

# ***SOFT TISSUE INJURIES OF THE SHOULDER IN THE CANINE ATHLETE***

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Dog owners can participate in a wide variety of dog sports. Agility, Hunting, Track Racing, Lure Racing, Obedience, Rally-O, Flyball, Scent Hurdle, Earth Dog, Weight pulling, Field Trialing, Sled Dog Racing, Skijoring, Freestyle, Carting, Tracking, Frisbee Dog, and Herding are some common dog sports. As dog sports become more popular, owners of these athletic dogs look to animal health care practitioners to answer their sport-specific questions, assess their sport-specific performance issues or non-specific lamenesses, and provide effective treatments applicable to their sporting animal. It is imperative that the veterinary community be better prepared to meet the needs of this unique population.

## **IDENTIFICATION OF SOFT TISSUE INJURIES OF THE SHOULDER**

Soft tissue injuries are often an under-diagnosed source of canine lameness.(Breur & Bevins 1997; Fitch et al 1997; Steiss 2002) Sporting and working dogs may be particularly at risk of suffering acute traumatic muscle strains, ligamentous sprains or chronic overuse degenerative tendinosis lesions resultant from poor healing of repetitive strain injuries. Less conditioned animals may also be at risk when performing infrequent burst activities or endurance tasks, much like the phenomenon known as weekend-warrior-syndrome in humans. Physical therapy skills and knowledge lend the ability to systematically assess, diagnose and conservatively treat soft tissue injuries in the canine patient.

### **Background**

Muscle strains may be caused by poor flexibility, inadequate warm-up, fatigue, sudden forceful contraction or forced extension/flexion, strength imbalances, intense interval training, insufficient breaks and overtraining.(Steiss 2002) The potential for certain muscles to be strained or torn is greater for some muscles than others. Multi-joint muscles are those that cross two or more joints and are at greatest risk for strain because they can be stretched by the movement at more than one joint.(Steiss 2002; Fitch et al 1997; Nielsen & Pluhar 2005) A strain may also occur when high forces are put through tendons and muscles, as occurs during eccentric muscle contractions (where a muscle is contracted during a stretch), when forces are applied quickly and obliquely, or during an explosive burst of movement.( Fitch et al 1997; Nielsen & Pluhar 2005; Sharma & Maffulli 2005; Steiss 2002) Muscle strains most often affect the muscle origin or insertion, typically at the musculotendinous and teno-osseous junctions but can occur within the muscle belly as well.(Steiss 2002; Maganaris et al 2004; Nielsen & Pluhar 2005)

Tendon injuries may be secondary to acute trauma or repetitive loading. (Wilson & Best 2005) The designation of tendon pain as “tendonitis” is often a misnomer as it implies an inflammatory process.(Wilson & Best 2005) Tendinopathy is a better generic descriptor that can

be used to include all pathologies that arise in and around tendons (i.e. tendonitis, tendinosis, or paratenonitis). (Khan et al 1999)

There is a lack of good quality histological data from symptomatic tendon disorders of short duration to unequivocally state that tendon lesions are actually inflammatory in nature. (Rees et al 2006) Marr et al (1993) described an inflammatory reaction in superficial digital flexor tendon injuries in horses, but only within the first 2 weeks. Other animal models suggest that an inflammatory reaction is present in acute situations but that a degenerative process soon supersedes this.(Rees et al 2006) Classic inflammatory changes are not frequently seen in chronic athletic tendon conditions, and it has been suggested that the time at which the tendon becomes symptomatic for pain does not coincide with onset of pathology.(Maganaris et al 2004; Rees et al 2006; Wilson & Best 2005) On a practical note, in clinical practice most tendinopathies are chronic (tendinosis lesions) by the time the patient (or animal owner) seeks medical attention.(Khan et al 1999; Wilson & Best 2005) So perhaps, the clinician should place only minimal, if any, focus on inflammation for tendon pain conditions.

