osteoarthritis in that heavier people have more severe OA than lighter people (Tepper and Hochberg, 1993; Manek et al., 2003). Mechanical and systemic effects play a role in the influence that BMI has on OA

and it is now clear that a high BMI, particularly in females, correlates with osteoarthritis (Weiss and Jurmain, 2007). The association of OA at one anatomical site and its presence at another site has also been examined. In one study, the progression of knee OA was found to be associated with the progression of OA in the lumbar spine and hip (Hassett et al., 2006). Other likely etiological agents include hormones, other arthropathies, rate of bone turnover, and diet; however, results are often conflicting regarding their overall contribution in development of the disease (Jurmain, 1999; Holderbaum et al., 1999; Valdes et al., 2004).

While it is apparent that significantly altered joint biomechanics or trauma can initiate secondary OA, there is little consensus regarding the role of repetitive stress on the initiation of primary, or idiopathic, OA (Radin, 1983; Jurmain, 1999; Otterness et al., 1998). Indeed, it has been stated that OA is “neither a good predictor of specific activities, nor a good indicator of overall levels of activity” (Weiss and Jurmain, 2007). Studies of mechanical factors and the effects of activity (such as occupational stress and/or sports) are conflicting and demonstrate inconsistent results (e.g. Eichner, 1989; Radin, 1982; Jurmain, 1977 and 1999; Sharma et al., 2000; Coggon et al., 2000; Nevitt et al., 2002; Otterness et al., 1998). For example, five groups of researchers studying long-distance and elite runners found no association between running and increased knee OA (Puranen et al., 1975; Sohn and Micheli, 1985; Panush et al., 1986; Lane et al., 1987, 1990, 1993; Konradsen et al., 1990), while three groups of researchers studying elite runners and runners presenting with pain did find an association between activity and increased knee OA (McDermott and Freyne, 1983; Marti et al., 1989; Coggon et al., 2000). A review of epidemiological studies that investigated the correlation of OA with

general levels of activity found 22 samples with positive correlations, 14 with no significant correlations, and 5 with mixed results. Likewise, of studies examined for the correlation of OA with specific occupational/sports activities, 48 found a positive correlation, 22 found no correlation, and 2 displayed mixed results (see Weiss and Jurmain, 2007, for a more detailed summary). The effect of exercise on the outcome of OA in clinical settings has also been examined and it was found that the kind of exercise performed is important. One study recommended regular aerobic activity of moderate intensity as well as muscle strengthening (like that recommended for *all* adults) with evidence suggesting that exercise can actually decrease knee pain and improve function. As the author points out, “the disability in OA is due not only to the arthritis but also to the inactivity associated with the disease and with aging” (Minor, 2004).

Other studies have examined the biomechanical implications of OA. A recent experiment on cadaveric thoraco-lumbar segments of elderly individuals (aged 64-92 years) tested whether degenerative changes in apophyseal joints are directly related to high levels of compressive load-bearing in these joints. The thoraco-lumbar segments were subjected to a compressive load and the resulting stress measurements were calculated to give the compressive force resisted by the disc. It was found that in elderly individuals apophyseal joint load-bearing above a threshold of 50% is associated with severe degenerative changes in cartilage and bone (Robson-Brown et al., 2008). Implications from some studies would appear to suggest that altered postures lead to altered functional anatomy. For example, Sarmiento (1985), in a study based on reviews of literature on wild orangutans and focal sampling of captive orangutans, demonstrated that the skeletal structures of captive and wild orangutans were modified by their

respective environments. Nevertheless, modifications of skeletal structures were documented in those traits with known environmental input, such as long bone torsions and adaptations for wrist mobility, as well as in those traits which were not expected to be altered, such as body proportions. Thus, it is still far from clear exactly what impact altered functional anatomy has on the likelihood of developing OA, or even if there is a causal relationship to the exclusion of a large number of other factors that differ between populations.

There has been some research to suggest that the age of onset of stressful activity might influence increased risk for developing OA, particularly if the stressful activity begins early in life. This idea has been both supported and denied by some research, but it appears likely that high amplitude stresses that begin early in life can initiate OA, at least in some cases (Weiss and Jurmain, 2007). For example, two reports found that OA was most frequent in agricultural farmers who farmed for more than 10 years and that many of these farmers began some tasks as children. One of these studies noted that many subjects started farm work in their early teens when the hip joint is not fully developed and that the hip may be particularly vulnerable to trauma or physical stress at that stage in life (Jurmain, 1999). Similar ideas have been proposed relating to sports activity begun early in life and later increased prevalence of OA. For example, baseball pitching, where severe, repetitive and early onset stress is common, *may* lead to a variety of degenerative elbow changes (Jurmain, 1999).

Research of Non-Human Primates: In terms of biology and behavior, primates are exceptionally diverse. Studies of non-human primates encompass myriad subjects, including such topics as fundamental behavior, intelligence, social systems, tool use,

demography, anatomy, and so on. Anthropologists and primatologists typically study primates in order to learn more about our basic primate natures as well as to speculate about our evolutionary origins. Prior to the 1960s, most studies of non-human primates concerned anatomy (eg: Schultz, 1935, 1940, 1941, 1950) and behavior, such as the laboratory studies of chimpanzees conducted by Yerkes and colleagues (Yerkes, 1925; Yerkes and Learned, 1925; and Köhler, 1927). While field studies began in the 1930s (Itani, 1996), it was not until the 1960s that long-term field research began in earnest when it was thought that studying our closest relatives in their natural habitat could provide clues to the behavior of early humans (Goodall, 1986). In 1960, Goodall began the longest-running field study of any apes in nature, studying the chimpanzee in Tanzania, and chimpanzees are now studied in at least five field sites that have been in continuous operation for more than 20 years (McGrew, 2004). Gorillas and orangutans have also been the subject of long term field studies such as those begun by Fossey and Galdikas (Fossey, 1983; Galdikas, 1995). Research on captive apes typically comprise observations of naturalistic groups (such as those in wildlife parks and accredited zoos), psychological testing in traditional behavior laboratories, and language acquisition studies (Goodall, 1994). Nevertheless, while lab studies can sometimes expand on and validate field impressions, many scientists are most interested in what happens in nature rather than what happens in the artificial environments of captivity.

In skeletal collections, the availability of primate skeletal material (at least in the United States and Canada) is heavily biased towards wild-caught animals. In part, this is due to the fact that up until the 1960/70s, when conservation-based principles were realized, it was quicker and easier for museums to obtain specimens from the wild rather

than relying on the unpredictability of obtaining specimens from zoos. This is particularly true prior to WWII, when big-game hunting and colonial attitudes influenced collection methods and large-scale collection by organized 'wild shoots' were common. In addition, wild specimens are seen to be of immense value for systematic, functional, and evolutionary studies, while specimens which experienced captivity are thought to be of lesser importance because of possible pathologies or lack of information about their provenience (Albrecht, 1982). Further, it is not clear how potential differences in diet, behavior, and access to veterinary care might impact skeletal development and disease. A further area of concern is the possibility of differing rates of aging between wild and captive animals; however, studies are conflicting in their results. For example, a study of chimpanzee dentition from three African field sites (Taï, Gombe, and Bossou) showed that wild chimpanzees demonstrate "an unambiguous pattern of a slower growth rate" than captive chimpanzees (Zihlman et al., 2004). However, a recent study of dental development in the Taï chimpanzees demonstrated that tooth formation stages largely overlapped in wild and captive specimens, thus suggesting that there is "a high degree of overlap in dental development between wild and captive chimpanzees" (Smith et al., 2009). While studies of dental development are conflicting, evidence from field studies on social and behavioral development appear to suggest that wild chimpanzees take up to three years longer to mature than captive animals (Boesch and Boesch-Achermann, 2000). Thus, while studies provide evidence of social and behavioral differences in the development of wild and captive animals, differences relating to dental development are less clearly established. Further, it is not known with certainty whether captive and wild ape teeth vary significantly in regard to dental wear rates or patterns. Evidence certainly

suggests that the pattern of wear is similar in wild and captive animals, although severity of wear may be less in captive animals (Nichols and Zihlman, 2002).

The Great Apes

The great apes have been the subjects of extensive studies both in wild and captive environments. The living apes and humans represent a small remnant of an ape lineage that was widespread and diverse throughout the Miocene (around 22-5 mya). Other than humans, there are three genera of living great apes: chimpanzees (*Pan*), gorilla (*Gorilla*), and orangutans (*Pongo*). It is now universally accepted that the orangutans were the first great ape to have diverged from the branch leading to the other living apes. Gorillas split off next from the line leading to chimpanzees, bonobos, and humans, with chimpanzees and bonobos being the most recently diverged from each other. Chimpanzees (including bonobos) and gorillas are found in Central and West Africa, while orangutans are found only on the islands of Borneo and Sumatra in Southeast Asia. The great apes exhibit a wide range of social systems and behaviors. Group sizes range from the largely solitary orangutan to chimpanzees with groups of up to 100 individuals. Orangutans are the most arboreal of the great apes living a largely solitary existence, while the African apes are all highly social but with different group structures (Caldecott and Miles, 2005). The difference in social systems and behaviors calls for more specific background information on each species.

Chimpanzee: The common chimpanzee (*Pan troglodytes*) is geographically widespread across Central and West Africa ranging from as far east as Tanzania to Guinea in the west. They inhabit a wide variety of habitats including primary and

secondary forests, dry woodland savanna, grassland, and tropical rain forests at altitudes from sea level to 2600 m in East Africa. Chimpanzees eat a diverse diet that includes fruit, leaves, flowers, seeds, insects, and animal prey, and are well-known as tool users, particularly with respect to the use of tools to obtain and extract food items, such as termites. They exhibit diverse behaviors with up to 39 cultural variants identified that include varied tool use techniques, social customs, and courtship activities (Caldecott and Miles, 2005). Male chimpanzees are slightly larger than females in all populations. The central chimpanzee (*P.t. troglodytes*) is larger and heavier than the other subspecies with weights ranging between 52 and 60 kg for males and 44 and 50 kg for females. The western chimpanzee (*P.t. verus*) is smaller with males weighing around 46-48 kg and females weighing as little as 21 kg. The eastern chimpanzee (*P.t. schweinfurthii*) is smaller and shorter-limbed than the central chimpanzee, with weights ranging from around 30-60 kg for males and 22-45 kg for females (Caldecott and Miles, 2005). Chimpanzees live in multi-male, multi-female groups in fission-fusion social units in which individuals associate in smaller temporary subgroups within the community range, but in some populations females often travel alone. Males are generally philopatric, remaining in their natal group, while young females commonly (but not always) disperse to join other communities. In the wild, chimpanzees have an estimated maximum lifespan of between 40 and 50 years (Caldecott and Miles, 2005). One study presenting synthetic life tables derived from mortality data gathered from five study populations show that life expectancy at birth is less than 15 years for both sexes. Infant mortality in the first year is about 20.0%, dropping to about 3.5% between the ages of 10-15. At age 15, life expectancy is about another 15 years. These life tables also suggest that males

experience higher mortality than females with 27.0% of all male and 41.0% of all female infants expected to survive to age 15. From age 15 to 40, 11.0% of males and 18.0% of females are expected to survive (Hill et al., 2001). The estimated total population size of wild chimpanzees is between 172,000 and 301,000 individuals (as of 2003). Habitat loss, illegal hunting, disease (primarily Ebola), and logging continue to affect population numbers (Caldecott and Miles, 2005). According to the IUCN red list, chimpanzees are listed as “endangered” (IUCN Red List, [online] accessed Jul 2008).

Gorilla: Gorillas (*Gorilla*) are found in West and Central Africa inhabiting swamp, lowland, and montane forests. Gorillas from all areas eat much the same kinds of foods, but with local ecological limitations in food availability. Food choices include fruit, leaves, stems, herbaceous vegetation, bark, and insects (Caldecott and Miles, 2005). Male gorillas are significantly larger than females weighing around 150-175 kg, while females weigh around 71-97 kg (Rowe, 1996). Gorillas live in cohesive groups of up to 50 individuals, with a typical group containing one or two silverback males, a few blackback (younger) males, and a number of adult females and their young offspring. Young females generally disperse to join another male’s group in which they stay and raise their young. Most males also leave their natal group, often associating with other males until they are mature enough to lead their own group. In the wild, gorillas have an estimated lifespan of between 35 and 45 years, although the maximum lifespan of wild gorillas is unknown (Caldecott and Miles, 2005). For western lowland gorillas, life-history data available from two study sites in the Republic of Congo showed that infant mortality up to three years of age, in populations not affected by Ebola, was 22.0% and

65.0%, respectively (Walsh et al., [online]; accessed Nov 2008). Recent reports from one of these study sites in northern Congo estimated that western gorillas are weaned at a later age when compared with mountain gorillas and have slower maturation of immature animals (Breuer et al., 2008). In mountain gorillas, it was found that trauma is the cause of 40.0% of gorilla mortality and is evenly distributed across age groups. Infanticide is the primary cause of traumatic deaths in infants, while in juveniles and adults intraspecific aggression and human-induced trauma are the main factors. Respiratory disease (24.0%) is the second-greatest cause of death in mountain gorillas and is also evenly spread across age groups (Cranfield, 2008). In mountain gorillas, it has also been found that, unlike birth rates, death rates vary with rainfall, with deaths clustered in the wettest months (Watts, 1998). Two species of gorilla, separated by the inner Congo Basin, are now recognized and each of these species has two subspecies: the eastern gorilla (*G. beringei*) is divided into the eastern lowland gorilla (*G.b. graueri*) and the mountain gorilla (*G. b. beringei*). The western gorilla (*Gorilla gorilla*) is divided into the western lowland gorilla (*G.g. gorilla*) and the Cross River gorilla (*G.g. diehli*) (Caldecott and Miles, 2005). Of the eastern gorillas, there are around 700 mountain gorillas and perhaps as many as 17,000 eastern lowland gorillas. But, due to hunting and warfare in the region, eastern lowland gorilla numbers have been difficult to ascertain and are likely to be significantly lower (Caldecott and Miles, 2005). Of the western gorillas, there are around 250-280 cross-river gorillas, and perhaps as many as 200,000 western lowland gorillas. Habitat loss, hunting, and Ebola continue to threaten western lowland gorilla numbers which in recent years have typically been estimated at around 95,000 (International Union for Conservation of Nature (IUCN), [online] accessed Jul 2008).

However, a recent census presented by the Wildlife Conservation Society at the International Primatological Society conference in August 2008, estimated that there were 125,000 western lowland gorillas in northern Congo alone. The census was based on nest count data that used a mathematical model to estimate gorilla numbers. Nevertheless, some experts advise caution in accepting these estimates based on potential problems with the estimation methods used (National Geographic [online], accessed Nov 2008). According to the IUCN red list, *Gorilla gorilla gorilla*, and *Gorilla gorilla diehl* are listed as “critically endangered.” The remaining gorillas are listed as “endangered” (IUCN Red List, [online] accessed Jul 2008).

Orangutan: Orangutans (*Pongo*) are found in forested areas on the islands of Borneo and Sumatra in Southeast Asia. They eat a diet of sugary, ripe fruit and undefended seeds, as well as leaf shoots, insects, flowers, and bark. Male orangutans are roughly twice the size of females, weighing an average of around 75-80 kg to a female's 40 kg (Rowe, 1996). Orangutans are wide-ranging, largely solitary, animals with large, but perhaps stable, overlapping home ranges. They are not territorial, although fully adult males are intolerant of each other. Male orangutans demonstrate a maturation process, unique among the apes and not yet fully understood, known as ‘bimaturism.’ This means that in the course of development there are two alternative pathways with some ‘flanged’ males reaching full socio-sexual maturity sooner than other ‘unflanged’ males, who maintain testosterone levels intermediate between fully-flanged males and juveniles (Caldecott and Miles, 2005). Female orangutans tend to stay near the range in which they were born, maintaining friendly relationships with local females who are

likely to be relatives. In the wild, orangutans have an estimated maximum lifespan of up to 45 years (Caldecott and Miles, 2005); although, one study of life history data on wild Sumatran orangutans estimated longevity to be at least 58 years for males and 53 years for females. This study concluded that orangutan life history is the slowest among extant great apes and estimated age at first reproduction to be 15.4 years with an average interbirth interval at 9.3 years (the longest ever recorded for any great ape). Age specific mortality was not found to differ between the sexes and was found to be significantly lower than that of wild chimpanzees (Wich et al., 2004). Bornean orangutans have an estimated age at first reproduction at between 10-15 years with an interbirth interval that can be as low as 5 years (World Wildlife Fund (WWF) [online], accessed Nov 2008). Traditionally, the two island populations have been regarded as sub-species and fertile hybrids have been produced in captivity; however, molecular studies suggest that the two taxa are “highly differentiated” at the genetic level with the genetic differentiation between the two orangutans comparable to those between well-recognized species (eg: chimpanzee vs bonobo, horse vs donkey) (Ryder and Chemnick, 2001; Zhang et al., 2001). Thus, two species of orangutan are now recognized: the Sumatran (*Pongo abelii*) and the Bornean (*Pongo pygmaeus*). No sub-species of Sumatran orangutan are recognized, while three sub-species of Bornean orangutan are now recognized: the northwest (*P.p. pygmaeus*), the central (*P.p. wurmbii*), and the northeast (*P.p. morio*) (Caldecott and Miles, 2005). There are currently around 7,300 Sumatran and 57,000 Bornean orangutans with numbers continuing to decline. The primary causes of the decline are logging, forest fires, illegal hunting, and the conversion of forests to farms and plantations (Caldecott and Miles, 2005). According to the IUCN red list, *Pongo*

abelii is listed as “critically endangered,” while *Pongo pygmaeus* is listed as “endangered” (IUCN Red List, [online] accessed Jul 2008).

Apes in Captivity: All species of great ape are commonly kept in captivity, although chimpanzees are currently the only great ape used in biomedical research. The 2006 North America regional chimpanzee studbook lists 283 chimpanzees living at 39 zoos in North America, with a historic population of approximately 1,324 animals (Ross, 2006). This number comprises only a portion of the total number of chimpanzees held in captivity in North America, the remainder being found in research laboratories, the entertainment industry, as well as other facilities. According to the Gorilla Species Survival Plan (SSP), 355 gorillas are currently housed in 52 accredited institutions (Gorilla SSP, [online] accessed September 2008). The 2006 gorilla studbook lists a historical population of 1,938 individuals (Wharton, 2006). The orangutan international studbook lists 869 animals held worldwide in 215 facilities, with a historical population of 2,521 individuals (Perkins, 2002).

Research Goals

The primary focus of this research is to examine whether status (captive vs wild), sex, and species’ differences affect disease prevalence in the great apes. Controlling for age is also essential because age is a known contributor to disease expression. However, the age of wild skeletons is typically not known and current aging methods utilized in many studies involve the use of suture closure where only broad, and potentially inaccurate, age categories are possible. Thus, development of improved aging techniques

is necessary, particularly for studies that examine diseases that are strongly influenced by age. Consequently, the aging of the sample is a significant issue that requires evaluation of a variety of methods with the hope that a more rigorous means of determining age in unknown-aged apes is possible. Therefore, aging of the skeletons will be discussed in detail in chapter 3, while chapters 4 and 5 deal with the various analyses relating to OA. A summary of the hypotheses to be examined (in chapter 5) is presented below:

Hypothesis 1: Of interest is whether status (wild vs captive), with age as a factor, effects disease prevalence in the great apes. Difference in prevalence of the disease is expected because research on other animal species indicates that OA is more common in captive animals (eg: Rothschild and Woods, 1992). Thus, it is anticipated that *status will affect disease prevalence in that captive animals will experience higher rates of OA than wild animals, even after controlling for age.*

Hypothesis 2: Of interest is whether sex, with age as a factor, affects disease prevalence. In both controlled clinical and archaeological studies of humans, disease prevalence is typically evaluated separately by sex because the frequency and patterning of expression varies by sex. It is, however, possible that body size may be a confounding factor in sex differences; although, the few studies that have controlled for body mass have found contradictory results (DeRousseau, 1988; Weiss and Jurmain, 2007). Evaluation of disease prevalence by sex is warranted to determine what, if any, differences exist. However, a body size effect may still be inferred to be present if, after controlling for age, the males in each species have more OA than females *and* species-level differences of OA in males and females mirror species-level differences in sexual

dimorphism. Thus, it is anticipated that *sex will affect disease prevalence in that males will have higher rates of OA than females, even after controlling for age.*

Hypothesis 3: Of interest is the whether species' differences, with age as a factor, effects disease prevalence. This expectation stems from research that found wild chimpanzees to be less frequently affected than wild gorillas (Jurmain, 2000). Thus, it is anticipated that *species' differences will affect disease prevalence in that chimpanzees will experience less OA than either gorillas or orangutans and that gorillas and orangutans will not differ from each other, even after controlling for age.*

Research Significance.

This research has broad significance for aging studies in both great apes and humans, both in regard to aging techniques and in gaining a better understanding of whether differences in prevalence of osteoarthritis actually exist between wild and captive animals. Further, results from this study could improve our understanding of osteoarthritis from an evolutionary perspective, as well as have implications for answering the question of why the disease displays such a high prevalence in modern humans.

Maintaining optimal health in captive animals will be important in the coming decades because an increasing percentage of the world's great apes are likely to live in captivity as populations of wild apes continue to decline dramatically. The results from this study could have implications for institutions that house animals in captivity, particularly as studies that aid in prevention and treatment of disorders benefit captive animals as well as humans.

Chapter 2. Methods

Description of the Sample: The skeletons examined are housed at 16 different locations. These locations were chosen because of the availability of captive specimens, which are the limiting factor. Table 1 below lists the location and numbers of specimens examined at each institution. At some locations, such as the National Museum of Natural History in Washington, DC, and the Natural History Museum in London, UK, among others, more wild skeletons were available for study than were examined. Appendix 1 lists the specimens examined by location, ID number, sex, and status (captive or wild).

Table 1: Location and Number of Specimens

Location	Chimp		Gorilla		Orangutan	
	Wild	Captive	Wild	Captive	Wild	Captive
American Museum of Natural History, New York	23	3	22	3	3	2
Arizona State University	0	5	0	2	0	2
California Academy of Sciences	0	3	1	1	0	2
Field Museum, Chicago	5	7	7	11	2	10
Holloman Primate Facility, Alamogordo, NM	0	1	0	0	0	0
Maxwell Museum, University of New Mexico	0	3	0	0	0	3
Museum of Comparative Zoology, Harvard University	8	0	15	1	5	0
Museum of Vertebrate Zoology, UC Berkeley	0	1	2	2	0	0
National Museum of Natural History, Washington DC	9	1	8	0	14	2
Natural History Museum, London	10	1	8	3	18	1
Peabody Museum, Harvard University	1	1	1	0	2	0
Primate Foundation of Arizona	0	15	0	0	0	0
Royal Belgian Institute for the Natural Sciences, Belgium	1	1	1	1	0	3
Royal Museum for Central Africa, Belgium	0	5	0	1	0	0
Sam Noble Oklahoma Museum of Natural History	0	1	0	0	0	0
University of Arkansas at Little Rock	0	1	0	1	0	2
William R. Adams Primate Skeletal Collection, Indiana University	0	9	0	3	0	4
Totals	57	58	65	29	44	31

The Wild Sample: Wild specimens were collected from a variety of locations, with most of these specimens being obtained early in the last century by collectors in the field. There were numerous expeditions to Africa and Asia in the early 1900s, with each collector sending the specimens they gathered to specific museums. Generally, each of these collectors went to one or two locations in Africa or, in the case of orangutans, to either Borneo or Sumatra, with the goal of collecting specimens for a specific museum. For example, on Monday, 12th December, 1921, a New York Times article states that “A family of five gorillas has been bagged for the American Museum of Natural History by the Carl E. Akeley expedition,” and that the expedition “went to Africa for that purpose” (New York Times archives, [online] accessed Jul 2008). Thus, at any one institution it is likely that the specimens are from a limited geographical range and/or from a particular population. Because analysis of skeletons was carried out at various institutions, a broad geographical area and multiple populations are represented by this sample. Tables 2 lists the countries as specified on museum records, number of specimens collected in each country, and sub-species assigned by the museum or assigned based on location, if known.

Table 2: Wild Specimen Sample: Geographical and Sub-Species Data

Species	Country	Number of specimens	Sub-species
Chimpanzee	No locality specified	2	Unknown
	Rwanda	1	Eastern
	Zaire	2	Eastern
	Uganda	3	Eastern
	Gabon	8	Central
	Congo	11	Central
	Cameroon	20	Nigerian
	Niger	1	Nigerian
	Cote d I'Voire	2	Western
	West Africa	3	Western
	Liberia	1	Western
	Guinea	3	Western
Gorilla	No locality specified	2	Unknown
	Spanish Guinea	5	Western
	Gabon	3	Western
	Cameroon	36	Western
	Uganda	1	Mountain
	Congo	9	Mountain
	Zaire	2	Mountain
	Rwanda	7	Mountain
Orangutan	No Locality	6	Unknown
	Borneo	27	<i>Pongo pygmaeus</i>
	Sumatra	11	<i>Pongo abelii</i>

Of the study sample, most of the wild chimpanzee specimens were collected in the 1920s and 30s, with the earliest date recorded in 1909 and the latest in 1971. Wild orangutans were collected, for the most part, in the 1920s and 30s with the earliest recorded date in 1905 and the latest in 1937. Most of the wild gorillas were collected in the 1920s and 30s, with the earliest date recorded in 1907 and the latest date in 1977. One exception is the sample of mountain gorillas housed at the National Museum of Natural History in Washington, DC. In general, this sample was collected by the late Dian Fossey at her Karisoke Research Center in Rwanda during the 1970s. Some of these individuals had previously been buried and were then exhumed by Fossey in 1979.

Thus, the Fossey sample comprises a specific group due to manner of death, known history of some of the specimens, and the condition of skeletal remains (exhumed individuals).

The Study Group: An assumption being made is that the study group is representative of the great ape species, namely chimpanzees, gorillas, and orangutans. One issue is whether the sample structure is representative of the age and sex structures of wild populations and whether the frequency of OA is representative of those in uncollected specimens or, to put it another way, could there be an over-representation of arthritic individuals in the wild sample? It could also be assumed that the wild sample will include healthier individuals because these individuals were shot when encountered by humans, and the likelihood of encounter was probably unrelated to the animals' health. One might assume that, in contrast, the captive animals died of old age or were euthanized due to illness. However, the death of captive animals is likely to be unrelated to osteoarthritis, as the disease is one that progresses slowly and does not *cause* death. It is interesting to note that there are parallels in cause of death between wild and captive apes. For example, trauma and respiratory disease are the main factors in the deaths of wild gorillas and chimpanzees (Cranfield, 2008; Williams et al., 2008). Likewise, the main causes of death in captive infant chimpanzees were pneumonia and trauma (Courtney, 2005). Thus, while it may be assumed that access to medical care provides an advantage to captive apes, this is not necessarily always the case because, regardless of the level of medical care and intervention, animals do die. Nevertheless, wild and captive apes differ in their exposure to some potentially lethal factors. In the wild, many threats are human-influenced, such as habitat destruction and exposure to snares (which

do not necessarily cause death immediately, but often induce sufficient damage to cause death over time), while in captivity, space limitations, which affect the ability of an animal to escape personal conflicts, can lead to injury or stress-induced factors that may increase mortality. Whether the problems identified above actually affect the sample structure is difficult to prove; however, given the collection methods of wild animals and cause of death in captive animals (described below), it is unlikely that this is the case. Nevertheless, if there were differences in the sample structure, this would be an example of differential mortality, one of the problems in the osteological paradox (Wood et al., 1992).

In natural primate groups it is reasonable to expect uneven representation of both sex and age given both the social composition of groups (in general, uni-male gorilla harems, multi-male/female chimpanzees, and solitary orangutans) and variation in mortality rates. Synthetic life tables based on five study populations of wild chimpanzees show that males have higher mortality than females with some inter-site variation. Generally, chimpanzee life expectancy at birth is less than 15 years with mean adult lifespan (after sexual maturity) being about 15 years. Infant mortality is around 20.0% in the first year, dropping to 3.5% between ages 10-15 and, by age 30, the annual mortality rate is around 8.5% with an additional eight years of life expectancy (Hill et al., 2001). Nevertheless, maximum lifespan estimates for the wild apes are as follows: chimpanzees – 40-50 years; gorillas – 35-45 years, and orangutans – 45 years (Caldecott and Miles, 2005).

It is possible that the methods of collection could affect the sample structure, particularly if collectors focused only on one sex (eg: silverback male gorillas) or adults

only. Some reports state that field workers tried to collect one large male and one typical female from each local population (Schultz, 1935). Other collectors, such as the Akeley expedition in 1929, went after entire families and, as mentioned previously, were successful. Some expeditions pursued entire families but managed to shoot only one individual. For example, in the Virunga Mountains of Uganda on 27 December, 1925, an expedition shot an adult male gorilla from a group of ten or more. A label attached to the specimen (Field Museum # 26065) reads that “three other adult males seen but backs less grayish white. This old male last of herd to retreat.” This label is interesting for a number of reasons. First, the “old male” who was shot was the last of the group to leave, which is typical of gorilla behavior where females and the young retreat first and males (particularly the oldest, silverback male) are the groups’ defenders. Second, it is possible that this individual was the oldest member of the group and, given that OA is age-related, we could assume that he was the one most likely to exhibit some form of arthritis. However, we do not know the age composition of the other adults in the group or even if this “old adult male” was, in fact, old. Third, the label mentions a herd of ten or more but only specifies “three other adult males.” It is possible that the collectors were more interested in pursuing the silverback male than the other members of the group or that clear views of all group members was not possible due vegetative obstructions and/or the males’ defense of their group, or simply that adult male gorillas are much larger and impressive and thus more memorable. Nevertheless, it is likely that all group members would have been shot if the opportunity had arisen.

Yet other expeditions appear to have pursued any individual they encountered and they were apparently very determined in their goal. A label attached to an adult male

gorilla skeleton (Field Museum #27551) reads that in Zaire, on 15th March, 1924, the expedition pursued a "solitary old male which lived in second growth bush or old shambas. Hunted for three days and charged hunters twenty times or more." It is possible that this individual could have been an arthritic, old male, perhaps ousted from his group by a younger, fitter silverback. However, it is also possible that he was a fit, solitary male traveling alone, that other group members escaped the hunters' notice, and that this male was old but fit enough to evade death for three days. Indeed, it is likely that this male ran out of energy due to the mental and nutritional stressors brought on by being pursued for such a long time. Thus, the conclusion is that, in general, collectors appear to have pursued whatever specimens they could get without any conscious preference for age, sex, or overall health.

For some collections, there are differences in the numbers of male versus female skeletons available for study. For example, at the National Museum of Natural History in Washington DC, male lowland gorillas are available in greater numbers than female lowland gorillas (lowland 31/16 respectively). For most collections, it appears that orangutans were collected without bias for sex, which perhaps is an indication of methods of opportunistic hunting given that orangutans lead primarily solitary lives. For chimpanzees, it seems apparent that more female chimpanzees than males were collected. Chimpanzees live in fission-fusion social groups that divide into a number of subgroups. Their sociality seems to vary considerably from population to population with chimpanzee females in eastern Africa being more solitary than males, while in western Africa, chimpanzee females are more social and forage together (Fleagle, 1999; Lehmann and Boesch, 2008). Hunters have been known to kill adult females of all the great ape

species in order to obtain infants for zoos, research facilities, or the pet trade (indeed, shooting animals for the bush meat trade and pet trade continues to thrive in many areas); however, this practice was not common until the 1950s and the study specimens were generally collected two or three decades earlier. The reasons why there is disparity between numbers of male and female chimpanzee specimens is unclear. Regardless, the sample for this study was chosen to ensure that bias towards one sex was minimized. Table 3 below shows the distribution, by sex, of the wild skeletal samples:

Table 3: Wild skeletal sample, by sex

Sex	Chimpanzee	Gorilla	Orangutan
Male	28	36	20
Female	29	29	24
Totals	57	65	44

The age at death of wild individuals is not indicated in museum records, and there are numerous instances where even a generalized age at death (i.e., juvenile, adult, old adult) is not indicated at all. While aging of juveniles can be approximated based on formation and eruption of the dentition and stages of bone growth, aging of adults is frustratingly problematic. For the purposes of this study, and because OA is an age-related disease, infants, juveniles, and sub-adults were not included. Because of the problems associated with aging the wild sample, aging of the skeletons will be discussed in more detail in chapter 3. But, to give a general overview of the potential ages of the sample, an initial age was assigned to each specimen based on age categories utilized by other researchers (Lovell, 1990), and these data will be presented here. The following

initial age categories were utilized (all of these categories include complete permanent dentition to distinguish the subjects from juveniles):

Sub-Adult – proximal humeral epiphyseal line open; basilar suture open.

Adult – proximal humeral epiphyseal line fused; basilar suture open.

Old Adult – proximal humeral epiphyseal line fused; basilar suture fused.

Table 4 below shows the initial age distribution of the wild sample. In cases in which there was no skull available, individuals were assigned to an adult (no skull) category. This is because the only difference between the adult and old adult category is based on the appearance of the basilar suture and the absence of the basilar suture for analysis means that there is uncertainty as to whether these individuals would be assigned to an adult or old adult category. Thus, the more conservative age category was selected. Based on these initial aging categories, there appears to be a strong bias of old adults in both the chimpanzee and gorilla samples, while the orangutan sample is less biased towards the old adult category; however, refer to chapter 3 for a more detailed discussion of the age distribution of the sample

Table 4: Initial age distribution of the wild sample

Age Category	Chimpanzee	Gorilla	Orangutan
Adult (no skull)	7	10	10
Adult	6	7	13
Old Adult	44	48	21
Totals	57	65	44

The Captive Sample: Skeletons of captive great apes are, unfortunately, limited in number. There are a number of potential reasons for this. First, museums have

traditionally focused their attention on gathering wild samples. Second, there appears to be a sense that wild animal specimens are much more valuable to researchers than their captive counterparts and this idea is supported by the fact that many researcher are not interested in, and do not use, captive specimens. However, comparative studies could be undertaken by researchers and it is possible that a lack of specimens deters such an approach. Third, some research facilities and zoos are not willing to donate their deceased animals to a museum. Sometimes, this is due to issues of ownership, but also could be due to a lack of communication between museums and the zoo community. In some cases, even if a zoo is willing to donate specimens, there are no pre-existing agreements set up and thus if an animal dies, disposing of the body quickly becomes the priority. For many facilities, this means incineration of the dead animal. Due to the limited number of captive skeletons available for study, bias towards one sex is a likely, but unavoidable, outcome. Table 5 below shows the distribution, by sex, of the captive skeletal samples:

Table 5: Captive skeletal sample, by sex

Sex	Chimpanzee	Gorilla	Orangutan
Male	29	17	20
Female	26	12	11
Unknown	3		
Totals	58	29	31

The captive specimens used in this study came from a variety of locations. Most originated in zoos, while some were donated to museums by research institutions and circuses. One research institution, the Primate Foundation of Arizona, has accumulated the largest number of captive chimpanzee skeletons available in any one location (n=28,

of which 15 were used in this study). The advantage of this collection is that the foundation has kept meticulous records on almost every individual (the exception is two skeletons that were exhumed elsewhere and donated to the foundation). It was originally anticipated that, in general, captive skeletons would be accompanied by detailed records on each individual, such as known age for captive-bred or estimated age for wild-caught animals, where the animal originated from and, perhaps, some medical history, such as cause of death. In some instances, individual detailed records were available, but this proved to be the exception rather than the rule. Some locations had partial records (such as name of zoo and date of death) that enabled an animal's history to be traced by utilizing studbooks or by contacting the zoo or research facility directly. Yet other locations had no records at all. In these cases, any hope of tracing the history of an animal was lost. It should be noted, however, that some of these specimens were donated to museums at a time when records were sparse or not kept at all. For example, the earliest captive chimpanzee was donated to a museum in 1894 by a circus and nine other chimpanzee specimens were donated to various museums prior to 1950. Four captive gorillas were donated in the 1930s and 40s while six orangutans were donated to various museums prior to 1950. The likelihood is that no records were available on these animals. Table 6 below details the origin of the captive sample and whether the animals were wild-caught (usually at 1-2 years of age), captive-bred, or of unknown origin. Decade of donation to the museum is also indicated. At first glance, it appears that a larger number of specimens have been donated to museums since 1980; however, given the relatively high number of unknown donation dates, this may or may not be true.

Table 6: Historical origin of captive specimens

Origin	Chimpanzee	Gorilla	Orangutan
Wild Caught	19	17	11
Captive Bred	5	4	3
Unknown	34	8	17
Donation:			
Unknown	16	6	8
Prior to 1920s	1	0	0
1920-1940s	9	4	6
1950-1970s	8	8	7
1980-1990s	19	10	7
2000 on	5	1	3

The age of death of captive individuals was certain in some cases, estimated to within 2 years in the case of wild-caught animals, while in others an estimated numerical age was not possible at all. In cases where age was not known or traceable through records, individual specimens were assigned to the same aging categories initially utilized for the wild sample.

It is often assumed that because captive individuals have the potential to live to a more advanced age than their wild counterparts, that the captive sample will be heavily biased towards old individuals. This is not the case for the known-aged chimpanzees and gorillas in this study, while for known-aged orangutans there is some bias towards older individuals. Of the 31 known-aged chimpanzees, 13 (41.9%) died before the age of 20, while only 3 (9.7%) lived into their 40s. Synthetic life tables for captive chimpanzees suggest that 35-50% of all individuals survive to age 30, while synthetic life tables for wild chimpanzees suggest that 11.0% of males and 18.0% of female chimpanzees are expected to survive from age 15 to 40 (Hill, et al., 2001). This particular sample appears to show a mortality trend more similar to that of wild chimpanzees; however, as

explained below, a particular infection (coccidioidomycosis) was a strong factor in the deaths of many of the chimpanzees in this sample. Of the 22 known-aged gorillas, 9 (40.9%) died before the age of 20, while none lived to reach 40 years of age. For orangutans, the story is different in that only 4 (23.5%) of the 17 known-aged orangutans died before the age of 20, while 11 (64.7%) lived past 30 years of age, with one individual reaching 45 years of age. Table 7 below provides details of the ages of the captive sample. Initially, there appears to be bias in individuals of unknown age with significantly more old adults; however, these age categories are the initial age assignments (based on closure of the basilar suture) and this issue is addressed in more detail in chapter 3.

Table 7: Age at Death of the Captive Sample:

Age at death	Chimpanzee	Gorilla	Orangutan
<20 years	13	9	4
20-29 yrs	9	8	2
30-39 yrs	6	5	10
41+ yrs	3	0	1
Adult (no skull)	6	2	2
Adult	2	0	2
Old Adult	19	5	10
Total	58	29	31

Of interest is why so many of these known-aged individuals died at a relatively early age. While the mortality of captive individuals is not the primary focus of this study, a brief summary of cause of death, if known, is provided. In the case of chimpanzees, 24 individuals had a known cause of death. Of these, 13 (54.0%) died of coccidioidomycosis, a systemic infection caused by a soil fungus and endemic to the southwestern United States, northern Mexico, and a few regions in Central and South

America. All of these chimpanzees lived in Arizona, an area known to be endemic for the disease (see Marzke and Merbs (1984) and Long and Merbs (1981) for more details). These chimpanzees ranged in age from 10-30 years (3 of unknown age) and died before the late 1980s when medical advancement in prevention of this disease was utilized for chimpanzees. Of the 11 remaining chimpanzees, 5 (20.8%) died of heart-related conditions (ages ~17, 18 (also had coccidioidomycosis), 22, 30 and 31). The remaining six died of the following causes: during childbirth (age 10), unidentified systemic infection (age 14), viral pneumonia (age 16 and also had coccidioidomycosis), septicemia (age 18), accidental suffocation (age 19), and euthanized (age 34; intestinal leiomyoma with necrosis causing secondary peritonitis).

Eleven gorillas had a known cause of death. Two were residents of Arizona, with one dying of coccidioidomycosis at age 20 and the other of intestinal lymphosarcoma at age 31 (this individual also had coccidioidomycosis). The remaining causes of death are as follows: mass on thymus (age 13), coronary thrombosis (age 15-16), toxemia (age 16 ½), myocardial infarction after dental procedure (age 20), colitis (age 20), died during attempted copulation (age 23), myocardial infarction during cage transfer (age 25), multiple organ failure (age 28), and euthanized (positive for Epstein-Barr and pneumonia, age 38). Of these 11 gorillas, 27.0% died of heart-related diseases with the remainder dying from a variety of other causes.

For orangutans, six known-aged individuals listed a cause of death as follows: senile (age 26), stroke (age 30), euthanized (no other details listed, age 34), cardiac failure (age 36), euthanized due to chronic kidney failure (age 38), and heart-related due to anesthesia (age 38 ½ - this individual lived in Arizona and also had

coccidioidomycosis). Of these six orangutans, 50.0% died of heart-related diseases with the remainder dying from a variety of other causes.

While the captive specimens were the limiting factor in this study, for the most part, only complete skeletons were utilized. In a few cases, skulls were missing but postcranial elements were present while, in other cases, only one side of the postcranial skeleton had been accessioned into a collection. In some cases, the skeletal elements were present, but full disarticulation and processing had not been accomplished. Thus, there were a few cases where the torso was left intact with ribs and vertebral elements remaining joined via cartilage, and this meant that scoring of the vertebrae could not be accomplished because the articular surfaces were not clearly visible. Further, in other cases, the condition of bones (i.e., presence of significant cartilage) meant that scoring could not be accomplished.

Methods of Analysis. One concern regarding analysis pertains to the sample size. In studies with limited resources, such as this one, power analysis is a useful tool to determine if sufficient power exists to find an expected difference. The power of a test is the probability that a given test will find an effect, assuming that one exists in the population. A desirable result would achieve a power of .8, which means that there is an 80% chance of detecting an effect if one genuinely exists (Field, 2005). Prior to data collection, a 2-sample binomial arcsine calculator was used to test the potential sample size at a significance level of 0.05. For wild specimens, OA prevalence data for chimps and gorillas was used for these species respectively (Jurmain, 2000). For orangutans, due to the lack of prevalence data for wild orangutans, the gorilla data was utilized because it

is the more conservative option given that wild gorillas have higher rates of OA than wild chimpanzees. Because studies on captive apes have not been undertaken, it is difficult to ascertain prevalence rates in captive samples, so samples were compared utilizing data from DeRousseau (1988) on macaques for the peripheral joints. This sample was chosen for two reasons: first, macaques are quadrupedal as are the African apes and second, the macaque sample used by DeRousseau was from a captive colony; thus, these macaques can be expected to have experienced similar conditions to other captive animals.

Prevalence rates in the four major joints of wild chimpanzees and gorillas are low ranging from an average of 0% in the elbow to 3.9% in the gorilla knee, while for macaques prevalence rates across age groups range from an average of 20% in the elbow to 43% in the hip (see table 9 below for prevalence rates in all joints tested). For VOP, prevalence rates found in wild apes were compared to a human sample (Jurmain, 2000). Pre-data collection results from the power analysis showed that the power was above 90% for all joints on all three great ape species ($\alpha = .05$), thereby indicating that sufficient analytical power exists.

One problem encountered with the sample was that actual numbers of specimens analyzed were significantly less than the pre-collection ‘anticipated’ numbers. In gathering data on the availability of samples, museum and facility databases were utilized. In some cases, databases were accessible via the internet, while in other cases information was requested, and received, directly from the research institution. Table 8 below shows the anticipated (pre-data collection) and actual numbers of skeletons used in this study:

Table 8: Numbers of skeletons available for study

	Pan	Gorilla	Pongo
Wild	Anticipated: >100 Actual: 57	Anticipated: >56 Actual: 65	Anticipated: ~62 Actual: 44
Captive	Anticipated: 97 Actual: 58	Anticipated: 46 Actual: 29	Anticipated: 60 Actual: 31

As previously mentioned, the limiting factor in this study was the availability of captive specimens. Wild specimen numbers were therefore chosen to approximate that of the captive specimens. There were several reasons for the reduction in numbers of skeletal specimens available. Data collection was discarded on a number of specimens because records, accessed prior to data collection, regarding age and/or other information were not specific enough. For example, at the American Museum of Natural History in New York, the database listed approximately 75 chimpanzee skeletons as available for study of which 26 adults were analyzed. Of the specimens listed in the museum's catalog, 31 were juvenile, 1 was missing, 11 were mounted, and 6 were casts of faces, meaning that none of these specimens could be utilized for this study. Analysis of orangutans at the museum was equally disappointing because of 13 potential captive specimens, 10 were juvenile, 1 had been exchanged, and only 2 were available for analysis. It is unfortunate that similar problems were encountered at other institutions with specimens either being out on loan, missing, fully articulated and mounted, only partially processed, or too young for this analysis. In most cases, the databases generally did not reflect all of the information that would be useful when compiling a dataset.

The question then becomes how the final numbers of specimens affect the power of the analysis. Calculating power after the data has been collected and when final

numbers of specimens are known is less useful as power calculations are primarily used for design and not analysis. Nevertheless, revised estimates of the power, given the final sample sizes, are presented in table 9 below. In general, the results indicate that sufficient power exists in all but two joints, the gorilla and orangutan knee. Thus, although the reliability of the results for the gorilla and orangutan knee are less certain, until more captive specimens are available for study, studies such as this provide the best estimates currently available.

Table 9: Revised Power Analysis

	Joint	Wild* %	Captive+ %	Power %
Chimpanzee Wild:N=57 Captive:N=58	VOP	0.4	34	99.9
	Shoulder	1.4	28	98.7
	Elbow	0.1	20	95.4
	Hip	2.1	43	100
	Knee	2.9	21	85.9
Gorilla Wild: N=65 Captive: N=29	VOP	1.4	34	98.2
	Shoulder	2.3	28	92.7
	Elbow	0.1	20	89.5
	Hip	2.4	43	99.6
	Knee	3.9	21	71.3
Orangutan Wild: N=44 Captive: N=31	VOP	1.4	34	96.9
	Shoulder	2.3	28	89
	Elbow	0.1	20	84.1
	Hip	2.4	43	99.3
	Knee	3.9	21	63.6

* Wild percentages are an average for chimps and gorillas from data contained in Jurmain (2000)

+ Captive percentages for the four major joints are an average across age groups for macaques from DeRousseau (1988). For the vertebrae (VOP), the data is from a human sample (Jurmain, 2000)

Data Analysis. The data were analyzed using SPSS 16.0 for Windows. Within SPSS, linear regression and logistic regression were utilized. Each of these methods is discussed below:

Linear Regression. Regression analysis is used to fit a predictive model to a set of data and to use that model to predict values of the dependent variable from one or more independent variables. In linear regression, the model fitted is a linear model meaning that it is based on a straight line. This fitted line is then used to predict the dependent variable for a given value of either a single independent variable (simple linear regression) or multiple explanatory variables (multiple linear regression). To find the line that best describes the data, the method of least squares is utilized. The general statistical model for linear regression takes the form of

$$y = \beta_0 + \beta_1x_1 + \beta_2x_2 + \dots + \beta_px_p + \varepsilon_{1\dots p}$$

where p represents the number of predictor variables used to calculate y , and ε represents the residual or error left over after the model is fitted. β_0 represents the intercept (or constant), where the line intersects the vertical axis at $x = 0$. The $\beta_{1\dots p}$ values represent the slope values (or coefficients) for each of the predictors. The regression analysis tests the null hypothesis that each β value is equal to zero, or has no effect on the dependent variable. Significant effects of predictor variables indicate that changing the value of the predictors influences the value of the dependent variable. Linear regression analysis assumes that the residuals in the model are random, normally distributed variables with a mean of 0, the values of the outcome variable are independent, the data displays linearity, and the predictors should have some variation in value (Field, 2005). In this study,

multiple linear regression is utilized to analyze skeletal age markers, to include molar dental wear, sternal rib ends, auricular surface, and the acetabulum.

Logistic Regression: Logistic regression is multiple regression but with an outcome variable that is a categorical dichotomy and predictor variables that are continuous or categorical. Hence, logistic regression can be used to predict a dependent variable on the basis of continuous and/or categorical independents and to determine the percent of variance in the dependent variable explained by the independents. This simply means that we can predict which of two categories an individual is likely to belong to given certain other information. There are some parallels between linear and logistic regression, but whereas linear regression assumes that the relationship between variables is linear, and thus cannot be applied directly to a situation with an outcome variable that is dichotomous, logistic regression expresses the multiple linear regression equation in logarithmic terms, which has the effect of making the form of the relationship linear while leaving the relationship itself as non-linear. Thus, logistic regression overcomes the problem of violating the assumption of linearity. In logistic regression, we predict the probability of y occurring given known values of X_I (or X_s). In its simplest form, when there is only one predictor variable X_I , the equation from which the probability of y is predicted is:

$$P(y) = \frac{1}{1 + e^{-(\beta_0 + \beta_1 X_1)}}$$

where $P(y)$ is the probability of y occurring, e is the base of natural logarithms, and the other coefficients form a linear combination much the same as in simple regression.

When there are several predictors, the equation becomes:

$$P(y) = \frac{1}{1 + e^{-(\beta_0 + \beta_1 X_1 + \beta_2 X_2 + \dots + \beta_n X_n)}}$$

(Field, 2005). The logistic regression equation described above is the appropriate model to use for dependent variables that are categorical dichotomies, as is the case in this study. Logistic regression is used to analyze disease prevalence where the dependent variables (skeletal markers) are categorical dichotomies (absent/present) and the independent variables are categorical (status, sex, species) or continuous (age). While the preferred method of analyses would be to examine issues of disease severity (as indicated by use of an ordinal scaling system for data collection), the data do not allow for this level of analyses and a binary response (absent/present) is utilized, the reasons for which will be discussed in more detail in chapter 5.

Chapter 3. Aging of the Sample

Introduction. In some research studies, it is often sufficient to distinguish skeletons as infant, juvenile, or adult. Attempts are sometimes made to separate adults into young adult and old adult categories; however, predicting age with some degree of accuracy for adult great ape skeletons (in particular) has proven both difficult and elusive. Nevertheless, in studies such as this establishing a realistic skeletal age is critical. This is because controlling for age is essential when studying a disease where age is a known contributor to disease expression.

It is not unreasonable to assume that apes will follow similar trajectories to humans with regards to age markers and age-related changes appear to follow some basic principles (Morbeck et al., 2002). Kerley (1966) concluded that the chronological age in years for chimps is about 2/3 that of humans of the same skeletal age and that chimps “fall between monkeys and man with a stronger resemblance to man” in aging changes.

While the age of infant and juvenile great ape skeletons can, in general, be reliably attained based primarily on sequence of dental eruption and stages of bone growth, aging of adult great ape skeletons is problematic. Early studies of ape skeletons focused primarily on growth and development (Schultz, 1940, 1941, 1945, 1950, 1969), and it is largely from these studies that the current age categories were formulated. These categories are typically very broad comprising infant, juvenile, young adult (or adult), and old adult. In general, adults are separated from infants and juveniles based on completion of the permanent dentition and fusion of the proximal humerus, while old adults are distinguished from the others based on closure of the basilar suture (Taylor, 2002; Lovell, 1990).

In order to determine which, if any, human skeletal age markers has potential use for aging adult great ape skeletons, data were taken on the basilar suture, sternal rib ends (3rd, 4th, and 5th ribs), auricular surface, acetabulum, and dentition. The ribs, auricular surface, and acetabulum were scored based on human aging standards (discussed below). Dental wear was scored based on methods developed for humans and African apes (discussed below). One human aging marker that was not utilized was the pubic symphysis. This was because Kerley (1966) reported that the pubic symphysis begins to fuse at about 18 years of age in the chimpanzee, and so it was anticipated that a relatively high percentage of pubic symphysis fusion would be evident thus negating the utility of data collection for this aging marker. Thus, data were only recorded on whether the pubic symphysis was fused or unfused. Table 10 below displays the results. Chimpanzees show the highest percentage of pubic symphysis fusion in 20.0% of cases, followed by gorillas in 12.7% of cases and orangutans in 10.7% of cases. As these percentages are relatively low, future research that analyzes the utility of the pubic symphysis for its use as an aging marker may be warranted.

Table 10: Pubic Symphysis Fusion in the Great Apes

Pubic Symphysis	# Fused	# Unfused	# missing/unobservable
Chimpanzee	23	86	6
Gorilla	12	74	8
Orangutan	8	64	3

Basilar Suture. The categorization of great ape skeletons as “adult” or “old adult” based on closure of the basilar suture is highly problematic. Suture closure usually occurs after all growth has ceased, and different sutures and different animals vary greatly in this respect. In the great apes, closure of all sutures begins early in

development, while in humans and most laboratory animals, sutures may never completely close (Pritchard et al., 1956). In humans, cranial suture closure is highly variable and is often seen as an unreliable method for aging generally only used when no other criteria are available or used in conjunction with other characteristics (Buikstra and Ubelaker, 1994). Buikstra and Ubelaker (1994) state that (in humans) “although cranial sutures generally close (fuse) with increasing age there is considerable variability in closure rates,” and that “such variation reduces the value of suture closure patterns for age estimation.” One study found human cranial vault suture closure to be highly variable with prolonged patency in some cases. It was concluded that the prolonged patency of some sutures may be due to external forces, such as the number of muscles affecting a suture. In addition, no significant differences were found between age and suture grade (Sabini and Elkowitz, 2006). However, while the sutures of the cranial vault (coronal, sagittal, squamosal, and lambdoid) form in a process of intramembranous growth, the basilar suture is of endochondral origin and thus similar to the epiphyses in timeframe of closure. Nevertheless, little is known about the age of basilar suture fusion in humans; although, it has been found to be completely fused in 100% of 21-year-old men (Kleplinger, 2006).

One study of non-human primates analyzed suture closure as an aging indicator. In the Darajani baboon, Bramblett (1969) found that vault suture closure exhibited marked sexual dimorphism (male sutures close earlier in life) and were extremely variable at age of obliteration. However, the basilar suture was found to be the least variable with closure beginning at around six years of age in females and slightly after six years of age in males. Closure of this suture was complete by 10 years of age in females

and 13 years of age in males (Bramblett, 1969). As baboons can live upwards of 30 years with females reaching maturity at roughly six years of age and males at around 10 years of age (Strum, 1987), this means that an “old age” category likely contains a significant proportion of adult or young adult individuals and spans two or more decades of life. This mirrors the problem associated with aging the apes based on closure of the basilar suture.

The great apes vary somewhat from humans in sequence of suture closure. The earliest suture to close in chimpanzees is the nasal suture (often closing in prenatal life), which is usually one of the last to become obliterated in humans (Schultz, 1940). In contrast, the premaxillary sutures close much earlier in humans than in apes (Schultz, 1941) as does the incisive suture (Braga, 1998). Schultz (1969) found that the main sutures of the chimpanzee neurocranium close at more advanced ages than other sutures, but that obliteration “*begins well before the dentition has been completed*” (emphasis added). This same study by Schultz reported that “the occipito-sphenoid or basilar suture is closed in the highest percentages of skulls, classified as adults by their full dentition.” Schultz also found that the occipito-mastoid suture was closed least frequently indicating that it likely remains open longer than the other sutures (Schultz, 1969). This is interesting given that the basilar suture, and not the occipito-mastoid, is the one used most frequently to categorize an individual as an “old adult.” Schultz (1940) also reported that the cranial sutures are “not completely obliterated *until all permanent teeth have erupted*...(emphasis added)” This means that suture closure occurs, except in abnormal cases, shortly after the permanent dentition and thus early in the second decade of life, leaving three or more decades accounted for by one ‘old adult’

category. Thus these age categories become particularly problematic when studying a disease that is thought to have a strong age-related component. This is particularly true when we consider that chimpanzees and gorillas reach adulthood by 11 years of age or perhaps even earlier, while orangutans are mature at between 10 and 12 years of age (Schultz, 1940, 1941, and 1945).

To determine the viability of using the basilar suture to age adult great ape skeletons, details on the state of suture closure were taken for all specimens, but with particular emphasis and analysis focused on known-aged individuals. For the purposes of this study, sutures were scored as open, partially closed, or closed. Open sutures demonstrated no evidence of any closure at the suture site. Partially closed sutures showed minimal signs of closure, while closed sutures are those that are completely fused and may even be completely obliterated. Table 11 below displays the results.

Table 11: Basilar Suture Closure of Known-Aged Captive Apes.

Basilar Suture	Chimpanzee	Gorilla	Orangutan
Open	1	1 (partially closed)	0
Closed	20	18	15
Age range of sample	10-48	13-39	13-45
Youngest age with closed suture	10	13	13
Oldest age with open suture	14	28 (partial)	n/a

Results: For known-aged captive specimens, only one chimpanzee, aged 14, had an open basilar suture. The remainder had closed sutures, with the youngest individual being 10 years of age. For gorillas, one individual demonstrated partial suture closure at age 28, while the remaining individuals had closed sutures with the youngest individual being 13 years of age. For orangutans, all basilar sutures were closed with the youngest individual being 13 years of age. Thus, while the basilar suture shows some variability

in age of closure, it is closed at a relatively early age in virtually all known-aged captive individuals. Thus, an “old adult” category most likely contains individuals who are, in fact, relatively young. This indicates that using the basilar suture as a means to categorize individual skeletons as “adult” or “old adult” is very imprecise.

Sternal Rib Ends. Methods for determining the age of adult human skeletons utilizing the sternal rib ends were developed in the 1980s. Initially, a phase analysis method was developed for age estimation of white males and white females using the 4th rib (Iskan et al., 1984 and 1985). Later, sex and race-related morphological variation was examined as was the applicability of the results to other populations. Iskan (1991) found morphological differences between both sex and race (black/white) and noted that population specificity might also be a problem. Đonić and Đurić (2005) tested Iskan’s phase method on a Balkan population finding significant differences, in both males and females, between real chronological age and phase method values, particularly in phases 6 and 7. Oettlé and Steyn (2000) tested Iskan’s method on black individuals from South Africa finding the method was less accurate for that population. They developed new phases with adjusted criteria and age ranges specific to that population.

Other ribs were tested for their usefulness in predicting age and studies also have evaluated the usefulness of the ribs for aging (eg: Loth et al., 1994, Russell et al., 1993, and Baccino et al., 1999). Loth et al., (1994) found that age could be assessed using the rib phase method on the 3rd and 5th ribs, while Atkas et al., (2004), testing the 3rd, 4th, and 5th ribs on a Turkish population, obtained mixed results finding some variation in the male sample but concordance in the female sample. In a study testing the applicability of

the phase method on the left and right 2nd, 3rd, and 5th through 9th ribs, questions of statistical significance and accuracy when using ribs other than the 4th arose and composite score were recommended instead (Yoder et al., 2000). Thus, while the 4th rib appears to provide the best age estimates, the 3rd and 5th ribs also provide reasonable results, while the remaining ribs are of questionable utility.

In general, Iscan's phase method is thought to be useful for aging adult human skeletons. The phases range from 0 – 8 covering ages of 16 and younger (phase 0) to 65 and older (phase 8) for males and 13 and younger (phase 0) to 70 and older (phase 8) for females (Iscan and Loth, 1993). Rib phase casts were developed by Iscan and Loth (1993) giving specific age ranges for males and females. These phases were developed separately for males and females because of the sufficiently different aging processes seen in the male and female ribs (Iscan et al., 1984 and 1985). For humans, the rib phases are based on changes noted in the form, shape, texture, and overall quality of the sternal rib end. In general, the rib features progress from a flat articular surface with billowy appearance and regular, rounded rim edges, to a surface pit that assumes a V-shape and that deepens with age. The rim progresses from a regular, rounded border to a scalloped shape, and then grows increasingly sharp and irregular. Later, the walls become thinner with sharper edges and the pit becomes noticeably deep with a wide U-shape. The bone becomes more porous later in life and the walls become thin and fragile with sharp, highly irregular edges and bony projections (see Iscan rib phase casts (1993) and Iscan et al., (1984 and 1985) for specific details of each phase).

Methods: Based on the morphological changes noted in human sternal rib ends, the following characters were scored for the left and right 3rd, 4th, and 5th ribs: margins –

flat, scalloped, irregular; pit depth – none, small, medium, deep; pit shape – none, V, U; wall quality – good, poor; porosity – absent, micro, macro; extremities – absent, present; ossified nodules – absent, present. Table 12 below gives the morphological descriptions for each variable and each state. Photographs are provided for further reference.

Table 12: Morphological Descriptions of Rib Aging Variables

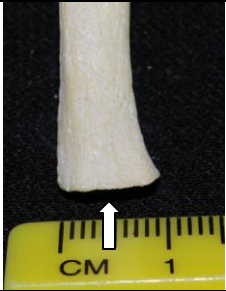
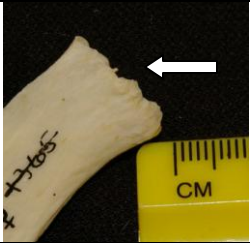



Variable	State	Code	Description	Photograph of states
Margins	Flat	1	The rim is rounded and regular with the bone being firm, smooth, and solid	
	Scalloped	2	The rim remains rounded and firm but takes on a scalloped appearance along the margin	
	Irregular	3	The rim becomes sharp and irregular in shape	
Pit Depth	None	1	The articular surface is relatively flat	
	Small	2	A small indentation, or pit, can be seen in the articular surface of approximately 1mm in depth	

Table 12: Morphological Descriptions of Rib Aging Variables continued...






	Medium	3	The articular surface pit increases in depth to approximately 2-3mm	
	Deep	4	The articular surface pit is significant in size and is greater than 3mm in depth	
Pit Shape	None	1	The articular surface pit is flat without shape	
	V	2	The articular surface pit forms a V-shape between the anterior and posterior walls	
	U	3	The articular surface pit forms a wide U shape between the anterior and posterior walls	

Table 12: Morphological Descriptions of Rib Aging Variables continued...









