

A Thoroughbred racehorse with a unicortical palmar lateral condylar fracture returned to training 14 days after surgery: a hypothesis on the role of a single bone screw on crack propagation

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A 2-year-old Thoroughbred racehorse had LF lameness that began post high-speed exercise and persisted for two days before the horse once again became sound. Diagnostic analgesia localized the lameness to the LF distal metacarpus, and a standing MRI identified a unicortical condylar fracture. A single 5.5 mm cortical screw was placed in lag fashion. The horse began hand walking at 14 days, racetrack jogging at 30 days, and racing at 5 months after the day of surgery. Placement of a single lag screw ahead of the tip of the crack in unicortical condylar fracture may be useful for reducing the recovery period for horses returning to training and racing.

Key words: crack propagation, fracture, MRI

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A 2-year-old intact male Thoroughbred racehorse was evaluated for left forelimb lameness that began immediately post high-speed exercise. The horse had been in race training for approximately six months previously (Table 1) and had his first race of five furlongs (F) at a south Florida racetrack on June 10, 2016. Following this race, a high-speed exercise event (work) at a distance of four F created grade 2/5 left forelimb lameness that persisted for two days prior to the lameness resolving.

The horse was evaluated during the time that the lameness was present. All physical examination findings identified were normal other than grade 2/5 lameness in the left front limb. The left front fetlock (LFF) was cold to the touch,

there was no joint distension, the horse exhibited a negative response to fetlock flexion, but diagnostic analgesia of the lateral metacarpal nerve eliminated lameness in the left front limb.

Standard dorsopalmar, lateromedial, dorsolateral-palmaromedial oblique, and dorsomedial-palmarolateral oblique projections of the LFF were unable to visualize a fracture. The flexed dorsodisto-palmaroproximal oblique projection was the only useful projection, and it identified a subtle but suspicious line in the lateral parasagittal groove of the distal lateral MC3 bone (Fig. 1).

Given the horse's training and lameness history, diagnostic blocking pattern, and suspicious radiographic appearance, a standing MRI was performed on the LFF using a 0.27 Tesla standing MRI (Hallmarq Veterinary Imaging, Guildford, Surrey, U.K.). The MRI images (Fig. 2) were collected using standard orthogonal planes (sagittal, dorsal, and transverse) and accepted sequences (T1W gradient echo, T2*W gradient echo, and Short tau inversion recovery [STIR]). The T1W images identified an area of moderate signal decrease in the periphery and a hyperintense line that

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Table 1. Timeline of high speed exercise in furlongs

Date	Event
Jun 10, 2016	5 F race
Jun 23	4 F work–fracture
Jul 5	Standing MRI
Jul 6	Surgery
Jul 20	Walk
Aug 3	Jog
Aug 31	Canter
Sep 28	2 F work
Oct 27	3 F work
Nov 4	3 F work
Nov 12	3 F work
Nov 19	4 F work
Nov 26	5 F work
Dec 3	5 F work
Dec 10	6 F race finished 2nd
Dec 29	4 F work
Jan 5, 2017	6 F race finished 1st
Jan 18	4 F work
Jan 26	4 F work
Feb 2	4 F work
Feb 12	6.5 F race–3rd
Feb 22	4 F work
Mar 5	4 F work
Mar 11	7.5 F race–8th
Mar 27	5 F work
Apr 4	3 F work
Apr 9	6 F race finished 1st
May 24	Sesamoid fracture–horse retired

Chronology of speed exercise in furlongs (F) from first race, through surgery and recovery to first race after surgery until the (named Wait) horse was retired (www.equibase.com).

emanated from the palmar lateral parasagittal groove. The T2*W images identified a peripheral area of moderate signal increase that was surrounded by a black line created by a phase cancellation artifact. A hyperintense line emanated from the palmar lateral parasagittal groove, acknowledging that fracture lines may also be bright on fluid sensitive sequences like T2*W and STIR images if the fracture line contains fluid. The STIR images (Fig. 2) identified an area of marked signal increase that had accumulated in the axial aspect of the lateral condyle and the parasagittal groove. The MRI findings were consistent with a unicortical palmar lateral condylar fracture or grade 4 bone stress injury (BSI) of the distal palmar lateral MC3 when using the Fredrickson BSI classification scale [2, 13]. There was no evidence of injury to the proximal sesamoid bones.

