Pathology of Gray Wolf Shoulders: Lessons in Species and Aging

DENNIS LAWLER,[1,2]* JULIA BECKER,[3] JENNIFER REETZ,[4] PAT GOODMANN,[5] RICHARD EVANS,[2] DAVID RUBIN,[6] BASIL TANGREDI,[7] CHRISTOPHER WIDGA,[1] JILL SACKMAN,[8] TERRENCE MARTIN,[1] LUCI KOHN,[9] AND GAIL SMITH[4]

[1]Illinois State Museum, Research and Collections Center, 1011 E Ash St., Springfield, Illinois 62703
[2]Pacific Marine Mammal Center, 20612 Laguna Canyon Rd., Laguna Beach, California 92651
[3]Tippecanoe Animal Hospital, 3818 State Rd. 38 E, Lafayette, Indiana 47905
[4]Department of Clinical Studies School of Veterinary Medicine, University of Pennsylvania, 3900 Spruce St., Philadelphia, Pennsylvania 19104
[5]Wolf Park, 4004 E 800 N, Battle Ground, Indiana 47920
[6]Mallinckrodt Institute of Radiology (Box 8131), Washington University School of Medicine, 510 S. Kingshighway Blvd, St Louis, Missouri 63110
[7]Department of Biology, Green Mountain College, Poultney, Vermont 05764
[8]Numerof & Associates, Inc, Four City Place Drive, St. Louis, Missouri 63141
[9]Department of Biological Sciences, Southern Illinois University, Edwardsville, Illinois 62026

ABSTRACT

We examined scapula glenoids ($n = 14$) and proximal articular humeri ($n = 14$) of seven gray wolves that were maintained in a sanctuary park setting. Immediately after death, observations were made visually *in situ* and by radiography. Further observations were made in a museum laboratory setting, prior to and following clearing of soft tissues. Selected dry bone specimens were evaluated using computed tomography. Significant cartilage erosion and osteoarthropathy were identified in all shoulder joints. No single evaluation method yielded maximal information. Plain film radiography revealed only more severe changes. Computed tomography yielded more detail and clarity than standard radiography. Direct examination of articular cartilage informed about joint soft tissue, and dry bone informed about externally visible bone pathology. These data provide a basis for biological, biomedical, ecological, and archaeological scientists to improve retrospective interpretations of bone lesions. They further support developing plausible differential diagnoses for features of ancient and modern animal bones. We noted a dog-like capacity for wolf longevity in a non-free-roaming environment. However, aged wolves' life spans far exceeded those of similar-sized domestic dogs and breeds, suggesting the possibility of an important species difference that should be explored. We suggest also a hypothesis that the driving force for joint pathology in sheltered non-domestic species may relate significantly to achieving the longevity that is possible biologically, but is uncommon in the wild because of differential stochastic influences. Anat Rec, 299:1338–1347, 2016. © 2016 Wiley Periodicals, Inc.

*Correspondence to: Dennis F. Lawler, 618-632-4950, 1105 Woodleaf Dr., O'Fallon IL 62269. E-mail: dlawler11@yahoo.com

Dr. Jill Sackman is currently at 29080 Inkster Rd., Blue Pearl Veterinary Partners, Southfield, MI 48034, USA.

Dr. Gail Smith is currently at 575 Rock Raymond Rd., Downington, PA 19335, USA.

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INTRODUCTION

The opportunity to examine normal or diseased joints of aged wild animals occurs infrequently. Existing literature supports the idea of numerous ecological impacts on joint diseases during aging (Peterson et al., 2010), including effects of the microecology of the internal environment of physical forces (Thomopoulos et al., 2007). However, there is little indication that degenerative joint disease is a direct outcome of a long-term sheltered ecology for wild animals. This should not be surprising, since degenerative joint disease is known to be nonspecific, with many possible contributing factors (Pedersen et al., 2000).

We found no literature reports of shoulder disease in gray wolves. By comparison, many studies of shoulder diseases of domestic dogs have focused on imaging modalities, osteochondroses (Olsson, 1982), and a variety of congenital or acquired problems involving tendon, ligament, muscle, joint capsule, and nerve structures that surround the joint (Sumner-Smith, 1993). Thus, parallels between gray wolves and domestic dogs need to be examined.

We describe pathological features of the scapula glenoids and proximal humeri of seven aged gray wolves (*Canis lupus* Linnaeus 1758, now *C. l. lupus*). Our observations include: gross postmortem evaluation; radiography and selected dry bone specimens for computed tomography; articular and surrounding soft tissue prior to bone clearing; and dry bone after soft tissues were cleared.

We provide data to support developing differential diagnoses of joint-related lesions in archaeological, ecological, and biosciences investigations. We suggest that the driving force for joint pathology in sheltered nondomestic species is not sheltering, but rather achieving biologically possible aging and longevity.

MATERIALS AND METHODS

Wolf Management

The study population consisted of 7 adult gray wolves (3 females and 4 males). Among the wolves were various causes of death, at ages ranging from 5 to 17 years. Postmortem evaluation was done by the attending veterinarian following the death of each wolf (Table 1).

None of the wolves that we evaluated were born in the wild. Six wolves (Chetan, Ruedi, Miska, Marion, Dharma, and Tristan) were life-long residents at the Wolf Park, Battle Ground IN. The seventh (Eclipse) spent part of her adult life at another sanctuary. We examined familial relationships among these seven wolves.

