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The effect of insertional suspensory branch desmitis on racing performance in juvenile Thoroughbred racehorses

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Summary

Reasons for performing the study: Limited information exists regarding the prognosis for juvenile racehorses sustaining injury to the suspensory ligament branch insertion (JISBI).

Objectives: To investigate the effect of JISBI on racing performance; and to assess whether the severity of JISBI is prognostically important.

Study design: Retrospective cohort study.

Methods: Records were reviewed for 896 juvenile Thoroughbreds in training, to identify horses with JISBI limited to one branch (cases). The ability of cases to start a race was compared to their cohorts. Race records of maternal siblings were compared to cases.

Results: The prevalence of JISBI for this population was 9.5%. Cohorts were 3.2 times more likely to start as a 2-year-old and 3.6 times more likely to start as a 3-year-old compared to horses with JISBI. Of 58 cases that were compared to maternal siblings, the total earnings per start (EPS), 2- and 3-year-old EPS were all reduced ($P<0.01$). Cases raced a mean of 3.8 times compared to 7.4 races for controls over the study period and were 113.2 days older by the time of first start ($P<0.01$). 'Speed figures' and EPS were, however, similar between cases that had raced and controls. Cases with a moderate–severe lesion (≥ *Grade 2*) were at significantly greater risk of reinjury than mild cases ($P<0.05$).

Conclusions: JISBI causes decreased racing ability as a 2-year-old. Mild cases perform similarly to controls by their 3-year-old season, but more severe cases demonstrate reduced ability as a 3-year-old, with an increased likelihood of reinjury. Although the wastage was higher in case horses with JISBI, individual cases that make it to a race have similar earnings per start as their controls.

Keywords: horse; suspensory branch; suspensory ligament; desmitis; ultrasound; sesamoid; Thoroughbred

Introduction

Suspensory ligament branch injury (SLBI) is an important cause of decreased performance in Thoroughbred racehorses [1,2]. To date, however, the effect of juvenile insertional-suspensory branch injury (JISBI) on a horse's future racing performance is unknown. For the purposes of this study, JISBI was defined as the development of clinical signs of suspensory branch injury at the insertion on the proximal sesamoid bone (PSB), prior to the start of the 2-year-old racing season.

Few studies have been undertaken regarding SLBI in racehorses and publications that do exist mainly base their prognoses on anecdotal observations and range from good to poor [3,4]. It is important, therefore, to examine objectively the relationship between JISBI and subsequent racing performance, to allow clinicians to provide evidence-based prognostic advice to their clients.

The suspensory apparatus of the horse consists of the suspensory ligament (SL) and its extensor branches, the proximal sesamoid bones and the distal sesmoidean ligaments [3]. Loss of integrity at the sesamoid attachment results in inability of the SL to function properly [4]. The SL bifurcates into lateral and medial branches one-half to two-thirds of the way down the metacarpus/tarsus, and the branches insert mainly onto the concave abaxial margins of the proximal sesamoid bones (PSBs) [1,3]. A recent *in vitro* study observed that fatigue of the muscular elements in the body of the SL may explain the high incidence of lesions at the insertion and the branches [5].

Clinical signs of SLBI depend on the degree of damage and chronicity of the lesion and include localised heat and swelling. Swelling of the branches may be accompanied by periligamentous oedema or fibrosis [6]. There may be associated distension of the metacarpophalangeal/metatarsophalangeal joint capsules owing to the intimate relationship between the SL and the joint capsule [6]. Pain on palpation is present in acute cases, but the presence of lameness is variable and may be absent [3,7].

Diagnosis is based on clinical signs and ultrasonography [2,3,7]. The lack of significant muscular tissue within the SL branch and its superficial location aid in both imaging and interpretation [8,9].

Disruption of fibre pattern, altered echogenicity, ligament enlargement and poor differentiation of ligament margins are all recognised ultrasonographic features [2]. The entire suspensory apparatus should be examined ultrasonographically as lesions may extend further proximally or distally [8–10].

The first objective of this study was to investigate the influence of JISBI on racing ability. Our hypotheses were that horses with JISBI would be less likely to race as a 2- or 3-year-old than their cohorts, and that the earning ability of affected horses would be reduced.

The second objective was to determine whether the severity of JISBI had an affect on future racing ability. Our hypothesis was that more severe cases of JISBI, as determined ultrasonographically, would have poorer racing ability than those horses less severely affected.

Materials and methods

Study population

Daily training charts and veterinary inspection forms, in addition to detailed veterinary notes, on juvenile Thoroughbred racehorses were reviewed from the commencement of training in September of their yearling year, until 31 May of their 2-year-old year for clinical signs of JISBI. Detailed histories were reviewed on 896 horses from September 2007 to May 2010. All yearlings were clinically normal when they entered race training and were located at a single training centre in Florida.

Cases were identified as juvenile Thoroughbreds-in-training with a first occurrence of JISBI in one branch of one limb prior to the start of their racing career. Cases with multiple suspensory branch involvement were excluded. For the purposes of this study, clinical injury was defined as: the presence of palpable pain and/or avoidance response to direct palpation; visible swelling or palpable thickening of the branch; or focal inflammation, with or without lameness.

