FLORIDA MUSEUM OF NATURAL HISTORY™

BULLETIN

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Laurie Wilkins, Julie M. Allen, Joan Coltrain, Shelly Flanagin, Terry D. Allen, & David L. Reed[1]

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Key Words: methods, museum specimens, osteology, health, stable isotopes, diet, Florida panther, *Puma concolor*

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PART I: OSTEOLOGY AS A MEANS OF ASSESSING FLORIDA PANTHER HEALTH

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ABSTRACT

Conservation efforts to reverse the negative effects of inbreeding in an isolated population of Florida panthers (*Puma concolor coryi*) resulted in the release of eight Texas females into Florida in 1995 (Seal 1994; Johnson et al. 1998). Since that time, Florida panthers have shown increased productivity, range expansion, and the reversal of a suite of deleterious morphological and physiological effects of inbreeding. (Land et al. 2005, Pimm et al. 2006). Previously described bone pathologies in the Florida panther may result from a compromised immune system due to inbreeding, poor health related to diet and nutrition, or the presence of previously undetected pathogenic diseases. We examine the current collection of 140 post-cranial skeletons to determine the frequency of trauma, infection, arthritis, and incidence of Harris Lines. Harris lines, visible from X-rays of long bones, represent a cessation of growth due to a major episode of starvation or illness. We compare the population born before and after 1995 to examine changes over a time line that includes genetic, biomedical, and management interventions. Our data support earlier findings that there are idiopathic bone pathologies that exist in the Florida population, and we explore possible causes. Multivariate analysis reveals that Harris Lines and osteopathologies increase with age, and those pathologies affect males more than females, and both show increases after two years of age. There is a reduction in the number and severity of osteopathologies in panthers born after 1995; however, the demographics of our population (as represented in the museum sample) have shifted from an "aged" population to one that includes a disproportionately large number of young animals (<2 years old). It is likely that more than one biological process is operating to produce this result, and the study of osteological material alone cannot provide definitive diagnoses. Advanced studies in the pathology of human arthritis offer intriguing insights, and we expect at least some of their findings to have application in wildlife disease studies. Our results, together with the rich resource of archival material, leads to new cooperative research opportunities between museums, wildlife biologists, and wildlife veterinarians in the efforts to improve the conservation status of Florida panthers.

INTRODUCTION

Post-mortem skeletal remains reveal a history of activity, injury or traumatic events in the life of an animal. This is expressed by scars, malformations, unusual lesions, or excessive bone deposition as animals overcome infections, disease, or injury in life. Unusual or abnormal osteological features that have been described in panthers include arthritis, evidence of infection, trauma and bone lesions of unknown cause. Harris Lines (HLs), internal osteological features, were also recorded in high frequency in panthers. Duckler and Van Valkenburgh (1998a) showed that 69% of Florida panthers (N=51) exhibited at least one post-cranial osteopatholgy compared to 46% in a sample of pumas (N=26) from other locations. In the same study, the prevalence of HLs in Florida panthers was significantly greater (56.9%) than that of non-Florida pumas (27%). They conjectured that the elevated incidence of HLs was due to more regular episodes of poor nutrition, perhaps exacerbated by health problems associated with inbreeding.

Harris Lines are visible in radiographs as dense lines of bone deposition in long bones of humans and other mammals (Fig. 1). They record episodes of arrested bone growth in young individuals (Park 1964), and have been experimentally induced in rats, rabbits, pigs and dogs by starvation, selective nutrient deficiency, and bacterial inoculation (Harris 1933; Wolbach 1947; Park & Richter 1953; Platt & Stewart 1962; Mays 1995). It was these experimental studies that linked the formation of HLs, with the occurrence of a physical stress (Grolleau-Raoux et al. 1997). In humans, HLs have been associated with episodes of childhood illness (for

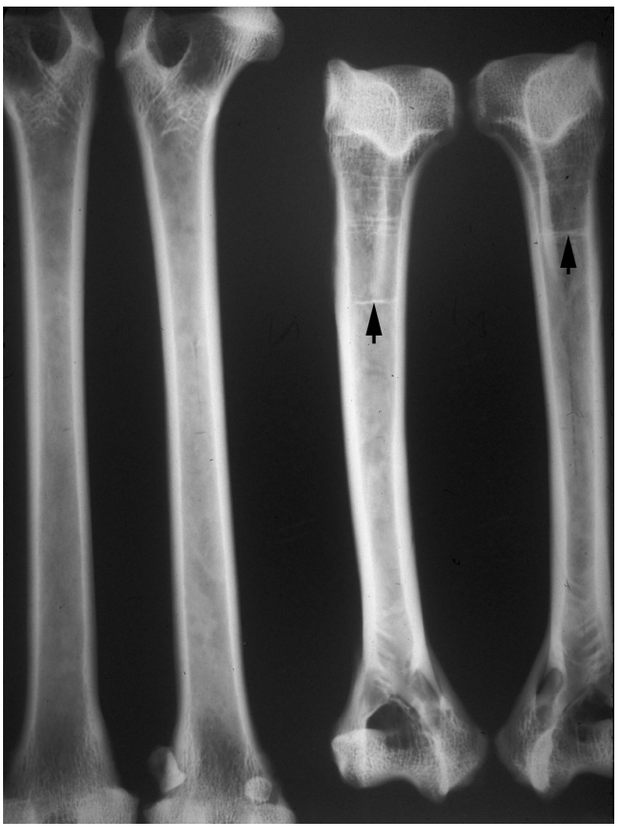


Figure 1: X-ray of humerii of Florida panther 15 , UF24563) showing Harris Lines. Harris Lines are transverse lines of bone density that represent a period of arrested growth followed by renewed growth.

review, see Garn et al. 1968). As a result, they have been used extensively in historical and archeological studies to characterize the health of human populations (Macchiarelli et al 1994; McHenry 1968; Rathbun 1987).

Current interpretations differ on the significance of HL formation. Some believe that HLs form as stress lines during accelerated growth spurts rather than as indicators of past illness, trauma, or malnutrition (Alfonso et al. 2005). The alternative explanation—that HLs represent renewed bone growth upon recovery from a serious illness—remains well supported (Mays 1995), although it has been shown that there is often no one-to-one correspondence between episodes of disease or nutritional deficiency and HL formation (Marshall 1968). Medical monitoring of Florida panthers during the 1980s showed cats to be in variable health declining southward towards the Everglades National Park (ENP) and the Fakahatchee Strand State Preserve (FSSP), a condition associated with the type and abundance of prey (Roelke 1990), and supported by food habit studies. Scat analysis showed the panthers living north of Alligator Alley were killing predominantly large prey (white-tailed deer and feral hogs), while those panthers living in the FSSP were taking a large number of small prey (raccoon and armadillo) (Roelke et al. 1986, Maehr et al. 1990). A relationship between diet, health, and presence of HLs may exist in this stressed population, but it is complex and requires further investigation.

Studies on the incidence of osteological abnormalities in a wild mammal population are rare, and frequently involve the description of a single or few specimens. This is often true because post-cranial skeletons were not archived, and are not available for study. The panther specimens in the FLMNH mammal collection today number more than 140 individuals, including those salvaged through mid-2006. This provides an unparalleled opportunity to expand the earlier study of morphology conducted by Duckler and Van Valkenburgh (1998a,b) and to further explore the nature of osteopathology in natural populations. The literature on bone pathology in mammals is vast, but often relates to veterinary studies on domestic animals; terminology and the diagnosis of skeletal abnormalities from skeletal assemblages is difficult and often confusing.

Arthritis is defined by the presence of osteophytes, which are bone spicules that develop around joint margins or sites of injury and represent the body's attempt to repair an injury. Arthritis involves proliferation of bone rather than loss of bone as in osteoporosis. The degree of osteophytosis in humans and animals can vary considerably—from single spicules, a ridge of new growth along an area of muscle attachment, to a massive outgrowth of bone. The presence of osteophytes is a common indicator of osteoarthritis (OA) or degenerative often age-related joint disease (DJD), which is common in humans (Rogers & Waldron 1995), wild mammals (Fox 1939; Greer et al. 1977) and domestic animals (Jubb et al. 1985). Degenerative joint disease is a progressive condition in which the articular cartilage is slowly degraded and the surrounding bone reacts by producing osteophytes. The disease affects many animal species, and is common in domestic breeds of cats. However, the presence of osteophytes may be associated with other conditions, as 'arthritis' is a general term that includes a broad spectrum of disorders or diseases that has been extensively studied in humans, and is beginning to emerge as a field of study in other mammals, as we will discuss in greater detail later.

In past studies, joint disorders have been broadly separated into two categories, non-inflammatory and inflammatory. Osteoarthritis (OA), or DJD, including joint disease resulting from trauma and developmental, metabolic, or dietary causes is generally considered to be non-inflammatory (Turnbull & Cowan 1999). Factors

cited in pathogenesis of DJD include genetics, abnormal joint alignment, excessive stress, trauma, local inflammation, and hormonal influences (McKeag 1992; Lane & Buckwalter 1993; O'Connor & Brandt 1993). Inflammatory arthritis includes a variety of disorders that typically involve reactive bone formation and fall under the general term spondyloarthropathy (SpA). One particular form of SpA is reactive arthritis (ReA), not to be confused with another type of arthritis, rheumatoid arthritis (RA). In humans (ReA) is mediated by a variety of infectious organisms to which panthers may also be susceptible. Variable manifestations of SpA include asymmetrical, pauciarticular (involving less than five joints), peripheral (appendicular) joint erosions and fusion, and axial (spine and pelvis) joint inflammations (Resnick & Niwayama 1988; Rothschild & Martin 1993).