Given the history, physical examination findings, diagnostic blocking pattern, and imaging findings from digital radiography and standing MRI, a diagnosis of incomplete non-displaced palmar lateral condylar fracture was made. Of the available frontal and transverse plane images, only the

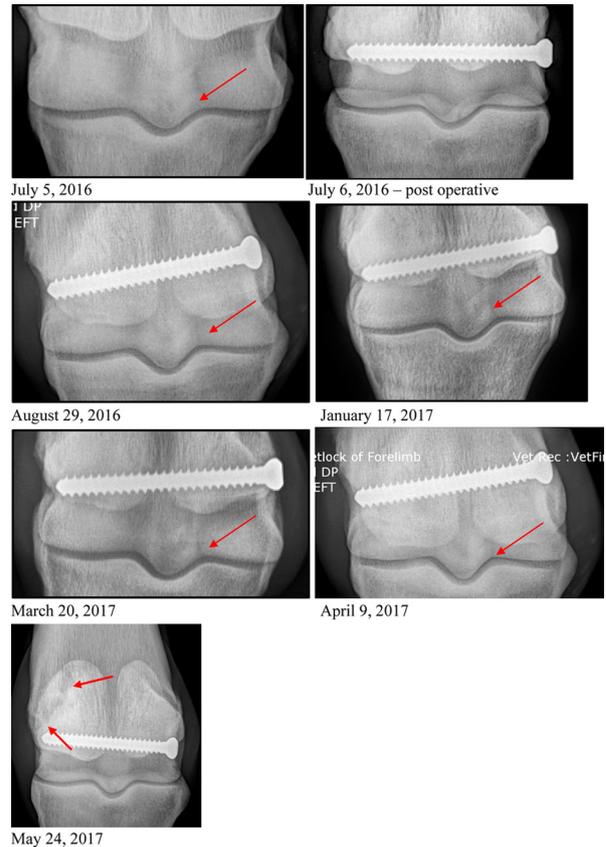


Fig. 1. Serial flexed dorsodisto-palmaroproximal projection of the left front fetlock taken over time. Initially, a suspicious line in the lateral parasagittal groove (top left–red arrow) was identified. The postoperative appearance of the LFF (top right) and the same projection of the fetlock was followed sequentially during the horse's racing career. The horse was ultimately retired due to an abaxial fracture of the medial sesamoid bone (red arrows in the radiograph at the bottom left).

most palmar and distal 2 slices identified the presence of the crack. Based on the location of the crack, it was evident that placing a single screw with a lateral to medial orientation in the supracondylar fossa would position the screw dorsal and proximal to the front of the crack. In preparation for surgery, the horse received preoperative antibiotics, phenylbutazone, and tetanus prophylaxis, was placed under general anesthesia, and positioned in right lateral recumbency. With the limb clipped, sterilely prepped, and draped, a single 5.5 mm lag screw was placed with a lateral to medial orientation in the lateral supracondylar fossa to compress the left front MC3 unicortical palmar lateral condyle fracture on July 6, 2016 using a previously described technique [24]. Currently, one of the authors (JGP) recommends doing this surgery in the standing patient.

