

# **Osteochondrosis in Beef Sires in Sweden**

*Y. Persson and S. Ekman*

*Swedish University of Agricultural Sciences (SLU), Uppsala, Sweden*

## **Abstract**

Swedish insurance companies report that a great number of their insured beef sires are culled because of lameness. To our knowledge, little is known about hind limb problems in beef cattle, but there are indications that osteochondrosis is a common cause of hind limb lameness in Swedish beef bulls used for natural service. The aim of the present study was to examine hind limbs of Swedish beef sires post mortem, regarding the presence of osteochondrosis. Right and left hind limb bones from 42 beef sires were examined post mortem to identify lesions in the stifle and tarsal joints. The bulls were slaughtered during or after the breeding season due to leg weakness. The bulls were of five different breeds, Charolais (n=16), Simmental (n=12), Aberdeen Angus (n=9), Hereford (n=4) and Limousine (n=1) and the mean age was 2.5 years (range 1-7 years). Thirty-eight of the bulls (90 %) had lesions in at least one joint. Thirty-four bulls (81 %) had lesions in the stifle and 14 bulls (33 %) had lesions in the tarsus. In a majority of the bulls (67 %), the lesions were bilateral. The most common location of the joint lesions was the lateral ridge of the femur trochlea. Three bulls had no joint lesions. In conclusion, the present results indicate that lesions compatible with osteochondrosis are common post mortem findings in beef sires with lameness.

## **Introduction**

The sustainability of the beef bull is essential for successful breeding and is also important from a welfare point of view. A Swedish insurance company (AGRIA) reports that a great number of the insured beef sires are culled because of lameness (Ohlén, personal communication 2004). In a group of healthy, performance-tested yearling beef bulls, 97.8% had joint lesions, at slaughter, compatible with osteochondrosis (OC) (Dutra et al. 1999).

Osteochondrosis of the articular-epiphyseal-cartilage complex (AECC) is characterized by disturbed endochondral ossification of the epiphyseal growth cartilage. The aetiology is not fully understood but it is suggested that focal failure of blood supply in the growth cartilage causes local ischemia, which in turn leads to focal necrosis of the growth cartilage (Ytrehus 2004). The disorder has been described in pigs (Reiland, 1978), horses (Rejnö & Strömberg, 1978), dogs (Olsson, 1987), man (Bohndorf 1998), poultry (Poulous et al., 1978) and cattle (Trostle 1998). The aetiology of OC is considered to be multifactorial. Heredity, gender, growth, weight, trauma, nutritional imbalance and anatomical conformation have been proposed as aetiological factors (for review see Ekman & Carlson, 1990). The focal necrosis of growth cartilage with impaired ossification is sometimes followed by osteochondrosis dissecans (OCD) (Olsson, 1978). The joint shape, growth rate and body weight have been suggested as factors influencing the local conditions of the tissue (Ytrehus 2004). OCD will cause a synovitis and secondary osteoarthritis (OA) (Olsson, 1978, Bailey 1985), with lameness as the main clinical symptom. Bilateral lesions are common in bulls (Trostle 1998) and the lameness can be difficult to observe. Hence, it can be problematic to diagnose joint lesions in beef bulls.

The aim of the present study was to examine hind limbs of Swedish purebred beef sires post mortem, in order to find possible causes to hind limb lameness.

## **Material and methods**

Stifle and tarsal joints from right and left hind limbs of 42 beef sires were examined post mortem. In some of the bulls, hip joints and sacrum were also examined. The bulls were slaughtered during or after the breeding season due to leg weakness. The bulls were of five different breeds, Charolais (n=16), Simmental (n=12), Aberdeen Angus (n=9), Hereford (n=4) and Limousine (n=1) and the mean age was 2.5 years (range 1-7 years).

The joints were disarticulated and examined macroscopically. The articular cartilage, synovial membrane/capsule, ligaments, menisci and subchondral bone were inspected for lesions such as; cartilage fraying, wear lines, erosion, ulceration, osteochondritis dissecans (OCD), cartilage retention osteophytes and villiformation. Osteochondrosis was diagnosed when cartilage retention was found in a predilection site, and OCD when a cartilage rupture or loose osteochondral body could be seen. The lesions were recorded as unilateral or bilateral and graded as normal, mild, moderate, severe or deformed osteoarthritis (OA). Mild OA in the stifle joint were characterized by superficial cartilage fraying of less than 30% of the articular cartilages with single erosion <1cm and superficial wear lines. Mild OA in the tarsal joints were characterized by single erosion or single OCD. Larger areas of cartilage fraying (>30% of the articular cartilage) with multiple erosions, single ulceration, single erosion >1cm and villiformation of the synovial membrane in the stifle joint and multiple OCD and/or erosions in the tarsal joint were considered as moderate OA. Severe OA in the stifle joint were characterized by single or multiple OCD, multiple ulcerations and single ulcer >1cm. Severe OA in the tarsal joint were characterized by ulcer with denuded bone >0.5cm. Deformed OA in both joints, were characterized by severe OA with osteophytes.

