# BONE SPAVIN IN ICELANDIC HORSES

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## Introduction

Osteoarthrosis (OA) or degenerative joint disease is a chronic disorder of synovial joints characterized by progressive deterioration of articular cartilage, accompanied by changes in the bone and soft tissue of the joint, including subchondral bone sclerosis and marginal osteophyte formation (McIlwraith and Vachon 1988).

Bone spavin is an OA of the distal tarsal joints, described as commonly occurring in all horse breeds including ponies (Barneveld 1983, Wyn-Jones 1988). Studies of German sport horses (Winter *et al.* 1996), young trotters (Hartung *et al.* 1983), young horses of different breeds (Laverty *et al.* 1991) and Dutch Warmblood foals (Barneveld and van Weeren 1999), have revealed high prevalences of radiographic and histological abnormalities compatible with bone spavin.

Radiography is considered essential for the diagnosis of bone spavin (Butler *et al.* 2000 b). The radiographic signs of OA in the distal tarsal joints (RS) include: periarticular osteophytes, subchondral bone lysis or rarifaction, and narrowing and collapse of joint spaces or ankylosis (Morgan 1972, Shelley and Dyson 1984, Park *et al.* 1996, Butler *et al.* 2000 b).

Clinical signs are most commonly found in mature horses in hard work (Gabel 1980) and include hindlimb lameness, positive flexion tests, reduced arc of the foot flight, reduced flexion of the hock, and wearing of the toe (Sullins 2002). The clinical manifestations are however variable and in many horses the relationship betwen pain associated with the distal joints of the hock and the radiographic abnormalities is poor (Butler *et al.* 2000b, Sullins 2002).

The aetiology of the disease is considered to be multifactorial (Wyn-Jones 1988). Inherited, poor tarsal conformation e.g. sickle hocks and cow hocks (Rooney 1969, Morgan 1972, Gabel 1980, Barneveld 1983, Wyn-Jones 1988), and special types of work (Rooney 1969, Gabel 1980) resulting in asynchronous movements of the tarsal bones (Rooney 1969) are examples of etiological factors which have been suggested. Specific injury (Sullins 2002) and periarticular trauma are also considered (Pool 1996). A genetic predisposition for OA in the distal tarsus was reported in Dutch warmbloods (Barneveld 1983), but the heritability was estimated to be low in German riding horses (Winter *et al.* 1996).

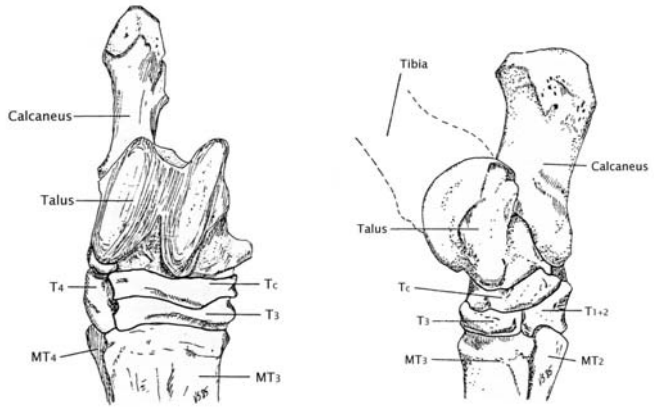




Figure 1. Schematic drawing of the tarsus, a) dorsal view, b) medial view.
Tc: os tarsi centrale, T1+2: Fuced os tarsale primum and os tarsale secundum, T3: os tarsale tertium, T4: os tarsale quartum, MT2-4: os metatarsale 2 – 4.

The tarsus consists of three rows of tarsal bones (Figure 1). Together with the distal tibia and proximal metatarsal bones, they form a composite joint, articulatio tarsi comprising the tarsocrural, the proximal intertarsal (PIT), the distal intertarsal (also named centrodistal interatarsal, CD) and the tarsometatarsal (TMT) joint. The tarsocrural joint is responsible for most of the movement of the tarsus, as the distal tarsal joints have flat surfaces limiting the motion (Nickel *et al.* 1986).