Depending on age, season, grouping, and activity, the shelter has housing enclosures of 17.0, 6.8, 1.5, 0.5, 0.2, and 0.06 acres. Free-roaming and behavioral exercise is facilitated by opportunities for roaming the 17-acre fenced pasture with a small herd of bison. Bison are formidable prey and were chosen because of their antipredation behaviors. Wolves walked, trotted, loped, sprinted, raced, and dodged, when engaging with bison. Beginning in late 2011, exercise in the 17.0-acre enclosure was curtailed in favor of increased use of the 6.8-acre enclosure, without bison.

The wolves' primary diet is raw whole animal carcass, apportioned so that all wolves have access to all carcass parts at least monthly. Animal carcass feeding includes primarily white-tailed deer, domestic cattle or deceased calves, and occasionally horse, donkey, goat, or sheep. Remains such as hog and fish usually are refused. The wolves self-supplement by hunting small animal prey, and may accept commercial dog foods. None of the wolves were obese during life or at the time of death.

The wolves are vaccinated for rabies, distemper, leptospirosis, parvovirus, coronavirus, adenovirus, parainfluenza virus, and lyme disease. Those that exhibit evidence of illness are isolated and examined, and given appropriate treatment by the attending veterinarian.

Tissue Extraction and Processing

Extraction of bone and joint tissue was done by Wolf Park permit. The attending veterinarian completed the following evaluations: (a) after death, the shoulders, spine, thorax, abdomen, and other major diarthrodial joints were radiographed; (b) the shoulder joint was opened from the lateral aspect, but not disarticulated, and photographs were taken of the glenoid fossa and proximal humeral articulation; (c) after *en bloc* shoulder extraction, radiographs again were taken to maximize radiographic image quality; (d) the remainder of the gross post-mortem examination was completed.

The specimens were transferred to the Illinois State Museum Research and Collections Center, Springfield IL, either frozen ($n = 6$ wolves) or preserved in 10% buffered neutral formalin (Miska). Specimen processing at the Research and Collections Center involved initial trimming of soft tissue, description and photography of intact cartilage and peri-articulum, and incubation in hot water (40°C−45°C) solutions to macerate remaining nonossified tissue by bacterial action. Following incubation of several weeks, soaking in ~15% ammonia solution promoted further removal of fat from bone. A brushing step in detergent frequently was needed to remove tightly adhered exuded fat. Following clearing, bones were rinsed in water and dried.

Shoulder Observations

The following features were evaluated at each stage of bone processing (Table 1).

Radiology. Articular surfaces; articular margins; peri-articular structures; sub-articular condition; humeral intertubercular groove.

TABLE 1. Life History Data of Seven Sanctuary *Canis lupus lupus*.

Wolf (sex)	Birth	Death	Cause	History
Dharma (f)	2010	2015	trauma	litter early onset cataracts; one pulmonic stenosis; post-litter ovariohysterectomy
Eclipse (f)	1997	2013	neoplasia	Rx for chronic foreleg lameness; renal tubulopapillary adenocarcinoma metastatic; part of life span at another sanctuary
Marion (f)	1998	2015	neoplasia	late life persistent estrus-like signs; neuromuscular failure hindquarters; quadruple mastectomy – chronic mastitis; stomatitis & squamous cell papilloma tongue; osteoblastic osteosarcoma metastatic; Rx for orthopedic pain in late life
Tristan (m)	1998	2013	neoplasia	vasectomy; spindle cell carcinoma metastatic (death); squamous cell carcinoma tongue; Rx for chronic spinal or hip pain
Ruedi (m)	2004	2013	unknown found dead	vasectomy; early life nutritional secondary hyperparathyroidism; chronic lameness; spinal deformities; Rx for chronic orthopedic pain
Chetan (m)	2005	2013	spondylosis hip dislocated; urine incontinence	periodic moist eczema; gradual weight loss late life
Miska (m)	1996	2015	rapid terminal decline from unknown cause	skin hepatoid adenoma surgical removal; Rx for chronic hindquarters lameness

TABLE 2. Genetic Relationships Among Seven Study Wolves.

Miska	Two siblings sired-Chetan and Marion
Tristan	Sire of Ruedi
Ruedi	Related only to Tristan
Chetan	Half-sibling of Eclipse (same dam) Half-sibling of Marion (same dam)
Eclipse	Half-sibling of Chetan and Marion (same dam)
Marion	Half-sibling of Chetan and Eclipse
Dharma	No relationship to other study wolves

In situ. Visible articular cartilage; visible articular margins and peri-articular soft tissue structures; visual color variation; shapes of features on articular surfaces; joint capsule.

Pre-clearing soft tissue. Articular cartilage; articular margins and peri-articular structures; visual color variation; shapes of features on articular surfaces; humeral intertubercular groove; joint capsule.

Cleared dried bone. Articular bone, articular margins and peri-articular structures; visual color variation; feature formation in relation to cartilage observations; humeral intertubercular groove.