Extremities	Absent	1	The rim can be flat, scalloped, or irregular but is without bony projections at the superior and inferior ends	
	Present	2	Bony projection(s) are evident at the superior and/or inferior ends. This projection can be small or large in size (as in example photograph). The rim is usually irregular in shape.	
Wall Quality	Good	1	The walls are firm and thick maintaining their shape. The walls feel firm when gently squeezed between the fingers.	
	Poor	2	The quality of the walls has deteriorated and they are thin and pliable when squeezed gently between the fingers	This condition was not observed in the ape skeletons. Photograph unavailable.
Porosity	Absent	1	No porosity is evident inside the pit. The floor of the pit is dense.	

Table 12: Morphological Descriptions of Rib Aging Variables continued...

	Micro	2	Pin-prick sized porosity is evident within the pit and the floor becomes less dense.	
	Macro	3	Loss of density with porosity seen easily with the naked eye when the rib is held at arms length. Microporosity is usually present.	
Nodules	Absent	1	No additional bony growth is seen within the pit. The pit is smooth and dense.	
	Present	2	One or more bony nodules are located in the interior of the pit	

Results: For all three species, linear regression utilizing the stepwise method was performed on both the pooled data (3rd, 4th, and 5th ribs) and on the 4th rib separately on all known-aged individuals. As mentioned earlier, there were 31 known-aged chimpanzees, 22 known-aged gorillas, and 17 known-aged orangutans. For all three species, of the seven variables scored on each rib, one variable (wall quality) remained constant, meaning that there was a lack of variation in this feature. In the pooled analyses (3rd, 4th, and 5th ribs), there were 36 predictors included in the model (6 variables on each of the left and right ribs). Due to missing data, only eight chimpanzees, five gorillas, and two orangutans were included in the analyses. Statistics could not be computed on the orangutans. Plots of the standardized residuals against standardized predicted values are shown for each test and should look like a random array of dots evenly dispersed around zero; however, in some cases it is difficult to interpret the plots due to few observations. Thus, any interpretation and suggestion of problems associated with these plots should be considered tentative. The results for chimpanzees and gorillas are as follows:

Chimpanzees: For chimpanzees, five variables were constant or there were too few complete cases to estimate the correlation, while the majority of the remaining variables were excluded from the analysis. Only two variables (5th rib left pit depth (Pearson's correlation = .787, $p=.010$) and 5th rib right porosity (Pearson's correlation = .710, $p=.024$)) remained in the final model. This final model had an R^2 of .909 and was significant at $p=.002$. The plot of the standardized residuals against standardized predicted values shown in figure 1 below demonstrates some evidence of non-linearity, and a histogram of the residuals shown in figure 2 below demonstrates evidence of non-normal distribution. A Kolmogorov-Smirnov (K-S) normality test of the standardized

residuals ($p=.034$) confirms that these deviations were significant. Scatterplots of the residuals of the outcome variable and each of the predictors in the model are shown in figures 3 and 4 below. The scatterplot of the 5th left rib pit depth (Rib5LPD) indicates a positive relationship with age, although there is indication of at least one outlier. The scatterplot of the 5th rib right porosity (Rib5RP) looks less linear with the pattern demonstrating a more random relationship of this variable with age.

Figure 1: Chimpanzee Pooled Ribs - Standardized Residuals plotted against Standardized Predicted Values

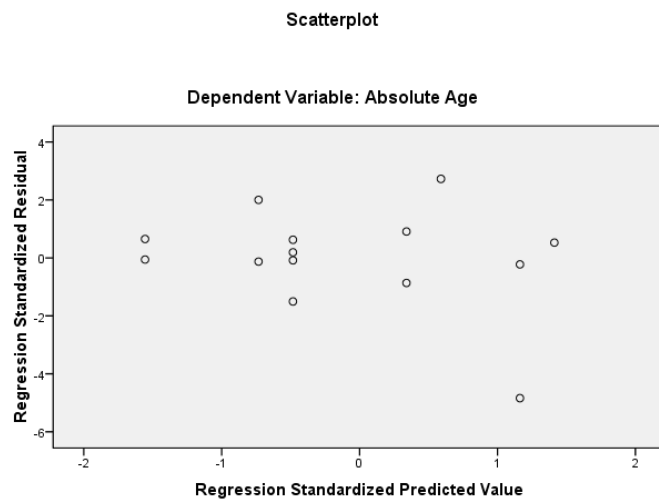


Figure 2: Chimpanzee Pooled Ribs – Histogram of Residuals

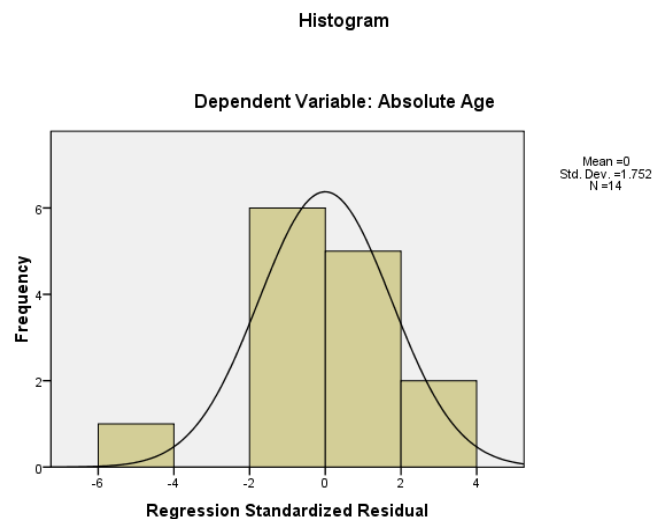


Figure 3: Chimpanzee Pooled Ribs - Partial Regression Scatterplot of the 5th left rib pit depth



Figure 4: Chimpanzee Pooled Ribs - Partial Regression Scatterplot of the 5th right rib porosity



Gorillas: For gorillas, six variables were constant or there were too few complete cases to estimate the correlation while the majority of the remaining variables were excluded from the analysis. Only one variable (5th rib right porosity (Pearson's correlation = -.954, $p=.006$) remained in the final model. This final model had an R^2 of .910 and was significant at $p=.012$. The plot of the standardized residuals against standardized predicted values shown in figure 5 below shows a non-linear relationship; although, this interpretation should be considered tentative as there are few observations. A histogram of the residuals shown in figure 6 below demonstrates evidence of non-normal distribution; although a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant.

Figure 5: Gorilla Pooled Ribs - Standardized Residuals plotted against Standardized Predicted Values

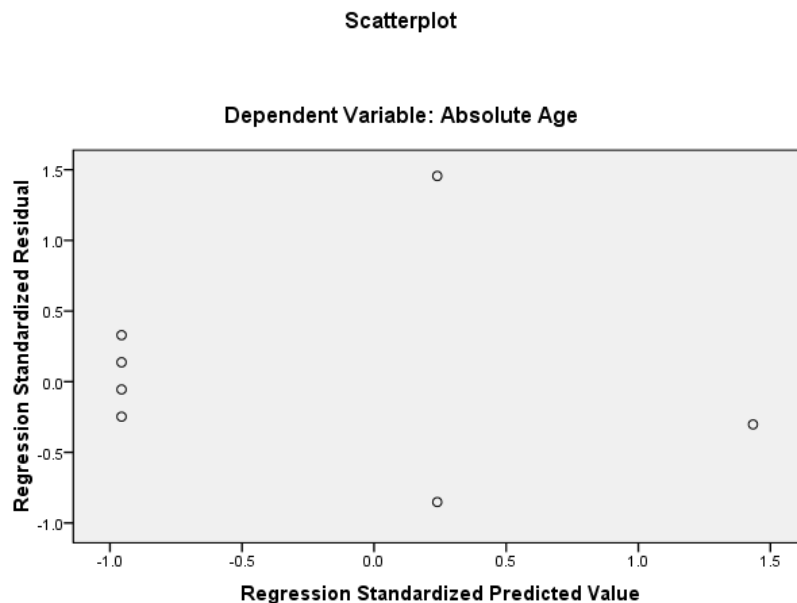
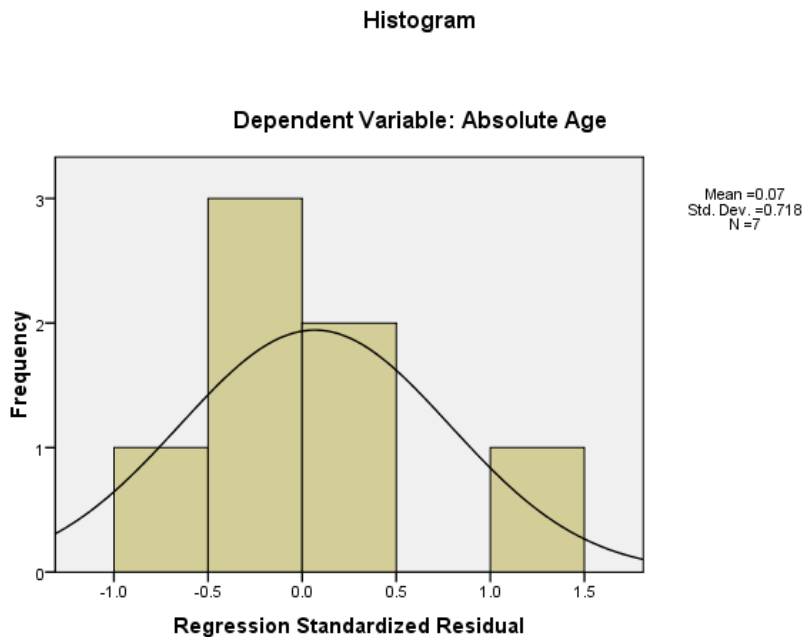


Figure 6: Gorilla Pooled Ribs – Histogram of Residuals



In the 4th rib-only analysis, there were 12 predictors included in the model (6 variables on each of the left and right ribs). Due to missing data, only 12 chimpanzees, 6 gorillas, and 6 orangutans were included in the analyses. The results are as follows:

Chimpanzees: For chimpanzees, one variable (left extremity) was constant or there were too few complete cases to estimate the correlation. Of the remaining 11 variables, only 1 variable (left pit depth (Pearson's correlation = .700, $p = .006$)) was included in the final model. This model had an R^2 of .490 and was significant at $p = .011$. The non-random nature of the plot of the standardized residuals against standardized predicted values shown in figure 7 below likely indicates a case of heteroscedasticity (non-constant variance), where at each point along any predictor variable the residuals are different; however, this assumption should be considered tentative due to the small number of observations. A histogram of the residuals shown in figure 8 below demonstrates a near-

normal distribution and a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant.

Figure 7: Chimpanzee 4th Rib - Standardized Residuals plotted against Standardized Predicted Values

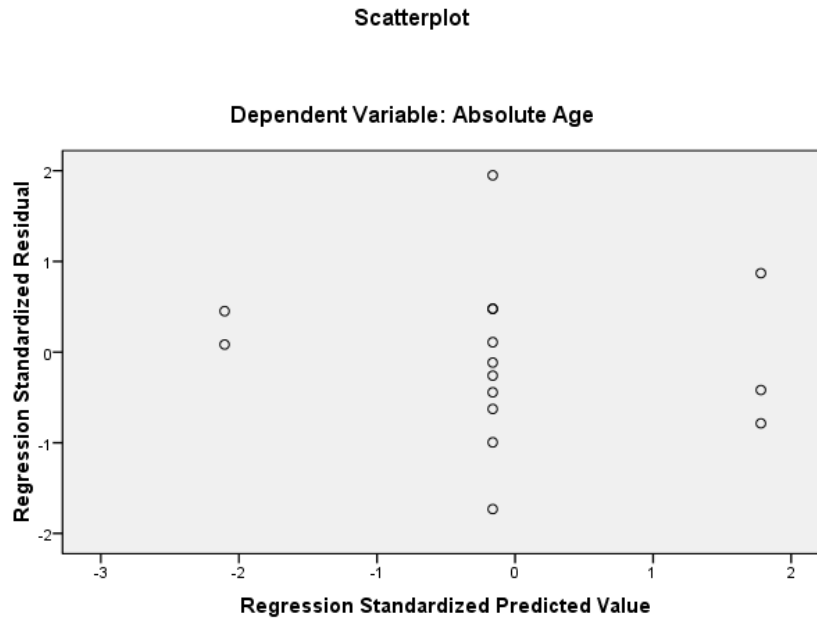
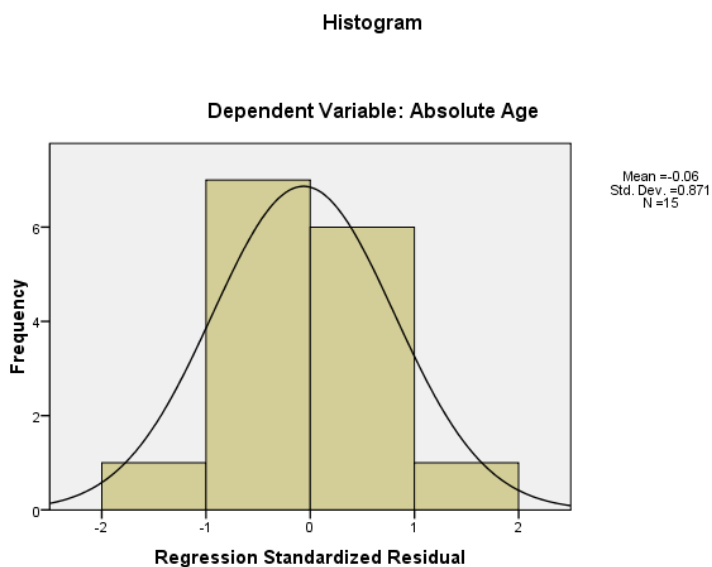


Figure 8: Chimpanzee 4th Rib – Histogram of Residuals



Gorillas: For gorillas, two variables (left and right margins) were constants or there were too few complete cases to estimate the correlation. Of the remaining 10 variables, 2 were included in the final model (left porosity (Pearson's correlation = $-.707$, $p=.058$) and right porosity (Pearson's correlation = $-.831$, $p=.020$)). This model had an R^2 of $.995$ and was significant at $p=.000$. The non-random nature of the plot of the standardized residuals against standardized predicted values shown in figure 9 below appears to indicate a case of heteroscedasticity; however, this assumption should be considered tentative due to the very small number of observations. A histogram of the residuals shown in figure 10 below demonstrates a non-normal distribution; although, a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant. The scatterplots of the 4th rib left porosity (Rib4LP) and 4th rib right porosity (Rib4RP) shown in figures 11 and 12 indicate a negative relationship with age with at least one outlier in each case.

Figure 9: Gorilla 4th Rib - Standardized Residuals plotted against Standardized Predicted Values

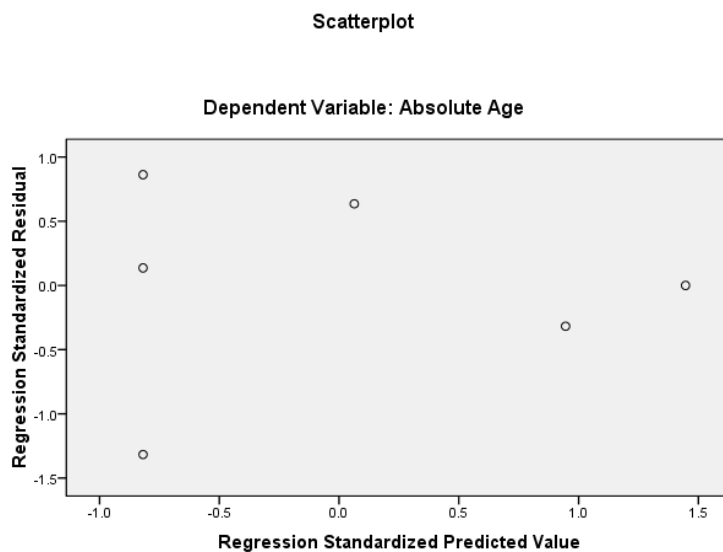


Figure 10: Gorilla 4th Rib – Histogram of Residuals

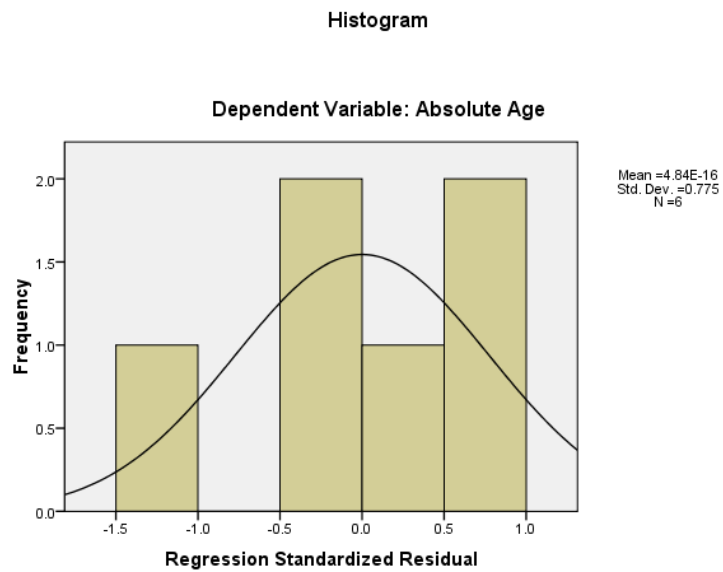
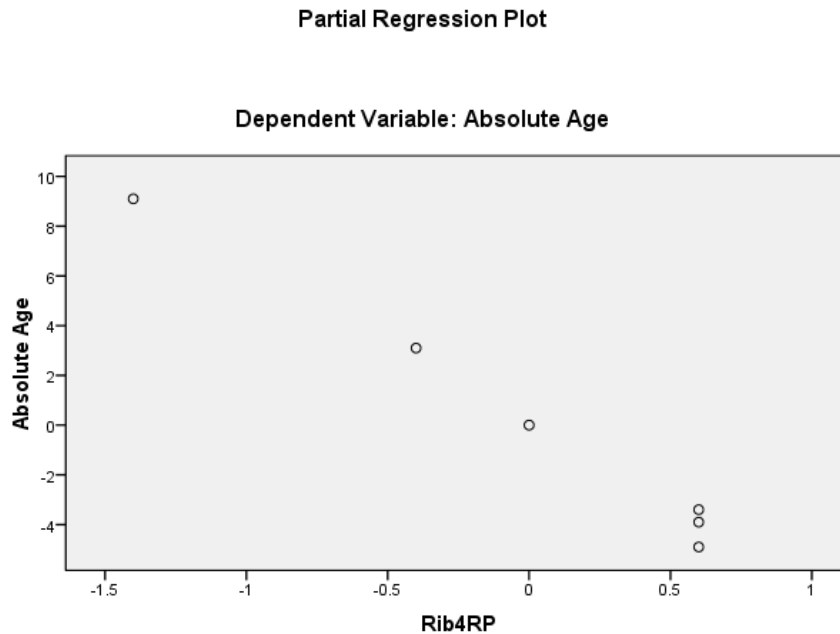


Figure 11: Gorilla 4th Rib - Partial Regression Scatterplot of the 4th left rib porosity



Figure 12: Gorilla 4th Rib - Partial Regression Scatterplot of the 4th right rib porosity



Orangutans: For orangutans, three variables were constants or there were too few complete cases to estimate the correlation. Of the remaining variables, the analyses could not be computed.

Summary: Overall, due to the limited number of variables that were significant only in the chimpanzee and gorilla, the ribs are not recommended as a useful indicator of age. The lack of fit could be due to the small sample or to a lack of relationship between features of the ribs and age. Plots of the residuals appear to demonstrate some evidence of non-linearity and non-normally distributed data, which would lend support to the idea that there is a lack of relationship between features of the ribs and age (although K-S normality tests of standardized residuals showed that, in general, these deviations were not significant); however, any suggestions of problems in the data should be considered

tentative due to the small number of observations. Thus a larger sample would be needed to verify the model. Aging of the apes utilizing features of the ribs appears to be further confounded due to potential differences as to which predictors (if any) are correlated with age among the three different species. As mentioned previously, in human aging studies morphological differences between sex, race (black/white), and different populations have been identified in the ribs (Iskan, 1991; Đonić and Đurić, 2005; Oettlé and Steyn, 2000). Thus, it is possible that because the same variables are not being highlighted as significant for each species, that these differences among the species reflect true morphological differences. However, given the small sample, this may or may not be true. Further investigation into potential species and/or sex differences may be warranted; however, the small sample negates this possibility at present.

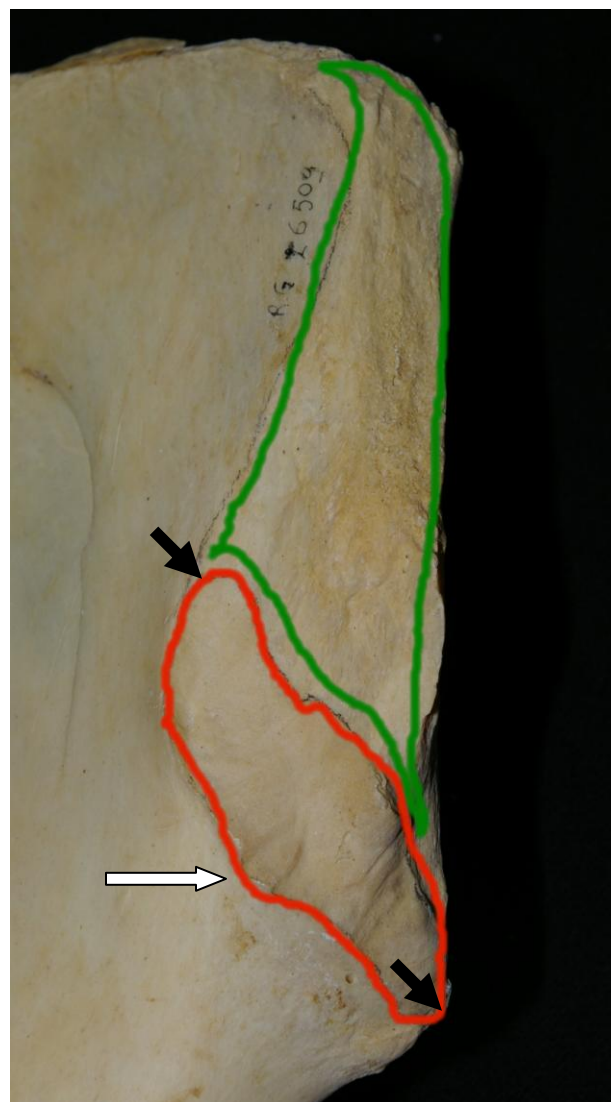
Auricular Surface. A method for determining the age of adult human skeletons utilizing the auricular surface of the os coxae was developed in the mid 1980s (Lovejoy, et al., 1985). A revised method based on Lovejoy et al., and utilizing composite scores was also proposed (Buckberry and Chamberlain, 2002). The Lovejoy et al.'s (1985) method has received some criticism that it does not give very accurate results, either underestimating the age at death of individuals older than 50 years or overestimating the age of younger individuals (Rissech et al., 2006); however, it is seen as a useful aging technique as the auricular surface is frequently preserved in archaeological settings (Buikstra and Ubelaker, 1994). The aging technique for the auricular surface is a phase method developed with phases ranging from 1 to 8 and ages ranging from 20-24 (phase 1) to 60+ (phase 8). The presence or absence of billowing, stria, and porosity are scored,

as is granularity and activity of the apical, retro, and surface extremities. In general, in stage 1 the auricular surface displays billowing and fine granularity with no porosity, retroauricular, or apical activity. As aging progresses, there is a loss of billowing, the presence of striae, and coarsening of granularity. Over time, the apical, retroauricular, and superior and inferior surfaces demonstrate change progressing from no activity (smooth appearance) to moderate and then pronounced activity (rugged appearance). Porosity (usually micro followed by macro) begins to appear in phase 3. In the later phases, there is complete loss of granularity, and marked surface irregularity, which becomes the principal feature. See Buikstra and Ubelaker (1994) or Lovejoy et al., (1985) for a more complete description of the phases and the corresponding morphological changes.

Methods. Based on morphological changes noted in the human auricular surface, the following characters were scored for the left and right auricular surface in the ape skeletons. Billowing – present, absent; Stria – absent, present; Fissure – absent, present; Porosity, absent, micro, macro; Apical activity – none/minimal, moderate, pronounced; Retro activity – none/minimal, moderate, pronounced; Superior activity – none/minimal, moderate, pronounced; Inferior activity – none/minimal, moderate, pronounced; Density (granularity) – superior facet – smooth, coarse; inferior facet – smooth, coarse; nodules – absent, present. Density was scored for superior and inferior facets as, in many of the ape skeletons, a distinct separation on this surface was evident. The presence, or absence, of fissures and nodules were also scored for their potential use as aging indicators. The diagram below (figure 13) shows the specific areas of the auricular surface. Billowing, stria, fissures, porosity, density, and nodules were scored on the demiface surface only.

Activity (apical, retro, superior and inferior) was scored at specific points as indicated in figure 13 below. The auricular surfaces of the three ape species differ somewhat in overall shape and size; however, the same principle for scoring and the area scored applies to all species. Figure 13 shows the features of the auricular surface, while table 13 below provides morphological descriptions of the variables along with the corresponding codes and photographs.

Figure 13: Features of the Auricular Surface



Auricular Surface Features:
Green line = retroauricular area
Red line = demiface surface
White arrow = apex
Black arrows = superior and inferior extremities

Table 13: Morphological Descriptions of the Auricular Aging Variables



Variable	States	Code	Description	Photograph of states
Billowing	Present	1	Broad, well-organized billows that cover most of the demiface surface; transverse organization	This condition was not seen in the apes. Photograph unavailable.
	Absent	2	Loss of billowing; demiface surface takes on a flatter, smooth appearance.	
Stria	Absent	1	Demiface surface has a relatively flat appearance, lacking grooves or striae.	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...



	Present	2	One or more striae (narrow grooves) appear on demiface surface.	
Fissure	Absent	1	The demiface surface is unmarred by blemishes or fissures.	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

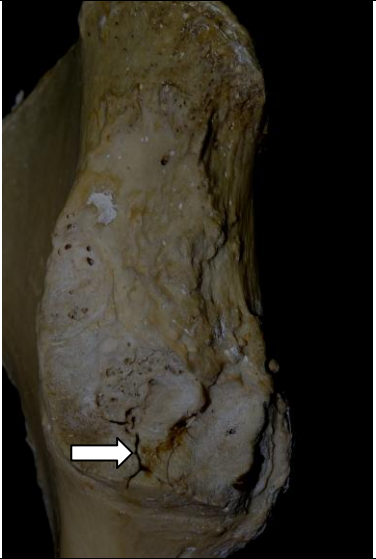
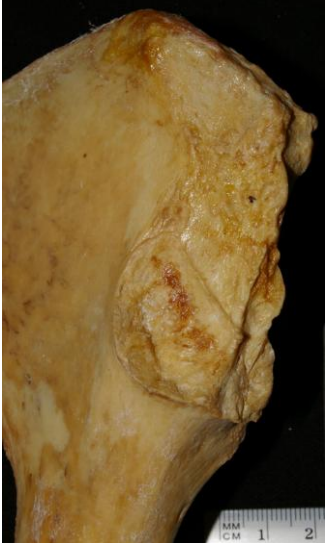
	Present	2	One or more fissures are noticeable in the demiface surface.	
Porosity	Absent	1	The demiface surface is unmarred by porosity maintaining a regular appearance	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

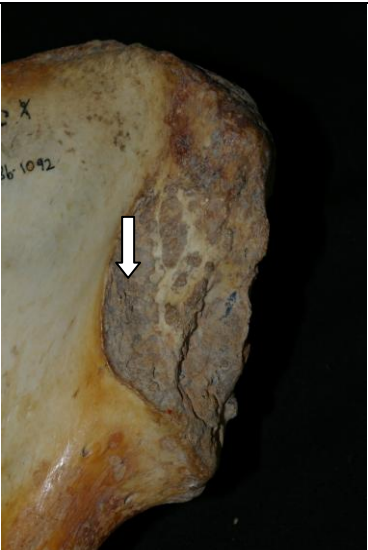
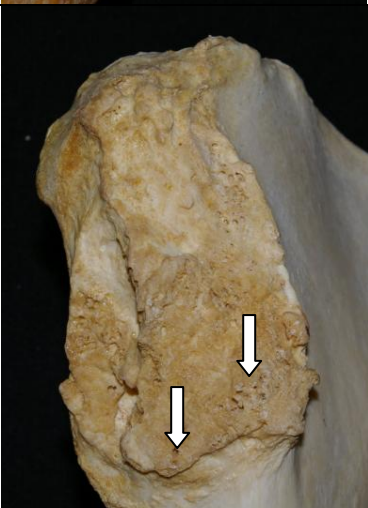
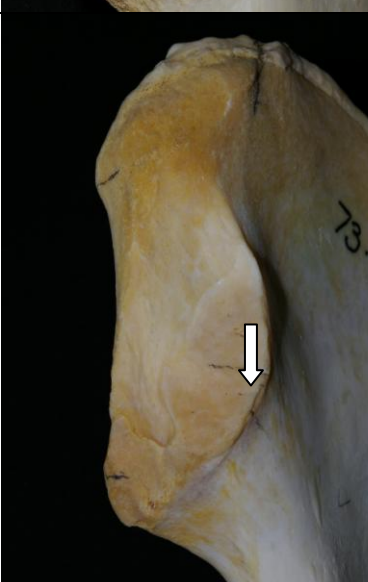
	Microporosity	2	Small pin-prick sized holes are evident on the demiface surface.	
	Macroporosity	3	Porosity on the demiface surface is large in size and can be seen easily with the naked eye when held at arms' length. Microporosity is usually present.	
Apical Activity	None/Minimal	1	The apex is rounded and relatively smooth to the touch. It might display a slightly bumpy appearance.	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

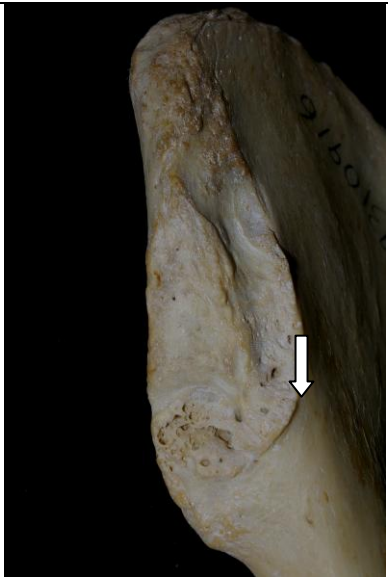

	Moderate	2	The apex is sharp with a small to moderate sized lip at the edge that is usually uniform in shape.	
	Pronounced	3	The apex is sharp with a large protruding lip that can be irregular in shape.	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...



Retro Activity	None/Minimal	1	The retroauricular area is smooth and has a relatively flat appearance.	
	Moderate	2	The retroauricular area displays slight ruggedness to the topography.	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

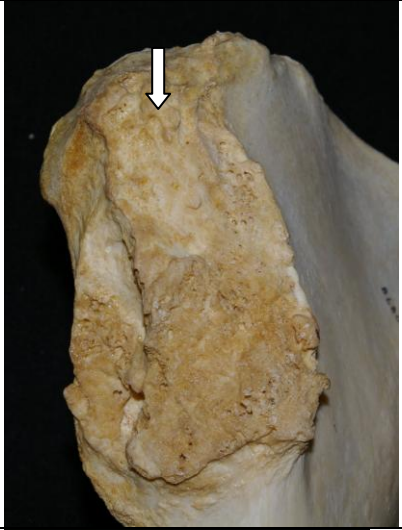
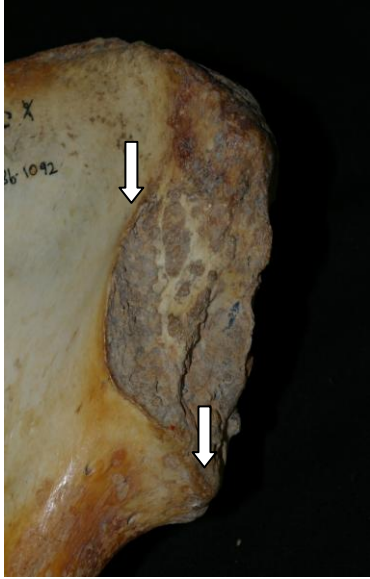
	Pronounced	3	The retroauricular area displays rugged topography that covers most of the surface	
Superior and Inferior Activity	None/Minimal	1	The superior and/or inferior margin is rounded and smooth to the touch. There may be a slight bump; however, this bump is rounded and smooth	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

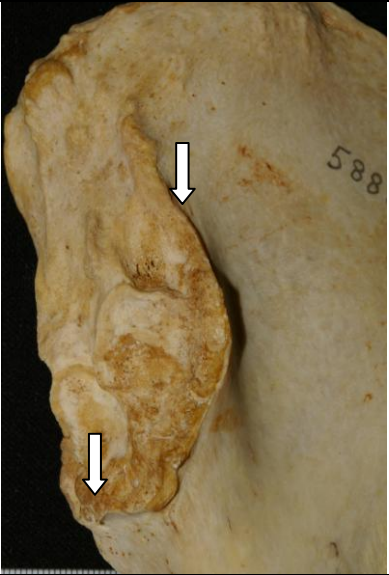
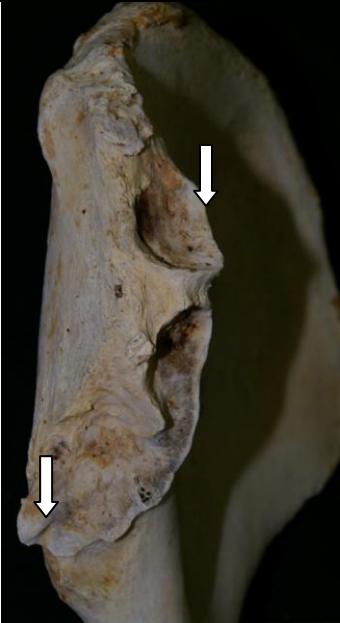
Moderate	2	The superior and/or inferior margin is sharp with a small to moderate sized lip at the edge that is usually uniform in shape.	
Pronounced	3	The superior and/or inferior margin is sharp with a large protruding lip that can be irregular in shape.	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

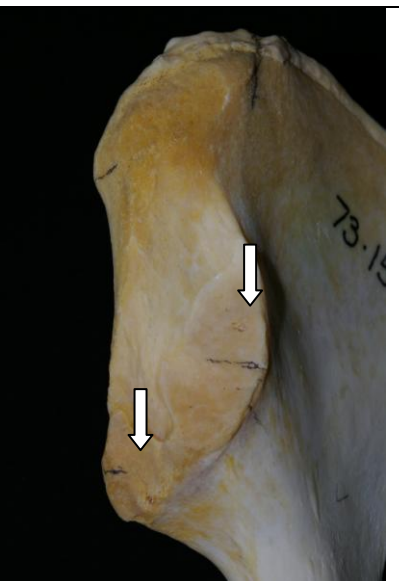



Density – Superior and Inferior	Smooth	1	Cortical bone on the surface has a fine-grained texture	
	Coarse	2	Cortical bone on the surface has a coarse- grained texture	

Table 13: Morphological Descriptions of the Auricular Aging Variables continued...

Nodules	Absent	1	Bony nodules are absent on the demiface surface.	
	Present	2	One or more bony nodules are evident on the demiface surface.	

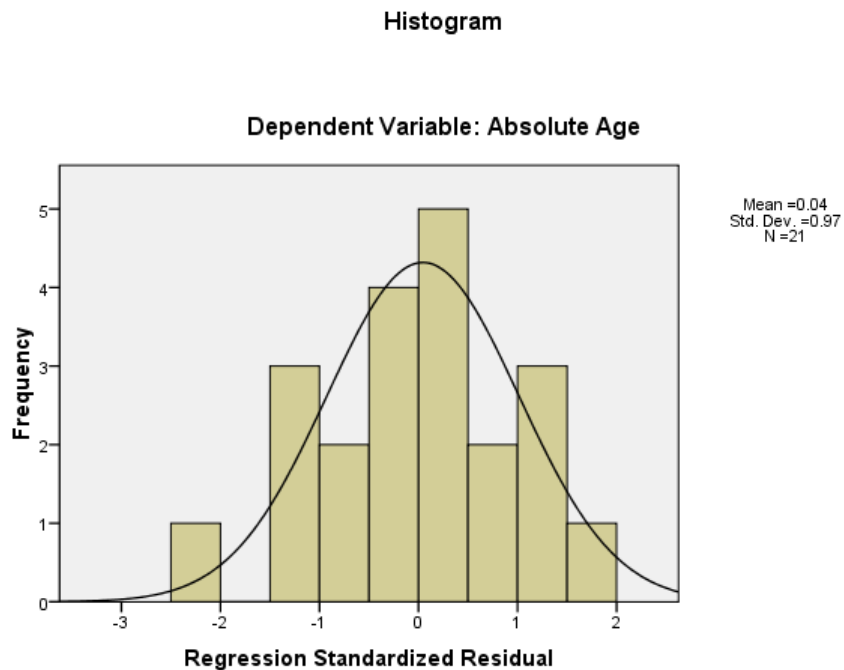
Results. For all three species, linear regression utilizing the stepwise method was performed on all known-aged specimens. As a reminder, there were 31 known-aged chimpanzees, 22 known-aged gorillas, and 17 known-aged orangutans. For all three species, of the 22 features scored on the left and right auricular surfaces (11 on each side), one variable (billowing) remained constant, meaning that there was a lack of variation in this feature. Plots of the standardized residuals against standardized predicted values are shown for each test and should look like a random array of dots evenly dispersed around zero; however, in some cases it is difficult to interpret the plots due to few observations. Thus, any interpretation and suggestion of problems associated with these plots should be considered tentative. The result for each species is as follows:

Chimpanzee: There were 20 chimpanzees included in the analyses. Of the 20 variables included in the analysis (10 on each of the left and right sides), 3 were constants or there were too few complete cases to estimate the correlation (left and right porosity, left nodules). Of the remaining 17 variables, only 1 (right retro activity (Pearson's correlation = .470, $p=.018$)) was included in the final model. This final model had an R^2 of .221 and was significant at $p=.036$. The non-random nature of the plot of the standardized residuals against standardized predicted values shown in figure 14 below likely indicates a case of heteroscedasticity (non-constant variance), where at each point along any predictor variable the residuals are different; however, this assumption should be considered tentative due to the small number of observations. A histogram of the residuals shown in figure 15 below demonstrates some evidence of a non-normal distribution; although, a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant.

Figure 14: Chimpanzee Auricular Surface - Standardized Residuals plotted against Standardized Predicted Values



Figure 15: Chimpanzee Auricular Surface – Histogram of Residuals



Gorilla: There were 8 gorillas included in the analysis. Of the 20 variables (10 on each of the left and right sides), 2 were constants or there were too few complete cases to estimate the correlation (left and right nodules). None of the remaining variables were individually significant and statistics could not be computed for this sample.

Orangutan: There were 12 orangutans included in the analysis. Of the 20 variables (10 on each of the left and right sides), all were included in the analysis. Only one variable (left inferior density (Pearson's correlation = .864, $p=.000$) was included in the final model. This final model had an R^2 of .746 and was significant at $p=.000$. The non-random nature of the plot of the standardized residuals against standardized predicted values shown in figure 16 below likely indicates a case of heteroscedasticity (non-constant variance), where at each point along any predictor variable the residuals are different; however, this assumption should be considered tentative due to the small number of observations. A histogram of the residuals shown in figure 17 below demonstrates evidence of a non-normal distribution, and a K-S normality test of the standardized residuals ($p=.049$) indicates that these deviations were significant.

Figure 16: Orangutan Auricular Surface - Standardized Residuals plotted against Standardized Predicted Values

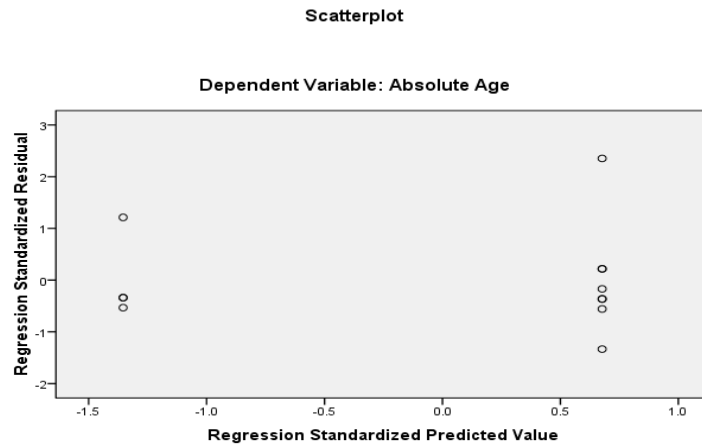
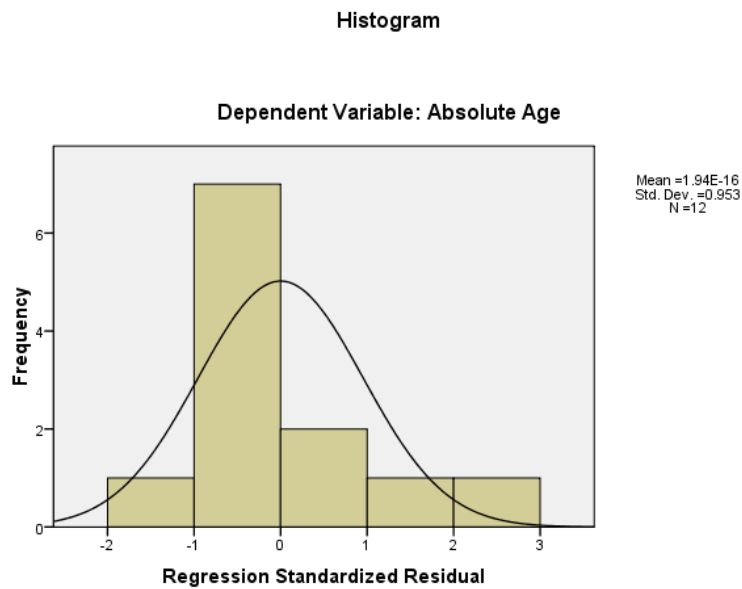


Figure 17: Orangutan Auricular Surface – Histogram of Residuals



Summary: Overall, due to the limited number of variables that were significant only in the chimpanzee and orangutan, the auricular surface is not recommended as a useful indicator of age. The lack of fit could be due to the small sample or to a lack of a

relationship between features of the auricular surface and age. Plots of the residuals appear to demonstrate some evidence of non-constant variance and non-normally distributed data, which would lend support to the idea that there is a lack of relationship between features of the auricular surface and age. K-S normality tests of standardized residuals showed that these deviations were significant in the orangutan but not in the chimpanzee; however, any suggestion of problems in the data should be considered tentative due to the small number of observations. Thus a larger sample would be needed to verify the model. Only two variables were significant (one each in the chimpanzee and orangutan) and the same variables are not highlighted as significant for each species. Thus, as with the ribs, it is possible that differences among the three ape species reflect true morphological differences among the species. However, given the small sample and limited number of variables that were significant, this may or may not be true. It is also possible that the apes do not show the significant variation seen in the auricular surface of older (50+) humans. Further, individual variation may play a greater role in the appearance of the aging markers, particularly as there were instances in which identical trait scores were found in both young and old individuals. Further investigation into potential species' differences may be warranted; however, the small samples negate this possibility at present.

Acetabulum. The development of aging techniques utilizing features of the human acetabulum are more recent, and perhaps less well-known, than techniques developed for the ribs and auricular surface. Age-related changes on the acetabulum show similarity to changes of the auricular surface in that during senescence, both

demonstrate porosity, osteophytic formation, and characteristics compatible with degenerative osteoarthritis (Rissech et al., 2006). The use of the acetabulum to determine age has, however, been criticized. One critique is that hip dysplasia, resulting from localized overloading and leading to localized cartilage attrition, might cause a bias in age estimation (Rougé-Maillart et al., 2004). It is also possible that morphological changes related to osteoarthritis could bias results. This raises the question of how it is determined whether the morphological changes seen are due to age, osteoarthritis, or age *and* osteoarthritis. Given that age and osteoarthritis show strong correlations, use of the acetabulum, a portion of the hip joint known to exhibit OA, might introduce uncertainty into any age-related analysis.

Two studies have utilized adult male skeletons to test the usefulness of the acetabulum as a means to estimate age at death. Rougé-Maillart et al., (2004) in a preliminary study of 30 male coxal bones, ranging in age from 24-81 years old, used four criteria as follows: (1) appearance of the rim; (2) appearance of the fossa; (3) lunate surface porosity and (4) apical activity. For each criterion, stages were developed, with the rim appearance classified into five stages, the fossa into four stages, porosity into three stages, and apical activity into three stages. The rim and fossa criteria produced a significant link between the various stages and age, while apical activity was said to be “noteworthy.” The lunate porosity criterion did not produce satisfactory results. Results did indicate a progressive trend with stage and age, but with overlap between the age groups and variation within each group. “Total porosity” results were significant.

In a study with a much larger sample size, Rissech et al., (2006) examined 242 male left os coxa ranging in age from 16 to 96 years old. Seven variables were utilized:

(1) acetabular groove, (2) rim shape, (3) rim porosity, (4) apex activity, (5) outer edge activity, (6) fossa activity, and (7) fossa porosity. Each of the variables was structured as a series of states. See Rissech et al., (2006) for detailed descriptions of the 7 variables and 41 states. The age ranges of the states for each variable covered ages from young to old with slight overlap. Results showed that the difference between known age and estimated age was within 10 years for more than 89.0% of the specimens. Approximately 67.0% were estimated within 5 years and 35.0% within 2 years of known age of death.

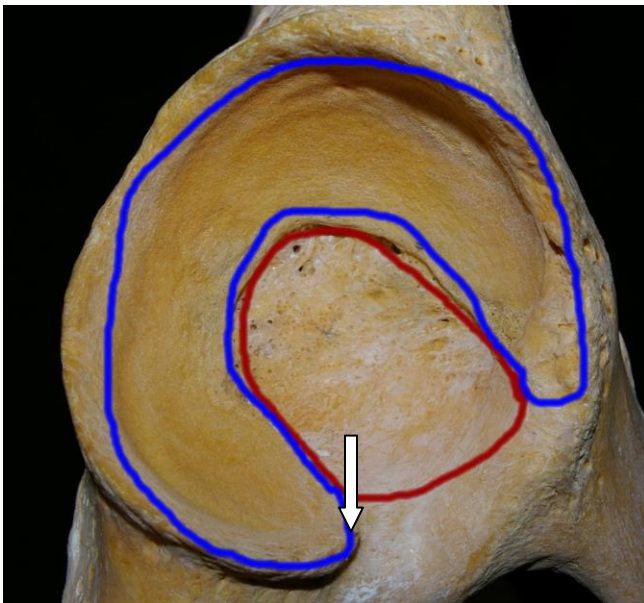
In general, with age, the acetabular rim progresses from blunt and rounded to form a sharp ridge that becomes crested due to osteophytic growth. Eventually, an extremely high crest can form and the rim may destructure, meaning that there is generalized bone loss around the entire rim. The apex starts out round and smooth then becomes sharp. Later, it develops a projection or spicule that can become quite elongated and large in size. The young acetabular fossa is dense and relatively smooth. With age, it develops perforations (smaller then larger) followed by exposure of trabecular bone. In addition, sclerotic bone may form thereby obscuring the fossa. The young lunate surface is smooth and level with the fossa. With age, the fossa becomes defined and appears to recede becoming deeper than the lunate surface. Activity, usually porosity followed by osteophyte production, can form on both the lunate and fossa surface. This activity can eventually obliterate the fossa and cover much of the lunate surface.

Methods. Based on morphological changes noted in human adult skeletons, the following characters were scored for both the left and right acetabulum of the ape skeletons. Rim – blunt/round; sharp; sharp and lipping; destruction. Fossa – dense < 1/3; dense 1/2-2/3; dense > 2/3; trabecular fully exposed; sclerosis. Lunate surface – defect

absent/present; microporosity absent/present; macroporosity absent/present; sclerosis.

Apex – rounded/sloped; sharp/angled; projection; destruction; fusion. Inner Fossa – none; barely discernible; clearly defined; destruction. Figure 18 below shows the features of the acetabulum, while table 14 below gives details of each variable along with descriptions, codes, and photographs of each state.

Figure 18: Features of the Acetabulum



<u>Features of the Acetabulum</u> Blue line – lunate surface Red line – fossa White Arrow - Apex

Table 14: Morphological Descriptions of the Acetabulum Aging Variables.

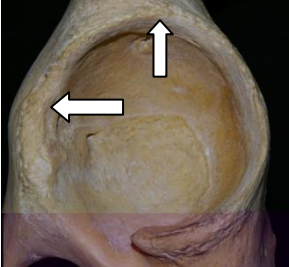
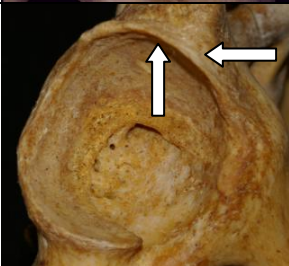
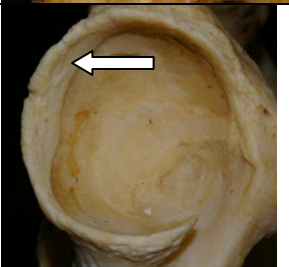
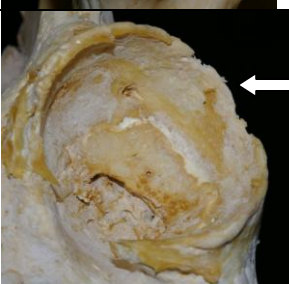
Variable	States	Code	Description	Photograph of States
Rim	Blunt/rounded	1	The acetabular rim is round and smooth along the entire length. This is a rim of young appearance.	
	Sharp	2	The acetabular rim loses the rounded, smooth appearance becoming narrower and sharp. Portions of the rim may still be rounded/blunt.	
	Sharp with lipping	3	The acetabular rim is sharp with osteophytes (lipping) around some or all of the rim. There is usually an obvious anatomical interruption between the lunate surface and the acetabular rim where osteophytic growth has occurred.	
	Destruction	4	The acetabular rim becomes irregular in shape with a somewhat jagged appearance. Osteophytes may still be present, but the rim takes on an uneven appearance where the bone has become destructured (loss of bone).	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...


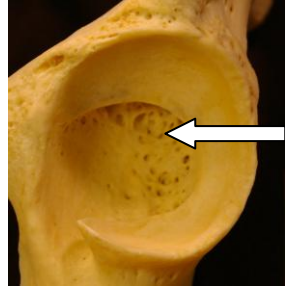

Fossa	Density < 1/3	1	The fossa is dense in appearance with perforations of the subchondral bone of the surface. These perforations cover < 1/3 of the fossa surface. In general, the perforations are small in size.	
	Density 1/3-2/3	2	The fossa is dense with perforations of the subchondral bone of the surface. These perforations cover between 1/3-2/3 of the fossa surface and become larger in size.	
	Density > 2/3	3	The fossa is dense with perforations of the subchondral bone of the surface. These perforations cover > 2/3 of the surface. The perforations may be quite large in size.	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...

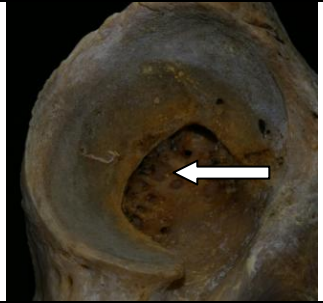
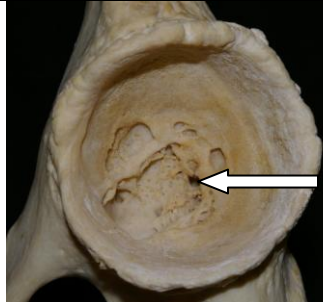

	Trabecular	4	The fossa is dense with exposure of trabecular bone. These perforations are very large in size.	
	Sclerosis	5	The fossa has sclerotic bone covering all or part of the surface.	
Lunate	Defect absent	1	The lunate surface is smooth and regular. This is a lunate of young appearance.	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...

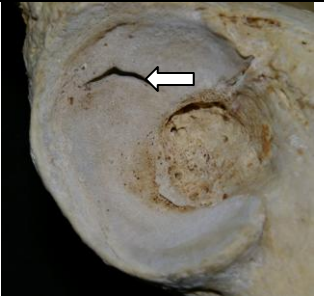


Lunate	Defect present	2	The lunate surface has one or more fissures (defect) present.	
	Microporosity and/or macroporosity absent	1	The lunate surface is smooth and regular without porosity.	
	Microporosity present	2	The lunate surface has one or more small pin-prick sized porous holes.	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...

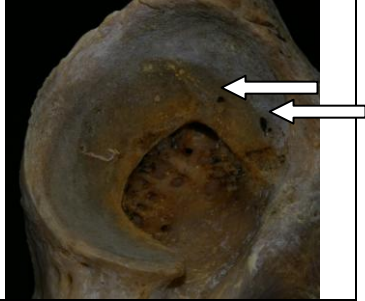
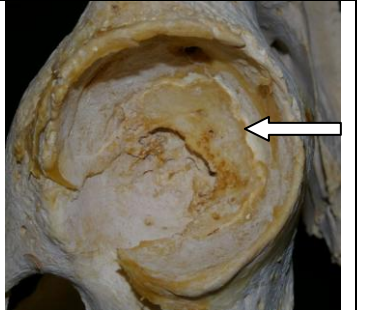
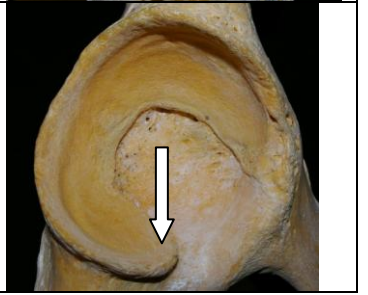
	Macroporosity present	2	The lunate surface has one or more large porous holes. Microporosity is often also present.	
	Sclerosis	3	The lunate surface is rough with areas of sclerotic bone covering either all or part of the surface.	
Apex	Rounded	1	The apex is rounded and blunt in appearance and feels smooth to the touch	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...

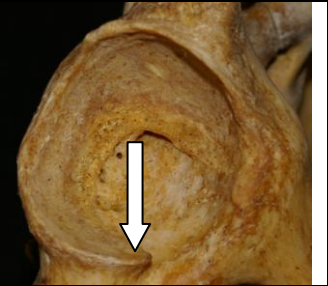
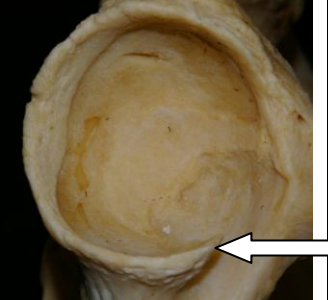

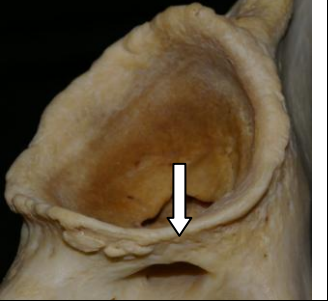
Apex	Sharp	2	The apex has a sharp edge and is more angled in appearance	
	Projection	3	The apex has developed a conspicuous osteophyte or spicule. This spicule can form a ledge or projection, which eventually becomes quite elongated.	
	Destruction	4	The apex has become destructured, thus giving the appearance of being missing with a 'bumpy' appearance	
	Fusion	5	The apex osteophyte has elongated to the point that fusion has occurred with the anterior horn	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...


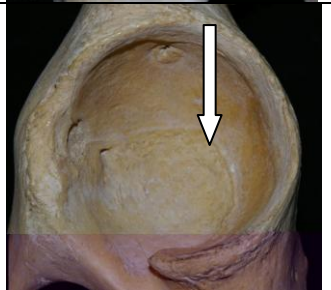
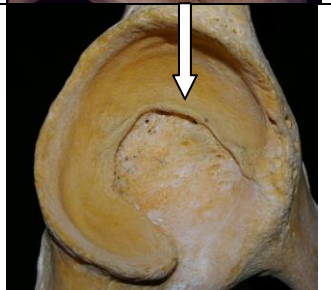
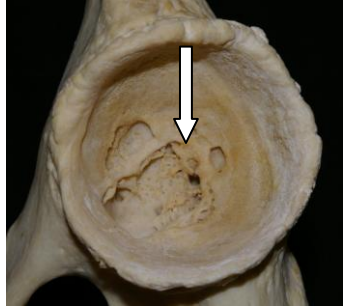
Inner Fossa	None	1	The area between the lunate surface and fossa is dense, smooth and appears even.	
	Barely discernible	2	The area between the lunate surface and fossa is dense with a thin, barely discernible line of separation. The fossa is beginning to recede.	
	Clearly defined	3	The area between the lunate surface and fossa is clearly defined. The fossa recedes and appears to be deeper than the lunate surface.	

Table 14: Morphological Descriptions of the Acetabulum Aging Variables continued...

	Destruction	4	The area between the lunate surface and fossa has sclerotic bone which obstructs the fossa surface and often obliterates any definition between the lunate surface and fossa. The sclerotic growth gives an uneven, rough appearance.	
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Results. For all three species, linear regression utilizing the stepwise method was performed on all known-aged specimens. As a reminder, there were 31 known-aged chimpanzees, 22 known-aged gorillas, and 17 known-aged orangutans. The result for each species is as follows:

Chimpanzee: There were 26 chimpanzees included in the analyses. Of the 12 variables examined (6 on each of the left and right sides), 2 were constants or there were too few complete cases to estimate the correlation (left and right inner fossa). Of the remaining 10 variables, only 2, right rim (AceRR) (Pearson's correlation = .616, $p=.000$) and right fossa (AceRF) (Pearson's correlation = .359, $p=.036$) were included in the final model. This final model had an R^2 of .561 and was significant at $p=.000$. The regression equation for this model, with unstandardized coefficients, is: $y = 8.061(\text{AceRR}) + 6.586(\text{AceRF})$. The plot of the standardized residuals against standardized predicted values shown in figure 19 below indicates a random distribution, which is indicative of a situation in which the assumptions of linearity and homoscedasticity have been met. Further, a histogram of the residuals shown in figure 20 below demonstrates a near normal distribution, and a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant. Partial regression plots for the right rim and right fossa are shown in figures 21 and 22 below. These scatterplots show some evidence of a linear relationship, although outliers are evident.

Figure 19: Chimpanzee Acetabulum - Standardized Residuals plotted against Standardized Predicted Values

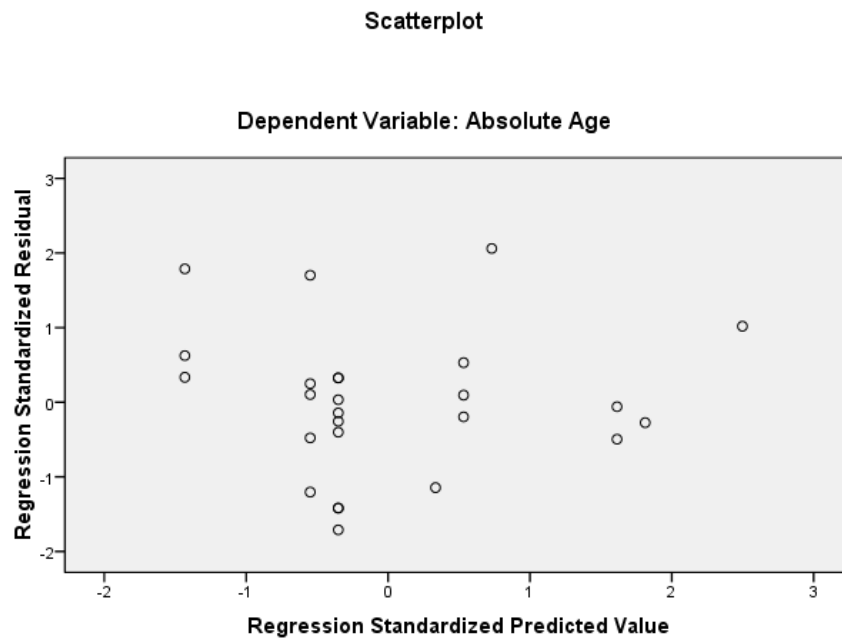


Figure 20: Chimpanzee Acetabulum – Histogram of Residuals

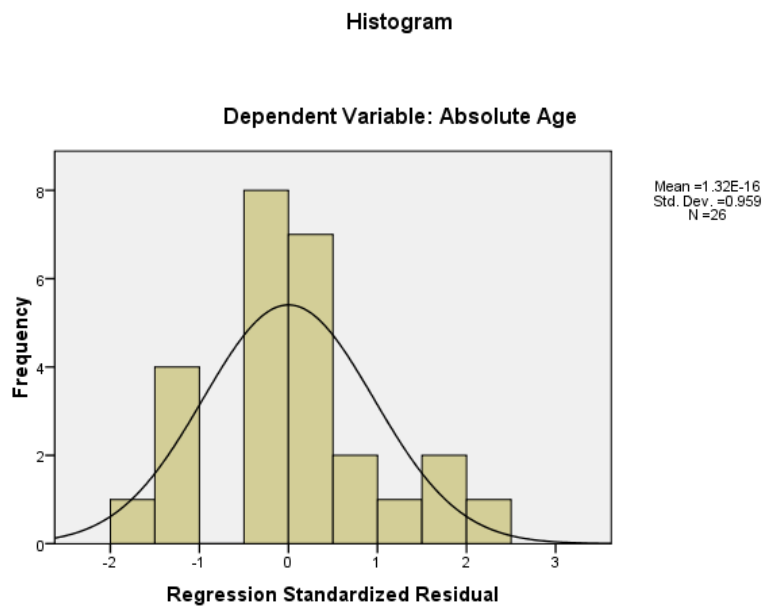


Figure 21: Chimpanzee Acetabulum – Partial Regression Plot of Acetabulum Right Rim



Figure 22: Chimpanzee Acetabulum – Partial Regression Plot of Acetabulum Right Fossa



Gorilla: There were 16 gorillas included in the analyses. All 12 variables were included in the model (6 on each of the left and right sides). Of these 12 variables, only 1 (right lunar porosity (AceRLP) (Pearson's correlation .600, $p=.007$) was included in the final model. This final model had an R^2 of .361 and was significant at $p=.014$. The regression equation for this model, with unstandardized coefficients, is:

$$y = 4.573(\text{AceRLP}).$$

The non-random nature of the plot of the standardized residuals against standardized predicted values shown in figure 23 appears to indicate a case of heteroscedasticity (non-constant variance), where at each point along any predictor variable the residuals are different; however, this interpretation should be considered tentative due to the relatively small number of observations. A histogram of the residuals shown in figure 24 below demonstrates evidence of non-normal distribution; although, a K-S normality test of the standardized residuals ($p=.053$) (lower bound)) indicates that these deviations were not significant.