The trainer evaluated each leg for palpable abnormalities on every horse once daily: all horses were evaluated for lameness each morning before training, and any abnormalities were documented. The second author evaluated all horses weekly during the study period in addition to those presented to him by the trainer. A veterinarian was present at this training facility every day and regular direct visualisation of each horse was routine.

Detailed veterinary notes on each horse in the cohort were available for review.

All suspected cases of JISBI were confirmed ultrasonographically as previously described [1–3] and images were stored for review. The entire SL was evaluated ultrasonographically and only those horses with lesions confined to the insertion of the suspensory ligament branch were included in this study.

Ultrasonographic technique

All ultrasonographic examinations were performed by an experienced clinician using a Logic E[a] ultrasound machine coupled to a variable frequency high-definition linear array probe, set between 8 and 10 mHz, and a standoff pad. The ultrasound images were transferred to a DICOM workstation for review. Limbs were clipped using a No. 40 blade, and cleaned with alcohol before applying coupling gel.

The horse was positioned with weight evenly distributed. Each affected branch was scanned in its entirety in both transverse and longitudinal planes. Three still images were reviewed for each branch: at the insertion; 1 cm above the insertion; and 2 cm above the insertion. The contralateral suspensory branch was scanned for comparison and images were recorded as for the affected branch. All measurements were recorded for subsequent analysis.

Prior to the initiation of this study, interobserver agreement was investigated in a pilot study and determined to be good (unpublished data).

The images were reviewed by the first and second authors and a consensus grade was assigned. The following parameters were recorded: altered fibre pattern; altered echogenicity; focal disruption; swelling; periligamentous oedema or fibrosis; or increased ligament size relative to the corresponding contralateral SLB. An arbitrary ordinal grading system was devised as follows (Fig 1):

Grade 1: enlargement of branch compared to contralateral branch. Hypoechoic areas and/or irregular fibre pattern without discrete core lesion.
Grade 2: enlargement of branch compared to contralateral branch with discrete core lesion representing <10% loss of fibre attachment at PSB.
Grade 3: enlargement of branch compared to contralateral branch with discrete core lesion representing >10% loss of fibre attachment at PSB.

The ultrasonographic appearance of the abaxial border of the PSB was also evaluated and the presence of PSB pathology was recorded as *present* or *absent*. This classification was based on the regularity of the bone surface; evidence of enlarged vascular channels; and/or the presence of mineralisation/fragments at the bone–ligament interface (Fig 2).

Treatment of cases

All case horses were treated conservatively without specific therapeutic intervention.

The horses were managed with appropriate trimming and shoeing, stall rest, and a controlled increasing exercise programme [11]. The horses were followed through their rehabilitation period with sequential ultrasound examinations and physical examinations every 30 days. Examination reports were reviewed for each case.

Racing data and performance ratings

Racing data were obtained from the Equibase[b] website. Dichotomous outcome variables included whether a horse started a race as a 2- or a 3-year-old. Categorical variables included total number of starts; number of 2-year-old starts; number of 3-year-old starts; and total combined starts. Continuous outcome variables included: combined average (total) earnings per start (EPS) as a 2- and 3-year-old; EPS as a 2-or 3-year-old; days from birth to first start; and days from injury to first start.

Additionally, the average Equibase speed figure was used as a measure of performance. The ‘speed figure’ is published by Equibase in the USA as an aid to gambling: it calculates how fast a horse ran in a race and is reported as one single number. It uses advanced algorithms based on actual race times in combination with other factors such as the condition of

