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Pediatric Bone Health Update

The importance of bone health for pediatric athletes: From juvenile osteochondritis dissecans to relative energy deficiency in sports

Louise Atadja, MD[1]; Jennifer Beck, MD[2]; Corinna Franklin, MD[1,*]

[1] Yale University School of Medicine, 47 College St, New Haven, CT, USA
[2] Boulder Medical Center, Boulder, CO, USA

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ABSTRACT

Vitamin D is pivotal for bone health, muscle strength, recovery, and overall athletic performance. This article discusses the effects of vitamin D deficiency among athletes, with a specific focus on its connection to sports-related conditions like Relative Energy Deficiency in Sport (RED-S) and Juvenile Osteochondritis Dissecans (JOCD). Additionally, we review the importance of vitamin D for muscle strength and muscle repair. Vitamin D deficiency exacerbates the risk of stress fractures in RED-S patients due to its impact on bone biology. Research points to an increased incidence of vitamin D deficiency in JOCD patients, indicating a potential connection between the condition and low vitamin D levels. Children and athletes should be screened for vitamin D deficiency as supplementation has been found to have positive outcomes. This article underscores the pivotal role of vitamin D in athletic performance. Further research is essential to elucidate how vitamin D deficiency contributes to sports-related pathologies and to establish the optimal vitamin D levels for injury prevention in athletes.

Introduction

Early reports of vitamin D deficiency were first described in the mid-1600s when physicians in Northern Europe began reporting on children living in industrialized cities with severe deformities of the skeleton and growth delay [1]. Bone mineralization is dependent upon vitamin D through its vital role in the regulation of calcium and skeletal homeostasis [2,3]. The best method of measuring vitamin D status is by measuring serum levels of 25(OH)D [1,2,4]. The American Academy of Pediatrics defines vitamin D deficiency as 25 (OH)D levels of < 20 ng/mL in children and adults and insufficiency as levels of 20 to 30 ng/mL [4,5].

Vitamin D deficiency in sports has been well-studied in the literature. A meta-analysis found that winter season sports, indoor sports, and living at higher latitudes increased the risk of vitamin D deficiency in athletes [6]. A substantial amount of the literature focuses on the influence of vitamin D deficiency and increased risk of stress fracture in athletes [7]. Miller et al. over a 3-year period found that 83% of the patients with imaging-confirmed stress fractures had deficiency levels of vitamin D [8]. Another study looked at a population of National Collegiate Athletic Association collegiate athletes and found a protective association against serum vitamin D levels and stress fractures [9,10]. In a national survey of cross-country runners across the nation, female runners showed a 2-times greater risk of stress fracture risk when compared to male counterparts [11]. The authors of this study believed that male subjects were more likely to get stress fractures based on overuse and participation in sports, whereas female patients were at greater risk due to bone composition from lack of nutritional support with supplements such as vitamin D [11]. However, in recent years, the literature on the role of vitamin D has grown beyond stress fracture management and includes its metabolic role in muscle repair and recovery and other pathologies such as Relative Energy Deficiency in Sport (RED-S) and Juvenile Osteochondritis Dissecans (JOCD).

Vitamin D in muscle strength and muscle repair

Vitamin D has been shown to be associated with muscle functional strength, repair, and recovery. Vitamin D receptors have been identified in many tissues, including skeletal muscle [12,13]. A study of healthy postmenarche girls showed vitamin D to be significantly associated with muscle strength [14]. This study showed serum 25(OH)D

* Corresponding author: Yale University School of Medicine, 47 College St, New Haven, CT 06510, USA.
E-mail address: corinna.franklin@yale.edu (C. Franklin).