Figure 23: Gorilla Acetabulum - Standardized Residuals plotted against Standardized Predicted Values

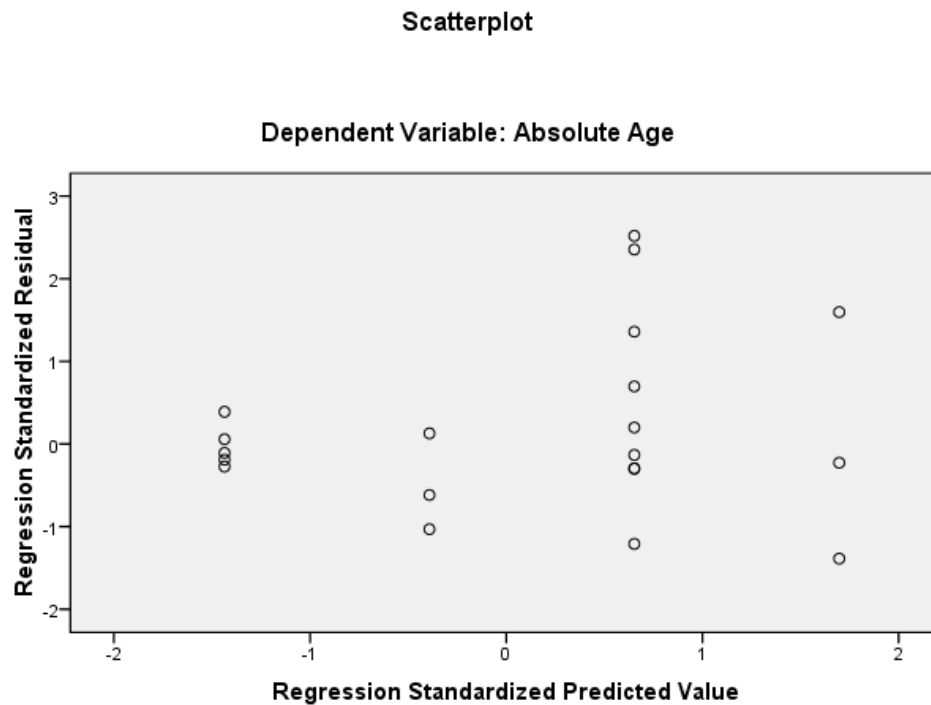
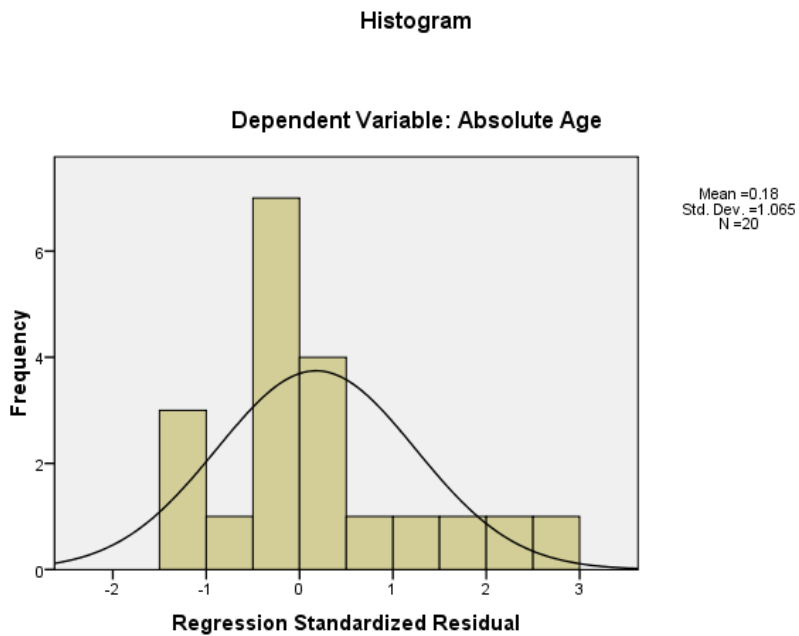


Figure 24: Gorilla Acetabulum – Histogram of Residuals



Orangutan: There were 14 orangutans included in the analyses. All 12 variables were included in the model (6 on each of the left and right sides). Of these 12 variables, only 1 (left rim (AceLR)) (Pearson's correlation .649, $p=.006$) was included in the final model. This final model had an R^2 of .421 and was significant at $p=.012$. The regression equation for this model, with unstandardized coefficients, is: $y = 7.597(\text{AceLR})$. The plot of the standardized residuals against standardized predicted values shown in figure 25 below demonstrates evidence of non-linearity and non-constant variance; however, this interpretation should be considered tentative due to the small number of observations. A histogram of the residuals shown in figure 26 below demonstrates evidence of non-normal distribution; although, a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant.

Figure 25: Orangutan Acetabulum - Standardized Residuals plotted against Standardized Predicted Values

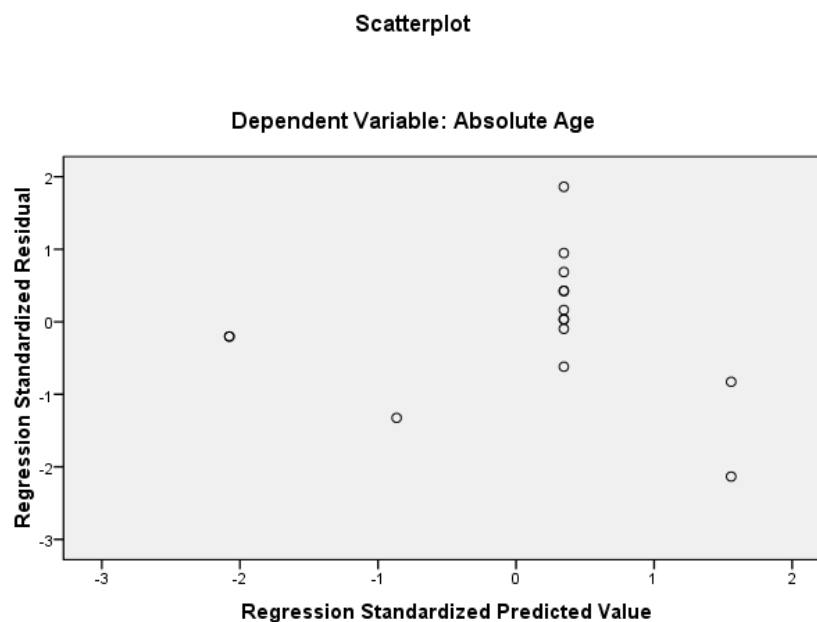
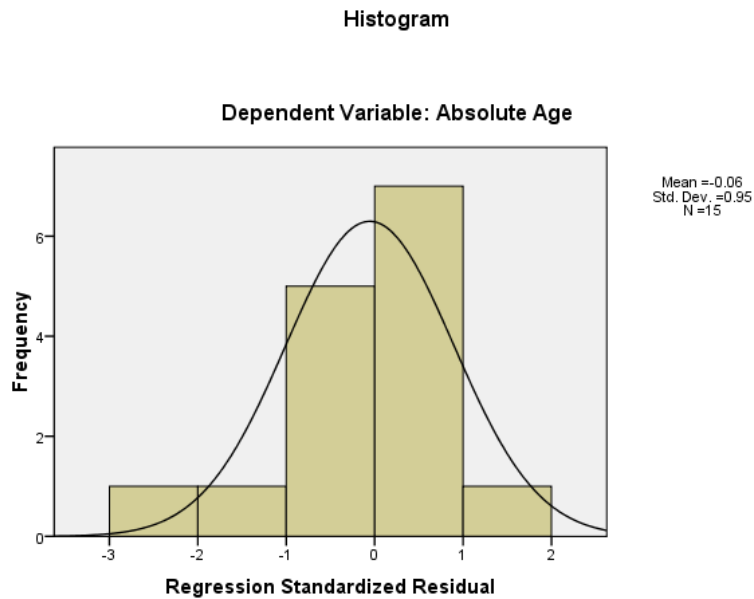


Figure 26: Orangutan Acetabulum – Histogram of Residuals



Summary: Overall, of the post-cranial skeletal markers analyzed, the acetabulum appears to be the most useful aging marker. Unlike the ribs and auricular surface, the acetabulum is significant in all three species, although the R^2 is low in the gorilla and orangutan. Both the gorilla and orangutan models demonstrate some evidence of non-linearity and non-normally distributed data; however, this interpretation should be considered tentative due to the small number of observations. K-S normality tests of the standardized residuals were not significant, indicating that potential problems are most likely due to the small samples. This is because distributions can look very non-normal in small samples even when they are normal (Field, 2005). It is also apparent that different variables are being highlighted as significant in each species. Thus, as with the ribs and auricular surface, it is possible that differences among the three ape species reflect true morphological differences among the species. However, given the small

sample and limited number of variables that were significant, this may or may not be true.

One possibility is that a reduction in the number of variables entered into a model may produce results that indicate a general equation or set of recommendations that is applicable to all three species; however, manipulating the data to a great extent also has the potential of producing a model that may not be generalizable. Nevertheless, to test the possibility of an ‘improved’ model, linear regression was run on 6 of the 12 variables. The variables selected were those that were highlighted as having potential relevancy to all three species. The results show that, for all three species, there was an increase in the R^2 value (Chimpanzees - $R^2=.582$; Gorillas - $R^2=.622$; Orangutans - $R^2 .481$) and the model was significant for chimpanzees and gorillas ($p=.006/p=.037$ respectively), but not in orangutans ($p=.238$). Thus, it appears likely that a model that is generalizable to all three species is possible; however, more data would be needed to verify this assumption.

While the acetabulum demonstrates potential for use as an aging indicator, one additional concern with using this skeletal feature to estimate age is that it has not been clearly established how much influence arthritic-related changes have and how they might influence the results. This is because the hip is one of the four major joints that is susceptible to osteoarthritis and the various features of the acetabulum are thus subject to both ‘ordinary’ senescent and arthritic-induced change. The issue is further complicated given the established relationship between OA and age. Thus, overall, using the acetabulum as the sole marker for age is inadvisable at present. However, further research into the utility of the acetabulum as an aging marker is warranted. Meanwhile,

using the acetabulum in conjunction with another aging marker may prove to be of value and will be discussed in more detail later in this chapter.

Dentition: Teeth are the elements most frequently found in the fossil record and the best-documented morphological adaptations to diet are found in the teeth (Fleagle, 1999). Dental features also are important in studies of health, disease, and genetic affiliation (Buikstra and Ubelaker, 1994). Teeth have proven particularly useful in aging of human immature remains as the sequence of formation and eruption of the teeth follow relatively uniform trajectories. Dental wear also has proven a major focus of numerous adult human age-related studies (eg: Molnar, 1971a and b; Scott, 1979; Tomenchuk and Mayhall, 1979; Lovejoy, 1985; Drusini, 1991; Cameriere et al., 2004; and Oliveira et al., 2006).

In non-human primates, eruption and decay of the permanent teeth was studied extensively by Schultz (1935) on *Pan*, *Gorilla*, *Pongo*, and *Hylobates* (gibbons), plus six species of Old World monkeys and five species of New World monkeys. More recent studies (Nissen and Riesen, 1964; Conroy and Mahoney, 1991) also examined emergence patterns in the permanent dentition of chimpanzees, while features of the dentition (such as attrition) have factored strongly in numerous age-related studies of the apes (eg: Schultz, 1940, 1941, 1950, 1969; Randall, nd; Kilgore, 1989; Lovell, 1990; Zihlman et al., 1990; Morbeck et al., 2002; and Nichols and Zihlman, 2002). Likewise, several studies have examined patterns of dental wear in some species of monkeys (eg: Gant, 1979; Dennis et al., 2004), while Bramblett (1969) developed an attrition stage system for aging the Darajani baboon.

Several techniques have been developed for age assessment on humans and other species based on utilization of features of the dentition. Some of these techniques, and the reasons why they were not chosen for this research, will be discussed here.

Lamendin Method: Lamendin et al., (1992) proposed a method for aging human adults based on root translucency and periodontal regression of single rooted teeth. The system is based on measurements (periodontosis height times 100/root height (P) and transparency of the root height times 100/root height (T)) taken on the labial surface of the entire tooth (Lamendin et al., 1992). It requires three measurements: total root length, gingival regression, and root translucency. Root translucency is best observed when the tooth is backlit, and is generally seen after age 20, with age estimates based on this translucency being most effective in adults over age 30 (Megyesi et al., 2006).

Lamendin et al., (1992) found the method to be “reasonably accurate” except for individuals under the age of 40 where other methods would be preferred. In a test of the Lamendin method on two historic skeletal samples, it was found that postmortem factors affect the applicability of the technique to archaeological and historical samples. Results of the study suggest that root translucency disappears over time or is obscured by taphonomic effects, and this, combined with overall tooth condition, was identified as having a significant effect on the traits used in the method and on the age estimates (Megyesi et al., 2006). While the method is not destructive in the sense that it damages or destroys a tooth, it does require extraction of one or more teeth, which can be considered a negative event. Further, the method is not found to be accurate until after the age of 30 and has questionable utility in archaeological or historical samples. Thus,

for these reasons as well as the necessity for tooth extraction, the method was not considered as appropriate for this research.

Cementum Annulus Counts: Cementum annulus counts of teeth have proven a reliable aging method in many mammalian species including bears, wolves, and sheep, among others (Craighead et al., 1970; Turner, 1977; Goodwin and Ballard, 1985). The basis for cementum aging is the cyclic nature of cementum growth, which results in an annual (ring-like) pattern formed in the tooth (like that formed in the wood of trees). Dark rings form during the winter, with lightly staining cementum forming in spring and summer. Some variation is seen, with animals in southern regions of North America displaying less distinct annuli than their counterparts in northern regions. Human teeth have similar annuli, but the pattern of deposition is irregular when compared to most wild mammals. The method, while successful for aging many mammal species, requires utilizing a specialized lab to perform the analysis and is destructive in that it requires tooth extraction with subsequent histological damage to the tooth caused by exposure to excessive heat and/or chemical agents. Because of the irregularity of annuli patterns seen in human teeth, cementum annulus counts would likely prove to be ineffective for aging humans. However, labs generally have no experience in the aging of human teeth (Matson's Lab, [online] accessed 2007). It is not currently known whether the system would be applicable to any of the ape species. It is possible that apes, like humans, would deposit cementum annuli in an irregular pattern. It is also possible that cementum deposition would be influenced by geographical and seasonal variation that would likely skew or influence the results. In addition, artificial seasonal and/or lighting conditions often found in captivity could also influence cementum deposition. However, studies of

macaque monkeys from Cayo Santiago, that utilized both tooth wear and cementum annuli on lower first molars, found that both measurements were significantly correlated with age. The authors found that neither measurement by itself was strongly enough correlated with age to provide a reliable guide to the true age of monkeys older than about 14 years of age; although, cementum annuli counts were found to provide a more reliable guide to age determination than was dental wear. However, a combination of tooth wear and annulus counts was found to be a better predictor of age explaining 79% of overall variance in age (Kay et al., 1984; Kay and Cant, 1988). Given the destructive nature of this technique and the, as yet, unknown applicability of the method to ape teeth, the method was not chosen for general use in this study. Nevertheless, given the results of the macaque study, a test of the system for ape teeth is warranted. The Museum of Comparative Zoology at Harvard University (which utilizes labs for annulus counts on myriad mammal species) generously agreed to test the method on a small sample of their ape teeth. Three gorilla teeth were tested, one with known age. It was found that while annuli are present in the gorilla teeth examined these annuli are less distinct than in some other species and occur in an irregular pattern. Further, the tooth from a known-aged, 38-year-old, gorilla generated an age estimate of 16-18 years (Chupasko, personal communication). Thus, results from these preliminary tests indicate that cementum annulus counts are likely to be unreliable for aging great ape skeletons.

Dental Wear: The occlusal surfaces of teeth wear in accordance with daily food preparation and mastication. Studies of dental attrition in human populations are abundant and have been ongoing since at least the 1920s. Some authors, such as Molnar (1971), Scott (1979), and Brothwell (1981) have developed attrition scoring techniques

that are widely utilized. These techniques are based on the direction of the occlusal surface as well as the surface form itself. Molnar (1971a) developed an ordinal scaling system for human dentition ranging from unworn (category 1) to roots functioning as occlusal surface (category 8). In general, teeth begin in a pristine, unworn state, progressing to obliteration of cusp pattern, followed by appearance of dentine patches. These dentine patches become larger, progressing from primary to secondary dentine exposure that start in small isolated patches and then coalesce to cover the entire tooth's surface. Eventually, only roots function as the occlusal surface, followed by complete bony resorption. Scott (1979), in a system designed to supplement Molnar's procedure, proposed a 0-10 scale for molar wear where the molars are divided into quadrants (see Scott, 1979, for detailed descriptions of each stage). Scott suggested that the quadrant-based system allowed for the possibility of more accurate estimations of the amount of enamel present on tooth surfaces than do whole tooth systems, but also concluded that what system a researcher chooses is dependent upon the objectives of the research (Scott, 1979).

The accuracy and utility of using dental wear as an aging indicator is not universally accepted. Diet and sex are often factors that have also been cited as influential in dental wear and both have been studied, although results are often conflicting. In humans, a higher degree of attrition was found among California Indian females (Molnar, 1971) while in Igloolik Eskimos, male molars wear more rapidly than female molars (Tomenchuk and Mayhall, 1979). However, not all populations display differences in wear between the sexes as was found by Lunt (1978) in a Danish population. It has also been suggested that differences in wear can be attributed to

differential bruxism, socioeconomic status, and non-dietary uses of the dentition (such as food preparation techniques and tool usage) (Molnar, 1971; Lovejoy, 1985; Tomenchuk and Mayhall, 1979). While the degree and kinds of tooth wear in humans may vary from population to population, evidence suggests that dental wear is, in fact, useful for estimating age. In a prehistoric Brazilian sample, age classification by occlusal molar wear was found to be a “useful tool” for estimating age with a discrepancy of less than 8.22 years between upper and lower limits (Oliveira et al., 2006). Lovejoy (1985) found that dental wear can be a “highly accurate indicator of age at death” in entire skeletal samples. Indeed, Lovejoy et al., (1985), in a multifactorial method testing five aging indicators (pubic symphysis, auricular surface, radiographs of proximal femur, dental wear, and suture closure), found that dental wear is “*the best single indicator for determining age at death in skeletal populations*” (emphasis added). While the overall results of the study indicated that the multifactorial method was superior to results obtained only by use of single indicators, dental wear estimates were consistently high in accuracy and “virtually without significant bias.” Thus, the evidence would seem to suggest that, even given potential population differences in wear rates, dental wear is an important tool for use in age determination.

While some studies have not utilized dental wear for estimating age in ape skeletons (eg: Lovell, 1990), other studies have focused on dental pathology and dental wear in relation to aging studies. A study of mountain gorilla dentition determined that enamel wear was present to some degree in all individuals (Lovell, 1990). Likewise, a study of the Gombe chimpanzee skeletons found that there was an “obvious relationship” between wear and age with enamel wear being present to some extent in all individuals

(Kilgore, 1989). A more recent study of the Gombe chimpanzee skeletons also found that missing teeth and extensive wear characterized older individuals, with severe wear and caries being common in those individuals over 33 years of age (Morbeck et al., 2002).

It is not known with certainty whether captive and wild ape teeth vary significantly in wear rates or patterns. Evidence certainly suggests that the pattern of wear is similar in wild and captive animals, although severity of wear might be less in captive animals (Nichols and Zihlman, 2002). For example, in a study of captive gorillas (albeit a very small sample of $n=5$), it was found that the pattern of enamel attrition is similar to that of wild gorillas but with a lessened degree of severity (Nichols and Zihlman, 2002). Differences in severity can likely be attributed to dietary constraints as well as dental intervention for captive animals. Nevertheless, how much variation there is in the mechanical and nutrient properties of captive and wild foods is unclear. Indeed, while captive and wild specimens might vary in wear severity, there also is the potential for differences to be found among distinct wild populations as well as in distinct captive populations. Thus, while dental wear might not prove to be an ideal method for determining age, given the current problems with aging of adult ape skeletons, it should certainly be considered a viable resource.

Methods: Based on dental wear methods developed for both humans and the African apes, dental wear stages were developed for analyzing all teeth. In particular, Kilgore's system (1989), which was based on Molnar's system but developed specifically for the African apes, was the main reference used to develop the dental wear technique utilized for this study. Kilgore's system utilized a modified version of Molnar's system

taking into account premolar and molar cusp patterns, heavy staining on the teeth, and extensive enamel attrition along the lingual surface of incisors and canines and along the distal edges of lower canines (Kilgore, 1989). Orangutans are generally considered to have thicker enamel and bunadont (low-cusped) teeth when compared to the African apes (and thus more like human teeth); however, recent research using 3-D whole crown enamel distribution (Kono, 2004) found that while humans had relatively thick enamel throughout the crown and gorillas had relatively thin enamel throughout the crown, chimpanzees and orangutans were intermediate in enamel thickness. Thus, any differences in orangutan molar thickness may not be as pronounced as is commonly thought. It was also evident that orangutan incisors and canines have a shape and wear pattern more consistent with the African apes. Because of these similarities, the fact that Kilgore's system is very similar to Molnar's system, and for purposes of consistency, only one scoring technique was utilized for this study. Table 15 below shows details of the dental wear stages for each type of tooth. Table 16 below provides photographs for additional reference. These dental wear stages were utilized to analyze all dentition present in the sample.

Although wear data were collected on all dentition, only the molars were analyzed for this research. This is because the molars are the most likely type of tooth to demonstrate age-related wear that is not as heavily influenced by cultural factors. In humans, unusual wear patterns have been attributed to activities that involve holding or pulling fibrous material with the teeth (Molnar, 1971). The incisors and canines are those teeth most often used in such activities, while the primary action of the molars is the mastication of food. All three species of apes are also expected to demonstrate wear

related to similar activities as their incisors and canines are often used in food preparation in such endeavors as stripping bark and piercing tough fruits. Stems and twigs are routinely stripped by pulling between the teeth and increased wear has been found in the anterior teeth of chimpanzees that is most likely a direct result of this action (Kilgore, 1989). In addition, while the molars are responsible for the bulk of chewing actions, it is plausible that wear would be more consistent on the 1st and 2nd molars than on the third. This is because, as a general rule, it becomes harder to balance the muscle force on both sides of the mandible to keep the TMJ from experiencing tension the further back on the tooth row an individual chews. Thus, analyzing the effectiveness of a model that uses all three molars to estimate age compared to a model that uses only the first and second molars will be undertaken.

Table 15: Ape Dental Wear Stage Description



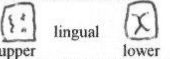
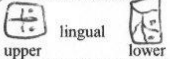


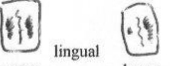
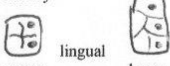


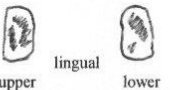
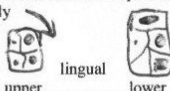


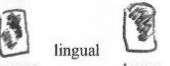
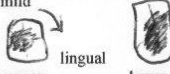


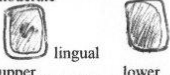
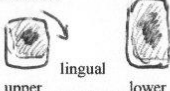



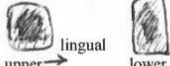








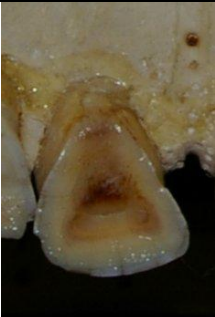




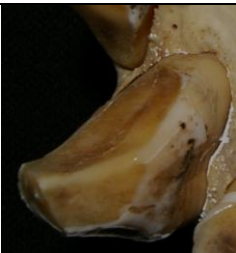




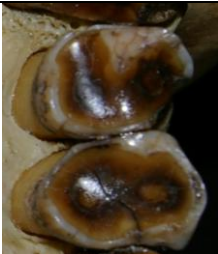





Stage	Incisors	Canines	Pre-Molars	Molars
0	Missing Unobservable Unworn	Missing Unobservable Unworn	Missing Unobservable Unworn	Missing Unobservable Unworn
1	Occlusal and lingual wear; small line of dentin on occlusal surface; occlusal tip no longer 'bumpy'  lingual	Wear facets at tip, distal, and lingual surfaces 	Facets on cusp surface, first appear lingually on uppers and buccally on lowers; no observable dentin  buccal upper lingual lower buccal	Facets on tips of cusps, first appear lingually on uppers and buccally on lowers; no observable dentin  buccal upper lingual lower buccal
2	Marked wear facet on median ridge; staining of ridge, esp. at base; dentin patch at occlusal tip; flat surface 	Facets marked on distal and lingual surface; small dentin patches at tip; small streak of dentin on distal surface (esp lowers); more conical appearance  distal	Small dentin patches on cusps, cusp pattern partially or completely obliterated  buccal upper lingual lower buccal	Small dentin patches on tips of cusps; appear first in uppers lingually and in lowers buccally  buccal upper lingual lower buccal
3	Wide dentin patch on tip beginning to extend lingually; may be dentin at base of ridge; polished appearance; tips becoming beveled; small patches of secondary dentin 	Distal notch with dentin exposure; dentin on distal and lingual surface; dentin on tip; secondary dentin absent-minor 	Upper – lingual dentin patch well established; patches separated by ridge Lower – dentin patch may be on entire distal surface  buccal upper lingual lower buccal	Upper – lingual dentin patches large and merging, but distinct from buccal separated by ridge; occlusal surface slopes bucco-lingually Lowers – buccal dentin patches larger or equal in size to lingual but separated by ridge; occlusal surface slopes lingually-buccally  buccal upper lingual lower buccal
4	Dentin exposure covers at least half of lingual surface; enamel rim present at each side; beveled appearance to tips; secondary dentin moderate 	Distal notch well established; dentin covers more than ¼ lingual and distal surface and tip; secondary dentin minimum-moderate 	Dentin patches on buccal and lingual surfaces merging; cusps obliterated; crown becoming blunt and flat; secondary dentin mild  buccal upper lingual lower buccal	Buccal and lingual patches merging or mostly merged; slope of crown present but becomes less obvious; secondary dentin mild  buccal upper lingual lower buccal
5	Dentin exposure on entire lingual surface; erosion of crown to enamel-cement junction; secondary dentine more extensive; narrow enamel ridge may still be intact; crown slopes from tip to base; lingual surface may be concave 	Dentin on entire lingual surface; lingually crown is worn to enamel-cement junction; distal notch becoming obliterated; pulp cavity becoming visible; secondary dentine moderate-extensive 	Exposed dentin on entire surface; crown sloped or flat; cusps now obliterated; lower crown may be worn to enamel-cement junction lingually; secondary dentin moderate  buccal upper lingual lower buccal	Occlusal surface flat; dentin exposed throughout; upper slopes lingually to root; crown mostly obliterated; secondary dentin moderate  buccal upper lingual lower buccal
6	Exposure of pulp cavity; Peg-like appearance 	Pulp cavity exposed; peg-like appearance 	Complete dentin exposure; secondary dentin extensive; crown (enamel) completely absent on at least one side 	Crown (enamel) completely absent on at least 1 side; secondary dentin extensive  buccal upper lingual lower buccal
7	Root only in socket	Root only in socket	Root only in socket; Pulp cavity exposed	Root only; pulp cavity exposed; portions of two roots remaining but no longer joined by crown
8	Total resorption	Total resorption	Total resorption	Total resorption

Table 16: Ape Dental Wear Stage Photographs

	Incisors	Canines	Pre-Molars	Molars
0	unworn	Unworn	unworn	unworn
1				
2				
3				
4				
5				
6				
7	Roots only	Roots only	Roots only	Roots only
8	Total resorption	Total resorption	Total resorption	Total resorption

Results: Linear Regression was performed on all known-aged specimens. As mentioned earlier, there were 31 known-aged chimpanzees, 22 known-aged gorillas, and 17 known-aged orangutans. The regression models were run to include all molars (12 variables: 3 molars in each quadrant) and then for molars 1 and 2 (8 variables: 2 molars in each quadrant). The second analysis was performed due to problems with the data for orangutans and gorillas as explained below. The results for each model and each species are as follows:

Chimpanzees: All molars: There were 26 known-aged individuals included in the analysis. The model displayed a good fit with an R^2 of .783, and was significant at $p=.011$. Table 17 shows the Pearson's correlations (bivariate correlations measuring the strength of the relationship between each marker and absolute age), and all except the left upper molar 1 (LUM1) and right lower molar 3 (RLM3) are significant. The regression equation for this model, with unstandardized coefficients, is:

$$y = -.471(\text{LUM1}) + 1.314(\text{LUM2}) + .916(\text{LUM3}) + -17.137(\text{LLM1}) + 6.858(\text{LLM2}) + -1.077(\text{LLM3}) + 4.362(\text{RUM1}) + 2.406(\text{RUM2}) + 4.746(\text{RUM3}) + 13.444(\text{RLM1}) + 2.035(\text{RLM2}) + -2.993(\text{RLM3})$$

The plot of the standardized residuals against standardized predicted values shown in figure 27 below indicates a random distribution, which is indicative of a situation in which the assumptions of linearity and homoscedasticity have been met. Further, a histogram of the residuals shown in figure 28 below demonstrates a near normal distribution and a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that any deviations were not significant. Partial regression plots for the molars are shown below in figures 29-40. In general, these scatterplots show some evidence of a linear relationship, although outliers are evident. Only the lower left molar

1 was individually significant at $p=.008$, while the right lower molar 1 was near significant at $p=.059$. Nevertheless, given the extremely high R^2 value and significance of the ANOVA, the indications are that all of the molars taken together as a composite are strong predictors of age.

Table 17: Chimpanzee Molar Linear Regression – Pearson’s Correlations

		Value	Sig (1-tailed)
Pearson Correlation	Absolute Age		
	LUM1	.284	.079
	LUM2	.526	.003
	LUM3	.398	.022
	LLM1	.481	.006
	LLM2	.648	.000
	LLM3	.438	.013
	RUM1	.595	.001
	RUM2	.617	.000
	RUM3	.521	.003
	RLM1	.580	.001
	RLM2	.599	.001
	RLM3	.292	.074

Figure 27: Chimpanzee Molar Wear - Standardized Residuals plotted against Standardized Predicted Values



Figure 28: Chimpanzee Molar Wear – Histogram of Residuals

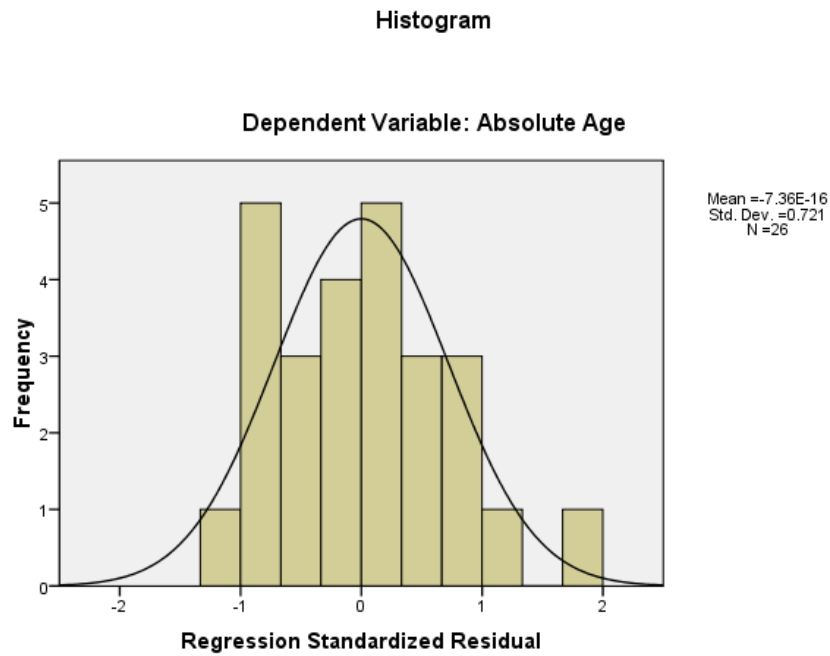


Figure 29: Chimpanzee Molar Wear – Partial Regression Plot Left Upper Molar 1

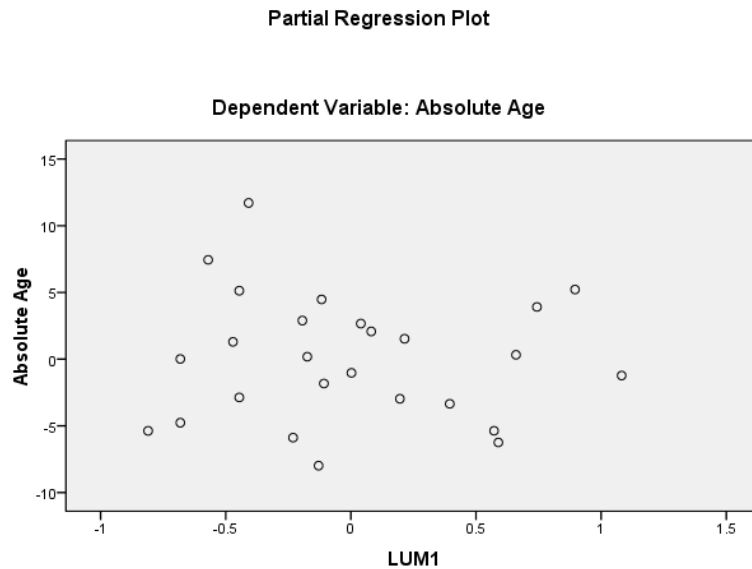


Figure 30: Chimpanzee Molar Wear – Partial Regression Plot Left Upper Molar 2

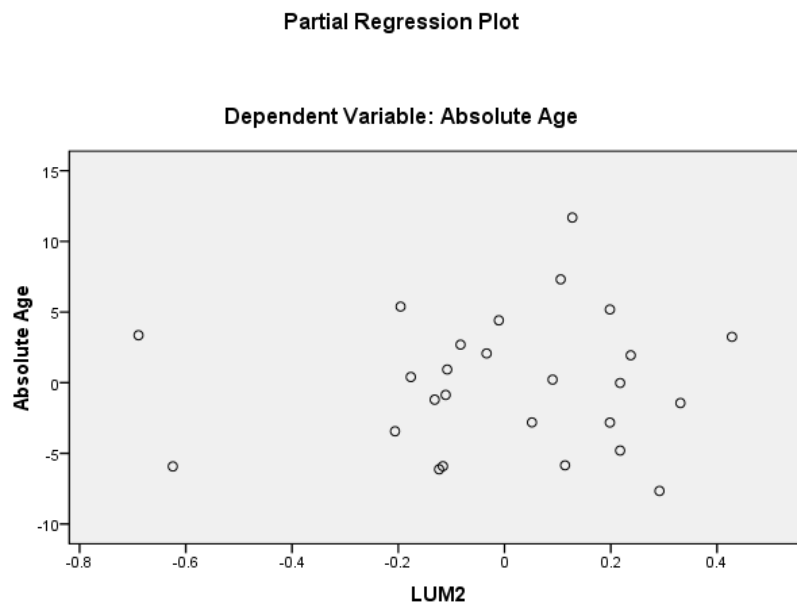


Figure 31: Chimpanzee Molar Wear – Partial Regression Plot Left Upper Molar 3

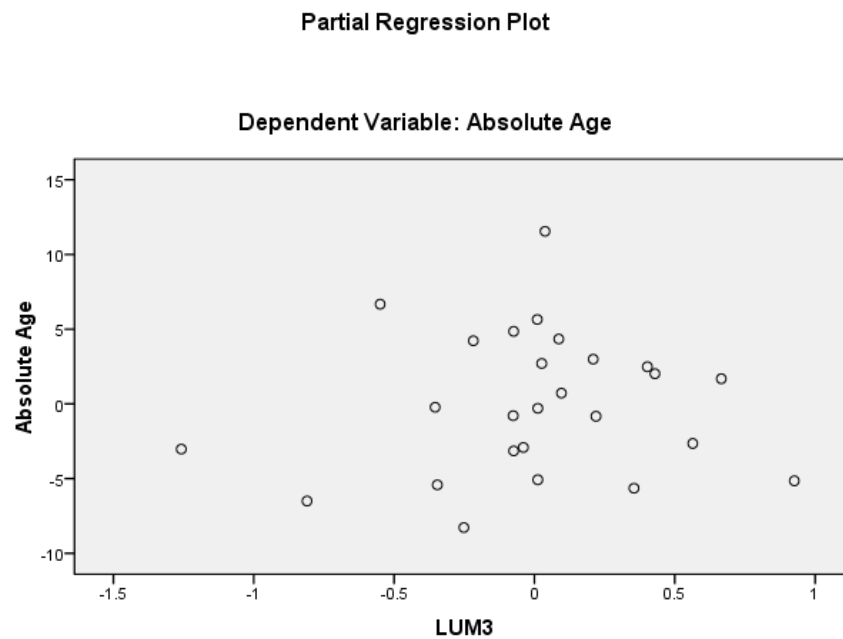


Figure 32: Chimpanzee Molar Wear – Partial Regression Plot Left Lower Molar 1

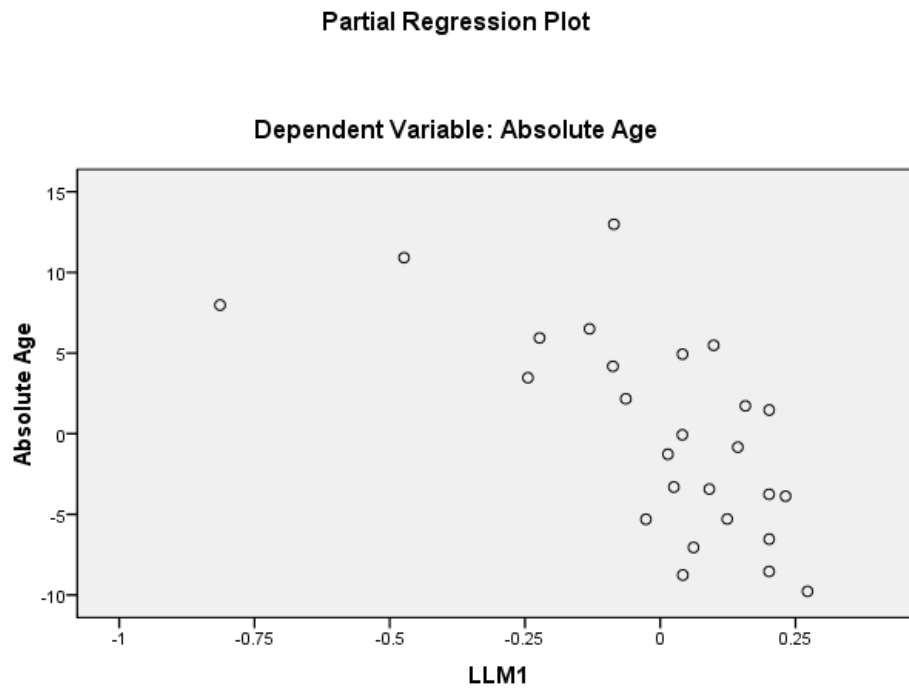


Figure 33: Chimpanzee Molar Wear – Partial Regression Plot Left Lower Molar 2

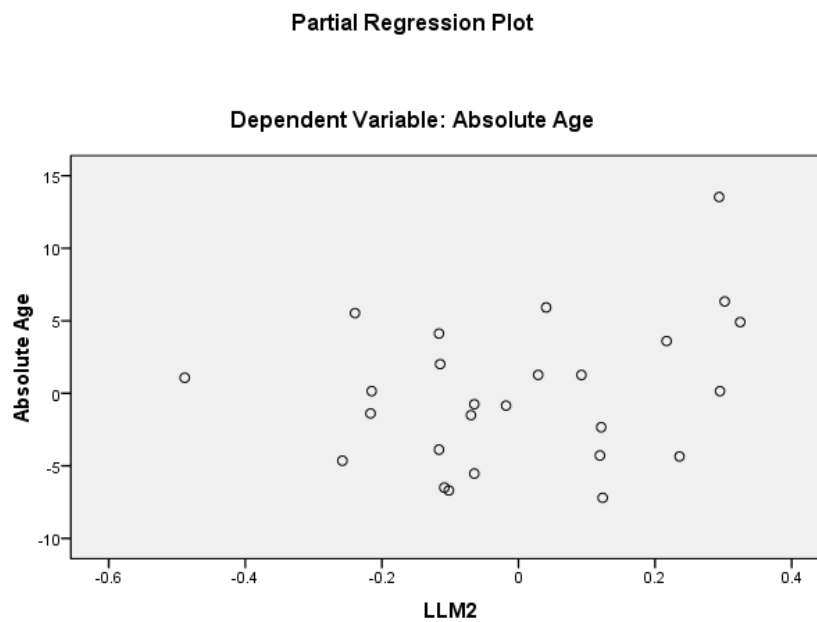


Figure 34: Chimpanzee Molar Wear – Partial Regression Plot Left Lower Molar 3

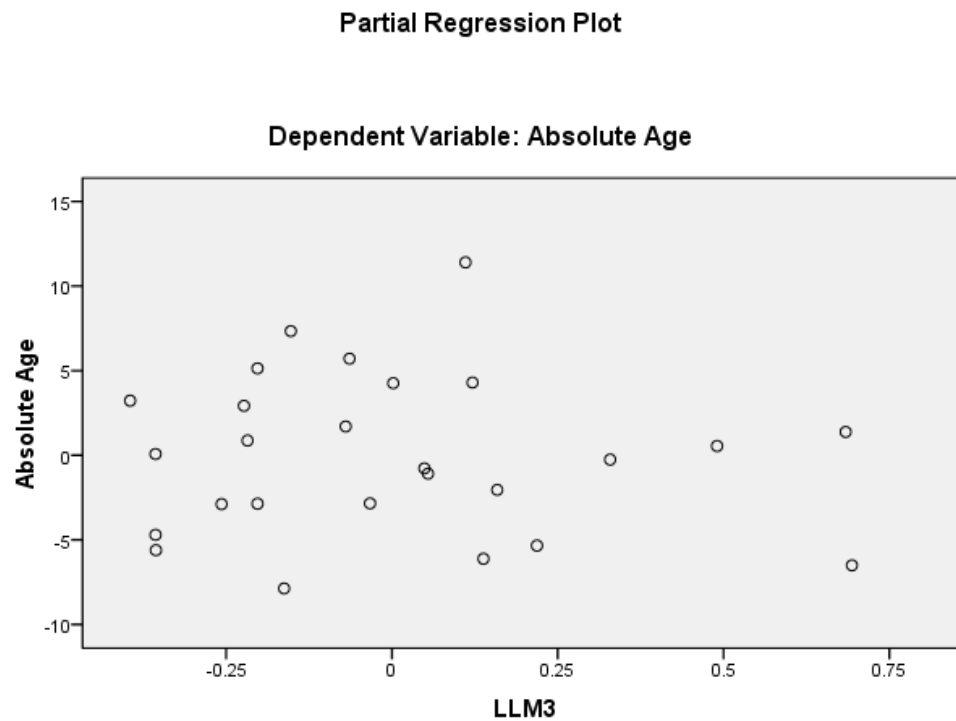


Figure 35: Chimpanzee Molar Wear – Partial Regression Plot Right Upper Molar 1

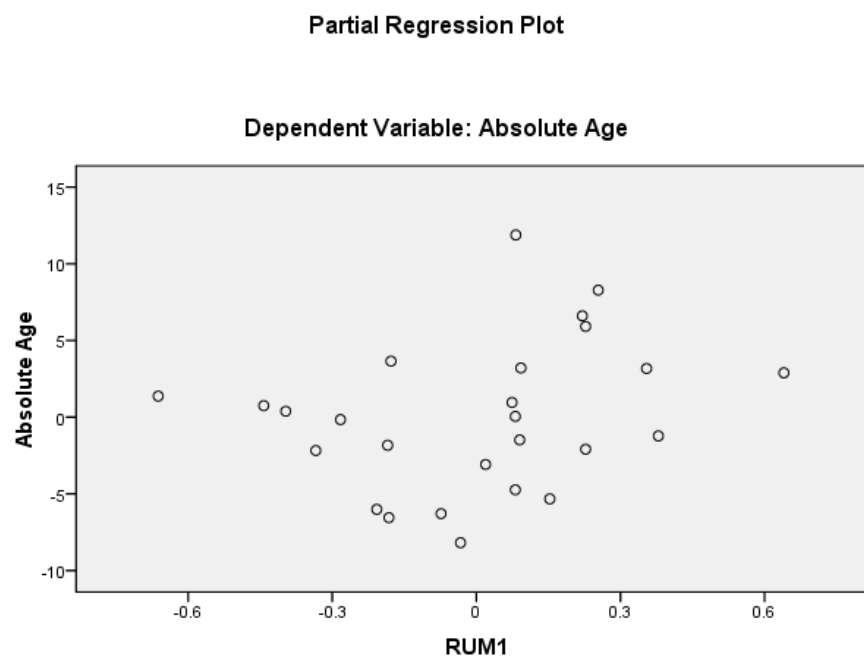


Figure 36: Chimpanzee Molar Wear – Partial Regression Plot Right Upper Molar 2



Figure 37: Chimpanzee Molar Wear – Partial Regression Plot Right Upper Molar 3

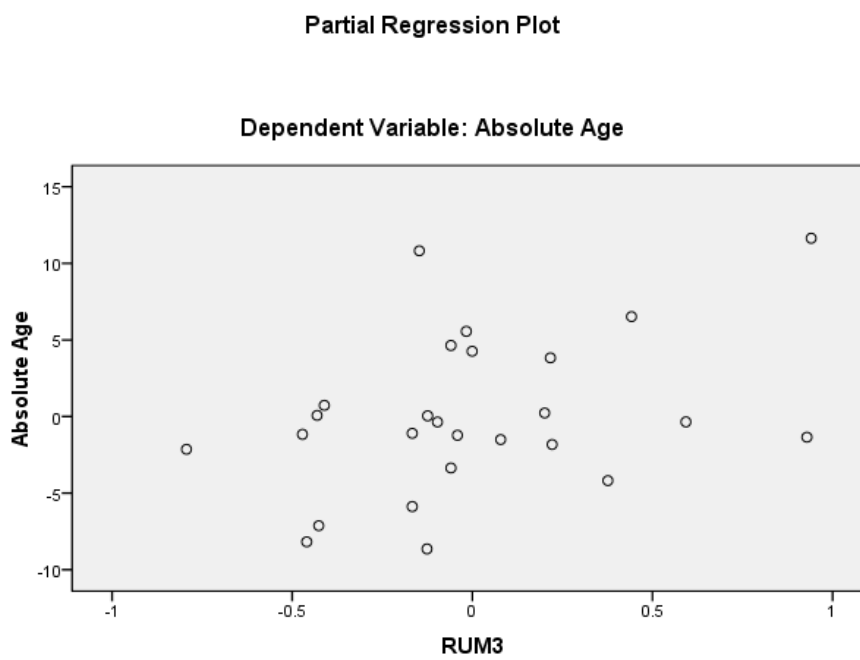


Figure 38: Chimpanzee Molar Wear – Partial Regression Plot Right Lower Molar 1

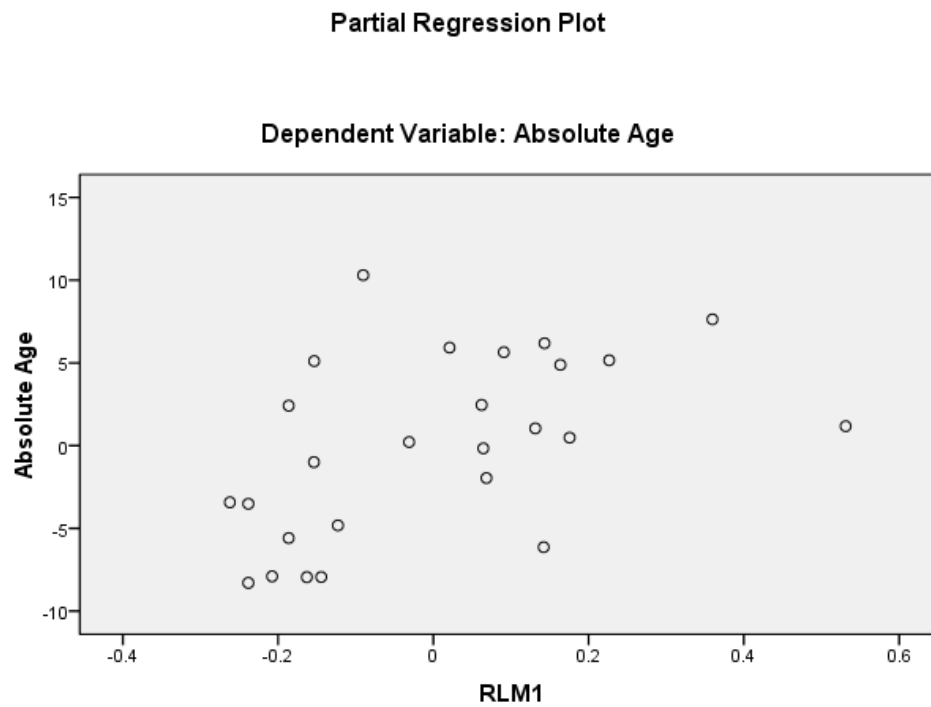
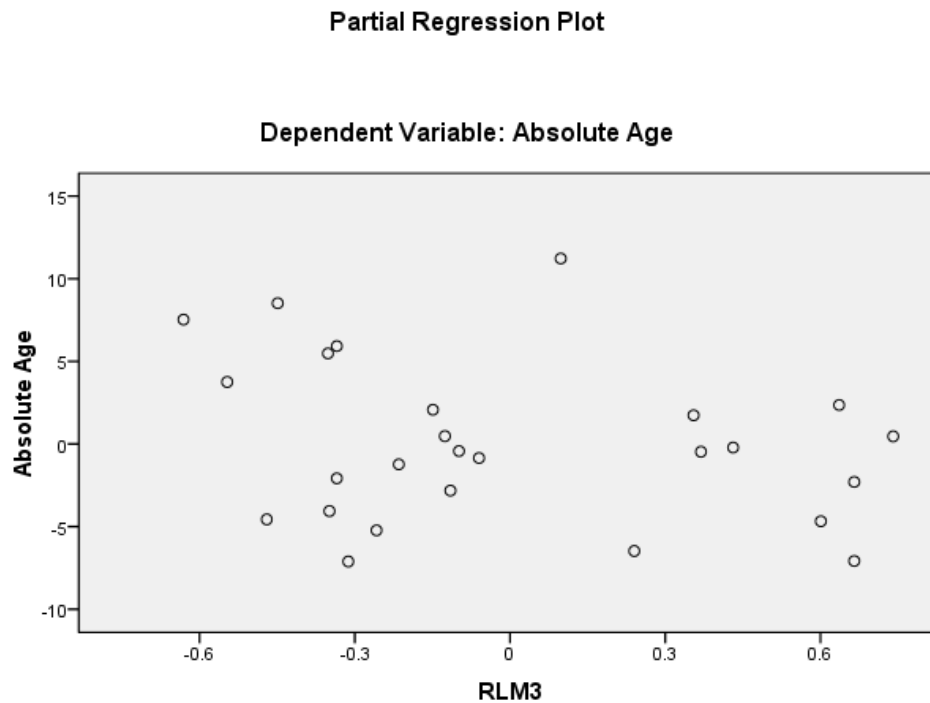


Figure 39: Chimpanzee Molar Wear – Partial Regression Plot Right Lower Molar 2



Figure 40: Chimpanzee Molar Wear – Partial Regression Plot Right Lower Molar 3



Molars 1 and 2: There were 28 known-aged individuals included in the analysis.

The model displayed a good fit with an R^2 of .688, and was significant at $p=.001$. The Pearson's correlations were significant for all of the molars included. The regression equation for this model, with unstandardized coefficients, is:

$$y = -1.304(\text{LUM1}) + 3.852(\text{LUM2}) - 13.128(\text{LLM1}) + 7.847(\text{LLM2}) + 3.503(\text{RUM1}) + 1.867(\text{RUM2}) + 11.610(\text{RLM1}) + .606(\text{RLM2})$$

The plot of the standardized residuals against standardized predicted values shown in figure 41 below indicates a random distribution, which is indicative of a situation in which the assumptions of linearity and homoscedasticity have been met. Further, a histogram of the residuals shown in figure 42 below demonstrates a near normal distribution, and a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that any deviations were not significant. Thus, the indications are that

either the full molar model or the reduced molar model works well for the chimpanzee data.

Figure 41: Chimpanzee Molars 1 and 2 Wear - Standardized Residuals plotted against Standardized Predicted Values

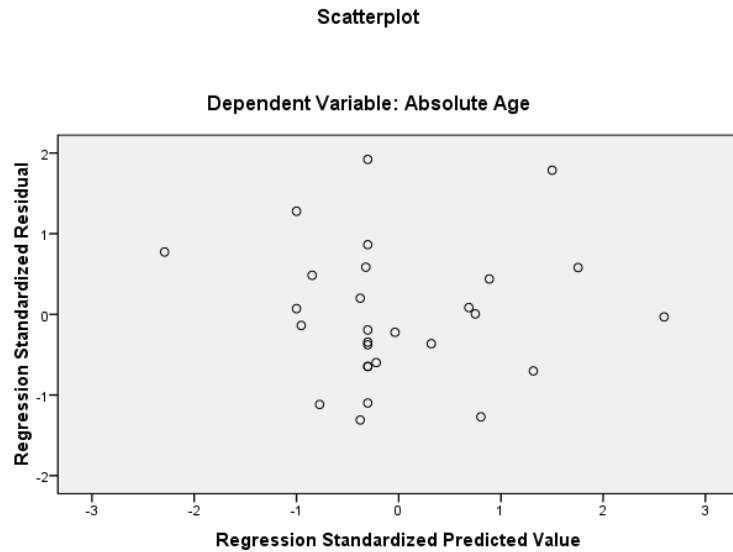
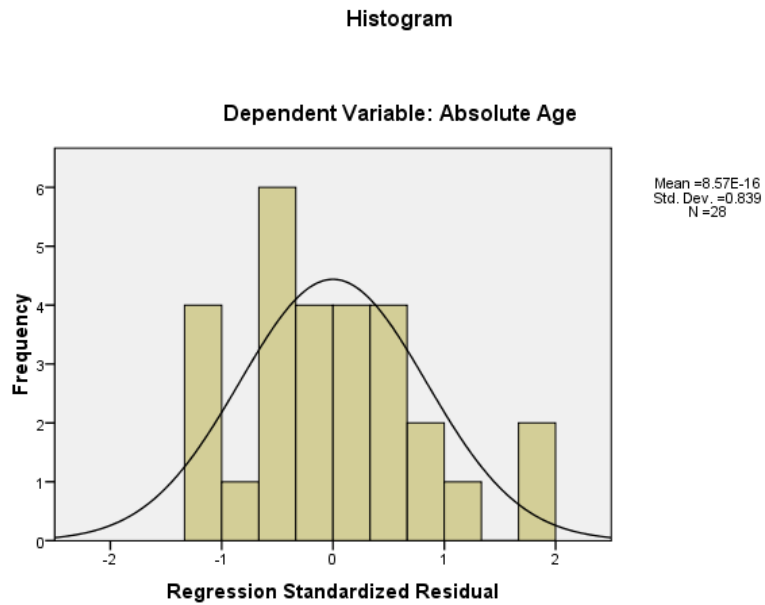


Figure 42: Chimpanzee Molars 1 and 2 Wear – Histogram of Residuals



Gorilla: All molars: There were 19 known-aged individuals included in the analysis. The model displayed a good fit with an R^2 of .787, but was not significant at $p=.231$. Table 18 shows the Pearson's correlations (bivariate correlations measuring the strength of the relationship between each marker and absolute age), and all except the left upper molar 1 (LUM1) are significant. The regression equation for this model, with unstandardized coefficients, is:

$$y = -3.829(\text{LUM1}) + -2.824(\text{LUM2}) + 4.411(\text{LUM3}) + 7.232(\text{LLM1}) + -0.970(\text{LLM2}) + -3.557(\text{LLM3}) + -1.092(\text{RUM1}) + 19.715(\text{RUM2}) + -7.693(\text{RUM3}) + -3.533(\text{RLM1}) + -2.522(\text{RLM2}) + -4.005(\text{RLM3})$$

Table 18: Gorilla Molar Linear Regression – Pearson's Correlations

		Value	Sig (1-tailed)
Pearson Correlation	Absolute Age		
	LUM1	.251	.150
	LUM2	.533	.009
	LUM3	.613	.003
	LLM1	.644	.001
	LLM2	.533	.009
	LLM3	.608	.003
	RUM1	.554	.007
	RUM2	.630	.002
	RUM3	.615	.003
	RLM1	.599	.003
	RLM2	.452	.026
	RLM3	.565	.006

The plot of the standardized residuals against standardized predicted values shown in figure 43 below indicates a near random distribution, which is indicative of a situation in which the assumptions of linearity and homoscedasticity have been met. However, a histogram of the residuals shown in figure 44 below demonstrates evidence of non-normality; although, a K-S normality test of the standardized residuals ($p=.037$)

indicates that these deviations were significant. Partial regression plots for the molars are shown below in figures 45-56. In general, these scatterplots show some evidence of a linear relationship, although outliers are evident.