The use of the term DJD is widespread in the both the wildlife and veterinary medicine literature but the descriptive terminology is confusing as its symptoms overlap with that of SpA. Non-inflammatory disorders can often lead to secondary inflammations, and inflammatory joint disorders commonly result in secondary, often severe degenerative changes (Turnbull & Cowan 1999). According to Rothschild et al. (1998, 2001), it is the more inflammatory form of arthritis, (namely SpA), that is the more likely condition in wild mammal populations, including large cats, whereas DJD is more often associated with domestic breeds and zoo animals. SpA has been described in hyenas, bears, canids, non-human primates, elephants, large felids, and mammalian and non-mammalian fossils (Rothschild & Rothschild 1994; Rothschild & Woods 1989, 1991, 1992; Rothschild et al. 1993, 1998, 2001). Turnbull and Cowan (1999) described synovial joint diseases in wild cetaceans that included both degenerative and infectious manifestations.

Medical management, genetic augmentation, and intervention to increase prey species may have contributed to improved health of Florida panthers over the years, which could reduce the expression of health-related skeletal anomalies. Our objective was to review earlier studies, investigate a larger sample of cats from a longer time period, and quantify the observed skeletal features to determine if there have been any changes in the frequencies found by Duckler and Van Valkenburgh (1998a). Further, using multivariate statistical methodology, we explore the interrelationship of HLs, skeletal anomalies observed per individual, and the degree of severity of those anomalies, and to what extent these vary by age, sex and habitat. Any general deviation from normal bone development is referred to as an Abnormal Osteological Feature (AOF), to distinguish it from the many specific types of pathologies that exist in nature and to have a consistent and easy reference. We hope to learn more about the causes of HLs and AOFs, or at least the conditions under which they form, utilizing a large data set and ample life history information about individual Florida panthers.

Considerable analogy with human arthritis research was used for insight into panther bone pathology, as others have done (Rothschild et al. 2001; Turnbull & Cowan 1999), to reveal new directions in arthritis research, and to search for explanations for the high frequencies of HLs and AOPs. The study of osteological material alone cannot provide a definitive diagnosis; therefore this is a first step to categorize an idiopathic disease process that may exist in the panther population of Florida.

METHODS

HARRIS LINES

To document frequency of HLs in Florida panthers, we X-rayed left and right humerii and femora of 69 males and 43 females including animals that died as recently as 2006. HLs were scored when the density line was perpendicular to the long axis of the bone and extended across the entire shaft. This is a more conservative measure as often HLs need only extend one-fourth to one-half way across the shaft of the bone. Ultimately, only humeral HLs were counted, since femora showed very few. Harris Lines do not always occur symmetrically, so we totaled the number of HLs present in L and R humerii. Age, sex, year of death, and use area were recorded for each specimen (Appendix 1). Our X-rayed individuals overlapped with those studied by Duckler and Van Valkenburgh, but did not completely duplicate their series.

ABNORMAL OSTEOLOGICAL FEATURES

Major skeletal elements of Florida panther specimens in FLMNH collections including 77 males 50 females, and one unknown (n=128) were inspected and abnormal osteological features (AOFs), including healed fractures, arthritis, infection, unusual lesions, malformations, and unknown pathologies were tabulated (Appendix 1). We gained insight into bone pathology by referencing studies in archaeology and forensics (Baker and Brothwell 1980; Buikstra & Ubelaker 1994; Rogers & Waldron 1995), paleopathology (Rothschild & Martin 1993), wildlife and veterinary science, and contemporary studies in human arthritis, as well as consultation with forensic specialists. Our results are reported and compared to the earlier study by Duckler and Van Valkenburgh (1998a,b).

We examined the cranial, axial, pelvic, and appendicular skeleton including the feet, and scored any ab-

normality within each. We recorded the total number of AOFs, being conservative in our estimate. For example, an AOF associated with two or more bones, such as tibia-fibula or humerus-radius-ulna joint was only counted once regardless of the number of affected bones. If there were two lesions on one bone, both would be counted only if they were qualitatively different, or located in a different region of the bone, although we could never be certain of the relationship between any two observations because of possible systemic involvement. The skeleton records a lifetime of insults, and there is no way to know for certain that this scoring system adequately distinguished one event from another. No fractures that were related to the cause of death were recorded; that is broken bones without evidence of bone remodeling.

An overall severity score (S) was assigned to each specimen, ranging from S1-Mild, S2-Moderate, to S3-Severe, depending on the nature and extent of the anomaly. Measures of severity suggest the greater or lesser expression of a condition or the later phase of a disease. While neither of these may have any basis in clinical practice (Rogers & Waldron 1995), they do provide a basis for comparison. A severity score of S3 (the most severe case) was assigned only in cases where bones were broken or the deformation from normal state was extreme, and potentially crippling, the latter having been noted either in life through observations and reports, or during necropsy. Since many skeletons had more than one incident that ranged in severity from S1 to S3, the highest single score was selected, since that one would have the greatest potential threat to the survival of that animal.

PANTHER STUDY GROUPS

In many cases, the ages calculated for Florida panthers were estimates, so age classes were established based on several references, including degree of fusion of cranial sutures and epiphyseal closure, direct comparison with animals of known age, and estimates provided in FWC annual reports (Land et al. 2005). Animals were grouped into five age classes (Class I = <1 year, Class II = 1-2 years, Class III = 2-4 years, Class IV = 4-10 years, and Class V = > 10 years. To gain an understanding of the relationship of health parameters (HLs, AOFs) to regions of Florida (north or south), we assigned a "Use Area"(UA) to each animal based on published reports or, in the case of panthers not radiocollared by the FWC, the location where they were found dead. Use areas were defined UA1) north of Interstate 75 (former SR 84 or Alligator Alley), and UA 2) south of Interstate 75 including southern Big Cypress National Preserve (sBCNP), Fakahatchee Strand State Preserve (FSSP), and the Everglades National Park (ENP)(see map in Part II). Past health and diet studies of panthers frequently delineated three distinct regions by considering ENP a separate region, but sufficient samples did not exist, so ENP cats were grouped into UA2.

ANALYSES

The dependent variables in the study are total number of abnormal osteological features (AOFs), severity index (SI) of AOFs, and HLs. The independent variables were sex, age class and use area. The model used to test the dependent variables included all three independent variables and all possible interactions. A Multivariate Analysis of Variance (MANOVA) was performed simultaneously testing all three dependent variables against the independent variables using SAS (SAS Institute Inc., Cary, NC). Once significance was established with the MANOVA, individual ANOVAs were run on each independent and dependent variable. A student's t was used to find differences between Age Class groups whenever significance was established for that variable. All of the individual ANOVAs were analyzed using JMP version 5.0 Statistical software for the Macintosh (SAS Institute Inc., Cary NC).

The relationships between the three dependent variables (HLs, AOFs, and SI) were analyzed using regression analysis. Animals with no osteopathologies were removed because these animals also had a scoring of zero for the AOF severity index. Therefore, only animals with at least one AOF were used to examine trends between the dependent variables. To determine the effect on HL or AOF frequency resulting from the introduction of Texas cats in 1995, we compared the prevalence of HLs and AOFs of animals that were born prior to 1995 (HL n=63, AOF n = 79), to those born since that time (HL n=49, AOF n = 49) using ANOVA.

RESULTS AND DISCUSSION

The MANOVA testing the differences among total AOFs, Severity of AOFs, and total HLs on sex, age class, and use area shows a significant multivariate effect for sex and age class. This test fails to reveal any significant multivariate effect for use area or any interactions of the three independent variables (Table 1).

HARRIS LINES

The average number of HLs increases with each Age Class (AC) for all animals combined (Fig. 2). There is a continuous increase in number of HLs with each successive AC and overall significant differences were found between the ACs (ANOVA; $_{F4,107}$ = 4.82, p =

Table 1: MANOVA results for Florida panthers from the Florida Museum of Natural History. The MANOVA examined three dependent variables; total AOFs, severity of AOFs, and Harris Lines.

Variables	Wilks' Lambda	F statistic	df	p
Sex	0.90	3.14	3/88	0.0292
Age Class	0.59	4.29	12/233.12	<0.0001
Age Class * Sex	0.83	1.37	12/233.12	0.1789
Use Area	0.98	0.46	3/88	0.7006
Use Area * Sex	0.98	0.50	3/88	0.6859
Age Class * Use Area	0.92	0.61	12/233.12	0.8299
Age Class * Use Area * Sex	0.96	0.28	12/233.12	0.9923

0.0013). A significant increase exists from AC-I to AC-IV as well as from AC-IV to AC-V. There is no difference between males and females with respect to HLs, nor was there any significant difference between the average number of HLs for animals from the north, above SR 84, and those from the south, including sBCNP, FSSP and the ENP (MANOVA results; Table 1).

The apparent continual accumulation of HLs throughout life is an unexpected result. Harris Lines develop at the metaphases during growth before the epiphyseal plate fuses to the long bone, at which point bone growth stops. For panthers, this would be approximately between the ages of three and four years. Because bone is remodeled over time, it is also expected that evidence of HLs would disappear with age as bone continuously remodels, so an older animal should have fewer HLs rather than more. One possibility for the continued visibility of HLs in older animals is that previously formed HLs become easier to detect as cats age, perhaps due to a reduction in the thickness of the bone cortex. This has not been studied, however, it is possible to measure cortical bone thickness in individuals

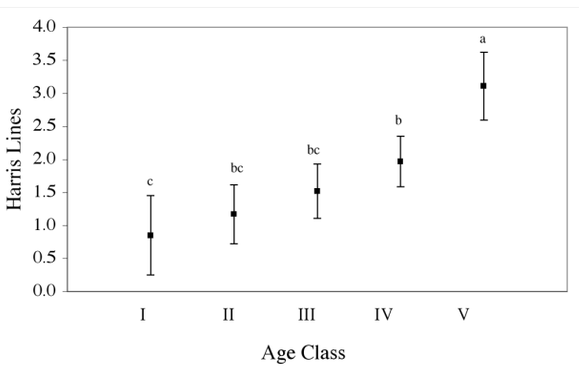


Figure 2: Mean number of Harris Lines for Florida panthers at each age class. Letters represent groups that are significantly different. Sample sizes are; Age class I (n=13), II (n=23), III (n=27), IV (n=31), V (n=18).

that vary in age to determine if this is the case. (A. Falsetti, C. A. Pound Human Identification Lab, pers. comm.). If it is assumed that HLs are indeed an indication of stress in the population, then a likely explanation is that the older animals in our study experienced greater health-related stress at a younger age than those in the younger age classes, a possibility also noted by Duckler and Van Valkenburgh (1998a). This might suggest that health or living conditions have improved since the mid-1980s. This is consistent with information from diet and prey studies conducted in the mid-1980s in the sBCPR and FSSP that reported that animals in the south were eating fewer deer and more smaller prey, and that the general health of animals living in the Fakahatchee was poor (Roelke et al. 1986, Maehr et al. 1990, Roelke 1990).