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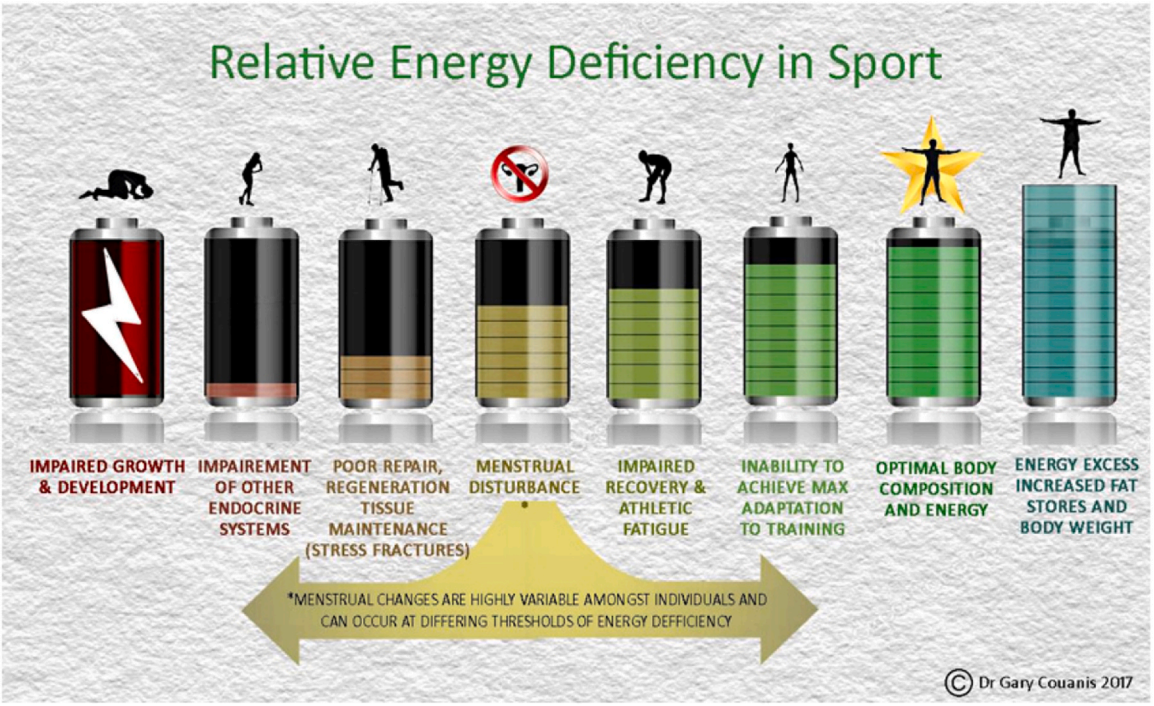
concentrations to be positively related to muscle power, force, velocity, and jump height [14]. One systematic review on the effect of vitamin D supplementation on muscle strength found significant improvement in proximal muscle, calf, and grip strength in patients with vitamin D deficiency [12]. Vitamin D supplementation has been shown to improve trabecular bone mineral density and muscle power [15]. Gilsanz et al. looked at muscle lipid content as a measure of muscle strength and found that low amounts of 25-(OH)D were significantly associated with increased fatty muscle infiltration in healthy young women [16].

Vitamin D has also been shown to affect muscle repair after injury, especially in athletes [12,13,17]. Pharmacological studies demonstrate vitamin D to be inversely correlated with lactic acid, creatine kinase, and total antioxidants after exercise. Higher serum levels of vitamin D have been associated with reduced injury rates and improved sports performance. Histopathological studies have shown muscle atrophy of type 2 fibers is associated with vitamin D deficiency [12]. Vitamin D deficiency contributes to muscle wasting and proximal muscle weakness by promoting oxidative stress which favors the production of free radicals within muscle fibers [13]. A recent randomized control trial of 20 healthy young men with low levels of 25(OH)D showed improved functional recovery from eccentric exercise when supplemented with vitamin D [13].

Relative energy deficiency in sport



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The understanding of RED-S, previously known as the Female Athlete Triad, has greatly changed over the past few years. Previously, RED-S was selectively studied in female high-level athletes with disordered eating habits who were at risk of osteopenia of amenorrhea [18]. The definition has since changed and in 2014, the International Olympic Committee changed the term “Female Athlete Triad” to RED-S to describe low energy availability with or without disordered eating patterns, amenorrhea, and low bone mineral density [19]. The International Olympic Committee (IOC) recently came out with an updated consensus statement on RED-S in September 2023 [20]. In this update, the IOC defined specifically the different phases of energy availability, ranging from low energy availability and adaptable energy availability to RED-S to describe the syndrome of collective physiological and psychological effects of prolonged or severe exposure to low energy availability [20].