Severity scores were assigned subjectively as normal (0), mild pathology (1), moderate pathology (2), or severe pathology (3). Some observations were difficult to score, even qualitatively; these were tabulated by presence in right and left shoulder tissues. Only limited statistical evaluation was considered appropriate. We compared subjective joint feature scores by summing within-wolf and comparing the 4 very elderly wolves with the 3 "younger" wolves for each evaluation method, using a chi-square test.

RESULTS

Health Observations

Among this group of seven wolves, one died spontaneously and six were euthanized. Three advanced-life deaths occurred from malignancy (15, 16, 17 years). A fourth aged wolf (19 years) experienced a rapid terminal deterioration from unknown underlying causes. Among three younger wolves, death resulted from trauma ($n = 1$, 5 years), severe multiple hip and spinal disease ($n = 1$, 8 years), and unknown spontaneous cause ($n = 1$, 9 years) (Table 1).

Diseases observed during life predominately were chronic symptomatic orthopedic disorders in six of the seven wolves (Table 1). Neoplasias most probably were short-term late-life events. Their collective medical histories otherwise were not remarkable. There was no suggestion of health (e.g., infection, metabolic diseases) or environmental (e.g., severe trauma, nutritional deficit or

TABLE 3. Combined Wolf Shoulder Scores

	Tristan 15 yr				Ruedi 9 yr				Miska 19 yr				Eclipse 16 yr				Chetan 8 yr				Dharma 5 yr				Marion 17 yr			
Scapula Glenoids	I*	S*	P*	C*	I	S	P	C	I	S	P	C	I	S	P	C	I	S	P	C	I	S	P	C	I	S	P	C
Irregular cartilage surface		3[a]	3			2.5	1.5			3	3			3	3			2	3			1				3		
Non-encircling periarticular osteophytes	1	3			1	3	1	1.5	1				1.5	2.5	3		0				0.5	0		0.5	1			
Encircling periarticular osteophytes			2	1.5						1.5	3	3				3		3	3	3					2		2
Impinging periarticular osteophytes		1.5	1	1	0	1	1	1	0	1.5	3	2.5	0	3	2	2.5	0	2.5	2.5	3	0	0		0	0	1		1
visual color variation articular surface		1.5	3	0.5		2	1.5	1		1.5	2	2		2.5	1	2		3	2.5	3		0		0		2.5		1
Irregular articular bone surface	0			1	0			1.5	0			2.5	0.5			3	0.5			3	0			0.5	0			2
Feature bands on articular surface		2	2.5	0		1.5	1	0		0.5	2.5	1.5		1.5	1	2		3	3	3		0		0		3		0
Erosion articular cartilage		3	3			2.5	2			3	3			3	3			2	3			0				3		
Sub-articular sclerosis	0				0				0				0				1				0							
Subtotal	1	14	14.5	4	1	12.5	8	5	1	11	16.5	11.5	2	15.5	13	12.5	1.5	15.5	17	15	0.5	1		1.5	1	14.5		6
Proximal Humeri																												
Irregular cartilage surface		3	3			2	2.5			2	3			3	3			2	3			1				3		
Non-encircling periarticular osteophytes	1	3			1		1		1	2			1.5	2			2				1	1			1			
Encircling periarticular osteophytes			2	3		1.5		2			2.5	2.5			2	3		3	3	3				1		2		3
Impinging periarticular osteophytes		2	1.5	2	0	1.5	1	2	0	1	2.5	3	0	3	1	3	0	2.5	3	3	0	0		0	0	1		3
Visual color variation articular surface		2	2	0		2.5	2.5	0.5		2	2.5	1		3	2	2		3	3	2.5		0		0		2.5		0.5
Irregular articular bone surface	0			0.5	0			2	0			1.5	1			3	0.5			2	0			0	0			2
Feature bands on articular surface		2.5	3	0		2	2	0.5		1.5	3	0.5		3	2.5	3		3	3	2.5		0		0		3		0
Erosion articular cartilage		3	3			2	2			2.5	3			3	3			3	3			0				3		
Sub-articular sclerosis	0				0				0				0				0				0							
Osteophytes intertubercular groove	0.5			2.5	0		1	0.5	0.5		1.5	3	1		3	3	1			3	0			0.5	0			2.5
Joint capsule thickening		2				2				2.5				3				3										
Subtotal	1.5	17.5	14.5	8	1	13.5	12	7.5	1.5	13.5	18	11.5	3.5	20	16.5	17	4	19.5	18	16	1	2		1.5	1	14.5		11
Column total	2.5	31.5	29	12	2	26	20	12.5	2.5	24.5	34.5	23	5.5	35.5	29.5	29.5	5.5	35	**35**	**31**	**1.5**	**3**		**2.5**	**2**	**29**		**17**

*I, imaging observations; *S, in situ* observations; P, pre-clearing observations; C, post-clearing observations.
[a§]Severity 0-normal; 1-mild pathology; 2-moderate pathology; 3-severe pathology.