This time period during the 1980s also reflects a time when deer hunting in the FSSP was legal, but later banned, and therefore panthers and hunters may have been competing for the same large prey species with the result that panthers in FSSP were eating small prey rather than deer and hog (Roelke 1990). This too is a complex issue because many factors affect prey density. Prior to 1980s there was no intentional management on public lands to increase prey (such as deer) for panthers. During the 1980s, actions were taken to reduce access to public lands, reduce hunting pressure and harvest on white-tailed deer and hogs, reduce the use of hunting dogs, and protect does and fawns (Schortemeyer et al. 1991, Beier et al. 2003). Additional factors affecting deer densities are length and intensity of hydroperiod in the ENP that might affect fawn survival and variable quality of vegetation and soil types (Land 1991, Fleming 1994). Improvements in prey base throughout the range of panthers would contribute to a healthier population.

Contemporary arguments exist that HLs do not form as a result of pathological stress or disease, but rather may reflect accelerated growth during specific

early periods of development (Alfonso et al. 2005). Many of the HLs observed were faint, spatially clustered and appeared more frequently in proximal humerii close to the epiphyseal closure. The clustered nature of the HLs may reflect many individual stresses associated with rapid growth during a brief period of time. It is unlikely that this study will clarify the debate on the cause of HLs in vertebrates at least not without further research. However, it should be noted that serious health incidents nearly always result in transverse lines of considerable density, and such lines tend to remain detectable despite subsequent bone remodeling or advanced age (Park 1964, Maat 1984).

Dense lines in panthers frequently occur in the humerus anywhere from slightly distal to the midpoint up to the upper proximal portion of the limb bone. No dense lines in the lower quadrants were observed. If HLs are caused by pathological stress or nutritional deficiencies, then those events may not occur during the earliest months after birth, but rather after cubs become independent and begin to disperse at approximately 14 months of age. This activity might be more stressful, especially for males who move a greater distance than females, who must compete with resident males in other locations (Maehr et al. 2002). Harris Lines formed in very young animals are more likely to disappear as the bone is continuously remodeled during the earliest months of growth. As pointed out by Duckler and Van Valkenburgh (1998a), HLs are more likely to be retained in the adult skeleton when they form close to the time of epiphyseal closure and growth termination.

Duckler and Van Valkenburgh (1998b) also showed that pumas throughout their range have higher incidence of HLs compared to other extant species, including mule deer *(Odocoileus hemionus*), bobcat (*Lynx rufus)*, and gray wolf (*Canis lupus*). This may be due to the stress to the bone brought about by one or a combination of larger size (compared to the wolves), and the reckless nature of their hunting style, in which leaping over boulders, jumping from trees, and incredible spurts of speed are legendary (Sunquist & Sunquist 2002). However, why Florida panthers have more HLs than puma from other geographic locations remains uncertain. A long history of poor health as a result of diet, as well as deleterious consequences of inbreeding manifested as cardiac defects, and high pathogen/parasite loads increased the potential for illness or disease and HL formation (Duckler and Van Valkenburgh 1998b). This remains a plausible explanation.

A superficial review of the life history of several young cats does not support the conventional view that HLs form as a result of one particular stressor or starvation. For example, Florida panther 22 was captured by FWC at six months old, subsequently abandoned by her mother, and was later recaptured by FWC in a starved and dehydrated condition. She was rehabilitated, released, and again in 3-4 months, as a result of another FWC capture attempt, she was separated from her mother and was recaptured again in a debilitated and dehydrated condition. She was brought into captivity and not released until 2 years later (M. Roelke, pers. comm.). In spite of two traumatic separations from her mother, two bouts of starvation, two capture events, and a two-year period in captivity, there is no evidence of HLs in this individual. Her sibling, FP 23 also captured twice had distinct HLs. Florida panther 8, who later in life was captured and found to be underweight and in poor health, had HLs, but they were in the middle portion of the limbs, indicating that the cause of the HL occurred when she was very young. These examples demonstrate that it is not possible to make a direct correlation between episodes of starvation and the presence of Harris lines, particularly in older individuals.

HLs reportedly form as a result of a variety of infectious diseases, as well as protein or vitamin deficiencies. There is also evidence to suggest that they may form in humans as a result of exposure to mercury and other heavy metals (Raber 1999). A study examining the presence of HLs as they relate to mercury levels recorded in individual panthers is underway (M. Cunningham, pers. comm.).

The importance of HLs as indicators of pathological stress has gone in and out of vogue over the past 50 years. Bone formation and homeostasis is a dynamic process, and many physiological processes affect bone health. Earlier research (Duckler and Van Valkenburgh 1998a) has shown that prior to 1995, Florida panthers had significantly more HLs (56%) than non-Florida populations (27%) (Table 3). We are unable to compare our results directly to theirs since we used total HLs for each animal specimen rather than presence per side. In addition, we did not have the opportunity to examine X-rays of the non-Florida sample. In our results, an ANOVA testing for HL differences between animals born before and after 1995 showed no significant difference ($F_{1,110} = 1.31$ $p = 0.2554$) although there is a slight trend showing a reduction in the average number of HLs of post-1995 animals, (1.53) compared to the pre-1995 animals (1.92). Presence of HLs in Florida panthers has not diminished over time in any significant way and HLs continue to be abundant in all age classes. Future research will investigate the relationship between HLs and variables such as overall health, mercury levels, and specific biomedical conditions reported for panthers.

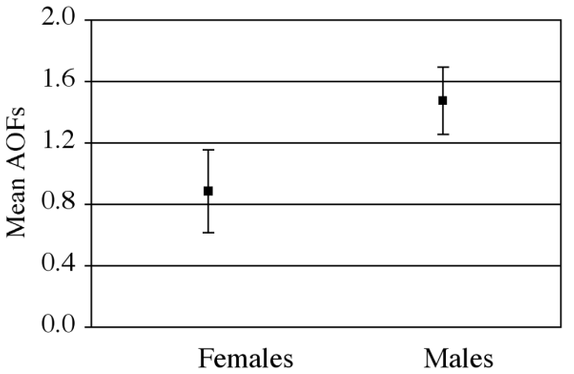


Figure 3: Mean Abnormal Osteological Features (AOFs) for female (n = 50) and male (n = 78) Florida panthers with error bars. Males have significantly more AOFs than females (ANOVA; $F_{1,126} = 4.45$, $p = 0.0369$).

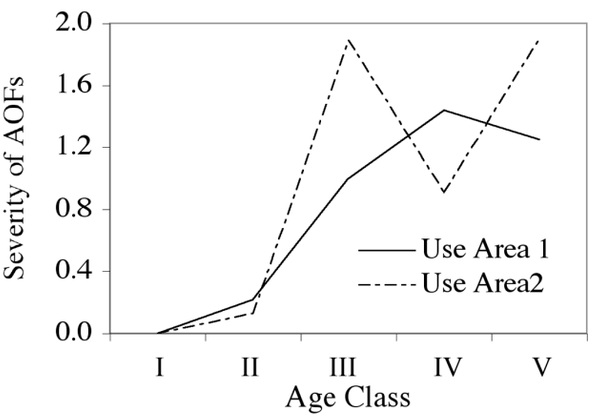


Figure 5: Interaction plot between Florida panthers from the two different use areas. Each point is a mean for each age class. Panthers in age class three show a significant difference between the two use areas (ANOVA $F_{9,116} = 2.55$ $p = 0.043$)

ABNORMAL OSTEOLOGICAL FEATURES

The total number of AOFs was significant for both sex and age class, in the MANOVA analyses (Table 1). Males had more AOFs than females (ANOVA; $F_{1,126} = 4.45$, $p = 0.0369$; Fig. 3), and older animals had more AOFs than younger animals with the total number increasing with each successive age class (ANOVA; $F_{4,123} = 12.84$; $p < 0.0001$; Fig. 4)

There were no significant results in the interaction between any two variables and our dependent variables in the MANOVA. However, when ANOVAs were run for just severity of AOFs, use area, and age class (Fig 5), a significant interaction was found ($F_{9,116} = 2.55$ $p = 0.043$; Fig. 5). These results suggest that a trend exists, but the original MANOVA is not sensitive enough to detect this. Alternatively, it could represent a type I error, but because results are significant it is necessary to mention the possible trend. The severity of the AOFs in Use Area (UA) 2 are more variable than UA 1, but generally animals in this group also have more severe AOFs. Severity (and occurrence) increases between AC-II and AC-III, between ages of approximately two to four. A nearly significant interaction was also found between sex and age class with severity index (SI) of AOF's ($F_{9,115} = 2.36$ $p = 0.057$; Fig. 6). Males and females have a similar low SI in AC-I and AC-II, but males increase dramatically in the severity of AOF's compared to females in AC-III and AC-IV. This corresponds to the period of time that males are dispersing and attempting to establish a territory and home range.