The horse was confined to a stall for 14 days (Table 1), hand walked for 15 min twice a day while confined to the

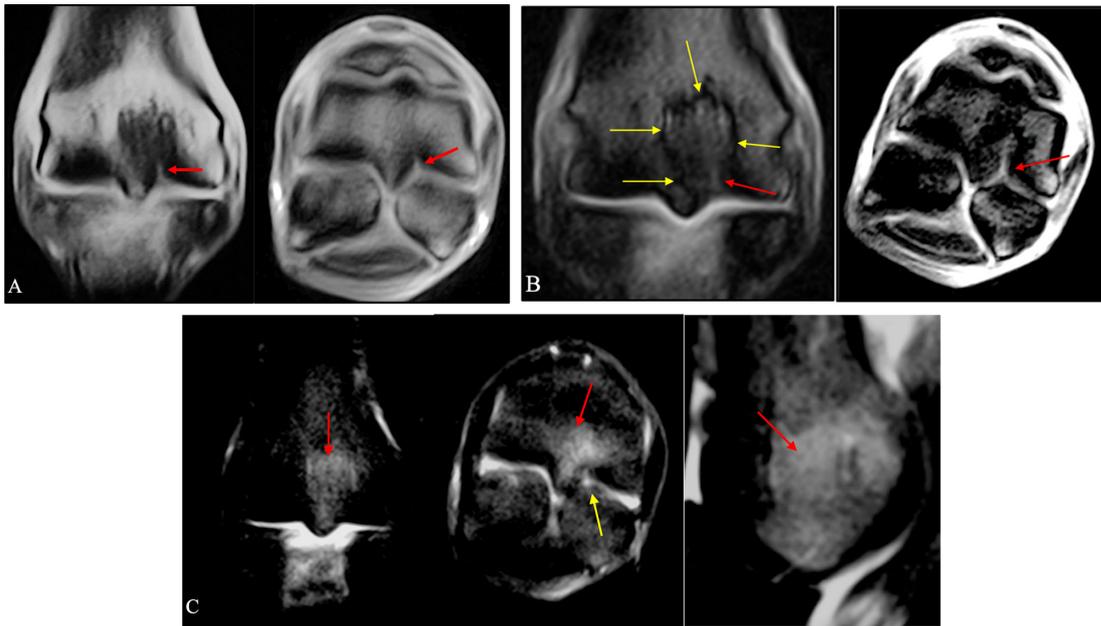


Fig. 2. Preoperative MRI images. (A) TIW GRE frontal plane (left) and transverse plane (right) MRI images of the left front fetlock. Images identify an area of moderate signal decrease in the periphery that is consistent with either bone densification or bone marrow edema. The red arrow identifies a hyperintense line that emanates from the lateral parasagittal groove and is consistent with a unicortical palmar lateral condylar fracture. (B) T2*W frontal plane (left) and transverse plane (right) MRI images of the left front fetlock. The images identify a peripheral area of moderate signal increase that is surrounded by a black line (yellow arrows) created by a phase cancellation artifact. The red arrow identifies a hyperintense line in the lateral parasagittal groove that is consistent with a lateral condylar fracture. (C) STIR FSE frontal plane (left), transverse plane (middle), and sagittal plane (right) MRI images of the left front fetlock. The generalized area of signal increase identified by the red arrows in the lateral parasagittal groove is consistent with bone marrow edema in the area of the developing fracture. The yellow arrow identifies a hyperintense line in the lateral parasagittal groove that is consistent with a palmar lateral condylar fracture.

stall for an additional 14 days, and then jogged with a rider up at the race track starting on August 3, 2016 for 30 days. The horse had his first 2 F high-speed event (work) on September 28, 82 days after the day of surgery. He had 6 more high-speed events (works) before his first race (Table 1), and on December 10, 2016, he finished second in a 6 F race just 157 days (5 months and 4 days) after the day of surgery. On January 5, 2017, the horse finished 1st by 5 1/2 lengths in a 6 F race just 183 days (5 months, 30 days) after the day of surgery. The horse continued to successfully train and competed in 3 additional races (Table 1). He fractured the abaxial aspect of the left front medial sesamoid bone on May 24, 2017, 322 days (10 months and 19 days) after surgery and was retired as a stallion on the owner's farm (Fig. 1). There was no evidence of injury to the LF medial sesamoid on evaluation of the original MRI images suggesting that this injury was new and unrelated to the early return to training and racing.