The underlying etiology of RED-S results when energy expenditure exceeds energy intake leading to a decrease in energy availability [21]. Athletes with RED-S have insufficient energy to support functional physiological performance let alone allow them to compete at high-intensity levels. RED-S affects several systems, including metabolic rate, menstrual function, bone health, immunity, protein synthesis, and cardiovascular health [18]. These health irregularities can lead to impaired training tolerance, increased risk of injury, and compromised sports performance [22,23]. In para-athletes, problematic low-energy availability may lead to impaired bone health and a higher rate of musculoskeletal injury due to the increased risk associated with altered skeletal loading in these athletes [20].

Female athletes with RED-S have also been shown to have an increased risk for stress fractures [24]. Barrack et al. showed a 30% to 50% increase in bone stress injury with RED-S associated symptoms and variables [25]. Stress fractures can develop due to repetitive overloading of the extremity when normal bone biology is altered due to deficiencies in essential bone building blocks like vitamin D in RED-S [26]. One study found that among female distance runners, 63% of the athletes had low energy availability and 45% had impaired bone health as diagnosed on bone dual-energy x-ray absorptiometry [27]. It is well-studied in the literature that adolescent girls are also at a higher risk of RED-S [28]. A 2019 study of 390 athletes showed a higher risk of stress fractures in teenage athletes aged 16 to 17 compared to athletes in their 20s [28]. The 2023 IOC consensus statement introduced an updated screening tool with 4 category risk stratifications to help identify the risk and/or severity of RED-S along with sport participation guidelines for each level (Table 1) [20]. These preventative measures may be

Table 1
International Olympic Committee consensus indicators of RED-S [20].

Severe primary indicators	Primary indicators	Secondary indicators
Primary Amenorrhea (failure to menstruate by age 15 in the presence of normal secondary sexual development (2 SD above the mean of 13 years), or within 5 years after breast development if occurring before age 10	Secondary Amenorrhea (the absence of 3-11 consecutive menstrual cycles) caused by Functional Hypothalamic Amenorrhea (FHA)	Oligomenorrhoea caused by FHA (> 35 days between periods for a maximum of 8 periods/year)
Prolonged Secondary Amenorrhea (absence of 12 or more consecutive menstrual cycles due to FHA.	Sub-clinically low total or free testosterone (males: within the lowest 25% (quartile) of the reference range)	History of 1 low-risk BSI (Bone Stress Injury) within the previous 2 years and absence of < 6 months from training due to BSI in the previous 2 years
Clinically low free or total testosterone	Sub-clinically or clinically low total or free T3 (within or below the lowest 25% (quartile) of the reference range) History of > 1 high-risk (femoral neck, sacrum, pelvis) or > 2 low-risk BSI (all other BSI locations) within the previous 2 years or absence of > 6 months from training due to BSI in the previous 2 years Pre-menopausal children/adolescents: BMD Z-score* < −1 at the lumbar spine, or TBLH or decrease in BMD Z-score from prior testing (can occur from bone loss or inadequate bone accrual) A negative deviation of pediatric or adolescent athlete's previous growth trajectory (height and/or weight) An elevated score for the EDE-Q global > 2.30 in females; > 1.68 in males) and/or clinically diagnosed eating disorder	Elevated total or LDL cholesterol (above reference range) Clinically diagnosed depression and/or anxiety (only one secondary indicator for either or both outcomes)