The joints are surrounded by a common joint capsule which encloses separate synovial sacs. The joints are stabilized by collateral ligaments on either side of the tarsus, proximal and distal tarsal ligaments (e.g. the dorsal tarsal ligament), and tarsometatarsal ligaments (Nickel *et al.* 1986). The tendon (the medial terminal branch) of m. tibialis cranialis runs obliquely over the medial aspect of the tarsus, over the cunean bursa, and is sometimes refered to as the "tendon of spavin" (Nickel *et al.* 1986, Kainer 2002).

The distal tarsal joints are low-motion joints, where the area of maximal weight bearing is nearly stationary during locomotion. They are therefore under a greater compression stress compared to the high-motion joints and hence more susceptible to non-physiologic loading and metabolic disturbance (Pool 1996).

**Aims of the investigation**

1. To determine the age of onset and the nature of changes in the cartilage and subchondral bone of the centrodistal joint of young Icelandic horses by high detail radiography and histology.
2. To estimate the prevalence of radiographic signs of OA in the distal tarsal joints (RS) in a population of Icelandic horses being used for riding and describe the radiographic findings.
3. To estimate the prevalence of hind limb lameness after flexion test of the tarsus in the Icelandic riding horse population and to assess the association between RS and lameness after flexion test.
4. To compare the rate of culling between horses with and without RS and lameness after flexion test and thus to determine the prognostic value of these diagnostic methods.
5. To evaluate intrinsic and environmental risk factors associated with RS and lameness after flexion test.
6. To estimate the heritability of RS and lameness after flexion test.

**Material and methods**

*Specimens*

The left CD joints, including the Tc and the T3, from 111 horses in the age range of 6 months to 6 ½ years were collected post-mortem at the slaughterhouse. The bones were sectioned with a bandsaw into 8-mm thick slabs in the frontal plane, dorsal to the mid-line of the central tarsal bone, for high detail radiography. Specimens from horses younger than 5 years (n=82) were then selected for histology. The majority of the horses were not broken to saddle. They were culled because of poor conformation, gaits or pedigree.

The bone sections were radiographed using high detail film and corresponding high detail intensifying screen (mammography system). The CD was evaluated on the high detail radiographs for sclerosis of the subchondral bone, defects of the subchondral bone plate, narrowing of the joint space and periarticular osteophytes.

Subsequently, the bone sections were decalcified, trimmed, embedded in paraffin, cut into approximately 6-µm thick section, coded and stained with hematoxylin & eosin and toluidine blue. The degree of articular cartilage lesions was graded as mild (a focal loss of the extracellular stain and chondrocytes, with formation of chondrocytes in clusters adjacent to the area), moderate (diffuse chondronecrosis containing more cluster formations) or severe

(marked chondronecrosis with loss of cartilage, often together with fibrosis and areas of fusion of the Tc and the T3) in the lateral and medial parts of the CD.

*Horses*

Horses in the age range of 6-12 years and in use for riding were invited to the field survey. Offspring from 17 selected sires were requested. This provided 420 horses. Additionally 194 horses, meeting the same criteria but sired by unselected stallions, were also included. Together, the material consisted of 614 horses, 24 (3.9%) stallions, 403 (65.6%) geldings and 187 (30.5%) mares. The mean age was 7.9 years.

The radiographic examination consisted of latero-5°-proximal-mediodistal (L5Pr - MD), dorso-35°-lateral-plantaromedial oblique (D35L - PlMO) and plantaro-45°-lateral-dorsomedial oblique (Pl45L - DMO) projections of each tarsus. Strict intraarticular diagnostic criteria were used for RS: rarefaction of the subchondral bone, narrowing or collapse of the intertarsal joint spaces. The location of the radiographic findings was identified by the specific joints involved and the extension of the lesions was graded as mild (radiographic signs in one or more of the distal intertarsal joints, in total up to a half joint space), moderate (in total between half and one joint space) or severe (in total more than one joint space). Presence of periarticular osteophytes on the dorsomedial aspect of the distal tarsus was recorded. The radiographic findings of active bone remodelling, defined as irregular and poorly demarcated bone proliferations (Butler *et al.* 2000 a) were noted as absent or present. The radiographs were coded and evaluated by two radiologists together. Equivocal findings were graded as the less severe alternative.

Hind limb lameness was first evaluated while trotting the horse by hand on a firm surface 25 - 30 meters straight away from the examiner and back, after which a flexion test was performed by flexion of each tarsus for one minute and repeating the above motion evaluation. Lameness before and after the flexion test was graded as mild, moderate, severe, very severe or non-weight bearing.

