Received: 15 February 2022 | Accepted: 11 May 2022

DOI: 10.1111/evj.13599

NARRATIVE REVIEW

Equine Veterinary Journal WILEY

A review of equine tibial fractures

Kristin Bowers[1] | Joshua T. Weinhandl[2] | David E. Anderson[1]

[1]Large Animal Clinical Sciences, University of Tennessee College of Veterinary Medicine, Knoxville, Tennessee, USA

[2]Department of Kinesiology, Recreation, and Sport Studies, University of Tennessee, Knoxville, Tennessee, USA

Correspondence
Kristin Bowers, Large Animal Clinical Sciences, University of Tennessee College of Veterinary Medicinem, Knoxville, TN, USA.
Email: kmbowerseq@gmail.com

Abstract

Equine tibial fractures are relatively infrequent in racing and non-racing sport horses, but limitations in successful treatment of tibial fractures in adult horses result in relatively high mortality compared with other musculoskeletal injuries. The aetiology of tibial fracture can be classified into two general categories: traumatic impact or fatigue failure. Tibial stress fractures, also known as fatigue fractures, are often rated as the second most common stress fracture in racing Thoroughbreds; young age, early stage in race training, and initiation of training after a period of rest are the reported risk factors. Both impact and fatigue fracture propagation are dependent on the magnitude of force applied and on the local composition/alignment of mineralised collagen in the tibial lamella. Extensive research has characterised the pattern of strain distribution and stress remodelling within the equine tibia, but in vivo measurement of load and angular moments are currently not feasible. Further research is warranted to correlate biomechanical theory of tibia fatigue fracture propagation with current histopathological data. Preventative measures for fatigue fractures aim to optimise diagnostic efficiency, reduce the interval between injury and diagnosis and modify racing and training conditions to reduce non-specific fracture risk. Treatment options for complete tibial fractures in adult horses are limited, but with careful case selection, successful outcomes have been reported after open reduction and internal fixation. On the other hand, tibial stress fractures and minimally displaced incomplete fractures are typically treated conservatively and have good prognosis for athletic recovery. This review aims to describe the current literature regarding tibial fracture aetiology, prevalence, risk factors, fracture biomechanics, treatment, prognosis and prevention.

KEYWORDS
biomechanics, fracture, horse, racehorse, stress fracture, tibia

1 | INTRODUCTION

Musculoskeletal injuries are among the most prevalent problems affecting horses and result in significant emotional distress and economic losses in the racing, sport horse, and show horse industries. These injuries are the most prevalent causes of euthanasia in racehorses with roughly 80% of deaths in California Thoroughbred racehorses attributed to musculoskeletal injury.[1] Complete tibial fractures are relatively infrequent in racing and non-racing horses. However, limitations in successful treatment of tibial fractures in adult horses result in relatively high mortality compared with other musculoskeletal

Abbreviations: ORIF, open reduction and internal fixation; IRU, increased radiopharmaceutical uptake.

injuries.[2] A complete, multimodal understanding of the pathogenesis of equine tibial fracture is crucial to optimising treatment and prevention modalities. The aims of this review are to describe the prevalence, risk factors, biomechanics and current practices regarding prevention and treatment for equine tibial fracture.

Equine tibial fractures can be broadly categorised as complete or incomplete. These categories are linked by two distinct aetiologies: trauma, such as a kick injury or automobile accident, and fatigue, such as exercise-induced stress remodelling.[3] This review will focus on complete fractures, incomplete fractures and stress microtrauma of the tibial diaphysis, but a few exclusions must be acknowledged. This review will focus on injuries of juvenile and adult horses; physeal fractures have been excluded because of their unique characteristics, resulting in dramatically different treatment modalities and prognosis.[3,4] Avulsion fractures of the tibial tuberosity and intercondylar eminence fractures have been excluded from this review because they differ in their aetiology and treatment.[3]

2 | PREVALENCE

The reported prevalence of equine tibial fractures has changed considerably over time, but common trends can be gleaned from these reports (Figure 1). Starting in earnest during the 1970s and 1980s, race-related tibial fractures have been characterised as unique entities in equine orthopaedics.[3] During the late 1990s, there was a marked increase in published work regarding race-related fracture localisation and biomechanical stresses affecting the equine tibia.[5–7] Digitisation of medical records facilitated the development of large-scale, multicentre epidemiological studies after 2000. These studies were used to assess the current state of tibial fracture with regards to prevalence in modern racing and non-racing populations.[8,9]

A survey conducted with 13 racehorse trainers in the United Kingdom, between 1998 and 2000, found that 148 fractures had been diagnosed out of 1178 horses (12.5%).[10] Twenty-one out of 148 fractures were stress fractures of the tibia (14.2%), making tibial fracture the third most common site of injury after the third metacarpal bone and pelvis.[10] Pelvic and tibial fractures were reported to account for almost a third of all fractures diagnosed, marking a notable increase in prevalence than had been previously reported.[11] In a retrospective epidemiological study focusing on all Thoroughbred racehorses examined at Randwick Equine Centre in Australia, tibial stress fractures were diagnosed in 9% of horses having nuclear scintigraphy done for musculoskeletal problems between January 1992 and March 2000.[7]

The California Horse Racing Board's Postmortem Program required postmortem examination of all horse deaths associated with racing in the state.[8] Between January 1990 and December 2018, 115 racehorses were euthanised for tibial fracture in California, yielding an annual median rate of four (range 1-10) fatal tibial fractures per year.[8] This represents a fracture rate of 0.106% for complete tibial fractures in Thoroughbred starters (101/95082). Of 101 horses having tibial fracture, only 18 tibial fractures were sustained by Thoroughbreds during a race; the majority of fatal tibial fractures occurred during training, often at sub-maximum speeds. Compared with Thoroughbreds, the tibial fracture rate in racing Quarter Horses was lower at 0.033% (8/24182) but a similar skewedness towards non-race-related tibial fracture was noted.[8]

Two recent epidemiologic studies focused on Thoroughbred racehorses under the jurisdiction of the Hong Kong Jockey Club. In a retrospective analysis of all Thoroughbreds in race training between August 2003 and July 2014, tibial fractures were the second most common bone fractured (35/119; 29.4%), second only to humerus fractures (59/119; 49.6%).[9] In an analysis of data utilising the same