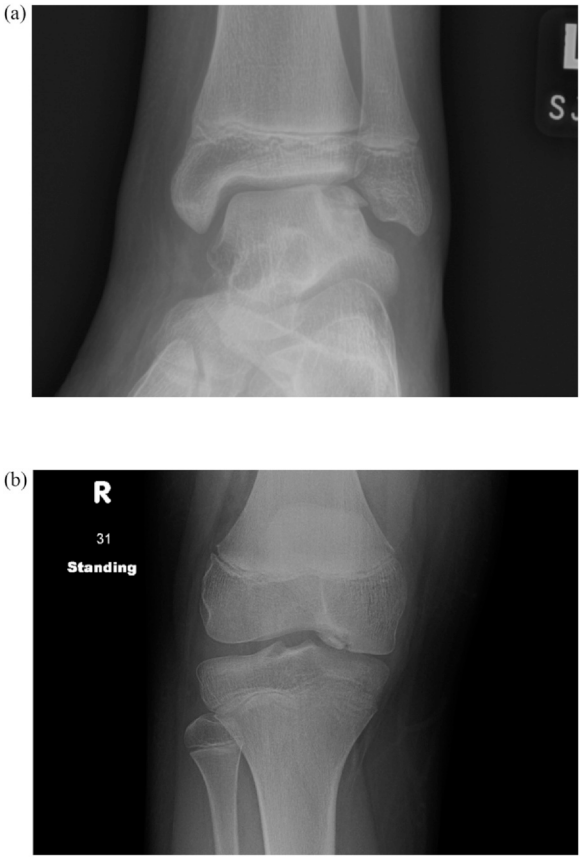

Figure 1. a) JOCD of the talus; b) JOCD of the knee.

useful for optimizing performance in athletes while reducing the risk of injury.

In RED-S, deficient nutritional intake leads to changes in hormone levels like GnRH and estrogen which may cause abnormalities in menstruation cycles [18,19,29]. Low estrogen also leads to decreased osteoblastic and increased osteoclastic levels creating an environment ripe for lower bone density [29]. One study looked at the dose-response relationship between bone turnover and energy availability and found a disturbance in the cycle of bone resorption and formation caused by severely deficient energy availability [30]. Even in situations of short-term low energy availability, studies show a decrease in markers of bone formation likely caused by the deficiency in vital nutrients necessary for the cycle of resorption and formation [31,32]. Male athletes are also at risk of RED-S. Those at risk include cyclists, rowers, runners, jockeys, and athletes in weight-class combat sports requiring cyclic changes in body mass and composition [33]. Low energy availability in male athletes is associated with decreased testosterone levels, decreased bone density, and decreased resting metabolic rate [34]. A recent study in animal models found that male mice who were subject to food restriction and access to an exercise wheel showed low bone mineral density and lower levels of osteoblastogenesis akin to patients with RED-S [35]. When these mice were given treatment with active vitamin D analogs, they found significant restoration of osteoblastic activities and improvement in bone mineral density [35]. Regular supplementation of vitamin D may have a positive effect on adolescent athletes, especially those at higher risk of RED-S, and may prevent future injury.

Juvenile osteochondritis dissecans

JOCD is a joint disorder of the subchondral bone and articular cartilage in young and active patients [36–39]. JOCD lesions form in subarticular epiphyseal cartilage and subchondral bone [36–38]. Trauma, whether in the acute setting or through micro-repetitive trauma, has generally been agreed upon in the literature as a major cause of JOCD [39–43]. However, the exact etiology of JOCD is largely unknown and may be multifactorial and include other factors such as inflammation, ischemia, vascular abnormalities, and genetic factors [36,42,44,45]. The clinical presentation of JOCD is highly variable, with some children having no symptoms at all [39]. The diagnosis of JOCD is dependent on imaging as other physical exam maneuvers have shown to be less sensitive for diagnosis in patients with radiographically proven OCD lesions [46]. Magnetic Resonance Imaging may be useful to distinguish between irregular lesion types and provide

information on size, character of lesion, stability on T2 sequences, and overall health of the articular surface [36,39,47]. Reported treatment strategies for JOCD vary widely in the literature, but surgery is usually indicated for patients with unstable JOCD lesions and lesions that fail non-operative intervention [36]. In stable lesions, some surgeons prefer to use arthroscopic drilling with rates of healing reported as between 82% and 98% [48]. After the failure of knee JOCD primary surgical intervention, alternative options may include the use of autograft and allograft options [49,50] (Fig. 1).