Fractures of metacarpal III/ metatarsal III (herein MC3/ MT3) are the most common type of fracture [5, 14] and the most common cause for euthanasia in Thoroughbred

racehorses in Europe [14]. In North America, it is the second most common cause for euthanasia [17–20] after biaxial proximal sesamoid bone fracture [6, 17]. In California, U.S.A., racehorse fatalities associated with Thoroughbred racing injure the jockey in 54% of horse spills [4]. Reducing the number and severity of condylar fractures of the fetlock is essential for the welfare of the jockey, the racehorse, the owner, and the Thoroughbred industry. Decreasing the time required to safely return horses to competition would reduce the economic hardship borne by owners associated with days lost to training and racing, improve the efficient use of resources for trainers and racetrack owners, and enhance the public's perception of the Thoroughbred industry.

The clinical signs of an MC3 / MT3 condylar fracture are variable and depend on the stage of the BSI within the pathologic continuum [21, 23, 27]. Occasionally mild lameness can precede more severe lameness, however, severe lameness following a race or high-speed exercise is often pathognomonic for a BSI of the condyle of the MC3 / MT3 [21, 23, 27].

With unicortical condylar fractures, the fetlock is often

cold to the touch, joint distension is absent, horses are negative to fetlock flexion, and they do not respond to intra-articular fetlock anesthesia [21, 23, 27]. Because the fracture is initiated within the interior of the MC3 / MT3 epiphysis (18), clinical signs like synovial effusion of the fetlock joint are often absent [21, 23, 27]. In essence, the lesion is extra-articular to the fetlock joint. The absence of localizing physical examination findings and the resolution of lameness within days of onset makes identification of a unicortical condylar fracture a diagnostic challenge [21, 23, 27] and provide the opportunity for the serious nature of this condition to be missed or overlooked [23]. Unicortical lateral condylar fractures with minimal joint damage and degenerative changes have an excellent prognosis, with greater than 90% of horses returning to racing. This statistic demonstrates that early treatment results in a more favorable prognosis [5, 14, 23, 24]. Horses with complete but nondisplaced lateral condylar fractures that are repaired surgically are typically on stall rest with controlled exercise for two months with some turnout in the third month and return to regular exercise in the fourth to fifth month post surgery [23, 24].

Complete bicortical condylar fractures are a progression of a BSI from the unicortical condylar fracture stage. Damage to the subchondral bone on the distopalmar aspect of the MC3 / MT3 is a common biomechanical disorder in the racing Thoroughbred that is initiated by the cyclic high loads of the gallop gait as the proximal sesamoid bones of the suspensory apparatus oppose the ground ward movement of the distal MC3 (MT3) [21, 22]. Bone can fail monotonically as a result of a single load that exceeds its failure stress, a rare event in the racing Thoroughbred, or gradually as a result of a repetitive submaximal load that creates fatigue [1, 3, 7, 8, 11, 12, 15, 16, 26, 29, 31–35]. The microcracks created by fatigue are physiologic and stimulate remodelling that leads to their removal with sufficient rest. With continued loading and insufficient rest, fatigue damage accumulates, remodelling is overwhelmed, and an overuse injury to bone, known as BSI, is created [3, 7, 15, 18, 32–35]. BSI progresses as a pathologic continuum and begins as a stress reaction with bone marrow edema being the distinguishing feature at the cellular level [18, 21–23], progresses to a stress fracture with a macroscopic fracture being the distinguishing feature, and then progresses to a gross fracture that is radiographically apparent as a complete fracture [3, 7, 8, 15, 32–35]. An unidentified BSI of the MC3 / MT3 that propagates during training or racing and produces a stable fracture can be repaired. A BSI that propagates and produces an unstable (catastrophic) fracture usually requires the horse to be euthanized (6, 17) and often injures the jockey [5].