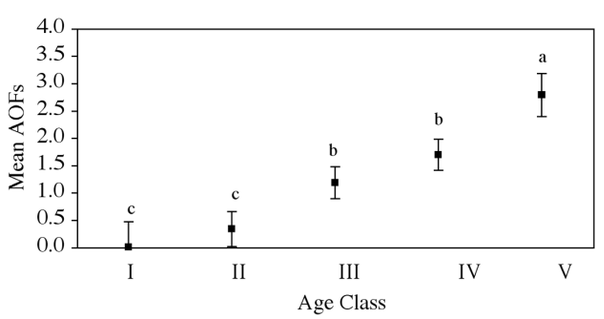


Figure 4: Mean AOFs for Florida panthers for each age class with error bars. Letters represent significant differences between groups. Sample sizes are: Age Class I (n=14), II (n=27), III (n=34), IV (n=35), V (n=18).

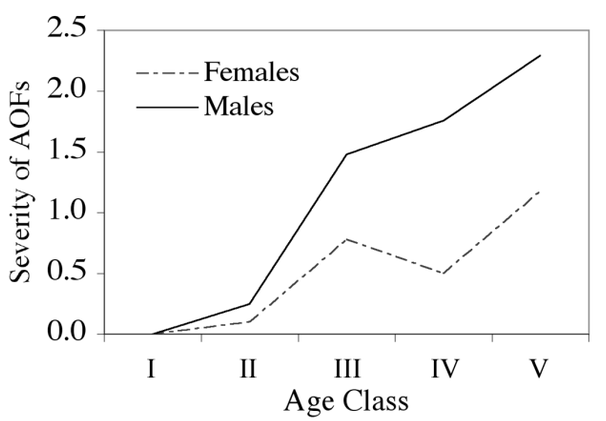


Figure 6: Interaction plot for female and male Florida panthers in each age class. Females and males from the 3rd, 4th and 5th age classes are significantly different from each other (ANOVA $F_{9,115} = 2.37$ $p = 0.057$).

Table 2: Number of Florida panthers in age class for animals born before and after 1995.

Age Class	Pre-1995	Post-1995	Total
1	6	8	14
2	9	18	27
3	21	13	34
4	26	9	35
5	17	1	18

Maehr et al. (2002) describe the process as beginning at approximately 14 months of age and continuing for 7.0 months for females and 9.6 months for males with males' efforts frequently frustrated by insufficient vacant range or range containing no individuals of the opposite sex. Independence and dispersal of young cats can, and probably does, increase food stress as well as encourage, especially in males, male-male conflicts. As males disperse, they also have a greater chance of injury (and death), thus accounting for both the increase in young animals in our collection, and increased evidence of trauma and AOFs.

Our sample consists of animals born prior to 1995 (n=79) and those born since 1995 (n=49; Table 2). There has been a significant reduction of both the number (ANOVA; $F_{1,12} = 13.53$, $p = 0.0003$; Fig. 7) and severity (ANOVA; $F_{1,124} = 6.97$, $p = 0.0093$; data not shown) of AOFs since 1995 in a sample that includes both males and females. There is only one animal in AC- V that was born after 1995 (n = 1; Table 2). This could bias these results because AOFs also increase with age class,

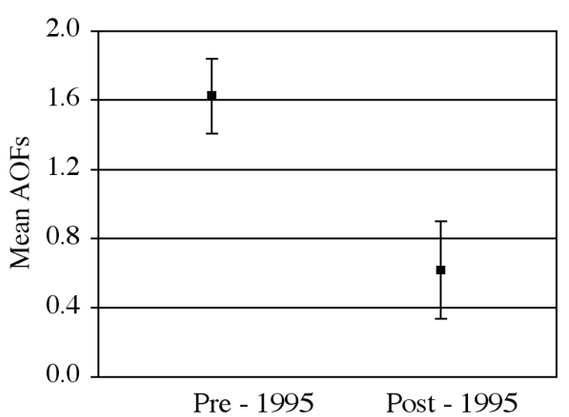


Figure 7: Mean AOFs and error bars for Florida panthers born before 1995 (n = 79) and after 1995 (n = 49). Panthers born after 1995 have significantly fewer AOFs (ANOVA; $F_{1,126} = 13.53$, $p = 0.0003$).

but when we repeat the analysis after removing AC-V, our results remain significant for total AOFs (ANOVA; $F_{1,108} = 9.61$ $p = 0.0025$). This leads us to conclude that there is a trend towards a reduction of AOFs and their severity since 1995.

Although no correlation exists between Harris Lines and total AOFs (Fig. 8A,B), a correlation exists between the number of AOFs and the severity of AOFs (Fig. 8C). This correlation is expected given that an increase in the number of AOFs will increase the probability that at least one of them is severe.

Panthers over the age of two born in Florida over the last 50 years, show a range of mild-to-severe, and potentially debilitating, osteological conditions (Appendix 1). These include trauma (breaks and bite wounds), arthritis, periostitis (disease of the periostium), enthesitis (inflammation of osseous attachment of tendons, ligaments, synovium) and other unidentifiable infections frequently observed in the feet, forearms, and axial skeleton.

The overall incidence of fractures and other trauma remain high when compared to the population of non-Florida panthers analyzed by Duckler and Van Valkenburgh 1998. (Table 3). The overall prevalence of all pathologies in our sample was 60.8% in the pre-1995 sample compared to 69% reported by Duckler and Van Valkenburgh (1998a). However, our scoring protocol differed in that we counted number of AOFs rather than a type and presence/absence per side. This resulted in more incidents of AOFs in our study. We also did not consider HLs in our final totals of AOFs, thereby potentially lowering the score. We preferred to treat HLs separately since there is disagreement as to whether HLs are only associated with illness or injury. Further, we hoped to learn more about the possible relationship of one to the other through these analyses. Regardless of how pathologies were counted, both studies found a greater than 50% incidence of abnormal osteological features in the population of Florida panthers. This is far greater than any published study of abnormal bone development in any wildlife species, although those studies are themselves very rare. Pathologies were less frequent in the post-1995 sample (n=49) with 18 individuals (36.7%) exhibiting one or more pathology compared to 48 (60.8% pre-1995). The fraction of the post-1995 population with pathologies is significantly lower than the pre-1995 sample. However, the proportions of old versus young animals in the two samples vary considerably. There is a 34% increase in animals under two years of age in the post- 1995 sample, and it has already been demonstrated that injuries and other or AOFs accumulate with age (Table 3).

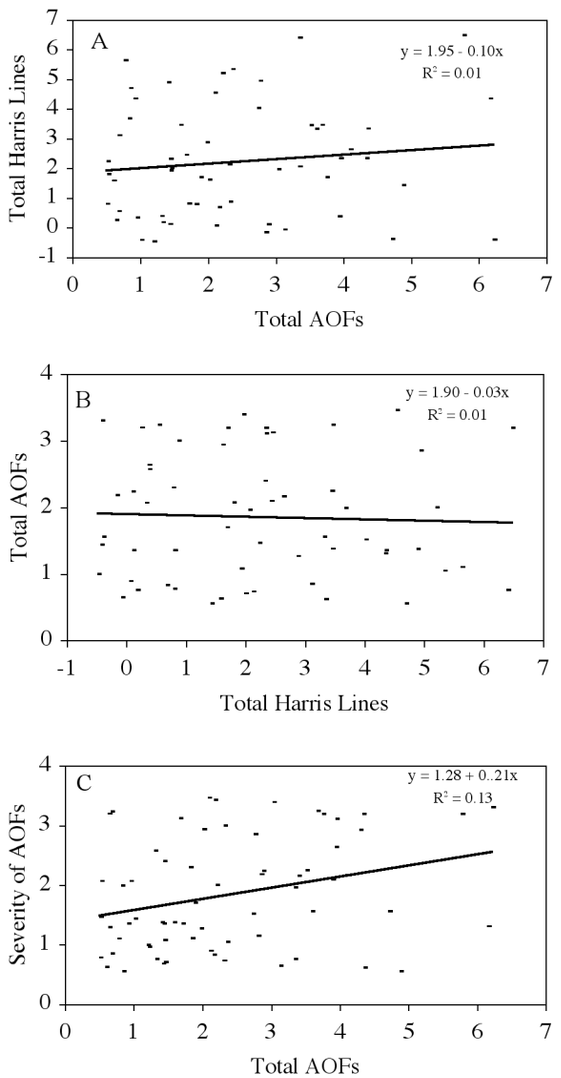


Figure 8: Correlations between total number of Harris Lines (A), total AOFs (B), and severity of AOFs (C) for Florida panthers. Points were jittered (offset) by adding a random number between –1 and 1 to each point so that the points don't lay on top of each other. Animals without any AOFs were removed from the analysis because they also had a zero Severity Index, which would influence the regression line.

The most abundant osteological expression was that of arthritis, which was present in 53.2% (n=42) of the sample of animals born before 1995, but only 30.6% (n=15) of those born after 1995. It might have been easy to dismiss the severe arthritis we observed as age-related, since many of the specimens acquired in the 1980s and early 1990s represent an "aged population" (Ballou et al. 1989; Roelke 1990; Duckler & Van Valkenburgh 1998a). However, arthritis, as the accumulation of osteophytes, even in an incipient form, was present in cats of all ages, except the very youngest (Age Class I). Young animals (those younger than two years of age) did not show significant arthritic lesions, and there are considerably more young animals (53.1%) in our post-1995 sample. Age is a significant factor to be considered in the interpretation of these results, as insults to the skeleton continue to accumulate through life.