Paratenonitis occurs when a tendon rubs over a bony protuberance and is alternately known as peritendinitis, tenosynovitis and tenovaginitis.(Khan et al 1999) It is clinically characterized by acute edema and hyperaemia of the paratenon with infiltration of inflammatory cells and within hours or days, fibrinous exudates fills the tendon sheath.(Khan et al 1999) Despite these results, pathologists and scientists in this field argue that inflammation of the paratenon is a rare occurrence.(Khan et al 1999)

Tendinosis describes intratendinous degeneration without clinical or histological signs of an inflammatory response.(Khan et al 1999) This form of tendinopathy is typically considered an overuse injury that involves excessive loading of the tendons, frequent cumulative micro trauma and subsequent mechanical breakdown of the loaded tendon.(Khan et al 1999; Rees et al 2006; Sharma & Maffulli 2005; Wang et al 2006; Wilson & Best 2005) In order to mediate the repair process, local tenocytes must maintain a fine balance between extracellular matrix network production and degradation, an unless fatigue damage is actively repaired, tendons will weaken and eventually rupture.(Sharma & Maffulli 2005) In humans, tendinosis is a common problem that that is characterized by persistent, localized, activity related pain and swelling associated with common calcaneal (Achilles), patellar, and supraspinatus tendons.(Fransson et al 2005) The histological appearance of tendinosis is that of collagen disorientation, disorganization, and fibre separation with an increase in mucoid ground substance, increased prominence of cells and vascular spaces with or without neovascularisation and focal necrosis or calcification.(Clancy 1990; Sharma & Maffulli 2005) Additionally, affected tendons are characterized by fibrocartilaginous metaplasia of tenocytes and hypercellularity.(Fransson et al 2005) On visual inspections of affected portions of a tendon, they are lacking their normal glistening-white appearance and have been reported to have a gray-brown or pink-yellow appearance.(Fransson et al 2005; Sharma & Maffulli 2005)

Barring a direct trauma muscle strain, it is more likely that a soft tissue injury is a tendinosis lesion, and the practitioner should be aware of the pathology.

## **SPECIFIC CANINE SHOULDER INJURIES**

Problems specific to the canine shoulder joint include tendinopathies of the supraspinatus, and subscapularis muscles, bicipital tenosynovitis or bursitis, medial shoulder ligamentous instability, and strains of the teres major muscle.

Supraspinatus calcification as well as tendinosis has been reported in veterinary literature.(Fransson et al 2005; Long & Nyland; Muir et al 1996; Laitinen & Flo 2000; Flo & Middleton 1990; Soslowky et al 2000; Bardet 1998) Calcification has been reported to be a cause of unilateral forelimb lameness in dogs, with an incidence of 2.8 – 7% in all clinically lame dogs.(Long & Nyland 1999) The indicated treatment is surgical excision.(Muir et al 1996; Laitinen & Flo 2000) However, mineralization of the supraspinatus tendon is a common finding in asymptomatic limbs, and while improvement in symptoms is reported following surgery, long term follow up reveals that supraspinatus tendon mineralization can recur within a 5 year post-operative period.(Laitinen & Flo 2000; Flo & Middleton 1990) Tendonosis lesions of the supraspinatus tendon have been described in dogs and may precede or be associated with calcium deposits in the tendon.(Fransson et al 2005; Long & Nyland 1999) Additionally, it has been shown that overuse injuries of the supraspinatus can be induced in an animal model with a simulation of repetitive eccentric muscle activity created by running rats on a decline treadmill.(Soslowky et al 2000) Surgical resections have been shown to yield ‘good’ results in clinically lame dogs (LaFuente et al 2009), but why not try conservative rehab management first, especially with a canine athlete?