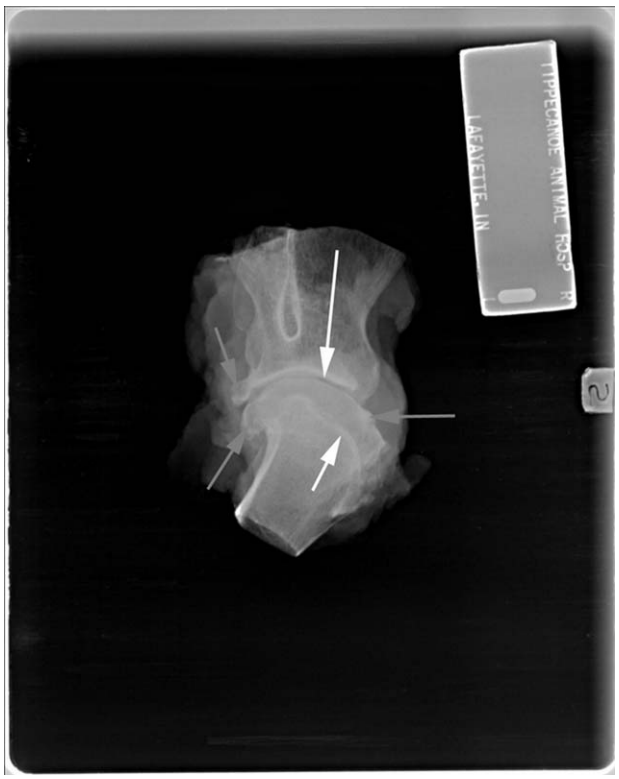

Fig. 1. Chetan, left shoulder radiograph: periarticular osteophytes—gray arrows; overly prominent glenoid articular margin-long white arrow; rim of osteophytes on the proximal humerus-short white arrow.

TABLE 4. Non-scored observations

Feature
regional soft tissues difficult to evaluate – left & right shoulders: Tristan, Ruedi, Chetan, Dharma, Marion
regional soft tissues appear normal – left & right shoulders: Miska, Eclipse
glenoid fossa incomplete bone plate fusion – right shoulder: Reudi; left shoulder: Miska
glenoid tuberosity foci calcified cartilage – left & right shoulders: Miska, Eclipse, Chetan
glenoid fossa focal calcified cartilage – left & right shoulders: Eclipse, Chetan; left shoulder: Miska
glenoid foci grey discoloration (sub-erosive) – left & right shoulders: Eclipse, Chetan; right shoulder: Tristan
humeral articular foci calcified cartilage – left & right shoulders: Miska, Eclipse, Chetan
humerus grey-tan surface color - deep erosion – left & right shoulders: Eclipse, Chetan; left shoulder:Ruedi
glenoid polished brown articular bone - contact – left & right shoulders: Chetan
humerus polished brown articular bone - contact – left & right shoulders: Chetan
humerus osteophytes on articular surfaces – left & right shoulders: Eclipse; left shoulder: Reudi
narrowed joint space – left shoulder: Chetan
matching lesions glenoid & proximal humerus – left & right shoulders: Eclipse, Chetan
flat focus caudal humeral head – left shoulder: Chetan
lesser tubercle humerus, irregular contour – left & right shoulders: Tristan, Eclipse; right shoulder: Chetan

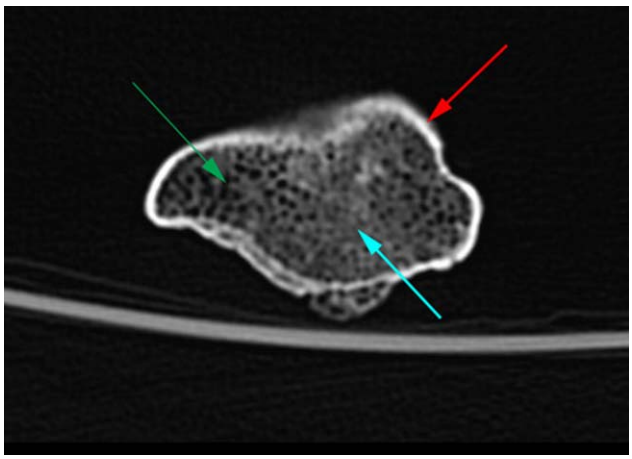

Fig. 2. Chetan, left glenoid fossa, computed tomography view: subchondral sclerosis-blue arrow; compare with nonsclerotic trabecular region-green arrow. Red arrow points to joint margin that is more dense than expected, suggesting pathological change.

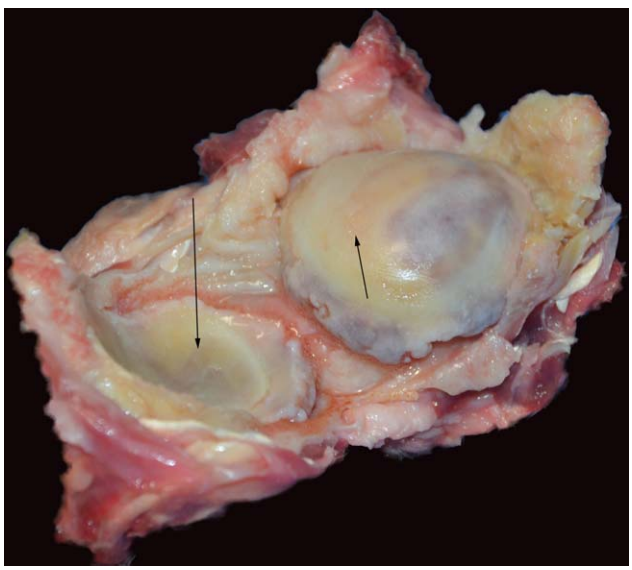

Fig. 3. Chetan, left shoulder at necropsy: glenoid fossa-long arrow; humeral articular surface-short arrow. Note tendency for features to occur in bands on both surfaces, and visible osteophytes impinging the articular margin.

excess) contributions to degenerative shoulder joint disease, with the possible exception of Ruedi. As a puppy, Ruedi experienced nutritional secondary hyperparathyroidism that left him with limb and spinal deformities that may have contributed to degenerative joint disease secondary to chronically altered force vectors during movement.