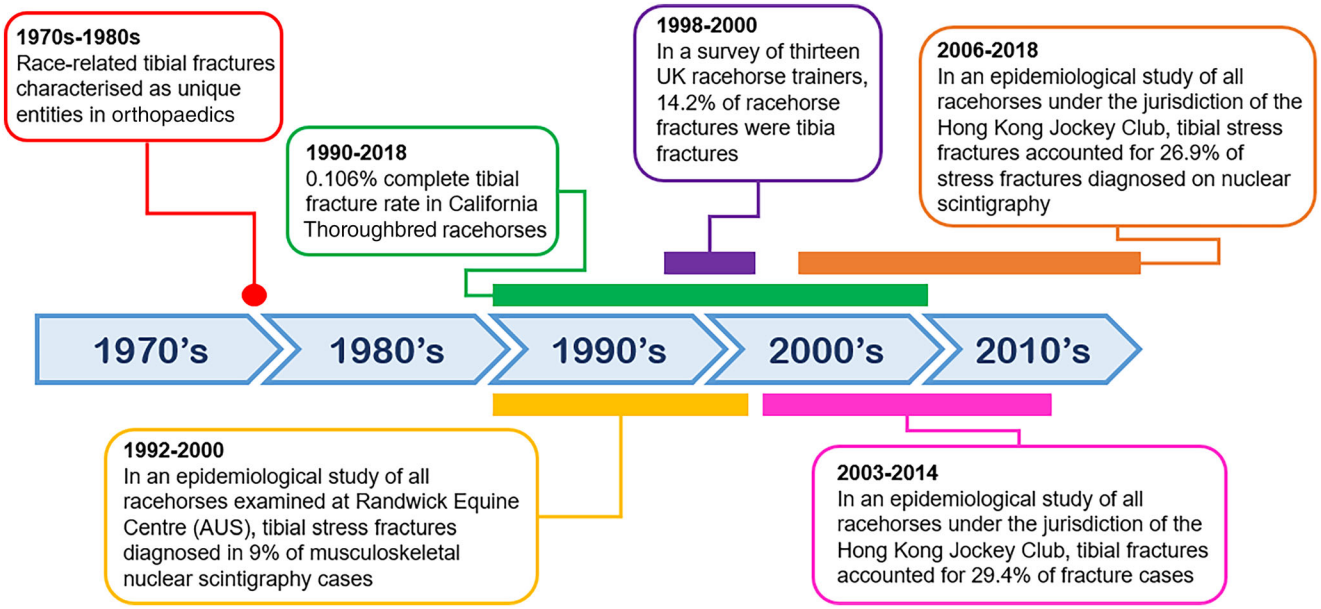

FIGURE 1 Overview of reported prevalence of tibial fracture in racehorses worldwide

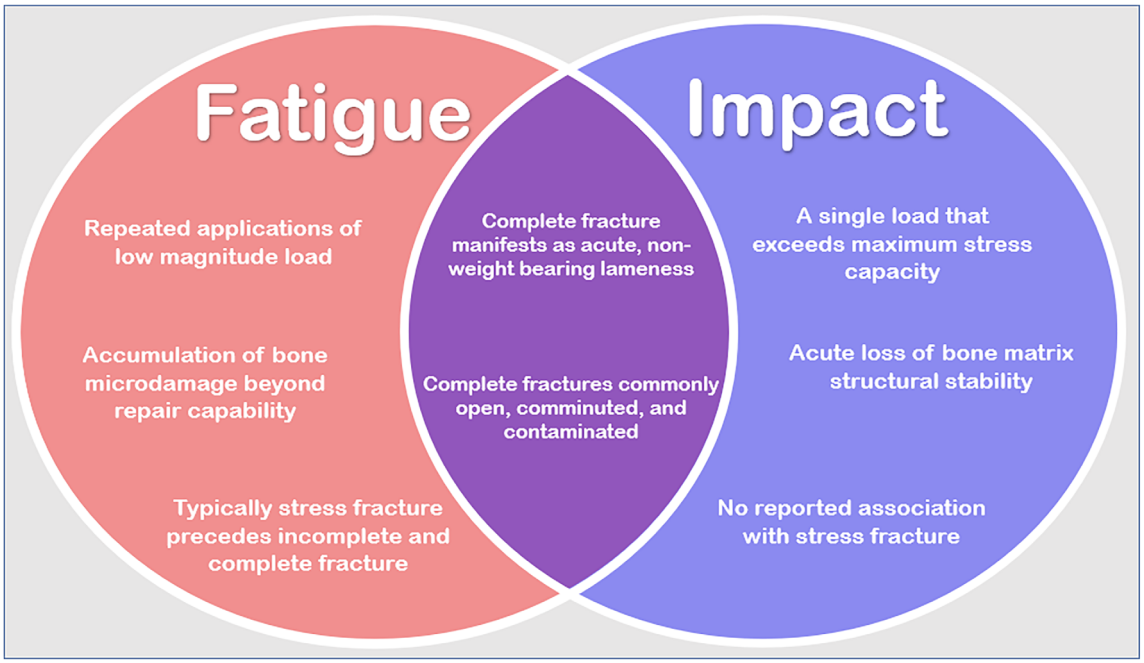

FIGURE 2 Overview of injury mechanisms and clinical presentation of fatigue-aetiology and impact-aetiology tibial fractures in horses

population database of horses racing between January 2006 and June 2018, stress fractures were diagnosed, based on nuclear scintigraphy, in the humerus (42.0%) and tibia (26.9%) most often.[12]

3 | INJURY CLASSIFICATION AND CONFIGURATION

Equine tibial fractures can be separated into broad aetiological classifications of impact and fatigue (Figure 2).[3,13] An impact fracture is the result of a single load acting on the bone that exceeds its maximum stress capacity.[14] The most common impact fractures in horses occur as a result of being kicked. Forces resulting from impact of a single hoof, without horseshoes, have been reported in the range of 9807 N peak force.[15] This impact force is well beyond the resistance threshold of the equine tibia, which has been reported to range between 1100 N and 2300 N.[15] On the other hand, a fatigue fracture forms from repeated applications of load of low magnitude which occurs frequently enough to prevent repair of bone microdamage attributed to significant strain.[16] Fatigue fractures are typically sustained during physical activity that causes muscle fatigue, reducing the muscle's ability to store energy and neutralise the stresses imposed on the bone.[13] In the literature, the terms 'fatigue fracture' and 'stress fracture' are often used interchangeably and are often implied when describing tibial fractures in racehorses.[3]

In 119 racehorses euthanised due to tibial fracture in California, 111 fractures (93%) were classified as complete and comminuted.[8] Eighty-two percent of fractures were diaphyseal, of which the proximal diaphysis was most commonly affected. Of the complete fractures, the fracture configurations were most often oblique, transverse or oblique with a short transverse component.[8] In an ex vivo model of kick injury, simulated kicks to the tibia most commonly resulted in incomplete fractures with a butterfly configuration or incomplete longitudinal fractures.[14] When subjected to the simulated kick in a modified three-point-bending trial, the degree of comminution was greater in tibial fractures as compared with radius fractures which the authors attributed to a difference in surface geometry between the equine tibia and radius.[14]