The biceps tendon is a major stabilizer of the canine shoulder joint impacting cranial, medial and lateral translations of the humerus relative to the glenoid cavity.(Sidaway et al 2004) Bicipital tenosynovitis is a commonly reported pathology of the biceps tendon. (Gilley et al 2002; Long & Nyland 1999; Kramer et al 2001; Bruce et al 2000; Davidson et al 2000) Biceps tenosynovitis has been described as an inflammation of the biceps tendon or origin, its tendon sheath and the bicipital bursa within the intertubercular groove in the proximal humerus.(Davidson et al 2000) While inflammatory pathology of this tendon does exist, in some dogs, this disease may be the result of a degenerative process rather than an inflammatory process.(Gilley et al 2002) Other disease processes localized to the biceps tendon include but are not limited to calcification, osseous metaplasia, bone chip in the tendon sheath, and osteophyte formation in the intertubercular groove or supraglenoid tubercle.(Davidson et al 2000; Gilley et al 2002; Long & Nyland 1999; Kramer et al 2001) Clinical evaluation of the biceps tendon includes the biceps tendon test (positioning the forelimb into shoulder joint flexion with the elbow extended), pain on focal digital pressure applied directly to the biceps origin and/or intertubercular groove and the biceps retraction test.(Bruce et al 2000; Davidson et al 2000; Gilley et al 2002) As well a history of chronic and/or progressive weight bearing lameness that is worse after exercise and affecting active middle-aged or older medium to large breed dogs is common.(Bruce et al 2000; Gilley et al 2002; Davidson et al 2000) However, Bardet (1998) proposed that the biceps tendon test appears to be more of an indicator of generalized shoulder joint pain than a pathognomonic sign of biceps tendon disorders. Common veterinary treatments for bicipital tendonopathies include oral administration of non-steroidal anti-inflammatory (NSAID) medications, local injection of corticosteroids or tenotomy or tenodesis of the tendon. Yet animal model studies have revealed that NSAID administration or corticosteroid injections inhibit or delay collagen repair of muscles / tendons following NSAID administration and if inflammation is not the source of the pathology, then their use would be unwarranted. (Almekinders & Gilbert 1986;

Obremsky et al 1994; Fransson et al 2005) As well, with the finding that the biceps tendon has a significant role in passive stability of the shoulder joint, there is a question as to whether any long-term adverse effects such as osteoarthritis in the shoulder joint may be caused by a mild instability after tenotomy or tenodesis.(Sidaway et al 2004) A recent study showed excellent recovery following biceps tenotomy for return of limb function over a long term follow-up (mean 26 months), without the progression of osteoarthritis. (Bergenhuyzen et al 2010) Human studies have not found the same however.(Szabo et al 2008) Thus, it would be interesting to compare and contrast conservative rehab management or early surgical interventions for canine athletes with a biceps tendon lesion.

