

## HEREDITARY LAMINITIS IN JERSEY CALVES IN ZIMBABWE

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### ABSTRACT

The clinical signs and radiological findings of a rare laminitis-like condition in Jersey calves (n=6) are described. Regular hoof-trimming proved very beneficial. Pedigree studies of the affected calves strongly suggest a recessive autosomal inheritance.

**Key words:** Jersey cattle, laminitis, hereditary

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### INTRODUCTION

In 1949 a laminitis-like syndrome in young Jersey calves was reported from California<sup>6</sup>. A hereditary origin was suspected and a 'recessive monogenic autosomal gene' was thought to be the cause of the disease, as was suggested by pedigree studies and a breeding experiment<sup>6</sup>. Later a similar condition was recognised in 19 Jersey calves in South Africa<sup>2</sup> and in 3 calves in the USA<sup>7</sup>. A breeding experiment carried out, using an affected bull in the South African study, very strongly suggested the presence of a simple autosomal recessive gene. Edwards<sup>3</sup> described 5 cases of hereditary laminitis in Jersey calves in the UK. In this group a direct link could be established with the bull incriminated in the South African study. In both groups, parentage could be traced to a common male ancestor, 16 generations back. This bull, registered on the Isle of Jersey in 1912, was thought to be the original mutant (H P A De Boom 1989 173 Marija Street, Wonderboom, RSA, personal communication).

The present report describes a case of hereditary laminitis in 6 pedigree heifer calves in a Jersey herd near Harare.

### CASE REPORT

Early in 1988, Jersey heifer calves (n=6) were presented with signs of acute lameness. The calves ranged from 3,5 to 6

months old and were housed and fed in groups amongst calves of the same age. They were reluctant to move, stood with arched backs and cow-hocked hindlegs. Weak muscular tremors were observed in the shoulder muscles of some of the calves. If forced to move, the calves walked stiff-legged and very gingerly on all 4 feet. Their general body condition was good and on physical examination, all vital signs appeared normal. On palpation, some hooves felt warm and sensitive at the coronary band, and in some cases a strong pulse could be felt in the common digital arteries in the front legs. No abnormalities were found in the limbs proximal to the hooves. A nutritional problem was suspected and the calves were taken off concentrates. The acute signs resolved within a few days, but within the next weeks the hooves became elongated and distinct horizontal laminitic rings started to appear on the walls. The anterior hoof walls became dished and the soles more prominent (Fig. 1 & 2). One of the affected calves was taken to the University of Zimbabwe Veterinary Hospital for a radiographic examination, together with an unaffected calf of exactly the same age for comparison. The radiographs of the affected calf, showed shortening of the third phalanx when compared to those of the unaffected calf. The animals remained slightly lame, but regular corrective foot-trimming (shortening of the toe and flattening of the sole) proved very beneficial. The natural pigmentation of the horn prevented any possible haemorrhages from being detected. Nine months after the first onset of signs, only one calf still needed regular trimming. The others

appeared normal, apart from slight flattening of the hooves and the occurrence of some horizontal lines. This calf which needed trimming was brought into the clinic for a follow-up radiographic study. This revealed a broken foot axis, flattening of the hoof, a convex anterior wall, shortening and upward rotation of P3. No abnormalities could be detected in P1, P2 and the articular surfaces.

All 6 calves grew normally and at 12 to 13 months of age, were artificially inseminated by the owner, using semen of unrelated sires. By this time all of them have calved. All calves born were clinically normal and have not developed any signs of the foot disorder.

A brief study of the parentage of these calves born to different dams, revealed that all had the same sire, an American bull (FO) of which semen was imported into Zimbabwe. Other calves born to different bulls, but raised under the same conditions did not show any signs of the disease. A hereditary defect was suspected and a questionnaire was sent to all Jersey breeders in Zimbabwe, who had purchased semen of the suspected carrier bull (FO). None of the addressed farmers had noted the condition on their farms in offspring of this particular bull.

An extensive study of the pedigree of the affected calves was carried out (Fig. 3) and revealed the following: five of the 6 calves had a maternal great-grand sire in common, a locally-bred Jersey bull (Sek). This bull was known to be a carrier of another hereditary defect known in the Jersey breed, namely recto-vaginal constriction. The sixth calf had, on its maternal side, a sire strongly related to the maternal grand sire of the sire of the calves.

Late in 1988, a full brother of one of the affected calves, was born with severe front-limb arthrogyposis and palatoschizis. This animal was euthanased. The postmortem examination supported the clinical findings. This condition has been proven to be of hereditary origin in Charolais, Hereford and Shorthorn breeds as well as in piglets<sup>5</sup>, and suggests that the American bull is a carrier of more than one hereditary defect.