Figure 43: Gorilla Molar Wear - Standardized Residuals plotted against Standardized Predicted Values

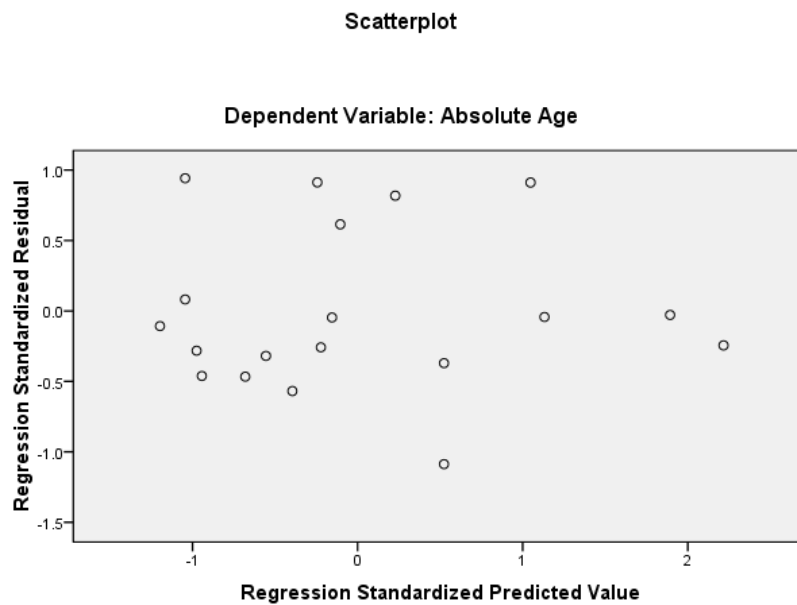


Figure 44: Gorilla Molar Wear – Histogram of Residuals

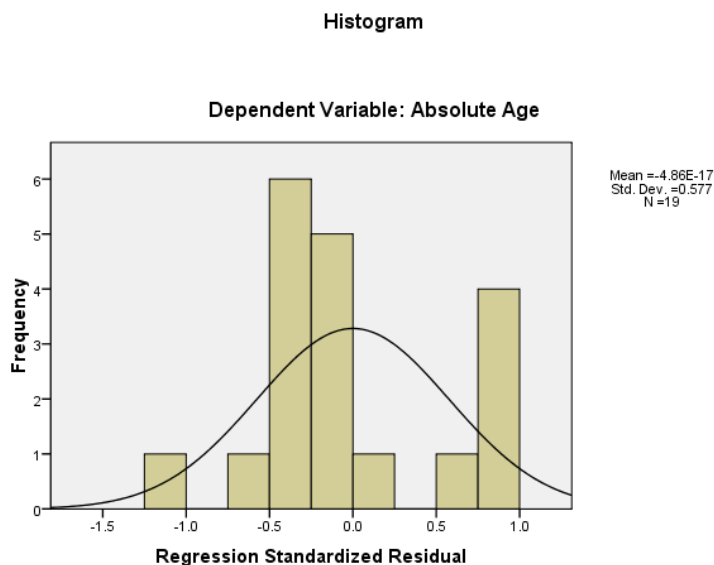


Figure 45: Gorilla Molar Wear – Partial Regression Plot Left Upper Molar 1

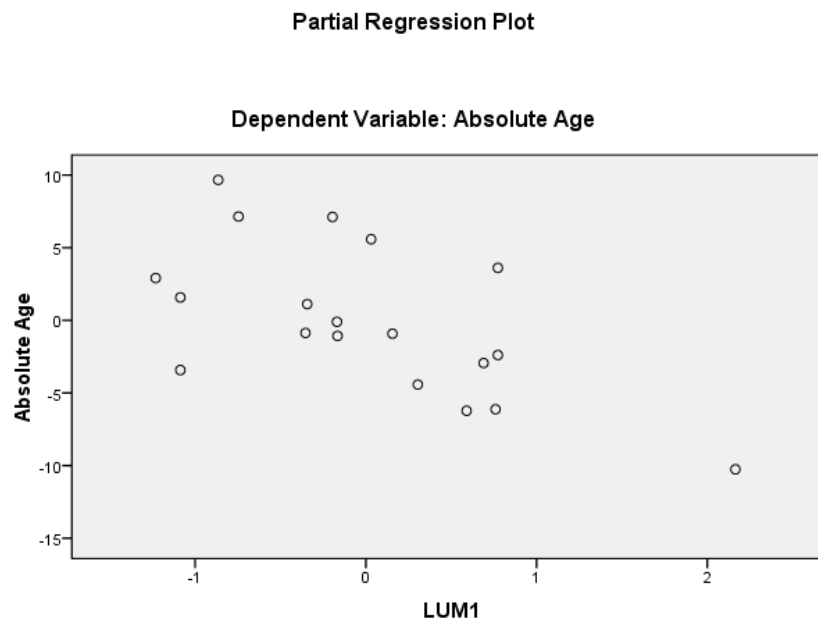


Figure 46: Gorilla Molar Wear – Partial Regression Plot Left Upper Molar 2

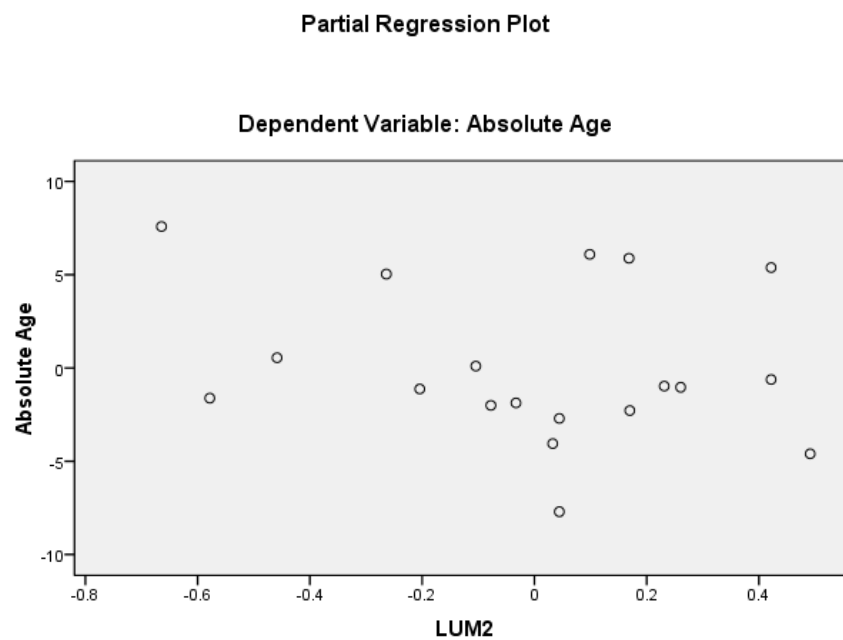


Figure 47: Gorilla Molar Wear – Partial Regression Plot Left Upper Molar 3

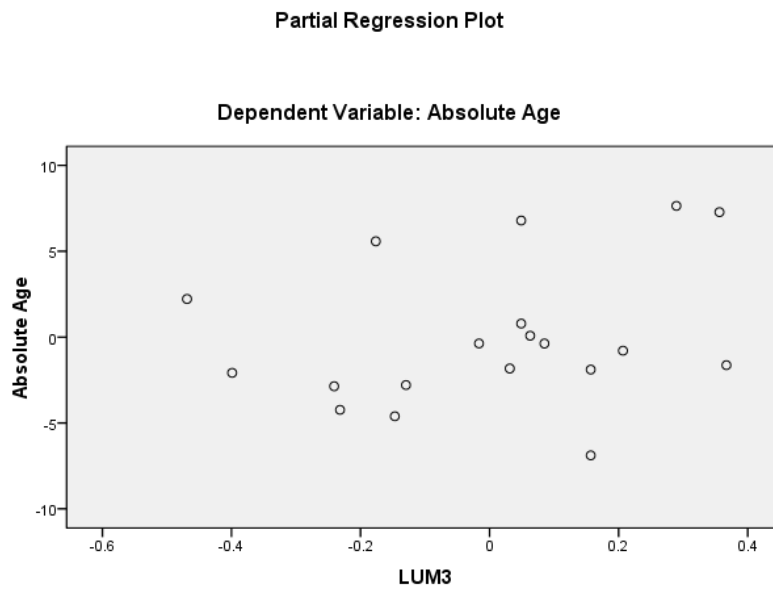


Figure 48: Gorilla Molar Wear – Partial Regression Plot Left Lower Molar 1

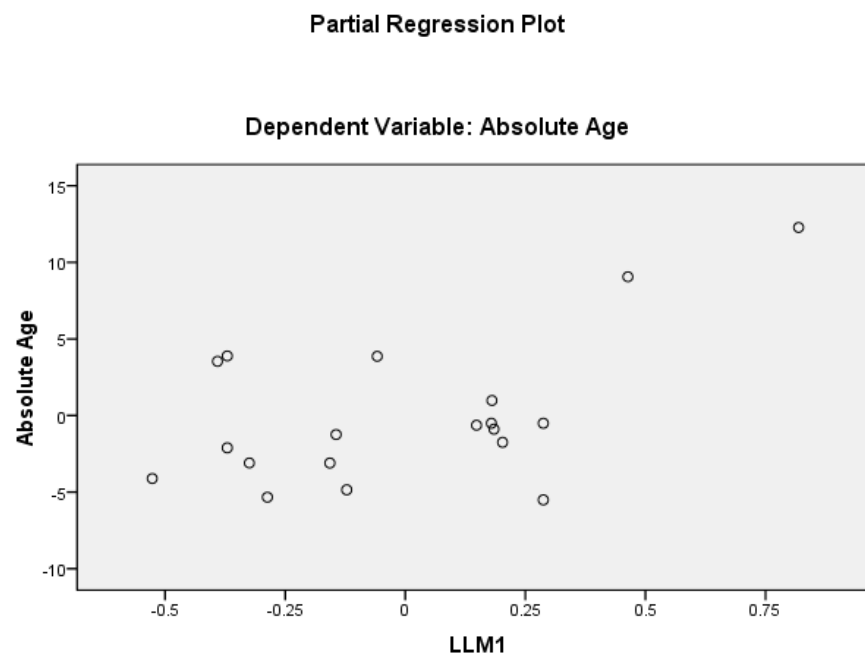


Figure 49: Gorilla Molar Wear – Partial Regression Plot Left Lower Molar 2

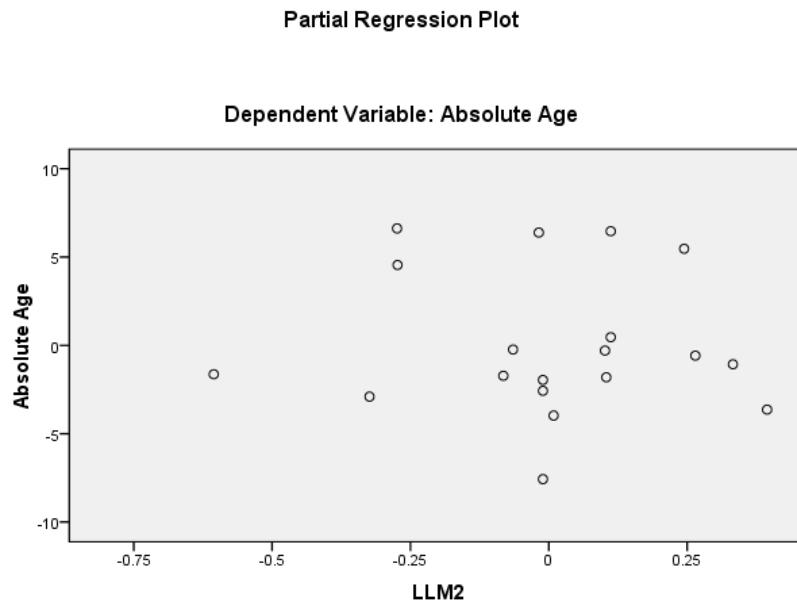


Figure 50: Gorilla Molar Wear – Partial Regression Plot Left Lower Molar 3

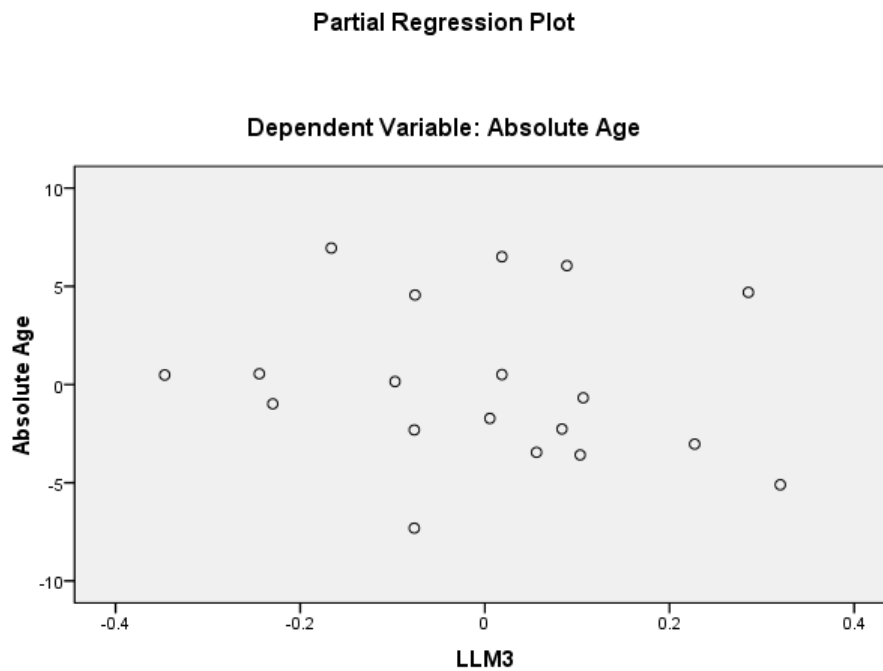


Figure 51: Gorilla Molar Wear – Partial Regression Plot Right Upper Molar 1

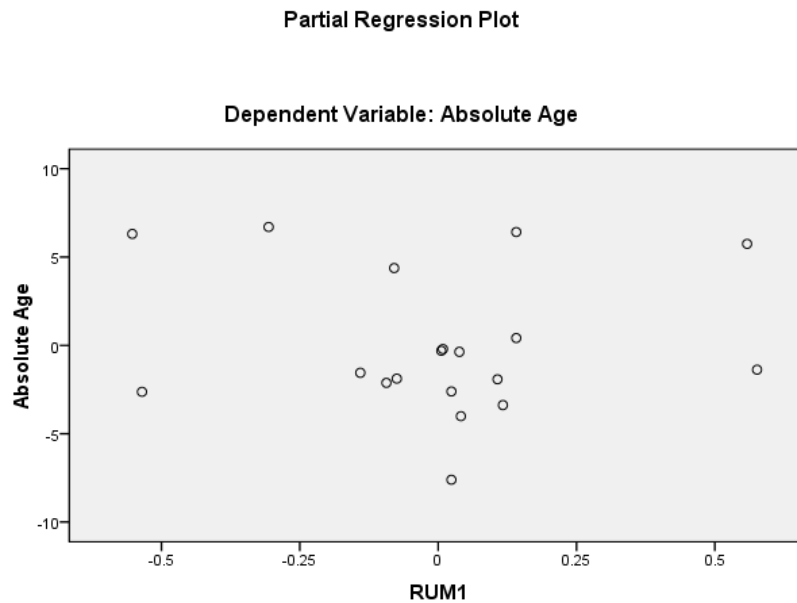


Figure 52: Gorilla Molar Wear – Partial Regression Plot Right Upper Molar 2



Figure 53: Gorilla Molar Wear – Partial Regression Plot Right Upper Molar 3

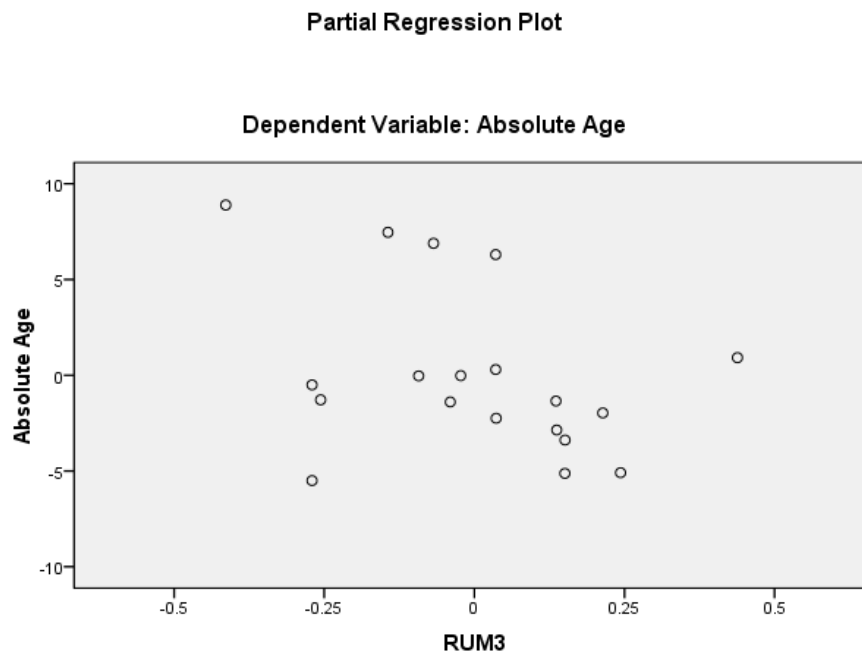


Figure 54: Gorilla Molar Wear – Partial Regression Plot Right Lower Molar 1

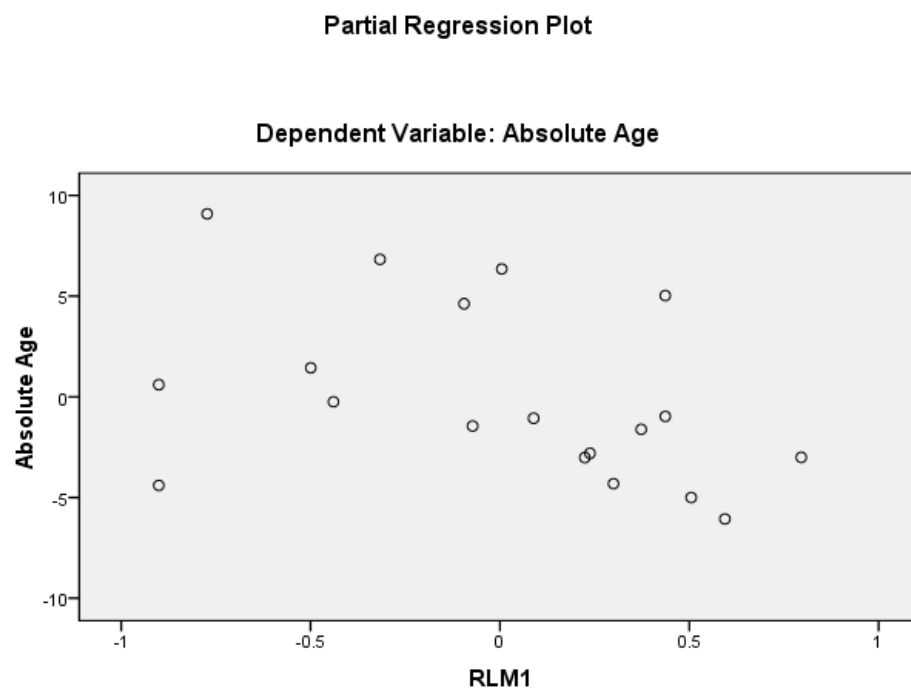


Figure 55: Gorilla Molar Wear – Partial Regression Plot Right Lower Molar 2



Figure 56: Gorilla Molar Wear – Partial Regression Plot Right Lower Molar 3



Molars 1 and 2: There were 19 known-aged individuals included in the analysis.

The model displayed a good fit with an R^2 of .701, and was marginally significant at $p=.057$. The regression equation for this model, with unstandardized coefficients, is:

$$y = -2.701(\text{LUM1}) + -0.990(\text{LUM2}) + 4.740(\text{LLM1}) + -5.862(\text{LLM2}) + -2.426(\text{RUM1}) + 9.674(\text{RUM2}) + -1.011(\text{RLM1}) + .343(\text{RLM2})$$

The plot of the standardized residuals against standardized predicted values shown in figure 57 below indicates a near random distribution, which is indicative of a situation in which the assumptions of linearity and homoscedasticity have been met. Further, a histogram of the residuals shown in figure 48 below demonstrates a near normal distribution, and a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that any deviations were not significant. Thus, the evidence indicates that utilizing only molars 1 and 2 provides a good fit for the gorilla data.

Figure 57: Gorilla Molar 1 and 2 Wear - Standardized Residuals plotted against Standardized Predicted Values

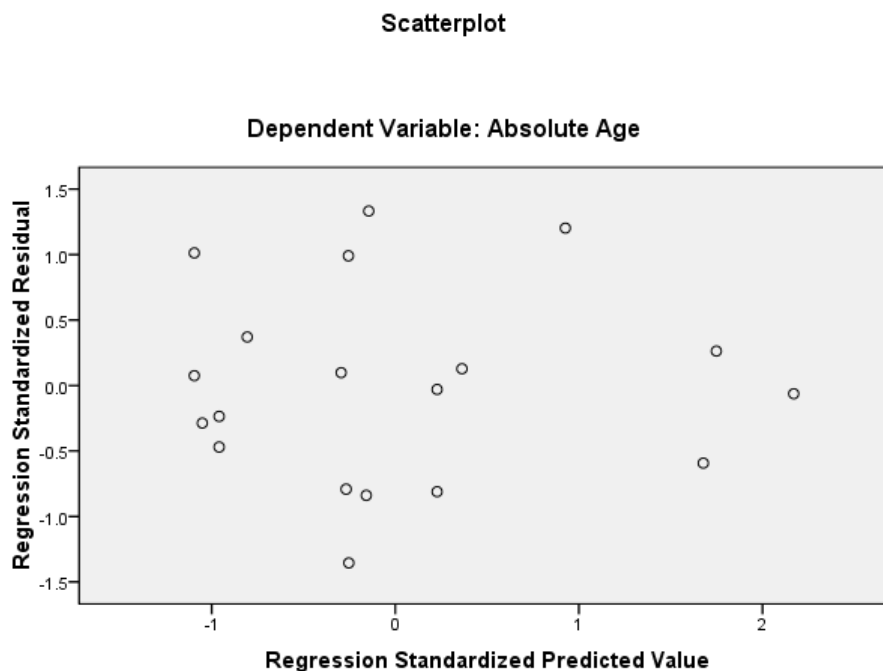
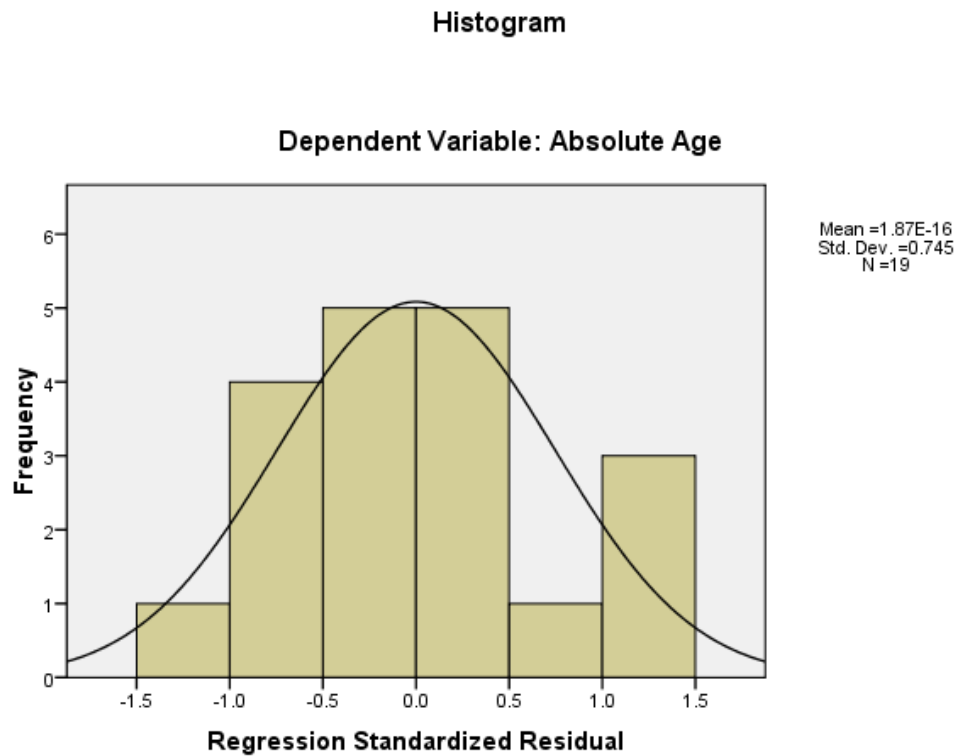


Figure 58: Gorilla Molar 1 and 2 Wear – Histogram of Residuals



Orangutans: All molars: There were 11 known-aged individuals included in the analysis; however, the regression model for the orangutan data could not be computed.

Molars 1 and 2: There were 15 known-aged individuals included in the analysis. The model displayed a reasonable fit with an R^2 of .405, although it was not significant at $p=.814$. Table 19 shows the Pearson's correlations (bivariate correlations measuring the strength of the relationship between each marker and absolute age), which are relatively low with only the right lower molar 2 (RLM2) being significant at $p=.044$; although, three others are near significant. The regression equation for this model, with unstandardized coefficients, is:

$$y = -1.448(\text{LUM1}) + -.865(\text{LUM2}) + .430(\text{LLM1}) + -.610(\text{LLM2}) + 1.720(\text{RUM1}) + -.229(\text{RUM2}) + 1.353(\text{RLM1}) + 1.620(\text{RLM2})$$

The plot of the standardized residuals against standardized predicted values shown in figure 59 below indicates a near random distribution, and a histogram of the residuals shown in figure 60 below demonstrates a distribution that deviates from normality; although, a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant. In general, scatterplots of the molars shown in figures 61-69 below demonstrate a slight linear relationship with outliers. Thus, although the molar 1 and 2 model is not significant, the evidence indicates that utilizing only these molars provides the best fit for the data. Further, it is likely that given a larger sample, with more variation in ages, that the molars would prove to be a reliable method for aging.

Table 19: Orangutan Molars 1 and 2 – Pearson’s Correlations

		Value	Sig (1-tailed)
Pearson Correlation	Absolute Age		
	LUM1	.047	.435
	LUM2	.367	.089
	LLM1	.427	.056
	LLM2	.427	.056
	RUM1	.335	.111
	RUM2	.274	.161
	RLM1	.436	.052
	RLM2	.455	.044

Figure 59: Orangutan Molar 1 and 2 Wear - Standardized Residuals plotted against Standardized Predicted Values

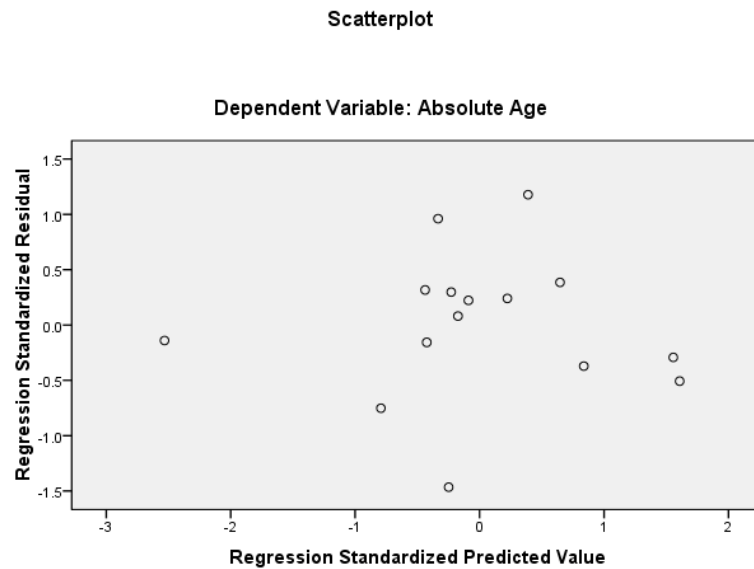


Figure 60: Orangutan Molar 1 and 2 Wear – Histogram of Residuals

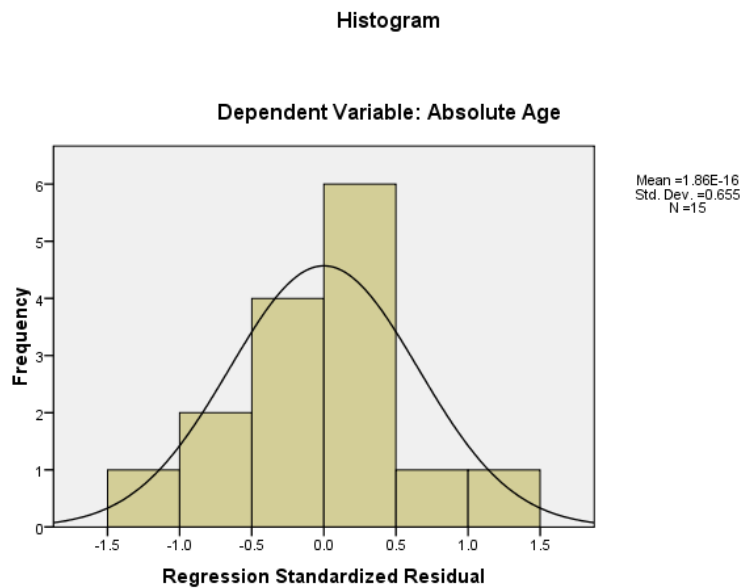


Figure 61: Orangutan Partial Regression Plot – Left Upper Molar 1

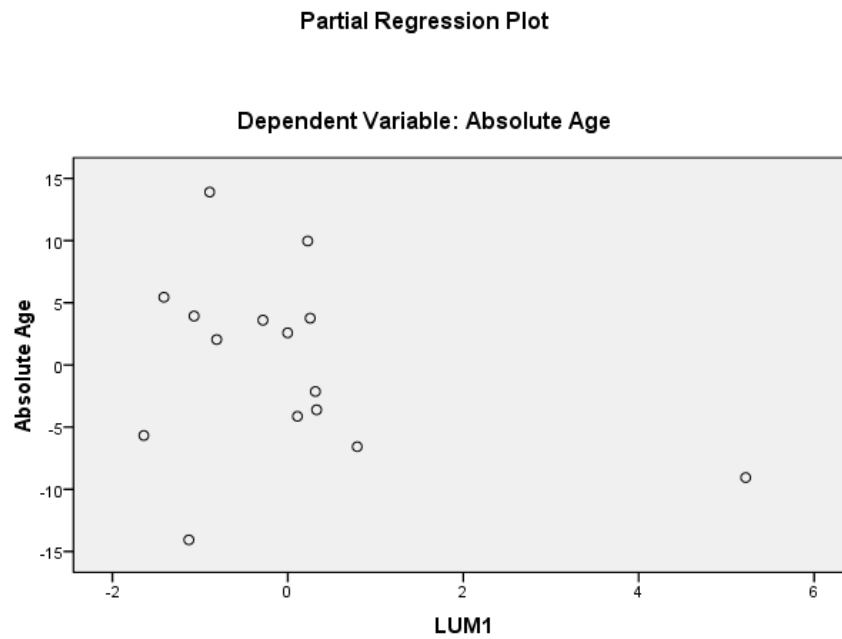


Figure 62: Orangutan Partial Regression Plot – Left Upper Molar 2

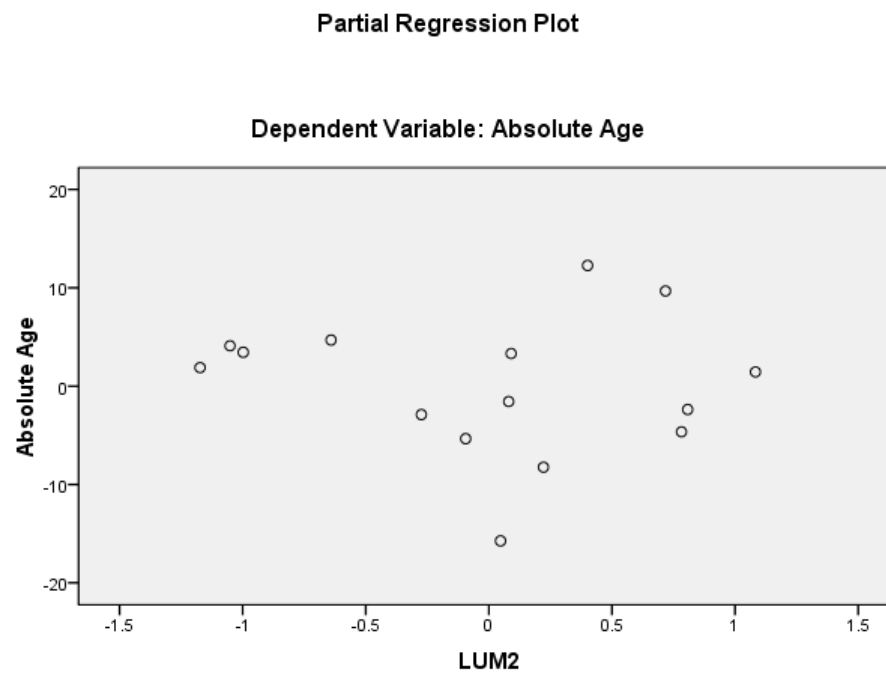


Figure 63: Orangutan Partial Regression Plot – Left Lower Molar 1

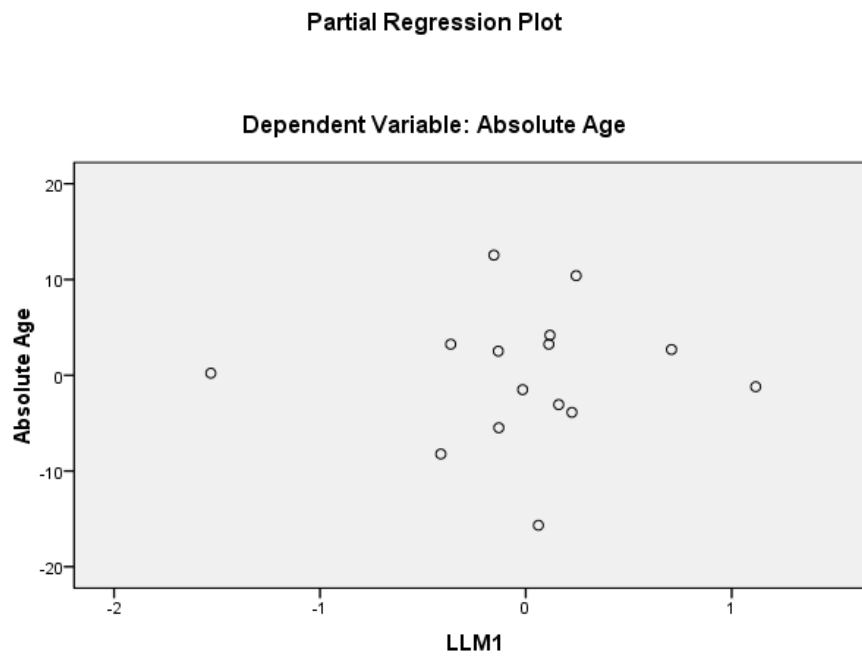


Figure 64: Orangutan Partial Regression Plot – Left Lower Molar 2

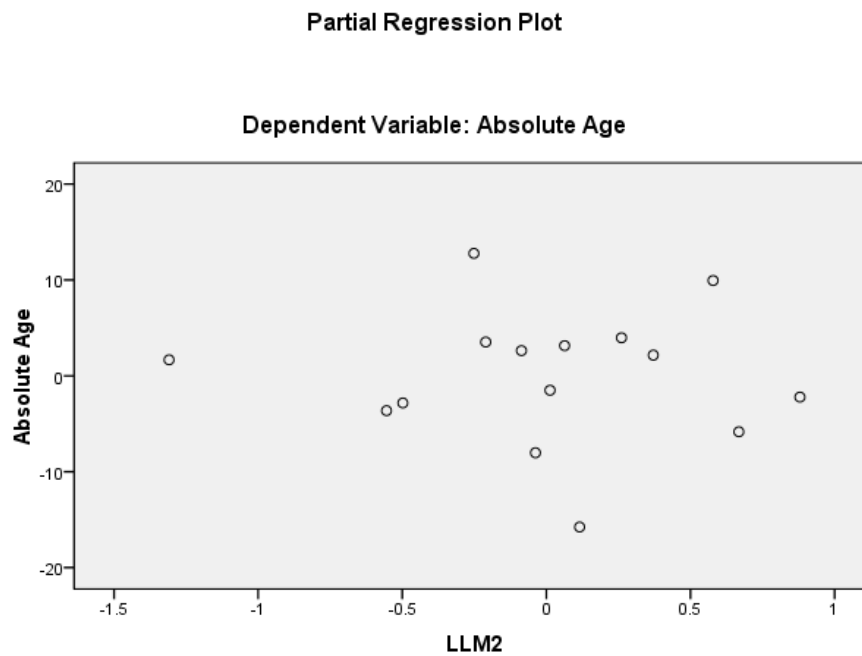


Figure 65: Orangutan Partial Regression Plot – Right Upper Molar 1

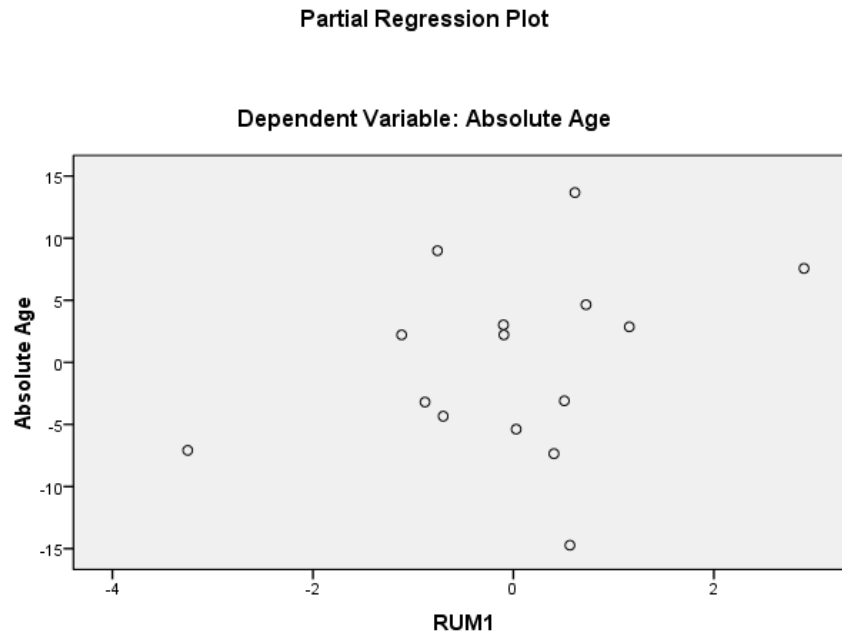


Figure 66: Orangutan Partial Regression Plot – Right Upper Molar 2

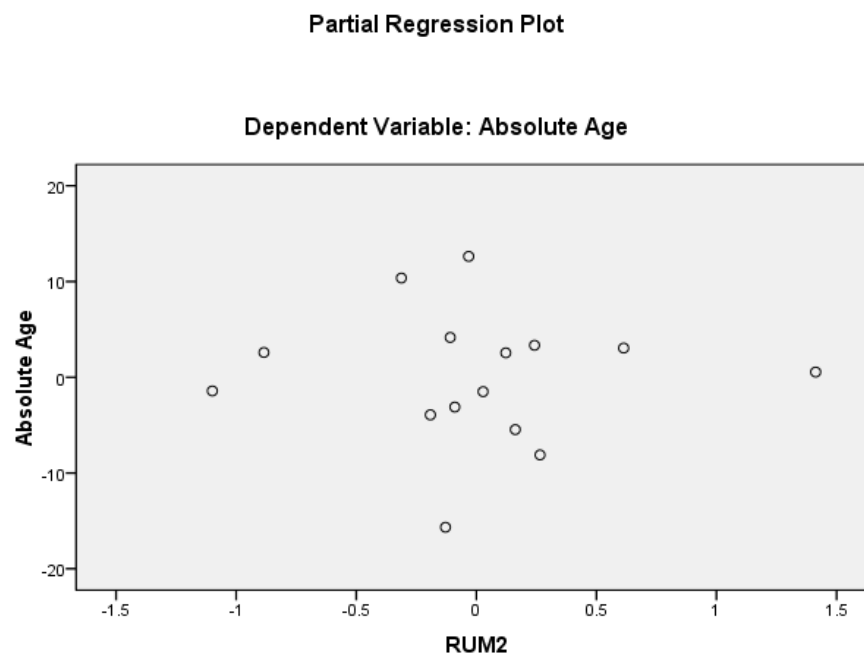


Figure 67: Orangutan Partial Regression Plot – Right Lower Molar 1

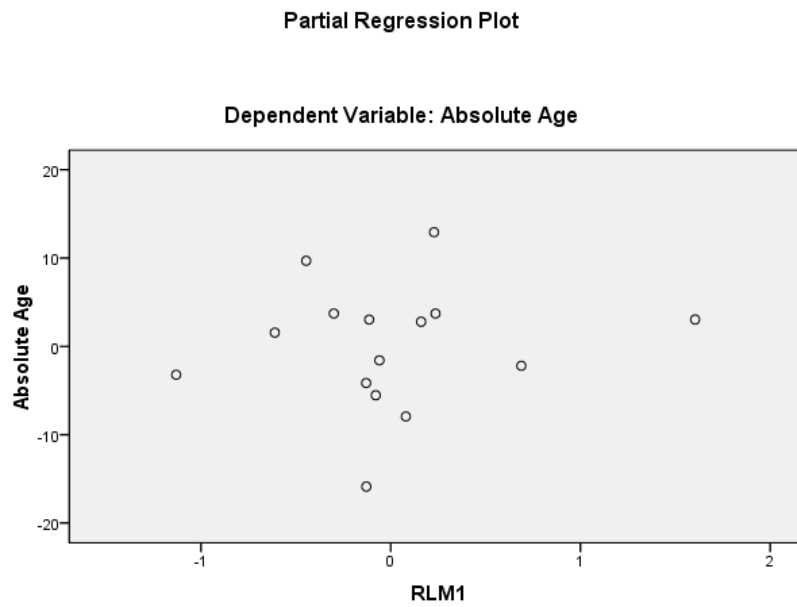


Figure 68: Orangutan Partial Regression Plot – Right Lower Molar 2



The evidence presented above suggests that the molars are the best single predictor for aging the great ape skeletons with some degree of accuracy. Further, the model that utilizes wear of molars 1 and 2 provides the best evidence for accurately aging individuals in all three species. Nevertheless, one concern with using these known-aged captive samples as the basis for estimating age in wild samples is the issue of severity of wear. While research suggests that wear occurs in a similar way in both wild and captive samples, it is not yet clear whether the rate of wear is equally comparable. This is because similar wear patterns are indicated since both wild and captive gorilla anterior dentition exhibits more wear than posterior dentition, despite the captive gorilla tendency towards malocclusion of maxillary and mandibular incisors. Further, it was found that the pattern of enamel attrition is similar to that of wild gorillas but with a lessened degree of severity (Nichols and Zihlman, 2002). Appendix B shows scatter plots of total molar wear by status (wild/captive). For chimpanzees, the spread of the data is relatively uniform for both the wild and captive sample. There are slightly more individuals with higher rates of wear in the wild sample, although both the captive and wild sample contains at least one individual with severe wear and the overall spread of data is similar. For gorillas, the data spread is similar, but with one outlier in the captive sample showing more severe wear. For orangutans, the captive sample shows more wear than the wild sample with severe wear displayed in at least three individuals; however, the spread of data for individuals with mild to moderate wear is similar for both captive and wild individuals. These results demonstrate that the wild and captive samples have similar wear patterns with relatively even spread. Outliers in the orangutan sample indicate more severe wear in the captive individuals and this is most likely attributed to the older age of

the known-aged individuals. Given the data spread of this dataset, it appears reasonable to utilize the known-aged captive sample to predict ages for the wild sample.

Despite the fact that molars 1 and 2 provide the best fit for the data in estimating age, it is also possible that utilizing the molars in conjunction with features of the acetabulum would be a viable option. This is because of the three postcranial skeletal markers tested the acetabulum demonstrated the most potential for use in aging and thus a model that includes both molar wear and features of the acetabulum might prove to be a stronger predictor of age than a model based on either the molars or acetabulum alone. Further, a model that utilizes postcranial features would be useful in estimating age in those individuals where only postcranial elements are available (i.e., no skull) or in cases where only partial scores are available. To determine if such a model is appropriate, linear regression was performed utilizing molars 1 and 2 (8 variables: 2 in each quadrant) and those features of the acetabulum that demonstrated the most likely evidence of age-related change (6 variables: left and right fossa, left and right rim, and left and right lunate porosity). The results are as follows:

Chimpanzees: There were 23 known-aged individuals included in the analysis. The model displayed a good fit with an R^2 of .914, and it was significant at $p=.008$. Table 20 shows the Pearson's correlations (bivariate correlations measuring the strength of the relationship between each marker and absolute age), which are significant in all but two variables. The plot of the standardized residuals against standardized predicted values shown in figure 69 below indicates a random distribution, although a histogram of the residuals shown in figure 70 below demonstrates a distribution with some deviation

from normality; although, a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant.

Table 20: Chimpanzee Molar/Acetabulum – Pearson’s Correlations

		Value	Sig (1-tailed)
Pearson Correlation	Absolute Age		
	LUM1	.359	.046
	LUM2	.498	.008
	LLM1	.379	.037
	LLM2	.542	.004
	RUM1	.605	.001
	RUM2	.629	.001
	RLM1	.538	.004
	RLM2	.521	.005
	AceLR	.599	.001
	AceLF	.456	.014
	AceLLP	-.069	.378
	AceRR	.619	.001
	AceRF	.369	.041
	AceRLP	.067	.381

Figure 69: Chimpanzee Molar/Acetabulum Model - Standardized Residuals plotted against Standardized Predicted Values

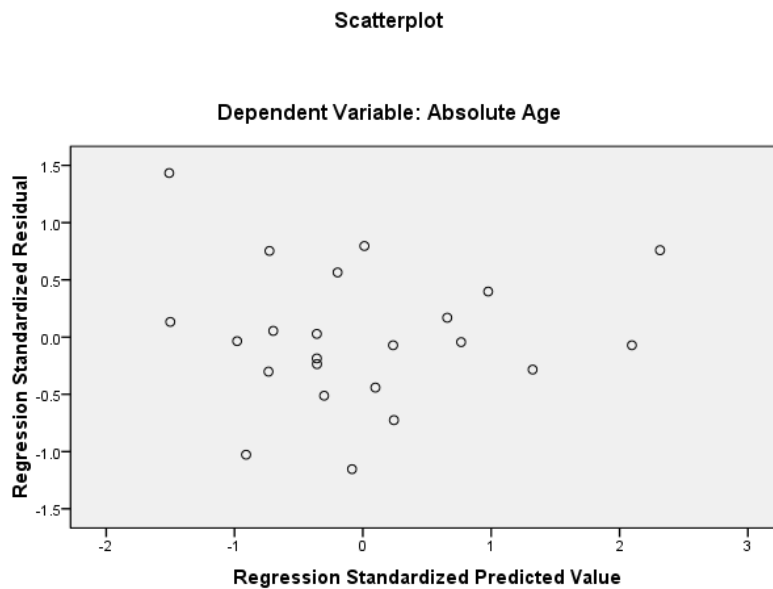
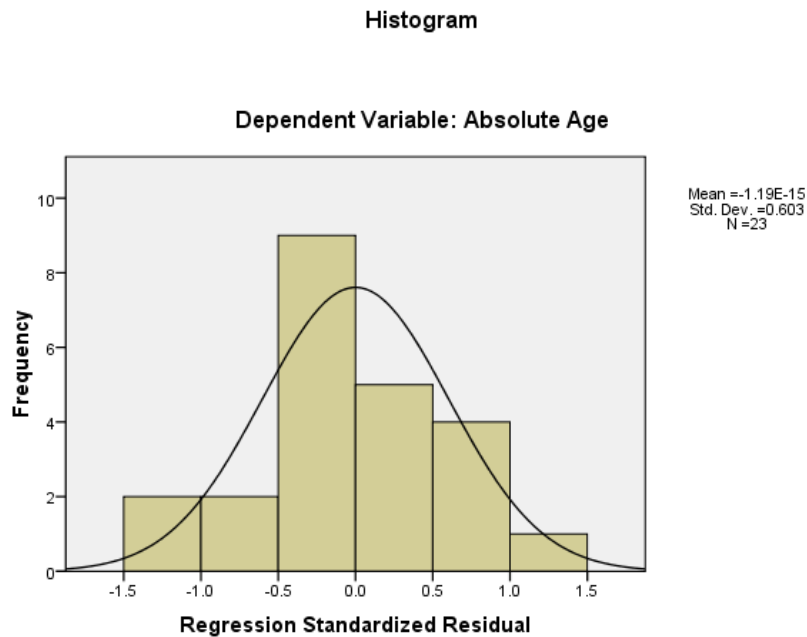


Figure 70: Chimpanzee Molar/Acetabulum Model – Histogram of Residuals



The regression equation, with unstandardized coefficients (shown in table 21 below), is:

$$y = -1.556(\text{LUM1}) + 5.123(\text{LUM2}) - 13.303(\text{LLM1}) + 8.989(\text{LLM2}) + 1.408(\text{RUM1}) + 2.049(\text{RUM2}) + 12.722(\text{RLM1}) + -4.030(\text{RLM2}) + 1.435(\text{AceLR}) + 3.552(\text{AceLF}) + -2.308(\text{AceLLP}) + 3.456(\text{AceRR}) + -0.155(\text{AceRF}) + 0.826(\text{AceRLP})$$

Table 21: Chimpanzee Molars 1 and 2/Acetabulum Coefficients

Model	Unstandardized Coefficients	
	B	Std. Error
1 (Constant)	-13.138	6.119
LUM1	-1.556	2.286
LUM2	5.123	4.339
LLM1	-13.303	5.456
LLM2	8.989	5.692
RUM1	1.408	4.746
RUM2	2.049	4.060
RLM1	12.722	6.270
RLM2	-4.030	4.056
AceLR	1.435	3.251
AceLF	3.552	3.590
AceLLP	-2.308	2.313
AceRR	3.456	3.100
AceRF	-.155	5.011
AceRLP	.826	3.058

a. Dependent Variable: Absolute Age

Gorillas: There were 15 known-aged individuals included in the analysis. The model displayed a good fit with an R^2 of .972, although it was not significant at $p=.452$. Table 22 shows the Pearson's correlations (bivariate correlations measuring the strength of the relationship between each marker and absolute age), which are significant in all but four variables. The plot of the standardized residuals against standardized predicted values shown in figure 71 below indicates a random distribution, although a histogram of the residuals shown in figure 72 below demonstrates a distribution with some deviation from normality; although, a K-S normality test of the standardized residuals ($p=.200$ (lower bound)) indicates that these deviations were not significant.

Table 22: Gorilla Molar/Acetabulum – Pearson’s Correlations

		Value	Sig (1-tailed)
Pearson Correlation	Absolute Age		
	LUM1	-.012	.483
	LUM2	.459	.043
	LLM1	.453	.045
	LLM2	.325	.119
	RUM1	.431	.054
	RUM2	.641	.005
	RLM1	.541	.019
	RLM2	.216	.220
	AceLR	.679	.003
	AceLF	.540	.019
	AceLLP	.663	.004
	AceRR	.679	.003
	AceRF	.533	.020
	AceRLP	.739	.001

Figure 71: Gorilla Molar/Acetabulum Model - Standardized Residuals plotted against Standardized Predicted Values

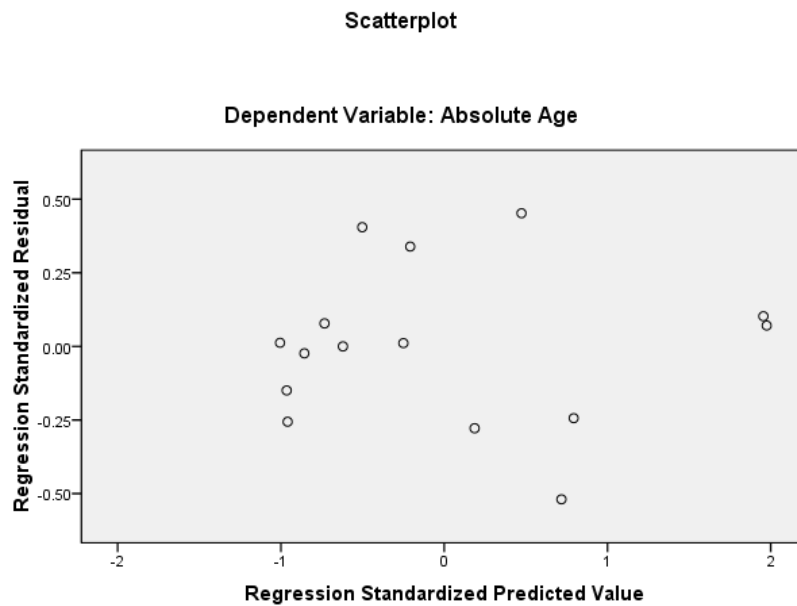
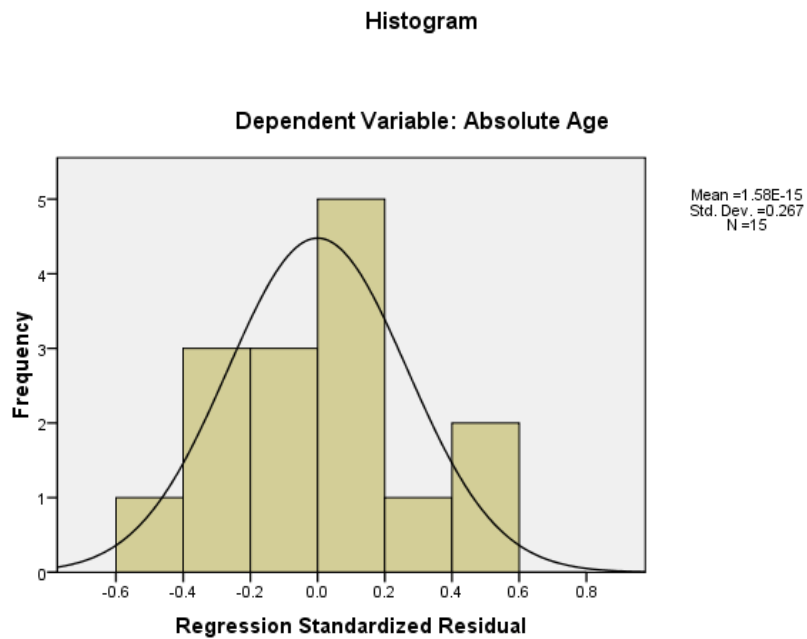


Figure 72: Gorilla Molar/Acetabulum Model – Histogram of Residuals



The regression equation, with unstandardized coefficients (shown in table 23 below), is:

$$y = 1.685(\text{LUM1}) + 23.314(\text{LUM2}) = 6.414(\text{LLM1}) + -16.325(\text{LLM2}) + .242(\text{RUM1}) + -27.902(\text{RUM2}) + .357(\text{RLM1}) + 13.044(\text{RLM2}) + -14.551(\text{AceLF}) + 3.706(\text{AceLLP}) + 28.996(\text{AceRR}) + 14.265(\text{AceRF}) + -9.363(\text{AceRLP})$$

Table 23: Gorilla Molars 1 and 2/Acetabulum Coefficients

Model	Unstandardized Coefficients	
	B	Std. Error
1 (Constant)	-42.889	26.988
LUM1	1.685	3.356
LUM2	23.314	14.011
LLM1	6.414	8.594
LLM2	-16.325	13.231
RUM1	.242	5.028
RUM2	-27.902	16.596
RLM1	.357	3.155
RLM2	13.044	8.463
AceLF	-14.551	12.897
AceLLP	3.706	3.502
AceRR	28.996	16.500
AceRF	14.265	14.038
AceRLP	-9.363	8.764

a. Dependent Variable: Absolute Age

Orangutans: There were 13 known-aged individuals included in the analysis.

The model displayed a good fit with an R^2 of .952, although it was not significant at $p=.528$. Table 24 shows the Pearson's correlations (bivariate correlations measuring the strength of the relationship between each marker and absolute age), which are significant in two variables, near significant, in three variables, and not significant in the remaining variables. The plot of the standardized residuals against standardized predicted values shown in figure 73 below indicates a near random distribution, although a histogram of the residuals shown in figure 74 below demonstrates a distribution with some deviation from normality; although, a K-S normality test of the standardized residuals ($p=.055$) indicates that these deviations were not significant.

Table 24: Orangutan Molar/Acetabulum Model – Pearson’s Correlations

		Value	Sig (1-tailed)
Pearson Correlation	Absolute Age		
	LUM1	-.083	.071
	LUM2	.340	.068
	LLM1	.370	.030
	LLM2	.384	.181
	RUM1	.292	.491
	RUM2	.243	.052
	RLM1	.374	.082
	RLM2	.460	.365
	AceLR	.715	.055
	AceLF	.331	.000
	AceRR	.715	.055
	AceRF	.175	.

Figure 73: Orangutan Molar/Acetabulum Model - Standardized Residuals plotted against Standardized Predicted Values

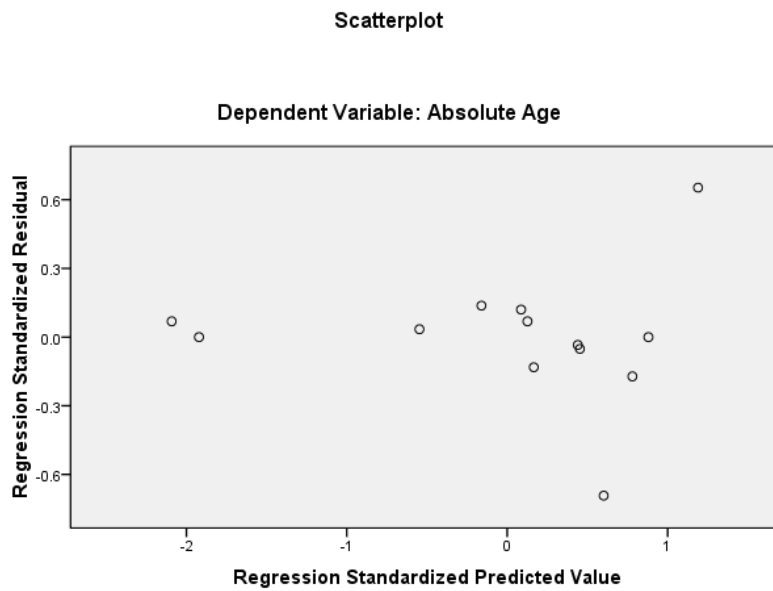
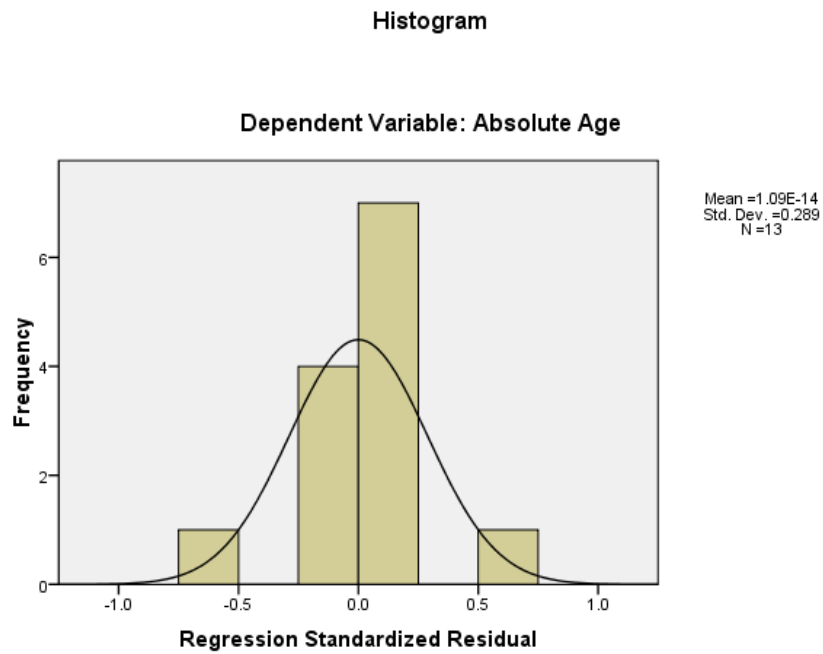


Figure 74: Orangutan Molar/Acetabulum Model – Histogram of Residuals



The regression equation, with unstandardized coefficients (shown in table 25 below), is:

$$y = 23.174(\text{LUM1}) + -11.629(\text{LUM2}) = -22.691(\text{LLM1}) + -27.417(\text{LLM2}) + 6.462(\text{RUM1}) + 21.955(\text{RUM2}) + 27.828(\text{RLM1}) + -17.894(\text{RLM2}) + 104.109(\text{AceLF}) + 12.746(\text{AceRR}) + -64.471(\text{AceRF})$$

Table 25: Orangutan Molars 1 and 2/Acetabulum Coefficients

Model		Unstandardized Coefficients	
		B	Std. Error
1	(Constant)	-200.389	119.818
	LUM1	23.174	13.399
	LUM2	-11.629	10.523
	LLM1	-22.691	11.470
	LLM2	-27.417	12.847
	RUM1	6.462	3.318
	RUM2	21.955	11.078
	RLM1	27.828	12.708
	RLM2	-17.894	8.804
	AceLF	104.109	47.692
	AceRR	12.746	6.349
	AceRF	-64.471	27.321

a. Dependent Variable: Absolute Age

In general, the results presented above provide evidence that a combined model utilizing wear in molars 1 and 2 and certain features of the acetabulum is a viable option for all three species. And, while the gorilla and orangutan models demonstrate some evidence of non-normality, this could be due to the small samples. Further, K-S normality tests of the standardized residuals indicate that the deviations are not significant. But, due to the lack of overall model significance in the gorilla and orangutan, and to explore the utility of correcting potential problems in these datasets, both log and square root transformations were performed. There was little improvement in the models and thus, for purposes of continuity, the untransformed data was utilized. Nevertheless, a greater number of known-aged individuals, particularly for orangutans and gorillas, would be required to verify the model presented.

Conclusion: Aging of adult great ape skeletons has been problematic and, particularly for wild specimens, age is often unknown. But, a more rigorous means of determining age in the ape sample is crucial because age effects are known to contribute to OA expression. In the great apes, methods that are commonly used assign ages based on closure of the proximal humerus (to distinguish adults) and basilar suture (to separate old adults from younger animals). Using this method, only broad age categories can be utilized and the accuracy of utilizing the basilar suture is highly questionable and, based on the results from this research, not recommended. This is because while the basilar suture shows some variability in age of closure, it is closed at an early age in virtually all known-aged captive individuals. Thus, an “old adult” category most likely contains individuals who are, in fact, relatively young.

Several other methods of aging, based on techniques developed for aging human skeletons, were tested. The ribs and auricular surface proved to be of limited use for aging the ape skeletons. Of the features that did have recordable data, there was generally not enough variation in the known-aged individuals. Further, evidence of non-linearity and non-normally distributed data lend support to the idea that there is a lack of relationship between features of the ribs and the auricular surface with age; however, the small number of observations included in these models, particularly for gorillas and orangutans, mean that this suggestion should be considered tentative. Aging of the apes utilizing the ribs and auricular surface appears to be further confounded due to potential differences as to which predictors (if any) are correlated with age among the three different species. Thus, the ribs and auricular surface were not considered to be viable

for use as aging markers in this study; however, future research that includes larger samples would be necessary to verify the utility of these aging markers.

Unlike the ribs and auricular surface, the acetabulum demonstrates some potential for use in aging. There was some evidence of non-linearity and non-normally distributed data in gorillas and orangutans, although K-S normality tests of the standardized residuals indicate that these deviations were not significant. Thus, based on the small number of observations, any suggestion of non-normality in the data is considered tentative. As with the ribs and auricular surface, different variables are highlighted in each species as being significant, and a reduction in the number of variables utilized resulted in an improved model; however, more data would be needed to verify this assumption. Further, one additional concern with using the acetabulum to estimate age is that it has not been clearly established how much influence arthritic-related changes have and how they might influence the results. Thus, using the acetabulum as the *sole* marker for age was not considered viable for this study.

While the acetabulum shows some value in estimating ages in the ape sample, molar dental wear proved to be the most useful aging indicator. The model utilizing upper and lower molars 1 and 2 (8 variables) provided a good fit, particularly in chimpanzees and gorillas. Although the orangutan model was not significant, this is most likely due to the limited number of known-aged specimens and a dataset that is skewed toward older individuals. As with all of the methods tested, more data would be needed to verify the model; however, the indications are that wear on molars 1 and 2 provides a reasonable estimate for aging the skeletons. Nevertheless, a model that included both molars 1 and 2 and certain features of the acetabulum also proved to be viable. In the

combined model, the R^2 was extremely high for all three species (above 90%), although it was significant only in the chimpanzee. Further, the Pearson's correlations, which test the strength of the relationship between each marker and age, are significant for nearly all of the markers and this lends further credence that this method is a viable option for predicting age.

Even though the combined model is considered the most appropriate method for aging the great ape skeletons (and was the one chosen here), it is acknowledged that this system is far from perfect. The biggest problem encountered is lack of known-aged individuals, particularly in the gorilla and orangutan sample. Another problem is that different variables are sometimes being highlighted as being significant in each species; however, this could be due to the small samples or to true morphological differences among the species. Based on the appearance of certain features of the ribs (especially margins, nodules, pit depth), it is probable that morphological differences among the species are a factor; future research may clarify this suggestion. The high R^2 values for many of the tests are not unexpected given that there are lots of predictor variables with relatively few individuals. Thus, it is possible that the high R^2 gives a false impression of how well the equation would work on other individuals. And yet, manipulating the dataset by reducing the variables to fit the data is inadvisable as this would likely result in a model that is not generalizable. Thus, future research needs to be conducted on samples with more known-aged individuals to allow the validity of the model presented to be verified. Nevertheless, the model presented here is considered to be an improvement over other methods commonly used (i.e., suture closure).