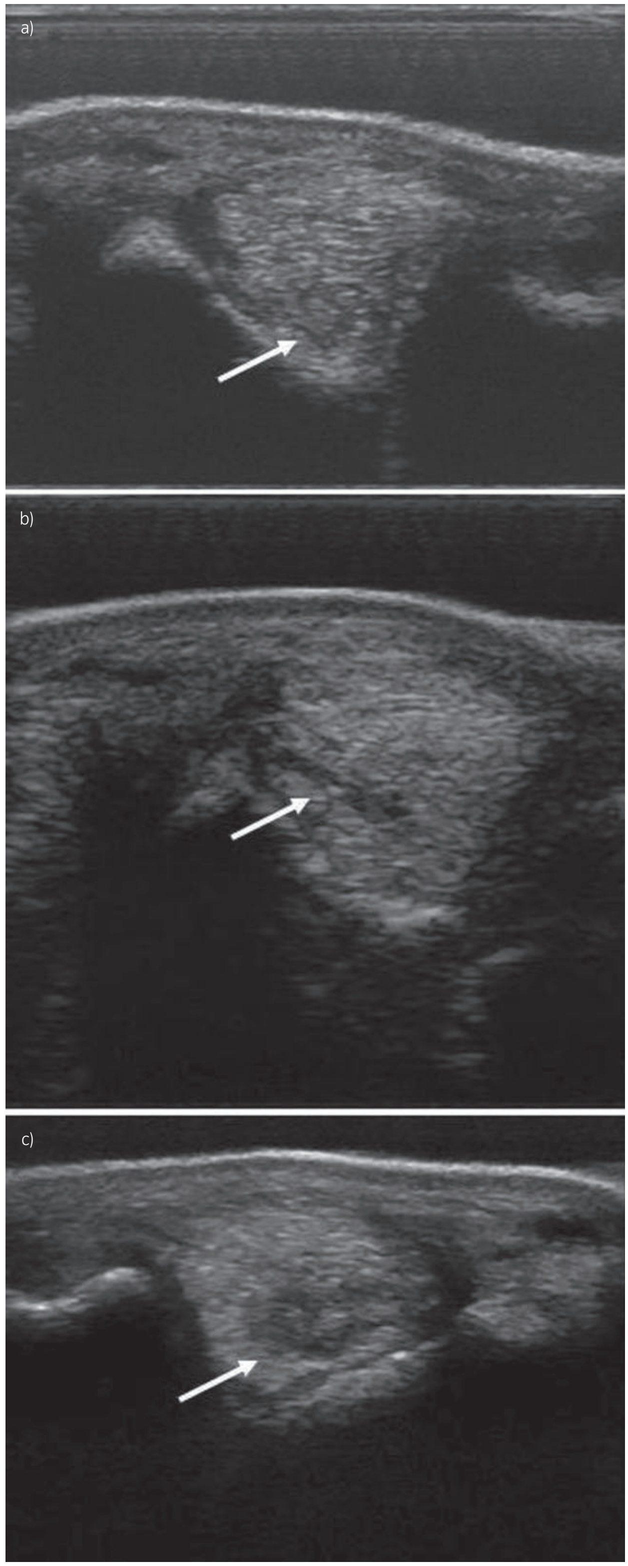

Fig 1: Different grades of suspensory pathology assessed on transverse ultrasound examination. Dorsal is to the left of the image. a) *Grade 1*: Hypoechoic areas and/ or irregular fibre pattern without discrete core lesion at insertion (arrow); b) *Grade 2*: discrete core lesion representing <10% loss of fibre attachment at the proximal sesamoid bone (arrow); c) *Grade 3*: discrete core lesion representing >10% loss of fibre attachment at the proximal sesamoid bone (arrow).

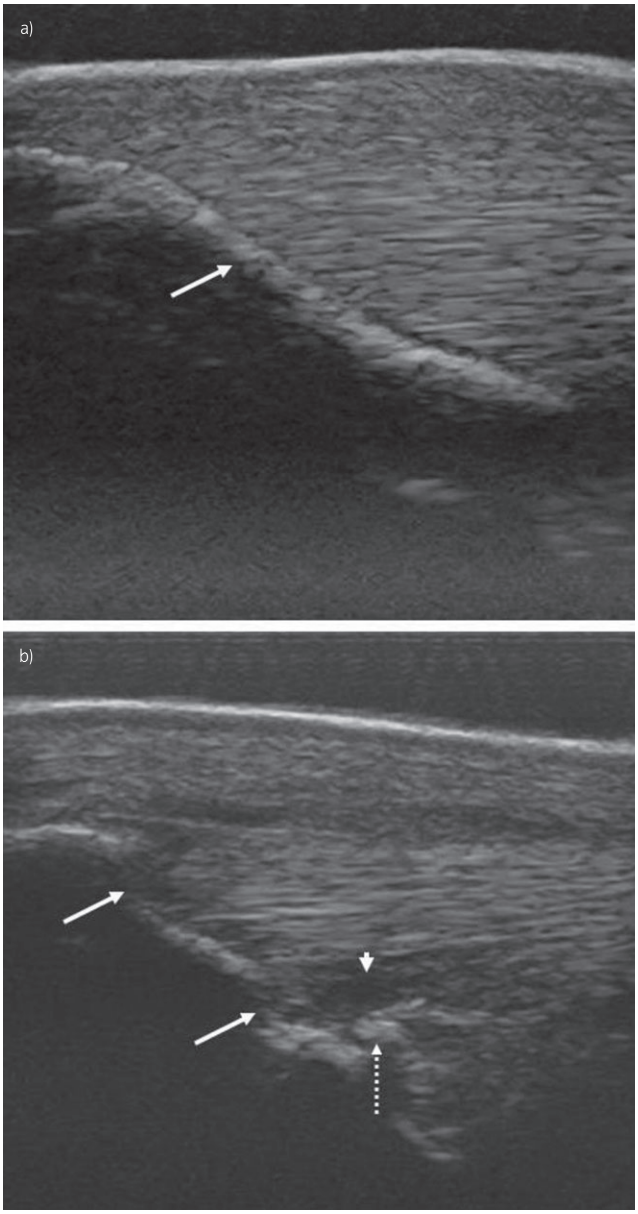

Fig 2: Longitudinal ultrasound of suspensory ligament attachment on the proximal sesamoid bone. Proximal is to the right of the image. a) Normal attachment with no loss of fibres and no obvious sesamoid pathology. This is a healthy reference image from a control. Note normal bone-ligament interface (arrow). b) Evidence of sesamoid pathology in association with suspensory pathology (arrowhead). Note the openings to enlarged vascular canals (arrows), and a small proximal fragment (dotted arrow).

the track. It has been equalised for different tracks, to ensure equality in comparison of speed figures between horses. This is similar to the *Racing Post Rating* used in European studies [12]. The average speed figure was calculated for each horse that ran.