Regardless of aetiology, complete tibial fracture typically manifests clinically as acute non-weightbearing lameness of the affected limb.[3] Diaphyseal fractures are associated with a high degree of soft tissue injury, and severe swelling and sensitivity of the overlying tissue is common. The limb often displays valgus angulation due to the inherent asymmetry of muscular attachments; the craniolateral muscle mass abducts the distal limb, forcing the distal fragment medially, and due to the lack of medial soft tissue coverage, complete tibial fractures often become open and contaminated.[3] Extensive comminution, especially when open and contaminated, greatly limits the prognosis for success repair and rehabilitation.[3]

4 | BIOMECHANICS

Bone is an organised matrix of mineralised collagen fibres (organic matrix dominated by Collagen Type I), and it is surrounded by and interspersed with specialised cells for first sensing the bone's environment and subsequently responding with appropriate upkeep of the mineralised (inorganic) matrix.[17,18] It is comprised of cortical and trabecular bone, both composed of lamellae, the organised units of collagen bundles and their cells.[18] The mechanical characteristics of axial

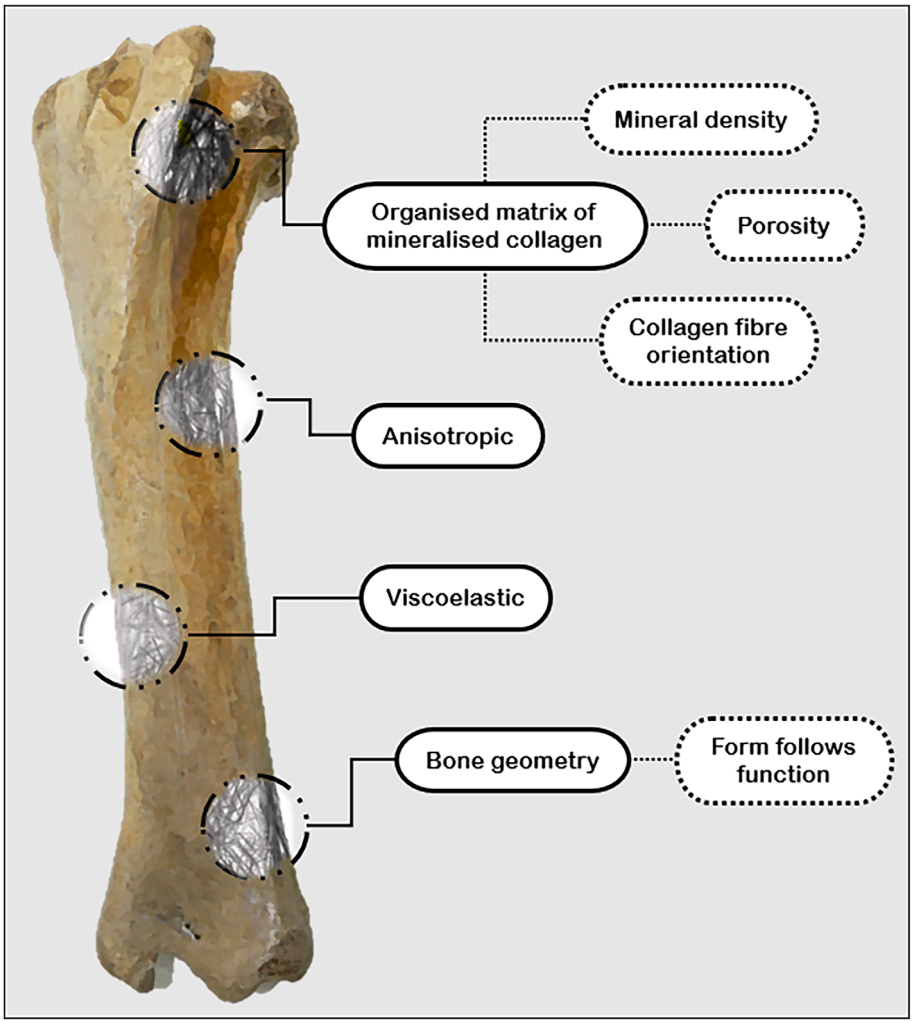

FIGURE 3 Compositional and biomechanical properties of the equine tibia that affect fracture initiation and propagation

bones can be described as anisotropic and viscoelastic. Anisotropy refers to the variable response of bone to mechanical stresses based on the direction of loading and the geometrical shape of the bone.[19,20] Viscoelasticity refers to the variable response of bone to mechanical stress based on the rate of loading.[19] Collagen fibre orientation within the bony lamellae was reported as a better predictor of bone strength compared with degree of mineralisation; in general, longitudinally oriented fibres are strongest in tension and transversely oriented fibres are strongest in compression.[21] In a study of compositional variables that affect mechanical properties of the cranial and caudal cortices of the equine radius, Riggs et al. noted a dichotomy of collagen organisation with longitudinal collagen fibres predominating in the cranial cortex and oblique and transverse fibres predominating in the caudal cortex.[17] In mechanical testing to failure of transverse sections of the radial diaphysis under both tensile and compressive loads, collagen fibre orientation accounted for 71% of the variation in tensile strength, 58% of the variation in elastic modulus and 43% of the variation in strain to failure.[17] Mineral density and porosity were the second and third most influential variables in the radial sections.[17] From the pattern of collagen fibre orientation, it was concluded that the radius, a relatively cylindrical bone, underwent predominantly bending forces during locomotion.[17] When compared with the radius, tibial geometry is less uniform, and geometric irregularities at soft tissue attachments may act as inherent stress risers (Figure 3).[13] The collagen fibre orientation in the equine tibia is most consistent with a combination of bending and torsional forces transmitted along the tibial diaphysis, and the consistent location of stress remodelling observed by Samol et al. corroborates this hypothesis.[8,13]

While equine hindlimb kinematics have been described, no studies that focus on the tibia alone have been published. In three dimensional radiosteriometric analysis of equine stifle biomechanics, Halley et al. noted significant external rotation of the tibia as the joint was extended from 110 degrees to full extension.[22] In a direct three-dimensional kinematic analysis using intra-cortical pins in the tibia and third metatarsus, Lanovaz et al. investigated angular stresses experienced in the equine tarsus, hypothesising that the screw motion accountable for the calculated angular stress would originate at the tarsus.[23] However, their hypothesis could not be confirmed, and the authors suggested that the angular stresses must arise from another joint.[23] Taken together, these biomechanical analyses confirm that with each stride, the tibia undergoes external rotation originating at the stifle, but the tarsocrural joint does not share the same degree of rotation; this means that the proximal tibia and distal tibia experience unequal rotational moments at the walk, resulting in significant

torsional stress with each stride, and its patterns of stress and strain distribution reflect such.