Based on the results presented above, either the molar-only model or the combined model could be used for predicting age. In this case, the combined model was chosen and was used for predicting ages in unknown-aged individuals using the equations presented on pages 140, 142, and 145 above. However, for some individuals it was necessary to validate the age assignments on a case by case basis due to missing variables (i.e., no skull). Appendix C lists the specimens with known and predicted ages as well as an age category assignment for each individual. While predicted ages were utilized in the analyses, age categories were assigned for comparative purposes. The following age categories were utilized:

Young Adult – category 2 – age up to 19

Middle Adult – category 3 – 20-30

Old Adult – category 4 – 31+

Table 26 below shows the numbers of specimens in each category by species, status, and sex. If the skull was missing, age assignment was based on features of the acetabulum. If the skull and acetabulum were missing and/or unobservable, the specimen was assigned to category 2, which is the more conservative choice as this avoids the possibility of over-estimating age.

Table 26: Age category by species, status, and sex

Species	Sex	Category 2 up to age 19		Category 3 Age 20-30		Category 4 Age 31+	
		Captive	Wild	Captive	Wild	Captive	Wild
Chimp	M	13	12	12	9	4	7
	F	8	12	12	13	6	4
	Total	21	24	24	22	10	11
	?	3					
Gorilla	M	4	9	6	20	7	7
	F	9	11	2	17	1	1
	Total	13	20	8	37	8	8
Orangutan	M	5	1	7	11	8	8
	F	1	6	4	12	6	6
	Total	6	7	11	23	14	14

In chapter 2, initial age categories were assigned based on closure of the basilar suture. Table 27 below shows the initial age assignments for the unknown-aged individuals.

Table 27: Initial Age Assignments for Unknown-aged Individuals

	Chimpanzee Captive	Wild	Gorilla Captive	Wild	Orangutan Captive	Wild
Adult (no skull)	6	7	2	10	2	10
Adult	2	6	0	7	2	13
Old Adult	19	44	6	48	10	21

When age assignments are compared, those based on the combined molar wear/acetabulum model exhibit results with an age spread that is likely to be more in line with what we might expect to occur naturally.

Chapter 4. Osteoarthritis – Problem, Methods, and Disease Patterns

Research Problem: Osteoarthritis has a long and pervasive history and is one of the most prevalent skeletal diseases in modern human populations. The disease creates debilitating effects in humans and other mammals but, despite the longevity and incidence of the disease, the cause is not completely known and there is no cure. It is, however, known that factors that contribute to the development of OA include age, obesity, genetics, and prior injury. Nevertheless, a common misconception is that OA is due solely to wear and tear. One of the reasons for this misunderstanding is because OA is typically correlated with age; however, this relationship between age and the development of OA merely illustrates that the disease is a process that takes time to develop. A second reason for this misconception is that research, particularly in the 1960s and 1970s, focused on defining the cause of OA as resulting from repetitive stress. While perspectives have shifted away from wear and tear as the sole cause of OA, skeletal analyses focusing on OA have become less common in recent years, even though understanding OA skeletal patterns is important in interpretation of disease (Weiss and Jurmain, 2007).

While osteoarthritis is found in non-human primates, it is thought to be relatively uncommon when compared to the frequency of the disease in modern humans (Jurmain, 2000). The disease has been studied in wild chimpanzee and gorillas skeletons, although studies that compare skeletal patterns and prevalence rates among wild and captive samples have not been undertaken. Because great apes experience many of the same age-related disorders as humans, and because they resemble humans genetically, biologically, and psychologically, research addressing disease processes is particularly important.

As mentioned earlier, the primary goal of this research is to compare the prevalence of osteoarthritis between wild and captive great ape skeletons. Research that addresses whether a difference in prevalence actually exists between wild and captive apes, with age as a factor, is a critical first step because differences in lifestyle can potentially relate directly to causal factors. In addition, variables such sex and interspecific variation also will be examined.

Research Methods: Data were collected on the shoulder, elbow, hip, knee, temporomandibular joint (TMJ), and the vertebral column. To control for the effects of secondary OA resulting from trauma, both the left and right sides were scored for all elements, if present. Thus, if asymmetric involvement is noted, it will indicate the possibility of the presence of secondary OA. Data were collected using ordinal scaling criteria with standards based on data collection methods specified by Buikstra and Ubelaker (1994, pgs 121-123). While some other methods have been developed (eg: Nagy, 2000), the standard used was chosen because it is in wide use and is recognized as very useful (Jurmain, personal communication).

Scoring Criteria: The four major peripheral joints (shoulder, elbow, hip, and knee), TMJ, and interfacetal joints of the spine were scored on both the joint surface and peri-articular surface. Each joint surface on each element was scored based on relevant anatomical features shown in table 28 below (it should be noted that the shoulder comprises two joints with separate joint capsules):

Table 28: Anatomical Features Scored on Each Joint

Joint	Bone	Feature
TMJ	Mandible Temporal	Mandibular Condyle Mandibular fossa
Shoulder (1)	Clavicle Scapula	Acromion facet Acromion process
Shoulder (2)	Scapula Humerus	Glenoid fossa Head
Elbow	Humerus Ulna Radius	Capitulum Trochlea Olecranon fossa Radial notch Olecranon process Trochlear notch Coronoid process Head
Hip	Os Coxae Femur	Acetabulum Head
Knee	Femur Patella Tibia	Medial condyle Lateral condyle Patellar surface Intercondylar fossa Facet Medial condyle Lateral condyle
Apophyseal	Vertebrae	Superior facet Inferior facet

The maximum score for any feature within a joint was assigned to the joint as a whole.

This scoring system was utilized because using prevalence scores (that indicate presence anywhere within a joint) is preferable to an average as they are a more accurate indicator of the actual prevalence of arthritis within a population (Bridges, 1993). Scoring of pitting at the peri-articular margins, and surface porosity, osteophytes, and eburnation are based on the scale shown in table 29 below:

Table 29: Skeletal Marker Scoring Criteria

Skeletal Marker	Scoring Criteria
Marginal Lipping	0 = none 1 – slight – small, elevated margin 2 – moderate – elevated spicules, may be irregular in size 3 – severe – extensive, curved spicules
Surface Osteophytes	0 = none 1 = slight – barely discernible 2 = moderate – regular, clearly discernible 3 = severe – irregular, clearly discernible
Surface Porosity	0 = none 1 = slight – microporosity only (pin-prick sized) 2 = moderate – macroporosity 3 = severe – micro and macroporosity – coalescence
Surface Eburnation	0 = none 1 = slight – barely discernible polish 2 = moderate – clearly discernible, polish only 3 = severe – polish with grooves and/or polish with secondary porosity/bone destruction

If present, the extent of each criterion was scored as follows: 1 = ($< 1/3$); 2 ($1/3$ - $2/3$); and 3 ($> 2/3$). The maximum score for each criterion within a joint was assigned to the joint as a whole.

The superior and inferior margins of vertebral bodies were scored for severity and extent of lipping (as mentioned previously, this is not true OA). For the vertebral bodies, scoring of the presence of lipping/osteophytes is based on the following scale: 0 = none/trace; 1 = slight (elevated ring); 2 = moderate (spicules); 3 = severe (curved spicules/fusion). If present, the extent of lipping was scored as follows: 1 = ($< 1/3$); 2 ($1/3$ - $2/3$); and 3 ($> 2/3$). The score for each vertebral segment (cervical, thoracic, lumbar) is taken as the maximum score on any of the elements.

In the spine, there are a number of diseases that affect the vertebral bodies. These spinal diseases (three of which are discussed below) are generally distinguished by location and appearance; although, they can be present at the same time.

Vertebral Osteophytosis (VOP) – Degeneration of the intervertebral discs leads to contact between the vertebrae. This contact between the vertebrae stimulates osteophytes to form along the vertebral margins. These osteophytes may eventually become large enough to fuse adjoining vertebrae resulting in osseous ankylosis. The lower cervical, lower thoracic, and lower lumbar are the most frequently affected spinal segments (Aufderheide and Rodriguez-Martin, 1998).

Diffuse Idiopathic Skeletal Hyperostosis (DISH) – Ossification of ligaments leads to ankylosing of the spine. There is fusion of at least four vertebrae, usually on the right side only. This disease presents as a flowing calcification on the anterolateral aspect, most commonly in the mid thoracic region, and often referred to as having a ‘candle wax’ appearance. In humans, the disease rarely occurs before the age of 40 (Aufderheide and Rodriguez-Martin, 1998).

Ankylosing Spondylitis – Joint inflammation and surface erosion leads to bony ankylosis that generates a bamboo-like appearance (segmented). In humans, age of onset is usually between 15 and 35 years. Prior to 1981, ankylosing spondylitis and DISH were not separated (Aufderheide and Rodriguez-Martin, 1998).

As discussed above, the typical features of osteoarthritis include lipping (bony spurs or osteophytes), porosity, osteophytes (which reflect the addition of compact bone to the joint surface) and eburnation (bone-on bone polish that develops following degeneration of the cartilage). Changes to peri-articular surfaces were analyzed

separately from joint surface changes because it is now recognized that marginal changes (i.e., osteophytes at the peri-articular edges of a joint) may be influenced by factors other than those which affect the joint surface. Recent clinical investigations, such as genetic information from twin studies and experimental work give additional support to this view (Weiss and Jurmain, 2007).

While porosity, surface osteophytes, and eburnation were scored for all joint surfaces, the focus of analysis is on the presence of eburnation only. The reason for placing the emphasis on eburnation only is discussed in chapter 1; however, a review of the information presented earlier follows. Eburnation is universally accepted as diagnostic of OA in both clinical and osteological fields, while porosity and surface osteophytes are of uncertain diagnostic value. Some research indicates that osteophytes develop in relation to biological aging and thus may not be reliable as an indicator of disease severity (Weiss and Jurmain, 2007). Research now indicates that porosity may be unrelated to osteoarthritis, and may occur independently from eburnation (Wood, 1995; Rothschild, 1997; Weiss and Jurmain, 2007). Rothschild (1997) found that “no significant relationship exists between porosity and osteoarthritis” and that “eburnation and porosity are unrelated.” He further recommended that porosity should not be used as an identifier of osteoarthritis. Porosity, while frequently utilized in OA evaluation, remains poorly defined. There are at least three different pathways by which porosity occurs: (1) thinning of the articular plate exposing vascular channels (probably not related to OA); (2) active vascular invasion of calcified cartilage (may be related to OA); and (3) perforation through the articular plate *subsequent* to eburnation (Jurmain, 1999). These different pathways produce different types of ‘holes’ that can be difficult to

differentiate. In addition, some researchers recommend that “...for most analyses OA should be assessed *solely* on the basis of eburnation” (Jurmain, 1999). Thus, while it is prudent to collect and record the maximum amount of data, eburnation is the only criterion that is universally accepted as truly indicative of severe OA and is the primary focus in this analysis.

To test what relationship exists, if any, among the joint markers used in this study, correlation analyses were utilized. A correlation is a measure of the linear relationship between variables and variables can be related in several ways, either positively, negatively, or not related at all. For this study, Kendall’s tau correlation coefficients were utilized. This method was chosen because Kendall’s tau is a non-parametric correlation that is used when the data set is small with a large number of tied ranks. It is also suggested that Kendall’s tau is a better estimate of the correlation in a population than other methods commonly used (such as Spearman’s) and that more accurate generalizations can be drawn from Kendall’s statistic than from Spearman’s (Field, 2005). Correlations were performed for all three species on each of the four major joints (shoulder, elbow, hip, and knee). Left and right sides of each joint were analyzed separately. Appendix D shows the correlation matrix results for all three species, while table 30 below summarizes the total number of significant or non-significant correlations between eburnation and each of the other joint markers.

Table 30: Number of significant or non-significant correlations between eburnation and the other joint markers

	Marginal Lipping	Porosity	Osteophytes
Significant	16	13	13
Not Significant	5	8	8

The results of the correlations between eburnation and the other OA markers (marginal lipping, porosity, and osteophytes) are mixed. The results tend to be either: low in magnitude, positive, significant; low in magnitude, positive, not significant; or low in magnitude, negative, not significant. There are also cases where correlations were not computed and this is due to empty cells within the matrix (i.e. absence of eburnation in any specimen). In general, of those correlations that are significant, the correlations are weak. Thus, given the results obtained from the correlation analyses and the information garnered from other research (discussed above and in chapter 1), eburnation is the primary criterion utilized in this study to diagnose the presence of OA in the four major joints, the TMJ, and the vertebral apophyseal joints.

Disease Patterns and Frequency: The remainder of this chapter describes the pattern of osteoarthritis, as diagnosed by the presence of eburnation, in each species. Statistical analyses and the results from these analyses will be discussed in chapter 5. As noted above, the focus is on the presence of eburnation in the joints and on the presence of lipping at the vertebral bodies. Each species will be discussed separately and example photographs are provided, where appropriate. All photographs were taken by the author, with credit annotated where appropriate. Photographs taken at the Museum of

Comparative Zoology (MCZ) are copyrighted as © President and Fellows of Harvard College. Permission was granted to publish these photographs in this dissertation and a copy of the letter of approval is included in Appendix E.

Chimpanzees: The sample consisted of 115 chimpanzees, of which 58 were captive and 57 were wild. There were a total of 56 males, 58 females, and 3 of uncertain sex. Of the captive chimpanzees, 27 were male, 28 were female, and 3 were of uncertain sex. Of the wild chimpanzees, 29 were male and 28 were female.

Osteoarthritis: Among the chimpanzee sample, the incidence of osteoarthritis, diagnosed by the presence of eburnation, is uniformly low. The captive and wild samples will be discussed separately as follows:

Captive Chimpanzee OA: Table 31 below shows the distribution of eburnation in the captive sample (note: the vertebrae details listed in this table are for apophyseal OA only):

Table 31: Presence of Eburnation in Captive Chimpanzees

Joint	Side	# individuals scored	# with eburnation	% affected
TMJ	Left	53	1	1.89
	Right	53	1	1.89
Shoulder	Left	58	0	
	Right	56	0	
Elbow	Left	57	0	
	Right	55	0	
Hip	Left	57	0	
	Right	56	0	
Knee	Left	55	1	1.82
	Right	54	1	1.86
Cervical	Left	53	0	
	Right	53	1	1.89
Thoracic	Left	50	0	
	Right	50	0	
Lumbar	Left	50	0	
	Right	50	0	

Three captive chimpanzees exhibited osteoarthritis as follows:

1. Specimen Number Indiana University (IU) 9910098: Male chimpanzee, predicted age 14, primary osteoarthritis in the left and right TMJ. The left and right mandibular condyles and mandibular fossae show signs of slight eburnation on $< 1/3$ of each surface. This individual demonstrated slight and moderate lipping of the thoracic (T10) and lumbar (L2-sacrum) vertebral bodies.
2. Specimen Number University of New Mexico (UNM) 2006.61.1: Male chimpanzee, age 30, primary osteoarthritis in the left and right knee. Both the left and right femora and the left and right tibiae exhibited moderate eburnation of moderate extent ($1/3$ - $2/3$) on the lateral condyles. This individual showed signs of slight lipping of the superior body of C7.

Figure 75: Specimen Number UNM 2006.61.1 - Right Distal Femur



Specimen Number UNM 2006.61.1: Right posterior distal femur. Eburnation (with marginal lipping and porosity) on lateral condyle

3. Specimen Number IU 10198: Female chimpanzee, age 42, primary osteoarthritis in the right cervical apophyseal facets. The right inferior facet of C1 exhibited moderate eburnation of moderate extent, while the right superior facet of C2 demonstrated severe eburnation of moderate extent (polish with grooves). This individual displayed slight and moderate lipping of the majority of the cervical, thoracic, and lumbar vertebral bodies.

Figure 76: Specimen Number IU 10198 - L3



Specimen Number IU 10198:
Lipping on superior and
inferior body of L3

Two other captive chimpanzees were notable as follows:

1. Specimen Number IU 9510332: Female chimpanzee, predicted age 29, right clavicle, scapula, and humerus malformed. The left and right clavicles and scapulae differed in size, while the right scapula and right humerus were malformed. While eburnation was not evident, the shoulder joint was significantly altered.

Figure 77: Specimen Number IU 9510332 - Clavicles



Specimen Number IU 9510332:
Clavicles – note difference in size

Figure 78: Specimen Number IU 9510332 - Right Scapula



Specimen Number IU 9510332:
Right scapula – note deformity
to glenoid cavity (indicated by
arrow)

Figure 79: Specimen Number IU 9510332 - Right Humerus



Specimen Number IU
9510332: Right proximal
humerus

2. Specimen Number Primate Foundation of Arizona (PFA) 2012: Female chimpanzee, age 34, deformed right femur and tibia. This individual was

originally a night club performer who, at a very young age, was given Progerone to retard her growth. The right hindlimb is approx 7.62cm shorter than the left. The facility that housed this individual, when she was ‘retired’ from performing, was informed that surgery had been completed to correct a congenital knee deformity. This individual displayed some marginal lipping in some joints, although not of severe levels or extent. Her skull, particularly the mandible and maxilla, also showed some evidence of deformity.

Figure 80: Specimen Number PFA 1012 – Right Proximal Tibia



Specimen Number PFA 1012:
Proximal anterior right tibia –
note deformity to condyles

Figure 81: Specimen Number PFA 1012 – Right Distal Femur



Specimen Number PFA
1012: Right lateral distal
femur: note the metal rod
surgically inserted in femur
(indicated by the white
arrow) and deformity of
condyles

Figure 82: Specimen Number PFA 1012 – Left and Right Leg Bones



Specimen Number PFA 1012:
Left and right leg bones – note
difference in size and length

Wild chimpanzee OA: Table 32 below shows the distribution of eburnation in the wild sample (note: the vertebrae details listed in the table are for apophyseal OA only):

Table 32: Presence of Eburnation in Wild Chimpanzees

Joint	Side	# individuals scored	# with eburnation	% affected
TMJ	Left	50	0	
	Right	50	0	
Shoulder	Left	57	1	1.75
	Right	57	2	3.51
Elbow	Left	55	2	3.64
	Right	57	2	3.51
Hip	Left	57	0	
	Right	57	1	1.75
Knee	Left	57	0	
	Right	57	1	1.75
Cervical	Left	57	0	
	Right	55	0	
Thoracic	Left	56	0	
	Right	56	0	
Lumbar	Left	50	0	
	Right	50	0	

Five wild chimpanzees exhibited osteoarthritis as follows:

1. Specimen Number American Museum of Natural History (AMNH) 201469:

Female chimpanzee, predicted age 37, secondary OA in right shoulder. The right glenoid cavity of the scapula exhibited porosity, on $>2/3$ of the surface, that is probably secondary to eburnation. Due to the degeneration of the glenoid cavity, the maximum score was attributed to this element. The right humeral head displayed severe eburnation (with secondary porosity) over $<1/3$ of the surface. This osteoarthritis is judged to be secondary OA, associated with a healed fracture that is clearly evident on the humeral shaft at approximately 6cm superior to the distal epiphyses. Lipping was absent in the vertebral elements.

Figure 83: Specimen Number AMNH 201469 – Right Distal Humerus



Specimen Number AMNH 201469: Right distal posterior humerus – healed fracture on the humeral shaft

Figure 84: Specimen Number AMNH 201469 – Right Proximal Humerus



Specimen Number
AMNH 201469: Right
proximal humeral head.
Eburnation (with
secondary porosity)

Figure 85: Specimen Number AMNH 201469 – Right Glenoid Cavity



Specimen Number
AMNH 201469: Right
glenoid cavity. Porosity
(most likely secondary
to eburnation) and minor
erosion of cavity

2. Specimen Number Museum of Comparative Zoology (MCZ) 26849: Female chimpanzee, predicted age 28, primary osteoarthritis of both the left and right shoulders and elbows. In the shoulder, the glenoid cavity of the left scapula exhibited moderate eburnation on $< 1/3$ of the surface, while the glenoid cavity of the right scapula demonstrated severe porosity on $1/3$ - $2/3$ of the surface that is likely secondary to eburnation. The left humeral head displayed slight eburnation on $< 1/3$ of each surface, while the right humeral

head displayed moderate eburnation on $< 1/3$ of the surface. In the elbow, the trochlea of the left humerus exhibited moderate eburnation over $< 1/3$ of the surface, while the trochlea of the right humerus exhibited severe eburnation (with secondary porosity) over $1/3$ - $2/3$ of the surface. The coronoid processes of the left and right ulnae demonstrated severe eburnation (with grooves) over $>2/3$ of each surface. This individual also showed evidence of alteration to the left knee joint, with severe marginal lipping and deformation of the lateral femoral condyle, although no eburnation was evident. The left tibia and fibula were fused proximally and the tibia displayed deformation to the condyles, although no eburnation was evident. The right limb bones did not exhibit any sign of alteration at the knee joint. Slight and moderate lipping was present on the vertebral bodies of C3-C6 and L1-sacrum.

Figure 86: Specimen Number MCZ 26849 – Right Glenoid Cavity



Specimen Number MCZ
26849: Right glenoid cavity -
eburnation (with secondary
porosity).
Museum of Comparative
Zoology, Harvard University

Figure 87: Specimen Number MCZ 26849 – Right Humeral Head



Specimen Number MCZ
26849: Right humeral head
– eburnation, with porosity
Museum of Comparative
Zoology, Harvard
University

Figure 88: Specimen Number MCZ 26849 – Right Distal Humerus



Specimen Number MCZ
26849: Right anterior distal
humerus - eburnation (with
porosity) on trochlea
Museum of Comparative
Zoology, Harvard University

Figure 89: Specimen Number MCZ 26849 – Left Proximal Ulna



Specimen Number MCZ 26849:
Left coronoid process of ulna –
eburnation, with grooves
Museum of Comparative
Zoology, Harvard University

Figure 90: Specimen Number MCZ 26849 – Left and Right Distal Femora



Specimen Number
MCZ 26849: Distal
anterior left and right
femora – note
deformity to left
femoral condyles
Museum of
Comparative Zoology,
Harvard University

3. Specimen Number British Museum of Natural History (BMNH) 1861.7.49.14:

Female chimpanzee, predicted age 11, primary osteoarthritis of the left and right elbows. The trochlea of both the left and right humeri exhibited moderate eburnation on 1-3/2/3 of each surface. The olecranon processes of both the left and right ulnae demonstrated moderate eburnation covering $< 1/3$ of each surface. The coronoid processes of the left and right ulnae displayed severe eburnation (with grooves) covering $> 2/3$ of each surface. Slight and moderate lipping was evident on the vertebral bodies of C3-C7.

Figure 91: Specimen Number BMNH 1861.7.49.14 – Right Distal Humerus



Specimen Number BMNH
1861.7.49.14: Right distal
anterior humerus –
eburnation (with
porosity) on trochlea

4. Specimen Number National Museum of Natural History (NMNH) 176229:
Female chimpanzee, predicted age 19, possible secondary osteoarthritis of the right knee. The right femoral medial and lateral condyles exhibited slight eburnation on $< 1/3$ of the surface. No other anomalies were noted and the joint was otherwise healthy. Lipping was absent from the vertebral elements.

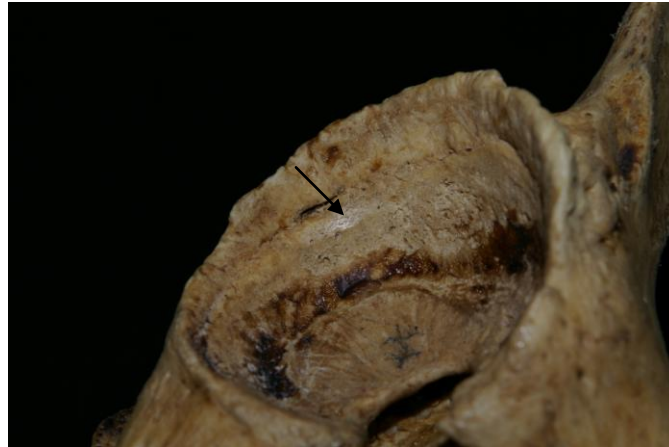
Figure 92: Specimen Number NMNH 176229 – Right Distal Femur



Specimen Number
NMNH 176229:
Eburnation on right
femoral condyles

5. Specimen Number BMNH 1951.9.27.8: Male chimpanzee, predicted age 38, secondary osteoarthritis of the right hip. The right acetabulum exhibited slight eburnation on $< 1/3$ of the lunate surface, while the right femoral head demonstrated moderate eburnation on $< 1/3$ of the surface. There was no obvious sign of trauma to the lower limb bones; however, the asymmetric involvement means that this is likely secondary OA. Slight and moderate lipping was evident on the vertebral bodies of C3-C6.

Figure 93: Specimen Number BMNH 1951.9.27.8 – Right Acetabulum



Specimen Number
BMNH 1951.9.27.8:
Eburnation on right
acetabular lunate
surface

Vertebral Body Lipping: Among the chimpanzee sample, the presence of lipping on the superior and inferior rims of the vertebral elements is relatively low. The captive and wild samples will be discussed separately as follows:

Captive chimpanzees: Lipping in the cervical, thoracic, and lumbar vertebrae is more common than joint OA; however, the incidence of severe lipping is uniformly low. In the cervical vertebrae, of the 54 individuals examined, 13 (24.1%) exhibited some form of lipping. Of these, 10 (18.5%) displayed slight lipping, 2 (3.7%) demonstrated moderate lipping, and only 1 (1.8%) exhibited severe lipping. In the thoracic vertebrae, 12 of 49 individuals (24.5%) showed evidence of lipping. Of these 6 (12.2%) displayed slight lipping, 6 (12.2%) demonstrated moderate lipping, and none exhibited severe lipping. In the lumbar vertebrae, 16 of 48 individuals (33.3%) showed evidence of lipping. Of these, 3 (6.2%) displayed slight lipping, 11 (22.9%) demonstrated moderate lipping, and 2 (4.2%) exhibited severe lipping.

Wild chimpanzees: Lipping in the cervical, thoracic, and lumbar vertebrae is uniformly low in wild chimpanzees. In the cervical vertebrae, of the 54 individuals

examined, 5 (9.3%) displayed some form of lipping. Of these, 2 (3.7%) demonstrated slight lipping, 3 (5.5%) displayed moderate lipping, and none exhibited severe lipping. In the thoracic vertebrae, 3 of 51 individuals (5.9%) showed evidence of lipping. Of these, 1 (2%) displayed slight lipping, 1 (2%) demonstrated moderate lipping, and 1 (2%) exhibited severe lipping. In the lumbar vertebrae, 3 of 48 individuals (6.3%) showed evidence of lipping. Of these, 1 (2.1%) displayed slight lipping, 1 (2.1%) demonstrated moderate lipping, and 1 (2.1%) exhibited severe lipping.

Summary: As mentioned previously, for all three species, the statistical analyses and results are presented in chapter 5, while a summary of the descriptive data discussed in this chapter follows. In chimpanzees, severe primary osteoarthritis is rare in both captive and wild individuals. Captive individuals did not exhibit obvious secondary osteoarthritis and, although evident in wild individuals, secondary OA is also rare. Only three captive individuals showed signs of joint OA, each in a different joint (TMJ, knee, cervical apophyseal joints), while five wild individuals exhibited joint OA, two with primary and three with secondary OA. In wild individuals, the shoulder and elbow joints demonstrated primary OA, while the shoulder, hip, and knee exhibited secondary OA. Thus, of the four major joints (shoulder, elbow, hip, and knee), captive individuals showed signs of OA only in the knee, while wild individuals evidenced OA in the shoulder and elbow. The distribution of disease expression in the wild and captive sample is in contrast to previous research. Rothschild and Woods (1992) found the elbow to be the most commonly affected joint in captive Old World monkeys and gibbons, while knees were affected most frequently in wild individuals. Other research has found that wild chimpanzees were most often affected in the knee (Woods, 1986;

Jurmain, 1989). While overall rates of severe OA are uniformly low in both wild and captive samples, the differences in distribution might suggest that compressive stress and load bearing could be factors in disease expression; however, given the contrast in results between this and earlier studies, this is unlikely to be the case for chimpanzees. Indeed, given the overall low rates of osteoarthritis exhibited in the chimpanzee sample, differences in disease expression and status (captive vs wild) appear to be of minimal interpretative value.

For spinal lipping, captive chimpanzees showed a higher rate of both slight and moderate involvement than do wild chimpanzees in all three vertebral segments. Both wild and captive individuals demonstrate low incidence of lipping for extreme involvement, with captive specimens exhibiting severe lipping in the cervical and lumbar regions only and wild individuals demonstrating severe lipping in the thoracic and lumbar regions only. Overall, spinal lipping is relatively low in both the captive and wild sample; however, given that captive chimpanzees demonstrate a higher rate of slight and moderate involvement, this may indicate the likelihood of an increased prevalence in captive individuals (i.e., they did not live long enough to develop severe lipping). Nevertheless, given the age range of the sample, it appears more likely that chimpanzees are not highly prone to develop vertebral body lipping and, if they do, it does not reach severe levels even in old individuals. It also appears that age is not a strong factor in disease development in captive chimpanzees because two of the oldest individuals in the sample (48 and 42 years old) did not show any signs of vertebral body lipping (the 42 year old did have apophyseal OA in the cervical vertebrae). Indeed, the 48-year-old (the

oldest chimpanzee in the captive sample) did not exhibit vertebral body lipping, eburnation, or marginal lipping in any joint.

In terms of sex, of those individuals with eburnation, male captive chimpanzees demonstrated joint OA more frequently than females (2:1); however, the two chimpanzees with notable joint deformities (discussed above) were both female, so this apparent disparity may or may not be true. In wild chimpanzees, of those with eburnation, females displayed more joint OA than males (4:1). Only wild female chimpanzees showed signs of primary joint OA, while the one male in the sample with OA displayed the secondary form of the disease. For vertebral body lipping, 24 captive individuals exhibited some form of lipping with 11 males, 10 females, and 3 of unknown sex affected. In wild chimpanzees, 9 individuals demonstrated some form of vertebral body lipping with 2 males and 7 females affected.

In terms of age, the captive chimpanzees with primary joint OA were aged 14 (predicted), 30, and 42, while the wild chimpanzees with primary joint OA were of predicted ages of 11 and 19. Thus, it would appear that wild individuals with primary OA tend to be younger than captive individuals with primary OA.

Gorillas: The sample consisted of 94 gorillas, of which 29 were captive and 65 were wild. There were a total of 54 males and 41 females. Of the captive gorillas, 17 were male and 12 were female, while of the wild gorillas, 36 were male and 29 were female.

Osteoarthritis: Among the gorilla sample, the incidence of osteoarthritis, diagnosed by the presence of eburnation, is low. The captive and wild samples will be discussed separately as follows:

Captive Gorilla OA: Table 33 below shows the distribution of eburnation in the captive sample (note: the vertebrae details in this table are for apophyseal OA only):

Table 33: Presence of Eburnation in Captive Gorillas

Joint	Side	# individuals scored	# with eburnation	% affected
TMJ	Left	23	0	
	Right	23	0	
Shoulder	Left	26	1	3.84
	Right	27	1	3.70
Elbow	Left	25	1	4.0
	Right	27	1	3.70
Hip	Left	27	1	3.70
	Right	28	1	3.57
Knee	Left	25	2	8.0
	Right	26	2	7.69
Cervical	Left	27	0	
	Right	27	1	3.70
Thoracic	Left	26	0	
	Right	26	0	
Lumbar	Left	23	0	
	Right	23	0	

Three captive gorillas exhibited osteoarthritis as follows:

1. Specimen Number Field Museum (FM) 126045: Male gorilla, aged 38, primary OA of the left and right shoulder, hip, and knee. This individual exhibited eburnation in three of the four major joints and, while eburnation was not present in the elbow joints, all of the elements that form the elbow (humerus, ulna, radius) showed evidence of marginal lipping, porosity, and osteophytes. The only joint that did not show any characteristic of OA was the temporomandibular joint. In general, marginal lipping was severe in this

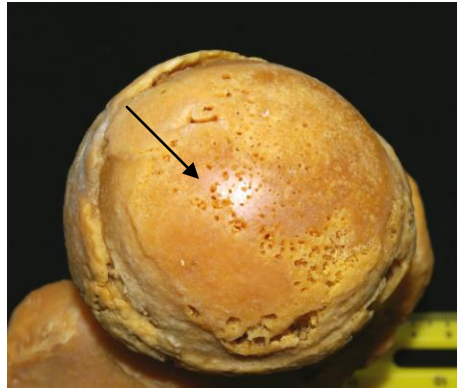
individual. In the shoulder, both the left and right glenoid cavities demonstrated moderate eburnation covering 1-3/2-3 of each surface. The right humeral head displayed moderate eburnation on $< 1/3$ of the surface. In the hip, the left and right acetabulae showed signs of moderate eburnation on 1-3/2-3 of each surface. The left femoral head displayed moderate eburnation on 1/3-2/3 of the surface, while the right femoral head demonstrated moderate eburnation on $> 2/3$ of the surface. In the knee, the left femoral medial and lateral condyles exhibited moderate eburnation on $< 1/3$ of each surface. The right femoral medial condyle displayed moderate eburnation on 1/3-2/3 of the surface, while the right lateral condyle demonstrated moderate eburnation on $< 1/3$ of the surface. The left tibial medial and lateral condyles showed signs of moderate eburnation on 1-3/2-3 of each surface, while the right tibial medial and lateral condyles displayed moderate eburnation on $< 1/3$ of each surface. Lipping was slight, moderate, and severe in the all three vertebral segments with complete fusion of T13-L3 and L4-sacrum. The appearance of the fused vertebrae suggests an extreme demonstration of an individual who is a “bone former.”

Figure 94: Specimen Number FM 126045 – Right Glenoid Cavity



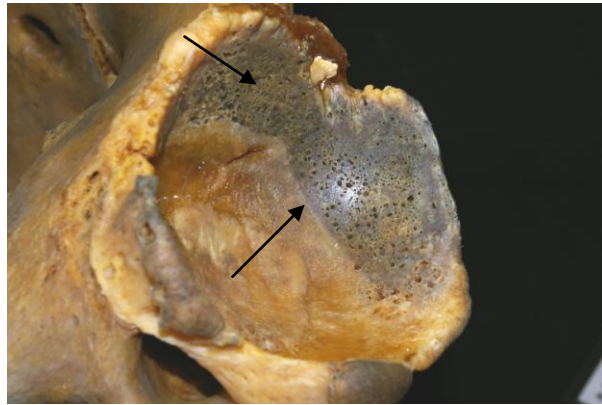
Specimen Number FM
126045: Eburnation in
right glenoid fossa

Figure 95: Specimen Number FM 126045 – Left Femoral Head



Specimen Number FM
126045: Eburnation on
left femoral head – note
severe marginal lipping

Figure 96: Specimen Number FM 126045 – Left Acetabulum



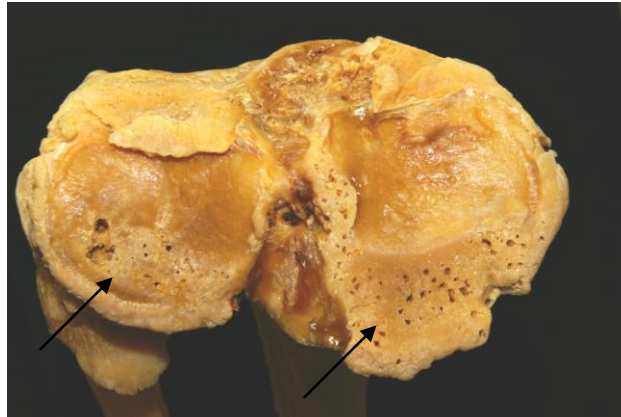
Specimen Number FM
126045: Eburnation of left
acetabulum (with
porosity)

Figure 97: Specimen Number FM 126045 – Left Distal Femur



Specimen Number FM
126045: Eburnation of left
medial condyle of distal
femur – note severe
marginal lipping of medial
condyle

Figure 98: Specimen Number FM 126045 – Right Proximal Tibia



Specimen Number FM
126045: Eburnation of
right tibial condyles (with
porosity)

Figure 99: Specimen Number FM 126045 – T13-L3



Specimen Number FM
126045: Complete fusion
of T13-L3 at vertebral
bodies.

2. Specimen Number BMNH 1978-1226: Male gorilla, age 32, primary OA of the left and right elbows. In the elbows, the left and right radial notch of the ulnae displayed moderate eburnation covering $< 1/3$ of each surface. Vertebral body lipping was present in all segments of the vertebral column. C4-C6 and T3-T11 showed slight and moderate lipping of the vertebral

bodies, the inferior body of T12 displayed severe lipping, T13-L3 were fused, and the sacrum demonstrated moderate lipping.

Figure 100: Specimen Number BMNH 1978-1226 – T13-L3



Specimen Number BMNH
1978-1226: Complete
fusion of T13-L3 at
vertebral bodies.

3. Specimen Number MCZ 62393: Male gorilla, age 38, primary OA of the left and right knees and right cervical apophyseal joints. In the knee, the left and right femoral medial condyles displayed moderate eburnation covering 1-3/2-3 of each surface. The left tibial medial condyle demonstrated moderate eburnation on 1-3/2-3 of the surface, while the right tibial medial condyle exhibited moderate eburnation on > 2/3 of the surface. In the vertebrae, the C1 right inferior facet and C2 right superior facets displayed severe eburnation (with grooves) over > 2/3 of each surface. With regards to vertebral body lipping, C1-C7 showed signs of slight, moderate, and severe

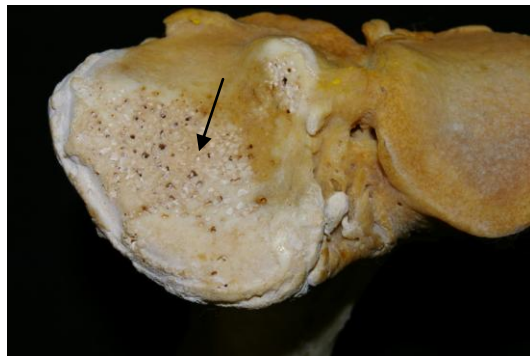
lipping, T1-T11 demonstrated slight and moderate lipping, while T12-L3 was completely fused and L4-sacrum was fused.

Figure 101: Specimen Number MCZ 62393 – Left Distal Femur



Specimen Number MCZ 62393: Left distal femoral condyles with eburnation and porosity on medial condyle.
Museum of Comparative Zoology, Harvard

Figure 102: Specimen Number MCZ 62393 – Left Proximal Tibia



Specimen Number MCZ 62393: Left proximal tibial condyles with eburnation on medial condyle (with porosity).
Museum of Comparative Zoology, Harvard University

Figure 103: Specimen Number MCZ 62393 – C1



Specimen Number MCZ 62393: C1 inferior right articular facet exhibiting eburnation with grooves (and porosity).
Museum of Comparative Zoology, Harvard University

Figure 104: Specimen Number MCZ 62393 – C2



Specimen Number MCZ 62393: C2 superior right articular facet exhibiting eburnation with grooves (and porosity).
Museum of Comparative Zoology, Harvard University

Figure 105: Specimen Number MCZ 62393 – T12-L3



Specimen Number MCZ 62393: Complete fusion of T12-L3 at vertebral bodies.
Museum of Comparative Zoology, Harvard University

One other captive gorilla was notable as follows:

Specimen Number FM 180677: Male gorilla, age 39, complete fusion of T8-sacrum. Only the skull, hip, and partial vertebrae were present. Thus, it is unknown whether this individual would have experienced OA in any of the four major joints.

Figure 106: Specimen Number FM 180677 – T8-Sacrum and Os Coxa



Specimen Number FM 180677:
Complete fusion of T8-sacrum
and os coxa.

Wild Gorilla OA: Table 34 below shows the distribution of eburnation in the wild sample

(note: the vertebrae details listed in this table are for apophyseal OA only):

Table 34: Presence of Eburnation in Wild Gorillas

Joint	Side	# individuals scored	# with eburnation	% affected
TMJ	Left	58	1	1.72
	Right	58	1	1.72
Shoulder	Left	65	0	
	Right	64	0	
Elbow	Left	65	2	3.08
	Right	65	1	1.54
Hip	Left	65	0	
	Right	65	0	
Knee	Left	65	1	1.54
	Right	64	3	4.69
Cervical	Left	62	0	
	Right	62	1	1.61
Thoracic	Left	62	0	
	Right	62	0	
Lumbar	Left	58	0	
	Right	58	0	

Five wild gorillas exhibited osteoarthritis as follows:

1. Specimen Number AMNH 99.9425 (Anthropology Dept): Male gorilla, predicted age 31, primary OA in the left TMJ. The left mandibular condyle and left fossa showed evidence of slight eburnation on $< 1/3$ of surface. As the right TMJ showed some early signs of OA (although not eburnation), this is probably primary in nature. Vertebral body lipping was absent from all of the vertebral elements.
2. Specimen Number AMNH 54355: Male gorilla, predicted age 25, primary OA of the right TMJ, left and right knee, and left and right cervical articular facets. Eburnation was evident in the right elbow and, given the side specificity and lack of diagnostic criteria on elements of the left elbow, this could be attributed to secondary OA; however, given the evidence for primary OA in other joints, this is more likely to be primary in nature. The right mandibular fossa displayed slight eburnation on $< 1/3$ of the surface and, although side specific, other criteria (lipping, porosity, osteophytes) were present on the left side. The trochlea of the right humerus demonstrated severe eburnation on $< 1/3$ of the surface (with grooves), while the coronoid process of the right ulna showed moderate eburnation on $1/3$ - $2/3$ of the surface. The medial condyle of the left femur displayed moderate eburnation on $> 2/3$ of the surface, while the lateral condyle of the right femur demonstrated moderate eburnation on $1/3$ - $2/3$ of the surface. The medial condyle of the left tibia exhibited moderate eburnation on $1/3$ - $2/3$ of the surface, while the lateral condyle of the right tibia showed evidence of

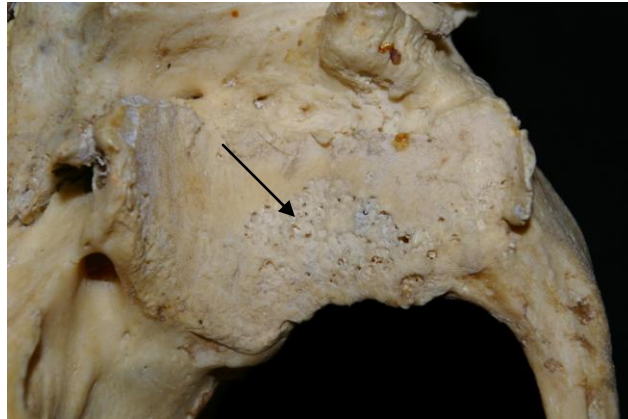
moderate eburnation on $< 1/3$ of the surface. In the cervical region, the fovea of C1 and the dens of C2 demonstrated moderate eburnation on $1/3$ - $2/3$ of each surface, while the inferior right articular facet of C1 showed slight eburnation on $< 1/3$ of the surface and the superior right articular facet of C2 showed evidence of moderate eburnation on $< 1/3$ of the surface. Vertebral body lipping was evident in the cervical vertebrae with slight lipping on C1 and C2. The thoracic vertebrae were also affected with slight lipping on T1-T2, T7-T8, and T10-T13. L1-L3 bodies were unobservable, while L4 demonstrated severe lipping on the inferior body and the sacrum displayed slight lipping.

Figure 107: Specimen Number AMNH 54355 – Left Distal Femur



Specimen Number AMNH 54355: Left distal medial condyle of femur with eburnation (and porosity) covering almost the entire articular surface

Figure 108: Specimen Number AMNH 54355 – Right Mandibular Fossa



Specimen Number AMNH 54355: Right mandibular fossa with eburnation (and porosity)

Figure 109: Specimen Number AMNH 54355 – Left Proximal Tibia



Specimen Number AMNH 54355: The medial condyle of the proximal left tibia with eburnation (and porosity)

Figure 110: Specimen Number AMNH 54355 – L4



Specimen Number AMNH 54355: Significant lipping on inferior body of L4

3. Specimen Number Royal Belgian Institute of Natural Sciences (RBINS) 7503:

Male gorilla, predicted age 32, primary OA in the right knee. Although eburnation was present only on the right side, the evidence of other characteristics (lipping, porosity) on the left side, indicate that primary OA is the more likely cause. The medial condyle of the right femur exhibited moderate eburnation on $< 1/3$ of the surface. For vertebral body lipping, C4-C7 and T1-T11 (with the exception of T8 which was unobservable) displayed slight-moderate lipping. T11-T12 and L1-sacrum were fused.

Figure 111: Specimen Number RBINS 7503 – Right Distal Femur



Specimen Number RBINS 7503: The medial condyle of the right distal femur with eburnation (and porosity)

Figure 112: Specimen Number RBINS 7503 – Thoracic and Lumbar Vertebrae



Specimen Number RBINS
7503: Thoracic and lumbar
vertebrae exhibiting lipping
and complete fusion

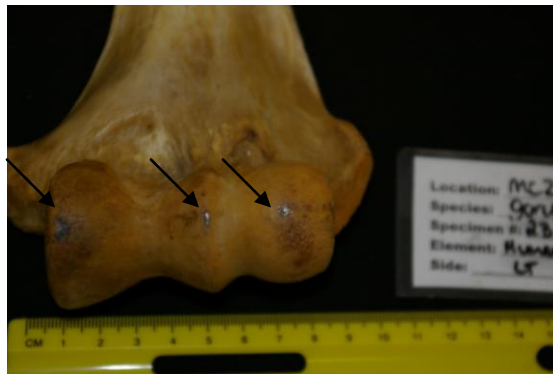
4. Specimen Number MCZ 23160: Male gorilla, predicted age 20, secondary OA of the left elbow and right knee. The capitulum and trochlea of the left humerus showed evidence of slight eburnation on $< 1/3$ of each surface, but were otherwise healthy. The medial and lateral condyles of the right femur exhibited slight eburnation on $< 1/3$ of each surface, but were otherwise healthy. The right femur of this individual demonstrated severe pathology (refer to photographs below). Vertebral body lipping was absent from the vertebral elements.

Figure 113: Specimen Number MCZ 23160 – Right Distal Femur



Specimen Number MCZ
23160: Slight eburnation
on the right femoral
condyles
Museum of
Comparative Zoology,
Harvard University

Figure 114: Specimen Number MCZ 23160 – Left Distal Humerus



Specimen Number MCZ
23160: Slight eburnation
to left distal humerus.
Museum of
Comparative Zoology,
Harvard University

Figure 115: Specimen Number MCZ 23160 – Right Proximal Femur



Specimen Number MCZ
23160: Pathological
right femur
Museum of
Comparative Zoology,
Harvard University

Figure 116: Specimen Number MCZ 23160 – Right Femur



Specimen Number MCZ 23160: Pseudo-arthritis of the right femur likely resulting from an unhealed old fracture in a limb that continued to be used.

Museum of Comparative Zoology, Harvard University

5. Specimen Number MCZ 38017: Male gorilla, predicted age 15, secondary OA of the left elbow. The capitulum of the left elbow displayed slight eburnation on $< 1/3$ of the surface and, given the side specificity and lack of diagnostic criteria on elements of the right elbow, this is attributed to secondary OA.

Slight vertebral body lipping was evident on T2-T4 only.

Figure 117: Specimen Number MCZ 38017 – Left Humerus



Specimen Number MCZ 38017: Eburnation to capitulum of left humerus
Museum of Comparative Zoology, Harvard University

Vertebral Body Lipping: Among the gorilla sample, the presence of lipping on the superior and inferior rims of the vertebral bodies was relatively common. The captive and wild samples will be discussed separately as follows:

Captive gorillas: Lipping in the cervical, thoracic, and lumbar vertebral bodies is evident in over 50% of the captive gorillas. In the cervical vertebrae, of the 28 individuals examined, 16 (57.1%) demonstrated some form of lipping. Of these, 6 (21.4%) displayed slight lipping, 6 (21.4%) exhibited moderate lipping, and 4 (14.3%) demonstrated severe lipping. In the thoracic vertebrae, 20 of 28 individuals (71.4%) showed evidence of lipping. Of these 5 (17.8%) displayed slight lipping, 6 (21.4%) demonstrated moderate lipping, and 9 (32.1%) exhibited severe lipping. In the lumbar vertebrae, 18 of 27 individuals (66.7%) showed evidence of lipping. Of these, 2 (7.4%) displayed slight lipping, 4 (14.8%) demonstrated moderate lipping, and 12 (44.4%) exhibited severe lipping.

Wild gorillas: Lipping in the cervical, thoracic, and lumbar vertebrae is relatively common in wild gorillas. In the cervical vertebrae, of the 62 individuals examined, 19 (30.6%) showed signs of some form of lipping. Of these, 13 (20.9%) displayed slight lipping, 5 (8.1%) demonstrated moderate lipping, and 1 (1.6%) exhibited severe lipping. In the thoracic vertebrae, 27 of 62 individuals (43.6%) showed signs of lipping. Of these, 20 (32.3%) displayed slight lipping, 6 (9.7%) demonstrated moderate lipping, and 1 (1.6%) exhibited severe lipping. In the lumbar vertebrae, 21 of 61 individuals (34.4%) showed evidence of some form of lipping. Of these, 5 (8.2%) displayed slight lipping, 5 (8.2%) demonstrated moderate lipping, and 11 (18%) exhibited severe lipping.

Summary: Captive individuals did not exhibit secondary OA, while secondary OA was evident in two wild gorillas. Only three captive individuals showed evidence of joint OA, with the knee being the most commonly affected joint. The joints affected in captive individuals include the cervical apophyses, shoulders, elbows, hips, and knees. Of the three captive individuals with primary OA of the joints, one individual demonstrated severe OA in three of the four main joints (shoulder, hip, and knee). This individual also had complete fusion in T13-L2. In wild individuals, the elbow and knee joints are most commonly affected with both primary and secondary OA evident in both joints. OA was also evident in the TMJ of two wild individuals, but was not seen in the captive sample. While overall rates of severe OA are uniformly low in both wild and captive samples, differences in distribution might suggest that compressive stress and load bearing could be factors in disease expression; however, given the fact that both knee and elbow OA is seen in both captive and wild individuals, this is unlikely to be the case for gorillas.

For vertebral body lipping, captive and wild gorillas demonstrate a high rate of involvement. In both wild and captive gorillas the incidence of severe lipping is seen most often in the lumbar region with complete fusion being relatively common. In stark contrast to chimpanzees, vertebral body lipping is common in both captive and wild gorillas. In terms of sex, all three captive gorillas with joint OA and all 5 wild gorillas with either primary or secondary joint OA were male. For vertebral body lipping, 22 captive individuals showed evidence of some form of lipping with 15 males and 7 females affected. In wild gorillas, 31 individuals displayed some form of lipping with 19 males and 12 females affected.

In terms of age, the captive gorillas with primary joint OA were aged 32, 38, 38, and 39, while the wild gorillas had predicted ages of 31, 25, 32 (primary) and 20, 15 (secondary). Thus, the ages for those individuals with primary OA are comparable in both the wild and captive sample.

Orangutans: The sample consisted of 75 orangutans, of which 31 were captive and 44 were wild. There were a total of 43 males and 35 females. Of the captive orangutans, 20 were male and 11 were female, while of the wild orangutans, 20 were male and 24 were female. There is disparity between the numbers of male versus female captive animals, with approximately 64% being male; however, as explained previously, bias towards one or other sex was unavoidable.

Osteoarthritis: Among the orangutan sample, the incidence of osteoarthritis, diagnosed by the presence of eburnation, is uncommon in the wild sample and appears to be more prevalent in the captive sample. The captive and wild samples will be discussed separately as follows:

Captive Orangutan OA: Table 35 below shows the distribution of eburnation in the captive sample (note: the vertebrae details are for apophyseal OA only):

Table 35: Presence of Eburnation in Captive Orangutans

Joint	Side	# individuals scored	# with eburnation	% affected
TMJ	Left	27	0	
	Right	27	1	3.73
Shoulder	Left	30	3	10
	Right	30	3	10
Elbow	Left	30	0	
	Right	28	3	10.71
Hip	Left	30	0	
	Right	31	0	
Knee	Left	29	3	10
	Right	29	4	13.79
Cervical	Left	27	0	
	Right	27	0	
Thoracic	Left	25	0	
	Right	25	0	
Lumbar	Left	24	0	
	Right	24	0	

Seven captive orangutans exhibited osteoarthritis as follows:

1. Specimen Number University of Arkansas at Little Rock (UALR) 9310878:

Female orangutan, age 30, primary OA of the knees. The medial condyles of the left and right femora and the medial condyle of the left tibia exhibited moderate eburnation on < 1/3 of each surface. In the spine, moderate to severe lipping was evident in C2-C7 with C4-C7 being fused. In the thoracic vertebrae, T1 displayed severe lipping, while T3-T10 demonstrated slight lipping. T11 (inferior) and T12 (superior) exhibited severe lipping. In the lumbar vertebrae, L1-sacrum is fused. The side specificity of the fusion seen in the lumbar region is indicative of DISH; however, the location and appearance are not completely in accord with this diagnosis. It is likely the fusion in this region is a result of ligamentous ossification.

Figure 118: Specimen Number UALR 9310878 – Left Distal Femur



Specimen Number UALR 9310878: Eburnation on lateral and medial condyle of left femur

Figure 119: Specimen Number UALR 9310878 – C4-C7



Specimen Number UALR 9310878: Fusion of C4-C7

Figure 120: Specimen Number UALR 9310878 – L1-Sacrum



Specimen Number UALR 9310878: Fusion of L1-sacrum, most likely a result of ligamentous ossification.

2. Specimen Number California Academy of Sciences (CAS) 3733: Male orangutan, age 32, primary OA of the right TMJ. The right mandibular condyle and right mandibular fossa displayed moderate eburnation of $< 1/3$ of each surface. Given the presence of other diagnostic criteria (lipping, osteophytes, porosity), it is probable that this OA is primary in nature. In the vertebral column, the cervical bodies demonstrated slight, moderate, or severe lipping. The thoracic vertebrae were missing, while the lumbar vertebrae exhibited moderate or severe lipping.

Figure 121: Specimen Number CAS 3733 – L2



Specimen Number CAS 3733: Severe lipping on the inferior body of L2

3. Specimen Number FM 168868: Male orangutan, age 36, primary OA of the shoulders, right elbow, and right knee. In the shoulder, the left glenoid cavity and left humeral head demonstrated moderate eburnation on $< 1/3$ of the surface, while the right glenoid cavity and right humeral head displayed moderate eburnation on $1/3$ - $2/3$ of the surface. In the right elbow, the capitulum of the humerus revealed moderate eburnation on $1/3$ - $2/3$ of the surface, while the radial head showed moderate eburnation on $> 2/3$ of the surface. In the right knee, the medial condyle of the femur exhibited moderate

eburnation on 1/3-2/3 of the surface, while the medial condyle of the tibia displayed moderate eburnation on < 1/3 of the surface. Although only the right elbow and knee showed evidence of eburnation, due to the presence of other diagnostic criteria (lipping, osteophytes, and porosity) on elements of the left side, this OA is considered to be primary. In addition, the primary OA demonstrated by the shoulders lends weight to this conclusion. In the vertebrae, slight, moderate, or severe lipping was present in all elements. In particular, C7-T1 were fused, while the lumbar displayed severe lipping.

Figure 122: Specimen Number FM 168868 – Right Humerus



Specimen Number FM
168868: Eburnation on the
capitulum of the right
humerus

Figure 123: Specimen Number FM 168868 – Right Radial Head



Specimen Number FM 168868: Eburnation on the right radial head – approximately $\frac{3}{4}$ of surface displays polish

Figure 124: Specimen Number FM 168868 – C7-T1



Specimen Number FM 168868: Severe lipping with fusion of C7-T1

4. Specimen Number UALR 1995-01: Female orangutan, age 38, primary OA of the knees. In the knees, the left medial and lateral condyles of the femur displayed moderate eburnation on $\frac{1}{3}$ - $\frac{2}{3}$ of each surface. The medial condyle of the right femur demonstrated moderate eburnation on $> \frac{2}{3}$ of the surface. In the Tibia, the left and right medial condyles and the left lateral condyle exhibited moderate eburnation on $\frac{1}{3}$ - $\frac{2}{3}$ of each surface. In the

vertebrae, C1-C5 showed signs of slight or moderate lipping, while all thoracic vertebrae had slight, moderate, or severe lipping. T7-T8 were fused, as were T11-T12. In the lumbar region, moderate and severe lipping was evident with L3-L4 being fused.

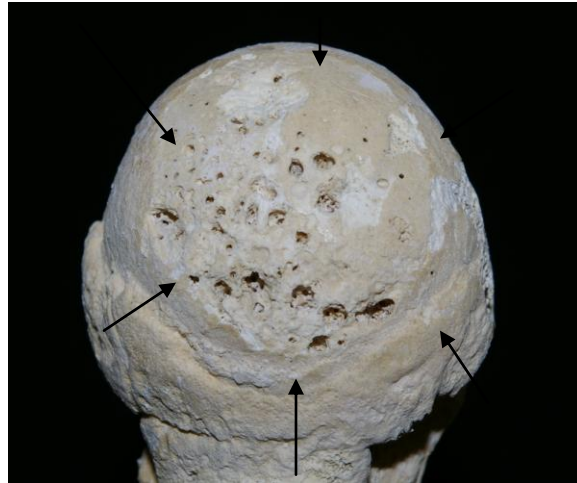
Figure 125: Specimen Number UALR 1995-01 – Right Distal Femur



Specimen Number UALR 1995-01: Eburnation (with porosity) on the right medial condyle of the femur

5. Specimen Number FM 160018: Female orangutan, age 45, primary OA of the shoulder. In the shoulder, the left and right glenoid cavities and humeral heads demonstrated breakdown of the joint surface with eburnation and secondary porosity. In the vertebrae, slight, moderate, or severe lipping was evident in all vertebral segments.

Figure 126: Specimen Number FM 160018 – Right Humeral Head



Specimen Number FM 160018: Eburnation (with porosity) on the right humeral head – arrows indicate the extent of involvement

Figure 127: Specimen Number FM 160018 – Left Glenoid Cavity



Specimen Number FM 160018: Eburnation (with porosity) on the left glenoid cavity with surface destruction

Figure 128: Specimen Number FM 160018 – Lower Spine



Specimen Number FM 160018: Fusion in the lower spine, most likely a result of ossification of the vertebral ligament.

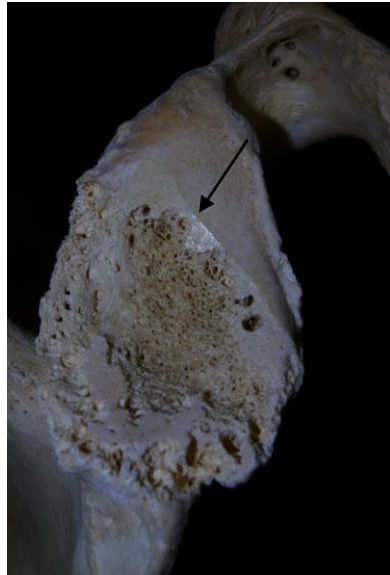
6. Specimen Number FM 153745: Male orangutan, predicted age 29, primary OA of the shoulders, right elbow, and knees. In the shoulders, the left and right elements demonstrated destruction of the surfaces with eburnation and severe secondary porosity. The left glenoid cavity displayed moderate eburnation on 1/3-2/3 of the surface, while the right glenoid cavity exhibited severe eburnation on $> 2/3$ of the surface. The left and right humeral heads demonstrated moderate eburnation on 1/3-2/3 of each surface. In the right elbow, the capitulum and radial head displayed moderate eburnation on $< 1/3$ of each surface. This is unlikely to be secondary OA due to the presence of diagnostic criteria on the left side and the presence of primary OA in other joints. In the knees, the left lateral condyle of the femur showed severe eburnation on $< 1/3$ of the surface, while the left and right lateral condyles of the tibia exhibited moderate eburnation on $< 1/3$ of each surface (both patellae were missing). In the vertebral column, all segments showed evidence of slight, moderate, or severe lipping, but without fusion of any elements.

Figure 129: Specimen Number FM 153745 – Right Humeral Head



Specimen Number FM 153745:
Eburnation (with secondary
porosity) on the right humeral
head. The arrows indicate the
extent of the eburnation.

Figure 130: Specimen Number FM 153745 – Right Glenoid Cavity



Specimen Number FM 153745:
Eburnation (with extensive secondary porosity) of the right glenoid cavity. Evidence of polish is seen only at the margin of the area where the surface is destroyed.

Figure 131: Specimen Number FM 153745 – Left Distal Femur



Specimen Number FM 153745: Eburnation on left lateral condyle of femur (with extensive secondary porosity).

7. Specimen Number UNM P-31: Female orangutan, age 14, secondary OA of right elbow. Complete fusion of right humerus and ulna at the elbow joint due to prior fracture. Right radial head deformed.

