International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE

STRESS FRACTURES IN ATHLETES: FROM PATHOPHYSIOLOGICAL MECHANISMS TO EVIDENCE-BASED DIAGNOSTIC AND THERAPEUTIC STRATEGIES

DOI https://doi.org/10.31435/ijitss.1(49).2026.5289

RECEIVED 14 January 2026

ACCEPTED 20 March 2026

PUBLISHED 25 March 2026

STRESS FRACTURES IN ATHLETES: FROM PATHOPHYSIOLOGICAL MECHANISMS TO EVIDENCE-BASED DIAGNOSTIC AND THERAPEUTIC STRATEGIES

Wiktoria Sobieraj
MD, Lower Silesian Center for Oncology, Pulmonology and Hematology, Wrocław, Poland
ORCID ID: 0009-0007-5412-6302

Julia Pająk
MD, 4th Military Clinical Hospital with Polyclinic, Wrocław, Poland
ORCID ID: 0009-0007-0882-9365

Olga Sławatyniec
MD, 4th Military Clinical Hospital with Polyclinic, Wrocław, Poland
ORCID ID: 0009-0007-0373-1588

Anna Kamosińska *(Corresponding Author, Email: anna.kamosinska@interia.pl)*
MD, 4th Military Clinical Hospital with Polyclinic, Wrocław, Poland
ORCID ID: 0009-0005-0718-2112

ABSTRACT

Background: Stress fractures (SFs) occur due to repetitive submaximal loading exceeding the bone's intrinsic repair capacity. SFs constitute a significant challenge among endurance athletes and substantially compromise athletic performance and disrupt training continuity, requiring a comprehensive and multidisciplinary therapeutic approach.

Objective: This review synthesises current evidence on the etiology, pathophysiology, diagnosis, and management of SFs in athletes.

Methods: A literature review was conducted to evaluate diagnostic standards and therapeutic strategies for bone stress injuries.

Results: SFs develop along a pathological continuum. Risk factors include abrupt training load transitions and metabolic disturbances like the Female Athlete Triad. While X-rays lack early sensitivity, MRI is the gold standard for diagnosis. Management is risk-stratified: low-risk fractures are typically managed conservatively, whereas high-risk fractures often require surgery or prolonged immobilisation.

Conclusion: Systematic workload management and early, risk-based intervention are essential for effective recovery and the prevention of training interruptions.

KEYWORDS

Stress Fractures, Athletes, Etiopathogenesis, Diagnosis, Management

CITATION

Wiktoria Sobieraj, Julia Pająk, Olga Sławatyniec, Anna Kamosińska. (2026) Stress Fractures in Athletes: From Pathophysiological Mechanisms to Evidence-Based Diagnostic and Therapeutic Strategies. *International Journal of Innovative Technologies in Social Science*. 1(49). doi: 10.31435/ijitss.1(49).2026.5289

COPYRIGHT

1. Introduction

Stress fractures (SF) arise from microdamage that occurs when bone is subjected to repetitive submaximal loading [1]. In athletes, particularly those engaged in endurance disciplines, repetitive loading of the lower limbs promotes the accumulation of microdamage in bone tissue. Over time, if mechanical loads exceed bone's intrinsic repair capacity, accumulated microdamage progresses through repeated cycles, ultimately resulting in macrostructural lesions and complete fractures [2].

Although regular physical activity confers numerous health benefits, it simultaneously increases the risk of overuse injuries, which are a major concern among athletes. Stress fractures account for 10-20% of injuries in sports medicine and approximately 10% of orthopedic injuries [3, 4]. Up to 40% of athletes may experience a stress fracture during their careers, and a prior occurrence constitutes a significant predisposing factor, increasing the risk of subsequent fractures five - to sixfold, independent of sex, with roughly one in seven athletes having a history of stress fracture [2, 3, 5].

Women are nearly twice as likely as men to develop stress fractures [6]. This risk is closely associated with components of the Female Athlete Triad, including menstrual disturbances, reduced bone mineral density (BMD), and low energy availability (LEA). Women with the most severe manifestations of the Triad are at markedly increased risk of stress fractures [7].

The incidence of stress fractures varies with sport type, being higher in disciplines such as cross-country running, gymnastics, athletics, and basketball [8, 9]. Early recognition and implementation of effective preventive strategies are crucial not only for reducing fracture risk but also for mitigating clinical symptoms and minimizing training interruptions [9].

The aim of this review is to provide a comprehensive overview of current knowledge regarding stress fractures, with particular emphasis on etiology, clinical presentation, diagnostic strategies, treatment approaches, and potential complications, underscoring their clinical and practical significance in sports medicine.

2. Pathophysiology of stress fractures

2.1. Bone physiology and remodelling

Bone is a highly specialised, mineralised connective tissue that serves as the body’s primary structural framework, providing protection for vital organs and mechanical support enabling locomotion, housing the bone marrow, and acting as a reservoir for calcium, growth factors and cytokines, as well as playing an important role in maintaining acid-base balance. It is an inherently dynamic organ with a complex metabolism that undergoes lifelong remodelling of its structure [10, 11]. Osseous tissue is organised into two main architectural components: dense cortical bone, which comprises approximately 80% of skeletal mass, and highly porous trabecular bone interspersed within the medullary compartment [11, 12]. Skeletal homeostasis is coordinated by osteoclasts, which are responsible for the resorption of old or damaged bone; osteoblasts, which synthesise and deposit new bone matrix; and osteocytes, which are former osteoblasts embedded within the calcified matrix that help maintain bone tissue and control extracellular concentrations of calcium and phosphate [10, 12].