Shoulder instability appears to be a common cause of lameness in medium and large-breed hyperactive dogs with a chronic permanent or intermittent foreleg lameness.(Bardet 1998) Other clinical signs of shoulder subluxation include atrophy of the shoulder muscles, non-weight-bearing lameness, spontaneous cries, signs and symptoms of disc disease or a 'wobblers walk' presentation, as well, abnormal craniocaudal or mediolateral translations (drawer tests) are reported to be consistent indicators of shoulder joint instability in dogs subsequently diagnosed by arthroscopic evaluation.(Bardet 1998) This same author suggested grading of the direction and degree of the drawer translation: Grade 1 – when the translocation of the head of the humerus on the glenohumeral joint is not appreciated; Grade 2 (Mild) – when the translocation is appreciated but is not enough to allow the head of the humerus to rise up on the rim of the glenoid cavity; Grade 3 (Moderate) – when the head of the humerus is appreciated but is not enough to allow the head of the humerus to rise up on the rim of the glenoid cavity; Grade 4 (Severe) – when the head of the humerus courses over the rim of the glenoid cavity and is dislocated. Additional clinical findings may include pain with the biceps tendon test and pain on shoulder joint hyperextension.(Bardet 1998) Cook et al (2005a) described clinical diagnostic testing utilizing measurement of shoulder abduction angles. In dogs diagnosed with instability, the mean abduction angles ( $53.7 \pm 4.7^\circ$  measured goniometrically) were significantly larger than for all unaffected shoulders ( $32.6 \pm 2.0^\circ$  measured goniometrically). They proposed that the difference between angles is substantial enough to suggest that a visual observation of this asymmetry may be all that is required to make a preoperative diagnosis of medial shoulder joint instability in dogs. Medial shoulder instability is attributable to pathology of the medial aspect of the joint capsule, the subscapularis tendon and or the medial glenohumeral ligaments and may precede glenoid cartilage or humeral head cartilage wear or defects and eventual degenerative joint disease.(Bardet 1998; Cook et al 2005a) A demonstrated treatment for this condition is radiofrequency-induced thermal 'shrinkage' of the lax tissue to induce tightening of the joint capsule followed by post operative care involving a Velpeau sling and physiotherapy treatments.(Cook et al 2005b) Surgery can result in good functional recovery and can re-establish normal abduction angles with medial shoulder instability. (Cook et al 2005; Pettitt et al 2007; Pucheu et al 2008) However, no studies have been published pertaining to return to sport following rehab only or surgical interventions. Presumably, the impact of the instability may be the deciding factor on how to proceed regarding treatment.

The teres major muscle originates from the caudal angle and caudal edge of the scapula and inserts into the eminence on the proximal 1/3 of the medial surface of the humerus and shares a common tendon of insertion with the latissimus dorsi.(Evans 1993) The teres major muscle is reported to flex the shoulder joint, however in analyzing the origin and insertion of teres major in the canine, this muscle can not only flex the shoulder but should also adduct and internally rotate

the shoulder when the front limb is in an outstretched position.(Edge-Hughes 2004b) The proposed mechanism of injury would be an exaggerated extension, abduction and external rotation which could occur when a dog is running at high speeds and makes a sudden turn.(Edge-Hughes 2004b) Clinical presentation is of acute or chronic forelimb lameness that improves with rest but returns when allowed to resume normal activities.(Edge-Hughes 2004a) Physical examination reveals mild discomfort with full shoulder extension, inclusive of scapulothoracic movement, an increase in discomfort with the addition of abduction and external rotation, and moderate to severe tenderness (patient yelp or muscle twitching) on palpation of the teres major muscle or its tendon of insertion located in the ‘roof’ of the caudal aspect of the axilla.(Edge-Hughes 2004a)

## **RETURN TO SPORT REHABILITATION**

### **Treatment of a tendinopathy lesion**

What may look like an Acute tendinopathy may be a well-advanced failure of a chronic healing response in which there is neither histologic nor biochemical evidence of inflammation.(Magra & Maffulli 2006) When an athlete first notices tendon pain, tissue damage may already be advanced.(Kahn et al 1999) Chronic ligamentous (or musculotendinous) injuries will have random collagen orientation, wound contracture, restrictive adhesions / scars and may be degenerative.(Kahn et al 1999; Maganaris 2004; Sharma & Maffulli 2005) Treatments for tendinosis would utilize alternate treatment strategies to that of tendonitis / acute tendon lesions.

Prolonged immobilization may have detrimental effects such as a tendon atrophy, decrease in tensile strength and strain at failure, decreased water and proteoglycan content of tendons and an increase in number of reducible collagen cross links.(Sharma & Maffulli et al 2005) Therefore a proper balance between guided activity and relative rest is imperative.

Stretching has been shown to increase collagen synthesis and improve collagen fibre alignment, resulting in higher tensile strength.(Sharma & Maffulli 2005) A recent study compared eccentric exercise training versus a stretching regime for Achilles tendon pain and found that both groups exhibited marked improvement in symptoms but no significant difference between the groups.(Norregaard et al 2006)

Hands on manual therapy treatments, such as deep transverse frictions have been proposed for soft tissue lesions.(Cyriax 1982) Studies have been unable to show a consistent benefit over control groups for improvement of pain.(Rees et al 2005) Massage, which is thought to increase blood supply and therefore promote healing, is another form of manual therapy that has not been adequately studied in these cases. Therefore, neither of these treatment methods have proven efficacy as therapies of choice for tendinosis.