Figure 132: Specimen Number FM 153745 – Right Elbow



Specimen Number
UNM P-31: Complete
fusion of right radius
and ulna

Wild Orangutan OA: Table 36 below shows the distribution of eburnation in the wild sample (note: the vertebrae details are for apophyseal OA only):

Table 36: Presence of Eburnation in Wild Orangutans

Joint	Side	# individuals scored	# with eburnation	% affected
TMJ	Left	33	0	
	Right	34	0	
Shoulder	Left	44	0	
	Right	44	0	
Elbow	Left	44	1	2.27
	Right	44	3	6.81
Hip	Left	44	0	
	Right	43	0	
Knee	Left	41	0	
	Right	42	0	
Cervical	Left	39	0	
	Right	39	0	
Thoracic	Left	38	1	2.63
	Right	38	1	2.63
Lumbar	Left	38	0	
	Right	38	0	

Four wild orangutans exhibited osteoarthritis as follows:

1. Specimen Number FM 33536: Female orangutan, predicted age 38, primary osteoarthritis of the elbows. The capitulum of the left and right humeri displayed moderate eburnation on 1/3-2/3 of each surface. The right olecranon process of the ulna showed moderate eburnation on 1/3-2/3 of the surface, while the trochlear notch demonstrated moderate eburnation on $< 1/3$ of the surface. The left radial head exhibited moderate eburnation on $> 2/3$ of the surface, while the right radial head displayed moderate eburnation on $< 1/3$ of the surface. In the vertebrae, C3-C5 exhibited slight lipping of the vertebral bodies.

Figure 133: Specimen Number FM 33536 – Left Humerus



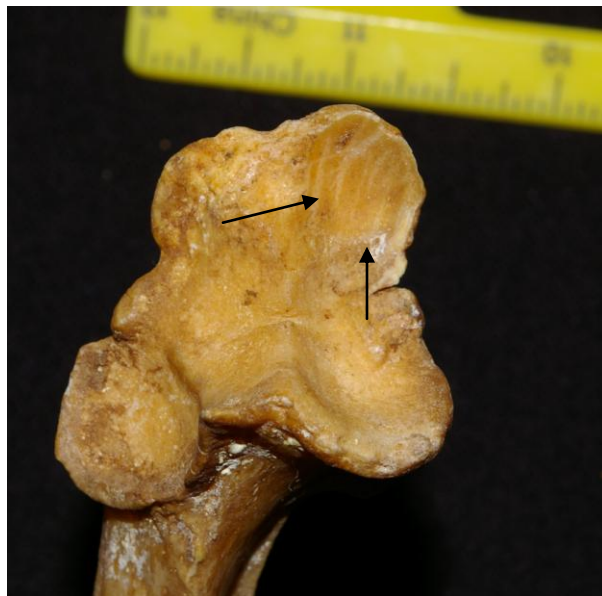
Specimen Number FM 33536: Eburnation on capitulum of left humerus

Figure 134: Specimen Number FM 33536 – Left Radius



Specimen Number FM 33536: Eburnation on left radial head – the entire surface displays a polished appearance

Figure 135: Specimen Number FM 33536 – Right Proximal Ulna



Specimen Number FM 33536: Eburnation on olecranon process of right ulna

2. Specimen Number BMNH 1880.4.10.1: Male orangutan, predicted age 15, osteoarthritis of the apophyseal articular facets in the thoracic vertebrae. The inferior left articular facet of T10 showed signs of moderate eburnation on < 1/3 of the surface, while the right articular facet demonstrated moderate

eburnation on 1/3-2/3 of the surface. The superior right articular facet of T11 exhibited moderate eburnation on < 1/3 of the surface.

Figure 136: Specimen Number BMNH 1880.4.10.1 – T10



Specimen Number BMNH 1880.4.10.1: Eburnation on inferior right articular facet of T10

3. Specimen Number BMNH 1986.1120: Male orangutan, predicted age 24, secondary osteoarthritis of the right elbow. An approximately 1cm circular area of moderate eburnation was evident on the capitulum of the right humerus and the right radial head. No other anomalies were noted.

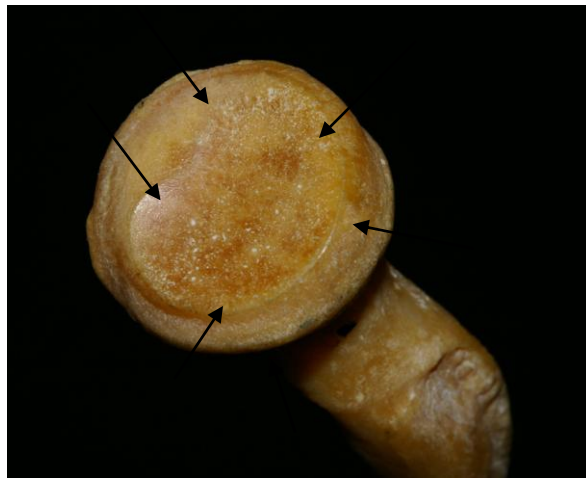
Figure 137: Specimen Number BMNH 1986.1120 – Right Distal Humerus



Specimen Number BMNH 1986.1120: Eburnation on capitulum of right humerus

4. Specimen Number MCZ 37363: Female orangutan, predicted age 27, secondary osteoarthritis of the right elbow. The right ulna is approximately 4 cm shorter than the left ulna, and the radial notch is distorted; however, no eburnation was evident. The right radius is approximately 2 cm shorter than the left radius and exhibits moderate eburnation on the entire surface ($> 2/3$). The right radial head is larger than the left.

Figure 138: Specimen Number MCZ 37363 – Right Radial Head



Specimen Number MCZ 37363: Eburnation on right radial head – arrows indicate the extent of the eburnation.
Museum of Comparative Zoology, Harvard University

Figure 139: Specimen Number MCZ 37363 – Left and Right Ulnae and Radii



Specimen Number MCZ 37363: Left and right ulnae and radii – note differences in length
Museum of Comparative Zoology, Harvard University

Vertebral Body Lipping: Among the orangutan sample, the presence of lipping on the superior and inferior rims of the vertebral bodies was relatively common. The captive and wild samples will be discussed separately as follows:

Captive orangutans: Lipping in the cervical, thoracic, and lumbar vertebrae is evident in over 77% of the captive orangutans. In the cervical vertebrae, of the 27 individuals examined, 15 (55.5%) showed evidence of lipping. Of these, 2 (7.4%) displayed slight lipping, 4 (14.8%) demonstrated moderate lipping, and 9 (33.3%) exhibited severe lipping. In the thoracic vertebrae, 17 of 25 individuals (68%) showed evidence of lipping. Of these 2 (8%) displayed slight lipping, 4 (16%) demonstrated moderate lipping, and 11 (44%) exhibited severe lipping. In the lumbar vertebrae, 20 of 26 individuals (76.9%) showed evidence of lipping. Of these, 1 (3.8%) displayed slight lipping, 4 (15.4%) demonstrated moderate lipping, and 15 (57.7%) exhibited severe lipping.

Wild orangutans: VOP in the cervical, thoracic, and lumbar vertebrae is uncommon in wild orangutans. In the cervical vertebrae, of the 38 individuals examined, 4 (10.3%) showed evidence of slight lipping. In the thoracic vertebrae, 6 of 37 individuals (16.2%) showed evidence of lipping. Of these, 5 (13.5%) displayed slight lipping, and 1 (2.7%) demonstrated moderate lipping. In the lumbar vertebrae, 7 of 38 individuals (18.4%) showed evidence of lipping. Of these, 5 (13.2%) displayed slight lipping and 2 (5.3%) exhibited moderate lipping. Moderate lipping was not seen in the cervical vertebrae, and severe lipping was not seen in any of the vertebral elements in wild orangutans.

Summary: In orangutans, severe primary joint osteoarthritis is uncommon in wild individuals but appears to be more common in captive individuals. Secondary OA is uncommon, and was seen in only one captive and two wild orangutans. Seven captive individuals showed evidence of severe joint OA, with the shoulders, elbows, and knees most commonly affected. One of these was a 14-year-old female with secondary OA of the right elbow resulting from a healed fracture. The TMJ was also affected in one captive individual. Of the six captive individuals with primary OA of the joints, two individuals demonstrated severe OA in three of the four main joints (shoulder, elbow, and knee) with extensive secondary porosity and degeneration of the joint surfaces. In wild individuals, the elbow was the most commonly affected joint. One individual exhibited primary OA in both elbows, while two individuals displayed secondary OA in the right elbow. OA was also evident in the apophyseal articular facets of one individual in the thoracic vertebrae (T10-T11 only). While both wild and captive orangutans demonstrate primary elbow osteoarthritis, only captive individuals had severe OA in the shoulder and knees. These differences in distribution might suggest that compressive stress and load bearing could be factors in disease expression; however, given the fact that both chimpanzees and gorillas do not demonstrate clear differences in these joints between the wild and captive samples, this may or may not be the case for orangutans.

For vertebral body lipping, wild orangutans demonstrate a uniformly low rate of involvement, while captive orangutans exhibit an extremely high rate of spinal involvement. Severe lipping was not seen in the vertebrae of wild orangutans and slight to moderate lipping was infrequent. In captive orangutans, spinal disease appears to be more common with the majority of individuals demonstrating severe involvement.

In terms of sex, in captive orangutans with eburnation, three males (9.7% of captive sample) and four females (12.9% of captive sample) were affected, while for wild orangutans with eburnation, two males (4.5% of wild sample) and two females (4.5% of wild sample) exhibited joint OA (of these one male and one female demonstrated secondary OA). In the vertebrae, 21 captive individuals showed evidence of lipping with 13 males (42.9% of captive sample) and 8 females (25.8% of captive sample) affected. In wild orangutans, 12 individuals showed evidence of lipping with 5 males (11.4% of wild sample) and 7 females (15.9% of wild sample) affected. The incidence of OA is almost equal in males and females in both wild and captive individuals, while vertebral body lipping is seen slightly more often in captive males and wild females. Thus, there is no clear sex bias in either captive or wild orangutans; however, given the bias towards the number of males in the captive sample (20 male/11 female), the fact that more captive females exhibited joint OA might indicate that females have a higher tendency to develop the disease. Nevertheless, more captive males exhibited some form of spinal lipping and thus any sex bias remains uncertain.

In terms of age, the captive orangutans with joint OA were aged 14 (secondary), 29 (predicted), 30, 32, 36, 38, and 45, while the wild orangutans with joint OA were predicted to be aged 15, 24 (secondary), 27 (secondary), and 38. The youngest captive individual exhibited secondary OA, while four out of five remaining individuals were over 30 years of age. The trend for wild individuals with primary OA is less clear.

OA Summary: In chimpanzees, severe primary osteoarthritis is rare in both captive and wild individuals. Of those individuals with OA in the four major joints (shoulder, elbow,

hip, and knee), captive individuals exhibited OA only in the knee, while wild individuals displayed OA in the shoulder and elbow. In gorillas, severe primary joint OA is uncommon in both captive and wild individuals. Of the captive gorillas with joint OA, the knee was affected most often. In wild gorillas, the elbow and knee joints are most commonly affected with both primary and secondary OA evident in both. In orangutans, severe primary joint osteoarthritis is uncommon in both captive and wild individuals. In captive orangutans the shoulders, elbows, and knees were most commonly affected, while in wild individuals, the elbow was the most commonly affected joint.

In terms of sex, for those individuals with primary OA of the joints, male captive chimpanzees exhibited OA more frequently than females while, in wild chimpanzees, females exhibited more joint OA than males. In gorillas, all captive and wild gorillas with either primary or secondary joint OA were male. In captive orangutans with osteoarthritis, three males and four females were affected, while for wild orangutans with osteoarthritis, two males and two females exhibited joint OA.

In terms of age, the majority of the individuals exhibiting OA in both the captive and wild sample were older adults. The younger individuals were those that tended to exhibit secondary OA. This fact lends support to the inference that OA is strongly related to age and counters the idea that the conditions of captivity might engender OA to develop at an earlier age.

For vertebral body lipping, both wild and captive chimpanzees exhibit a low incidence of lipping, particularly for severe involvement. Captive and wild gorillas exhibit a higher rate of involvement with severe lipping most often seen in the lumbar

vertebrae. Wild orangutans exhibit a uniformly low rate of vertebral body lipping, while captive orangutans exhibit an extremely high rate of spinal involvement.

To summarize the findings, Table 37 below shows the frequency of osteoarthritis in captive and wild great apes by specific joint. Table 38 below shows the combined frequencies of OA involvement for the four peripheral joints, TMJ, and apophyseal joints.

Table 37: Frequency of osteoarthritis in captive and wild great apes by specific joint.

	Status	TMJ				Shoulder				Elbow				Hip				Knee			
		Left		Right		Left		Right		Left		Right		Left		Right		Left		Right	
		N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%	N	%
Primary OA																					
Chimpanzee	Captive	53	1.7	53	1.7	58	0	56	0	57	0	55	0	57	0	56	0	55	1.8	53	1.8
	Wild	50	0	50	0	57	1	57	1	55	1.8	57	1.7	57	0	57	0	57	0	57	0
Gorilla	Captive	23	0	23	0	26	3.8	27	3.7	25	4	27	3.7	27	3.7	28	3.6	25	8	26	7.7
	Wild	58	1.7	58	1.7	65	0	64	0	65	1.5	65	1.5	65	0	65	0	65	1.5	65	3.1
Orangutan	Captive	27	0	27	3.7	30	10	30	10	30	0	30	6.7	30	0	30	0	29	10.3	29	13.8
	Wild	33	0	34	0	44	0	44	0	44	2.3	44	2.3	44	0	43	0	41	0	42	0
Secondary																					
Chimpanzee	Captive	53	0	53	0	58	0	56	0	57	0	55	0	57	0	56	0	55	0	53	0
	Wild	50	0	50	0	57	0	57	1	55	0	57	0	57	0	57	1.7	57	0	57	1.7
Gorilla	Captive	23	0	23	0	26	0	27	0	25	0	27	0	27	0	28	0	25	0	26	0
	Wild	58	0	58	0	65	0	64	0	65	3.1	65	1.5	65	0	65	0	65	0	65	1.5
Orangutan	Captive	27	0	27	0	30	0	30	0	30	0	28	3.6	30	0	31	0	29	0	29	0
	Wild	33	0	34	0	44	0	44	0	44	0	44	4.5	44	0	43	0	41	0	42	0

Species	Status	Cervical Apophyseal				Thoracic Apophyseal				Lumbar Apophyseal			
		Left		Right		Left		Right		Left		Right	
		N	%	N	%	N	%	N	%	N	%	N	%
Chimpanzee	Captive	53	0	53	1.9	50	0	50	0	50	0	50	0
	Wild	55	0	55	0	56	0	56	0	50	0	50	0
Gorilla	Captive	27	0	27	3.7	26	0	26	0	23	0	23	0
	Wild	62	0	62	1.6	62	0	62	0	58	0	58	0
Orangutan	Captive	27	0	27	0	25	0	25	0	24	0	24	0
	Wild	39	0	39	0	38	2.6	38	2.6	38	0	38	0

Table 38: Combined frequencies of OA by species and status

Species	Status	Sample size	# with OA	Overall %	Primary %	Secondary %
Chimpanzee	Captive	58	3	5.17	5.17	0
	Wild	57	5	8.8	3.50	5.26
Gorilla	Captive	29	3	10.34	10.34	0
	Wild	65	5	7.69	4.61	3.07
Orangutan	Captive	31	7	22.58	19.35	3.22
	Wild	44	4	9.09	4.54	4.54

Captive chimpanzees exhibit the lowest overall frequency of OA of any species, either captive or wild. But, wild chimpanzees exhibit the lowest frequency of primary OA and the highest frequency of secondary OA. In terms of species, the chimpanzee is the least frequently affected. This result mirrors that found by Jurmain (2000) where chimpanzees were rarely affected and gorillas were more commonly affected than chimpanzees (orangutans were not included in that study). Statistical analyses in chapter 5 will examine prevalence rates and other potential differences between the captive and wild samples to determine if any perceived differences are, in fact, statistically significant.

Chapter 5. Osteoarthritis - Comparative Analyses

While Chapter 4 described disease patterns and frequency, this chapter will focus on whether any perceived differences are statistically significant. For the vertebral bodies, the presence of vertebral body lipping will be examined. For the four main joints, TMJ, and vertebral apophyseal joints, the analysis will focus on eburnation and marginal lipping. The effect that status (captive/wild), sex, and species have on disease prevalence will be examined. Age will be considered a factor in each of the analyses because controlling for age is essential as age is expected to have an effect on disease prevalence. For all tests, logistic regression was utilized and individuals exhibiting secondary OA were removed from the analysis.

Computational Issues: The first part of Chapter 4 detailed the collection methods and use of an ordinal scaling system. While the preferred method of analyses would be to examine issues of disease severity (as indicated by use of an ordinal scaling system for data collection), the data do not allow for this level of analyses and a binary response (absent/present) will be utilized. This is because there were many instances (particularly for eburnation) where cell frequencies were zero. In general, statistical analyses require all expected cell frequencies to be > 1 , with at least 80% of cells having a frequency of > 5 (Sirkin, 1995). When there are cell frequencies of zero (as is the case here), computational issues occur and the results are considered to be uninformative. Thus, reducing the severity scale to two levels (absent/present) is appropriate. This method was chosen for the analyses of all three disease markers (eburnation, marginal lipping, and vertebral body lipping) because (for all three species) cell frequencies of zero were

encountered in at least one joint for each marker. Scores for the left and right sides of each joint were combined, partly because the focus of analysis is on primary osteoarthritis, which is typically a bilateral condition, and also to avoid any potential for further computational errors. It should also be noted that, in some instances, the “present” condition was not evident in either the captive or wild sample and for those joints the analyses were not computed.

Hypotheses: For each of the skeletal markers, the following hypotheses will be tested:

Hypothesis 1: Of interest, is whether status (wild vs captive), with age as a factor, effects disease prevalence in the great apes. Difference in prevalence of the disease is expected because research on other animal species indicates that OA is more common in captive animals (eg: Rothschild and Woods, 1992). Thus, it is anticipated that *status will affect disease prevalence in that captive animals will experience higher rates of OA than wild animals, even after controlling for age.*

H₀: Status has no effect on disease prevalence in the great apes, even after controlling for age.

H_A: Status has an effect on disease prevalence in the great apes, even after controlling for age.

Hypothesis 2: Of interest is whether sex, with age as a factor, effects disease prevalence. In both controlled clinical and archaeological studies of humans, disease prevalence is typically evaluated separately by sex because the frequency and patterning of expression varies by sex. It is, however, possible that body size may be a confounding factor in sex differences; although, the few studies that have controlled for body mass have found contradictory results (DeRousseau, 1988; Weiss and Jurmain, 2007).

Evaluation of disease prevalence by sex is warranted to determine what, if any, differences exist. However, a body size effect may still be inferred to be present if, after controlling for age, the males in each species have more OA than females *and* species-level differences of OA in males and females mirror species-level differences in sexual dimorphism. Thus, it is anticipated that *sex will affect disease prevalence in that males will have higher rates of OA than females, even after controlling for age.*

H₀: Sex has no effect on disease prevalence in the great apes, even after controlling for age.

H_A: Sex has an effect on disease prevalence in the great apes, even after controlling for age.

Hypothesis 3: Of interest is the whether species' differences, with age as a factor, effect disease prevalence. This expectation stems from research that found wild chimpanzees to be less frequently affected than wild gorillas (Jurmain, 2000). Thus, it is anticipated that *species' differences will affect disease prevalence in that chimpanzees will experience less OA than either gorillas or orangutans and that gorillas and orangutans will not differ from each other, even after controlling for age.*

H₀: Species' differences have no effect on disease prevalence, even after controlling for age.

H_A: Species' differences have an effect on disease prevalence, even after controlling for age.

Hypothesis 1: Of interest, is whether status (wild vs captive), with age as a factor, effects disease prevalence in the great apes. It should be noted that an interaction effect (status*age) was included to test whether captive vs wild specimens followed different patterns with age. However, when the interaction effect was included, the results (for all three variables) were not significant. Thus, the interaction effect does not explain much of the variation, and including this effect in the model increased the degrees of freedom which thereby reduced the power of the model pertaining to the two remaining variables (status, age). Consequently, the model without the interaction variable was utilized and the results for each species are as follows:

Chimpanzee:

Vertebral Body Lipping: Table 39 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold). The results show that status is a significant predictor of disease prevalence for all three vertebral segments, while age is a significant predictor of disease prevalence in the cervical vertebrae only. This means that captive chimpanzees have significantly more vertebral body lipping than wild chimpanzees in all three vertebral segments and that older chimpanzees are more likely to exhibit cervical body lipping.

Table 39: Chimpanzee Logistic Regression Summary by status – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		105	12.113	.002				
	Status				1.463	5.242	.026	4.137
	Age				.083	6.182	.013	1.087
Thoracic Body Lipping		97	8.265	.016				
	Status				1.673	.696	.016	5.331
	Age				.048	1.687	.194	1.049
Lumbar Body Lipping		93	14.298	.001				
	Status				2.073	8.933	.003	7.952
	Age				.062	3.139	.076	1.064

Eburnation only: Table 40 below shows the overall test of the model and the results for the coefficients of status and age. The results show that hypothesis 1 is not supported in any joint. Thus, it is concluded that there is no evidence that status or age are significant predictors of disease prevalence in chimpanzees.

Table 40: Chimpanzee Logistic Regression Summary by status – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		101	2.973	.226				
	Status				17.109	.000	.997	2.693E7
	Age				-.240	.953	.329	.787
Shoulder		112	1.800	.401				
	Status				-17.197	.000	.997	.000
	Age				.065	.328	.567	1.067
Elbow		111	3.273	.195				
	Status				-17.954	.000	.997	.000
	Age				-.054	.317	.573	.948
Hip	Not computed *							
Knee		107	1.966	.374				
	Status				17.176	.000	.997	2.881E7
	Age				.075	.586	.444	1.078
Cervical Apophyseal		105	5.935	.051				
	Status				16.783	.000	.997	1.944E7
	Age				.248	2.704	.100	1.281
Thoracic Apophyseal	Not computed *							
Lumbar Apophyseal	Not computed *							

* Eburnation was not present in either the wild or captive sample

Marginal Lipping only: Table 41 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold). The results show that status is a significant predictor of disease prevalence in all joints with the exception of the TMJ. Age is a significant predictor of disease prevalence in all joints except the knee and cervical apophyseal facets. This means that captive chimpanzees have significantly more marginal lipping than wild chimpanzees in most joints, and that older chimpanzees are more likely to exhibit marginal lipping in most joints.

Table 41: Chimpanzee Logistic Regression Summary by status – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		101	17.941	.000				
	Status				.596	1.147	.284	1.815
	Age				.125	13.285	.000	1.133
Shoulder		112	23.114	.000				
	Status				1.063	5.425	.020	2.896
	Age				.110	15.078	.000	1.116
Elbow		111	23.210	.000				
	Status				1.065	6.033	.014	2.902
	Age				.107	14.779	.000	1.113
Hip		111	24.442	.000				
	Status				1.065	6.033	.014	2.902
	Age				.107	14.779	.000	1.113
Knee		109	8.321	.016				
	Status				1.364	6.846	.009	3.911
	Age				.021	.626	.429	1.022
Cervical Apophyseal		105	15.134	.001				
	Status				1.869	11.333	.001	6.485
	Age				.037	1.666	.197	1.037
Thoracic Apophyseal		103	30.262	.000				
	Status				2.651	20.190	.000	14.169
	Age				.068	4.483	.035	1.071
Lumbar Apophyseal		107	35.939	.000				
	Status				2.755	19.268	.000	15.728
	Age				.117	10.801	.001	1.124

Gorilla:

Vertebral Body Lipping: Table 42 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold). The results show that status is a significant predictor of disease prevalence for all three vertebral segments, while age is a significant predictor of disease prevalence in the lumbar vertebrae only. This means that captive gorillas have significantly more vertebral

body lipping than wild gorillas in all three vertebral segments and that older gorillas are more likely to exhibit lumbar body lipping.

Table 42: Gorilla Logistic Regression Summary by status – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		88	6.895	.032				
	Status				1.092	5.184	.023	2.980
	Age				.041	1.726	.189	1.042
Thoracic Body Lipping		89	7.916	.019				
	Status				1.094	4.946	.026	2.987
	Age				.052	2.789	.095	1.053
Lumbar Body Lipping		88	11.182	.004				
	Status				1.255	6.254	.012	3.509
	Age				.068	4.572	.033	1.071

Eburnation only: Table 43 below shows the overall test of the model and the results for the coefficients of status and age. The results show that hypothesis 1 is not supported in any joint. Thus, it is concluded that there is no evidence that status or age are significant predictors of disease prevalence in gorillas.

Table 43: Gorilla Logistic Regression Summary by status – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		79	2.418	.298				
	Status				-17.986	.000	.998	.000
	Age				.098	1.039	.308	1.103
Shoulder		91	8.238	.016				
	Status				29.253	.000	.993	5.062E2
	Age				2.626	.000	.991	13.817
Elbow		91	1.655	.437				
	Status				.846	.341	.559	2.330
	Age				.105	1.283	.257	1.111
Hip		92	6.676	.036				
	Status				18.223	.000	.996	8.210E7
	Age				.549	.854	.335	1.731
Knee		90	6.547	.038				
	Status				1.395	2.019	.155	4.035
	Age				.129	4.024	.045	1.138
Cervical Apophyseal		87	3.319	.190				
	Status				.714	.232	.630	2.042
	Age				.165	2.589	.108	1.179
Thoracic Apophyseal	Not computed *							
Lumbar Apophyseal	Not computed *							

* Eburnation was not present in either the wild or captive sample

Marginal Lipping Only: Table 44 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold). The results show that status is a significant predictor of disease prevalence in all joints with the exception of the TMJ. Age is a significant predictor of disease prevalence, but only in some joints. This means that captive gorillas have significantly more marginal lipping than wild gorillas in most joints, and that older gorillas are more likely to exhibit marginal lipping in the hip, knee, cervical apophyseal, and thoracic apophyseal joints only.

Table 44: Gorilla Logistic Regression Summary by status – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		78	.794	.672				
	Status				-.425	.351	.553	.654
	Age				.029	.474	.491	1.030
Shoulder		91	6.204	.035				
	Status				1.092	5.271	.022	2.982
	Age				.031	1.051	.305	1.031
Elbow		91	21.095	.000				
	Status				2.186	16.848	.000	8.903
	Age				.053	2.385	.122	1.054
Hip		92	11.973	.003				
	Status				1.101	4.980	.026	3.009
	Age				.082	6.422	.011	1.065
Knee		90	22.399	.000				
	Status				2.067	12.238	.000	7.900
	Age				.119	8.957	.003	1.126
Cervical Apophyseal		87	24.349	.000				
	Status				2.439	13.767	.000	11.459
	Age				.124	8.068	.005	1.132
Thoracic Apophyseal		88	28.479	.000				
	Status				2.715	19.497	.000	15.110
	Age				.073	3.825	.050	1.076
Lumbar Apophyseal		82	13.381	.001				
	Status				1.644	9.115	.003	5.177
	Age				.063	3.394	.065	1.065

Orangutan:

Vertebral Body Lipping: Table 45 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold). The results show that status is a significant predictor of disease prevalence for all three vertebral segments, while age is a significant predictor of disease prevalence in the cervical and lumbar vertebrae only. This means that captive orangutans have significantly more vertebral body lipping than wild orangutans in all three vertebral

segments and that older orangutans are more likely to exhibit cervical and lumbar body lipping.

Table 45: Orangutan Logistic Regression Summary by status – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		63	35.626	.000				
	Status				3.415	13.273	.000	30.430
	Age				.212	10.960	.001	1.236
Thoracic Body Lipping		59	21.078	.000				
	Status				2.583	14.799	.000	13.240
	Age				.066	3.106	.078	1.068
Lumbar Body Lipping		61	25.487	.000				
	Status				2.726	15.097	.000	15.265
	Age				.102	6.486	.011	1.107

Eburnation only: Table 46 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold).

The results show that hypothesis 1 is not supported in any joint. Thus, it is concluded that there is no evidence that status or age are significant predictors of disease prevalence in orangutans.

Table 46: Orangutan Logistic Regression Summary by status – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		59	1.804	.406				
	Status				17.873	.000	.998	5.780E7
	Age				.058	.139	.709	1.060
Shoulder		72	10.709	.005				
	Status				19.854	.000	.997	4.192E8
	Age				.242	3.093	.079	1.274
Elbow		71	3.198	.202				
	Status				1.214	.877	.349	3.366
	Age				.113	2.186	.139	1.120
Hip	Not computed *							
Knee		69	9.932	.007				
	Status				19.518	.000	.997	2.996E8
	Age				.124	2.114	.146	1.132
Cervical Apophyseal	Not computed *							
Thoracic Apophyseal		60	7.399	.025				
	Status				-79.865	.000	.989	.000
	Age				-12.579	.000	.981	.000
Lumbar Apophyseal	Not computed *							

* Eburnation was not present in either the wild or captive sample

Marginal Lipping only: Table 47 below shows the overall test of the model and the results for the coefficients of status and age (significant results are highlighted in bold). The results show that status is a significant predictor of disease prevalence in all joints with the exception of the TMJ. Age is a significant predictor of disease prevalence in all but the TMJ and thoracic apophyseal joints. This means that captive orangutans have significantly more marginal lipping than wild orangutans in most joints, and that older orangutans are more likely to exhibit marginal lipping in most joints.

Table 47: Orangutan Logistic Regression Summary by status – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		59	3.113	.211				
	Status				.931	1.014	.314	2.536
	Age				.085	1.633	.201	1.088
Shoulder		72	27.737	.000				
	Status				2.635	14.240	.000	13.939
	Age				.126	8.802	.003	1.134
Elbow		71	29.752	.000				
	Status				2.748	17.642	.000	15.618
	Age				.107	7.610	.006	1.113
Hip		72	26.284	.000				
	Status				2.154	12.463	.000	8.617
	Age				.126	1.244	.001	1.134
Knee		69	31.878	.000				
	Status				4.238	12.358	.000	69.297
	Age				.120	5.470	.019	1.128
Cervical Apophyseal		61	13.155	.001				
	Status				1.762	6.910	.009	5.823
	Age				.092	4.698	.030	1.096
Thoracic Apophyseal		60	19.877	.000				
	Status				2.548	15.780	.000	12.784
	Age				.033	.838	.360	1.034
Lumbar Apophyseal		59	16.634	.000				
	Status				2.045	10.161	.001	7.726
	Age				.086	5.010	.025	1.090

Hypothesis 2: Of interest is whether sex, with age as a factor, affects disease

prevalence. The results for each species are as follows:

Chimpanzee:

Vertebral Body Lipping: Table 48 below shows the overall test of the model and the results for the coefficients of sex and age (individuals of unknown sex (n=3) were removed from the analysis) (significant results are highlighted in bold). The results show that sex is not a significant predictor of disease prevalence in any vertebral segment. Age is a significant predictor of disease prevalence in the cervical and thoracic vertebrae only. Thus, it is concluded that there is no evidence that sex is a significant predictor of disease prevalence in chimpanzees, while older chimpanzees are more likely to exhibit vertebral body lipping in the cervical and thoracic vertebrae.

Table 48: Chimpanzee Logistic Regression Summary by sex – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		102	8.548	.014				
	Sex				-.089	.023	.880	.915
	Age				.094	7.521	.006	1.099
Thoracic Body Lipping		94	1.425	.490				
	Sex				-.099	.029	.866	.906
	Age				.040	1.354	.245	1.041
Lumbar Body Lipping		90	5.334	.069				
	Sex				-.074	.017	.896	.928
	Age				.077	4.929	.026	1.080

Eburnation only: Table 49 below shows the overall test of the model and the results for the coefficients of sex and age (individuals of unknown sex (n=3) were

removed from the analysis). The results show that hypothesis 2 is not supported in any joint. Thus it is concluded that there is no evidence that sex or age are significant predictors of disease prevalence in chimpanzees.

Table 49: Chimpanzee Logistic Regression Summary by Sex – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		101	2.632	.068				
	Sex				16.801	.000	.998	1.980E7
	Age				-.200	.735	.391	.819
Shoulder		109	1.736	.420				
	Sex				-17.189	.000	.997	.000
	Age				.055	.264	.608	1.056
Elbow		108	3.162	.164				
	Sex				-18.100	.000	.997	.000
	Age				-.075	.481	.408	.927
Hip	Not computed *							
Knee		104	1.984	.371				
	Sex				17.274	.000	.997	3.177E1
	Age				.085	.598	.439	1.089
Cervical Apophyseal		102	5.401	.067				
	Sex				-16.142	.000	.997	.000
	Age				.236	2.297	.130	1.267
Thoracic Apophyseal	Not computed *							
Lumbar Apophyseal	Not computed *							

* Eburnation was not present in the sample

Marginal Lipping only: Table 50 below shows the overall test of the model and the results for the coefficients of sex and age (individuals of unknown sex (n=3) were removed from the analysis) (significant results are highlighted in bold). The results show that sex is not a significant predictor of disease prevalence in any joint. Age is a significant predictor of disease prevalence in most joints. Thus, it is concluded that there

is no evidence that sex is a significant predictor of disease prevalence in chimpanzees, while older chimpanzees are more likely to exhibit marginal lipping in all joints except the knee, cervical apophyseal, and thoracic apophyseal joints.

Table 50: Chimpanzee Logistic Regression Summary by Sex – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		101	16.901	.000				
	Sex				-.200	.136	.712	.819
	Age				.123	13.270	.000	1.130
Shoulder		109	22.039	.000				
	Sex				-.159	.124	.725	.853
	Age				.120	16.905	.000	1.128
Elbow		108	19.208	.000				
	Sex				.306	.521	.470	1.358
	Age				.108	15.196	.000	1.114
Hip		108	12.385	.002				
	Status				-.574	1.907	.167	.563
	Age				.076	9.038	.003	1.079
Knee		106	1.934	.380				
	Sex				.101	.042	.838	1.106
	Age				.038	1.916	.166	1.039
Cervical Apophyseal		102	1.875	.392				
	Sex				-.014	.001	.976	.986
	Age				.037	1.825	.177	1.037
Thoracic Apophyseal		100	4.094	.129				
	Sex				-.453	1.031	.310	.635
	Age				.045	2.744	.098	1.046
Lumbar Apophyseal		94	10.056	.007				
	Sex				-.067	.021	.884	.936
	Age				.087	8.855	.003	1.091

Gorilla:

Vertebral Body Lipping: Table 51 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that hypothesis 2 is not supported in any joint. Thus, it is concluded that there is no evidence that sex or age are significant predictors of disease prevalence in gorillas.

Table 51: Gorilla Logistic Regression Summary by sex – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		88	4.432	.109				
	Sex				.767	2.806	.094	2.154
	Age				.029	.848	.357	1.029
Thoracic Body Lipping		89	4.274	.118				
	Sex				.562	1.587	.208	1.754
	Age				.039	1.693	.193	1.040
Lumbar Body Lipping		88	6.119	.047				
	Sex				.566	1.520	.218	1.762
	Age				.055	3.182	.074	1.056

Eburnation only: Table 52 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that hypothesis 2 is not supported in any joint. Thus it is concluded that there is no evidence that sex or age are significant predictors of disease prevalence in gorillas.

Table 52: Gorilla Logistic Regression Summary by Sex – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		79	2.881	.237				
	Sex				17.948	.000	.998	6.234E7
	Age				.066	.485	.486	1.068
Shoulder		91	8.238	.016				
	Sex				76.072	.000	.986	1.090E33
	Age				11.918	.001	.975	149975.123
Elbow		91	3.279	.194				
	Sex				17.713	.000	.998	4.926E7
	Age				.091	.794	.373	1.096
Hip		92	6.047	.049				
	Status				19.371	.000	.996	2.587E8
	Age				.825	.806	.369	2.282
Knee		90	5.189	.075				
	Sex				.920	.617	.432	2.510
	Age				.124	3.506	.061	1.132
Cervical Apophyseal		87	5.150	.076				
	Sex				17.715	.000	.998	4.936E7
	Age				.177	2.025	.155	1.193
Thoracic Apophyseal	Not computed *							
Lumbar Apophyseal	Not computed *							

* Eburnation was not present in the sample

Marginal Lipping only: Table 53 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that sex is not a significant predictor of disease prevalence in any joint. Age is a significant predictor of disease prevalence in some joints. Thus, it is concluded that there is no evidence that sex is a significant predictor of disease prevalence in gorillas, while older gorillas are more likely to exhibit marginal lippling in the hip, knee, and cervical apophyseal joints only.

Table 53: Gorilla Logistic Regression Summary by Sex – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		78	.493	.781				
	Sex				.168	.068	.794	1.183
	Age				.024	.325	.569	1.025
Shoulder		91	1.247	.536				
	Sex				.291	.411	.521	1.337
	Age				.023	.572	.450	1.023
Elbow		91	3.530	.171				
	Sex				.640	1.980	.159	1.897
	Age				.028	.829	.362	1.028
Hip		92	7.822	.020				
	Status				-.475	1.090	.297	.622
	Age				.083	6.910	.009	1.086
Knee		90	8.620	.013				
	Sex				.118	.052	.820	1.126
	Age				.096	7.173	.007	1.101
Cervical Apophyseal		87	8.904	.012				
	Sex				.571	.982	.322	1.770
	Age				.097	6.243	.012	1.102
Thoracic Apophyseal		88	3.399	.183				
	Sex				.413	.835	.361	1.512
	Age				.041	1.833	.176	1.042
Lumbar Apophyseal		82	3.278	.194				
	Sex				-.126	.073	.787	.881
	Age				.055	3.106	.078	1.052

Orangutan:

Vertebral Body Lipping: Table 54 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that sex is not a significant predictor of disease prevalence in any vertebral segment. Age is a significant predictor of disease prevalence in the cervical and lumbar vertebrae only. Thus, it is concluded that there is no evidence that sex is a significant predictor of disease prevalence in orangutans, while older orangutans are more likely to exhibit vertebral body lipping in the cervical and thoracic vertebrae.

Table 54: Orangutan Logistic Regression Summary by sex – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		63	15.376	.000				
	Sex				-.260	.171	.679	.771
	Age				.156	10.315	.001	1.169
Thoracic Body Lipping		59	3.023	.221				
	Sex				-.217	.156	.693	.805
	Age				.054	2.641	.104	1.056
Lumbar Body Lipping		61	5.934	.051				
	Sex				-.059	.012	.914	.942
	Age				.080	5.123	.024	1.084

Eburnation only: Table 55 below shows the overall test of the model and the results for the coefficients of sex and age. The results show that hypothesis 2 is not supported in any joint. Thus it is concluded that there is no evidence that sex or age are significant predictors of disease prevalence in orangutans.

Table 55: Orangutan Logistic Regression Summary by Sex – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		59	1.657	.437				
	Sex				17.840	.000	.998	5.595E7
	Age				.078	.274	.699	1.081
Shoulder		72	4.043	.132				
	Sex				.432	.110	.740	1.540
	Age				.139	3.379	.668	1.149
Elbow		71	2.424	.298				
	Sex				.527	.171	.679	1.694
	Age				.102	2.120	.145	1.107
Hip	Not computed *							
Knee		69	2.197	.333				
	Sex				-.148	.020	.889	.862
	Age				.091	2.129	.145	1.095
Cervical Apophyseal	Not computed *							
Thoracic Apophyseal		60	3.646	.162				
	Sex				16.853	.000	.998	2.085E7
	Age				-.247	1.387	.239	.781
Lumbar Apophyseal	Not computed *							

* Eburnation was not present in the sample

Marginal Lipping only: Table 56 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that sex is not a significant predictor of disease prevalence in any joint. Age is a significant predictor of disease prevalence in most joints. Thus, it is concluded that there is no evidence that sex is a significant predictor of disease prevalence in orangutans, while older orangutans are more likely to exhibit marginal lipping in all joints except the TMJ and thoracic apophyseal joints.

Table 56: Orangutan Logistic Regression Summary by Sex – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		59	2.042	.360				
	Sex				-.011	.000	.990	.989
	Age				.089	1.926	.165	1.093
Shoulder		72	10.658	.005				
	Sex				.640	1.293	.255	1.897
	Age				.105	7.838	.005	1.111
Elbow		71	8.692	.013				
	Sex				.688	1.744	.187	1.989
	Age				.086	6.312	.012	1.090
Hip		72	12.550	.002				
	Status				.503	.910	.340	1.654
	Age				.114	9.539	.002	1.121
Knee		69	5.538	.063				
	Sex				.596	.968	.325	1.814
	Age				.076	4.248	.039	1.079
Cervical Apophyseal		61	5.515	.063				
	Sex				-.031	.003	.960	.969
	Age				.088	4.782	.029	1.092
Thoracic Apophyseal		60	1.820	.403				
	Sex				.547	1.019	.313	1.728
	Age				.029	.858	.354	1.030
Lumbar Apophyseal		59	6.222	.045				
	Sex				.625	1.168	.280	1.868
	Age				.079	4.678	.031	1.083

Hypothesis 3: Of interest is the whether species' differences, with age as a factor, affect disease prevalence. The results are as follows:

Vertebral Body Lipping: Table 57 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that in the cervical vertebrae, gorillas have significantly more cervical body lipping than orangutans, and that chimpanzees and orangutans do not differ from each other. In the thoracic vertebrae, chimpanzees have significantly less thoracic

body lipping than orangutans while gorillas have significantly more thoracic body lipping than orangutans. Thus, in the thoracic vertebrae, chimpanzees are the least frequently affected followed by orangutans and gorillas. In the lumbar vertebrae, chimpanzees have significantly less lumbar body lipping than orangutans, while gorillas and orangutans do not differ from each other. Thus, chimpanzees are the least frequently affected species in all three vertebral segments, while gorillas are the most frequently affected species in the cervical and thoracic vertebrae. In addition, age is a significant factor in all three vertebral segments with older individuals being more likely to exhibit vertebral body lipping.

Table 57: Logistic Regression Summary by species – Vertebral Body Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
Cervical Body Lipping		256	31.731	.000				
	Species1				-.431	1.143	.285	.650
	Species2				.936	5.780	.016	2.551
	Age				.078	16.446	.000	1.081
Thoracic Body Lipping		245	34.419	.000				
	Species1				-1.046	6.889	.009	.351
	Species2				.723	3.972	.046	2.060
	Age				.046	6.336	.012	1.047
Lumbar Body Lipping		242	26.623	.000				
	Species1				-.831	4.764	.029	.436
	Species2				.326	.806	.369	1.385
	Age				.062	11.694	.001	1.064

Indicator variables: Species1 = chimp; Species2 = gorilla; reference category = orangutan

Eburnation only: Table 58 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that species is not a significant predictor of disease prevalence in any joint. Age is a significant predictor of disease prevalence, but only in some joints. Thus,

it is concluded that there is no evidence that species' differences are a significant predictor of disease prevalence, while older individuals are more likely to exhibit eburnation in the shoulder, knee, and cervical apophyseal joints only.

Table 58: Logistic Regression Summary by Species – Eburnation

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		239	.722	.868				
	Species1				-.454	.098	.755	.635
	Species2				.508	.157	.692	1.662
	Age				.018	.081	.775	1.018
Shoulder		275	9.536	.023				
	Species1				-1.170	.960	.327	.310
	Species2				-.617	.254	.614	.540
	Age				.143	6.261	.012	1.153
Elbow		273	2.549	.466				
	Species1				-.651	.477	.490	.522
	Species2				-.388	.163	.686	.679
	Age				.056	1.598	.206	1.58
Hip		275	6.432	.092				
	Species1				1.061	.000	1.000	.289
	Species2				18.670	.000	.996	1.283E8
	Age				.329	2.223	.136	1.389
Knee		266	11.706	.008				
	Species1				-1.560	1.860	.173	.210
	Species2				.518	.480	.488	1.678
	Age				.105	6.675	.010	1.111
Cervical Apophyseal		255	9.830	.020				
	Species1				17.283	.000	.997	3.207E7
	Species2				18.790	.000	.997	1.447E8
	Age				.205	5.875	.015	1.228
Thoracic Apophyseal		249	5.874	.118				
	Species1				-18.406	.000	.996	.000
	Species2				-17.854	.000	.996	.000
	Age				-.295	1.634	.201	.745
Lumbar Apophyseal	Not computed (Eburation was not present in any species)							

Indicator variables: Species1 = chimp; Species2 = gorilla; reference category = orangutan

Marginal Lipping only: Table 59 below shows the overall test of the model and the results for the coefficients of sex and age (significant results are highlighted in bold). The results show that species' differences are a significant predictor of disease prevalence, but only in some joints. Age is a significant predictor of disease prevalence in all joints. Thus, older individuals are more likely to exhibit marginal lipping. In the TMJ, chimpanzees and gorillas have significantly more marginal lipping than orangutans, with chimpanzees having higher odds of exhibiting marginal lipping in the TMJ than the other two species. In the elbow, chimpanzees have significantly more marginal lipping than orangutans, while gorillas and orangutans do not differ from each other. In the hip, gorillas have more marginal lipping than chimpanzees and orangutans, while chimpanzees and orangutans do not differ from each other. In the lumbar apophyseal joints, gorillas have more marginal lipping than orangutans, while chimpanzees and orangutans do not differ from each other. There are no significant differences among the species in the shoulder, knee, cervical apophyseal facets, and thoracic apophyseal facts. Thus, for marginal lipping chimpanzees are the most affected species in the TMJ and elbow, while gorillas are the most affected in the hip and lumbar apophyseal joints.

Table 59: Logistic Regression Summary by Species – Marginal Lipping

Joint	Variable	N	Test of Model Coefficients		Variables in the Equation			
			Chi Square	Sig	β	Wald	Sig	Exp(B)
TMJ		238	19.203	.000				
	Species1				1.296	6.119	.013	3.653
	Species2				1.095	3.855	.050	2.990
	Age				.088	14.702	.000	1.093
Shoulder		275	23.939	.000				
	Species1				.605	3.020	.082	1.832
	Species2				.674	3.459	.063	1.963
	Age				.078	21.027	.000	1.081
Elbow		273	35.460	.000				
	Species1				.730	4.779	.029	2.075
	Species2				.265	.588	.443	1.304
	Age				.075	20.351	.000	1.078
Hip		275	36.313	.000				
	Species1				.384	1.346	.246	1.468
	Species2				.699	4.146	.042	2.013
	Age				.079	22.404	.000	1.082
Knee		268	10.707	.013				
	Species1				.159	173	.677	1.172
	Species2				.469	1.444	.229	1.599
	Age				.056	9.817	.002	1.057
Cervical Apophyseal		253	11.884	.008				
	Species1				.285	.525	.469	1.330
	Species2				.142	.117	.732	1.153
	Age				.062	10.957	.001	1.064
Thoracic Apophyseal		251	8.952	.030				
	Species1				-.261	.550	.458	.771
	Species2				.268	.571	.450	1.307
	Age				.038	5.355	.021	1.039
Lumbar Apophyseal		238	18.396	.000				
	Species1				.292	.626	.429	1.339
	Species2				.745	3.862	.049	2.107
	Age				.069	15.112	.000	1.072

Indicator variables: Species1 = chimp; Species2 = gorilla; reference category = orangutan

Summary: A summary of the results is shown in tables 60-62. The results show that hypothesis 1 is partially supported. Status is a significant predictor of disease prevalence for vertebral body lipping and marginal lipping (except in the TMJ), but is not a significant predictor of the prevalence of eburnation. Captive animals have more vertebral body lipping and marginal lipping than wild animals, but wild and captive animals do not differ significantly from each other in the prevalence of eburnation. Thus, although status has some effect, the lack of significantly more eburnation in captive apes suggests that the conditions of captivity do not *always* engender arthritic disease development and progression. Hypothesis 2 is not supported. Sex is not a significant predictor of disease prevalence for vertebral body lipping, marginal lipping, or eburnation. Thus, a body size effect is not inferred to be present because, even after controlling for age, the males in each species do not have more disease than females *and* there was no evidence of species-level differences of disease expression in males and females that mirror species-level differences in sexual dimorphism. Hypothesis 3 is partially supported in that, for vertebral body lipping, chimpanzees are the least frequently affected followed by orangutans and gorillas. However, there is no evidence of species-level differences in the prevalence of eburnation, while for marginal lipping, the results are mixed. In general, for marginal lipping, orangutans are the least frequently affected species. In terms of age, older individuals exhibit more vertebral body lipping and marginal lipping than younger individuals in many joints. Age has less of an effect on the prevalence of eburnation, which is most likely due to the rarity of eburnation in the great apes. The finding that older individuals are more likely to exhibit these diseases lends support to the inference that there is a strong age related component and counters

the idea that the conditions of captivity might engender diseases to develop at an earlier age.

In summary, while status, species' differences, and age are factors in the development of vertebral body lipping and marginal lipping in many joints, the prevalence of OA, as diagnosed by the presence of eburnation, is extremely rare in the great apes with very few individuals being affected regardless of status, sex, species, or age.

Table 60: Summary of Results for Vertebral Body Lipping:

Hypotheses	Species	Cervical	Thoracic	Lumbar
1 Status	Chimpanzee	✓*	✓	✓
	Gorilla	✓	✓	✓*
	Orangutan	✓*	✓	✓*
2 Sex	Chimpanzee	×*	×	×*
	Gorilla	×	×	×
	Orangutan	×*	×	×*
3 Species	Chimpanzee	Least +*	Least*	Least*
	Gorilla	Most*	Most*	Most +*
	Orangutan	Least +*	Middle*	Most +*

* indicates age is a significant predictor of disease prevalence

+ indicates these species do not differ significantly from each other

Table 61: Summary of Results for Eburnation:

Hypotheses	Species	TMJ	Shoulder	Elbow	Hip	Knee	Cervical facets	Thoracic facets	Lumbar facets
1 Status	Chimpanzee	×	×	×	NC	×	×	NC	NC
	Gorilla	×	×	×	×	×*	×	NC	NC
	Orangutan	×	×	×	NC	×	×	×	NC
2 Sex	Chimpanzee	×	×	×	NC	×	×	NC	NC
	Gorilla	×	×	×	×	×	×	NC	NC
	Orangutan	×	×	×	NC	×	×	×	NC
3 Species		×	×*	×	×	×*	×*	×	NC

NC = not computed: eburnation was not present in any individual for the joints specified

* indicates age is a significant predictor of disease prevalence

Table 62: Summary of Results for Marginal Lipping:

Hypotheses	Species	TMJ	Shoulder	Elbow	Hip	Knee	Cervical facets	Thoracic facets	Lumbar facets
1 Status	Chimpanzee	×*	✓*	✓*	✓*	✓	✓	✓*	✓*
	Gorilla	×	✓	✓	✓*	✓*	✓*	✓*	✓
	Orangutan	×	✓*	✓*	✓*	✓*	✓*	✓	✓*
2 Sex	Chimpanzee	×*	×*	×*	×*	×	×	×	×*
	Gorilla	×	×	×	×*	×*	×*	×	×
	Orangutan	×	×*	×*	×*	×*	×*	×	×*
3 Species	Chimpanzee	Most*	×*	Most*	Least+*	×*	×*	×*	Least+*
	Gorilla	Middle*	×*	Least+*	Most*	×*	×*	×*	Most*
	Orangutan	Least*	×*	Least+*	Least+*	×*	×*	×*	Least+*

+ indicates these species do not differ significantly from each other

* indicates age is a significant predictor of disease prevalence

Chapter 6 – Summary and Conclusions

The primary goal of this study was to examine the prevalence of osteoarthritis in wild versus captive great ape skeletons. A secondary, but equally important, goal was to examine the issue of aging of great ape skeletons. A summary of these two areas of interest is presented below.

Aging of the Sample

There is no doubt that aging of great ape skeletons is challenging. Predicting age with some degree of accuracy for adult great ape skeletons (in particular) has proven both complex and elusive. Chapter 3 focused on the problems associated with aging of adult great ape skeletons. In order to test the validity of the current aging categories for adults specimens and to test the potential of other skeletal aging markers, data was taken on the basilar suture, sternal rib ends (3rd, 4th, and 5th ribs), auricular surface, acetabulum, and dentition. The ribs, auricular surface, and acetabulum were scored based on commonly used methods developed for aging human skeletons, while dental wear stages were created and are based on methods developed for humans and African apes (Molnar, 1971b and Kilgore, 1989). However, utilizing human-based standards for assessing the age of ape skeletons may incur potential inaccuracy due to intrinsic differences between humans and apes. This may be especially true for the postcranial features tested, in which difference in locomotion between apes and humans and the concomitant morphological adaptations may have an effect on the outcome.

Results of tests on known-aged captive specimens show that most of the skeletal markers of age did not work especially well. While the basilar suture shows some

variability in age of closure, it is closed at a relatively early age in virtually all known-aged captive individuals. Thus, an “old adult” category most likely contains individuals who are, in fact, relatively young. This indicates that using the basilar suture as a means to categorize individual skeletons as “adult” or “old adult” is very imprecise. Of the other aging methods tested, the ribs and auricular surface proved to be of limited use because there was generally not enough variation in the known-aged individuals. In addition, there appear to be potential differences as to which predictors (if any) are correlated with age among the three different species. The small number of observations associated with these aging markers, particularly in the gorilla and orangutan, mean that any suggestion of non-normality in the data is tentative. Thus, the ribs and auricular surface were not used for aging the skeletons of unknown age in this study. However, at least for the ribs, based on the appearance of certain aging indicators (especially margins, nodules, pit depth), it is probable that morphological differences among the species are apparent. Future research is needed to explore this pattern of species-related differences in morphology and senescent changes.

Unlike the ribs and auricular surface, the acetabulum demonstrates some potential for use in aging. There was some evidence of non-linearity and non-normally distributed data in gorillas and orangutans; although, K-S normality tests of the standardized residuals indicate that these deviations were not significant. Thus, indications of non-normality in the histograms are most likely due to the small sample size. As with the ribs and auricular surface, different variables are highlighted in each species as being significant. A reduction in the number of variables resulted in an improved model; however, given the small sample and limited number of variables that are significant

overall, more evidence is needed to confirm the effectiveness of using the acetabulum as an aging indicator. Thus, using the acetabulum as the *sole* marker for age is not currently recommended.

While the acetabulum shows some merit in aging the ape sample, molar dental wear proved to be the most useful single aging indicator. Two models were tested, one using all molars and the second using only molars 1 and 2. The model utilizing only molars 1 and 2 provided the best fit, particularly in chimpanzees and gorillas. However, a model that included both molars 1 and 2 and certain features of the acetabulum also proved to be viable. In the combined model, the R^2 was extremely high for all three species (above 90%), although it was significant only in the chimpanzee. The combined model was chosen as the primary method for predicting age in the unknown-aged skeletons, in part because features that combine both cranial and postcranial data are considered more useful in cases where skeletal parts are missing (i.e., no skull). Further, Pearson's correlations, which test the strength of the relationship between each marker and age, are significant for nearly all of the markers. This lends further credence that this method is a viable option for predicting age.

Nevertheless, although the aging method utilized is considered to be an improvement over more commonly used methods, it is acknowledged that this system is far from perfect. The biggest problem encountered was a lack of known-aged individuals, particularly in the gorilla and orangutan sample. Another problem is that different variables are sometimes being highlighted as being significant in each species; however, this could be due to the small samples or to true morphological differences among the species. Nevertheless, manipulating the dataset by reducing the variables to fit

the data is inadvisable as this would result in a model that is not generalizable. For chimpanzees, the sample of known-aged individuals was sufficient to obtain results for all tests, while for gorillas and orangutans some tests could not be computed due to the small number of observations. The age bias toward older individuals in the captive orangutan sample likely introduced a higher probability of error. Given the broad similarities in the great ape species, it is reasonable to assume that with a larger sample of gorillas and orangutans, the results for each species would be comparable. However, it may be wise to analyze more closely the utility of developing aging models that are specific to each species rather than using a generalized model as was done here. Thus, future research needs to be conducted on samples with more known-aged individuals to allow the validity of the models presented to be verified.

One other potential problem is that it is not known with certainty whether wild and captive apes demonstrate any major differences in rate of skeletal aging. Researchers who have studied skeletal aging in the apes tend to focus on either wild or captive specimens, which are usually from specific populations (eg: Morbeck et al., 2002). Indeed, comparative studies of wild and captive ape skeletons are extremely rare. This is also true for systematic studies that compare the behavior of aged and young great apes, either in wild or captive settings (Tarou et al., 2002). It is true, however, that age-related skeletal changes in adult mammals follow some basic principles and thus it is not unreasonable to assume that age-related changes are similar in wild and captive specimens. Evidence from this research suggests that captive and wild specimens show similar age-related changes in the features studied. This is supported by concordance among the aging indicators in the wild and captive sample as evidenced by the rates of

total molar wear in the sample. It should be noted, however, that a certain amount of individual variation likely plays a role in skeletal aging; this is to be expected in myriad species and does not inherently affect the results. Thus, while problems were encountered with developing the new aging techniques, the outcome is a step in the right direction and provides the best age estimates currently available.

Osteoarthritis

Chapters 4 and 5 dealt with the issue of osteoarthritis in the great ape sample. Chapter 4 was primarily descriptive in nature, while chapter 5 focused on statistical analyses of the data. Three hypotheses were tested relating to (1) wild vs captive status, (2) sex, and (3) species' differences. Age was examined as a co-factor as, in humans, age is commonly acknowledged as being closely related to disease expression. Three markers were examined and analyzed separately: vertebral body lipping, eburnation, and marginal lipping. A brief summary of the results is as follows:

Hypothesis 1: Status.

Vertebral Body Lipping: Wild versus captive status is a significant predictor of disease prevalence in all three species for all three vertebral segments. It was found that captive apes suffer significantly more vertebral body lipping than their wild counterparts. Further, age is a significant predictor of disease prevalence, although not in all vertebral segments, with older individuals exhibiting more vertebral body lipping than younger individuals in some vertebral segments.

Eburnation only: There is no evidence that status is a significant predictor of disease prevalence in any species. Age has an effect, but only in the gorilla knee.

Marginal Lipping only: Status is a significant predictor of disease prevalence in all three species for most joints (except the TMJ) where it was found that captive apes suffer significantly more marginal lipping than their wild counterparts. Further, age is a significant predictor of disease prevalence in most joints with older individuals exhibiting more marginal lipping than young individuals.

Hypothesis 2: Sex

Vertebral Body Lipping: There is no evidence that sex is a significant predictor of disease prevalence in any of the three species. Age has some effect in the chimpanzee and orangutan cervical and lumbar vertebrae only, where older individuals exhibit more vertebral body lipping than younger individuals.

Eburnation only: There is no evidence that sex, even after factoring in age, is a significant predictor of disease prevalence in any of the three species.

Marginal Lipping only: There is no evidence that sex is a significant predictor of disease prevalence in any of the three species, while age has an effect in some joints.

Hypothesis 3: Species' Differences

Vertebral Body Lipping: There are significant differences among the three species with chimpanzees being the least affected species in all three vertebral segments. Orangutans are intermediate in the cervical and thoracic vertebrae, while gorillas are the most frequently affected in these segments. Orangutans and gorillas do not differ from

each other in the lumbar vertebrae. Age is also a factor with older individuals exhibiting more vertebral body lipping than younger individuals.

Eburnation only: There is no evidence that there are significant differences among the three species, with or without accounting for age.

Marginal Lipping only: The results are mixed; however, in general, orangutans are the least frequently affected species. Age has an effect in many joints with older individuals exhibiting more marginal lipping than younger individuals.

Research Significance

The results of this research provide insights into several areas: Aging of ape skeletons, diagnostic methods, and disease prevalence and interpretation.

Aging of Ape Skeletons: As mentioned previously, estimating age is frustratingly problematic in adult ape skeletons; however, this research has contributed valuable information to the on-going dilemma of how age in unknown-aged ape specimens may be evaluated. It is clear that the use of the basilar suture to categorize specimens as either adult or old adult is very inexact. The results of this study on the known-aged specimens support the findings of previous research that suture closure in the great apes occurs, except in abnormal cases, shortly after the permanent dentition and thus early in the second decade of life (eg: Schultz 1940, 1969). The age of suture closure in the apes is similar to those found in some baboons where sutures close early after an individual reaches adulthood (Bramblett, 1969). Thus, it is clear that utilizing the basilar suture to assign age categories of young adult and old adult is unwise.

Of the aging techniques analyzed, the results of this study lend support to previous research on both humans and apes that found that dental wear is correlated with age (eg: Molnar, 1971; Lovejoy, 1985; Kilgore, 1989; Morbeck et al., 2002). Indeed, in this study, the model that was developed utilizing molar wear provided the highest level of accuracy in estimating age of all the other aging methods examined. It was found that the aging methods that are commonly used on humans (eg: auricular surface, acetabulum, sternal rib ends) do not work particularly well for the great apes. Of the postcranial markers studied, the acetabulum demonstrated the most potential, while there was generally not enough variation in the ribs and auricular surface of known-aged individuals. However, the small number of observations in this study could be a contributing factor in this assumption.

Overall, the results of this research indicate that many of the senescent changes in ape dentitions and joints are, in fact, correlated with age; although, they do not appear to be as strongly correlated as might be anticipated in order to produce *highly* accurate estimates of the age of wild individuals or captive animals of undocumented age. This could mean that apes and humans follow very different aging trajectories (that make comparisons difficult) that are strongly influenced by life history factors and structural differences pertaining to locomotion. Thus differences between humans and apes and among the three ape species may have a greater impact on the development of aging techniques for aging ape skeletons than was originally anticipated. The analyses were somewhat confounded due to the low number of observations in the sample, and so it is difficult to determine whether skeletal aging patterns vary significantly among species. Nevertheless, the model used in this research, based on wear of molars 1 and 2 and

certain features of the acetabulum, while not perfect, provides the most reliable age estimates currently available. Further, correlation analyses are significant and moderate in strength for nearly all of the markers in this model. This lends further credence that this method is a viable option for predicting age. Nevertheless, it may be wise to analyze more closely the utility of developing aging models that are specific to each species rather than using a generalized model as was done here. Due to the novelty of this research, separate analyses were not considered appropriate at this time.

Diagnostic Methods: This research has highlighted an on-going issue pertaining to which diagnostic criteria should be utilized to analyze OA in skeletal material.