There are subtle clinical signs associated with these

prodromal injuries, and progression from a unicortical to complete bicortical condylar fractures can take weeks to months [21, 23]. With bicortical condylar fractures, clinical signs include lameness, synovial effusion, subperiosteal and periarticular swelling, and heat and pain on flexion of the fetlock joint [5, 14, 21, 23, 24, 27, 35]. Horses with displaced fractures require longer periods of rest, and their prognosis for returning to performance is less favorable [5, 14, 24, 35], demonstrating the prognostic benefit of identifying a condylar fracture at the unicortical condylar fracture stage before it has progressed to a bicortical condylar fracture. The average period from surgery to the first race for horses with a surgically repaired bicortical condylar fractures is about 11 months [24]. In a case series that dealt exclusively with unicortical condylar fractures [23], the majority of patients were treated conservatively with variable periods of stable confinement and horse walker exercise. The most common (17/45 horses) rehabilitation schedule include a total of 8 weeks stable confinement/horse walker exercise usually comprising an initial 2–4 weeks of total stable confinement followed by stable confinement with 30–90 min of daily horse walker exercise. The horse in this case report was returned to hand walking at 14 days, rider up and jogging at the racetrack at 30 days, had 6 high-speed events (works), and raced at 5 months and 4 days after the day of surgery. This is half the time typically required for a bicortical condylar fracture, and a more aggressive schedule when compared to the case series of unicortical condylar fractures, recognizing that few horses in this series were treated with a lag screw (5/45 horses) [23, 35].

The use of MRI in equine practice has been limited in the past due to availability, financial constraints, time, and the health risks associated with both recovery from general anesthesia and recovery of an orthopedic injury from general anesthesia [18, 21–23, 30]. Recently, the use of standing MRI (sMRI) has become more frequent for the detection of condylar fractures because only MRI can identify bone marrow edema, which represents evidence of a BSI on a cellular level and the earliest stage that a developing fracture can be identified using advanced 3-D imaging [2, 3, 7, 8, 13–16, 18, 21–23, 30].

Approximately 25% of the weight of bone is collagenous fibers that provide tension strength and stiffness, and 50% of the weight of bone is rigid crystals of calcium that reinforce the collagen fibers and provide compression strength and stiffness [11, 29, 31]. Bone adapts to training (Wolff's law) by increasing the portion of rigid crystals of calcium (mineral density). A 5–8% increase in bone mineral density (BMD) can improve the toughness of bone by over 60% [32] but also increases its brittleness [29]. A 2003 report identified that equine compact bone that is stiff and strong in compression is also associated with brittle post-yield

behavior, a material property that is similarly identified in ceramics [1, 10, 12, 26].

The majority of condylar fractures are parasagittal in orientation [5, 24, 35], and begin as unicortical fracture in the palmar (plantar) aspect of the lateral (medial) parasagittal groove of the distal condyle [18, 23], and propagate in a proximal dorsal direction towards the dorsal cortex of the MC3/ MT3 to become bicortical fracture [5, 18, 21, 22, 24, 25, 35]. There are three ways a force is applied to a material to permit a crack to propagate [1, 11, 26, 29, 31]. A mode 1 (opening mode) force is the most common type and creates a tensile force applied perpendicular to the plane of the crack. In the galloping Thoroughbred, there is evidence that a transverse tensile force (mode 1 or opening force) acts at the distal palmar aspect of the MC3 between the condyles and the sagittal ridge due to a lateromedial deformation caused during loading [28]. This force and the remodelling process stimulate the trabecular epiphyseal bone to nucleate. In theory, negating this lateromedial tensile (opening mode 1) force could help eliminate propagation of the crack and ultimately fracture formation [1, 26]. Recognizing that nucleation and remodelling are normal events as bones adapt (Wolff's law), managing microcracks and macrocracks that may propagate is a rational way to manage the pathologic continuum of a BSI.