Multiple studies have found that many patients with traumatic or idiopathic JOCD have higher rates of vitamin D deficiency. A case-control study done by Maier et al. found a high prevalence of vitamin D deficiency in children diagnosed with JOCD [6]. A study of 80 patients showed that those diagnosed with JOCD presented with a lower mean 25(OH)D level compared with age-matched controls [6]. A study done by Oberti et al. looked at a population of patients diagnosed with JOCD and found patients who required surgery had a higher incidence of vitamin D deficiency compared to those who were treated conservatively [51]. In JOCD of the ankle, studies have shown a similar trend. Patients with osteochondral lesions of the talus showed higher rates of vitamin D deficiency when compared to age-matched controls with ankle sprains [52,53]. While the literature is still developing, vitamin D supplementation may be useful for preventing JOCD. A prospective pilot study of 23 patients with JOCD showed a 78% rate of vitamin D deficiency among the entire study cohort [54].

Conclusion

Vitamin D deficiency in athletes should be evaluated because it can impact athletic performance. RED-S and JOCD are 2 examples of pathology seen in sports where vitamin D deficiency can have a negative effect and where vitamin D supplementation has shown some promise. Athletes with RED-S have insufficient energy to support functional physiological performance let alone allow them to compete at high intensity levels. Stress fractures can develop due to repetitive overloading of the extremity when normal bone biology is altered due to deficiencies in essential bone building blocks like vitamin D in RED-S. Many patients with traumatic or idiopathic JOCD have higher rates of vitamin D deficiency, and patients who required surgery had a higher incidence of vitamin D deficiency compared to those treated conservatively. Vitamin D supplementation can also lead to protective and positive effects on muscle repair and recovery. Protecting bone health in the skeletally immature athlete population is essential as developing bone relies upon sufficient levels of vitamin D. Further research is needed to understand how vitamin D deficiency directly affects sports-related pathology and what the optimal level of vitamin D may be for preventing injury.

Author contributions

Corinna Franklin: Supervision, Writing – review & editing. **Jennifer Beck:** Supervision, Writing – review & editing. **Louise Atadja:** Writing – original draft, Writing – review & editing.

Declarations of competing interests

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

References

[1] Holick MF. The D-lightful vitamin D for child health. JPEN J Parent Enter Nutr 2012;36(1 Suppl):9S–19S. https://doi.org/10.1177/0148607111430189.

[2] Williams K, Hughes D, Horan M. Vitamin D trends in the pediatric orthopaedic population: a survey. J Pedia Orthop 2020;40(1):58–62. https://doi.org/10.1097/BPO.0000000000001394.

[3] Misra M, Pacaud D, Petryk A, Collett-Solberg PF, Kappy M. Vitamin D deficiency in children and its management: review of current knowledge and recommendations. Pediatrics 2008;122(2):398–417. https://doi.org/10.1542/peds.2007-1894.

[4] Vojinovic J, Cimaz R. Vitamin D—update for the pediatric rheumatologists. Pediatr Rheumatol Online J 2015;13:18. https://doi.org/10.1186/s12969-015-0017-9. Published 2015 May 29.

[5] Peroni DG, Trambusti I, Di Cicco ME, Nuzzi G. Vitamin D in pediatric health and disease. Pedia Allergy Immunol 2020;31(S24):54–7. https://doi.org/10.1111/pai.13154.

[6] Maier GS, Lazovic D, Maus U, Roth KE, Horas K, Seeger JB. Vitamin D deficiency: the missing etiological factor in the development of juvenile osteochondrosis dissecans? J Pedia Orthop 2019;39(1):51–4. https://doi.org/10.1097/BPO.0000000000000921.

[7] Abrams GD, Feldman D, Safran MR. Effects of vitamin D on skeletal muscle and athletic performance. J Am Acad Orthop Surg 2018;26(8):278–85. https://doi.org/10.5435/JAAOS-D-16-00464.

[8] Miller JR, Dunn KW, Ciliberti LJJ, Patel RD, Swanson BA. Association of vitamin D with stress fractures: a retrospective cohort study. J Foot Ankle Surg Publ Am Coll Foot Ankle Surg 2016;55(1):117–20. https://doi.org/10.1053/j.jfas.2015.08.002.

[9] Książek A, Zagrodna A, Słowińska-Lisowska M. Vitamin D, skeletal muscle function and athletic performance in athletes-a narrative review. Nutrients 2019;11(8). https://doi.org/10.3390/nu11081800.