In ex vivo biomechanical modelling of the walking gait, tensile strain is localised to the craniolateral aspect of the tibial cortex in the middle and proximal portions of the tibia diaphysis.[7] Moving distally, strain is more torsional and relatively inconsistent between tibia specimens. This pattern of strain distribution at a walk was confirmed in vivo using rosette strain gauges applied to the tibia in four sites: cranial, caudal, medial, and lateral.[24] Notably, the tensile axis of strain was identified in the craniolateral diaphysis, as reported by O'Sullivan et al., but the highest overall magnitude of strain was torsional, not tensile.[7,24] Hartman et al. repeated in vivo strain measurements using six rosette strain gauges applied on the cranial, caudal, and medial surfaces of the tibia, and noted a geometric pattern of strain distribution.[25] As previously documented in the radius, tensile strain predominated on the cranial surface of the tibia and compression predominated on the caudal surface; however, torsional strain was superimposed on the craniocaudal bending pattern and the localisation of strain was unaffected by a loss of muscular stabilisation from the cranial tibial muscle.[25] In a similar in vivo biomechanical study, Schamhardt et al. noted the same localisations of tensile, compressive and torsional strains in Shetland ponies and documented no significant change in strain distribution at the walk following transection of the peroneus tertius.[26]

While the localisation and magnitude of equine tibial strain has been well documented in the literature, limited data exist on the magnitudes of loads that the tibia or surrounding joints experience during locomotion. Martig et al. suggest that this limitation stems from the technical difficulty of in vivo load measurement without alteration of physiological loading conditions.[27] Published data from metacarpal and tibia strain gauges are helpful in load estimation but are limited by accessible bony surface on which gauges can be mounted. Computational modelling has allowed for the calculation of hindlimb loads. However, these calculations are a direct function of ground reaction force and increasing speed will amplify the load at joint surfaces.[27] In vitro modelling of tibial cyclic deformation allowed for the calculation of Young's Modulus of the tibial diaphysis at 19.4 GPa on the caudolateral surface and 27.7 GPa on the craniolateral surface.[28] Cyclic loading with stress amplitudes well below the elastic limit resulted in fatigue damage (microcracks) of equine tibial sections. The microcrack growth behaviour was highly dependent on the magnitude and components of local stresses, and in general, fatigue fracture propagated at an oblique angle with microcracks extending along the osteons (described in human literature as 'osteon pull out').[28] Further research is warranted to explore this proposed biomechanical method of fracture propagation in the equine tibia and to correlate this theory with postmortem analysis of tibia fatigue fractures.

5 | FATIGUE ('STRESS') FRACTURES

Fatigue is defined as degradation of material properties when a material or structure is repetitively loaded with forces that are less than the monotonic force required to cause catastrophic failure.[27] The fatigue life of a bone is the number of cycles of a specified loading regimen that can be sustained by a structure before catastrophic failure, and fatigue life is affected by the material properties of the bone, the magnitude of load and the bone's geometry.[27] Microdamage, the debonding between the mineral and collagen phases of bone on a molecular level resulting in the breakage of collagen fibres, is the basic building block of fatigue fracture propagation, but it also is essential for healthy bone remodelling.[27] As stated in Wolff's Law, bone has to be loaded cyclically within a narrow physiological range to retain its functionality, and bone health relies on an equilibrium between the development of microcracks and natural regeneration by basic multicellular units.[28] If the rate of damage exceeds the regenerative capability of a given bone, fatigue fractures will form. This tentative equilibrium can be altered by a multitude of factors including age, body conformation, track and race related factors, high-speed distances, and gender[27]

Tensile and compressive loads have unique effects on microcrack patterns, and propagation characteristics differ by the type of force.[13] In tensile surface failure, a rapid transverse propagation of fracture is observed, often accompanied by a butterfly fragment if perpendicular impact is the source of tension. In compressive surface failure, the fracture develops more slowly as a result of microcrack coalescence and branching, and it is hypothesised that a bone-specific threshold exists between microcrack coalescence and the propagation of a single, catastrophic fracture line.[13]

Evidence of the fatigue remodelling process has been documented in case reports of equine tibial fracture,[7,29] but recently, a retrospective postmortem analysis of racehorse tibial fracture described a significant correlation between fatigue remodelling and catastrophic fracture.[8] Performed over a period of 28 years, periosteal callus was only described in detail during the second half of the reporting window. This is likely associated with increasing awareness of a link between complete tibial fracture and stress remodelling. Thus, of 119 cases, only 53% of necropsy reports note examinations for the presence of callus.[8] Of these 119 cases, 64% of the horses had periosteal callus, evidence of fatigue remodelling, and apparent bridging of the fracture line. In many cases, vascular woven bone, indicative of active remodelling, was noted on the surface of a chronic, established callus, suggesting cyclical exacerbation of stress fractures. Calluses were often associated with cortical osteopenia, which could have served as a stress riser for fracture propagation.[8] While discrete causation cannot be proven from the current literature, the clinical significance of bone fatigue and a loss of microcrack/remodelling equilibrium is a well-accepted mechanism for fatigue fractures in racehorses.[27,30]

The availability of nuclear scintigraphy for diagnosis of lameness in horses has allowed for the identification of previously underdiagnosed stress-induced remodelling and stress fractures of the tibia.[5] Although evidence is limited to small case series or case reports, three consistent predilection sites have been documented in the tibia: proximolateral cortex, caudal cortex of the mid-diaphysis and caudal cortex of the distal tibia. Notable similarities in stress fracture configuration and stress-induced remodelling localisation were documented in both Thoroughbred and Standardbred racehorses, even though the two breeds employ a different racing gait.[6,31] These similarities between case series sparked interest in stress fracture diagnosis.

Thoroughbred racehorses offer an opportunity to study a population having relatively similar exercise and management risk factors throughout the industry.

6 | IMPACT-ASSOCIATED TIBIAL FRACTURE

Case data have been published for a general population of horses, regardless of use, which were treated at the University of Zurich teaching hospital between 1990 and 2014. In this population, 1845 horses presented with fractures, of which 1144 had sufficient diagnostic, treatment and follow-up documentation for inclusion in epidemiologic analysis.[16] Eighty-four of 1144 fractures (7.3%) occurred in the tibia, and 61 of these 84 fractures were secondary to a kick injury.[16] Donati et al. have published one of the few, if not only large-scale epidemiologic analysis of equine fracture injuries in a non-racing population of horses.[16] Consistent with previous case reports and series, this study concluded that tibial fractures of non-racing horses occur most commonly secondary to external trauma, incidence is highly variable and frequency is dependent on management of trauma-associated risk factors.[3,16,32]