Cracks propagate due to mutual competition between the intrinsic components ahead of the tip of the crack and extrinsic components behind the tip of the crack (crack wake) [1, 26]. Extrinsic toughening mechanisms in the crack wake are more important in materials with little or no ductility like ceramics and bone [1, 26]. The role of a metal screw in changing the material properties of bone and its impact on the tip of the crack, the wake of the crack, propagation of the crack, and primary bone healing requires further investigation in a materials testing machine [1, 9, 11, 12, 26, 29, 31].

In isolation, neither a standing MRI nor lag screw fixation of a condylar fracture is novel. Rather, the combination of identifying either a BSI or a condylar fracture when it is small by using the standing MRI, and the impact of a single steel screw on the propagation of the crack make this case report novel. The term unicortical condylar fracture was only recently described [23], and it is a term that is appropriate when using 3 dimensional standing MRI images (Hallmarq Veterinary Imaging, Guilford, Surrey, U.K.). Recognizing that MRI is the only imaging modality that can identify bone marrow edema, MRI provides the earliest opportunity to identify a BSI [3, 7, 8, 15, 32–34]. The theory that the single screw described in this case report affected fracture propagation requires further study using a materials testing machine. This case indicates only that placement of a single lag screw ahead of the tip of the crack in unicortical

condylar fractures may be useful for reducing the recovery period when returning horses to training and racing. It is intuitive that the earliest possible diagnosis and treatment of a small developing condylar fracture would facilitate a shorter recovery period, a better prognosis, fewer euthanized horses, reduced risk to jockeys from catastrophic fractures, and a better return on the financial investment of owners.

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References

1. Acevedo, C., Stadelmann, V.A., Pioletti, D.P., Tamara Alliston, T., and Ritchie, R.O. 2018. Fatigue as the missing link between bone fragility and fracture. *Nat. Biomed. Eng.* **2**: 62–71. [[CrossRef](#)]
2. Fredericson, M., Bergman, A.G., Hoffman, K.L., and Dillingham, M.S. 1995. Tibial stress reaction in runners. Correlation of clinical symptoms and scintigraphy with a new magnetic resonance imaging grading system. *Am. J. Sports Med.* **23**: 472–481. [[Medline](#)] [[CrossRef](#)]
3. Harrast, M.A., and Colonno, D. 2010. Stress fractures in runners. *Clin. Sports Med.* **29**: 399–416. [[Medline](#)] [[CrossRef](#)]
4. Hitchens, P.L., Hill, A.E., and Stover, S.M. 2016. The role of catastrophic injury or sudden death of the horse in race-day jockey falls and injuries in California, 2007–2012. *Equine Vet. J.* **48**: 50–56. [[Medline](#)] [[CrossRef](#)]
5. Jacklin, B.D., and Wright, I.M. 2012. Frequency distributions of 174 fractures of the distal condyles of the third metacarpal and metatarsal bones in 167 Thoroughbred racehorses (1999–2009). *Equine Vet. J.* **44**: 707–713. [[Medline](#)] [[CrossRef](#)]
6. Johnson, B.J., Stover, S.M., Daft, B.M., Kinde, H., Read, D.H., Barr, B.C., Anderson, M., Moore, J., Woods, L., Stoltz, J., and Blanchard, P. 1994. Causes of death in racehorses over a 2 year period. *Equine Vet. J.* **26**: 327–330. [[Medline](#)] [[CrossRef](#)]
7. Kahanov, L., Eberman, L.E., Games, K.E., and Wasik, M. 2015. Diagnosis, treatment, and rehabilitation of stress fractures in the lower extremity in runners. *Open Access J. Sports Med.* **6**: 87–95. [[Medline](#)] [[CrossRef](#)]
8. Kiuru, M.J., Pihlajamäki, H.K., and Ahovuo, J.A. 2004. Bone stress injuries. *Acta Radiol.* **45**: 317–326. [[Medline](#)] [[CrossRef](#)]
9. Kurtz, S.M., Kocagöz, S., Arnholt, C., Huet, R., Ueno, M., and Walter, W.L. 2014. Advances in zirconia toughened alumina biomaterials for total joint replacement. *J. Mech. Behav. Biomed. Mater.* **31**: 107–116. [[Medline](#)] [[CrossRef](#)]