Information on registration number, age, gender and pedigree was obtained from the owners.

In the five year follow-up the owners were interviewed by telephone and asked if the horses were still used for riding. If not, they were asked when and why the horses were sold, selected for breeding or culled.

*Data analysis.*

Bivariate and multivariate logistic regressions were used to examine the effect of age, gender and potential risk factors on RS and lameness, and the association between the findings. Also the effect of age on lesions found by high detail radiography and histology and their association were examined by these methods. Chi Square test and Chi Square for linear trend were used to analyze the effect of age on categorical dependent variables (details of radiographic findings) and their association to lameness in the same limb. The minimum level of significance was chosen as $P<0.05$.

The survival function and the culling rate was estimated by the Kaplan-Meier estimator or the Product limit estimator (Klein and Moeschberger 1997). The association between the hazard function and the explanatory variables was analyzed by Prentice and Glockler's (1978) models for grouped data approximated by an exponential regression model including a time dependent explanatory variable (Ducrocq 1999).

The heritability was analyzed by two statistical models based on the threshold liability concept, a non-linear sire model and a linear animal model. Survival analysis with Prentice and Glockler's (1978) model was used to estimate the heritability of age-at-onset of RS.

**Results and discussion**

*The early signs*

Histological examination of the centrodistal joint (CD) collected from young horses at slaughter revealed chondronecrosis of the articular cartilage from the age of 6 months with an increasing frequency up to the age of 4 ½ years, in total 33% of the specimens. The lesions were found both in the medial and the lateral parts of the joints without significant difference between the two locations. Sclerosis of the subchondral bone detected by high detail radiography was commonly found in the medial part of the joints with an increasing frequency up to the age of 6 ½ years. As it was not associated with the presence of chondronecrosis, but strongly related to age, it was considered to be an adaptation to dynamic strain (Kawcak 2001). However, in the lateral part of the joint, sclerosis of the subchondral bone was an infrequent finding. In all cases with lateral bone sclerosis, chondronecrosis was also found, but chondronecrosis could be present without bone sclerosis. These findings suggest that lateral bone sclerosis is secondary to, or not related to the degeneration of the articular cartilage. Hence, subchondral bone sclerosis did not appear to be a primary factor in the development of OA in the CD joint but is a result of an uneven distribution of biomechanical forces within the joint. Radiographic defects of the subchondral bone plate were on the other hand significantly associated with chondronecrosis in the corresponding articular cartilage.

*Radiographic findings*

Radiographic signs of OA in the distal tarsal joints (RS) were recorded in 30.3% of the horses (n = 186) and the total number of affected limbs was 306. The prevalence of RS was strongly correlated to age (OR = 1.3 / year, P<0.001), increasing from 18.4% in the 6-year-old horses up to 54.2% in the 12-year-old horses, or by 6% as an average for every year in the age range. There was no significant difference in frequency of RS between stallions, mares and geldings. The results correspond well to radiographic and clinical findings of bone spavin reported from Icelandic horses in Sweden (Eksell *et al.* 1998), taking in account the difference in the mean age. The RS were found bilaterally in 65% of the affected horses and the unilateral findings were evenly distributed between the left and the right limbs. Most commonly the CD was the only joint affected (n = 159), or the CD together with the TMT (n = 124). The TMT joint was seldom affected alone (n = 19) and the PIT was only affected in four limbs. The frequency of bilateral appearance of RS was not influenced by age, but the number of affected joints in the same limb increased significantly with age. In most limbs (n = 260), the extent of the RS was graded as moderate or severe and there was a linear trend with the grading increasing with age.

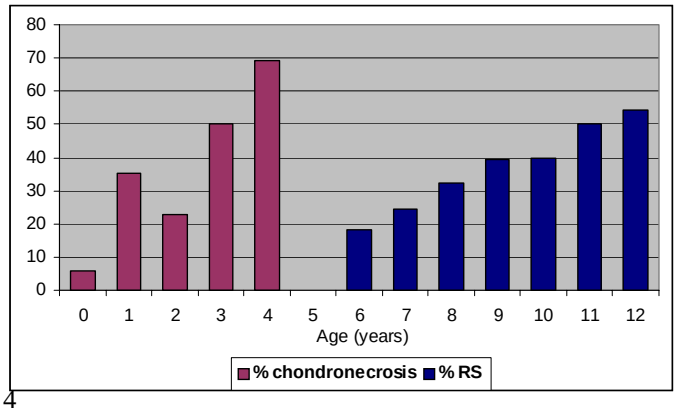


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Fig.2. The frequency of chondronecrosis in the centrodistal joint in specimens from ½ to 4 ½ year old horses and radiographic signs of OA in the distal tarsal joints of 6 – 12 year old horses.