Although the typical features used to diagnose the presence of osteoarthritis include lipping, surface osteophytes, porosity, and eburnation, there remains a lack of consensus on the diagnostic value of each of these markers. Research shows that porosity may be unrelated to osteoarthritis and may occur independently from eburnation (Woods, 1995; Rothschild, 1997; Weiss and Jurmain, 2007), while marginal lipping and eburnation may be affected by discrete genetic and/or physiological mechanisms (Weiss and Jurmain, 2007). Largely because of these issues, some researchers (eg: Jurmain, 1999) have recommended using eburnation only to diagnose the presence of OA. One possible factor is whether the different pathways that affect the skeletal markers apply as well to the great apes as they do to humans. It is conceivable that there are differences in the development of these features between apes and humans; however, it is reasonable to assume that this is not the case. This is because the differences that are commonly noted in humans, such as three different pathways for development of porosity, were also

evident in the ape sample, and also because correlation analyses of the ape sample showed that of those correlations that were significant, they were generally weak. Thus, these findings tend to support the results from the previous research alluded to above and indicate that it may be prudent to use eburnation only to diagnose the presence of OA.

While it appears sensible to utilize eburnation only to diagnose OA, it may also be necessary to adjust the current scoring criteria commonly used. Use of an ordinal scaling system like that suggested by Buikstra & Ubelaker (1994) is considered to be the standard, but even so researchers have not always followed the recommended method of separate scoring for marginal lipping and eburnation nor have they adopted scores that enables analysis by levels of severity. In this study, marginal lipping and eburnation were scored and analyzed separately and the results provide interesting insights. This is because it was found that while marginal lipping was significant in most joints, eburnation was not significant in any joint (by wild or captive status). But, if marginal lipping and eburnation scores had been aggregated, the difference by status would have provided a neat answer to the question of whether wild and captive animals vary in their prevalence of OA (i.e., captive animals would have significantly more disease than wild animals); however, the answer is clearly not that simple. This leads to several interesting questions: first, how wise is it to use aggregate scores for analyzing skeletal markers of disease? It is suggested that aggregate scores do not provide a clear picture of disease prevalence and progression and that separate scoring and analysis should be the standard. Second, what does the difference in prevalence of the skeletal markers analyzed in this study suggest? It is possible that there are, in fact, at least two different diseases in progress. As mentioned elsewhere in this paper, other researchers have suggested that

marginal lipping and eburnation might be under the influence of differing factors (such as genetic, anatomical, and mechanical factors) and that porosity occurs through at least three different pathways. Thus, the results of this research lend support to these findings and provide further evidence of the complexity surrounding this issue. The suggestion made here is that marginal and joint surface changes are likely influenced by different factors and that caution should be used when analyzing these skeletal changes. Third, are the severity scores typically used valid? Analysis of eburnation often utilizes scores of 0 = none; 1 = slight – barely discernible polish; 2 = moderate – clearly discernible, polish only; 3 = severe – polish with grooves (see Buikstra & Ubelaker, 1994). The sample in this study was scored using a severity scale and, even though the small sample size ultimately meant that issues of severity could not be analyzed statistically, an adjustment in the scoring criteria is recommended. This is because research suggests that it appears clear that one of the three pathways by which porosity occurs is via perforation through the articular plate *subsequent* to eburnation (Jurmain, 1999). Further, based on the appearance of some specimens examined in this study, it seems plausible that the type of porosity that is subsequent to eburnation is indicative of a level of severity above that of eburnation with grooves. Thus, it may be more useful to score eburnation as 0 = none; 1 = slight, polish only; 2 = moderate, polish with grooves; 3 = severe, polish with secondary porosity/bone destruction (grooves may or may not be evident depending on the extent of bone destruction).

Disease Prevalence and Interpretation: This research has contributed valuable information that enables a better understanding of disease prevalence in the great apes and also in disease patterns/interpretation in humans. There appears to be a general

assumption that all captive animals will suffer significantly more osteoarthritis than their wild counterparts. This is because previous research appears to support this idea but also because issues of exercise, diet, substrate, and age are often cited as potentially important differences between wild and captive specimens. The results from this study indicate that captive or wild status is a significant predictor of marginal lipping and vertebral body lipping, with captive apes suffering significantly more disease than their wild counterparts (supporting the above assumption); however, status has no effect on the prevalence of eburnation. Thus, it appears that the conditions of captivity do not *necessarily* engender disease development and progression in the great apes. Nevertheless, it is worth examining the various etiological factors that *may* affect osteoarthritis. One factor that is frequently cited as a likely cause of osteoarthritis is the type of substrate to which an animal is commonly exposed. Unlike wild apes, captive apes are exposed to concrete substrate, which is hypothesized to have a deleterious effect on osteoarthritis. However, how much this exposure to concrete affects the skeleton is unclear and is difficult to substantiate. This is because, in the past three decades, the conditions of captivity have improved dramatically from small concrete cages with limited natural substrate to large, naturalistic environments with limited concrete substrate. In addition, while indoor enclosures are primarily made of concrete, they are routinely provided with soft material (such as straw, clothing, and hammocks made from fire hoses) that alleviates prolonged exposure to concrete. Thus, the differences in substrate between wild and captive apes are difficult to quantify and may not be sufficiently different to cause osteoarthritis. This issue could be examined more closely by comparing the skeletons of captive animals housed in concrete-only enclosures to

those housed in naturalistic environments with the idea that those housed in concrete-only enclosure are more likely to exhibit osteoarthritis. However, due to the small sample of captive apes and lack of information available for many of these individuals, the data do not allow for this level of comparison. It is worth pointing out that for captive animals, and contrary to popular opinion, the conditions of captivity vary considerably from group to group, with variation not only in substrate, but also in diet, type of enclosure, level of exercise, and psychosomatic influences to name a few. Indeed, the variation in conditions experienced by captive animals means that potential etiological factors (such as diet, substrate, and exercise) are difficult to test appropriately, and caution should be used when making generalizations and/or assumptions that may not be generalizable to a particular group (i.e., captive or wild).

Even though the captive environment is more varied, and perhaps more naturalistic than is commonly assumed, studies in other non-human primates tend to find that animals in captivity have higher rates of disease than their wild counterparts; although, the overall rates of disease are often low (eg: Rothchild & Woods, 1992a and b). Studies in Old World monkeys (baboons and macaques) found relatively high rates of involvement while studies of wild African apes indicate a low rate of involvement (Bramblett, 1967; DeRousseau, 1988; Lovell, 1990; Jurmain, 2000); although, it is not entirely clear why these differences exist. Jurmain (2000) suggested that a “variety of potential confounders” could produce higher prevalence rates in the Old World monkeys and that one potential complication was that the macaques were from a captive colony. The results of this research tend to support this view because vertebral body lipping and marginal lipping were significantly higher in the captive animals in this study; however,

as alluded to earlier, the issue is not entirely straightforward because captive apes do not have significantly higher rates of eburnation than wild apes. It is worth pointing out that much of the data from Old World monkeys comes from the Cayo Santiago macaque colony. Cayo Santiago is a small island off the coast of Puerto Rico (approximately 16 hectares) that is home to a colony of roughly 1,000 macaques (Primate Cognitive Neuroscience Lab [online] accessed Nov 2009). These macaques are the descendants of around 400 monkeys that were imported onto the island in the early 1930s for the purpose of biomedical experimentation. The animals are considered “free-ranging”; the main difference between the Cayo Santiago macaques and the ‘average’ captive ape group is the availability of space (although the high population density on the island means that this is debatable). The Cayo Santiago monkeys receive regular feedings (although this practice was sporadic from the 1930s to 1960s), are trapped annually for physical examinations, undergo routine experimentation, and are culled “as needed” (Rawlins and Kessler, 1986), and thus these macaques are perhaps best thought of as a captive colony. Whether the macaques experience sufficiently different behaviors/conditions to wild animals that would engender osteoarthritis is debatable. It has been found that the Cayo Santiago macaques use locomotor behaviors on the ground and in branches and vines that correspond to captive monkey locomotion on comparable supports (poles, ropes, etc). It has also been found that, unlike captive monkeys, infant macaques begin to walk using a diagonal-sequence pattern and that swimming is common (Dunbar, 1989). Thus, these macaques have locomotor behaviors that parallel both wild and captive monkeys. With regard to osteoarthritis, data indicate that the Cayo Santiago monkeys experience high rates of spinal and peripheral joint diseases (similar to

the captive gorillas and orangutans in this sample), but it is important to note that methodological differences (i.e., aggregate scores combining marginal lipping and eburnation) may bias the results and thus generalizations are difficult to draw. However, it is likely that the macaque data parallels the captive ape data from this research and, given the conditions experienced by the macaques, this result is not surprising. It is also interesting to note that dietary differences between the Cayo Santiago macaques and the average captive ape group may not be sufficiently different to influence either osteoarthritis or dental wear. This is because both the macaques and captive apes are provisioned with monkey chow, a dry nutritional supplement (somewhat like dry dog food) that is relatively coarse. Food provisions are often distributed in captive enclosures by being tossed into the exhibit which means that grit and dirt are likely to be present. Thus, any differences in food properties are unlikely to be sufficient to bias either dental wear or the prevalence of osteoarthritis. Given the fact that diet is not substantially different between the macaques and captive apes, it is likely that other etiological factors play a greater role in the prevalence of osteoarthritis. But, as pointed out earlier, the issue is complex because levels of eburnation are uniformly low in the apes and captive chimpanzees are less affected than captive orangutans or gorillas.

In terms of disease in the vertebral column, studies in wild chimpanzees, gorillas, and bonobos have found relatively low rates of involvement, with chimpanzees being the least affected and gorillas the most affected of the great apes (eg: Lovell, 1990; Jurmain, 2000). It has been suggested that the high rates of spinal involvement found in humans could be explained by the biomechanical adaptations of bipedality (Jurmain, 2000). One study that compared the African apes to data from two human archaeological sites (one in

Central California (Ala-329) and one from a group of Inuit in Alaska) found that the apes displayed significantly less spinal disease than a human group. For spinal involvement, the Ala-329 sample showed moderate and severe involvement of 31.2% in the cervical vertebrae, 26.2% in the thoracic vertebrae, and 51.7% in the lumbar vertebrae (see Jurmain, 2000 for more details). Table 63 below shows the percentage of individuals in this study affected by moderate and severe vertebral body lipping. Both wild and captive chimpanzees are consistently less involved when compared to the human group, and wild gorillas and orangutans are also consistently less involved when compared to the human group. This is especially true for severe involvement where wild orangutans are the least affected of any species. For severe involvement, captive gorillas and orangutans (with the exception of the cervical vertebrae in gorillas) have levels of disease that are comparable to the human group. It is clear that chimpanzees, regardless of status, are the least frequently affected species when compared to the other apes and to the human sample. Thus, the results from this study support the finding that chimpanzees are the least affected of the great apes, but also indicate that the method of locomotion a species exhibits may not, in fact, be a strong factor in spinal involvement because captive gorillas and orangutans display levels of disease on par with the human group. As mentioned earlier, it has also been reported that in macaques, spinal involvement is relatively frequent, increasing with age (DeRousseau, 1985), which also adds weight to the argument that bipedal locomotion is not necessarily a strong contributing factor in spinal disease. Nevertheless, the paucity of data from nonhuman primates, potential for problems in analysis with reference to methodology (alluded to earlier), and the fact that

chimpanzees, regardless of status, demonstrate low levels of spinal involvement, suggest that the issue is complex and that generalizations are difficult to draw.

Table 63: Percentage of Apes affected with Vertebral Body Lipping

% affected with Vertebral Body Lipping		Captive		Wild	
		Moderate	Severe	Moderate	Severe
Cervical	Chimpanzee	3.7	1.8	5.5	0
	Gorilla	21.4	14.3	8.1	1.6
	Orangutan	14.8	33.3	0	0
Thoracic	Chimpanzee	12.2	0	2	2
	Gorilla	21.4	32.1	9.7	1.6
	Orangutan	16	44	2.7	0
Lumbar	Chimpanzee	22.9	4.2	2.1	2.1
	Gorilla	14.8	44.4	8.2	18
	Orangutan	15.4	57.7	5.3	0

In terms of peripheral joint OA, the issue is also complex. One study that examined prevalence rates of peripheral joint OA in wild great apes and four human groups found that, for moderate and severe involvement of all major joints, the human groups exhibited overall percentages of 4.0 (Ala-329), 34.6 (Inuit), 42.1 (U.S. White, all), and 16.9 (U.S. White, ages 21-50). The study found that, in general, wild apes were less involved than most human groups (this is particularly true for chimpanzees which were less involved than all human groups) (see Jurmain, 2000 for more details). Table 64 below shows the percentage of individuals in this study affected with eburnation of the four major joints (primary and secondary), while Table 65 below shows the human group data taken from Jurmain (2000). The results from this study are similar to those of Jurmain (2000), who reported lower rates of involvement for all three great ape species than those for most human groups. However, it is difficult to draw any further conclusions from this data given the likely differences in the scoring criteria used (i.e.,

the ape data presented here is based on the presence of eburnation only while the human data appears to include scoring from additional skeletal markers (most notably, marginal lipping).

Table 64: Percentage of Apes affected with Eburnation – Appendicular Joints

% affected		Shoulder		Elbow		Hip		Knee	
		Left	Right	Left	Right	Left	Right	Left	Right
Chimpanzee	Captive	0	0	0	0	0	0	1.8	1.8
	Wild	1.7	3.5	3.6	3.5	1.7	0	0	1.7
Gorilla	Captive	3.8	3.7	4.0	3.7	3.7	3.6	8.0	7.7
	Wild	0	0	3.1	1.5	0	0	1.5	4.7
Orangutan	Captive	10	10	0	10.7	0	0	10	13.8
	Wild	0	0	2.3	6.9	0	0	2.6	2.6

Table 65: Percentage of Humans with appendicular joint OA, moderate and severe involvement (taken from Jurmain, 2000)

% affected		Shoulder		Elbow		Hip		Knee	
		Left	Right	Left	Right	Left	Right	Left	Right
Ala-329		1.6	4.0	5.4	5.1	0.7	0.7	6.5	8.7
Inuit		33.3	37.9	36.6	37.9	31.7	28.2	32.4	39.5
U.S. White (all)		53.4	51.5	13.0	15.9	53.9	49.8	34.5	17.2
U.S. White (21-50)		26.3	22.0	1.1	3.5	26.7	24.7	17.2	14.9

One final point on the prevalence rates of spinal and peripheral joint OA in the great apes is that prior to this study it was not known where orangutans would fit. The results show that, while species' differences are not evident in the prevalence of eburnation, for vertebral body lipping, orangutans are intermediate between chimpanzees and gorillas. For marginal lipping, the results were mixed but, in general, orangutans are the least frequently affected species.

In studies that attempt to compare prevalence of osteoarthritis, age must be considered because it is well established that age is a contributing factor in disease

expression. Research in humans consistently demonstrates that OA is strongly related to age, and the results from this study tend to support these findings because, in general, older individuals are more affected than younger individuals; however, age does not appear to be a major factor in the prevalence of eburnation in the apes. There is evidence that age is a factor in the development of vertebral body lipping and marginal lipping; although, not in all joints. Thus the evidence suggests that the conditions of captivity do not engender these diseases to develop at an earlier age. Rather, the general finding is that age is a potential factor in disease progression with older individuals exhibiting more disease than young individuals in both captive and wild settings. The fact that age is not significant in the ape sample in all joints could be due to the differential effects of age among species because humans, on average, live longer and thus are more likely to develop arthritic conditions. Nevertheless, greater longevity in humans does not necessarily explain the variation seen because senescent changes occur in each species relative to that species' life history. Thus, the suggestion is apt that OA is not merely an "old age" disease because it may be absent in an individual of 80 and present in an individual of 35 (Comroe, 1944). Further, it is interesting to note that even in the oldest wild and captive animals of known age, spinal and/or peripheral involvement can be absent. For example, in an examination of the Gombe chimpanzees, Jurmain (2000) found that even the oldest chimpanzees of known age (>40) had no evidence of spinal disease. Likewise, in this study, of those animals of known age, two of the oldest chimpanzees in the sample (48 and 42 years old) did not show any evidence of vertebral body lipping. Indeed, the 48-year-old (the oldest chimpanzee in the captive sample) did not exhibit any evidence of arthritis in any joint. Thus, although age has a well

established pattern of contributing to osteoarthritis, it must be remembered that old age does not necessarily equate to disease presence.

Apart from factoring in age, this study also examined potential differences between the sexes, in part because this is commonly done in human samples. It was also hypothesized that body size may be a confounding factor in sex differences and that a body size effect would be inferred to be present if, after controlling for age, the males in each species had more disease than females *and* there was evidence that species-level differences of disease expression in males and females mirrored species-level differences in sexual dimorphism and, more generally, the differences in average body size between species. Previous research that controlled for body mass has found contradictory results (DeRousseau, 1988; Weiss and Jurmain, 2007) and thus the results from this study help to elucidate these findings. The results show that sex is not a significant predictor of disease prevalence for any skeletal marker tested (vertebral body lipping, marginal lipping, eburnation). Therefore, given the strong sexual dimorphism present in *Gorilla* and *Pongo* and the more moderate dimorphism in *Pan* a body size effect is not inferred to be present in the great apes. This result supports research that has found no significant body size correlation with osteoarthritis.

One other area that needs to be addressed is the merit of using the presence/prevalence of osteoarthritis to interpret behavior and/or activity patterns in human populations (past and present). Anthropologists have a long history of attempting to reconstruct behavior by examining prevalence of osteoarthritis. Although this type of analysis has become considerably less common in recent years, researchers do still use the presence/severity of osteoarthritis to examine issues of activity and adaptive strategies

in past human populations (Lieverse et al., 2007). However, arguments against studying activity patterns have been proposed, particularly in relation to spinal involvement (Knüsel et al., 1997). Thus, opinions appear somewhat divided as to the utility of using osteoarthritis to interpret behavior/activity in humans; although, it should be noted that anthropologists generally are cautious when trying to explain past activity and/or behavior. Nevertheless, a commonly held notion is that a clear cut relationship has been established between levels of activity and arthritis. The basic idea is that chronic overuse and/or long term wear and tear (sometimes called the ‘stress hypothesis’) is a major cause of osteoarthritis. However, clinical literature reveals that there is little consensus regarding the role of repetitive stress on the initiation of primary OA (Radin, 1983; Jurmain, 1999; Otterness et al., 1998), and it is likely that OA is “neither a good predictor of specific activities, nor a good indicator of overall levels of activity” (Weiss and Jurmain, 2007). Jurmain (1999) indicates that several factors should be considered when addressing the issue of mechanical stress and the development of osteoarthritis. These factors are (1) amplitude; (2) periodicity; (3) duration; (4) age of onset; (5) predisposing factors; (6) other systemic influences; and (7) regional variations in loading both between different joints and within joints. The results of this research indicate that some of these other factors should be investigated further. When prevalence rates among captive and wild animals are compared the assumption that chronic overuse leads to osteoarthritis is not clearly supported. This is because captive animals are not expected to experience the same levels of exercise/mechanical stress; rather, the level of activity in captive animals is likely to be significantly less than the level of activity in their wild counterparts. And yet, captive animals exhibit more vertebral body lipping and marginal lipping than their

wild counterparts. One possibility is that the periodicity of activity (how constant the stress is) is a factor in disease development and progression. The suggestion is that the comparable levels of disease seen in captive animals and humans are potentially a result of similarities in periodicity of activity. One possible theory is that humans and captive apes have a tendency to be active in an all-or-nothing way (i.e., short periods of intense exercise followed by long periods of inactivity), while wild animals engage in more constant and steady motion in their daily foraging activities. However, even this suggestion is not easily supported by this research because captive chimpanzees do not exhibit the same levels of disease as captive gorillas/orangutans and humans, and eburnation is not affected by status in any of the ape species. Thus, other systemic factors and/or influences (such as genetics) are likely strong contributors to disease expression. What is clear is that there are no definitive answers. What can be stated with certainty is that the reality is that a clear cut relationship has *not* been established between levels of activity and arthritis; rather, the etiopathogenesis of osteoarthritis is complex and conclusions relating to functional aspects are not likely to be well supported.

Overall, this study has contributed valuable information that helps improve our knowledge of skeletal aging and how status, sex, and age affects disease prevalence in the great apes. These results help elucidate problems associated with aging of adult ape skeletons and the scoring criteria commonly utilized to diagnose OA. Results show that status has an effect on vertebral body lipping and marginal lipping only, while sex has no effect on disease prevalence. Age is a factor with older individuals being more likely to exhibit these diseases than young individuals. Nevertheless, the presence of eburnation is rare in the great apes with very few individuals being affected regardless of status, sex,

species, or age. Thus, the results highlight the complex nature of osteoarthritis and enforce the idea that osteoarthritis is markedly multi-factorial and that disease prevalence and patterns are not easily understood or interpreted.

Future Research

This study highlights three areas of importance for future research. First, the availability of primate skeletal material (at least in the United States and Canada) is heavily biased towards wild-caught animals because these specimens are seen to be of immense value for systematic, functional, and evolutionary studies, while specimens which experienced captivity are thought to be of lesser importance. Comparative studies between wild and captive skeletal specimens are uncommon, which may be partly due to the lack of captive specimens. Thus, facilities that house captive great apes as well as those facilities (such as museums) that house skeletal specimens are encouraged to work closely to ensure a continuing accumulation of captive specimens.

Second, the methods utilized for diagnosing OA in skeletal material can vary, while common skeletal markers used to identify OA may need to be re-evaluated. This is because it is becoming clear that the various skeletal markers most commonly used to diagnose OA in skeletal samples have varying degrees of diagnostic value (Wood, 1995; Rothschild, 1997; Jurmain, 1999, Weiss and Jurmain, 2007). Based on what is currently known, it would appear that eburnation is the ‘best’ marker, although its use in isolation does not entirely escape criticism. Further research into the efficacy of the various skeletal markers used in diagnosing OA in skeletal samples would be useful.

Third, techniques used for aging adult great ape skeletons need to be refined. This is important not only to studies such as this, but to all research that utilizes great ape skeletons. At the very least use of the basilar suture as a means to distinguish between old adults and young adults should be discontinued. Further refinement and development of the aging model presented here is recommended. In particular, it would be useful to utilize known-aged wild animals to examine potential differences in wear rates between wild and captive apes of known age. Given the problems of bias towards old individuals in the known-aged orangutan sample, a re-examination of orangutan dental wear would be worthwhile if more known-aged orangutans, with a wider spread in age, become available for study.

Fourth, further research that examines the multi-factorial nature of osteoarthritis would be useful and, in particular, research that examines potential differences in captive/wild specimens has the potential to contribute valuable information to what is currently known. Given the complexities of the physical and psychological environment experienced by captive apes, research that addresses potential etiological influences will be challenging. However, genetic research will likely provide the most reliable data and may be the most apt given the apparent high heritability found in humans. Nevertheless, the complex nature of osteoarthritis will ensure that ideas and theories relating to its etiology and progression will continue to be challenged.

APPENDIX A: Specimens Examined

Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status
Chimpanzee				Gorilla				Orangutan			
AMNH	10276	0	0	AMNH	99.1.2055	1	1	AMNH	239847	1	0
AMNH	51202	0	1	AMNH	99.1.1577	0	1	AMNH	61585	0	0
AMNH	51278	0	1	AMNH	99.1.1578	1	1	AMNH	140426	0	1
AMNH	51376	1	1	AMNH	99.9425	0	1	AMNH	200898	1	1
AMNH	51377	0	1	AMNH	202932	0	1	AMNH	200900	1	1
AMNH	51379	0	1	AMNH	54091	1	1	ASU	Ben	0	0
AMNH	51381	0	1	AMNH	54090	0	1	ASU	Billy	0	0
AMNH	51382	0	1	AMNH	54092	1	1	CAS	20751	0	0
AMNH	51393	0	1	AMNH	54327	1	1	CAS	3733	0	0
AMNH	51394	0	1	AMNH	90289	0	1	FM	153717	0	0
AMNH	54330	0	1	AMNH	90290	0	1	FM	153732	0	0
AMNH	81854	0	1	AMNH	54355	0	1	FM	153744	1	0
AMNH	90189	0	1	AMNH	54356	1	1	FM	153745	0	0
AMNH	90190	0	1	AMNH	167335	0	1	FM	160018	1	0
AMNH	90191	1	1	AMNH	167338	0	1	FM	168868	0	0
AMNH	90292	1	1	AMNH	81651	0	1	FM	47411	0	0
AMNH	165763	0	0	AMNH	81652	1	1	FM	49832	0	0
AMNH	167341	0	1	AMNH	201459	0	1	FM	57231	0	0
AMNH	167342	0	1	AMNH	201460	0	1	FM	91723	0	0
AMNH	167343	1	1	AMNH	201471	0	1	FM	33533	1	1
AMNH	167344	0	1	AMNH	167339	1	1	FM	33536	1	1
AMNH	167346	0	1	AMNH	167340	1	1	IU	9510182	1	0
AMNH	174860	1	1	AMNH	115609	0	0	IU	BR2	0	0
AMNH	174861	0	1	AMNH	235603	0	0	IU	110245	1	0
AMNH	201469	1	1	AMNH	239597	0	0	IU	9510001	0	0
AMNH	202874	1	0	ASU	Hazel	1	0	Peabody	59940	1	1
APF	None	0	0	ASU	BJ	0	0	Peabody	1482	0	1
ASU	ASU 300	0	0	CAS	4980	0	1	UALR	1995.01	1	0
ASU	ASU 301	0	0	CAS	20943	1	0	UALR	9310878	1	0

Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status
Chimpanzee				Gorilla				Orangutan			
ASU	ASU 302	0	0	FM	126045	0	0	UNM	P-104	0	0
ASU	ASU 304	0	0	FM	180677	0	0	UNM	P-103	0	0
ASU	ASU 305	0	0	FM	57202	0	1	UNM	P-31	1	0
BMNH	1861.7.29.10	0	1	FM	27551	0	1	RBINS	6856	1	0
BMNH	1861.7.49.14	1	1	FM	26065	0	1	RBINS	864B	0	0
BMNH	1864.12.1.7	1	1	FM	18402	0	1	RBINS	4381	0	0
BMNH	1901.8.9.10	0	1	FM	18397	1	1	MCZ	50960	0	1
BMNH	1901.8.9.84	0	1	FM	18396	0	1	MCZ	50958	1	1
BMNH	1939-3366	1	1	FM	16344	0	1	MCZ	37365	1	1
BMNH	1939-3367	1	1	FM	134482	0	0	MCZ	37363	1	1
BMNH	1951.9.27.8	1	1	FM	153779	1	0	MCZ	37362	0	1
BMNH	1968.6.27.1	1	1	FM	163212	0	0	NMNH	270807	1	0
BMNH	1976-437	1	1	FM	180665	1	0	NMNH	588109	1	0
BMNH	ZD 1981.749	1	0	FM	57131	1	0	NMNH	143590	0	1
CAS	9806	0	0	FM	57408	1	0	NMNH	143593	0	1
CAS	26598	0	0	FM	60272	0	0	NMNH	143596	1	1
CAS	26673	1	0	FM	99092	1	0	NMNH	143597	1	1
FM	18409	0	1	FM	135290	0	0	NMNH	143598	1	1
FM	18410	1	1	MCZ	17684	1	1	NMNH	143601	1	1
FM	27529	0	1	MCZ	20038	0	1	NMNH	143602	1	1
FM	27542	1	1	MCZ	20039	0	1	NMNH	A22937	1	1
FM	27552	1	1	MCZ	20043	1	1	NMNH	142170	1	1
FM	44866	0	0	MCZ	23160	0	1	NMNH	142169	1	1
FM	47321	0	0	MCZ	23162	0	1	NMNH	145304	0	1
FM	51319	0	0	MCZ	26850	1	1	NMNH	145302	1	1
FM	127419	0	0	MCZ	23182	0	1	NMNH	145301	0	1
FM	137078	1	0	MCZ	29047	1	1	NMNH	145300	1	1
FM	137079	1	0	MCZ	29048	0	1	BMNH	1973.157	0	0
FM	180116	0	0	MCZ	62393	0	0	BMNH	1845.10.2.1	0	1
IU	10198	1	0	MCZ	29049	0	1	BMNH	1880.4.10.1	0	1
IU	9310916	2	0	MCZ	37264	1	1	BMNH	1880.4.10.2	1	1

Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status
Chimpanzee				Gorilla				Orangutan			
IU	9410306	1	0	MCZ	38017	0	1	BMNH	1939-1006	0	1
IU	9410338	2	0	MCZ	38326	1	1	BMNH	1948.10.25.1	0	1
IU	9510313	1	0	MCZ	57482	0	1	BMNH	1948.7.6.1	1	1
IU	9510332	1	0	MVZ	38930	0	1	BMNH	1948.9.9.2	0	1
IU	9510337	0	0	MVZ	38931	0	1	BMNH	1976.438	1	1
IU	9710291	2	0	MVZ	174521	1	0	BMNH	1986-1092	0	1
IU	9910098	0	0	MVZ	183656	1	0	BMNH	1986-1097	0	1
MCZ	10736	1	1	Peabody	60351	1	1	BMNH	1986-1101	1	1
MCZ	19187	0	1	RBINS	7503	0	1	BMNH	1986-1102	1	1
MCZ	20041	0	1	RBINS	869E	0	0	BMNH	1986-1114	1	1
MCZ	23164	0	1	NMNH	395636	0	1	BMNH	1986-1118	0	1
MCZ	23167	1	1	NMNH	396935	1	1	BMNH	1992.156	0	1
MCZ	26847	1	1	NMNH	395934	0	1	BMNH	2003.362	0	1
MCZ	26849	1	1	NMNH	545039	0	1	BMNH	2003.363	0	1
MCZ	48686	0	1	NMNH	545048	0	1	BMNH	1986.1120.	0	1
MVZ	183658	0	0	NMNH	545042	1	1				
NMNH	176227	1	1	NMNH	545045	1	1				
NMNH	176229	1	1	NMNH	174698	1	1				
NMNH	220062	1	1	UALR	9410342	0	0				
NMNH	220063	1	1	IU	9410343	1	0				
NMNH	220064	1	1	IU	9510003	0	0				
NMNH	236971	1	1	IU	9510181	0	0				
NMNH	256973	0	0	RMCA	17202	1	0				
NMNH	395820	0	1	BMNH	1978-1226	0	0				
NMNH	477333	1	1	BMNH	1981-758	1	0				
NMNH	481803	1	1	BMNH	1981-757	0	0				
Peabody	3942	1	0	BMNH	1948.12.20.2	1	1				
Peabody	60353	0	1	BMNH	1948.3.31.2	1	1				
PFA	999	1	0	BMNH	1948.3.31.1	1	1				
PFA	1002	1	0	BMNH	1976-439	1	1				
PFA	1018	1	0	BMNH	1976-440	1	1				

Location	Specimen ID	Sex	Status	Location	Specimen ID	Sex	Status
Chimpanzee				Gorilla			
PFA	1020	1	0	BMNH	1916.11.1.1	1	1
PFA	1021	1	0	BMNH	1864.12.1.1	1	1
PFA	1024	1	0	BMNH	1864.12.1.5	1	1
PFA	1027	1	0				
PFA	1030	1	0				
PFA	2005	0	0				
PFA	2018	0	0				
PFA	2019	0	0				
PFA	2021	0	0				
PFA	LO	1	0				
PFA	SD	1	0				
PFA	1012	1	0				
RBINS	4387	1	0				
RBINS	none	1	1				
RMCA	7426	1	0				
RMCA	26509	0	0				
RMCA	30660	0	0				
RMCA	93153M1	0	0				
RMCA	9942M02	0	0				
SNOMNH	9032	1	0				
UALR	Mikiba	1	0				
UNM	2005.93.4	0	0				
UNM	2006.61.1	0	0				
UNM	P-30	1	0				

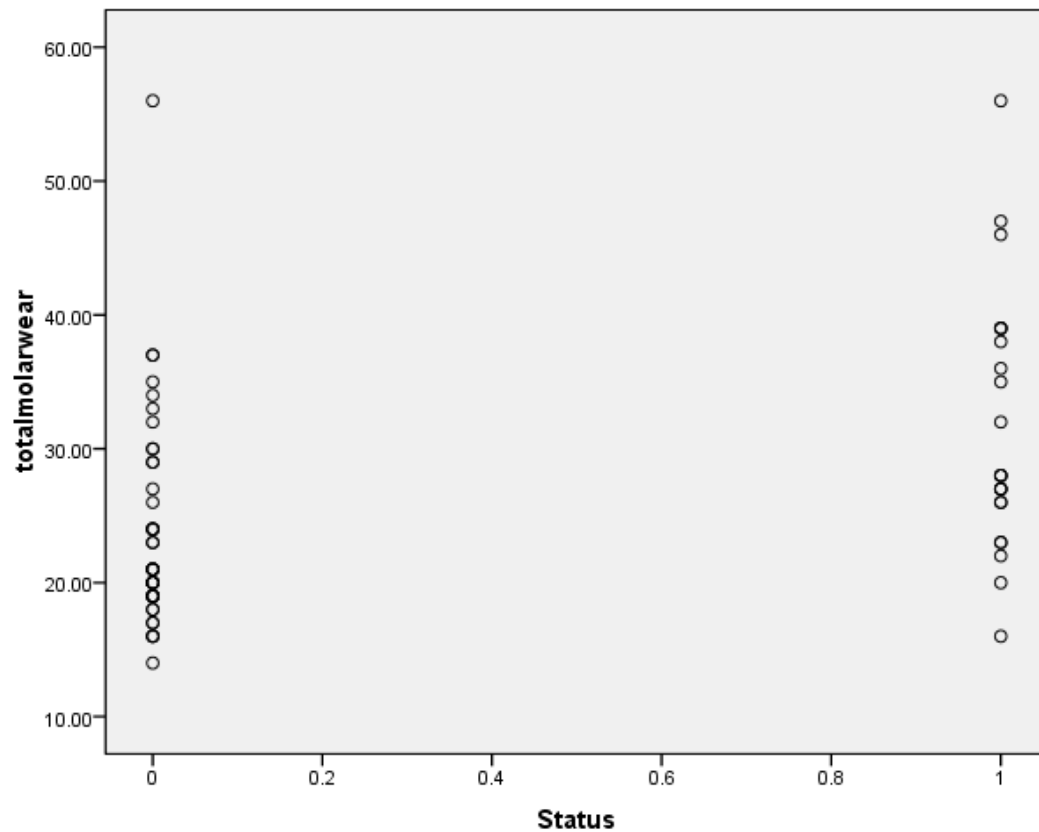
Location: AMNH = American Museum of Natural History, New York; ASU = Arizona State University; CAS = California Academy of Sciences; FM = Field Museum, Chicago; APF = Holloman Primate Facility, Alamogordo, NM; UNM = Maxwell Museum, University of New Mexico; MCZ = Museum of Comparative Zoology, Harvard University; MVZ = Museum of Vertebrate Zoology, UC Berkeley; NMNH = National Museum of Natural History, Washington DC; BMNH = British Museum of Natural History, London; Peabody = Peabody Museum, Harvard University; PFA = Primate Foundation of Arizona; RBINS = Royal Belgian Institute of Natural Sciences, Belgium; RMCA = Royal Museum of Central Africa, Belgium; SNOMNH = Sam Noble Oklahoma Museum of Natural History, Oklahoma; UALR = University of Arkansas at Little Rock; IU = William R. Adams Primate Skeletal Collection, Indiana University.

Status: 0 = captive, 1 = wild. Sex: 0 = male, 1 = female, 2 = unknown

APPENDIX B: Dental Wear Scatter Plots and Box Plots by Status

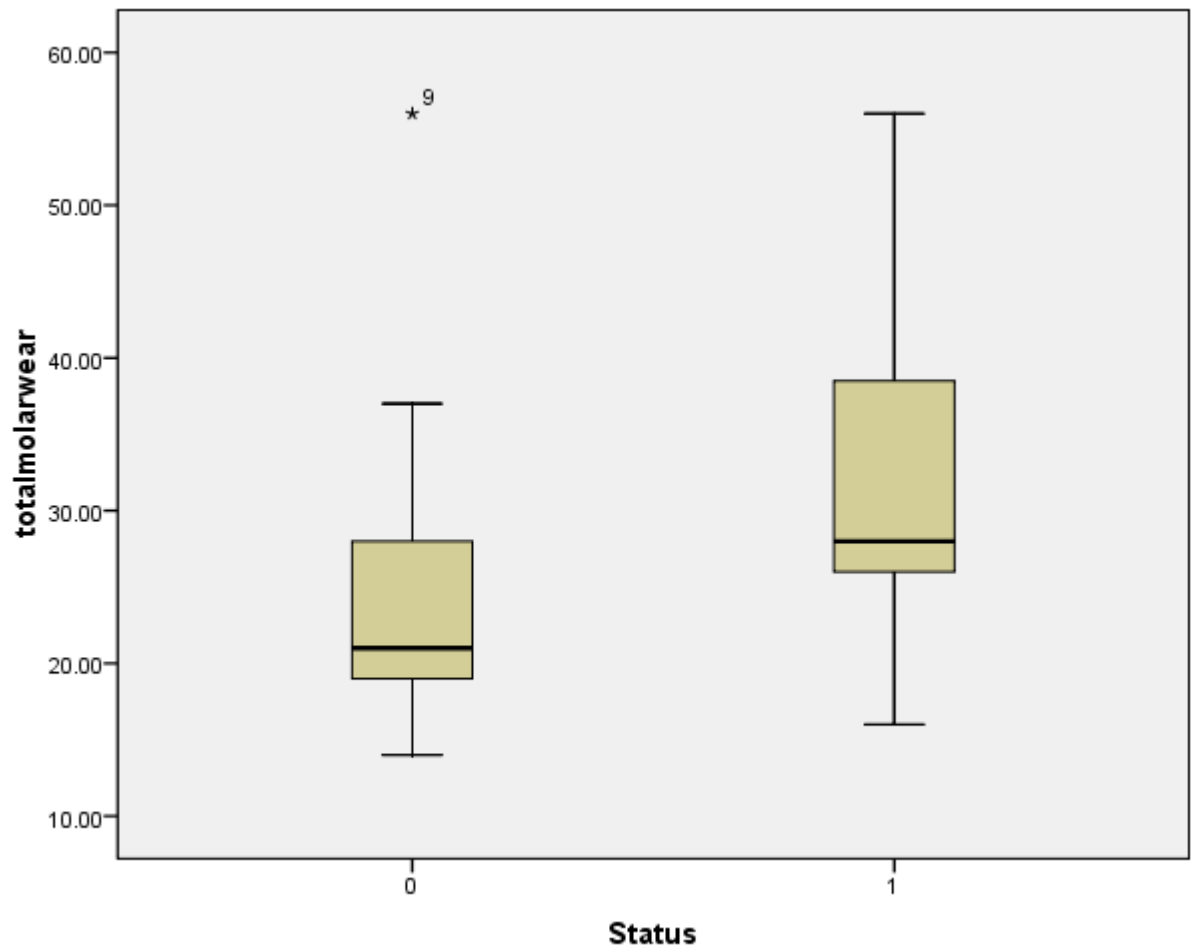
Total molar wear was calculated by summing the scores for each tooth (scale = 0-8, as specified in tables 14 and 15 of Chapter 3) for all 12 molars for each individual.

Chimpanzee total molar wear scatter plot by status:



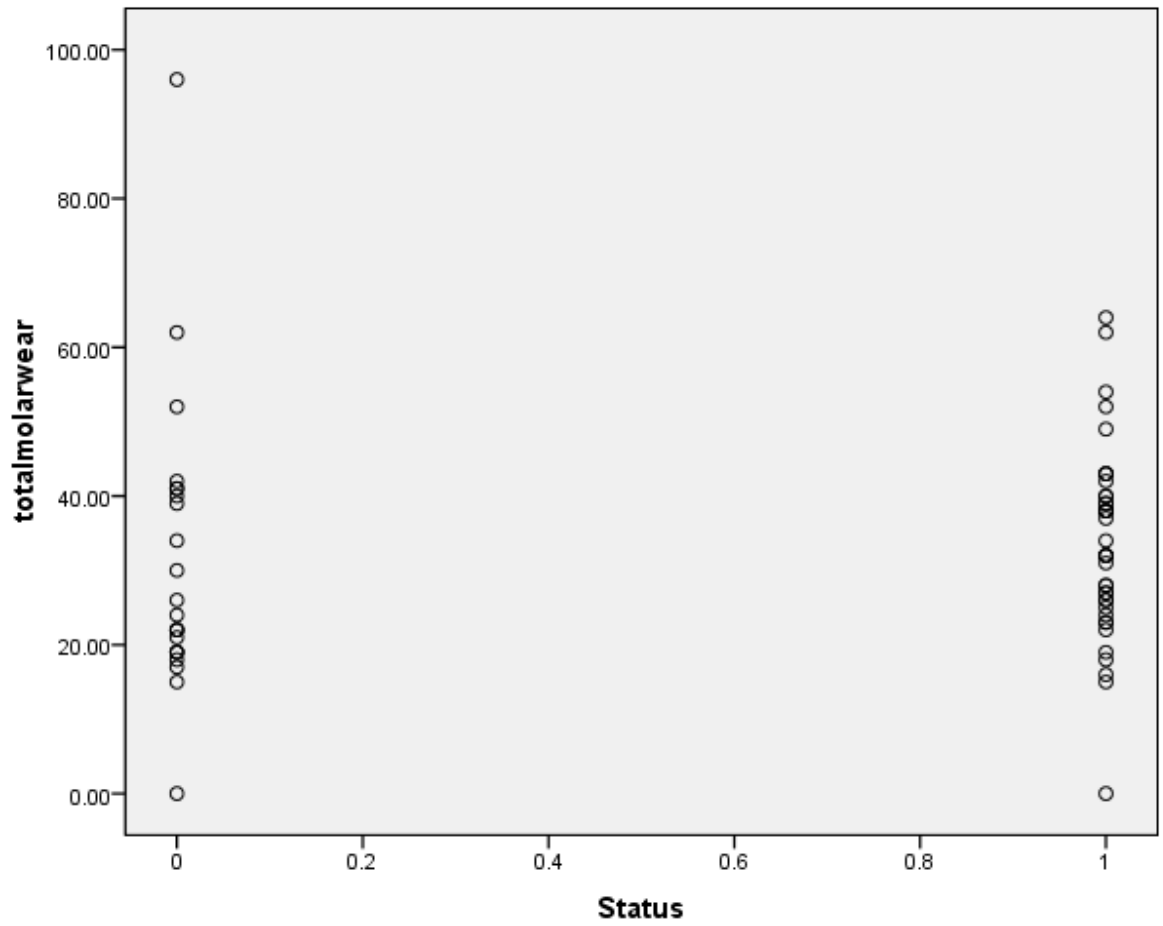
Status: 0 = captive; 1 = wild

Chimpanzee total molar wear box plot by status:



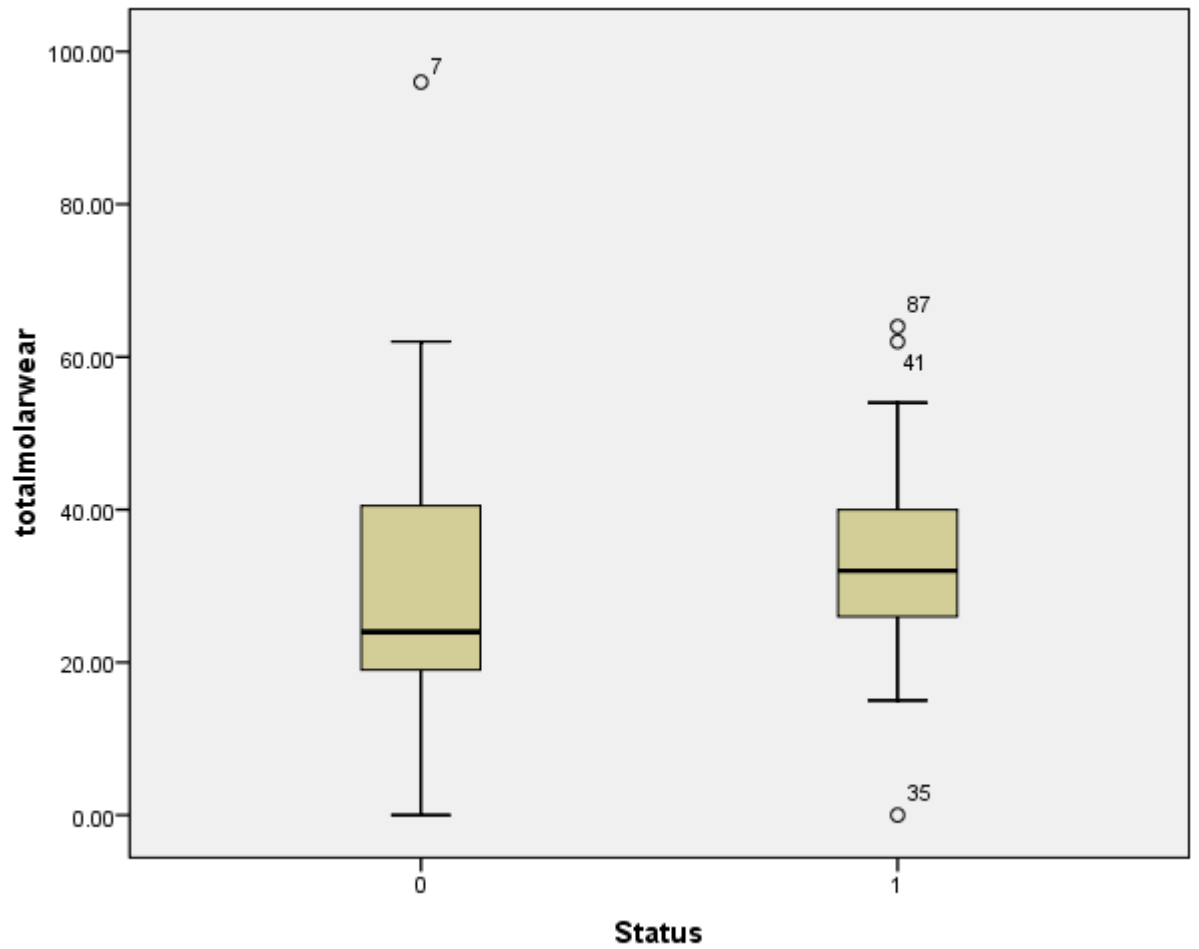
Status: 0 = captive; 1 = wild

Gorilla total molar wear scatter plot by status:



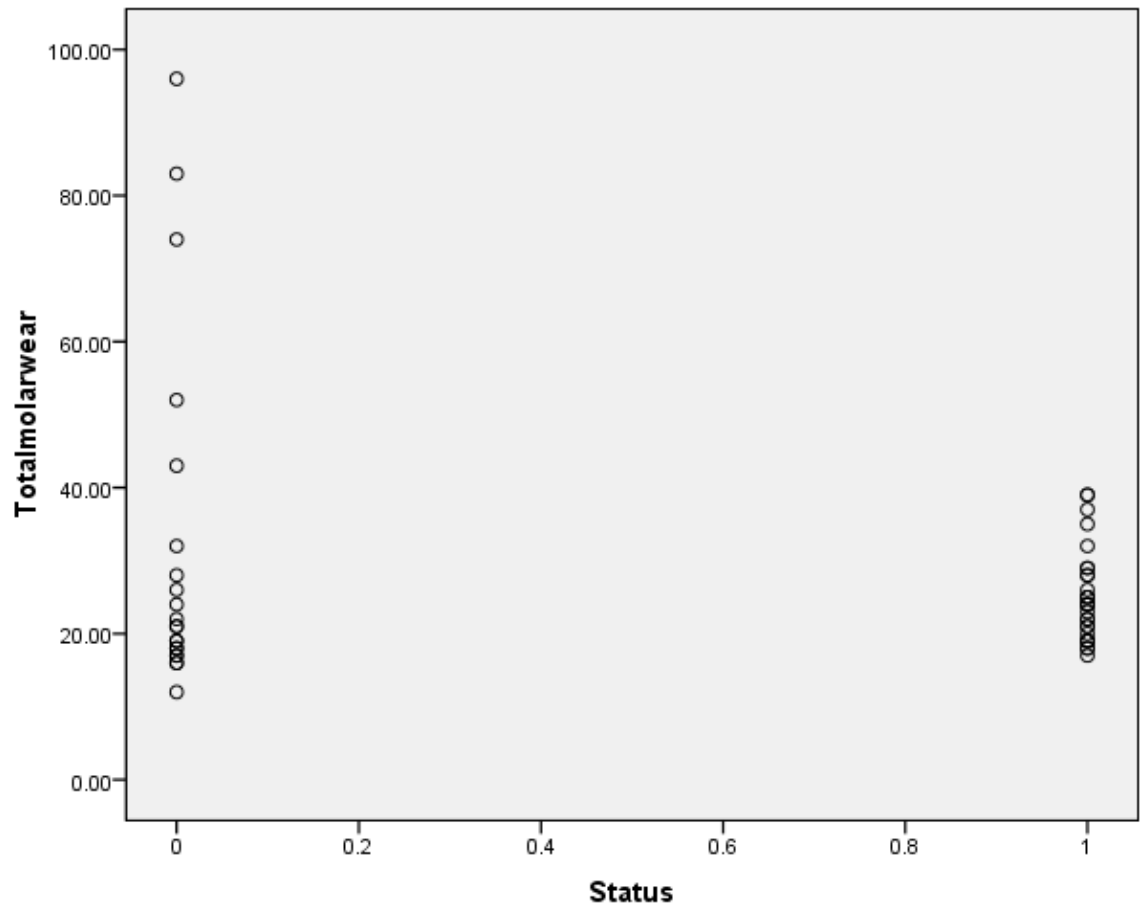
Status: 0 = captive; 1 = wild

Gorilla total molar wear box plot by status:



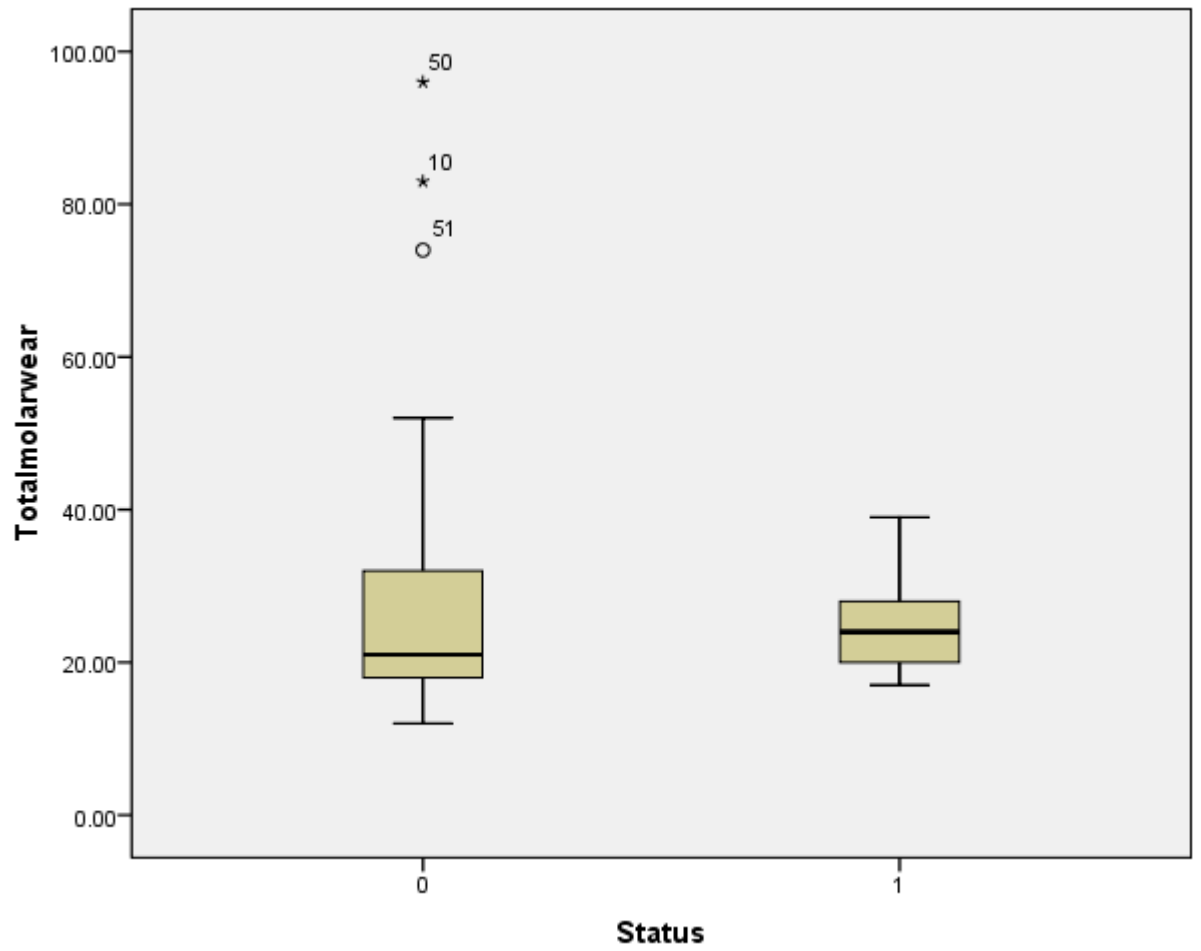
Status: 0 = captive; 1 = wild

Orangutan total molar wear scatter plot by status



Status: 0 = captive; 1 = wild

Orangutan total molar wear box plot by status



Status: 0 = captive; 1 = wild

APPENDIX C: Predicted Age Utilizing Molar Wear (Molars 1 and 2) and Acetabulum

The following tables list the specimen ID number, species, sex (0 = male, 1 = female), status (0 = captive, 1 = wild), known age, predicted age, and age category assignment for all specimens examined.

Chimpanzee

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
AMNH 10276	Pan t	0	0	-	12	2
AMNH 165763	Pan t	0	0	-	19	2
AMNH 202874	Pan t	1	0	-	14	2
APF Brooks	Pan t	0	0	22	22	3
ASU 300	Pan t	0	0	-	15	2
ASU 301	Pan t	0	0	-	21	3
ASU 302	Pan t	0	0	10	10	2
ASU 304	Pan t	0	0	15	15	2
ASU 305	Pan t	0	0	22	22	3
BMNH ZD 1981.749	Pan	1	0	-	27	3
CAS 26598	Pan t	0	0	45	45	4
CAS 26673	Pan t	1	0	24	24	3
CAS 9806	Pan t	0	0	-	26	3
FM 127419	Pan t	0	0	20	20	3
FM 137078	Pan t	1	0	34	34	4
FM 137079	Pan t	1	0	-	33	4
FM 180116	Pan t	0	0	24	24	3
FM 44866	Pan t	0	0	-	20	3
FM 47321	Pan t	0	0	-	15	2
FM 51319	Pan t	0	0	10	10	2
IU 10198	Pan t	1	0	42.1	42	4
IU 9310916	Pan t	2	0	-	14	2
IU 9410306	Pan t	1	0	-	28	3
IU 9410338	Pan t	2	0	-	13	2
IU 9510313	Pan t	1	0	48	48	4
IU 9510332	Pan t	1	0	-	29	3
IU 9510337	Pan t	0	0	-	15	2
IU 9710291	Pan t	2	0	-	17	2
IU 9910098	Pan t	0	0	-	14	2
MVZ 183658	Pan t	0	0	30	30	3
NMNH 256973	Pan t	0	0	8	8	2
Peabody 3942	Pan	1	0	-	29	3
PFA 1002	Pan t	1	0	19	19	2
PFA 1012	Pan t	1	0	34	34	4
PFA 1018	Pan t	1	0	22	22	3
PFA 1020	Pan tt	1	0	30	30	3

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
PFA 1021	Pan tv	1	0	25	25	3
PFA 1024	Pan tv	1	0	18.78	19	2
PFA 1027	Pan t	1	0	14	14	2
PFA 1030	Pan t	1	0	17	17	2
PFA 2005	Pan t	0	0	27	27	3
PFA 2018	Pan t	0	0	12	12	2
PFA 2019	Pan t	0	0	16	16	2
PFA 2021	Pan t	0	0	18	18	2
PFA 999	Pan t	1	0	20	20	3
PFA LO	Pan t	1	0	-	23	3
PFA SD	Pan t	1	0	-	26	3
RBINS 4387	Pan t	1	0	-	14	2
RMCA 26509	Pan t	0	0	-	28	3
RMCA 30660	Pan t	0	0	-	33	4
RMCA 7426	Pan ts	1	0	-	23	3
RMCA 93153M1	Pan t	0	0	-	25	3
RMCA 9942MO2	Pan t	0	0	-	26	3
SNOMNH 9032	Pan t	1	0	17	17	2
UALR Mikiba	Pan t	1	0	10	10	2
UNM 2005.93.4	Pan t	0	0	31	31	4
UNM 2006.61.1	Pan t	0	0	30	30	3
UNM P-30	Pan t	1	0	-	38	4
AMNH 167341	Pan tt	0	1	-	30	3
AMNH 167342	Pan tt	0	1	-	34	4
AMNH 167343	Pan tt	1	1	-	26	3
AMNH 167344	Pan tt	0	1	-	38	4
AMNH 167346	Pan tt	0	1	-	23	3
AMNH 174860	Pan tt	1	1	-	31	4
AMNH 174861	Pan tt	0	1	-	37	4
AMNH 201469	Pan tt	1	1	-	37	3
AMNH 51202	Pan ts	0	1	-	23	3
AMNH 51278	Pan ts	0	1	-	11	2
AMNH 51376	Pan ts	1	1	-	17	2
AMNH 51377	Pan ts	0	1	-	13	2
AMNH 51379	Pan ts	0	1	-	30	3
AMNH 51381	Pan ts	0	1	-	15	2
AMNH 51382	Pan ts	0	1	-	31	4
AMNH 51393	Pan ts	0	1	-	12	2
AMNH 51394	Pan ts	0	1	-	40	4
AMNH 54330	Pan tt	0	1	-	38	4
AMNH 81854	Pan ts	0	1	-	19	2
AMNH 90189	Pan tt	0	1	-	29	3
AMNH 90190	Pan tt	0	1	-	27	3
AMNH 90191	Pan tt	1	1	-	24	3

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
AMNH 90292	Pan tt	1	1	-	24	3
BMNH 1861.7.29.10	Pan tt	0	1	-	27	3
BMNH 1861.7.49.14	Pan tt	1	1	-	11	2
BMNH 1864.12.1.7	Pan	1	1	-	19	2
BMNH 1901.8.9.10	Pan ts	0	1	-	38	4
BMNH 1901.8.9.84	Pan	0	1	-	31	4
BMNH 1939-3366	Pan tt	1	1	-	27	3
BMNH 1939-3367	Pan tt	1	1	-	17	2
BMNH 1951.9.27.8	Pan tt	1	1	-	38	4
BMNH 1968.6.27.1	Pan tt	1	1	-	21	3
BMNH 1976-437	Pan tt	1	1	-	13	2
FM 18409	Pan t	0	1	-	15	2
FM 18410	Pan t	1	1	-	23	3
FM 27529	Pan ts	0	1	-	24	3
FM 27542	Pan t	1	1	-	15	2
FM 27552	Pan ts	1	1	-	29	3
MCZ 10736	Pan t	1	1	-	19	2
MCZ 19187	Pan t	0	1	-	10	2
MCZ 20041	Pan t	0	1	-	10	2
MCZ 23164	Pan t	0	1	-	15	2
MCZ 23167	Pan t	1	1	-	14	2
MCZ 26847	Pan t	1	1	-	43	4
MCZ 26849	Pan t	1	1	-	28	3
MCZ 48686	Pan t	0	1	-	11	2
NMNH 176227	Pan t	1	1	-	21	3
NMNH 176229	Pan t	1	1	-	19	2
NMNH 220062	Pan t	1	1	-	20	3
NMNH 220063	Pan t	1	1	-	33	4
NMNH 220064	Pan t	1	1	-	16	2
NMNH 236971	Pan t	1	1	-	12	2
NMNH 395820	Pan t	0	1	-	16	2
NMNH 477333	Pan t	1	1	-	28	3
NMNH 481803	Pan t	1	1	-	17	2
Peabody 60353	Pan tt	0	1	-	14	2
RBINS (Marit)	Pan t	1	1	-	25	3

Gorilla						
Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
AMNH 115609	Gorilla	0	0	15.5	16	2
AMNH 235603	Gorilla	0	0	22	22	3
AMNH 239597	Ggg	0	0	14	14	2
ASU BJ	Ggg	0	0	20	20	3
ASU Hazel	Ggg	1	0	31	31	4
BMNH 1978-1226	Ggg	0	0	32	32	4
BMNH 1981.757	Gorilla	0	0	-	15	2
BMNH 1981.758	Gorilla	1	0	-	15	2
CAS 20943	Ggg	1	0	22	22	3
FM 126045	Ggg	0	0	38	38	4
FM 134482	Ggg	0	0	25	25	3
FM 135290	Ggg	0	0	20	20	3
FM 153779	Ggg	1	0	17	17	2
FM 163212	Ggg	0	0	13	13	2
FM 180665	Ggg	1	0	13.5	14	2
FM 180677	Ggg	0	0	39	39	4
FM 57131	Gorilla	1	0	13	13	2
FM 57408	Ggg	1	0	16	16	2
FM 60272	Ggg	0	0	23	23	3
FM 99092	Ggg	1	0	16.5	16	2
IU 9410343	Ggg	1	0	-	14	2
IU 9510003	Ggg	0	0	-	32	4
IU 9510181	Gorilla	0	0	-	32	4
MCZ 62393	Ggg	0	0	38	38	4
MVZ 174521	Ggg	1	0	27	27	3
MVZ 183656	Ggg	1	0	15	15	2
RBINS 869E	Ggg	0	0	-	31	4
RMCA 17202	Ggg	1	0	-	15	2
UALR 9410342	Ggg	0	0	28	28	3
AMNH 167335	Gorilla	0	1	-	12	2
AMNH 167338	Ggg	0	1	-	18	2
AMNH 167339	Ggg	1	1	-	10	2
AMNH 167340	Ggg	1	1	-	24	3
AMNH 201459	Gorilla	0	1	-	25	3
AMNH 201460	Ggg	0	1	-	23	3
AMNH 201471	Ggg	0	1	-	23	3
AMNH 202932	Ggg	0	1	-	31	4
AMNH 54090	Gorilla	0	1	-	30	3
AMNH 54091	Ggb	1	1	-	10	2
AMNH 54092	Ggb	1	1	-	22	3
AMNH 54327	Ggg	1	1	-	30	3
AMNH 54355	Ggg	0	1	-	25	3
AMNH 54356	Ggg	1	1	-	25	3

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
AMNH 81651	Ggg	0	1	-	30	3
AMNH 81652	Ggg	1	1	-	20	3
AMNH 90289	Ggg	0	1	-	21	3
AMNH 90290	Ggg	0	1	-	25	3
AMNH 99.1.1577	Gorilla	0	1	-	32	4
AMNH 99.1.1578	Gorilla	1	1	-	27	3
AMNH 99.1.2055	Gorilla	1	1	-	23	3
AMNH 99.9425	Gorilla	0	1	-	31	4
BMNH 1864.12.1.1	Gorilla	1	1	-	12	2
BMNH 1864.12.1.5	Ggg	1	1	-	12	2
BMNH 1916.11.1.1	Ggg	1	1	-	26	3
BMNH 1948.12.20.2	Ggg	1	1	-	21	3
BMNH 1948.3.31.1	Ggg	1	1	-	23	3
BMNH 1948.3.31.2	Ggg	1	1	-	28	3
BMNH 1976.439	Gorilla	1	1	-	10	2
BMNH 1976.440	Gorilla	1	1	-	14	2
CAS 4980	Ggb	0	1	-	23	3
FM 16344	Ggg	0	1	-	26	3
FM 18396	Ggg	0	1	-	20	3
FM 18397	Ggg	1	1	-	11	2
FM 18402	Ggg	0	1	-	23	3
FM 26065	Ggb	0	1	-	17	2
FM 27551	Ggb	0	1	-	36	4
FM 57202	Ggg	0	1	-	21	3
MCZ 17684	Ggg	1	1	-	43	4
MCZ 20038	Ggg	0	1	-	17	2
MCZ 20039	Ggg	0	1	-	23	3
MCZ 20043	Ggg	1	1	-	24	3
MCZ 23160	Ggg	0	1	-	20	3
MCZ 23162	Ggg	0	1	-	37	4
MCZ 23182	Ggb	0	1	-	24	3
MCZ 26850	Ggg	1	1	-	19	2
MCZ 29047	Ggg	1	1	-	28	3
MCZ 29048	Ggg	0	1	-	36	4
MCZ 29049	Ggg	0	1	-	15	2
MCZ 37264	Ggg	1	1	-	30	3
MCZ 37326	Ggg	1	1	-	16	2
MCZ 38017	Gorilla	0	1	-	15	2
MCZ 57482	Ggg	0	1	-	20	3
MVZ 38930	Ggg	0	1	-	21	3
MVZ 38931	Ggg	0	1	-	21	3
NMNH 174698	Ggb	1	1	-	25	3
NMNH 395636	Ggb	0	1	-	17	2
NMNH 395934	Ggb	0	1	-	11	2

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
NMNH 396935	Ggb	1	1	-	17	2
NMNH 545039	Ggb	0	1	-	18	2
NMNH 545042	Gorilla	1	1	-	29	3
NMNH 545045	Ggb	1	1	-	22	3
NMNH 545048	Ggb	0	1	-	22	3
Peabody 60351	Gorilla	1	1	-	15	2
RBINS 7503	Ggb	0	1	-	32	4

Orangutan:

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
AMNH 239847	P sp.	1	0	-	26	3
AMNH 61585	Pp	0	0	-	23	3
ASU Ben	P sp.	0	0	17.5	18	2
ASU Billy	Pp	0	0	38.5	38	4
BMNH 1973.157	Pp	0	0	14	14	2
CAS 20751	P sp.	0	0	22	22	3
CAS 3733	P sp.	0	0	32	32	4
FM 153717	Pp	0	0	34	34	4
FM 153732	Pp	0	0	31	31	4
FM 153744	Pp	1	0	-	34	4
FM 153745	Pp	0	0	-	29	3
FM 160018	Pp	1	0	45	45	4
FM 168868	Pp	0	0	36	36	4
FM 47411	Pp	0	0	-	23	3
FM 49832	Pp	0	0	-	10	2
FM 57231	Pp	0	0	26	26	3
FM 91723	Pp	0	0	13	13	2
IU 110245	Pp	1	0	31	31	4
IU 9510001	Pp	0	0	34	34	4
IU 9510182	P sp.	1	0	-	25	3
IU BR2	P sp.	0	0	-	15	2
NMNH 270807	Pa	1	0	-	24	3
NMNH 588109	Pp	1	0	32	32	4
RBINS 4381	Pp	0	0	-	23	3
RBINS 6856	Pp	1	0	-	30	3
RBINS 864B	Pp	0	0	-	23	3
UALR 1995-01	P sp.	1	0	38	38	4
UALR 9310878	Pp	1	0	30	30	3
UNM P-103	P sp.	0	0	-	33	4
UNM P-104	P sp.	0	0	-	36	4
UNM P-31	Pp	1	0	14	14	2
AMNH 140426	P sp.	0	1	-	32	4
AMNH 200898	Pp	1	1	-	41	4
AMNH 200900	Pp	1	1	-	31	4
BMNH 1845.10.2.1	Pp	0	1	-	44	4
BMNH 1880.4.10.1	Pp	0	1	-	15	2
BMNH 1880.4.10.2	Pp	1	1	-	15	2
BMNH 1939-1006	Pp	0	1	-	36	4
BMNH 1948.10.25.1	Pa	0	1	-	28	3
BMNH 1948.7.6.1	Pa	1	1	-	33	4
BMNH 1948.9.9.2	Pa	0	1	-	21	3
BMNH 1976.438	Pp	1	1	-	20	3
BMNH 1986-1092	Pp	0	1	-	30	3

Specimen ID	Sp.	Sex	Status	Known Age	Predicted Age	Age Category
BMNH 1986-1097	Pp	0	1	-	24	3
BMNH 1986-1101	Pp	1	1	-	19	2
BMNH 1986-1114	Pp	1	1	-	28	3
BMNH 1986-1118	Pp	0	1	-	23	3
BMNH 1986.1102	Pp	1	1	-	27	3
BMNH 1986.1120	Pp	0	1	-	24	3
BMNH 1992.156	P sp.	0	1	-	35	4
BMNH 2003.362	P sp.	0	1	-	26	3
BMNH 2003.363	P sp.	0	1	-	51	4
FM 33533	P sp.	1	1	-	35	4
FM 33536	P sp.	1	1	-	38	4
MCZ 37362	Pp	0	1	-	38	4
MCZ 37363	Pp	1	1	-	27	3
MCZ 37365	Pp	1	1	-	34	4
MCZ 50958	Pp	1	1	-	25	3
MCZ 50960	Pp	0	1	-	22	3
NMNH 142169	Pp	1	1	-	22	3
NMNH 142170	Pp	1	1	-	22	3
NMNH 143590	Pa	0	1	-	33	4
NMNH 143593	Pa	0	1	-	22	3
NMNH 143596	Pa	1	1	-	19	2
NMNH 143597	Pa	1	1	-	16	2
NMNH 143598	Pa	1	1	-	22	3
NMNH 143601	Pa	1	1	-	20	3
NMNH 143602	Pa	1	1	-	29	3
NMNH 145300	Pp	1	1	-	32	4
NMNH 145301	Pp	0	1	-	22	3
NMNH 145302	Pp	1	1	-	19	2
NMNH 145304	Pp	0	1	-	31	4
NMNH A22937	Pa	1	1	-	21	3
Peabody 1482	Pp	0	1	-	24	3
Peabody 59940	Pp	1	1	-	22	3

APPENDIX D – Joint Marker Correlations for Shoulder, Elbow, Hip, and Knee

Note: Most of these correlations could be described as low in magnitude, but positive.