Days at risk were recorded for each horse as the number of days of canter or gallop exercise over the study period.

Cohort and maternal sibling controls

Training records and veterinary histories were reviewed for all noncase horses within the cohort. Prior to the commencement of the study, strict criteria for the selection of matched controls from the unexposed (noncase) cohort were established. The race record of one maternal sibling was compared to each case sibling. Eligible siblings, being selected from the cohort, were known to be free from JISBI. The closest (by age) elder or younger maternal sibling was chosen as a matched control. As a prerequisite, controls had to be free from any history of any significant musculoskeletal injury, including JISBI, over the 'juvenile' study period. By the nature of the training establishment studied, siblings were, by default, matched for owner, racetrack and race trainer. For the matched sibling analysis, a detailed history was obtained from the racetrack trainer, to ensure that no other significant variable may have arisen subsequent to the juvenile study period (for example a horse sustaining another significant but unrelated injury). Only cases and siblings with no documented history of other significant injury were included in this analysis.

Data analysis

All statistical analyses were performed using a commercially available software package (Excel)[c] and a freely available online tool (Statpages.org)[d]. Fisher's exact test and Chi-squared tests of independence were used to determine relationships between descriptive variables in the cohort. For dichotomous outcomes, the presence of confounding variables within the cohort was identified by Mantel-Haenszel testing. The influence of confounding on binomial outcome variables was assessed by multivariate logistic regression analysis, and adjusted odds ratios (adjOR), corrected for confounding variables, were calculated. For the case–sibling analysis, measurement variables were assessed for normality. Where the data, or the log-transformed data, were found to be normally distributed, parametric analyses were performed using paired *t* tests for matched data between siblings. Tests were run for all possible configurations of data.

For all tests, P<0.05 was considered significant.

Results

Four juvenile horses died (2 colic, 2 catastrophic injury) during the study period. The cohort comprised 896 horses. Race records, or trainer follow-up for nonstarters, were available for the remaining 892 horses. Eighty-five juvenile horses (51 colts, 34 fillies) demonstrated JISBI during the study period. The prevalence was 9.5% with an incidence rate of 0.47 cases of JISBI/1000 horse days at risk. Multivariate analysis demonstrated that sex was not a confounding variable (P>0.1), but the presence of significant tendon and ligament injury unrelated to JISBI (such as superficial digital flexor tendon injury), during the study period, was a significant confounding variable (P<0.05). The odds ratios presented for binomial variables were adjusted for this confounding variable.

Fifty-six (65.9%) horses with JISBI had started a race by the end of their 3-year-old season compared to 88.9% of the unaffected individuals (P<0.001, adjOR 4.2, 95% confidence interval [CI] 2.0–8.8). Only 27.1% of cases ran as a 2-year-old, and 62.3% as a 3-year-old, compared to 57.3% and 81.9%, respectively, for unaffected individuals. Unaffected horses were 3.2 times more likely to race as 2-year-olds (P<0.001, adjOR 3.2, 95% CI 1.8–8.5), and 3.6 times more likely to start as 3-year-olds (P = 0.01, adjOR 3.6, 95% CI 1.9–8.1) than horses with JISBI. Case horses had fewer total starts (3.8 vs. 7.4), 2-year-old starts, and 3-year-old starts, than unaffected individuals (P<0.001, P<0.001, P<0.02, respectively).

Fifty-eight horses (37 colts, 21 fillies) satisfied the selection criteria for maternal sibling comparison. Twenty-seven juvenile horses were excluded owing to failure to meet rigorous selection criteria: 9 had no matched sibling within the cohort; 8 had matched siblings which sustained other injuries; 6 sustained unrelated injury; 3 had insufficient follow-up; and one died due to colic during the study period.

When total EPS, 2-year-old EPS and 3-year-old EPS were compared, all 3 parameters were significantly reduced in case horses (P<0.005, P<0.001, P<0.01 respectively) vs. matched controls. By using data only from paired samples that ran successfully, however, no difference was observed in the speed figure (P = 0.19) or earnings, although horses suffering JISBI required, on average, 1085 days (851–1844 days) from birth to first start. This was significantly longer than the 977 days (766–1263) observed in their maternal controls (P<0.01). These results are summarised in Table 1.

TABLE 1: Results for juvenile suspensory branch insertional injury (JISBI) on racing performance