Three family groupings were identified: Ruedi and Tristan were son and sire, respectively, but were unrelated to the other five wolves. Dharma was entirely unrelated to the other wolves. Miska, Chetan, Eclipse, and Marion formed a loose family grouping built on an "uncle" and several half-sibling relationships (Table 2).

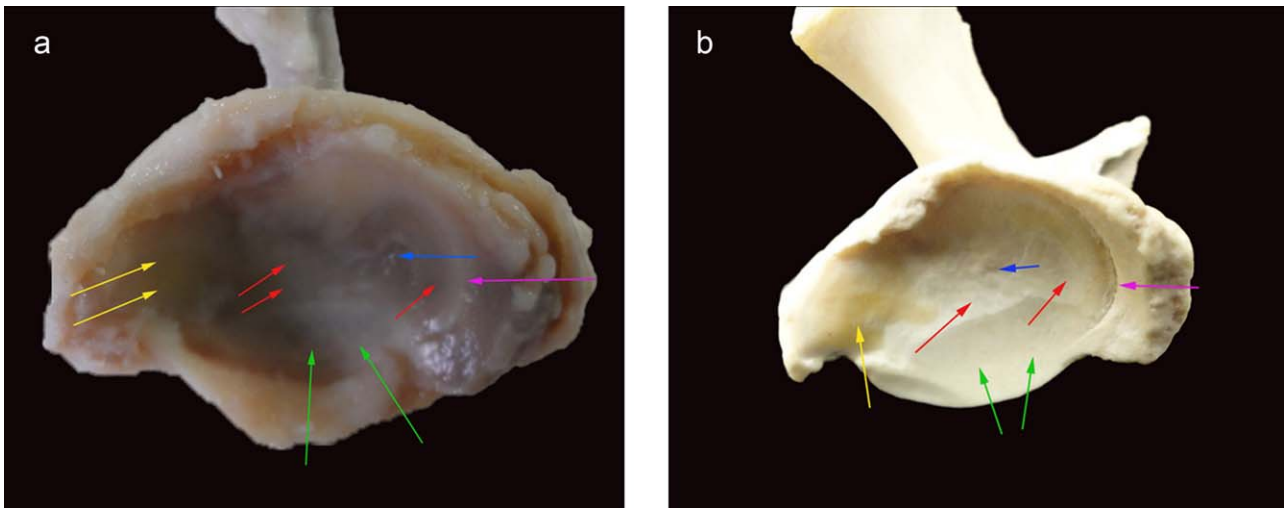

Fig. 4. (a) Chetan, uncleared left glenoid: centrolateral zone of eburnation-green arrows; caudolateral shelf margin-violet arrow; deep erosion focus-blue arrow; severe surface erosion zone-red arrows; yellow calcified cartilage zone with thin cartilage covering-yellow arrows. Note tendency for features to occur in bands. See color-matched arrows Figure 4b, identifying same features. Note severe articular margin/periarticular osteophytosis. (**b**) Chetan, cleared left glenoid: centrolateral zone of eburnation-green arrows; caudolateral shelf bony margin-violet arrow; deep erosion focus-blue arrow; severe surface erosion zone-red arrows, faint gray cast suggesting residual necrotic material; calcified cartilage zone, probably retained deepest cartilage layer-yellow arrows. Note tendency for features to occur in bands. See color-matched arrows Fig. 4a, identifying same features. Note severe articular margin/periarticular osteophytosis.

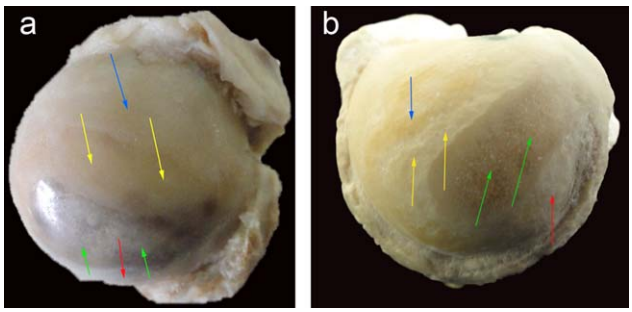

Fig. 5. (a) Chetan, uncleared left proximal humeral articular surface: dark zone of eburnation-green arrows; zone of severe cartilage surface erosion-yellow arrows; zone of moderate cartilage surface erosion-blue arrow; peripheral zone of transition to osteophyte production-red arrow. Note tendency for features to occur in bands. See color-matched arrows Fig. 5b, identifying same features. (**b**) Chetan, cleared left proximal humeral articular surface: dark zone of eburnation-green arrows, note subsurface bony spicules; zone of severe cartilage erosion-yellow arrows; zone of moderate cartilage surface erosion-blue arrow; peripheral zone with faint gray cast suggesting residual necrotic material and bordering articular margin/periarticular osteophytes-red arrow; zone of probable retained, calcified lowest cartilage layer-blue arrow. Note tendency for features to occur in bands. See color-matched arrows Fig. 5a, identifying same features.