Cryotherapy is believed to decrease blood flow and tendon metabolic rate and hence reduce swelling and inflammation.(Rees et al 2006) While tendinosis lesions are not inflamed, this therapy could help if paratenonitis is present or for its analgesic effects.(Kahn 1999; Rees et al 2006)

The use of modalities such as ultrasound, laser and pulsed electromagnetic field may be beneficial in the treatment of muscle and tendon lesions. Both laser and ultrasound have been studied and proven to have a beneficial effect for tendon healing.(Demir et al 2004) One recent

study found that 5.4 Joules per point of a 904 nm wavelength infrared, 20 mW laser was effective in increasing pressure pain threshold and reducing prostaglandin E2 concentrations in Achilles tendon lesions.(Bjordal et al 2006) Pulsed Magnetic Field Therapy (at 17 Hz) has been shown to improve collagen fibre alignment and increase the force to breakage, yet other studies failed to find improvement in adhesion formation with use in lacerated tendons.(Sharma & Maffulli 2005)

Perhaps the strongest evidence for treatment of tendinosis lesions lies in eccentric strength training protocols. Mechanical loading has been identified as an accelerant of tenocyte metabolism.(Kahn et al 1999) Eccentric muscular training is described as an event where the muscle contraction is purposely less than the opposing outside force, hence allowing for a slow controlled lengthening of the muscle or musculotendinous unit.(Magee 1987) Eccentric training consisting of twice daily exercises of several repetitions for 12-weeks was able to produce a decrease in tendon thickening, resolution of neovascularization, and an increase in patient satisfaction in the Achilles tendons as well as showing similar improvement with patellar tendinopathy and supraspinatus lesions.(Alfredson et al 1998; Ohberg et al 2004; Rees et al 2006) Gravare et al (2001) utilized a 12 week program aimed at increasing local blood circulation, improving ROM, plus balancing and gait exercises and specific eccentric exercises, which graduated in intensity to eventually incorporate quick rebounding exercises to address an Achilles tendinopathy. This program was shown to provide subjective improvement in symptoms over a training program that utilized concentric exercise training. An additional study for patellar tendinopathy utilized eccentric squats on a decline board, doing 3 sets of 15 reps, twice a day for 12 weeks, while adding 5kg increments of weighting to progress the exercise.(Bahr et al 2006) This study did allow for a resumption of cycling, jogging on a flat surface, or exercise in water at the 8 week mark if pain was not involved in the exercise. Yet another study of Achilles tendinosis in soccer players, utilized 12-weeks of heavy resistance eccentric training while allowing the participants to continue with their regular soccer training so long as the pain did not increase in doing so.(Langberg et al 2006) This study did find an increase in collagen synthesis rate with this protocol despite the lack of relative rest prescribed in many other protocols. A few of the studies cited above mentioned the importance in acceptance of pain during eccentric loading in order to obtain excellent results with this therapy.(Bahr et al 2006; Langberg et al 2006; Norregaard et al 2006) With this information, all muscular and tendinous lesions should undergo eccentric training at some point in time during their rehabilitation.