### DISCUSSION

The clinical signs and the radiographic findings, although less severe, correlate closely with those described by other

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Fig. 1 & 2: **Hind feet of a 14-month old Jersey heifer with hereditary laminitis. Note the laminitic rings and the dished appearance of the hooves**

A single recessive autosomal gene is suspected to be responsible for the condition. Criteria, which taken together, strongly suggest a recessive trait<sup>3,7,8</sup>, are:

1. The parents of affected animals need not be necessarily affected, the defect may skip generations.
2. All offspring of 2 affected parents are affected.
3. Approximately equal numbers of males and females are affected.
4. The average genetic relationship bet-

authors<sup>1,2,3,4,7</sup>. In the present cases, the animals did not develop a severe chronic laminitis necessitating slaughter on humane grounds. A possible reason for this could be that in the present outbreak, the calves were presented at an early stage and almost from the onset, the condition was treated by regular corrective hoof trimming. This therapy may be of considerable value in managing the disorder.

The fact that only 6 half-sibling calves, were affected amongst a group of calves, born to different bulls, under the same high standard management and nutritional conditions, makes a hereditary origin very likely and a nutritional or metabolic aetiology very remote, and ties in with the findings of previous studies. The pedigree of the sire of the calves (FO) showed a high degree of inbreeding and a common ancestor in the maternal line of 5 of the 6 calves (SEK). Unfortunately, insufficient information on the parentage of this Zimbabwean bull is available and it proved impossible to establish whether or not a relationship exists with the South African and British lines, as reported by Edwards<sup>3</sup>.

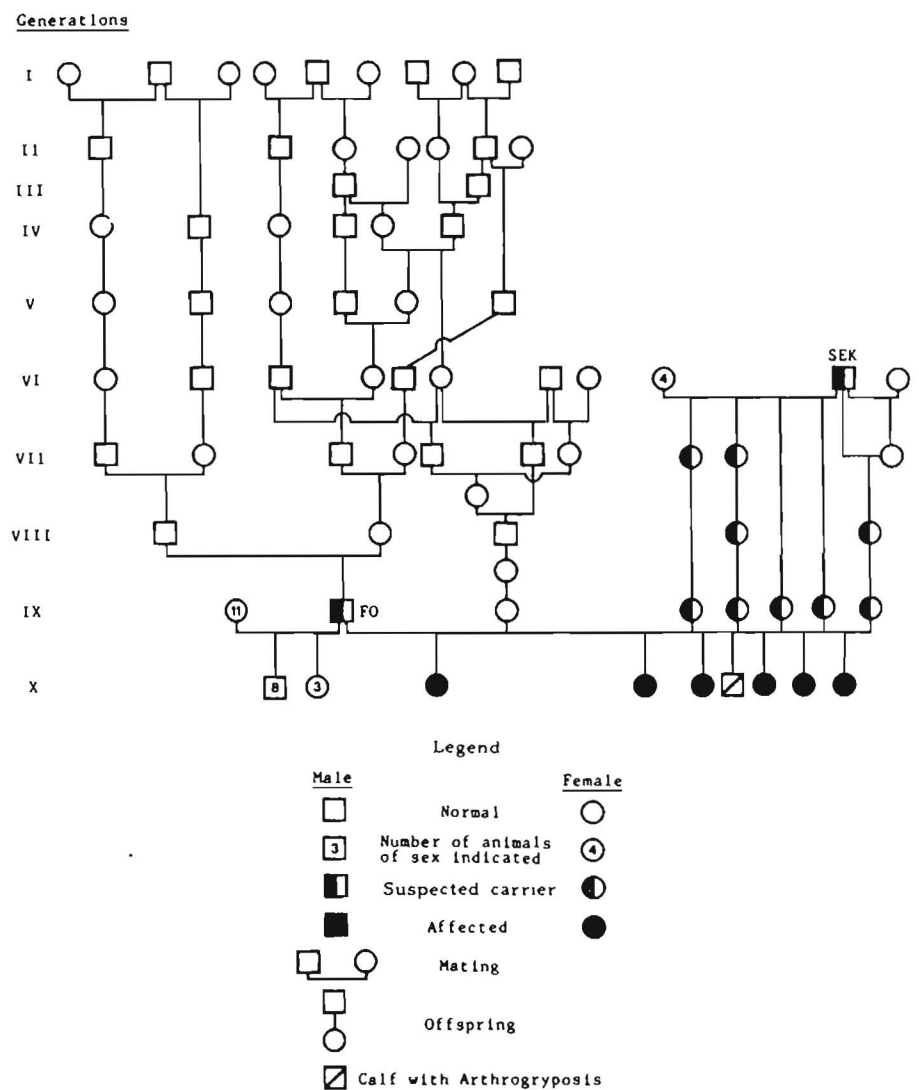


Fig. 3: **Genealogic diagram showing the close relationship between the 6 Jersey calves affected with hereditary laminitis and the inbreeding which occurred in the pedigree of the American Jersey bull FO. The bull SEK in the diagram is the locally-bred Zimbabwean Jersey bull**

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ween normal parents of affected individuals is greater than between normal parents that have not produced affected offspring.

Criteria 1 and 4 were met in this case. Criterium 2 was proven by De Boom et al.<sup>2</sup> in 1968 where breeding experiments with an affected bull and affected cows produced 100% affected calves. Unfortunately, it is not known whether the third criterium was fulfilled, as all bull calves born on this farm are culled within a few days following birth.

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