10. Les, C.M., Stover, S.M., Keyak, J.H., Taylor, K.T., and Kaneps, A.J. 2002. Stiff and strong compressive properties are associated with brittle post-yield behavior in equine compact bone material. *J. Orthop. Res.* **20**: 607–614. [[Medline](#)] [[CrossRef](#)]
11. Martin, R.B., Burr, D.B., Sharkey, N.A., and Fyhrie, D.P. 2015. Fatigue and fracture resistance of bone. pp. 423–482. *In: Skeletal Tissue Mechanics*, Springer, New York.
12. Naebe, M., Abolhasani, M.M., Khayyam, H., Amini, A., and Fox, B. 2016. Crack damage in polymers and composites: a review. *Polym. Rev. (Phila. Pa.)* **56**: 31–69. [[CrossRef](#)]
13. Nattiv, A., Kennedy, G., Barrack, M.T., Abdelkerim, A., Goolsby, M.A., Arends, J.C., and Seeger, L.L. 2013. Correlation of MRI grading of bone stress injuries with clinical risk factors and return to play: a 5-year prospective study in collegiate track and field athletes. *Am. J. Sports Med.* **41**: 1930–1941. [[Medline](#)] [[CrossRef](#)]
14. Parkin, T.D. 2008. Epidemiology of racetrack injuries in racehorses. *Vet. Clin. North Am. Equine Pract.* **24**: 1–19. [[Medline](#)] [[CrossRef](#)]
15. Pathria, M.N., Chung, C.B., and Resnick, D.L. 2016. Acute and stress-related injuries of bone and cartilage: pertinent anatomy, basic biomechanics, and imaging perspective. *Radiology* **280**: 21–38. [[Medline](#)] [[CrossRef](#)]
16. Pegrum, J., Crisp, T., and Padhiar, N. 2012. Diagnosis and management of bone stress injuries of the lower limb in athletes. *BMJ* **344**: e2511. [[Medline](#)] [[CrossRef](#)]
17. Peloso, J.G., Mundy, G.D., and Cohen, N.D. 1994. Prevalence of, and factors associated with, musculoskeletal racing injuries of thoroughbreds. *J. Am. Vet. Med. Assoc.* **204**: 620–626. [[Medline](#)]
18. Peloso, J.G., Cohen, N.D., Vogler, J.B. 3rd., Marquis, P., and Hilt, L. 2019. Association of catastrophic condylar fracture with bony changes of the third metacarpal bone identified by use of standing magnetic resonance imaging in forelimbs from cadavers of Thoroughbred racehorses in the United States. *Am. J. Vet. Res.* **80**: 178–188. [[Medline](#)]
19. Pool, R.R., and Meagher, D.M. 1990. Pathologic findings and pathogenesis of racetrack injuries. *Vet. Clin. North Am. Equine Pract.* **6**: 1–30. [[Medline](#)] [[CrossRef](#)]
20. Pool, R.R. 1996. Pathologic manifestations of joint disease in the athletic horse. pp. 87–104. *In: Joint Disease in the Horse*, 1st ed. (McIlwraith C.W., and Trotter, G.W. eds.), W. B. Saunders, Philadelphia.
21. Powell, S.E., Ramzan, P.H., Head, M.J., Shepherd, M.C., Baldwin, G.I., and Steven, W.N. 2010. Standing magnetic resonance imaging detection of bone marrow oedema-type signal pattern associated with subcarpal pain in 8 racehorses: a prospective study. *Equine Vet. J.* **42**: 10–17. [[Medline](#)] [[CrossRef](#)]
22. Powell, S.E. 2012. Low-field standing magnetic resonance imaging findings of the metacarpo/metatarsophalangeal joint of racing Thoroughbreds with lameness localised to the region: a retrospective study of 131 horses. *Equine Vet. J.* **44**: 169–177. [[Medline](#)] [[CrossRef](#)]