In a general population of horses, both racing and non-racing, Donati et al. reported that tibial fractures most commonly resulted from kick injury, and the risk of kick injury increased with group/herd housing, especially in herds with unestablished social dominance structure (Figure 4).[16] Impact fracture was often sustained shortly after new horses were introduced into pre-existing herds or during brief acclimation periods when stalled horses were transitioned to group turnout. Gender was a significant factor in this analysis with mares overrepresented in the tibial fracture group; Donati et al. hypothesised that the factors of social dominance and gender were linked, and that aggressive hierarchical behaviour would be common in group-housed mares.[16]

7 | TREATMENT

Regardless of fatigue or impact aetiology, complete diaphyseal fractures in adult horses are often catastrophic and fatal.[3] Marked comminution of the bone reduces the potential to reconstruct tibial fractures. Also, the tibia's limited regional soft tissue and muscular attachments contribute to complete fractures often being open and contaminated.[3] Rarely, a horse will present with a complete, closed and minimally comminuted diaphyseal tibial fracture that is a candidate for reconstruction with plate fixation. In low body mass equids such as ponies, miniature horses and donkeys, prognosis is considered to be better for surgical stabilisation. Cast stabilisation, alone, is not recommended for treatment of complete fracture of the tibia. In vivo strain measurements, before and after full limb cast application, showed a shift in the principal axis of tensile strain on the distal metaphysis but did not reduce the magnitude of strains measured.[24]

The current standard for surgical fixation of complete tibial fracture is open reduction and internal fixation (ORIF) using two plates of either locking compression plates, dynamic compression plates, or a combination of the two.[3] Two bone plates, perpendicular to each other, are placed on the tibia based on the individual fracture to maximise fixation stability. Minimally invasive options are available and encouraged when applicable.[3] In a retrospective analysis of fracture fixation and surgical site infections between 2008 and 2016 at the New Bolton Center, six tibial fractures were repaired surgically, four of which were closed at presentation and two of which were open. No surgical site infections or fatal complications were reported in the short-term in this case series, but the authors noted that careful case selection and surgical experience are critical factors in successful fracture reconstruction.[33]

According to Watkins et al., incomplete tibial fractures pose a 'management dilemma' as a clinician must balance the risk of propagation to catastrophic fracture against the risk of surgical complication.[3] In general, the gold standard of care for an incomplete tibial fracture is conservative management with cross-tying or sling placement to prevent recumbency. Tibial stress fractures usually are managed conservatively. Several studies report prognosis for recovery and athletic performance following tibial stress fracture diagnosis in racehorses (Table 1).[7,9,10] Of 74 tibial stress fracture cases identified at Randwick Equine Centre between 1992 and 2000, 61 horses had documented follow up after conservative treatment consisting of staged stall rest, hand-walks and exercise restriction for 3 to 4 months after diagnosis.[7] Eighty percent (49/61) of horses returned to racing and 39 logged at least three recorded starts after tibial stress fracture treatment; there was no significant change in earnings per start associated with treatment for tibial stress fracture.[7] Out of 35 tibial stress fracture cases in horses recorded in the Hong Kong Jockey Club database, 77.1% returned to racing after a median of 136 days of convalescence. No difference was noted in first placings or in months to retirement between affected horses and matched controls.[9]

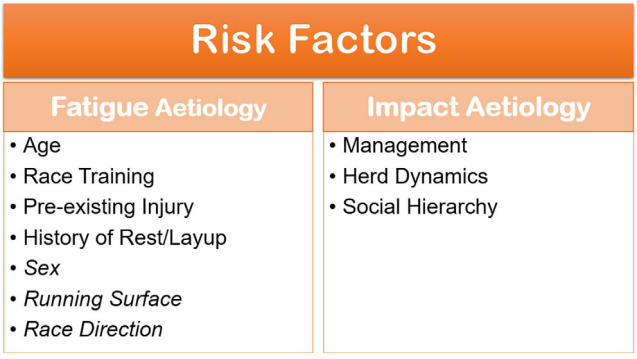

FIGURE 4 Overview of risk factors for tibial fracture; italics signify proposed risk factors that have not been unequivocally proven

8 | RISK FACTORS

Based on evaluation of published research regarding risk factors for fatigue-related tibial fracture, these injuries most commonly occur in racing populations and often are associated with stress-induced

TABLE 1 Overview of reported tibia fracture treatments, outcomes and prognoses organised by fracture characterisation

Author and date of publication	Population description	Sampling time frame	Sample size (tibia fracture)	Fracture characterisation	Treatment modalities	Prognosis/outcome	Notes	Citation
Ruggles AJ et al., 1996	Racing Standardbreds (USA)	1989–1993	13	Tibia stress fractures	Stall rest (8–16 weeks) followed by limited pasture turnout (4–12 weeks)	No complications associated with tibia stress fracture reported by owners or trainers; catastrophic tibia fracture did not occur; 10 of 13 returned to racing		6
Verheyen KLP et al., 2004	Racing Thoroughbreds (UK)	1998–2000	21	Tibia stress fractures	Conservative treatment – details not provided	No complications associated with tibia stress fracture reported by trainers	Prospective epidemiologic study focused on all fractures in Thoroughbred racehorses	10,11
Johnston AS et al., 2021	Racing Thoroughbreds (Hong Kong)	2006–2018	26	Tibia stress fractures	Conservative treatment – details not provided	70.8% (17/26) horses returned to barrier trials within one year of stress fracture	Retrospective epidemiologic study focused on stress fractures diagnosed by nuclear scintigraphy in racing Thoroughbreds	12
O'Sullivan CB et al., 2003	Racing Thoroughbreds (Randwick Equine Centre, Australia)	1992–2000	74	Tibia stress fractures	Exercise restriction (3–4 months) consisting of a minimum of 4 weeks of stall rest, 4 weeks of small paddock turnout, and 4 weeks pasture turnout	No complications associated with tibia stress fracture reported; catastrophic tibia fracture did not occur; 49 of 61 case horses with follow-up records returned to racing		7
Faillace V et al., 2017	Single case (University of Camerino, Italy)	Date not specified; case report received August 2016	1	Complete, comminuted tibia fracture	Internal fixation with a medial ten-hole neutralisation plate (eight screw stabilisation) and multiple lag screws; platelet rich plasma injected percutaneously at the fracture site 50 days postoperatively due to delayed callus formation	Delayed but successful fracture healing; case outcome qualified as excellent		32
Curtiss AL et al., 2019	Horses diagnosed with tibia fracture and treated by internal fixation (New Bolton Center, USA)	2008–2016	6	Tibia fracture	Open reduction internal fixation; further details not provided	No reported cases of surgical site infection (SSI); see 'Notes'	Retrospective study focused on surgical site infection associated with equine internal fixation; surgical details beyond the absence of SSI in tibia fracture cases	33

remodelling of the bone. Three risk factors for tibial fracture have been described based on epidemiologic analysis: age, race training and pre-existing injury (Figure 4). In a review of California race data, spanning 28 years of euthanasia performed because of tibial fracture, two- and three-year-old racehorses were significantly overrepresented and accounted for 73% of all tibial fractures during that time period.[8] Post hoc logistic regression modelling revealed that a greater proportion of two- and three-year-old racehorses were affected with tibial fracture compared with other musculoskeletal injuries. On the other hand, horses 4 years of age and older were significantly underrepresented in the tibial fracture population when compared with current California racehorse demographics.[8] A similar age distribution was noted in a retrospective analysis of tibial fracture cases diagnosed at Randwick Equine Centre between 1992 and 2000, with 71% of tibial fracture cases occurring in 2-year-old racehorses. This association remains true in recent epidemiological analyses based on racehorse data from the Hong Kong Jockey Club.[7,9,12,34]