Histology is a more sensitive for detecting early OA compared with radiology, as shown by a higher frequency of chondronecrosis in the articular cartilage from the young horses than the prevalence of RS in 6-12 year horses (Fig 2). Although the two materials are reflecting sub-

populations that might have different frequencies of OA in the distal tarsal joints, the results indicate a progressive nature of the disease from young age. However, if one assumes that all the chondronecrotic lesions will eventually progress to RS, the period required must be extremely variable.

*Clinical findings and their relation to RS*

Hind limb lameness after flexion test of the tarsus was found in 32.4% of the horses (n = 199) and 6.7% (n = 41) of these were lame at presentation. The lameness after flexion test was most often unilateral (85.0%), equally distributed between left and right limbs, and graded as mild (88.6%). The prevalence of lameness after flexion test was not significantly correlated with age and not influenced by gender.

The combination of RS and lameness after flexion test was found in 16.4% of the horses, 13.8% had RS exclusively and 16% showed only lameness after flexion test. The association between RS and lameness after flexion test was strong (OR = 4, P<0.001) as lameness after flexion test was found in 54.3% of the horses with RS, compared to 22.9% of the horses without RS. The absence of periarticular osteophytes reduced the risk of lameness after flexion test from 54.3% to 30.0% while other details of the radiographic findings: the joints involved, the extent of the RS, and signs of active bone remodelling were not associated with lameness in the same limb.

*Prognosis*

In the five year follow-up study, information about the rate of culling was obtained for 508 of the 614 horses originally examined in the survey. The rate of culling was low up to the age of 11 years. The presence of RS affected the survival function in the 5 years period significantly and the difference was highest for 14-year-old horses. The risk ratio of culling was twice as high in horses with RS compared to horses without RS (P< 0.001). The most common disease as cause of culling was hind limb lameness and the risk ratio of culling because of hind limb lameness was 5.5 times higher for the horses with RS (P< 0.001).

*Predisposing factors*

Several potential risk factors for RS and lameness after flexion test were studied separately, which included both intrinsic (age, gender, sire, conformation, gaits, temperament, forelimb action) and environmental variables (place of birth and examination, management and workload). The final multivariate model for RS included: age, tarsal angle and birthplace. Horses with a larger tarsal angle had a lower prevalence (OR/degree = 0.83, P<0.05) confirming that the tarsal conformation is associated with OA in the distal tarsus, probably by altering the biomechanics of the distal tarsal joints. Horses born in the north and south regions of Iceland had a lower prevalence of RS (OR = 0.27, P<0.001 and 0.41, P<0.05 respectively) compared with the other parts of the country. This is most likely an indirect genetic effect because of clustering of dams in the specific regions.

The final multivariate model for lameness after flexion test included: sires, age when broken to saddle, entered at stud show, height at croup and gaits. Two progeny groups had significant higher (OR = 8.26, P<0.05 and OR = 8.93, P<0.05 respectively) and one significant lower (OR = 0.05, P<0.05), indicating genetic predisposition. Breaking to saddle at a young age and especially entering stud show at a young age is expected to result in high workload on relative young (4-6 year) horses. However, horses broken to saddle at 6 years or older had a higher prevalence of lameness than horses broken to saddle at 4 years of age (OR = 12.07, P<0.001) as well as horses not entered at stud shows (OR = 3.74, P<0.001). These results indicate that the workload does not contribute to the development of the disease. The lack of a significant effect of the training intensity and competing status on any of the dependent variables, RS or lameness after flexion test supports this. The mean height for sound horses was 6 mm higher than for horses with lameness after flexion test. Although significant, the difference is too small

to be of value. Four gaited horses (walk, trot, *toelt* and gallop) was the only gait group having a significantly lower prevalence of lameness after flexion test (OR = 0.30, P<0.001) compared with the group of pace-like toelters. The effect of gaits is difficult to interpret, as the ability to perform the different gaits may be a consequence of the disease rather than a predictor. In any case, there was no indication of toelt or other riding qualities being a risk factor for the disease.