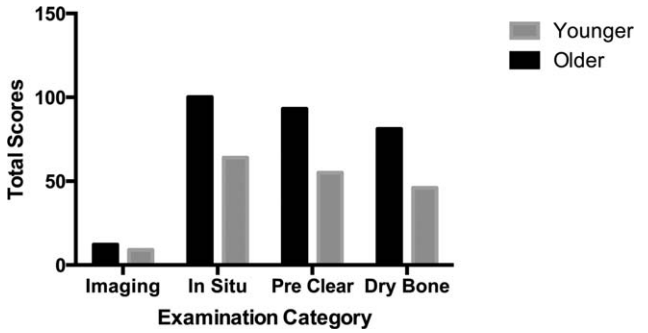

Fig. 6. Histogram showing total subjective pathology scores by examination category.

Shoulder Observations

The exposures that were taken post-extraction offered the best quality images for radiographic interpretation. Plain film radiography underestimated osteoarthropathy identified by other observational methods (Table 3) (Fig. 1). Narrowed left shoulder radiographic joint space in the most severely affected wolf (Chetan) did correctly predict severe loss of articular cartilage. Regional soft tissues were difficult to evaluate because of projection limitations and post-mortem artifact (Table 4).

Bones from Chetan and Miska were selected for computed tomography to evaluate subchondral bone, confirming that subchondral sclerosis was present (Fig. 2). As expected, computed tomography afforded observation in greater detail, compared with plain film radiography.

In situ studies physically exposed more tissue than would be seen by arthroscopy, and enabled recognition of thickened capsules of all joints. The examinations also revealed irregular articular cartilage surfaces; periarticular osteophytes; osteophytes impinging articular surfaces; cartilage color variation; feature orientation in bands across articular surfaces; and cartilage erosion (Table 3) (Fig. 3).

As with radiography, whether peri-articular osteophytes were encircling the articular surface or were nonencircling was not predicted well *in situ*. As expected, articular bone changes remained masked by overlying

articular cartilage at *in situ* examination (Table 3) (Fig. 3).

Pre-clearing visual studies suggested some modification of superficial articular cartilage following *in situ* evaluation, even though the shoulders were either frozen or formalin-preserved. Irregular and eroded articular cartilage surfaces; peri-articular and joint surface-impinging osteophytes; cartilage color variation; feature orientation in bands across articular surfaces; and osteophytes along the humeral intertubercular groove, all were recognized by visual inspection (Table 3) (Figs. 4a and 5a). The minor modification of superficial articular cartilage by freezing and thawing did not affect outcomes.

Dry bone, evaluated post-clearing, revealed pathological changes that were obscured by previous evaluations. Observations included irregular articular bone surfaces; peri-articular and joint surface-impinging osteophytes; bone color variation; feature orientation in bands across articular surfaces; and osteophytes along the humeral intertubercular groove (Table 3) (Figs. 4b and 5b).

Additional observations revealed that pathological features observed on cleared bone related spatially to overlying cartilage pathology during life. Further, the severity of pathological changes on dry bone reflected the severity of overlying cartilage pathology (Figs. 4a,4b and 5a,b).

Limited statistical evaluation (Table 3) showed that (a) imaging was less revealing than direct observation; (b) older wolves had generally higher numerical pathology scores (Fig. 6). No association was found between examination method and age group ($X^2 = 0.49$; df = 3; $P = 0.92$).

DISCUSSION

Davis (1949) published a treatise on shoulder morphology and function in Ursidae. The work described the planar alignment of the tetrapod carnivore shoulder and forelimb, functioning: (a) in load transmission (body to limb); (b) as a strut system of lateral extrinsic (origin off the limb, insertion on limb bones) muscles opposing medial extrinsic muscles for support and movement; and (c) as an intrinsic (origin and insertion on limb bones) muscle levering system to achieve motion on anteroposterior, transverse, and rotational axes.

Skeletal and functional features among Carnivora suggest that the range of expression of degenerative joint changes might be similar across the Order, Ursids being thus typical in this respect. In this context, we consider potential implications of our observations.

Population Implications

Gray wolves are immediate ancestors to domestic dogs, and the two are biologically capable of interbreeding. Considering the close species relationship, we suggest several population implications of our results:

1. Observing similar pathological joint changes among closely related species likely reflects phylogenetic conservation of physiological response capacity (Lawler, 2011), as well as fixed traits that define physical limits for morphological responses (Ham, 1969). A question for further study involves implications of similar degenerative joint pathology among species in circumstances of distant phylogenetic relationship, especially co-occurring with ecological dissimilarity. The latter could imply broad evolutionary conservation of response capacities, and would raise the additional question of the role(s) of conserved joint biology in the aging process (Lawler, 2011, 2015).

2. Mech (1970) estimated that 20% of an Ontario wolf population was over age 5 years, and showed (1998) that 6% of an Alaska wolf population was over age 5 years. Halfpenny (2003) described 20% loss of Yellowstone (USA) wolves by age 2 years, with survival beyond 2 years bringing an expected 5 years' longevity; 10 years longevity was uncommon. Mech (2006) described a wolf population in northeastern Minnesota, indicating that just 12% of "non-pups" were over age 5 years. Mech (1988) also summarized studies suggesting that sheltered wolves might live 9 − 16 years.