Analysis of risk factors is important to development of prevention strategies to minimise or eliminate these injuries from occurring. A striking case report of bilateral tibial stress fracture in a racing Quarter Horse poses the question of association between race training and supra-physiological stresses on the equine tibia.[29] Tibial stress fractures are more commonly observed in racing populations and there are relatively few reports of non-racehorse tibial stress fracture diagnosis.[3,9,16,31] The occupational risk of tibia fatigue fracture is greatest in flat racing horses, but an interesting association between pre-injury time in training and distance worked should be considered. When compared with all California racehorses euthanised for a musculoskeletal reason, horses sustained tibial fractures more frequently during sub-maximal training than other musculoskeletal injuries which occurred when racing at maximum speeds.[8] When compared with matched control horses, California racehorses with tibial fractures had fewer official timed workouts, logged fewer active days, and accumulated less overall distance in workouts and events.[8] These findings were supported by analysis of records maintained by the Hong Kong Jockey Club, and a significant association between number of rest or layup days and incidence of tibial stress fracture was identified.[7,12] Another widely accepted risk factor for complete tibial fracture in a racing population is previous injury in the form of tibia stress remodelling. On gross necropsy of horses that sustained complete tibial fracture, the catastrophic fracture line was most commonly associated with, if not stemming from, an area of stress remodelling; this association was confirmed with computed tomographic and histopathologic analysis.[8]

A variety of additional risk factors associated with tibial fracture in racehorses have been debated in the literature including gender, trainer effect and running surface (Figure 4). According to Samol et al., females were overrepresented in the catastrophic tibial fracture group (51.4% of cases) compared with the proportion of females that were euthanised for other musculoskeletal injury.[8] However, the authors admit that this finding may be confounded by a skewed representation of stallions in the tibial fracture group and question the significance of gender as a risk factor. Of the large-scale retrospective epidemiologic studies, only Donati et al. found a significant association between female gender and trauma-related tibial fracture, and this was outside of a racing population.[7,9,12,16]

Two risk factors of racing surface and trainer effect have been proposed but are extensively affected by confounders such as geographic region, racing regulations, reporting discrepancies, socioeconomic status and other factors. In an analysis of racing surface and stress fracture incidence, MacKinnon et al. noted that hindlimb stress fractures were more commonly diagnosed after a switch from dirt to synthetic track surface.[35] However, when the cases were grouped by trainer, tibial stress fractures were more commonly seen in horses trained on dirt tracks as opposed to turf grass and synthetic racetrack surfaces. Trainer effect was skewed by the number of horses per trainer; tibial fractures were most commonly associated with trainers having the most horses enrolled in the study, and the authors could not draw any conclusions due to significant confounding.[35] Similar findings were reported by Verheyen et al., who noted that two of 13 trainers were associated with significantly higher risk of stress fracture compared with baseline on univariate analysis, but the overall univariable effect of trainer (with track surface held as a confounder) was not statistically significant.[11] Therefore, the overall relationship between hoof-ground interaction and development of tibial stress fracture remains unknown, and trainer effect, while a logical risk factor, has not been unequivocally proven in the literature.

9 | DIAGNOSTICS AND PREVENTION

Currently, no specific and targeted preventative measures exist for complete tibial fractures, so prevention focuses on an understanding of fracture aetiology, identification of risk factors, and early and accurate diagnosis. Prevention of tibia impact fractures hinges on herd management and housing.[16] Continuing education and extension programmes help to educate horse owners on the basics of equine psychology and herd hierarchy, equipping owners to make informed decisions to reduce the risk of kick injuries when introducing new horses into a herd.[36] Nevertheless, impact fractures are an inherent risk, and rapid diagnostics and treatment following impact are important.

The topic of fatigue fracture prevention in racehorses has been extensively debated in the scientific community, horse industries and public at large. In review of the current literature, three overarching goals of prevention can be elucidated: optimise diagnostic efficiency, reduce the interval between injury and diagnosis and optimise racing and training conditions to reduce non-specific fracture risk.

Historically, radiographic analysis of the equine tibia has been the gold standard for tibial fracture diagnosis. A five-view study consisting of standard lateromedial, caudocranial, caudolateral-craniomedial oblique and caudomedial-craniolateral oblique views plus an additional plantaro-15°-distal-85°-lateral dorsoproximal oblique is recommended to screen for macroscopic stress remodelling and/or incomplete fracture.[37] However, the sensitivity of radiographic examination alone is low for minimally displaced fractures and early stress

remodelling. Most recently, a radiographic processing algorithm for human medicine application has been described, which enables automated callus detection and quantification using published corrections for magnification, exposure and relative area in contact to the parent bone.[38] With species-specific correction and calibration, there is hope that similar technology may be applied to equine tibial radiographic screening and serial monitoring of stress reactions without invasive, expensive or imprecise measures.

With the advent of nuclear medicine, diagnostic nuclear scintigraphy has replaced radiography as the gold standard for tibia fatigue fracture diagnosis and monitoring.[8] At its core, nuclear scintigraphy requires only four elements: a radiopharmaceutical agent (technetium-99 m labelled diphosphonates for musculoskeletal diagnostics), the patient, a radiation detector (a gamma camera), and a method for processing and storing data.[39] Predetermined timing of images after radiopharmaceutical administration allows for physiologic analysis of multiple systems in phases.[39] Due to the bioavailability and function of diphosphonates, radionucleotide uptake will increase in areas of high bone turnover and remodelling, and a stress fracture will manifest as a focal area of increased radiopharmaceutical uptake (IRU). Comparison of age-standardised normal images to clinical diagnostic images allows for diagnosis and relative localisation of areas of stress remodelling or fractures.[7,31]