[10] Millward D, Root AD, Dubois J, Cohen RP, Valdivia L, Helming B, et al. Association of serum vitamin D levels and stress fractures in collegiate athletes. Orthop J Sports Med 2020;8(12). https://doi.org/10.1177/2325967120966967. 2325967120966967 Published 2020 Dec 9.

[11] Beck JJ, Mahan ST, Nowicki P, Schreiber VM, Minkowitz B. What Is New in Pediatric Bone Health. J Pediatr Orthop 2021;41(8):e594–9. https://doi.org/10.1097/BPO.0000000000001896.

[12] Stockton KA, Mengersen K, Paratz JD, Kandiah D, Bennell KL. Effect of vitamin D supplementation on muscle strength: a systematic review and meta-analysis. Osteoporos Int 2011;22(3):859–71. https://doi.org/10.1007/s00198-010-1407-y.

[13] Owens DJ, Sharples AP, Polydorou I, Alwan N, Donovan T, Tang J, et al. A systems-based investigation into vitamin D and skeletal muscle repair, regeneration, and hypertrophy. Am J Physiol Endocrinol Metab 2015;309(12):E1019–31. https://doi.org/10.1152/ajpendo.00375.2015.

[14] Ward KA, Das G, Berry JL, Roberts SA, Rawer R, Adams JE, et al. Vitamin D status and muscle function in post-menarchal adolescent girls. J Clin Endocrinol Metab 2009;94(2):559–63. https://doi.org/10.1210/jc.2008-1284.

[15] Hradsky O, Soucek O, Maratova K, Matyskova J, Copova I, Zarubova K. Supplementation with 2000 IU of Cholecalciferol Is Associated with Improvement of Trabecular Bone Mineral Density and Muscle Power in Pediatric Patients with IBD. Inflamm Bowel Dis 2017;23(4):514–23. https://doi.org/10.1097/MIB.0000000000001047.

[16] Gilsanz V, Kremer A, Mo AO, Wren TAL, Kremer R. Vitamin D status and its relation to muscle mass and muscle fat in young women. J Clin Endocrinol Metab 2010;95(4):1595–601. https://doi.org/10.1210/jc.2009-2309.

[17] Chiang C-M, Ismaeel A, Griffis RB, Weems S. Effects of vitamin D supplementation on muscle strength in athletes: a systematic review. J Strength Cond Res 2017;31(2):566–74. https://doi.org/10.1519/JSC.0000000000001518.

[18] Dave SC, Fisher M. Relative energy deficiency in sport (RED - S). Curr Probl Pedia Adolesc Health Care 2022;52(8):101242. https://doi.org/10.1016/j.cppeds.2022.101242.

[19] Vopat L, Mackay MJ, Vopat BG, Mulcahey MK. Relative Energy Deficiency in Sport: an orthopaedic perspective. J Am Acad Orthop Surg 2021;29(1):e14–21. https://doi.org/10.5435/JAAOS-D-20-00460.

[20] Mountjoy M, Ackerman KE, Bailey DM, Burke LM, Constantini N, Hackney AC, et al. International Olympic Committee's (IOC) consensus statement on Relative Energy Deficiency in Sport (REDs). Br J Sports Med 2023;57(17):1073–97. https://doi.org/10.1136/bjsports-2023-106994.

[21] Cabre HE, Moore SR, Smith-Ryan AE, Hackney AC. Relative Energy Deficiency in Sport (RED-S): scientific, clinical, and practical implications for the female athlete. Dtsch Z Sport 2022;73(7):225–34. https://doi.org/10.5960/dzsm.2022.546.

[22] Joy E, De Souza MJ, Nattiv A, Misra M, Williams NI, Mallinson RJ, et al. 014 female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad. Curr Sports Med Rep 2014;13(4):219–32. https://doi.org/10.1249/JSR.0000000000000077.

[23] Heikura IA, Burke LM, Bergland D, Uusitalo ALT, Mero AA, Stellingwerff T. Impact of energy availability, health, and sex on hemoglobin-mass responses following live-high-train-high altitude training in elite female and male distance athletes. Int J Sports Physiol Perform 2018;13(8):1090–6. https://doi.org/10.1123/ijspp.2017-0547.