		Number of cases (%)		P value	
Location	Left fore lateral	15 (17.65)	Left fore overrepresentation	**0.02**	
	Left fore medial	19 (22.35)			
	Right fore lateral	9 (10.59)			
	Right fore medial	10 (11.76)			
	Left hind lateral	9 (10.59)			
	Left hind medial	7 (8.24)			
	Right hind lateral	12 (14.12)			
	Right hind medial	4 (4.71)			
	Total	85			
			Cohorts		
All JISBI cases in cohort		Number of cases (%)	Number of cohorts (%)		
Total		85	811		
Started a race		56 (65.88)	721 (88.90)	**<0.001**	**adjOR 4.2 (95% CI 2.0–8.8)***
Started as a 2-year-old		23 (27.06)	465 (57.33)	**<0.001**	**adjOR 3.2 (95% CI 1.8–8.5)***
Started as a 3-year-old		53 (62.35)	664 (81.87)	**0.01**	**adjOR 3.6 (95% CI 1.9–8.1)***
		Mean (range)	Mean (range)		
No. of starts		3.81 (0–12)	7.45 (0–22)	**<0.001**	
No. of starts as 2-year-old		0.66 (0–7)	2.36 (0–9)	**<0.001**	
No. of starts as 3-year-old		3.27 (0–11)	5.1 (0–14)	**<0.02**	
		Versus matched siblings			
		58 cases	58 matched siblings		
		Mean (range)	Mean (range)		
EPS ($)		2966.6 (0–30,557)	3972.63 (0–32,966)	**<0.005**	
EPS 2-year-old ($)		2068.19 (0–45,525)	2183.01 (0–26,691)	**<0.001**	
EPS 3-year-old ($)		2267.44 (0–13,101)	4605.93 (0–75,000)	**<0.01**	
Speed figure		78.8 (22–115)	84.1 (44–114)	>0.1	Runners only
Av (days) to first start		1085.26 (851–1844)	972.13 (766–1263)	**<0.01**	Runners only
EPS ($) (runners)		5301.23 (500–30,557)	6102.69 (1324–32,966)	>0.5	Runners only
EPS ($) 2-year-old (runners)		4208.91 (823–45,525)	3989.53 (500–26,691)	>0.2	Runners only
EPS ($) 3-year-old (runners)		5890.42 (725–13,101)	6459.06 (1402–75,000)	>0.5	Runners only
No. remaining in training after age 3 years		32 55.20%	45 77.60%	**<0.01**	

*Odds ratio adjusted for presence of significant tendon/ligament injury unrelated to JISBI. EPS: total average earnings per start over 2- and 3-year-old seasons; adjOR: adjusted odds ratio; CI: confidence interval.

Desmitis was identified at the insertion of each of the 8 possible suspensory branches, although it was a more common forelimb finding (62.4%). Injury to the left forelimb was overrepresented (P = 0.02) and accounted for 40% of all cases of branch desmitis (17.6% lateral, 22.3% medial). No significant difference in any outcome parameter was observed when forelimb cases were compared to hindlimb cases. This was also true for any combination of right/left/medial/lateral cases, including the over-represented left forelimb versus all other combinations.

When cases were stratified according to severity of JISBI and paired with their matched controls (Table 2), those with *Grade 1* lesions raced fewer

TABLE 2: Effect of juvenile suspensory branch insertional injury severity on racing performance vs. maternal siblings

	Grade 1 Mean (range)	Sibling Mean (range)	Significance	*Grade 2* Mean (range)	Sibling Mean (range)	Significance	*Grade 3* Mean (range)	Sibling Mean (range)	Significance
Total no. starts	4.1 (0–12)	7.1 (0–22)	*>0.05*	3.5 (0–12)	7.6 (0–21)	***<0.01***	4 (0–9)*	7.7 (0–20)	*
No. 2-year-old starts	0.7 (0–4)	2.3 (0–9)	***<0.02***	0.7 (0–7)	2.5 (0–8)	***<0.01***	0.2 (0–1)*	1.9 (0–8)	*
No. 3-year-old starts	3.8 (0–11)	4.8 (0–14)	*>0.1*	2.7 (0–7)	5.1 (0–14)	***<0.05***	3.7 (0–9)*	5.9 (0–12)	*
Total EPS ($)	5385.5 (0–30,557)	4464.3 (0–32,966)	*>0.1*	3218.4 (0–13,101)	4174.0 (0–28,240)	***<0.05***	3031.9 (0–13,436)*	1879.5 (0–532.5)	*
EPS 2-year-old ($)	2180.7 (0–45,525)	3239.1 (0–26,691)	***<0.05***	2335.1 (0–13,101)	4743.6 (0–28,240)	***<0.05***	3975.9 (0–31,800)*	622.9 (0–4228)	*
EPS 3-year-old ($)	5480.3 (0–6910)	5506.9 (0–75,000)	*>0.05*	3267.7 (0–12,020)	3976.6 (0–9735)	***<0.05***	2960.6 (0–11,141)*	1550.8 (0–5322.5)	*
'Speed' figure	75.1 (33–100)	81.6 (51–108)	*>0.2*	83.6 (58–103)	82.4 (44–114)	*>0.2*	71.3 (22–115)*	77.6 (53–99)	*
Avearage (days) to first start	1084.9 (851–1281)	979.2 (766–1263)	***<0.05***	1056.8 (912–1246)	953.9 (778–1250)	***<0.02***	1096.3 (902–1844)*	995 (817–1191)	*

*Group too small for meaningful analysis (insufficient power). EPS: total average earnings per start over 2- and 3-year-old seasons.