*Heritability*

The heritability analysis according to the linear animal model resulted in the following estimates on the underlying scale: for RS, $h^2$ = 0.10 (SE = 0.06) and not significantly different from zero. For lameness after flexion test, $h^2$ = 0.42 (SE = 0.13), and for the combined trait RS and lameness after flexion test, $h^2$ = 0.22 (SE = 0.08). The non-linear threshold sire model yielded similar results but higher standard errors. The estimated genetic correlation between RS and lameness after flexion test was of the order of 0.70. The phenotypic correlation was estimated as 0.30 and the corresponding environmental correlation was 0.22. The high genetic correlation estimate supports the assessment that lameness after flexion test was most often caused by OA and using the flexion test as a selection criteria would reduce the prevalence of bone spavin. The different heritability estimates of RS and lameness after flexion test can probably be explained by the different effect of age on the two dependent variables. If the effect of age was partly genetic, including it as a fixed effect in the linear model can have removed genetic effects as well as environmental effects and caused an underestimation of the heritability estimates of RS. Exclusion of age from the model would on the other hand have caused an overestimation of the heritability, since the progeny groups were unevenly distributed across the age classes.

An alternative way to analyze the data of RS was to apply survival analysis for censored data. The horse's age at examination was treated as a discrete time scale variable and the presence of RS was registered as the age-at-onset (failure) while negative findings represented censored data. The heritability estimates of the age-at-onset of RS (failure time) yielded the effective heritability estimate of $h^2$ = 0.33. Simulations of greater data sets provided strong evidence for the conclusion that this estimate is far less biased than the low heritability estimates of RS obtained by the binary threshold model.

Although reliable results from genetic analysis would have required much larger data than were available for this study, these results strongly indicate that the age-at-onset of RS, which reflects the predisposition for OA in the distal tarsal joints, is a trait with medium-high heritability.

## General discussion

The material of the survey reflects the population of active riding horses in Iceland. Offspring from 17 selected sires, representing all the major breeding lines, were selected to allow heritability estimates as well as the prevalence assessment. Results from the heritability assessments indicated that the dams represented a random sample. The slaughter house material did not reflect the riding horse population but was applicable to detect the first morphological lesions compatible with OA in the distal tarsal joints of Icelandic horses.

The high prevalences and the characteristics of chondronecrosis in the CD joint and RS in the CD and TMT joints confirm that bone spavin is a common disease in Icelandic horses that starts at young age. High frequency of similar histological lesions has been found in young horses of other breeds (Barneveld and van Weeren 1999, Laverty *et al.* 1991) but comparable information about the prevalence of RS in mature horses of other breeds is not available.

The increasing frequency of chondronecrosis and RS with age is most likely due to accumulation of affected horses within the population rather than an increased hazard rate. The variation in time-of-onset of RS is proposed to reflect a variance in the predisposition for the disease within the breed. Comparison of the frequency of histological findings in the young

horses to the frequency of radiographic findings in the mature horses strongly indicates that the chondronecrosis will in most cases progress to radiographic changes. The effect of age on the grading of RS and the number of joints involved also demonstrate the progressive nature of the disease. This relationship would probably be stronger if the exact age-of-onset was known.

According to the follow-up study, horses are seldom culled because of hind limb lameness before the age of 13 years and many of the horses with RS or lameness after flexion test can be used for riding for many years without becoming clinically affected. It can be concluded that OA in the distal tarsal joints is often subclinically manifested in Icelandic horses. However, some horses become severely lame because of bone spavin. This is probably due to further progression of the disease causing more destructive lesions of the bone and inflammation of the soft tissues of the joints. A radiographic study of 60 Icelandic horses presented with clinical suspicion of bone spavin (Sigurdsson 1991) support this.

Workload in young age or in older age was not found to be a risk factor for OA in the distal tarsal joint. This was supported by the frequent histological findings in the young horses, strongly suggesting that the initiation of the disease is unrelated to the use of the horses for riding. The characteristic gait "*toelt*" was ruled out as a risk factor. In agreement with other reports (Rooney 1969, Morgan 1972, Gabel 1980, Barneveld 1983, Wyn-Jones 1988, Eksell, 1998) reduction in the tarsal angle predisposed for the disease. Although the mean tarsal angle was smaller than 150° for both horses with RS and without RS, indicating sickle hocks to be a common conformation in the breed, the grade of the conformational defect was of importance. It can, however, not be excluded that the small reduction in the tarsal angle for horses with RS, is a consequence of the disease rather than a risk factor.