[24] Abbott A, Bird ML, Wild E, Brown SM, Stewart G, Mulcahey MK. Part I: epidemiology and risk factors for stress fractures in female athletes. Phys Sport 2020;48(1):17–24. https://doi.org/10.1080/00913847.2019.1632158.

[25] Barrack MT, Gibbs JC, De Souza MJ, Williams NI, Williams NI, Nichols JF, Rauh MJ, et al. Higher incidence of bone stress injuries with increasing female athlete triad-related risk factors: a prospective multisite study of exercising girls and women. Am J Sports Med 2014;42(4):949–58. https://doi.org/10.1177/0363546513520295.

[26] Hoenig T, Ackerman KE, Beck BR, Bouxsein ML, Burr DB, Hollander K, et al. Bone stress injuries. Nat Rev Dis Primers 2022;8(1):26. https://doi.org/10.1038/s41572-022-00352-y. Published 2022 Apr 28.

[27] Melin A, Tornberg ÅB, Skouby S, Møller SS, Sundgot-Borgen J, Faber J, et al. Energy availability and the female athlete triad in elite endurance athletes. Scand J Med Sci Sports 2015;25(5):610–22. https://doi.org/10.1111/sms.12261.

[28] Nose-Ogura S, Yoshino O, Dohi M, Kigawa M, Harada M, Hiraike O, et al. Risk factors of stress fractures due to the female athlete triad: differences in teens and twenties. Scand J Med Sci Sports 2019;29(10):1501–10. https://doi.org/10.1111/sms.13464.
[29] Lloyd T, Myers C, Buchanan JR, Demers LM. Collegiate women athletes with irregular menses during adolescence have decreased bone density. Obstet Gynecol 1988;72(4):639–42.
[30] Ihle R, Loucks AB. Dose-response relationships between energy availability and bone turnover in young exercising women. J Bone Min Res J Am Soc Bone Min Res 2004;19(8):1231–40. https://doi.org/10.1359/JBMR.040410.
[31] Wright AA, Taylor JB, Ford KR, Siska L, Smoliga JM. Risk factors associated with lower extremity stress fractures in runners: a systematic review with meta-analysis. Br J Sports Med 2015;49(23):1517–23. https://doi.org/10.1136/bjsports-2015-094828.
[32] Varley I, Stebbings G, Williams AG, Day S, Hennis P, Scott R, et al. An investigation into the association of bone characteristics and body composition with stress fracture in athletes. J Sports Med Phys Fit 2021;61(11):1490–8. https://doi.org/10.23736/S0022-4707.21.11871-7.
[33] da Rocha Lemos Costa TM, Borba VZC, Correa RGP, Moreira CA. Stress fractures. Arch Endocrinol Metab 2022;66(5):765–73. https://doi.org/10.20945/2359-3997000000562.
[34] Cupka M, Sedliak M. Hungry runners—low energy availability in male endurance athletes and its impact on performance and testosterone: mini-review. Eur J Transl Myol 2023;33(2):11104. https://doi.org/10.4081/ejtm.2023.11104. Published 2023 Apr 11.
[35] Ito E, Sato Y, Kobayashi T, Soma T, Matsumoto T, Kimura A, et al. Low energy availability reduces bone mass and gonadal function in male mice. J Bone Min Metab 2023;41(2):182–92. https://doi.org/10.1007/s00774-023-01413-2.
[36] Edmonds E.W., Polousky J. A Review of Knowledge in Osteochondritis Dissecans: ¨ nig to the ROCK 123 Years of Minimal Evolution from Ko Study Group. Published online 2013:1118-1126. doi:〈10.1007/s11999–012-2290-y〉.
[37] Johnson C.P., Wang L., Nelson B.J., Laprade R.F. Insights into the Epiphyseal Cartilage Origin and Subsequent Osseous Manifestation of Juvenile Osteochondritis Dissecans with a Modified Clinical MR Imaging Protocol: A. 2021;282(3).
[38] Kessler J.I., Nikizad H., Shea K.G., Jr J.C.J., Bebchuk J.D., Weiss J.M. The Demographics and Epidemiology of Osteochondritis Dissecans of the Knee in Children and Adolescents. Published online 2011:320-326. doi:〈10.1177/0363546513510390〉.