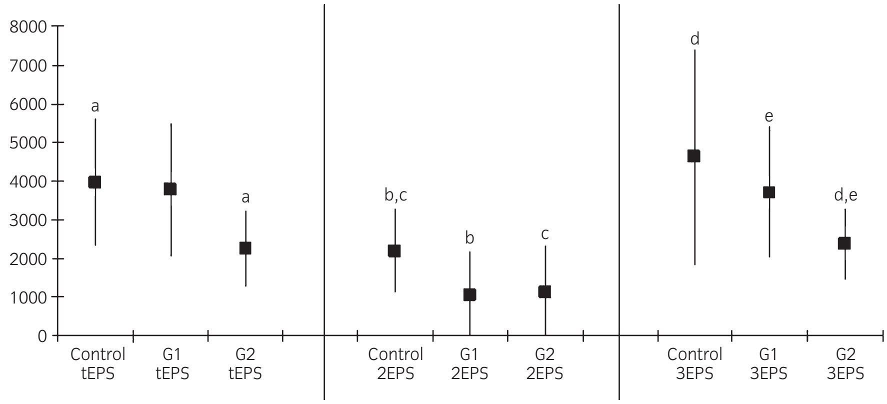

Fig 3: Mean (boxes) and 95% confidence interval (whiskers) of total average earnings per start over 2- and 3-year-old seasons (EPS), 2-year-old average earnings per start (2EPS), 3-year-old average earnings per start (3EPS) for pooled controls, *Grade 1* (G1) and *Grade 2* (G2) cases. *Y* axis is in US Dollars. Categories with matching letters are significantly different.

times as 2-year-olds (P<0.02) and had lower 2-year-old EPS (P<0.05) than their maternal siblings. *Grade 2* cases, in addition, had fewer total starts (P<0.01), 2-year-old starts (P<0.01) and 3-year-old starts (P<0.05) than their siblings, in addition to having lower total EPS (P<0.05), 2-year-old EPS (P<0.05) and 3-year-old EPS (P<0.05). Figure 3 summarises the EPS data for *Grade 1*, *Grade 2* and pooled controls. By the end of their 3-year-old year, 77.6% of controls remained in training, compared to 69.6% of *Grade 1* cases and 55.5% of *Grade 2* cases (P<0.05 control vs. *Grade 2*). Insufficient numbers of *Grade 3* lesions precluded further analysis of this group. Sesamoid pathology was visualised ultrasonographically in 34 (40%) cases. The presence of sesamoid pathology was significantly related to the grade of JISBI (P<0.05), being a more common finding in cases with *Grade 2* JISBI or above. When cases were divided on the basis of sesamoid pathology *present* or *absent*, those with pathology had fewer total starts, 2-year-old starts, and 3-year-old starts (P<0.005, P<0.001, P>0.05 respectively), in addition to lower total EPS, 2-year-old and 3-year-old EPS and a significantly longer time to first start (P<0.05).

Discussion

In this population of TB racehorses, a significant proportion (9.5%) was found to have JISBI. This correlates well with previous studies that have shown desmitis of the branches of the SL in the forelimbs and hindlimbs to be a relatively common condition [1,3,13].

The forelimb was the most common location for a branch injury, a fact that has been reported previously [13,14]. Although JISBI was identified in all 8 possible locations, the left forelimb (LF) was the injured limb in 40% (34/85) of cases (P = 0.02), with injury to the left front medial suspensory branch occurring 22.3% of the time compared to 17.6% for the lateral branch. This may be a result of anticlockwise training placing increased stress on the LF SLB. It could also be related to the track surface or the tight turns on North American training tracks, or to the horse's conformation. Foot imbalance and shoeing are often recognised as predisposing factors for SLBI [11], with abnormal conformation resulting in asymmetrical loading of the metacarpophalangeal/metatarsophalangeal region [1].

Despite the overrepresentation of LF injury, no change in performance was observed between LF injuries and injuries in all other locations. Hindlimb SLBs were injured 37.6% (32/85) and forelimbs 62.4% (53/85) but no difference was observed in performance outcomes when forelimbs and hindlimbs were compared to each other. This is in contrast to a previously proposed theory that injuries to the apparatus in the forelimb carry a worse prognosis than in the hindlimb [15]. It may be that the presence of the lesion is more significant than its specific anatomical location, but this would require further investigation.

Horses with JISBI were significantly less likely to have raced by the end of their 3-year-old season (P<0.001) than their cohorts, with only 66% starting at all: 27% raced as 2-year-olds; 62% raced as 3-year-olds. In comparison, 89% of their cohorts had started a race by the end of their 3-year-old season: 58% raced as 2-year-olds and 82% raced as 3-year-olds. This is in agreement with our first hypothesis that horses with JISBI would be less likely to race. When adjusted for possible confounding variables, cohorts were 4 times more likely to have started a race by the end of their 3-year-old season than horses suffering from JISBI.

This may be due to several factors, such as the presence of a JISBI that had not yet healed sufficiently to permit racing during the study period. It is also possible that trainers elected not to run these cases until they were 4-year-olds. This is, however, unlikely as all cases sustained their initial injury prior to the start of the 2-year-old racing season, thus it can be assumed they should have resumed training and have run by the end of their 3-year-old season.

It may also have been due to reinjury to the affected branch: 37.5% of cases with a *Grade 3* insertional lesion reinjured the same branch prior to the end of their 3-year-old season, as did 29.2% of *Grade 2* cases and 10.2% of *Grade 1* lesions. These rates of reinjury are greater than the 9.5% prevalence observed in the general population and are likely to account for much of the wastage and failure to run observed in this study. It is widely accepted that recurrence of SBLI is common [3], but to the authors' knowledge this is the first time that it has been quantified.