   Population-based age estimates can vary for many reasons. Influences that change population demographics over time can include climate impacts, population health, food availability, deliberate persecution, predation, accidents, and some level of sheltering by humans. Further, methods of age assessment sometimes suffer from biological inadequacy (Landon et al., 1998) and social or logistical barriers (Musiani and Paquet, 2004), thus compromising interpretations. Nonetheless, the survival estimates from the aforementioned studies are consistent with unlikely survival into late life for wolves in the wild. Further, the documented advanced ages of the wolves we evaluated clearly demonstrate the genomic capacity for advanced age in this species.

   More generally, Olshansky (2010) observed that aging-related diseases are uncommon outside of domestication or a sheltered environment. Frailty, malnutrition, and predation reflect the rigors of free-roaming existence, and mean life spans in populations are reduced in result (Olshanksy, 2010). On the other hand, events observed in sheltered populations reflect genomically possible population outcomes when stochastic causes of death are minimized and advanced life occurs more frequently. Our data serve as another confirmation of Olshansky's observations, and underscore the importance of excluding stochastic causes for mortality in studies of natural biological aging.

3. Another genomic consideration concerns our demonstration that gray wolves have the same capacity for longevity as do domestic dogs. It is further noteworthy that the older wolves expressed a capacity for longevity that rarely is observed in the largest domestic dogs. The explanation for this difference presently is unclear, but we advance a hypothesis that post-Victorian breeding selection practices applied to domestic dogs by humans have created a subspecies contrast in this respect. The IGF-1 gene of domestic dogs was found to explain 50% of population variation in breed size (Sutter et al., 2007), which with our observations, supports considering new research in comparative quantitative trait inheritance among canids. Comparing circulating IGF-1 levels in young and elderly wolves to those of large and giant breed dogs of different ages especially should be enlightening.

4. None of the wolves that we evaluated were born in the wild. Does sheltering lead to the pathology that we observed? The wolves in our study were not free-roaming, but were not confined in the manner of a zoological garden or domestic pets. The wolves' clinical histories document no lifetime history of obesity, ruling out a ubiquitous major causal co-morbidity for degenerative joint disease of domestic dogs (Lawler et al., 2008). The most and least severely affected wolves were Dharma and Chetan, the two youngest at ages 5 and 8 years, which reflects variability expected in domestic dog populations (Lawler et al., 2008; Chase et al., 2011). By favorable comparison, a recent study of a Japanese village population offers species-comparative data, revealing an age basis for shoulder joint osteoarthritis, with greater occurrence in persons over age 65 years (Kobayashi et al., 2014).

   It has been reported that onset of overt symptoms of degenerative joint disease in dogs frequently follows morphological onset by several years (Lawler et al., 2008). Thus, the time of morphological onset cannot be defined without having serial radiographs over the lifetime of dog or wolf. The clear implication is that extrapolative interpretation of (longevity x pathology) outcomes is tenuous without longitudinal data.

Pathology Implications

1. In a previous report of shoulder pathology of domestic dogs, a high percentage of a study population had radiographic evidence of osteoarthritis by age 8 years, with progression in the population from the 6th year (Runge et al., 2008). By comparison, 6 of the 7 wolves in our study were $\geq$ age 8 years, and all 7 had radiographically visible shoulder pathology. The comparative observation raises the question of whether an "aging threshold" exists in the gray wolf, as lifetime data appear to suggest for the domestic dog (Lawler et al., 2008). Beyond such a threshold, various degenerative processes should be more readily identified, as shown by the late life occurrence of neoplasia in our study population. Only a quantity of new research will clarify the threshold question.

   Our observations also align with those reported from post-mortem examinations of 88 domestic dogs of similar chronological ages as the wolves (Ljunggren and Olsson, 1975). Lesions in the dogs were caudocentralized on the humeral and glenoid articular surfaces. Peripheral fibrosis and osteophytosis occurred with more advanced cartilage degradation. The authors noted relatively high frequency of age-related lesions, with lesion patterns that did not respect sex or body size. Also noted was lack of clear inciting causes beyond aging (Ljunggren and Olsson, 1975). The bone pathologies reported by Ljunggren and Olsson are spatially and characteristically the same as those we recorded for the gray wolf, except that our study did not involve osteochondrosis that tends to occur in younger subjects (Ljunggren and Olsson, 1975).

   We observed a spatial relationship between cartilage erosion and underlying bone features, consistent with chronic, deep erosive processes in cartilage that involve underlying bone secondarily. Simon and colleagues (1973) demonstrated that scapular and humeral articular contact varies from 47% (flexed) to 62% (standing) in domestic dogs, the humeral articular surface being much larger. Thus, in the canine shoulder, the scapular glenoid, and articular proximal humerus, are not in constant full point-to-point contact. Korvick and Athanasiou (1997) evaluated scapular and humeral articulations from seven normal dogs, observing that cartilage damage does not occur in adult dogs as the result of differential mechanical properties between scapular and humeral cartilage. Thus, the accumulated data suggest that cartilage degradation and its associated progressive histological inflammatory process are responsible for the aligned articular bone features that we observed in the gray wolves.