23. Ramzan, P.H., Palmer, L., and Powell, S.E. 2015. Unicortical condylar fracture of the Thoroughbred fetlock: 45 cases (2006–2013). *Equine Vet. J.* **47**: 680–683. [[Medline](#)] [[CrossRef](#)]
24. Richardson, D.W. 2006. Third metacarpal and metatarsal bones. pp. 1325–1338. *In: Equine Surgery*, 4th ed. (Auer, J.A., and Stick, J.A. eds.), Saunders Elsevier, St. Louis.
25. Riggs, C.M., Whitehouse, G.H., and Boyde, A. 1999. Pathology of the distal condyles of the third metacarpal and third metatarsal bones of the horse. *Equine Vet. J.* **31**: 140–148. [[Medline](#)] [[CrossRef](#)]
26. Ritchie, R. 1999. Mechanisms of fatigue-crack propagation in ductile and brittle solids. *Int. J. Fract.* **100**: 55–83. [[CrossRef](#)]
27. Ross, M.W. 2005. Bone scintigraphy. Lessons learned from 5000 bone scans. *Proc. Annu. AAEP* **51**: 6–21.
28. Rubio-Martínez, L.M., Cruz, A.M., Inglis, D., and Hurtig, M.B. 2010. Analysis of the subchondral microarchitecture of the distopalmar aspect of the third metacarpal bone in racing Thoroughbreds. *Am. J. Vet. Res.* **71**: 1148–1153. [[Medline](#)] [[CrossRef](#)]
29. Seref-Ferlengez, Z., Kennedy, O.D., and Schaffler, M.B. 2015. Bone microdamage, remodeling and bone fragility: how much damage is too much damage? *Bonekey Rep.* **4**: 644. [[Medline](#)] [[CrossRef](#)]
30. Tapprest, J., Audigie, F., Radier, C., Anglade, M.C., Voisin, M.C., Foucher, N., Collobert-Laugier, C., Mathieu, D., and Denoix, J.M. 2003. Magnetic resonance imaging for the diagnosis of stress fractures in a horse. *Vet. Radiol. Ultrasound* **44**: 438–442. [[Medline](#)] [[CrossRef](#)]
31. Turner, C.H. 2006. Bone strength: current concepts. *Ann. N. Y. Acad. Sci.* **1068**: 429–446. [[Medline](#)] [[CrossRef](#)]
32. Warden, S.J., Hurst, J.A., Sanders, M.S., Turner, C.H., Burr, D.B., and Li, J. 2005. Bone adaptation to a mechanical loading program significantly increases skeletal fatigue resistance. *J. Bone Miner. Res.* **20**: 809–816. [[Medline](#)] [[CrossRef](#)]
33. Warden, S.J., Davis, I.S., and Fredericson, M. 2014. Management and prevention of bone stress injuries in long-distance runners. *J. Orthop. Sports Phys. Ther.* **44**: 749–765. [[Medline](#)] [[CrossRef](#)]
34. Wright, A.A., Hegedus, E.J., Lenchik, L., Kuhn, K.J., Santiago, L., and Smoliga, J.M. 2016. Diagnostic accuracy of various imaging modalities for suspected lower extremity stress fractures: a systematic review with evidence-based recommendations for clinical practice. *Am. J. Sports Med.* **44**: 255–263. [[Medline](#)] [[CrossRef](#)]
35. Zekas, L.J., Bramlage, L.R., Embertson, R.M., and Hance, S.R. 1999. Characterisation of the type and location of fractures of the third metacarpal/metatarsal condyles in 135 horses in central Kentucky (1986–1994). *Equine Vet. J.* **31**: 304–308. [[Medline](#)] [[CrossRef](#)]