It was decided not to follow these case horses into their 4-year-old season. Owing to the decline in the number of cases successfully competing through the 3-year-old season, it was surmised that insufficient cases of differing severity would be present by the end of the 4-year-old season on which to perform statistical analysis.

One limitation of this retrospective study is that the cohort was not physically inspected by the authors once it departed to race. Analysis of race records, therefore, cannot account for all subsequent confounding variables, such as unrelated injury, which may have affected any horse's ability to race. While any such factor may be likely to affect cases and cohorts with equal frequency, the effect may be diluted by the larger cohort group, and amplified in the smaller case group. By undertaking case–sibling analysis, we attempted to limit the exposure to such unknown confounders by obtaining a detailed history for each case and sibling up to the end of its 3-year-old season.

Significantly fewer cases started as 2-year-olds (27%) than as 3-year-olds (62%). In the general population, fewer starts were made as 2-year-olds (58%) than as 3-year-olds (82%), so the finding of reduced 2-year-old starts among cases is not surprising. It is probable, though, that the fewer 2-year-old starts made by cases was due to the extended time from birth to first start exhibited in case horses: on average, 1085 days from birth to first start, compared to 977 days for control horses. This increased time represents missed training during the convalescence and rehabilitation period, and partly explains why fewer 2-year-old starts were made by cases: they were not back in race training until midway through their 2-year-old season.

Significantly fewer total starts were made by case horses over the study period, with an average of 3.8 starts compared to cohorts, which had on average 7.4 starts by the end of their 3-year-old season (P<0.001). This may be a function of early retirement due to reinjury, or it may be that case horses were more lightly raced by trainers who were aware of the horses' underlying pathology. One flaw of retrospective studies using race records is that the effect of trainers' actions on results cannot be accounted for and may bias the results. Bias may also be encountered from comparison of multiple race outcomes or comparing EPS from different racing seasons.

Race records, however, represent an established method for comparing outcomes in racehorses; and the use of average EPS as an outcome variable reduced this bias and highlighted that, as a group, their performance was reduced compared to their matched controls.

In agreement with our second hypothesis, cases exhibited a decrease in total EPS, 2-year-old EPS, and 3-year-old EPS relative to their controls. The reduction in total EPS, 2-year-old EPS and 3-year-old EPS seen in cases indicates that they earned less money than their matched controls. It was presumed that this decrease in performance would also be demonstrated by comparison of the average speed figure between cases and controls. No significant difference was observed, however. This may be because the average speed figure is not a sensitive indicator of a horse's ability, or it may be because this variable is only evaluated in horses that run, and is therefore not affected by nonrunners. When total EPS, 2-year-old EPS and 3-year-old EPS were compared using parametric testing only for cases that ran, for example, no significant difference was observed in earnings between cases and controls. This is in agreement with the speed figure information and would suggest that, in cases that actually race successfully, their performance in any given race is equivalent to that of their matched controls.

The speed figure has not been previously used as an outcome parameter, although a similar European parameter, the Racing Post Rating, has been used successfully [12].

Our final hypothesis was that horses with more severe injuries would not perform as well as those with less severe injuries. When cases were stratified based on severity of JISBI, all grades raced fewer times and earned less money as 2-year-olds than their matched controls. This was probably related to lost training during the 2-year-old season for all grades of JISBI. Insufficient numbers of *Grade 3* lesions precluded meaningful analysis of many parameters. Larger study populations are necessary to investigate the effect of more severe JISBI on performance.

Grade 2 or *3* lesions were, however, found to be at significantly greater risk of reinjury ($P<0.05$) than *Grade 1* lesions. *Grade 2* lesions required longer from the point of injury until first start than *Grade 1* lesions. This may have been due to the fact that healing was monitored by serial ultrasound examinations and cases with core lesions, obviously, required longer to return to training than those without.

When cases with *Grade 1* lesions were compared to their sibling controls they had fewer starts as a 2-year-old and earned less money as a 2-year-old. Their number of starts, and total earnings as a 3-year-old, however, were not significantly different from their matched controls. They were as likely to remain in training by the end of their 3-year-old season as their matched controls. From this information we can surmise that horses with a *Grade 1* lesion will take longer to get to their first race than controls, owing to rehabilitation time, but by their 3-year-old year they should perform as well as horses that did not suffer from this injury.

Grade 2 cases, however, earned less money and started fewer times than their matched controls in both their 2- and 3-year-old seasons. This phenomenon may have been due to the greater incidence of reinjury in *Grade 2* cases. It also highlights that although *Grade 2* cases perform similarly to *Grade 1* cases as 2-year-olds, they do not appear to have the longevity required to successfully race throughout their 3-year-old season. Our results indicate that horses with *Grade 2* or above JISBI do not hold up well to training: only 55.5% of *Grade 2* cases remained in training at the end of their 3-year-old season, compared to 77% of their controls.