Some types of lesions were more prevalent, whereas others were more notable because of their severity. Among the most common AOFs were inflammation and osteophytosis, an inflammatory arthritis, in forelimbs, including elbow and wrist. A 3-year old male (FP 89), reportedly small for his age, had extensive proliferation of bone around the articular surfaces of both the left and right humerus/radius/ulna joints, which was also observed in life as an open wound at the elbow (M. Cunningham, pers. comm.) (Fig. 9). A similar, but even more extreme case is that of FP16 from Dade County. Evidence of osteoarthritis was present in the spine and feet of this 14-year old male, however, all bones of the forelimb showed an advanced joint disease, possibly caused by a broken right radius/ulna, which calcified into a large mass in healing (Fig. 10A). Deep eburnations visible in the trochleae of both humerii and the trochlear notch of both ulnae reflect the loss of the protective cartilage resulting in bone-on-bone contact (Fig. 10B). Long-term instability and/or inflammation were indicated by the excessive bone deposition around both joints suggesting a debilitating and presumably painful condition. In addition, the distal fibulae where they attach to tibiae, both appeared to have been deformed or possibly broken, a condition that is difficult to explain (Fig. 10C).

While these were the most extreme cases, there were other cats with less severe inflammations, but that also involve the olecrenon process, the attachment site of the complex triceps brachii muscle. Often the condition was expressed in both limbs, which might suggest a chronic front limb dysplasia creating the potential for instability, injury and inflammation. Some of these same animals also showed osteophyte formation at distal radius/ulna above the level of the wrist, which may be a related condition. These cases, particularly in the young panther FP89, where there was no obvious evidence of a wound or injury that could result in an infection, alerted us to the possibility that there was a predisposition to inflammation at certain sites. This led us to scrutinize those sites (e.g., elbow joints) where we observed the early signs of inflammation or distortion. One Florida panther (FP 2; UF 20777), shows areas of inflammation at the proximal ulnae, and, both distal left and right ulnae show evidence of injury with subsequent osteophyte formation (Fig. 11). FP 205 (UF26520), approximately 1-1/

Table 3. Number and per cent of individuals with AOFs and Harris Lines in 2 populations of *Puma concolor* over time and a comparison of two studies of Florida panthers. Total individuals[a] (w/combined[3]Osteopathologies/ AOPs)

	Sample (N)	Arthritis	Infection	Trauma	Unknown	Total individuals[a]	Harris Lines[4]	S3 severity[5]	# aged 2 yrs
Previous Study[1]	*Florida*								
Skull	53	0	1 (2%)	1 (2%)	2 (4%)	4 (8%)	0		
Skeleton	51	9 (18%)	4 (8%)	4 (8%)	6 (12%)	35 (69%)	29 (57%)		
	Non-Florida								
Skull	443	0	3 (0.7%)	3 (0.7%)	8 (2%)	14 (3%)	0		
Skeleton	26	3 (12%)	0	4 (15%)	5 (19%)	12 (46%)	7 (27%)		
This Study[2]	*Florida*								
Skull +Skeleton	*AOP/HL*								
Pre-1995	79/63	42 (53.2%)	9 (11.4%)	12 (15.2%)	12 (15.2%)	48 (60.8%)	42 (66.7%)	13 (31.0%)	15 (23.8%)
Post-1995	49/49	15 (30.6%)	1 (2.04%)	5 (10.2%)	3 (6.1%)	18 (36.7%)	29 (59.2%)	5 (17.2%)	26 (53.1%)
Total	128/112					66 (51.6%)	91 (81.3%)		

[1] Duckler and Van Valkenburgh 1998a

[2]The two studies used different parameters in measuring pathology and are not directly comparable; our definition for arthritis was more inclusive of minor skeletal changes

[3-4] Harris Line count in current study is not included in Osteopathology (as it is in the Duckler & Van Valkenburgh study). We do not consider HLs to be strictly
a function of pathology, see text.

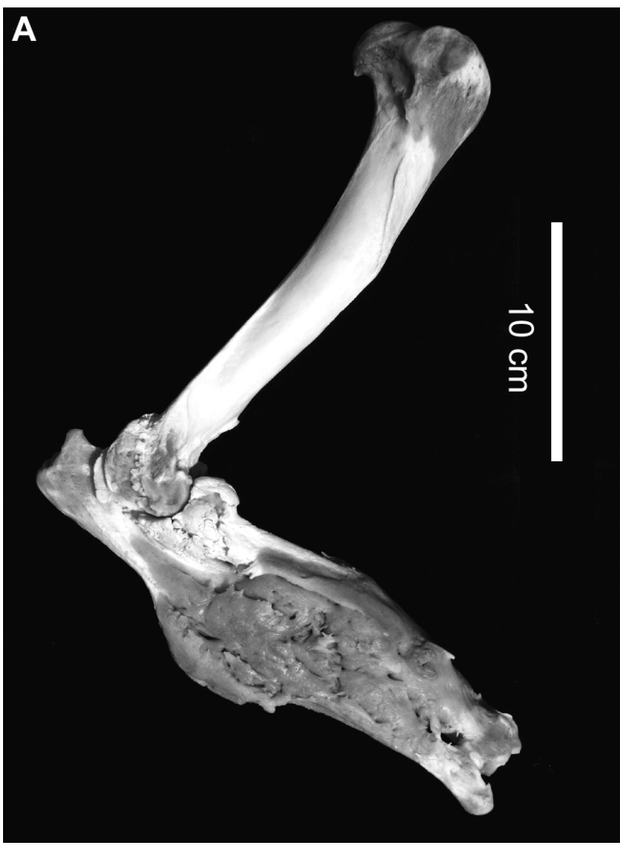
[5] The highest measure of severity represents broken bones, advanced debilitating arthritis, or other extreme deviations from normal bone development; the percentage shown
is based on the number of individuals with a severe condition among the total number of number of individual animals expressing one or more pathologies.

Figure 9: Example of severe inflammatory arthritis in ca. 3-year old Florida panther (FP89, UF30064) that died of intraspecific aggression. Left are the right and left humerii, and corresponding ulnae are seen at the right. In life an open wound was observed at the elbow.

2 years old, shows osteophyte formations at the metaphases (Fig. 12). Note that FP205 was not included in the statistical analyses, because he spent time in captivity. However, it is noteworthy that this animal died of an infection due to esophageal laceration, and the osteophyte formation visible in the photograph may be a systemic reaction to that infection.

A second very distinctive osteopathology worth noting was a severely degraded, porous, and rugose distal ulna in panther FP 10 (UF23986), who was killed by another Florida panther. The remnants of a periostitis infection, suggested by a layer of black film that covered most of the cleaned limb bones (Fig. 13) might have been a contributing factor. There were several young animals that exhibited a very porous state in the distal radius/ulna region of the forearm. Without knowing more about the developmental process we cannot say this is abnormal, only that young cats were vulnerable to osteophyte formation and infections in the distal ulna.

Frequencies of fractures and other trauma are high compared to the population of non-Florida panthers and much higher than in other vertebrates such as deer and wolf (Duckler & Van Valkenburgh 1998a,b). Few comparative studies exist of traumatic, degenerative or developmental lesions among wild carnivores or any mammal species. Among the few is a study of wolves (*Canis lupus*) and coyote (*Canis latrans*) in Saskatchewan (Wobeser 1992). Wolves showed a much greater number of broken bones (22.8%), but degenerative joint disease, involving the spinal column and limb joints similar to that reported here, was found in only seven wolves and two coyotes. As with panther FP16 (UF29821), the articular cartilage of a single wolf was eroded from the condyles of both femurs with eburnation of the underlying bone and extensive periarticular osteophytes were present. One would expect a greater number of injuries in carnivores, such as wolves and puma, than other mammals because hunting is a dangerous activity. Although the osteopathologies of non-Florida wild living puma were high (n=12, 46%), they were not as high as those observed in the Florida population (Duckler and Van Valkenburgh 1998a). Now, ten years later with a sample



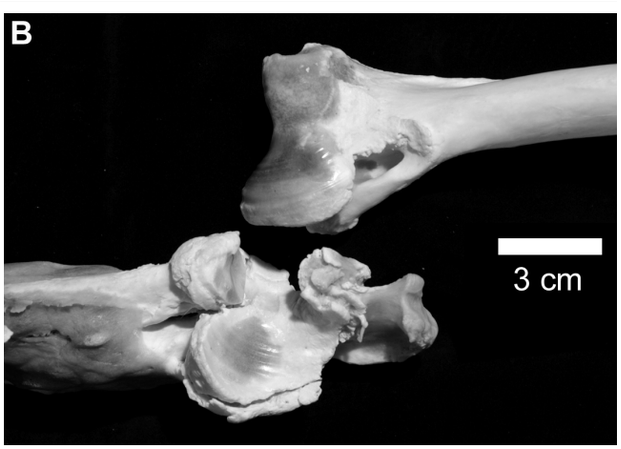
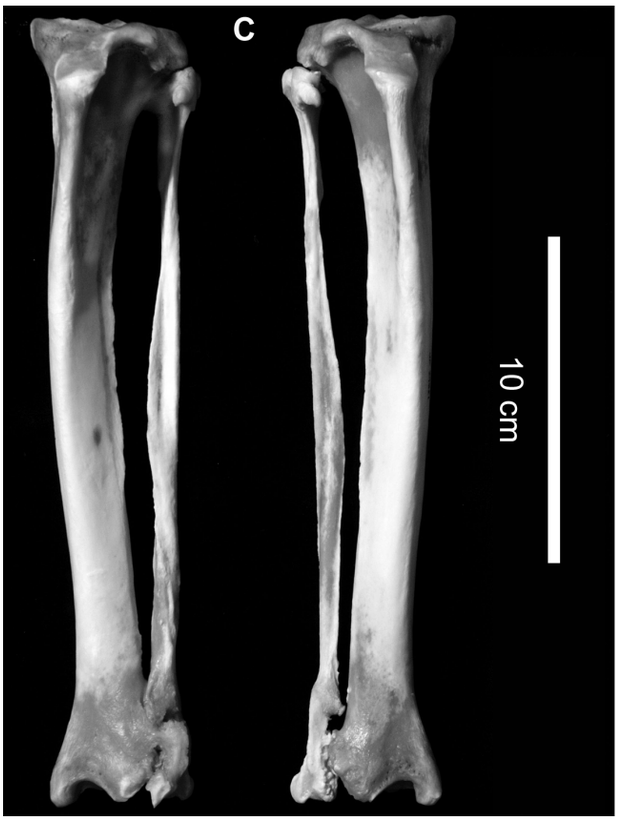