[39] Hughes JA, Cook JV, Churchill MA, Warren ME. Juvenile osteochondritis dissecans: a 5-year review of the natural history using clinical and MRI evaluation. Pedia Radiol 2003;33(6):410–7. https://doi.org/10.1007/s00247-003-0876-y.
[40] Fairbank HB. Osteochondritis dissecans. Br J Surg 1933;21:67–73.
[41] SMILLIE IS. Treatment of osteochondritis dissecans. J Bone Jt Surg Br 1957;39-B (2):248–60. https://doi.org/10.1302/0301-620X.39B2.248.
[42] Cahill BR. Osteochondritis dissecans of the knee: treatment of juvenile and adult forms. J Am Acad Orthop Surg 1995;3(4):237–47. https://doi.org/10.5435/00124635-199507000-00006.
[43] Uozumi H, Sugita T, Aizawa T, Takahashi A, Ohnuma M, Itoi E. Histologic findings and possible causes of osteochondritis dissecans of the knee. Am J Sports Med 2009;37(10):2003–8. https://doi.org/10.1177/0363546509346542.
[44] Woods, L.; Harris I. No Title. Hournal Bone Jt Surg - Br Vol. 1995;77-B(2):331.
[45] Shea K.G., Jacobs J.C., Bs J., Carey J.L., Anderson A.F., Oxford J.T. Osteochondritis Dissecans Knee Histology Studies Have Variable Findings and Theories of Etiology. Published online 2013:1127-1136. doi:〈10.1007/s11999–012-2619–6〉.
[46] Hefti F, Beguiristain J, Krauspe R, Möller-Madsen B, Riccio V, Tschauner C, et al. Osteochondritis dissecans: a multicenter study of the European Pediatric Orthopedic Society. J Pedia Orthop B 1999;8(4):231–45.
[47] Ludwig KD, Johnson CP, Zbýň Š, Nowacki A, Marette S, Takahashi T, et al. MRI evaluation of articular cartilage in patients with juvenile osteochondritis dissecans (JOCD) using T2∗ mapping at 3T. Osteoarthr Cartil 2020;28(9):1235–44. https://doi.org/10.1016/j.joca.2020.04.001.
[48] Chambers HG, Shea KG, Carey JL. AAOS clinical practice guideline: diagnosis and treatment of osteochondritis dissecans. J Am Acad Orthop Surg 2011;19(5):307–9. https://doi.org/10.5435/00124635-201105000-00008.
[49] Martinčič D, Radosavljevič D, Drobnič M. Ten-year clinical and radiographic outcomes after autologous chondrocyte implantation of femoral condyles. Knee Surg Sports Trauma Arthrosc 2014;22(6):1277–83. https://doi.org/10.1007/s00167-013-2778-3.
[50] Beck JJ, Sugimoto D, Micheli L. Sustained results in long-term follow-up of Autologous Chondrocyte Implantation (ACI) for distal femur Juvenile Osteochondritis Dissecans (JOCD). Adv Orthop 2018;2018:7912975. https://doi.org/10.1155/2018/7912975.
[51] Oberti V, Sanchez Ortiz M, Allende V, Masquijo J. Prevalence of hypovitaminosis D in patients with juvenile osteochondritis dissecans. Rev Esp Cir Ortopédica Y Trauma 2021;65(2):132–7. https://doi.org/10.1016/j.recote.2020.07.006.
[52] Telleria JJM, Ready LV, Bluman EM, Chiodo CP, Smith JT. Prevalence of vitamin D deficiency in patients with talar osteochondral lesions. Foot ankle Int 2018;39(4):471–8. https://doi.org/10.1177/1071100717745501.
[53] Fraissler L, Boelch SP, Schäfer T, Walcher M, Arnholdt J, Maier G, et al. Vitamin D deficiency in patients with idiopathic and traumatic osteochondritis dissecans of the talus. Foot ankle Int 2019;40(11):1309–18. https://doi.org/10.1177/1071100719864325.
[54] Bruns J, Werner M, Soyka M. Is vitamin D insufficiency or deficiency related to the development of osteochondritis dissecans? Knee Surg Sports Trauma Arthrosc 2016;24(5):1575–9. https://doi.org/10.1007/s00167-014-3413-7.