Extracorporeal Shockwave Therapy (ESWT) has been shown to create neovascularization and nitric oxide synthase and may promote healing of experimentally induced Achilles tendinopathy lesions in rats.(Sharma & Maffulli 2005) ESWT may be useful for calcifying tendinopathies of the shoulder and possibly heel pain but there is little evidence of benefit for others.(Rees et al 2006) Users of ESWT should be warned that there may be the possibility of dose dependent tendon damage including fibrinoid necrosis, fibrosis and inflammation.(Rees et al 2006)

Prolotherapy can be an effective treatment for tendinosis. It has been shown to be beneficial in Achilles tendinosis lesions, its use when combined with eccentric exercise provided more rapid improvements.(Yelland et al 2009) Stem cell therapy may also be an effective option. This technique has been used in both small and large animal models; for example, mesenchymal stem cells do promote healing in a rabbit Achilles tendon, and using autologous bone marrow-derived

stromal cells, researchers have developed a treatment for the management of injuries to the digital flexor tendons in horses. (Rees JD et al 2009)

Treatment of acute muscle injuries would simply follow traditional therapeutic principles. Incorporation of rest, ice, compression and 'protection' (since elevation is not always applicable) is utilized in the very acute stages. Following is the regeneration phase, which requires therapies to address tissue regeneration and promote newly constructed fibres to align properly, increasing circulation and restore coordination and body awareness. Lastly in the remodelling phase, strengthening, muscle extensibility, joint mobility, advanced neuromuscular retraining and return to sport conditioning are needed. Details on all of these components are beyond the intention of this paper.

## **RETURN TO SPORT FOLLOWING INJURY OR SURGERY**

### **Neuromuscular retraining**

Neuromuscular retraining is a term coined to describe the rehabilitative retraining of coordination, skill training, and higher levels of strengthening. It is imperative to the end functioning and return to sport for canine athletes to be instructed in and led through this kind of reconditioning following an injury or surgery. Considerations for this 'end-stage / advanced level' or rehabilitation may include the following (Edge-Hughes c): Exercises up and down hills, trotting exercises, acceleration / deceleration activities, cutting or rapid turning exercises, jumping exercises, concentric strengthening, eccentric strengthening, plyometrics, endurance, static balancing, and dynamic balancing. Reintroducing and retraining sport-specific movements is imperative to successful reintegration to an athletic career.

## **INJURY PREVENTION STRATEGIES**

### **Stretching**

Stretching is a popular prescription among health care professionals, athletic trainers and in fitness/coaching personnel, all of whom have an interest in improving flexibility in both healthy and injured clientele. Stretching has been touted to enhance athletic performance, prevent musculotendinous strain injuries and reduce delayed onset muscle soreness. However confusion and controversy exists over when stretching is most effective, and some claims and common uses of stretching are not supported by research. (Decoster et al 2005)

Stretching has been shown to be effective in increasing joint mobility about the knee, hip, trunk, shoulder and ankle joints including muscle length and flexibility .(Davis et al 2005, Decoster et al 2005; Knudson 1999; Magnusson et al 1998; Power et al 2004; Thacker et al 2004) Studies have shown that regular stretching can improve eccentric and concentric force production, velocity of contractions, maximal volitional contractions, counter-movement jump height, 50 yard dash and athletic performance. (Hunter et al 2002; Shrier 2004) One study found that regular stretching was able to induce hypertrophy in immobilized muscles and another speculated that this effect may actually improve performance in the long term.(Coutinho et al 2004; Shrier 2004)

Results varied for the optimum time required to obtain the most favourable muscle lengthening/joint range of motion (ROM). Studies found that passive stretches of 15 to 30 seconds were more effective than stretches of shorter duration and just as effective as stretches of

longer durations. (Decoster et al 2005; Thacker et al 2004) Reports of other studies found that the overall time of stretching was most important and found that 6 repetitions of 10 seconds each was just as effective as 2 repetitions of 30 seconds. Three sets of 15 second stretches were effective as well. The greatest gains in flexibility were made if stretching occurred on a regular basis over time. (Decoster et al 2005) One study reported that a static stretch of 1 repetition for 30 seconds, 3 days a week for 4 weeks, significantly increased hamstring length/flexibility. (Davis et al 2005) Consensus was that passive static stretching was more effective than proprioceptive neuromuscular facilitation techniques, active assisted or dynamic stretching protocols in improving muscle length.(DeCoster et al 2005; Shrier 2004)