While regions of abnormal bone turnover can be identified on a single scintigraphic study, consistent correlation between the degree of IRU and pathologic severity has not been proven. Ramzan et al. explored the theory that a standardised grading scale, adapted from human radiologic literature, would correlate with the severity of lesions on necropsy.[5] A significant association between lesion site and scintigraphic localisation was reported, but, unlike in human nuclear scintigraphy, scintigraphic and radiographic grades were unreliable indicators of lesion severity.[5] In a multicentre, retrospective analysis of tibial stress fractures diagnosed with both nuclear scintigraphy and radiology, a different scintigraphic grading scale using quantitative IRU ratios was not a significantly accurate predictor of lesion severity.[40] Both studies emphasise the confounding factor of soft tissue coverage asymmetry between the proximal and distal tibia and the limitation of inconsistent/unknown time between injury and diagnostics; scintigraphy was unable to distinguish between acute trauma and ongoing remodelling over time.[5,40] Nevertheless, nuclear scintigraphy remains the gold standard for early detection of stress fractures in racehorses, and this early detection has reduced the incidence of catastrophic tibial fractures in the modern day.[8,9,12]

10 | CONCLUSION

Equine tibial fractures occur as a result of traumatic impact or fatigue failure. The incidence of traumatic tibial fracture is not well described in the literature but is logically influenced by risk factors such as management, herd dynamics and social hierarchy. Tibial stress fractures are often rated as the second most common stress fracture in racing Thoroughbreds. Young age, early stage in race training and initiation of training after a period of rest are reported to be risk factors for tibial stress fracture. Additional factors to consider include gender, training, running surface, and race direction. Both impact and fatigue fracture propagation are dependent on the local composition/alignment of mineralised collagen in the tibial lamellae and on the magnitude of force applied. Extensive research has characterised the pattern of strain distribution and stress remodelling within the equine tibia, but in vivo measurement of load and angular moments are currently not feasible in the equine model. Further research is warranted to correlate biomechanical theory of tibia fatigue fracture propagation through areas of chronic remodelling with current histopathological data.

Preventative measures for fatigue fractures align with three overarching goals: to optimise diagnostic efficiency, reduce the interval between injury and diagnosis and modify racing and training conditions to reduce non-specific fracture risk. The current gold standard for early tibial stress fracture diagnosis is nuclear scintigraphy, and the increased availability and utilisation of this imaging modality are credited with an overall reduction in catastrophic tibial fractures over time. Treatment options for complete tibial fractures in adult horses are limited. In smaller horses (breed, weight, age) with minimally comminuted fractures, successful outcomes have been reported after ORIF. Tibial stress fractures and minimally displaced incomplete fractures are typically treated conservatively with extended stall rest, exercise restriction for roughly 4–6 months and rehabilitation. Conservatively managed stress fractures have fair to good prognosis for recovery and return to racing with no reported difference in career length and earnings from matched controls.

AUTHOR CONTRIBUTIONS

K. Bowers prepared the draft manuscript. J. Weinhandl and D. Anderson were responsible for final approval of the manuscript.

ACKNOWLEDGEMENTS

None to declare.

PEER REVIEW

The peer review history for this article is available at https://publons.com/publon/10.1111/evj.13599.

DATA AVAILABILITY STATEMENT

Data sharing is not applicable to this article as no new data were created in this study.

ETHICS STATEMENT

Research ethics committee oversight not required by this journal: retrospective analysis of publicly available data.

ORCID

Kristin Bowers https://orcid.org/0000-0002-0536-5135

REFERENCES

1. Johnson BJ, Stover SM, Daft BM, Kinde H, Read DH, Barr BC, et al. Causes of death in racehorses over a 2 year period. Equine Vet J. 1994; 26(4):327–30. https://doi.org/10.1111/j.2042-3306.1994.tb04395.x