## **Results**

Thirty-eight of the 42 bulls (90 %) had lesions in at least one joint. Twenty-four bulls had lesions in the stifle, 2 bulls had lesions in the tarsus and 12 bulls had lesions in both the stifle and the tarsus. Four bulls also had lesions in the hip and 3 bulls had fractures in the sacrum. In a majority of the bulls (67%), the lesions were bilateral. The most common location of the joint lesions was the lateral ridge of the femur trochlea (19/38), followed by the intercondylar eminentia of tibia (10/38). Almost all OA lesions (33/38) were characterized as lesions secondary to osteochondrosis or OCD. Four bulls had no lesions at all. Six bulls had mild lesions, 10 bulls had moderate lesions, 14 bulls had severe lesions and 8 bulls had deformed OA.

## **Discussion**

The present results indicate that lesions compatible with osteoarthritis (OA) are common post mortem findings in beef sires, regardless of clinical history. The results also indicate that lameness in beef bulls often appears to be caused by OA. Most of the OA lesions in this study seem to be secondary to osteochondritis dissecans, a phenomenon that is well recognized (Bailey 1985).

A majority of the bulls in this study had bilateral, symmetrical lesions. This might contribute to the difficulties to diagnose joint lesions in the field. The most common location of the lesion was the lateral trochlear ridge of the femur trochlea, similar to that have been reported in previous studies (Reiland 1978, Weisbrode SE 1982, Trostle 1997, Tryon 1999). In conclusion, the present results indicate that lesions compatible with osteochondrosis are common post mortem findings in beef sires with lameness.

## **Acknowledgements**

Appreciation is expressed to SLF for funding this study, to Thomas Ohlén, Agria insurances, and Urban Wahlström and others at Swedish Meats for supplying me with bulls, to Hasse, Lasse and Johan at SVA/pathology for helping me with my “bones”.

## **References**

- Bailey JV. 1985. Bovine arthritides. Classification, diagnosis, prognosis, and treatment.
- Bane A., Hansen HJ: 1962. Spinal changes in the bull and their significance in serving ability. *Cornell Vet.* 3:364-384.
- Bellenger CR. 1971. Bull wastage in beef cattle. *Austr. Vet. Jour.* 47:83-90
- Bohndorf K. 1998. Osteochondritis (osteochondrosis) dissecans: a review and new MRI classification. *Eur Radiol.* 8:103-12.
- Dutra, F., Carlsten, J and Ekman, S. 1999. Hind limb skeletal lesions in 12-month-old bulls of beef breeds. *J Vet Med A.* 46:489-506.
- Ekman S., Carlsson CS. 1998. The pathophysiology of osteochondrosis. *Vet Clin North Am Small Anim Pract.* 28: 17-32.
- Hill BD, Sutton RH, Thompson H. 1998. Investigation of osteochondrosis in grazing cattle. *Aust Vet J:* 3: 171-175
- Olsson SE. 1978. Osteochondrosis in domestic animals I. *Acta Radiol. Suppl.* 358: 9-12.
- Olsson SE. 1987. General and local aetiologic factors in canine osteochondrosis. *The Vet. Quart.* 3:268-278.
- Poulos PW, Reiland S, Elwinger K, Olsson SE. 1978. Skeletal lesions in the broiler, with special reference to dyschondroplasia (osteochondrosis). *Acta Radiol. Suppl.* 358: 229-275..
- Reiland, S. 1978. The effect of decreased growth rate on frequency and severity of osteochondrosis in pigs. *Acta Radiol Suppl.* 358:107-22.
- Reiland S, Stromberg B, Olsson SE, Dreimanis I, Olsson IG. 1978. Osteochondrosis in growing bulls. Pathology, frequency and severity on different feedings. *Acta Radiol Suppl.* 358:179-96.
- Rejnö S, Strömberg B. Osteochondrosis in the horse. 1978. *Acta Radiol Suppl.* 358:153-78.
- Trostle SS., Nicoll RG., Forrest, L.J., Markel, M. 1997. Clinical and radiographic findings, treatment, and outcome in cattle with osteochondrosis: 29 cases (1986-1996). *JAVMA* 211: 1566-70.
- Trostle, S.S., Nicoll, R.G., Forrest, L.J., Markel, M., Nordlund, K. 1998. Bovine osteochondrosis. *The Compendium.* 20; 7, 856-863.

Tryon KA, Farrow CS. 1999. Osteochondrosis in cattle. *Vet Clin North Am Food Anim Pract.* 15: 265-274

Weisbrode SE, Monke DR, Dodaro ST, Hull BL. 1982. Osteochondrosis, degenerative joint disease, and vertebral osteophytes in middle aged bulls. *J Am Vet Med Assoc.* 7: 700-5

Ytrehus B. 2004. Osteochondrosis; A morphological study of aetiology and pathogenesis. Thesis. The Norwegian School of Veterinary Science