It is in the use of stretching immediately before exercise or testing where the adverse effects of this technique are seen. It has been shown in human studies, that acute bouts of stretching does not improve maximal volitional force output, jump height, running speed, static balance, reaction time, or movement time. (Behm et al 2004; Knudson 1999; Power et al 2004; Shrier 2004; Thacker et al 2004) In this format, stretching results in a decrease in isokinetic performance, velocity of contraction, muscle force produced with contractions, musculotendinous unit compliance and a reduced ability to store elastic energy in the eccentric phase. (Fletcher et al 2004; Shrier 2004) These negative effects have been reported to last up to 1 hour following stretching. (Thacker et al 2004) A reduction in running economy has been reported as a result of stretching (Thacker et al 2004), however Nelson et al (2001) dispelled that assumption in their study by showing that VO<sub>2</sub>peak was not affected by a chronic stretching program.

Several studies and reviews have looked into the use of pre-event stretching to reduce the risk of injury. (Hart 2005; Herbert & Gabriel 2002; Pope et al 2000; Thacker et al 2004; Witvrouw et al 2004) Most have shown or reported that pre-event stretching does not reduce the risk of injury. Witvrouw et al (2004) suggested in their review that pre-event stretching was useful in preventing injuries in sports with high stretch shortening cycle movements (i.e. football or soccer), however stretching was always incorporated with an active warm up, which may have contributed to the reduction of injury. As well, studies of athletes that suffered muscle lesions were found to have less muscular flexibility than those without injury. One should not make the assumption that pre-event stretching would have benefited these athletes more or less than a regular stretching program. It should also be reported as a fallacy, that stretching before or after exercise does not confer protection from muscle soreness. (Herbert & Gabriel 2002) Additionally, it has been suggested that in sports that do not require burst or flexibility (i.e. jogging or cycling) that a certain amount of stiffness in the musculo-tendinous structures would in fact be beneficial. (Witvrouw et al 2004)

In any sprint activity, muscle flexibility should be adequate enough to allow the full range of joint motions required for the activity (deVries 1986), but not overt flexibility which would impede the immediate transference of musculotendinous forces to the bones and potentially reduce the speed of movement.(Witvrouw et al 2004) However, a recent study of racing greyhounds found that dogs that had received race training had greater flexibility, possibly due to training having an active stretching role on muscles, tendons and other structures limiting the hip.(Nicholson et al 2007) Presumably, stretching to gain flexibility would not be necessary for injury prevention in endurance athletes such as sled dogs, as the gait and speed at which an endurance race is run only utilizes the mid ranges of the extremity joints.([Witvrouw et al 2004)

## **Warming-up**

Warming-up the animal prior to racing or exercise is of great importance to achieve superior performance and prevent injuries.(Tyler et al 1996; Steiss 2003) In horses, a warm-up of 5 to 10 minutes is more beneficial for improving oxygen kinetics than a shorter warm-up period.(Tyler et al 1996) There are conflicting citations however, as to whether warming-up has any effect on performance in sprinting activities in people. (deVries 1986) A recent human study however revealed that both active warm-up (10 minutes at VO<sub>2</sub>max) or passive warm up (hot water submersion) were superior to no warm up for speed.(Brown et al 2008) Some literature also cites that endurance athletes perform better with five minutes of vigorous high-intensity warm-ups that include some sprinting.(deVries 1986) Essentially, heating of muscle tissues can improve musculotendinous extensibility and may thereby reduce its susceptibility to strain injury.(Stickler et al 1990)

## **CONCLUSION**

It is important for veterinarians and other animal health care practitioners to be aware of canine sporting injuries. Knowledge of stretching, warming up, and soft tissue injury identification and treatment options for the canine shoulder will benefit the welfare of canine athletes, as veterinarians become more able to offer suggestions for injury prevention and management to this unique clientele.

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