The genetic predisposition for the disease stands out as the most important aetiological factor. It was presumed that the presence of RS is a quantitative threshold trait with an underlying normal distribution of multigenetic effect. The genetic contribution can be, at least partly, via the conformation of the hock or the shape of the distal tarsal joints. It is suggested that the medium high heritability estimate reflect an inheritent variation in conformation or stability of the distal tarsal joints, resulting in predisposition to the disease.

Selection based on radiographic examination and flexion test of the tarsus can be expected to be useful in reducing the prevalence of bone spavin in the Icelandic horse population. The lack of specificity of the flexion test makes the radiographic examination preferable. It seems most important that sires and dams that develop RS early in their life are excluded from the breeding stock as they are expected to have the highest predisposition for bone spavin. The late appearance of RS will, however, reduce the expected profit as RS will in many cases not be detected until the sires have been used for breeding for many years. Identifying conformational traits with a strong genetic correlation to RS, would therefore be of great importance. The subclinical manifestation may have prevented a natural and artificial selection against the disease.

Sickle hock and narrow hock conformations might result in abnormal loading of the distal tarsal joints. From the caudal view, 77% of the horses in the survey were found to have narrow hock conformation, but none was classified as wide hock. Although the narrow hock conformation was not significantly associated with the presence of RS, it has not been ruled out as risk factor for bone spavin. A more precise grading of the conformation may detect an effect lost in the coarse grading "narrow hock" used here. As the initiation of the disease occurs in the first years of life in many cases medial instability due to laxity of periarticular support structures in foals (Auer 1999) and following valgus deformity should also be considered as an etiological factor.

It is suggested that the conformational defects, probable together with poor intra-articular or extra-articular architecure of the distal tarsal joints contribute to static strain laterally and instability medially. Depending on the severity of the deformity, an attempt at stabilization would result in proliferation of the periarticular soft tissue and formation of periarticular osteophytes, preferable on the medial side of the joints. The immobilization in the lateral part (or the whole joint after stabilization) would favour primary bone healing with resorption of the

subchondral bone plate and spongiosa, resulting in fibrous and bony ankylosis as described by Pool (1996). In a radiographic and scintigraphic study, Eksell *et al.* (1999) found the dorsolateral part of the distal tarsus to be the predilection site of bone spavin in Icelandic horses, indicating that the reparative response of the bone starts there in many cases.

## Conclusions

1. In young horses (1-4 year-old), chondronecrosis of the articular cartilage was commonly found on histology, both medially and laterally in the CD joint. The characteristics of the histological findings strongly indicated early degenerative joint disease. The pattern of radiographic subchondral sclerosis indicated asymmetrical distribution of biomechanical forces within the joint.
2. The prevalence of radiographic signs of OA in the distal tarsal joints (RS) was 30.3% in the population of 6-12 year-old Icelandic horses in use for riding, and was strongly correlated to age.
3. The prevalence of hind limb lameness after flexion test of the tarsus was 32.4% in the population There was a significant correlation between RS and lameness after flexion test and 16.4% of the horses had both of them.
4. The presence of RS and lameness after flexion test affected the survival function significantly in a five years period, with strongest effect in the age range of 12 – 16 years. As the only finding, RS had higher prognostic value than lameness after flexion test, but the presence of both RS and lameness after flexion test had the highest prognostic value.
5. The intrinsic factors age and tarsal angle were significantly associated with RS. Birthplace, which was considered to be an indirect genetic effect because of clustering of dams, was also associated with RS. Workload and other environmental factors tested did not influence the prevalence of RS. Lameness after flexion test was influenced by sire and inverse related to the environmental variables age when broken to saddle and entered at stud show.
6. The heritability of age-at-onset of RS, reflecting the predisposition of OA in the distal tarsal joints, was estimated to be $h^2 = 0.33$ and the estimation of the heritability of lameness after flexion test yielded $h^2 = 0.42$. Together, the results suggest a medium high heritability of OA in the distal tarsal joints.

## The overview is based on following papers

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