Chimpanzee Joint Marker Correlation Results:

Shoulder:

Correlations

			SMLR	SSPR	SSOR	SSER
Kendall's tau_b	SMLR	Correlation Coefficient	1.000	.483**	.256**	.264**
		Sig. (2-tailed)	.	.000	.005	.004
		N	113	113	113	113
	SSPR	Correlation Coefficient	.483**	1.000	.293**	.201*
		Sig. (2-tailed)	.000	.	.001	.024
		N	113	113	113	113
	SSOR	Correlation Coefficient	.256**	.293**	1.000	.411**
		Sig. (2-tailed)	.005	.001	.	.000
		N	113	113	113	113
	SSER	Correlation Coefficient	.264**	.201*	.411**	1.000
		Sig. (2-tailed)	.004	.024	.000	.
		N	113	113	113	113

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburation)

The last letter refers to L for left or R for right

Shoulder:

Correlations

			SSPL	SSOL	SSEL	SMLR
Kendall's tau_b	SSPL	Correlation Coefficient	1.000	.203 [*]	.155	.534 ^{**}
		Sig. (2-tailed)	.	.021	.080	.000
		N	115	115	115	113
	SSOL	Correlation Coefficient	.203 [*]	1.000	.370 ^{**}	.228 [*]
		Sig. (2-tailed)	.021	.	.000	.011
		N	115	115	115	113
	SSEL	Correlation Coefficient	.155	.370 ^{**}	1.000	.186 [*]
		Sig. (2-tailed)	.080	.000	.	.040
		N	115	115	115	113
	SMLR	Correlation Coefficient	.534 ^{**}	.228 [*]	.186 [*]	1.000
		Sig. (2-tailed)	.000	.011	.040	.
		N	113	113	113	113

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Elbow:

Correlations

			EMLL	ESPL	ESOL	ESEL
Kendall's tau_b	EMLL	Correlation Coefficient	1.000	.374**	.242**	.234**
		Sig. (2-tailed)	.	.000	.006	.009
		N	112	112	112	112
	ESPL	Correlation Coefficient	.374**	1.000	.197*	.198*
		Sig. (2-tailed)	.000	.	.026	.028
		N	112	112	112	112
	ESOL	Correlation Coefficient	.242**	.197*	1.000	.077
		Sig. (2-tailed)	.006	.026	.	.410
		N	112	112	112	112
	ESEL	Correlation Coefficient	.234**	.198*	.077	1.000
		Sig. (2-tailed)	.009	.028	.410	.
		N	112	112	112	112

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Elbow:

Correlations

			EMLR	ESPR	ESOR	ESER
Kendall's tau_b	EMLR	Correlation Coefficient	1.000	.405**	.330**	.247**
		Sig. (2-tailed)	.	.000	.000	.006
		N	112	112	112	112
	ESPR	Correlation Coefficient	.405**	1.000	.100	.201*
		Sig. (2-tailed)	.000	.	.247	.024
		N	112	112	112	112
	ESOR	Correlation Coefficient	.330**	.100	1.000	.207*
		Sig. (2-tailed)	.000	.247	.	.025
		N	112	112	112	112
	ESER	Correlation Coefficient	.247**	.201*	.207*	1.000
		Sig. (2-tailed)	.006	.024	.025	.
		N	112	112	112	112

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Hip:

Correlations

			HMLL	HSPL	HSOL	HSEL
Kendall's tau_b	HMLL	Correlation Coefficient	1.000	.419**	.473**	.
		Sig. (2-tailed)	.	.000	.000	.
		N	114	114	114	114
	HSPL	Correlation Coefficient	.419**	1.000	.398**	.
		Sig. (2-tailed)	.000	.	.000	.
		N	114	114	114	114
	HSOL	Correlation Coefficient	.473**	.398**	1.000	.
		Sig. (2-tailed)	.000	.000	.	.
		N	114	114	114	114
	HSEL	Correlation Coefficient
		Sig. (2-tailed)
		N	114	114	114	114

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Hip:

Correlations

			HMLR	HSPR	HSOR	HSER
Kendall's tau_b	HMLR	Correlation Coefficient	1.000	.403**	.388**	.176
		Sig. (2-tailed)	.	.000	.000	.055
		N	113	113	113	113
	HSPR	Correlation Coefficient	.403**	1.000	.381**	.197*
		Sig. (2-tailed)	.000	.	.000	.030
		N	113	113	113	113
	HSOR	Correlation Coefficient	.388**	.381**	1.000	.277**
		Sig. (2-tailed)	.000	.000	.	.003
		N	113	113	113	113
	HSER	Correlation Coefficient	.176	.197*	.277**	1.000
		Sig. (2-tailed)	.055	.030	.003	.
		N	113	113	113	113

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Knee:

Correlations

			KMLL	KSPL	KSOL	KSEL
Kendall's tau_b	KMLL	Correlation Coefficient	1.000	.445**	.365**	.207*
		Sig. (2-tailed)	.	.000	.000	.025
		N	112	112	112	112
	KSPL	Correlation Coefficient	.445**	1.000	.247**	.183*
		Sig. (2-tailed)	.000	.	.007	.044
		N	112	112	112	112
	KSOL	Correlation Coefficient	.365**	.247**	1.000	-.023
		Sig. (2-tailed)	.000	.007	.	.812
		N	112	112	112	112
	KSEL	Correlation Coefficient	.207*	.183*	-.023	1.000
		Sig. (2-tailed)	.025	.044	.812	.
		N	112	112	112	112

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Knee:

Correlations

			KMLR	KSPR	KSOR	KSER
Kendall's tau_b	KMLR	Correlation Coefficient	1.000	.298**	.311**	.133
		Sig. (2-tailed)	.	.001	.001	.156
		N	110	110	110	110
	KSPR	Correlation Coefficient	.298**	1.000	.095	.096
		Sig. (2-tailed)	.001	.	.302	.298
		N	110	110	110	110
	KSOR	Correlation Coefficient	.311**	.095	1.000	-.026
		Sig. (2-tailed)	.001	.302	.	.783
		N	110	110	110	110
	KSER	Correlation Coefficient	.133	.096	-.026	1.000
		Sig. (2-tailed)	.156	.298	.783	.
		N	110	110	110	110

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Gorilla – Joint Marker Correlation Results:

Shoulder:

Correlations

			SMLL	SSPL	SSOL	SSEL
Kendall's tau_b	SMLL	Correlation Coefficient	1.000	.386**	.339**	.216*
		Sig. (2-tailed)	.	.000	.001	.033
		N	91	91	91	91
	SSPL	Correlation Coefficient	.386**	1.000	.281**	.133
		Sig. (2-tailed)	.000	.	.004	.180
		N	91	91	91	91
	SSOL	Correlation Coefficient	.339**	.281**	1.000	.330**
		Sig. (2-tailed)	.001	.004	.	.002
		N	91	91	91	91
	SSEL	Correlation Coefficient	.216*	.133	.330**	1.000
		Sig. (2-tailed)	.033	.180	.002	.
		N	91	91	91	91

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Shoulder:

Correlations

			SMLR	SSPR	SSOR	SSER
Kendall's tau_b	SMLR	Correlation Coefficient	1.000	.385**	.357**	.219*
		Sig. (2-tailed)	.	.000	.000	.032
		N	91	91	91	91
	SSPR	Correlation Coefficient	.385**	1.000	.335**	.140
		Sig. (2-tailed)	.000	.	.001	.158
		N	91	91	91	91
	SSOR	Correlation Coefficient	.357**	.335**	1.000	.363**
		Sig. (2-tailed)	.000	.001	.	.000
		N	91	91	91	91
	SSER	Correlation Coefficient	.219*	.140	.363**	1.000
		Sig. (2-tailed)	.032	.158	.000	.
		N	91	91	91	91

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Elbow:

Correlations

			EMLL	ESPL	ESOL	ESEL
Kendall's tau_b	EMLL	Correlation Coefficient	1.000	.317**	.374**	.227*
		Sig. (2-tailed)	.	.001	.000	.026
		N	90	90	90	90
	ESPL	Correlation Coefficient	.317**	1.000	.252**	.170
		Sig. (2-tailed)	.001	.	.009	.088
		N	90	90	90	90
	ESOL	Correlation Coefficient	.374**	.252**	1.000	-.024
		Sig. (2-tailed)	.000	.009	.	.818
		N	90	90	90	90
	ESEL	Correlation Coefficient	.227*	.170	-.024	1.000
		Sig. (2-tailed)	.026	.088	.818	.
		N	90	90	90	90

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Elbow:

			EMLR	ESPR	ESOR	ESER
Kendall's tau_b	EMLR	Correlation Coefficient	1.000	.449**	.395**	.244*
		Sig. (2-tailed)	.	.000	.000	.014
		N	92	92	92	92
	ESPR	Correlation Coefficient	.449**	1.000	.331**	.110
		Sig. (2-tailed)	.000	.	.001	.260
		N	92	92	92	92
	ESOR	Correlation Coefficient	.395**	.331**	1.000	.134
		Sig. (2-tailed)	.000	.001	.	.186
		N	92	92	92	92
	ESER	Correlation Coefficient	.244*	.110	.134	1.000
		Sig. (2-tailed)	.014	.260	.186	.
		N	92	92	92	92

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Hip:

Correlations

			HMLL	HSPL	HSOL	HSEL
Kendall's tau_b	HMLL	Correlation Coefficient	1.000	.259**	.304**	.170
		Sig. (2-tailed)	.	.004	.001	.084
		N	92	92	92	92
	HSPL	Correlation Coefficient	.259**	1.000	.296**	.127
		Sig. (2-tailed)	.004	.	.002	.190
		N	92	92	92	92
	HSOL	Correlation Coefficient	.304**	.296**	1.000	.193
		Sig. (2-tailed)	.001	.002	.	.058
		N	92	92	92	92
	HSEL	Correlation Coefficient	.170	.127	.193	1.000
		Sig. (2-tailed)	.084	.190	.058	.
		N	92	92	92	92

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Hip:

Correlations

			HMLR	HSPR	HSOR	HSER
Kendall's tau_b	HMLR	Correlation Coefficient	1.000	.222 ⁺	.389 ^{**}	.171
		Sig. (2-tailed)	.	.015	.000	.080
		N	93	93	93	93
	HSPR	Correlation Coefficient	.222 ⁺	1.000	.315 ^{**}	.132
		Sig. (2-tailed)	.015	.	.001	.171
		N	93	93	93	93
	HSOR	Correlation Coefficient	.389 ^{**}	.315 ^{**}	1.000	.191
		Sig. (2-tailed)	.000	.001	.	.058
		N	93	93	93	93
	HSER	Correlation Coefficient	.171	.132	.191	1.000
		Sig. (2-tailed)	.080	.171	.058	.
		N	93	93	93	93

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Knee:

Correlations

			KMLL	KSPL	KSOL	KSEL
Kendall's tau_b	KMLL	Correlation Coefficient	1.000	.580**	.492**	.453**
		Sig. (2-tailed)	.	.000	.000	.000
		N	90	90	90	90
	KSPL	Correlation Coefficient	.580**	1.000	.577**	.388**
		Sig. (2-tailed)	.000	.	.000	.000
		N	90	90	90	90
	KSOL	Correlation Coefficient	.492**	.577**	1.000	.352**
		Sig. (2-tailed)	.000	.000	.	.001
		N	90	90	90	90
	KSEL	Correlation Coefficient	.453**	.388**	.352**	1.000
		Sig. (2-tailed)	.000	.000	.001	.
		N	90	90	90	90

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Knee:

Correlations

			KMLR	KSPR	KSOR	KSER
Kendall's tau_b	KMLR	Correlation Coefficient	1.000	.534**	.629**	.366**
		Sig. (2-tailed)	.	.000	.000	.000
		N	90	90	90	90
	KSPR	Correlation Coefficient	.534**	1.000	.425**	.367**
		Sig. (2-tailed)	.000	.	.000	.000
		N	90	90	90	90
	KSOR	Correlation Coefficient	.629**	.425**	1.000	.229*
		Sig. (2-tailed)	.000	.000	.	.028
		N	90	90	90	90
	KSER	Correlation Coefficient	.366**	.367**	.229*	1.000
		Sig. (2-tailed)	.000	.000	.028	.
		N	90	90	90	90

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Orangutan Joint Marker Correlation Results:

Shoulder:

Correlations

			SMLL	SSPL	SSOL	SSEL
Kendall's tau_b	SMLL	Correlation Coefficient	1.000	.711**	.759**	.406**
		Sig. (2-tailed)	.	.000	.000	.000
		N	74	74	74	74
	SSPL	Correlation Coefficient	.711**	1.000	.598**	.372**
		Sig. (2-tailed)	.000	.	.000	.001
		N	74	74	74	74
	SSOL	Correlation Coefficient	.759**	.598**	1.000	.250*
		Sig. (2-tailed)	.000	.000	.	.028
		N	74	74	74	74
	SSEL	Correlation Coefficient	.406**	.372**	.250*	1.000
		Sig. (2-tailed)	.000	.001	.028	.
		N	74	74	74	74

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Shoulder:

Correlations

			SMLR	SSPR	SSOR	SSER
Kendall's tau_b	SMLR	Correlation Coefficient	1.000	.640**	.765**	.406**
		Sig. (2-tailed)	.	.000	.000	.000
		N	74	74	74	74
	SSPR	Correlation Coefficient	.640**	1.000	.539**	.347**
		Sig. (2-tailed)	.000	.	.000	.002
		N	74	74	74	74
	SSOR	Correlation Coefficient	.765**	.539**	1.000	.475**
		Sig. (2-tailed)	.000	.000	.	.000
		N	74	74	74	74
	SSER	Correlation Coefficient	.406**	.347**	.475**	1.000
		Sig. (2-tailed)	.000	.002	.000	.
		N	74	74	74	74

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Elbow:

Correlations

			EMLL	ESPL	ESOL	ESEL
Kendall's tau_b	EMLL	Correlation Coefficient	1.000	.560**	.423**	.211
		Sig. (2-tailed)	.	.000	.000	.058
		N	74	74	74	74
	ESPL	Correlation Coefficient	.560**	1.000	.340**	.183
		Sig. (2-tailed)	.000	.	.002	.100
		N	74	74	74	74
	ESOL	Correlation Coefficient	.423**	.340**	1.000	.021
		Sig. (2-tailed)	.000	.002	.	.852
		N	74	74	74	74
	ESEL	Correlation Coefficient	.211	.183	.021	1.000
		Sig. (2-tailed)	.058	.100	.852	.
		N	74	74	74	74

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Elbow:

Correlations

			EMLR	ESPR	ESOR	ESER
Kendall's tau_b	EMLR	Correlation Coefficient	1.000	.549**	.524**	.336**
		Sig. (2-tailed)	.	.000	.000	.002
		N	73	72	72	73
	ESPR	Correlation Coefficient	.549**	1.000	.474**	.360**
		Sig. (2-tailed)	.000	.	.000	.001
		N	72	72	72	72
	ESOR	Correlation Coefficient	.524**	.474**	1.000	.231*
		Sig. (2-tailed)	.000	.000	.	.042
		N	72	72	72	72
	ESER	Correlation Coefficient	.336**	.360**	.231*	1.000
		Sig. (2-tailed)	.002	.001	.042	.
		N	73	72	72	73

** . Correlation is significant at the 0.01 level (2-tailed).

* . Correlation is significant at the 0.05 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Hip:

Correlations

			HMLL	HSPL	HSOL	HSEL
Kendall's tau_b	HMLL	Correlation Coefficient	1.000	.276**	.423**	.
		Sig. (2-tailed)	.	.009	.000	.
		N	74	74	74	74
	HSPL	Correlation Coefficient	.276**	1.000	.161	.
		Sig. (2-tailed)	.009	.	.141	.
		N	74	74	74	74
	HSOL	Correlation Coefficient	.423**	.161	1.000	.
		Sig. (2-tailed)	.000	.141	.	.
		N	74	74	74	74
	HSEL	Correlation Coefficient
		Sig. (2-tailed)
		N	74	74	74	74

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Hip:

Correlations

			HMLR	HSPR	HSOR	HSER
Kendall's tau_b	HMLR	Correlation Coefficient	1.000	.407**	.444**	.
		Sig. (2-tailed)	.	.000	.000	.
		N	74	74	74	74
	HSPR	Correlation Coefficient	.407**	1.000	.317**	.
		Sig. (2-tailed)	.000	.	.004	.
		N	74	74	74	74
	HSOR	Correlation Coefficient	.444**	.317**	1.000	.
		Sig. (2-tailed)	.000	.004	.	.
		N	74	74	74	74
	HSER	Correlation Coefficient
		Sig. (2-tailed)
		N	74	74	74	74

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Knee:

Correlations

			KMLL	KSPL	KSOL	KSEL
Kendall's tau_b	KMLL	Correlation Coefficient	1.000	.649**	.786**	.434**
		Sig. (2-tailed)	.	.000	.000	.000
		N	70	70	70	70
	KSPL	Correlation Coefficient	.649**	1.000	.752**	.450**
		Sig. (2-tailed)	.000	.	.000	.000
		N	70	70	70	70
	KSOL	Correlation Coefficient	.786**	.752**	1.000	.420**
		Sig. (2-tailed)	.000	.000	.	.000
		N	70	70	70	70
	KSEL	Correlation Coefficient	.434**	.450**	.420**	1.000
		Sig. (2-tailed)	.000	.000	.000	.
		N	70	70	70	70

** . Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Knee:

Correlations

			KMLR	KSPR	KSOR	KSER
Kendall's tau_b	KMLR	Correlation Coefficient	1.000	.265 [*]	.671 ^{**}	.482 ^{**}
		Sig. (2-tailed)	.	.020	.000	.000
		N	71	71	71	71
	KSPR	Correlation Coefficient	.265 [*]	1.000	.336 ^{**}	.366 ^{**}
		Sig. (2-tailed)	.020	.	.004	.002
		N	71	71	71	71
	KSOR	Correlation Coefficient	.671 ^{**}	.336 ^{**}	1.000	.448 ^{**}
		Sig. (2-tailed)	.000	.004	.	.000
		N	71	71	71	71
	KSER	Correlation Coefficient	.482 ^{**}	.366 ^{**}	.448 ^{**}	1.000
		Sig. (2-tailed)	.000	.002	.000	.
		N	71	71	71	71

*. Correlation is significant at the 0.05 level (2-tailed).

**. Correlation is significant at the 0.01 level (2-tailed).

Codes:

The first letter refers to the joint (S = shoulder; E = elbow; H = hip; K = knee)

The second letter refers to M for margin or S for surface

The third letter refers to the specific marker (L = lipping; P = porosity; O = osteophytes; E = eburnation)

The last letter refers to L for left or R for right

Appendix E: MCZ letter of permission to use copyrighted material

MUSEUM OF COMPARATIVE ZOOLOGY
The Agassiz Museum



HARVARD UNIVERSITY
26 Oxford Street
Cambridge, Massachusetts 02138

December 19, 2008

Demelza J. Poe
7656 Thacher Drive
Department of Anthropology
University of New Mexico
Toano, Virginia 23168

Dear Ms. Poe,

Permission has been given to use the following material from the Museum of Comparative Zoology at Harvard University:

Mammalogy Department, MCZ specimen 26849, 62393, 23160, 38017, 37363

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Sincerely,

Catherine Weisel

Catherine Weisel
Museum Projects Coordinator
cweisel@oeb.harvard.edu

Enclosure

References:

- Albrecht, G. H. (1982) Collections of Nonhuman Primate Skeletal Materials in the United States and Canada. *American Journal of Physical Anthropology* 57:77-97.
- Atkas E.O., A. Koçak, S. Atkas, and A. Yemişçigil (2004). "Intercostal variation for age estimation-are the standards for the right 4th rib applicable for other ribs?" *Coll Antropol* 28 Supp 2: 267-72.
- Aufderheide A.C., and C. Rodriguez-Martin. (1998) *The Cambridge Encyclopedia of Human Paleopathology*. Cambridge University Press.
- Baccino E., D.H. Ubelaker, L.A. Hayek, and A. Zerilli (1999). "Evaluation of seven methods of estimating age at death from mature human skeletal remains." *Journal of Forensic Science* 44(5): 931-6.
- Bailey A.J., and D. Metz. (2001) "The role of bone in osteoarthritis." *Age and Ageing* 30:374-378.
- Boesch, C. and Boesch-Achermann, H. (2000) *The Chimpanzees of the Tai Forest: Behavioural Ecology and Evolution*. Oxford University Press.
- Braga J. (1998) "Chimpanzee Variation Facilitates the Interpretation of the Incisive Suture Closure in South African Plio-Pleistocene Hominids." *American Journal of Physical Anthropology* 105:121-135.
- Bramblett C. A., (1969). "Non-Metric Skeletal Age Changes in the Darajani Baboon." *American Journal of Physical Anthropology* 30:161-172.
- Bramblett C.A., (1967) "Pathology in the Darajani Baboon." *American Journal of Physical Anthropology*. 26:331-340.
- Breuer, T., M. Breuer-Ndoundou Hockemba, C. Olejniczak, R. J. Parnell, and E.M. Stokes (2008) "Physical Maturation, Life-History Classes and Age Estimates of Free-Ranging Western Gorillas-Insights from Mbeli Bai, Republic of Congo. *American Journal of Primatology* 70:1-14
- Bridges P.S. (1991) "Degenerative Joint Disease in Hunter-Gatherers and Agriculturalists from the Southeastern United States." *American Journal of Physical Anthropology* 85:379-391.
- Bridges, P.S. (1993) "The Effect of Variation in Methodology on the Outcome of Osteoarthritic Studies." *International Journal of Osteoarcheology*. Vol 3:289-295.

- Brothwell D. R. (1981) "*Digging Up Bones.*" Natural History Museum Publications, London.
- Buckberry J.L., and A. T. Chamberlain (2002) "Age estimation from the auricular surface of the ilium: a revised method." *American Journal of Physical Anthropology*. 119:231-9.
- Buikstra J.E., and D. H. Ubelaker (1994) "*Standards for Data Collection from Human Skeletal Remains*". Arkansas Archeological Survey Research Series No. 44. Arkansas Archeological Survey, AR.
- Caldecott, J. and L. Miles Editors (2005) "*World Atlas of Great Apes and their Conservation.*" Prepared at the UNEP World Conservation Monitoring Center, University of California Press, Berkeley, USA
- Cameriere, R. L. Ferrante, D. Math, and M. Cingolani (2004) "Variations in Pulp/Tooth Area Ratio as an Indicator of Age: A Preliminary Study. *Journal of Forensic Science* 49(2) 1-3.
- Carlson C.S., M. J. Jayo, D. S. Weaver, M. R. Adams, and C. P. Jerome (1994). "Osteoarthritis in cynomolgus macaques: a primate model of naturally occurring disease. *Journal of Orthopedic Research* 12:331-339.
- Carlson C.S., R. F. Loeser, C. B. Purser, J. F. Gardin, and C.P. Jerome (1996) "Osteoarthritis in cynomolgus macaques III. Effects of age, gender, and subchondral bone thickness on the severity of disease." *Journal of Bone Mineral Research* 11:1209-1217
- Center for Disease Control (CDC) (2008). "Arthritis". [online: accessed January 2008]. <http://www.cdc.gov/arthritis/>.
- Christensen, R. (1996) *Analysis of Variance, Design and Regression*. Boca Raton: Chapman and Hall.
- Coggon D., P. Croft, S. Kellingray, D. Barret, M. McLaren, and C. Cooper (2000). "Occupational Physical Activities and Osteoarthritis of the Knee." *Arthritis and Rheumatism*. 43(7):1443-1449.
- Comroe, B. I. (1944) *Arthritis and Allied Conditions*. Lea and Febiger Publishers.
- Conroy G. C., and C. J. Mahoney (1991) "Mixed longitudinal study of dental emergence in the chimpanzee, *Pan troglodytes* (primates, pongidae)." *American Journal of Physical Anthropology* 86(2): 243-254.

- Courtney, J. (2005) "Infant mortality in mother-reared captive chimpanzees at Taronga Zoo, Sydney." *Zoo Biology* 7-1:61-68.
- Craighead, J. J., Craighead, F.C. Jr., and McCuthen, H.E. (1970) "Age Determination of Grizzly Bears from Fourth Premolar Tooth Sections" *The Journal of Wildlife Management* 34-2: 353-363.
- Cranfield, M.R. (2008) "Mountain Gorilla Research: The Risk of Disease Transmission Relative to the Benefit from the Perspective of Ecosystem Health." *American Journal of Primatology* 70:751-754
- DeRousseau C.J. (1985) "Aging in the musculoskeletal system of rhesus monkeys: II. Degenerative joint disease." *American Journal of Physical Anthropology* 67: 177-184.
- DeRousseau C.J. (1988) "Osteoarthritis in Rhesus Monkeys and Gibbons. " *Contributions to Primatology, Vol. 25.* Karger Publishers.
- Dieppe, P. (1987) "Osteoarthritis and related disorders." In *Oxford Textbook of Medicine*, D.J. Weatherall, and J.G.G. Ledingham, Eds. Oxford University Press: New York. 16.76-16.84.
- Dennis J.C., P. S. Ungar, M. F. Teaford, and K E. Glander (2004) "Dental topography and molar wear in *Alouatta palliata* from Costa Rica." *American Journal of Physical Anthropology* 125(2): 152-161
- Dequeker J., L. Mokassa, J. Aerssens, and S. Boonen (1997). "Bone density and local growth factors in generalized osteoarthritis." *Microsc. Res. Tech.* 37:358-371.
- Đonić D., and M Đurić (2005) "Reliability of the individual age assessment at the time of death based on sternal rib-ends' morphology in Balkan population." *Vojnosanitetski preglod* 62:6, 441-446
- Drusini A., I. Calliari, and A. Volpe (1991) "Root dentine transparency: Age determination of human teeth using computerized densitometric analysis. *American Journal of Physical Anthropology* 85(1): 25-30.
- Duckworth W.L.H. (1911) "On the Natural Repair of Fractures, as seen in the Skeletons of Anthropoid Apes." *Journal of Anatomy and Physiology.* 46:81-85.
- Dunbar D.C. (1989) "Locomotor behavior of rhesus macaques (*Macaca mulatta*) on Cayo Santiago." *P R Health Sciences Journal* 8-1: 79-85.
- Duncan H. (1987) "Osteoarthritis." *Henry Ford Hospital Medical Journal.* 27:6-9.
- Eichner E. (1989) "Does running cause osteoarthritis? An epidemiological perspective." *The Physician and Sports Med.* 17:147-154.

- Field, A. (2005) *"Discovering Statistics Using SPSS (2nd Ed)." SAGE Publications, London.*
- Fleagle, J. (1999) *"Primate Adaptation and Evolution (2nd Ed)" Academic Press, New York.*
- Fossey, D. (1983) *"Gorillas in the Mist" Houghton Mifflin Company, Boston.*
- Fox H. (1939) "Chronic Arthritis in Wild Mammals." *Transactions of the American Philosophical Society*, n.s. 31:71-149.
- Galdikas, B.M.F. (1995) *"Reflections of Eden" Little, Brown, and Company, New York.*
- Gant, D.G. (1979) "Patterns of dental wear and the role of the canine in Cercopithecinae." *American Journal of Physical Anthropology* 51(3): 353-359.
- Garson, G.D. (2008) "Path Analysis" [online: accessed December 2008]
<http://faculty.chass.ncsu.edu/garson/PA765/path.htm>
- Goodall, J. (1986) *"The Chimpanzees of Gombe: Patterns of Behavior."* The Belknap Press of Harvard University Press, Cambridge, MA and London, England.
- Goodall, J. (1994) *"Chimpanzees Cultures."* Forward by Jane Goodall.
R.W. Wrangham, W.C. McGrew, F.B.M deWaal, and P.HG.Heltne (Eds)
Chicago Academy of Sciences.
- Goodwin, E.A. and Ballard, W.B. (1985) "Use of Tooth Cementum for Age Determination of Gray Wolves." *The Journal of Wildlife Management* 49-2: 313-316
- Gorilla SSP (2008). "Gorillas in Zoos." [online: accessed September 2008]
<http://www.clemetzoo.com/gorillassp/>
- Greer M., J.K. Greer, and J. Gillingham (1977). "Osteoarthritis in selected wild mammals." *Proceedings of the Oklahoma Academy of Sciences*. 57:39-43.
- Hassett, G., D.J. Hart, D.V. Doyle, L. March, and T.D. Spector (2006). "The relation between progressive osteoarthritis of the knee and long term progression of osteoarthritis of the hand, hip, and lumbar spine." *Annals of Rheumatic Diseases*. 65:623-628.
- Hill, K., C. Boesch, J. Goodall, A. Pusey, J. Williams, and R. Wrangham. (2001) "Mortality rates among wild chimpanzees." *Journal of Human Evolution* 40:437-450
- Holderbaum D., T. M. Haqqi, and R.W. Moskowitz RW (1999). "Genetics and Osteoarthritis: Exposing the Iceberg." *Arthritis and Rheumatism* 42(3): 397-405.

- Hough, A. J. Jr., (1993) "Pathology of Osteoarthritis" in *Arthritis and Allied Conditions* (12th Ed.), pp 1699-1721. D.J. McCarty and W.J. Cooman, Eds, Lea and Febiger: Philadelphia.
- International Union for Conservation of Nature (IUCN) (2007) "*IUCN Red List of Threatened Species*" [online: accessed July 2008].
<http://www.iucn.org/themes/ssc/redlist.htm>
- Iscan M.Y., S. R. Loth, and R. K. Wright (1984). "Age Estimation from the Rib by Phase Analysis: White Males." *Journal of Forensic Science* 29-4: 1094-1104.
- Iscan M.Y., S. R. Loth, and R. K. Wright (1984). "Age Estimation from the Rib by Phase Analysis: White Females." *Journal of Forensic Science* 30: 853-863.
- Iscan M.Y. (1991) "The Aging Process in the Rib: An Analysis of Sex- and Race-Related Morphological Variation." *American Journal of Human Biology* 3:617-623
- Iscan M.Y., and S. R. Loth (1993) "*Casts of Age Phases from the Sternal End of the Rib for White Males and Females.*" France Casting, Bellvue, CO.
- Itani, J. (1996) Afterword: a new milestone in great ape research. In "*Great Ape Societies.*" W.W. McGrew, L.F. Marchant, and T. Nishida Eds. Cambridge University Press.
- Jurmain R. (1977) "Stress and Etiology of Osteoarthritis." *American Journal of Physical Anthropology*. 46:353-366.
- Jurmain R. (1980) "The Pattern of Involvement of Appendicular Degenerative Joint Disease." *American Journal of Physical Anthropology* 53:143-150.
- Jurmain, R. (1989) "Trauma, Degenerative Disease, and Other Pathologies among the Gombe Chimpanzees." *American Journal of Physical Anthropology*. 80:229-237.
- Jurmain R, (2000) and L. Kilgore. (1995) "Skeletal evidence of osteoarthritis: a palaeopathological perspective." *Annals of Rheumatic Diseases*. 54(6):443-450.
- Jurmain R. (1999) "*Stories from the Skeleton: Behavioral Reconstruction in Human Osteology.*" Gordon and Breach, California.
- Jurmain, R. (2000) "Degenerative joint disease in African great apes: an evolutionary perspective." *Journal of Human Evolution*. 39:185-203.
- Kay, R.F., T. Rasmussen, and C. Beard. (1984) "Cementum Annulus Counts Provide A Means for Age Determination in *Macaca mulatta* (Primates, Anthroidea)" *Folia Primatologica* 42-2:85-95.

- Kay R. F., and J. G. H. Gant (1988) "Age Assessment Using Cementum Annulus Counts and Tooth Wear in a Free-Ranging Population of *Macaca mulatta*." *American Journal of Primatology* 15:1-15.
- Kerley E.R. (1966). "Skeletal Age Changes in the Chimpanzee." *Tulane Studies in Zoology* 13: 71-82.
- Kilgore, L. (1989) "Dental Pathologies in Ten Free-Ranging Chimpanzees from Gombe National Park, Tanzania." *American Journal of Physical Anthropology* 80:219-227.
- Kleplinger, L.L. (2006) "*Fundamentals of Forensic Anthropology*." (pp 52). John Wiley and Sons, New Jersey
- Knüsel, C.J., S. Göggel, and D. Lucy (1997) "Comparative Degenerative Joint Disease of the Vertebral Column in the Medieval Monastic Cemetery of the Gilbertine Prior of St. Andrew, Fishergate, York, England." *American Journal of Physical Anthropology* 103:481-495.
- Köhler W. (1927) "*The Mentality of Apes (2nd Ed)*" Routledge and Kegan Paul Ltd., London.
- Kono, R. (2004) "Molar enamel thickness and distribution patterns in extant great apes and humans: new insights based on a 3-dimensional whole crown perspective." *Anthropological Science* 112(2):121-146
- Konradsen L., E. M. Hansen, and L. Sundergaard (1990) "Long distance running and osteoarthritis." *American Journal of Sports Medicine*. 18:379-381.
- Lamendin H., E. Baccino, J.F. Humbert, J. C. Tavernier, R.M. Nossintchouk, and A. Zerilli (1992) "A Simple Technique for Age Estimation in Adult Corpses: The Two Criteria Dental Method." *Journal of Forensic Sciences* 37(5) 1373-1379
- Lane N.E., D. A. Bloch, and P.D. Wood (1987). "Aging, long-distance running and the development of musculoskeletal disability: A controlled study." *American Journal of Medicine*. 82:772-780.
- Lane N.E., D.A. Bloch, and H.B. Hubert (1990). "Running, osteoarthritis, and bone density: Initial 2-year longitudinal study." *American Journal of Medicine*. 88:452-459.
- Lane N.E., B. Michel, and Bjorkengren. (1993) "The risk of osteoarthritis with running and aging: A 5-year longitudinal study." *Journal of Rheumatism*. 20:461-468.

- Lawrence R.C., D.T. Felson, C.G. Helmick, L.M. Arnold, H. Choi, R.A. Deyo, S. Gabriel, R. Hirsch, M.C. Hochberg, G.G. Hunder, J.M. Jordan, J.N. Katz, H.M. Kremers, and F. Wolfe. (2008) "Estimates of the Prevalence of Arthritis And Other Rheumatic Conditions in the United States, Part II." *Arthritis and Rheumatism* 58-1:26-35.
- Lehman, J. and C. Boesch. (2008) "Sexual Differences in Chimpanzee Sociality." *International Journal of Primatology* 29:65-81.
- Lieverse, A.R., A.W. Weber, V.I. Bazaliiskiy, O.I. Goriunova, and N.A. Savel'ev. (2007) "Osteoarthritis in Siberia's Cis-Baikal: Skeletal Indicators of Hunter-Gatherer Adaptation and Cultural change." *American Journal of Physical Anthropology* 132:1-16.
- Lim K.K., M.J. Kessler, K. P. Pritzker, J. E. Turnquist, and P. A. Dieppe (1996). "Osteoarthritis of the hand in nonhuman primates: a clinical, radiographic, and skeletal survey of Cayo Santiago rhesus macaques. *Journal of Medical Primatology* 25:301-308.
- Long, J. C., and C. F. Merbs (1981). "Coccidioidomycosis: A Primate Model. " In, *Prehistoric Tuberculosis in the Americas*. J. Buikstra, Ed. Evanston: Northwestern University Archeological Program, pp 69-83.
- Loth S.R., M.Y. Iscan, and E. H. Scheuerman (1994). "Intercostal variation at the sternal end of the rib." *Forensic Science International* 65(2): 135-43.
- Lovell, N.C. (1990a) *Patterns of Injury and Illness in Great Apes: A Skeletal Analysis*. Smithsonian Institution Press, Washington.
- Lovell, N.C. (1990b) "Skeletal and dental pathology of free-ranging mountain gorillas." *American Journal of Physical Anthropology* 81(3):399-412.
- Lovejoy C.O. (1985) "Dental Wear in the Libben Population: Its Functional Pattern and Role in the Determination of Adult Skeletal Age at Death." *American Journal of Physical Anthropology* 68:47-56
- Lovejoy C.O., R. S. Meindl, T. R. Pryzbeck, and R. P. Mensforth (1985). "Chronological metamorphosis of the auricular surface of the ilium: a new method for the determination of the adult skeletal age at death. *American Journal of Physical Anthropology* 68:15-28.
- Lovejoy C.O., R.S. Meindl, R.P. Mensforth, and T.J. Barton (1985) "Multifactorial determination of Skeletal Age at Death: A Method and Blind Tests of Its Accuracy." *American Journal of Physical Anthropology* 68:1-14.

- Lunt, D.A., (1978) "Molar attrition in Medieval Danes. In *"Development, Function, and Evolution of Teeth."* PB Butler and KA Joysey (Eds), Academic Press, New York, pp. 465-482.
- Manek N.J., D. Hart, T.D. Spector, and A. J. MacGregor. (2003) "The association of body mass index and osteoarthritis of the knee joint." *Arthritis and Rheumatism*, 48:1024-1029. DOI: 10.1002/art.10884.
- Marti B., M. Knobloch and A. Tschopp (1989) "Is excessive running predictive of degenerative hip disease? Controlled study of former athletes." *British Medical Journal*, 289:91-93.
- Marzke, M.W., and C. F. Merbs (1984). "Evidence of Hypertrophic Pulmonary Osteoarthropathy in a Chimpanzee, *Pan troglodytes*." *Journal of Medical Primatology* 12: 135-145
- Matson's Lab (2007) "A wildlife laboratory offering cementum aging and tetracycline biomarker screening." [online: accessed 2007] <http://www.matsonslab.com/>
- McDermot M., and P. Freyne (1983) "Osteoarthrosis in runners with knee pain." *British Journal of Sports Medicine*. 17:84-87.
- McGrew, W. (2004) *"The Cultured Chimpanzee: Reflections on Cultural Primatology."* Cambridge University Press
- Meeham, T.P., and L. J. Lowenstine (1994) "Causes of mortality in captive lowland Gorillas: a survey of the SSP population." *Proceedings of the Conference of the American Association of Zoo Veterinarians*, Hawaii, pp 216-218
- Megyesi M.S., D. H. Ubelaker, and N. J. Sauer (2006) "Test of the Lamendin Aging Method on Two Historic Skeletal Samples." *American Journal of Physical Anthropology* 131:363-367.
- Molnar S. (1971a) "Human Tooth Wear, Tooth Function and Cultural Variability." *American Journal of Physical Anthropology* 34: 175-190.
- Molnar S. (1971b) "Sex, Age, and Tooth Position as factors in the production of Tooth Wear." *American Antiquity* 36(2): 182-188.
- Morbeck M.E., A. Galloway, and D. R. Sumner (2002) "Getting Old at Gombe: Skeletal Aging in Wild-Ranging Chimpanzees" in *Aging in Nonhuman Primates*, Vol 31: pp 48-62. Interdisciplinary Topics in Gerontology. J.M. Erwin & P.R Hoff (Eds), Basel, Karger
- Nagy B.L.B. (2000) "The Life Left in Bones: Evidence of Habitual Activity Patterns in Two Prehistoric Kentucky Populations." PhD Dissertation, Arizona State University.

- Nakai M. (2003) "Bone and Joint Disorders in Wild Japanese Macaques from Nagano Prefecture, Japan". *American Journal of Primatology* 24(1):179-195
- National Geographic (5 Aug 2008) "Gorilla paradise found: May double world numbers" [online: accessed Nov 2008]
http://news.nationalgeographic.com/news/2008/08/080805-gorillas-congo_2.html
- Nevitt M.C., L. Xu, Y. Zhang, L. Y. Lui, W. Yu, N.E. Lane, M. Qin, M.C. Hochberg, S. R. Cummings, and D. T. Felson (2002). "Very low prevalence of hip osteoarthritis among Chinese elderly in Beijing, China, compared with whites in the United States: The Beijing osteoarthritis study." *Arthritis and Rheumatism* 46(7): 1773-1779.
- Newson, J.T. (2008) Lecture 12 "More on Chi-Square". [online: accessed Jun 2008]
<http://www.upa.pdx.edu/IOA/newsom/pa551/lectur13.html>
- Nichols K. A., and A. L. Zihlman (2002). "Skeletal and Dental Evidence of Aging in Captive Western Lowland Gorillas: A Preliminary Report. In *Aging in Nonhuman Primates*, Interdisciplinary Topics in Gerontology. Vol 31, pp 22-31. J. M. Erwin, Ed. Basel, Karger.
- Nissen H.W., and A. H. Riesen (1964) "The Eruption of Permanent Dentition of Chimpanzee." *American Journal of Physical Anthropology* 22: 285-294.
- New York Times (2008) "Bags Five Gorillas for Museum Here." New York Times Archives [online: accessed Jul 2008]
<http://query.nytimes.com/gst/abstract.html?res=9905E3DE113EEE3ABC4A52DFB467838A639EDE>
- Oettlé A.C., and M. Steyn (2000) "Age estimation from sternal ends of the rib by phase analysis in South African Blacks." *Journal of Forensic Science* 45(5): 1071-9.
- Oliveira R.N., S.F.S.M. Silva, A. Kawano, and J. L. F. Antunes (2006) "Estimating Age by Tooth Wear of Prehistoric Human Remains in Brazilian Archaeological Sites." *International Journal of Osteoarchaeology* 16: 407-414.
- Otterness I.G., J.D. Eskra, M. L. Bliven, A.K. Shay, J.P. Pelletier, and A.J. Milici (1998) "Exercise protects against articular cartilage degeneration in the hamster." *Arthritis and Rheumatism*. 41(11): 2068-2076.
- Panush R.S., C. Schmidt, and J. R. Caldwell. (1986) "Is running associated with degenerative joint disease? *Journal of the American Medical Association* 255:1152-1154.
- Perkins, L. (2002) International Studbook of the Orangutan. Lincoln Park Zoo, Lori Perkins, Studbook Keeper.

- Primate Cognitive Neuroscience Laboratory (2009) "Cayo Research" [online: accessed Nov 2009]
<http://www.wjh.harvard.edu/~mnkylab/media/cayo/cayo.index.html>
- Pritchard, J.J., J.H. Scott, and F.G. Girgis (1956). "The Structure and Development of Cranial and Facial Sutures." *Journal of Anatomy* 90:73-86.
- Punzi, L. F. Oliviero, and M. Plebani. (2005) "New biochemical insights into the pathogenesis of osteoarthritis and the role of laboratory investigations in clinical assessment." *Critical Reviews in Clinical Laboratory Sciences* 42:279-309.
- Puranen J., L. Ala-Ketola, and P. Peltokallio P (1975) "Running and primary osteoarthritis of the hip." *British Medical Journal* 2:424-425.
- Radin E.L. (1982) "Mechanical Factors in the Causation of Osteoarthritis." *Rheumatism*. 7:46-52.
- Radin E.L. (1983) "The Relationship between Biological and Mechanical Factors in the Etiology of Osteoarthritis." *Journal of Rheumatism* (supp 9) 10:20-21.
- Randall, F. E. (nd) "The Skeletal and Dental Development and Variability of the Gorilla." *Human Biology* 23-65.
- Rawlins, R.G., and M.T. Kessler (1986) *The Cayo Santiago macaques: history, behavior and Biology*" State University of New York Press, Albany, New York.
- Rissech, C., G. F. Estabrook, E. Cunha, and A. Malgosa (2006) "Using the Acetabulum to Estimate Age at Death of Adult Males." *Journal of Forensic Science* 51(2): 213-229.
- Robson Brown, K., P. Pollintine, and M.A. Adams (2008) "Biomechanical implications of degenerative joint disease in the apophyseal joints of human thoracic and lumbar vertebrae." *American Journal of Physical Anthropology* 136:318-326.
- Rogers J., T. Waldron, P. Dieppe, and I. Watt. (1987) "Arthropathies in Palaeopathology: The Basis of Classification according to Most Probable Cause." *Journal of Archaeological Science*. 14:179-193.
- Rogers J. and T. Waldron. (1995) "A Field Guide to Joint Diseases in Archaeology." John Wiley Press, New York.
- Rogers J., L. Shepstone, and P. Dieppe. (2004) "Is Osteoarthritis a Systemic Disorder of Bone?" *Arthritis and Rheumatism*. 50(2):452-457.
- Ross, S. (2006) "North America Regional Chimpanzee Studbook." Lincoln Park Zoo, Steve Ross, Studbook Keeper.

- Rothschild B.M., and R. J. Woods (1992a) "Erosive Arthritis and Spondyloarthropathy in Old World Primates." *American Journal of Physical Anthropology* 88:389-400.
- Rothschild B.M., and R. J. Woods (1992b) "Osteoarthritis, Calcium Pyrophosphate Deposition Disease, and Osseous Infection in Old World Primates." *American Journal of Physical Anthropology* 87:341-347.
- Rothschild, B.M. (1997) "Porosity: A curiosity without diagnostic significance." *American Journal of Physical Anthropology* 104(529-533).
- Rothschild, B.M. and F. J. Ruhli (2005a) "Comparison of Arthritis Characteristics in Lowland *Gorilla gorilla* and Mountain *Gorilla beringei*." *American Journal of Primatology* 66:205-218.
- Rothschild, B.M. and F. J. Ruhli (2005b) "Etiology of Reactive Arthritis in *Pan paniscus*, *P. troglodytes troglodytes*, and *P. troglodytes schweinfurthii*." *American Journal of Primatology* 66:205-218.
- Rougé-Maillart C., N. Telmon, C. Rissech, A. Malgosa, and D. Rougé (2004) "The Determination of Male Adult Age at Death by Central and Posterior Coxal Analysis – A Preliminary Study. *Journal of Forensic Science* 49(2): 1-7
- Rowe, N. (1996). "*The Pictorial Guide to the Living Primates*." Pogonias Press, New York.
- Russell K.F., S.W. Simpson, J. Genovese, M.D. Kinkel, R.S. Meindl, and C.O. Lovejoy (1993) "Independent test of the fourth rib aging technique." *American Journal of Physical Anthropology* 92(1): 53-62.
- Ryder, O.A., and L.G. Chemnick (1993). "Chromosomal and Mitochondrial DNA Variation in Orang Utans." *Journal of Heredity* 84(5):405-409.
- Sabini, R.O., and D.E. Elkowitz (2006) "Significance of Differences in Patency Among Cranial Sutures." *Journal of the American Osteopathic Association* 106-10: 600-604.
- Sarmiento E.E. (1985) "Functional differences in the skeleton of wild and captive orangutans and their adaptive significance." PhD Dissertation, New York University.
- Schultz A. H. (1935) "Eruption and Decay of the Permanent Teeth in Primates." *American Journal of Physical Anthropology* Vol XIX, No 4: 489-581.
- Schultz A. H. (1935) "Eruption and Decay of the Permanent Teeth in Primates." *Human Biology* 2:303-438.

- Schultz A.H. (1940) "Growth and Development of the Chimpanzee." *Contributions to Embryology No 170*:1-63.
- Schultz A. H. (1941) "Growth and Development of the Orangutan." *Contributions to Embryology No 182*:59-110.
- Schultz A.H. (1945) "Morphological Observations on Gorillas." *Raven Memorial Volume* 227-249.
- Schultz, A.H. (1950) Morphological Observations on Gorillas, Part Five, in "*Anatomy of the Gorilla*", Columbia University Press, New York.
- Schultz A.H. (1969) "*The Skeleton of the Chimpanzee*" in *The Chimpanzee*, Vol 1: 50-103, Karger, New York. G. H. Bourne, Ed.
- Schultz A.H. (1969) "*The Life of Primates*." Universe Books, New York.
- Scott E.C. (1979) "Dental Wear Scoring Technique" *American Journal of Physical Anthropology* 51: 213-218.
- Sharma L., C. Lou, S. Cahue, and C.D. Dunlop (2000) "The Mechanism of the Effect of Obesity in Knee Osteoarthritis." *Arthritis and Rheumatism*. 43(3):568-575.
- Sirkin, R.M (1995) "*Statistics for the Social Sciences*". Sage Publications, p 363.
- Smith, T.M., and B.H. Smith (2009) "Dental Development in the Tai Forest Chimpanzees Reappraised." *American Journal of Physical Anthropology* 138(S48):243
- Sohn R.S., and L. J. Micheli (1985) "The effect of running on the pathogenesis of osteoarthritis of the hips and knees. *Clinical Orthopedics*. 198:106-109.
- Sokoloff, L. (1969) "*The Biology of Degenerative Joint Disease*." The University of Chicago Press.
- StatSoft Electronic Textbook. (2008) "Generalized Linear Models(GLZ)." [online: accessed June 2008]. <http://www.statsoft.com/textbook/stglz.html>
- Strum, S.C. (1987) "*Almost Human: A Journey into the World of Baboons*" Random House, New York.
- Tarou, L.R, M.A. Bloomsmit, M.P. Hoff, J.M. Erwin, and T.L. Maple (2002) "The Behavior of Aged Great Apes." in *Aging in Nonhuman Primates*. Interdisciplinary Topics in Gerontology. Vol 31, pp 209-231. J.M. Erwin & P.R Hoff (Eds), Basel, Karger.
- Taylor, A. (2002). "Masticatory Form and Function in African Apes". *American Journal of Physical Anthropology* 117:133-156.

- Tepper, S. and M. C. Hochberg, (1993) "Factors associated with hip osteoarthritis: data from the First National Health and Nutrition Examination Survey." *American Journal of Epidemiology*. 137:1081-1088.
- Tomenchuk J., and Mayhall J.T. (1979) "A correlation of tooth wear and age among Modern Igloodik Eskimos." *American Journal of Physical Anthropology* 51(1): 67-77.
- Trochim, W.M.K. (2006) "General Linear Model." [online: accessed Dec 2008] <http://www.socialresearchmethods.net/kb/genlin.php> (last updated 10/20/2006)
- Turner, J.C., (1977) "Cemental Annulations as an Age Criterion in North American Sheep" *The Journal of Wildlife Management* 41-2: 211-217.
- UCLA: Academic Technology Services, Statistical Consulting Group. (2007) [online: accessed Jan 2009], http://www.ats.ucla.edu/stat/mult_pkg/whatstat/nominal_ordinal_interval.htm
- Valdes A.M., D. J. Hart, K. A. Jones, G. Surdulescu, P. Swarbrick, D. V. Doyle, A. J. Schafer, and T. D. Spector (2004). "Association Study of Candidate Genes for the Prevalence and Progression of Knee Osteoarthritis." *Arthritis and Rheumatism* 50(8):2497-2507.
- Walsh, P.D., Tutin, C.E.G., Baillie, J.E.M., Maisels, F., Stokes, E.J. and Gatti, S. (2008). *Gorilla gorilla ssp. gorilla*. In: IUCN 2008. 2008 IUCN Red List of Threatened Species. <www.iucnredlist.org>. [online: accessed Nov 2008].
- Watts, D.P (1998) "Seasonality in the Ecology and Life History of Mountain Gorillas (*Gorilla gorilla berengei*)" *International Journal of Primatology* 19-6: 929-948.
- Weiss, E and R. Jurmain. (2007) "Osteoarthritis Revisited: A Contemporary Review of Aetiology." *International Journal of Osteoarchaeology* 17:437-450.
- Wells, C. (1973) "The Paleopathology of Bone Disease." *Practitioner* 210(257):384-391.
- Wharton D. (2006). "International Gorilla Studbook." Chicago Zoological Society, Brookfield Zoo.
- Wich, S.A., S.S. Utami-Atmoko, T. Mitra Setia, H.D. Rijksen, C. Schürmann, J.A.R.A.M. van Hooff, and C.P. van Schaik. (2004) "Life history of wild Sumatran orangutans (*Pongo abelii*)." *Journal of Human Evolution* 47-6:385-398.
- Williams, F. M. K. and T. D. Spector. (2006) "Osteoarthritis" *Medicine* 34:9, 364-368.
- Williams, K.M., E.V Lonsdorf, M.L. Wilson, J. Schumacher-Stankey, J. Goodall, and A. Pusey (2008) "Causes of Death in the Kasekela Chimpanzees of Gombe National Park, Tanzania." *American Journal of Primatology* 70:766-777.

- Wood, J.W., G. R. Milner, H.C. Harpending, and K.M. Weiss (1992). "The Osteological Paradox: Problems of Inferring Prehistoric Health from Skeletal Samples." *Current Anthropology* 33-4:343-370.
- Woods, R.J. (1985) "Biomechanics and osteoarthritis of the knee." PhD thesis, Ohio State University, Columbus, OH.
- Woods R.J. (1986) "Biomechanics and degenerative joint disease in humans, gorillas, and Chimpanzees." M.A. Thesis, Kent State University, Kent Ohio.
- World Health Organization (WHO) (2008). "Chronic Diseases and Health Promotion" [online: accessed January 2008]. <http://www.who.int/chp/topics/rheumatic/en/>
- World Wildlife Fund (2008) "Orangutans – Ecology and Habitat." http://www.panda.org/about_wwf/what_we_do/species/about_species/species_factsheets/great_apes/orangutans/orangutans_ecology_habitat/index.cfm [online: accessed Nov 2008].
- Yerkes, R.M. (1925) "*Almost Human*" Jonathan Cape, London.
- Yerkes, R.M. and Learned B.W. (1925) "*Chimpanzee Intelligence and it's Vocal Expressions.*" The Williams and Wilkins Company, Baltimore.
- Yoder C., D. H. Ubelaker, J. F. Powell (2001) "Examination of Variation in Sternal Rib End Morphology Relevant to Age Assessment." *Journal of Forensic Science* 46(2): 223-227.
- Zhai, G., D.J. Hart, B.S Kato, A. MacGregor, and T. D. Spector. (2006) "Genetic influence on the progression of radiographic knee osteoarthritis: a longitudinal twin study." *OsteoArthritis and Cartilage*. Doi: 10.1016/j.joca.2006.09.004.
- Zhang, Y., O.A. Ryder, and Y.Zhang. (2001) "Genetic Divergence of Orangutan Subspecies (*Pongo pygmaeus*)" *Journal of Molecular Evolution* 52:516-526
- Zihlmann A. L., M. E. Morbeck, and J. Goodall (1990) "Skeletal biology and individual Life history of Gombe chimpanzees." *Journal of Zoology, London*. 221: 37-61.
- Zihlmann, A., D. Bolter, and C. Boesch (2004) Wild Chimpanzee dentition and its Implications for assessing life history in immature hominin fossils. *Proceedings of the National Academy of Sciences* 101-29: 10541-10543.