Figure 10: All bones of forelimb show advanced joint disease in this 14-year old male (FP16, UF29821), probably initiated by the broken right radius/ulna (A). Deep eburnations visible in the trochleae of both humerii and the trochlear notch reflect the loss of the protective cartilage, resulting in bone-on-bone contact (B). Distal fibulae, at the attachment site of tibiae, both appeared to have been deformed or previously broken (C).

twice that of Duckler and Van Valkenburgh's study (1998a), we do see a decline in trauma and arthritis. However, once again, it is necessary to consider the proportionate difference in age classes. Following



Duckler and Van Valkenburgh (1998a,b) we have documented a high incidence of injury, arthritis and other pathology associated with the skeletons of Florida panthers. We are not able to attribute them to any specific type of arthritis, since so many have been described (see following discussion of human arthritis studies) but it is apparent that some level of infection is operating, and it is likely that more than one causal agent exists. In several specimens, the degree of osteophytosis alone, whether initially due to an injury with secondary infection, would suggest there is a tendency for a systemic and inflammatory condition to prevail. The consistent involvement of the enthesis of the olecrenon process and the distal radius/ulna suggests abnormal joint alignment or ligamentous instability, particularly because these were often symmetric. This may also cause inflamation. Another consideration is that this front limb joint is more vulnerable to injury since it bears significant stress in many locomotor activities (see McGonagle et al. 2001 below). Thirty-eight panthers have been killed by intraspecific aggression (Land et al. 2005); several of these were severely debilitated.

NEW ARTHRITIS RESEARCH

Before we attempt a discussion of possible causes AOFS, we would like to present a summary of the intensive research in human arthritis during the last two

decades. This provides new information and intriguing insights into the pathogenesis the spondyloarthropathies (SpA), also referred to as arthropathies. References to SpA in wildlife studies are rare, but do exist (Turnbull and Cowan 1999), and recall that they have been identified in museum specimens (Rothschild and his colleagues, op cit.). Spondyloarthpathies include a diverse suite of related conditions in humans, including ankylosing spondylitis, reactive arthritis, psoriatic arthritis, and inflammatory, bowel-disease (Benjamin & McGonagle 2001). Collectively these arthropathies are characterized by inflammatory arthritis, extra-articular inflammation, preceding bacterial infection, seronegativity for rheumatoid factor, and a strong genetic association (HLA-B27) (Dougados et al. 1991; Calin & Taurog 1998). In humans, an important factor associated with the susceptibility of an individual to reactive arthritis is HLA-B27. It appears that B-27 positive individuals are affected more severely, although the pathogenesis is still not fully understood (Toivanen and Toivanen 2004). Today SpA is commonly referred to as enthesopathy because of the involvement of the enthesis, the insertion site of a tendon, ligament, or articular capsule into bone. Enthesitis, the inflammation of an enthesis, is believed to be a unifying concept for SpA (McGonagle et al. 1998). Numerous enthesitic arthritic lesions, some incipient and others grotesquely abundant, were observed at the olecrenon process and distal radius ulna of several young panthers. Enthesitis can accompany many disorders, including traumatic, degenerative, inflammatory, endocrine, and metabolic conditions. In some cases, enthesitis represents the initial or predominant manifestation of disease (Resnick & Niwayama 1983). Furthermore, one aspect of SpA, namely ReA, (reactive arthritis) is now considered in humans to be a disease, triggered by a host of infectious agents including bacteria, parasites, and viruses (Toivanen & Toivanen 2001a,b). The inflammatory expression is the interaction between the infectious agent and the host immune response (Schoen 2000). A hypothesis in the study of psoriatic arthritis is that enthesitis arises at sites of high shear and compression forces, with the additive interaction between mechanical stress, microtrauma, tissue repair mechanisms, and bacterial molecules variably leading to inflammation (McGonagle et al. 2001).

Whether it is possible to extend human research to panthers is as yet unknown. The tendency for front limb inflamation at the enthesis of the humero-radius ulna joint may be due to the biomechanical stresses of the front limbs associated with puma hunting behavior and occasional injury. However, it is also well known that panthers are immuno-compromised and vulnerable

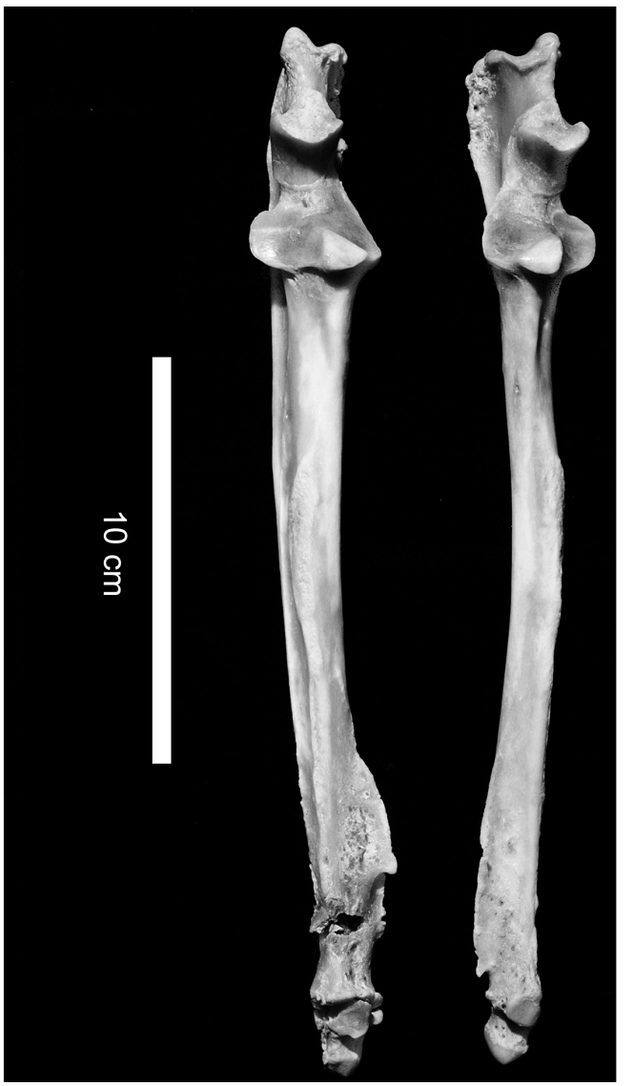


Figure 11: Areas of unusual rugosity or inflamation at the proximal ulnae and osteophyte formation at the distal, possibly due to injury or instability caused by dysplasia (FP 2, UF20777).

to a variety of pathogens (bacterial, viral, and parasitic) possibly as a result of low genetic diversity (Roelke et al. 1993a,b; Glass et al. 1994; Rotstein et al. 2000). Panthers exhibit a variety of congenital abnormalities, such as atrial septic defect and cryptorchidism related to low genetic variability (Beier et al. 2003; Pimm et al. 2006). Elbow dysplasia in dogs is influenced by multifactorial processes including genetic predisposition, and if untreated is known to progress to crippling osteoarthrosis (Pool 2002). Dysplasia and/or the increased susceptibility to osteological inflammation as a result of injury or infection could be another previously unsuspected manifestation of panther inbreeding. Further, inflammations associated with SpA, such as those identified in the el-

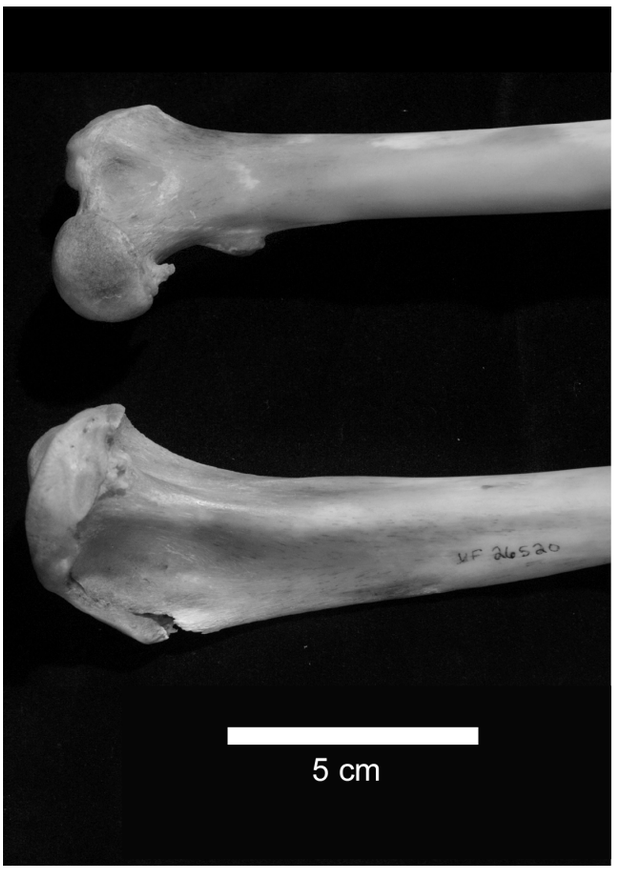


Figure 12: 1.5 year old Florida panther (FP 205, UF26520), shows early osteophyte formation at the metaphysis. This animal died from an infection due to esophageal laceration.