The failure of these *Grade 2* horses to stand up to training may be partly explained by the fact that horses cannot recreate the exact structure of the natural insertion of the SLB following injury. Although healing may occur, the insertion of the SLB becomes reconstituted with scar tissue, and it is improbable that any type of scar tissue will be able to function in a similar fashion to the undamaged SLB [4]. It is believed that when the level of training/racing is sufficiently demanding, the fibrous tissue repair of the ligamentous insertion will fail [4].

The cases in this study returned to training following previously described guidelines, which recommend return to full work either when: 1) the lesion has 'healed' ultrasonographically; or 2) there is no appreciable change in the ultrasonographic appearance between 2 examinations 3 months apart and the horse has been rested for at least 6 months [9,11]. However, in the face of persistent ultrasonographic lesions it is difficult to predict accurately when a horse may be able to return to work successfully, and the persistence of ultrasound abnormalities beyond the return to clinical normality and athletic activity is not uncommon and can lead to confusion [9]. It was, therefore, difficult to assess when many of these *Grade 2* cases were sufficiently healed to re-enter work. This uncertainty related to quality of healing may also explain the high rate of reinjury and reduced performance.

One further limitation of this study design was the use of ultrasonography and clinical examination as the principal methods of diagnosis and follow-up. Although palpation can localise injury to the SLB, through the presence of pain and/or periligmentous soft tissue swelling, it is not specific to SLBI. Moreover, palpation can be an insensitive indicator of SLBI, as some cases demonstrate few localising signs, especially if partial tears are present [16]. Both the sensitivity and the specificity of ultrasound examination for detection of SLBI have previously been questioned: it has been reported that, in some horses with clinical features indicative of SLBI, ultrasonography failed to reveal abnormalities [17]. In contrast, ultrasonographic abnormalities can exist in a small but significant proportion of horses without clinical indication of injury [3]. These shortcomings have the potential for misleading results, although by combining the results of clinical examination with ultrasound imaging, the authors felt confident about the accurate diagnosis of cases in this study.

Sesamoid pathology was present in 34 cases (40%). This study demonstrates that the severity of JISBI was significantly related to the presence of sesamoid pathology ($P<0.05$), with sesamoid pathology being a more common finding in cases with *Grade 2* or above suspensory pathology. It has previously been reported that horses exhibiting sesamoid pathology have reduced performance [18], and it is probable that the triad of sesamoid pathology, JISBI and reduced racing ability are linked [13]. It has been recently shown that the presence of significant sesmoiditis is a risk factor for the development of suspensory branch injury [13]. This is in agreement with other research that states that active sesmoiditis and inflammation in the bone causes bone resorption that further weakens the SLBI, which predisposes to further damage in a cyclical progressive manner [4].

Therefore, when providing prognostic information on JISBI it is prudent to note the presence of concurrent sesamoid pathology.

One potential limitation of this study was that limbs were not routinely radiographed. Obtaining radiographic projections of the affected PSB and distal splint bones would have been prudent [9] because fractures, enlarged vascular channels, cystic or linear lucent lesions and modelling of the abaxial margins of the sesamoid bones may occur concurrently with suspensory branch desmitis [3]. Any of these concurrent conditions may have influenced racing performance and hence the results of this study. It has been reported, however, that these conditions are also visible with ultrasound, and that radiography may only be required if suspicions of such pathology exist subsequent to ultrasonographic examination [1,19,20]. Any horses demonstrating visible pathology consistent with such injuries were excluded from the case population; therefore, the authors were confident that none of these conditions existed in these cases. Although useful to a complete examination, radiographic projections were not obtained in all cases.

To the authors' knowledge, there are no studies specifically correlating the association between radiographic and ultrasonographic findings of PSB pathology, and this is an important area for future investigation.

It was decided to only incorporate horses into this study that had been treated conservatively. This decision was made in an attempt to limit the variability inherent from the numerous treatment options available, many of which are used empirically and currently have no published literature to substantiate their use [17]. The results obtained in this paper demonstrate the prognosis for racing following insertional SLB injury in horses treated conservatively, and represents a baseline prognostic reference for this specific injury. Future studies are required to analyse individual interventional treatment options, particularly the increasingly popular regenerative treatment options, to evaluate any post treatment change in prognosis.

The authors decided to exclude horses that had JISBI involving multiple limbs. There were insufficient cases of multiple limb involvement to interpret results adequately. Larger studies are required to elucidate the effect of multiple limb, ipsilateral, contralateral, and contra-axial orientations of JISBI on racing ability.

Conclusion

Overall, JISBI causes decreased racing ability as a 2- and 3-year-old. Mild cases perform similarly to controls by their 3-year-old season but more severe cases demonstrate reduced ability as a 3-year-old.

An important finding from this study is that although the wastage was higher in cases with JISBI, individual cases that make it to a race have similar earnings per start to their controls.

The likelihood of repeat injury is greater for the more severe cases of JISBI.

The presence of sesamoid pathology coincides with more severe JISBI and reduced racing performance.

These factors should be considered when providing prognostic advice to clients regarding JISBI.

Authors' declaration of interests

No competing interests have been declared.

Ethical animal research

Ethical review not required by this journal: retrospective study of clinical and racing records.

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Authorship

Dr Plevin and Dr McLellan contributed equally to the design, data collection, and execution of this study. Dr McLellan performed the data analysis and both authors contributed equally to the creation of this manuscript.

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