2. Estberg L, Stover SM, Gardner IA, Johnson BJ, Case JT, Ardans A, et al. Fatal musculoskeletal injuries incurred during racing and training in thoroughbreds. J Am Vet Med Assoc. 1996;208(1):92–6.
3. Watkins JP, Sampson SN. Fractures of the tibia. Equine Fracture Repair. Hoboken, NJ: Wiley-Blackwell; 2019. p. 648–63.
4. Levine DG, Aitken MR. Physeal fractures in foals. Vet Clin North Am Equine Pract. 2017;33(2):417–30. https://doi.org/10.1016/j.cveq.2017.03.008
5. Ramzan PHL, Newton JR, Shepherd MC, Head MJ. The application of a scintigraphic grading system to equine tibial stress fractures: 42 cases. Equine Vet J. 2010;35(4):382–8. https://doi.org/10.2746/042516403776014253
6. Ruggles AJ, Moore RM, Bertone AL, Schneider RK, Bailey MQ. Tibial stress fractures in racing standardbreds: 13 cases (1989-1993). J Am Vet Med Assoc. 1996;209(3):634–7.
7. O'Sullivan CB, Lumsden JM. Stress fractures of the tibia and humerus in thoroughbred racehorses: 99 cases (1992-2000). J Am Vet Med Assoc. 2003;222(4):491–8. https://doi.org/10.2460/javma.2003.222.491
8. Samol MA, Uzal FA, Hill AE, Arthur RM, Stover SM. Characteristics of complete tibial fractures in California racehorses. Equine Vet J. 2021;53(5):911–22. https://doi.org/10.1111/evj.13375
9. McGlinchey L, Hurley MJ, Riggs CM, Rosanowski SM. Description of the incidence, clinical presentation and outcome of proximal limb and pelvic fractures in Hong Kong racehorses during 2003-2014. Equine Vet J. 2017;49(6):789–94. https://doi.org/10.1111/evj.12696
10. Verheyen KLP, Wood JLN. Descriptive epidemiology of fractures occurring in British thoroughbred racehorses in training. Equine Vet J. 2010;36(2):167–73. https://doi.org/10.2746/0425164044868684
11. Verheyen KLP, Newton JR, Price JS, Wood JLN. A case-control study of factors associated with pelvic and tibial stress fractures in thoroughbred racehorses in training in the UK. Prev Vet Med. 2006;74(1):21–35. https://doi.org/10.1016/j.prevetmed.2006.01.004
12. Johnston AS, Sidhu ABS, Riggs CM, Verheyen KLP, Rosanowski SM. The effect of stress fracture occurring within the first 12 months of training on subsequent race performance in thoroughbreds in Hong Kong. Equine Vet J. 2021;53(3):460–8. https://doi.org/10.1111/evj.13324
13. Markel MD. Fracture biomechanics. Equine Fracture Repair. Hoboken, NJ: Wiley-Blackwell; 2019. p. 12–23.
14. Oswald S, Jäggin S, Piskoty G, Michel S, Auer JA, Fürst AE. Fracture configurations of the equine radius and tibia after a simulated kick. Vet Comp Orthop Traumatol. 2008;21(01):49–58. https://doi.org/10.3415/vcot-07-03-0022
15. Piskoty G, Jäggin S, Michel SA, Weisse B, Terrasi GP, Fürst A. Resistance of equine tibiae and radii to side impact loads. Equine Vet J. 2012;44(6):714–20. https://doi.org/10.1111/j.2042-3306.2012.00560.x
16. Donati B, Fürst AE, Hässig M, Jackson MA. Epidemiology of fractures: the role of kick injuries in equine fractures. Equine Vet J. 2018;50(5):580–6. https://doi.org/10.1111/evj.12819
17. Riggs CM, Vaughan L, Evans GP, Lanyon L, Boyde A. Mechanical implications of collagen fibre orientation in cortical bone of the equine radius. Anat Embryol. 1993;187(3):239–48.
18. Reznikov N, Chase H, Brumfeld V, Shahar R, Weiner S. The 3D structure of the collagen fibril network in human trabecular bone: relation to trabecular organization. Bone. 2015;71:189–95. https://doi.org/10.1016/j.bone.2014.10.017
19. Ferguson VL. Deformation partitioning provides insight into elastic, plastic, and viscous contributions to bone material behavior. J Mech Behav Biomed Mater. 2009;2(4):364–74. https://doi.org/10.1016/j.jmbbm.2009.01.004
20. Creel JA, Stover SM, Martin RB, Fyhrie DP, Hazelwood SJ, Gibeling JC. Compliance calibration for fracture testing of anisotropic biological materials. J Mech Behav Biomed Mater. 2009;2(5):571–8. https://doi.org/10.1016/j.jmbbm.2008.11.005
21. Evans FG, Vincentelli R. Relation of collagen fiber orientation to some mechanical properties of human cortical bone. J Biomech. 1969;2(1):63–71. https://doi.org/10.1016/0021-9290(69)90042-6
22. Halley SE, Bey MJ, Haladik JA, Lavagnino M, Arnoczky SP. Three dimensional, radiosteriometric analysis (RSA) of equine stifle kinematics and articular surface contact: a cadaveric study. Equine Vet J. 2014;46(3):364–9. https://doi.org/10.1111/evj.12127
23. Lanovaz JL, Khumsap S, Clayton HM, Stick JA, Brown J. Three-dimensional kinematics of the tarsal joint at the trot. Equine Vet J. 2010;34(S34):308–13. https://doi.org/10.1111/j.2042-3306.2002.tb05438.x
24. Schneider RK, Milne DW, Gabel AA, Groom JJ, Bramlage LR. Multidirectional in vivo strain analysis of the equine radius and tibia during dynamic loading with and without a cast. Am J Vet Res. 1982;43(9):1541–50.
25. Hartman W, Schamhardt HC, Lammertink JL, Badoux DM. Bone strain in the equine tibia: an in vivo strain gauge analysis. Am J Vet Res. 1984;45(5):880–4.
26. Schamhardt HC, Hartman W, Lammertink JLMA. In vivo bone strain in the equine tibia before and after transection of the peroneus tertius muscle. Res Vet Sci. 1985;39(2):139–44. https://doi.org/10.1016/S0034-5288(18)31736-3
27. Martig S, Chen W, Lee PVS, Whitton RC. Bone fatigue and its implications for injuries in racehorses. Equine Vet J. 2014;46(4):408–15. https://doi.org/10.1111/evj.12241
28. Fleck C, Eifler D. Deformation behaviour and damage accumulation of cortical bone specimens from the equine tibia under cyclic loading. J Biomech. 2003;36(2):179–89. https://doi.org/10.1016/S0021-9290(02)00364-0
29. Peloso JG, Watkins JP, Keele SR, Morris EL. Bilateral stress fractures of the tibia in a racing American quarter horse. J Am Vet Med Assoc. 1993;203(6):801–5.
30. Nunamaker DM, Butterweck DM, Provost MT. Fatigue fractures in thoroughbred racehorses: relationships with age, peak bone strain, and training. J Orthop Res. 1990;8(4):604–11. https://doi.org/10.1002/jor.1100080417
31. Pilsworth RC, Webbon PM. The use of radionuclide bone scanning in the diagnosis of tibial 'stress' fractures in the horse: a review of five cases. Equine Vet J. 1988;20:60–5. https://doi.org/10.1111/j.2042-3306.1988.tb04649.x
32. Faillace V, Tambella AM, Fratini M, Paggi E, Dini F, Laus F. Use of autologous platelet-rich plasma for a delayed consolidation of a tibial fracture in a young donkey. J Vet Med Sci. 2017;79(3):618–22. https://doi.org/10.1292/jvms.16-0400
33. Curtiss AL, Stefanovski D, Richardson DW. Surgical site infection associated with equine orthopedic internal fixation: 155 cases (2008–2016). Vet Surg. 2019;48(5):685–93. https://doi.org/10.1111/vsu.13216
34. Whitton RC, Walmsley EA, Wong ASM, Shannon SM, Frazer EJ, Williams NJ, et al. Associations between pre-injury racing history and tibial and humeral fractures in Australian thoroughbred racehorses. Vet J. 2019;247:44–9. https://doi.org/10.1016/j.tvjl.2019.03.001
35. Mackinnon MC, Bonder D, Boston RC, Ross MW. Analysis of stress fractures associated with lameness in thoroughbred flat racehorses training on different track surfaces undergoing nuclear scintigraphic examination. Equine Vet J. 2015;47(3):296–301. https://doi.org/10.1111/evj.12285
36. Keiper RR. Social Structure. Vet Clin North Am Equine Pract. 1986;2(3):465–84. https://doi.org/10.1016/S0749-0739(17)30701-0
37. Huguet EE, Porter EG, Chapman KA, Saunders FC, Davis B, Berry CR. Radiographic anatomy of the equine distal tibia. Vet Radiol Ultrasoun. 2020;61:497–506. https://doi.org/10.1111/vru.12875
38. Porter SM, Dailey HL, Hollar KA, Klein K, Harty JA, Lujan TJ. Automated measurement of fracture callus in radiographs using portable software. J Orthop Res. 2016;34(7):1224–33. https://doi.org/10.1002/jor.23146

39. Hoskinson JJ. Equine nuclear scintigraphy: indications, uses, and techniques. Vet Clin North Am Equine Pract. 2001;17(1):63–74. https://doi.org/10.1016/S0749-0739(17)30075-5
40. Valdés-Martínez A, Seiler G, Mai W, Bolt DM, Mudge M, Dukti SA, et al. Quantitative analysis of scintigraphic findings in tibial stress fractures in thoroughbred racehorses. Am J Vet Res. 2008;69(7):886–90. https://doi.org/10.2460/ajvr.69.7.886

How to cite this article: Bowers K, Weinhandl JT, Anderson DE. A review of equine tibial fractures. Equine Vet J. 2023;55(2):171–81. https://doi.org/10.1111/evj.13599