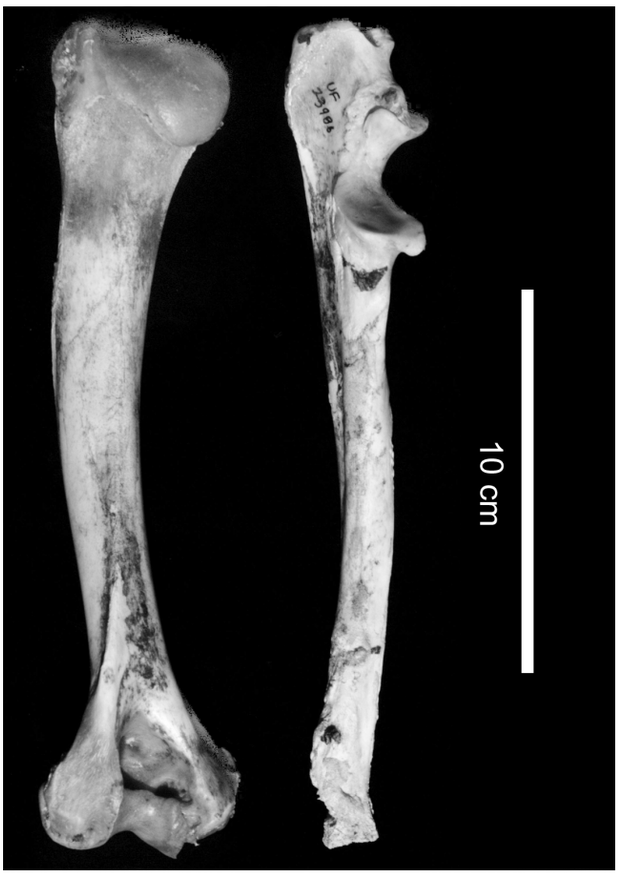


Figure 13: Distal ulna is abnormally porous and rugose; possible cause is a perostitis, an infection of the periostium, suggested by a layer of black film that covered most of the cleaned limb bones (FP 10, UF23986).

bow joint of panthers in this population, have been described in other wild mammals, including cetaceans, and are considered to be debilitating and a significant mortality factor if allowed to progress (Turnbull and Cowan 1999).

Veterinary involvement with the panther began in 1983. Since that time, biomedical research to further the understanding of disease, nutrition, and reproductive physiology has been an integral part of the Florida panther recovery efforts (Roelke 1990, Dunbar 1994). Among the many thousands of biological samples taken over the years including blood, urine, skin biopsy, feces, hair, saliva, viral and bacterial culture swabs, one is of particular interest here; namely Feline Syncytia-forming Virus (Roelke, pers. comm.). Feline Syncytia-forming virus (FeSFV) is common in healthy as well as sick domestic cats, but has been reported in conjunction with chronic progressive polyarthritis. Pedersen et al. (1980) reported that in a study 20 domestic cats with CPA between 1.5 and 5.0 years of age, only males were affected, and two distinct forms of the disease were manifested. The following is their actual account:

"The most prevalent form was characterized by osteopenia and periosteal new bone formation surrounding the affected joints. The second form was characterized by severe subchondral marginal erosions, joint instability, and deformities. The periosteal proliferative forms resembled Reiter's arthritis of man, and the deforming type resembled human rheumatoid arthritis. The disease began as tenosynovitis and synovitis, with subsequent changes in the articular cartilage and periosteal bone. Histopathologic changes in these cats were similar to those occurring in both chronic Reiter's and rheumatoid arthritis of man. Chronic progressive polyarthritis of cats was not caused by identifiable bacteria or

mycoplama, but was etiologically linked to feline leukemia virus (FeLV) and feline syncytia-forming virus (FeSFV) infections."

Pedersen et al. (1980) postulated that polyarthritis was an uncommon manifestation of FeSFV that occurred in predisposed male cats. Feline leukemia virus may not have been directly involved in the disease, but may have acted in some way to potentiate the pathogenic effects of FeSFV. Of all panthers tested for FeSFV from 1987-1992, 59% were found to be infected with FeSFV (original number not disclosed; Dunbar 1994). Sixteen of those panthers reside in the FLMNH collection and some of the males showed signs of severe arthritis, whereas females were less affected. Seven males (FP7, 10, 12, 13, 16, 17, and 20), demonstrated the following arthritis or unknown infections: SI of 3 (n=2), SI of 2 (N=4), and SI of 1(N=1), and five of seven males had three or more episodes of arthritis and infection that we would describe as arthritic, infectious, or having a component that would coincide with a description of enthesititis (inflamation in region of enthesis). Of the eight females represented in our collection, most showed no evidence (N=2), or mild arthritis (N=3), moderate infection on the rear feet (N=1), and more extreme episodes were apparent in old females (N=2, FP18 and 21, 11 and 14 years of age, respectively). It is difficult to come to any conclusions based on this small sample, but males had both more AOFs and more severe AOFs. Tests for FeSFV were discontinued after 1994, however, archived tissue samples exist and it may be worthwhile to explore this medium as a possible contributing factor to the high incidences of AOFs in Florida panthers.

Environmental contaminants such as methylmercury might further compromise the animals' ability to resist disease (Roelke et al. 1991). As well, they may be a contributing factor in the formation of Harris Lines and/or other osteological pathologies. Elevated levels of mercury have been reported in Florida panthers (Newman et al. 2005), and the death of at least one animal (FP27, UF 24557) from the Everglades was suspected to be the result of mercury toxicosis. Mercury is known to interfere with bone metabolism and calcium homeostasis (Suzuki et al. 2004), although it is not known if it exacerbates the inflammatory process. Presence of mercury in south Florida environments may also eventually explain the high incidence of Harris Lines. Dense metaphyseal banding has many possible causal agents, but heavy metals including lead and mercury, can induce physiological changes that result in increased calcium deposition (Raber 1999). An examination of whether Mercury plays a role in the high number of osteopathologies in Florida panthers is warranted.

CONCLUSIONS AND RECOMMENDATIONS

We present preliminary results of an exploration into presence and causes of abnormal osteological features, some of which may have a pathogenic basis, with respect to a timeline that includes genetic out-breeding, biomedical monitoring, and habitat and prey-base improvements. Our study demonstrates that HLs accumulate over time throughout all age classes, but there is no difference in the frequency of HLs between males and females, no difference in the number of HLs observed on cats living in the north versus the south, nor is there any significant difference between pre-1995 and post-1995 populations with respect to number of HLs. Harris Lines remain common in Florida panthers, however their cause is enigmatic and somewhat controversial. Food stress, illness, and infection cannot be eliminated as possible causes for HLs. Exposure to heavy metals, including Mercury, are known to cause HLs in humans as well. We cannot eliminate the possibility that at least some of the HLs are a response to accelerated growth at certain times in the growth cycle. Inasmuch as HLs were also frequent in Puma from other geographic regions, and in much higher frequencies than in other large mammals in general. Another plausible explanation for HLs might be high compression forces operating on the forelimbs. As we are able to gather further information about causes of AOFs, we may be better able to understand HLs and their relationship to bone pathology.

Osteopathology, and specifically joint disease, expressed through the formation of arthritic lesions in the forelimbs, of Florida panthers appears to exist out side the normal limits of a healthy population. Both AOF numbers and severity increase with age, with males showing a more dramatic increase than females, which may correspond to the risks that dispersal places on young animals, particularly males. The potential causes of AOFs are multiple, complex, and probably interrelated. Multiple physiological inflammatory responses to a broken bone are likely, as it presents an opportunity for a pathogenic invasion (Woodard & Riser 1991). We have seen that a break can create a systemic response, with equally well developed and even grotesque arthritic lesions on the opposite limb. Of course, a broken bone creates instability, not only in the damaged area but on other joints as well. The forelimb has shown a much higher incidence of osteophytic development than other limbs even though it is sometimes incipient and difficult to detect. High compression forces operating at this joint may account for the frequency of enthesitis or le-

sions observed at both the elbow and wrist joints. Microtrauma can provide an opportunity for bacterial involvement leading to inflamation and subsequent arthritis. At least some of these osteophytic expressions may be due to one or more of the spondeloarthropathies described in the human medical literature. Collectively, these arthropathies are characterized by inflammatory arthritis, preceding bacterial infection, a strong genetic predisposition, and a propensity for inflammation at the sites where tendons, ligaments, and joint capsules attach at the entheses (Dougados et al. 1991 Calin & Taurog 1998). The role that FeSFV plays in the elevated incidents of AOFs is unknown, but provides at least one link to the presence of polyarthritis in this population.

There was a significant interaction effect between Age Class and Use Area in the severity (and occurrence) of AOFs, with a greater severity occurring in Use Area 2. This is consistent with earlier studies implying that animals in the south were less healthy than in the north, but many management strategies to improve prey base have been implemented. We must consider that we are looking at animals of different ages through a time lens of perhaps 50 years, so it is difficult to pinpoint the specific time frame of the interaction.

The prevalence of total AOFs prior to 1995 (60.8%) was significantly higher than in animals that were born after 1995 (36.7%). Arthritis was the most common AOF in both the pre- and post-1995 samples. There has been a decrease in the incidence of pathology over time. The presence of many more young animals in the current population (relative to pre-1995 populations) may bias the results. However, we cannot rule out, and we can hope, that animals living today are healthier, less genetically compromised, and therefore less likely to develop severe pathologies. Whatever gains have been made as a result of increased variability through outbreeding or prey species management could be reversed as the increase in panther numbers create greater competition for habitat and prey, and offspring continue to mate with each other.