   A thought should be added with respect to pain-related debilitation. Individuals perceive pain differently, some being more sensitive than others (Johnston, 2000). Maddox et al. (2013) noted that computed tomography findings in dogs were not always associated with shoulder pain, and that age was a risk factor for shoulder osteoarthritis. Thus, the degree of morphological severity of degenerative joint disease is not an obligate indicator of the degree of debilitation. Inferring a severity-debilitation relationship in postmortem and archaeological specimens is possible only in a general way. Interpreting the relationship as the cause of death from archaeological specimens is tenuous at best.

   Lastly, the range of fatal and non-fatal diseases that were recognized in the wolves we studied is not unlike those that can be found in domestic dog populations (Ljunggren and Olsson, 1975; Lawler et al., 2008, Chase et al., 2011). Again, the comparative evaluation suggests a high degree of similarity or identity between wolf and dog joint aging, and raises the new question of similar parallels among other Canidae.

2. Data from field studies of wolf mortality are weighted heavily toward traumatic death (Wobeser, 1992), documenting early-to-midlife attrition from stochastic causes. Fritts and Caywood (1980) described a radio-tracked *Canis lupus lupus* with severe osteoarthrosis of shoulders and other evidence of prior trauma. Cross (1940) described two adult timber wolves (*Canis lupus lycaon*), both with osteoarthrosis of diarthrodial joints and vertebral pathology, along with other evidence of prior trauma. Both authors probably misdiagnosed the wolves' ages by overestimation, given that dental condition reflects nutrition, environment, behavior, and health in various ways. It appears that one author also overlooked the role of repeated trauma in chronic bone changes. Even so, the pattern of wolf osteoarthropathy resembled that seen in domestic dogs.

3. Methodological comparison of shoulder osteochondrosis using magnetic resonance imaging (MRI), arthrography, arthroscopy, and histology, revealed correlations among the methods (van Bree et al., 1993). In that study, MRI demonstrated low-signal foci coincident with inflammation of subchondral bone, as well as mixed signaling from degenerative cartilage.

In another study, ultrasonographic examination was shown to be useful as an imaging approach to canine shoulder osteochondrosis, joint mice, joint effusion, and (quoting the authors) "distinct new bone formation" (Vandevelde et al., 2006). A more recent report indicated that both methodology (including MRI) and positioning for evaluation are important to diagnosis of osteochondrosis (Wall et al., 2014), and the authors recommended more frequent use of advanced imaging in suspected occurrences.

Our data indicate that computed tomography yielded more information than radiography, while direct examination of postmortem specimens afforded greater opportunity to interpret other observations. Thus, imaging modalities that are used should reflect study circumstances as well as the information that investigators are seeking.

4. Olsson (1971) reported morphology of subchondral sclerosis and joint margin osteophytes, again reflecting compatibility with our observations of gray wolves. Involved subchondral bone becomes hypomineralized, microfractured, and develops microstructural alterations resembling cysts (Li et al., 2013). When interpreting archaeological canid dry bone specimens, it is useful to retain this mental view of the overall process of degenerative joint disease, as observed in the domestic dog.

5. Gross morphology and histology of the glenoid labrum in dogs has defined the relationship among articular cartilage, subchondral bone, and the collection of supporting structures and their attachments (Sager et al., 2013).

The close association among the tendon of origin of the biceps muscle, fibrocartilagenous labrum, medial and lateral glenohumeral ligaments, and a meniscoid fold, illustrate the complexity of the mammalian shoulder support system (Sager et al., 2013). The elucidated anatomical relationships underscore the potential for soft tissue injury immediately proximate to the articular surface, and thus expand the list of potential differential diagnoses in biomedical and archaeological investigations of degenerative joint disease. Clearly, further studies of degenerative joint disease in wildlife should include investigation of the shoulder joint support system.

6. Studies of induced or naturally occurring osteoarthritis have identified various cytokines, chemokines, and matrix metalloproteases that can be detected in biological fluids. Further elucidating molecular events that occur during development of canid degenerative joint disease will support new understanding of response commonality across species (Garner et al., 2011) and the nature of its origins in genetic conservation.

CONCLUSION

The data that we present here support developing improved and more accurate interpretation of archaeological observations of articular and periarticular bone. Imaging technologies can be very useful, but should be chosen carefully, based on the study circumstances and the information that is desired.

The spatial alignment of cartilage degeneration and effects on subchondral bone, while not unexpected, were well-defined in the wolf shoulders, and provide reference for investigators studying features of dry articular and periarticular bone.

The startling longevity noted in sanctuary wolves, compared to free-roaming wolves, strongly indicates that stochastic events shorten genomically possible life span, rather than sheltering "inducing" increased longevity. An important corollary observation is that gray wolves appear to have a subspecies-related capacity for longevity that far exceeds that of the largest domestic dogs. Further research examining this observation would be instructive, and especially informing to the idea of programmed aging.

Thus, the question of comparative age-based relationships to the observed pathologies might be phrased: Is there a biologically programmed age basis for degenerative joint pathology, and could naturally-occurring joint disease in the dog and wolf serve as a model for programmed aging (Lawler 2011)?

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