Our initial goal of assessing the effect of out-breeding on the health of Florida panthers vis-à-vis their osteology was limited by sample sizes in the post-1995 higher age classes. There was only one animal in AC-V and nine in AC-IV of the post-1995 sample, compared to 17 and 26, respectively, in pre-1995 sample. Our sample sizes in each group were also skewed with 79 pre-1995 and 49 post-1995. We cannot say with certainty that the out-breeding initiative played an important role in the improved health represented by our results as only eight animals known to be the product of the genetic cross between Texas and Florida cats were included in our study. Most other panthers, with the exception of the Everglades cats, showed at least one of the inbred panther characters of kinked tail or cowlick, and therefore our sample included many more of the original inbreed panther stock. Without the benefit of genetic diversity, we are left with the same basic problems of an inbred population: namely lowered disease resistance and reduced vitality (Roelke, et al. 1993a,b).

We have identified that a predisposition for joint disease may exist in Florida panthers and offer this assessment as a first step in developing a protocol to characterize the nature of this disease. That bone is limited in the way it can respond to any particular "insult" may limit the ability to distinguish among diseases when studying museum skeletons. Therefore, we recommend follow-up with studies that include soft tissue analyses, histology, bone density tests and radiology of joints including the forearm and pelvis joints as the latter is often involved in SpA.

There is new information revealed by this study and the potential for new paths of discovery. Recommendations by a panther advisory commission encourages analyses to consider research in toxins, diseases, and panther health, as well as how the prevalence of abnormalities in panthers is correlated with, or interacts with, genetic status (Beier et al. 2003). Our results, together with the rich resource of archival material, leads to new cooperative research opportunities between museums, wildlife biologists, and wildlife veterinarians in the efforts to improve the conservation status of Florida panthers.

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Appendix I. Florida Panthers listed by FLMNH number (UFID), Fish and Wildlife Conservation Commission number (FP#), age class, pre or post 1995 group, use area, total number of abnormal osteological features (AOFs), severity of AOFs, specific AOFs of Arthritis (AT), Infection (IN), Unknown Cause (UN), Gross Trauma (GT), and number of Harris Lines.

UF ID	FP#	AGE CLASS	PRE-POST 1995[1]	USE AREA[2]	TOTAL AOFs	OSTEO SEVERITY[3]	AT[4]	IN	UN	GT	TOTAL HARRIS LINES
9789		3	1	N	2	1	AT1		UN		
10424	UCFP 06	3	1	S	1	1		IN			2
11915		2	1	S	0	0					0
11927	UCFP 05	2	1	N	0	0					.
14390	UCFP 02	2	1	S	0	0					.
14699	R.ALLEN	3	1	N	0	0					
16374	6	4	1	S	2	2	AT1			GT	1
18798	3	4	1	S	0	0					3
18847	14	5	1	S	1	2	AT2				4
18944	PCO 047	3	1	N	3	2	AT1		UN		.
19077	CB 17	3	1	N	0	0					
19090		4	1	N	0	0					5
19096	1	5	1	S	4	3	AT1	IN		GT	2
20777	2	5	1	S	4	3	AT2	IN		GT	0
20957	UCFP 13	2	1	S	0	0					.
20958	UCFP 12	4	1	S	0	0					.
20973	UCFP 14	2	1	S	0	0					
22409	7	4	1	S	3	2	AT2				0
22529	4	5	1	S	2	1	AT1				1
23849	UCFP 34	2	2	N	0	0					0
23986	10	2	1	N	5	2	AT1	IN	UN		0
24042	Volusia Co.	4	1	N	4	3	AT1				2
24096	13	4	1	N	1	1	AT1				0
24160		4	1	S	1	1				GT	.
24267	8	5	1	S	1	1		IN			4
24268	PCO 059	4	1	S	1	1		IN			.
24314	20	4	1	N	1	2		IN			2
24315	25	4	1	N	1	1	AT				0
24316	24	4	1	N	0	0					3
24557	27	3	1	S	2	3	AT3			GT	.
24561		3	1	N	4	3	AT1		UN	GT	.
24563	15	4	1	S	0	0					3
24595	33	3	1	N	0	0					0
24611	35	1	1	N	0	0					0
24621	30	2	1	S	0	0					0
24644	39	3	1	S	3	1	AT1		UN		0
24645	UCFP 19	1	1	N	0	0					1
24646	17	4	1	N	3	2	AT2				2
24928	18	5	1	N	6	1	AT1			GT	4
24929	41	3	1	N	0	0					1
24931	37	4	1	N	1	1	AT1				.
25908	76	3	2	S	0	0					2
25914	84	2	2	N	0	0					0
25922	PCO 192	1	1	N	0	0					0
26083	43	3	1	N	0	0					1
26157	28	4	1	N	3	2	AT1		UN		0
26159	29	3	1	N	3	1	AT1				

UF ID	FP#	AGE CLASS	PRE-POST 1995[1]	USE AREA[2]	TOTAL AOFs	OSTEO SEVERITY[3]	AT[4]	IN	UN	GT	TOTAL HARRIS LINES
26161	22	4	1	S	0	0					0
26840	47	2	1	S	0	0					3
26841	44	3	1	S	1	3				GT	0
26842	53	1	1	N	0	0					0
26843	50	3	1	N	1	1	AT2				2
26844	34	4	1	N	1	2	AT1				2
26845	UCFP 21	1	1	N	0	0					.
26856	UCFP 22	3	1	N	0	.					0
26938	UCFP 23	1	1	N	0	0					0
26939	26	4	1	N	2	3	AT3				5
27148	31	5	1	N	2	1	AT1				3
27370	38	4	1	N	4	2	AT1	IN	UN		3
27616	12	5	1	N	4	3	AT3	IN		GT	2
27618	52	3	1	N	0	0					3
27700	42	4	1	S	5	1	AT1		UN		1
28713	UCFP 30	2	1	S	0	0					4
28802	58	3	1	N	0	0					0
28980	40	5	1	S	2	1	AT1				1
29199	UCFP 25	3	1	N	2	1	AT1				0
29242	UCFP 26	3	1	S	4	2	AT2			GT	3
29250	68	4	1	N	2	2	AT3	IN			5
29262	45	4	1	N	4	2	AT3				2
29263	51	4	1	S	2	2	AT2			GT	2
29273	72	3	1	N	2	3	AT3			GT	1
29370	46	4	1	N	4	3	AT2		UN		3
29532	64	3	2	N	1	3				GT	1
29566	UCFP 33	1	2	N	0	0					5
29567	74	3	2	N	0	0					1
29621	36	5	1	S	1	1	AT1				6
29819	63	4	2	N	3	3	AT1			GT	2
29821	16	5	1	S	6	3	AT3		UN		6
29826	80	4	2	N	1	1	AT1	`			0
30022	UCFP 35	2	2	N	0	0					0
30023	UCFP 36	2	2	N	0	0					0
30064	89	3	2	S	2	3	AT3				2
30178	90	2	2	N	3	2	AT1			GT	4
30366	UCFP 39	1	2	S	0	0					1
30367	UCFP 38	2	2	N	0	0					0
30374	UCFP 43	3	2	N	1	2				GT	0
30391	11	5	1	N	2	1	AT1				3
30393	23	5	1	N	3	3	AT1	IN	UN		5
30398	UCFP 40	1	2	S	0	0					3
30399	UCFP 41	2	2	N	0	0					2
30430	UCFP 29	3	1	N	0	0					0
30431	97	2	2	N	0	0					1
30433	UCFP 27	2	2	N	0	0					6
30434	105	4	2	N	3	1	AT1	IN	UN		6
30935	49	5	1	N	0	0					5
30936	UCFP 46	1	2	N	0	0					0
30937	UCFP 45	3	2	N	1	3			UN		0
30938	96	2	2	N	0	0					1
30948	98	4	2	N	0	0					2
30957	111	5	1	N	0	0					2

UF ID	FP#	AGE CLASS	PRE-POST 1995[1]	USE AREA[2]	TOTAL AOFs	OSTEO SEVERITY[3]	AT[4]	IN	UN	GT	TOTAL HARRIS LINES
30958	92	3	2	N	1	1	AT1				2
30959	UCFP 42	2	2	N	0	0					0
30960	32	5	1	N	2	1	AT1				5
31010	UCFP 52	2	2	N	0	0					0
31011	67	4	2	N	0	0					0
31012	UCFP 48	1	2	N	0	0					0
31018	106	3	2	N	1	1	AT1				2
31019	UCFP 49	2	2	N	0	0					0
31020	108	2	2	N	0	0					2
31021	78	3	2	N	1	1	AT1				3
31022	UCFP 54	1	2	N	0	0					1
31023	UCFP 58	1	2	N	0	0					0
31024	UCFP 50	3	2	N	1	1	AT1				5
31025	UCFP 53	3	2	N	0	0					3
31026	82	4	2	N	0	0					1
31101	91	4	2	S0	0						0
31103	UCFP 66	2	2	S0	.						0
31104	K 94	3	2	S1	1		AT1				5
31106	55	5	2	S4	1		AT1		UN		3
31108	UCFP 62	1	2	S0	0						0
31109	UCFP 65	2	2	N	0	0					4
31110	114	2	2	N	0	0					0
31161	UCFP 69	2	2	S1	1		AT1				0
31162	UCFP 63	3	2	S2	3		AT1			GT	2
31163	59	4	1	N	4	2	AT2		UN		3
31165	115	4	2	N	2	1	AT1				2
31182	69	4	2	N	1	1	AT1				1
31183	109	5	1	N	6	3	AT3				0

[1]Designates animal born before (1) or after (2) 1995
[2]Panther used areas to the North or South of Alligator Alley
[3]Measure of Severity (1) slight (2) moderate (3) represents broken bones, advanced debilitating arthritis, or other extreme condition