Bone remodelling, an adaptive mechanism intended to preserve skeletal strength and mineral homeostasis, is the key to understanding bone tissue function and its pathologies, such as stress fractures [11, 13]. This process is carried out by functional teams of cells known as basic multicellular units (BMUs), which operate at both randomly distributed remodelling sites and in targeted areas that require repair. The remodelling cycle within a BMU can be divided into multiple phases, beginning with the activation of the bone surface prior to resorption, subsequent dissolution of the mineral matrix by osteoclasts, the reversal phase, bone formation and mineralisation. Under physiological conditions, the amount of bone formed in one such cycle should be equal to the amount of bone resorbed, ensuring structural stability [11, 14].

2.2. Mechanobiology and pathophysiology of overload

The skeletal response to mechanical demand is governed by Wolff’s Law, which states that bone adapts its density and internal structure to the specific loads placed upon it [15, 6]. This concept was further refined by Harold Frost into the “mechanostat” theory. The theoretical framework he proposed identified two key mechanoadaptive processes in bone: modelling and remodelling. These processes explain how load-bearing bones adjust their mass and morphology through dynamic feedback loops [16, 17]. Within this system, osteocytes act as the primary mechanosensors, detecting changes in mechanical strain through the movement of interstitial fluid within the lacunar-canalicular system and initiating bone formation response.

The mechanostat operates based on specific Minimally Effective Strains (MES), where strain is defined as the change in length per unit length of a bone and is often expressed in microstrain ($\mu\varepsilon$). If strains fall below the MESr (remodelling threshold), disuse-mode remodelling occurs, leading to a net loss of bone mass. Conversely, when strains exceed the MESm (modelling threshold), formation modelling is activated, stimulating osteoblastic activity. Microdamage occurs when strains surpass the MESp (bone's operational microdamage threshold) at approximately 3000 $\mu\varepsilon$, although exact values largely depend on genetic factors. It is above the MESp that the rate of damage formation begins to outpace the innate repair mechanisms and the pathology continuum begins [17, 18].

The biological imbalance between load-induced microdamage formation and its removal results in a stress reaction, the initial stage of bone stress injury (BSI). If loading continues, this can progress to more advanced stages, including stress fracture and, ultimately, a complete bone fracture. BSI is a direct consequence of bone's inability to withstand repetitive submaximal loading, due to inadequate time for tissue recovery and other factors [2, 19, 20]. The clinical progression of BSI is illustrated as a chain of events in figure 1.

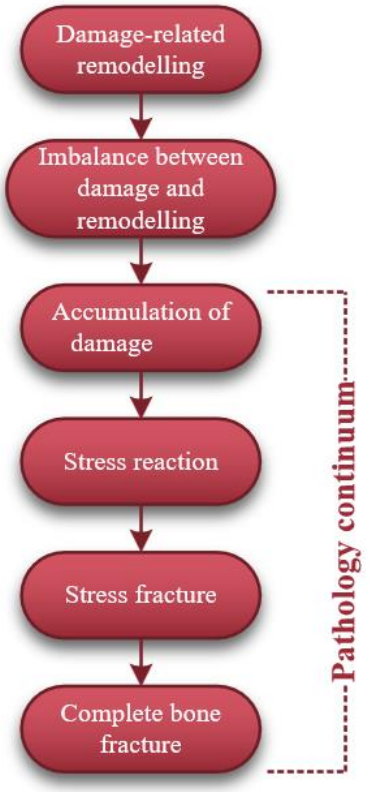

Fig. 1. *Bone stress injury continuum.*

Pathophysiologically, load-induced microcracks within the bone matrix initiate targeted remodelling [18, 20]. However, because bone resorption precedes formation in the remodelling cycle, an increase in active remodelling units creates a transient period of increased bone porosity [15, 19]. This temporary reduction in local bone density, if loading persists, facilitates the coalescence of microdamage into a visible fracture line, perpetuating a positive feedback loop of structural weakening that results in a stress fracture [2, 15, 19].

2.3. Extrinsic and intrinsic risk factors

Risk factors for bone stress injuries, including stress fractures, are multifactorial and typically categorised into extrinsic and intrinsic variables. The most common extrinsic causes involve abrupt transitions in training load, characterised by sudden increase in the volume, intensity, or frequency of activity [18, 21]. Participation in specific athletic disciplines further modulates the risk of stress injury and different sports may predispose to site-specific fractures. For instance, runners typically experience tibial and metatarsal fractures, whereas hurdlers are more prone to patellar fractures. Additionally, some activities, such as gymnastics and ballet, require maintenance of a lean physique, which may compound mechanical stress with metabolic disturbances [3, 6]. Type of training surface and footwear, especially in sports involving high volumes of jumping and running, are relatively modifiable but significantly contribute to the risk of BSI [20]. Furthermore, nutritional deficits, particularly insufficient intake of calcium and vitamin D, have been shown to considerably impact stress fracture risk. An original study by Nieves et al. found that a daily calcium intake of 800 mg in female runners was associated with 6-fold increased risk of stress fractures compared to a group with an intake of 1500 mg [2, 23].

Among intrinsic risk factors (related to characteristics within individuals), a prior history of BSI serves as one of the strongest predictors of future skeletal failure, often reflecting underlying structural or physiological predispositions [3, 18, 21, 22]. Other intrinsic variables encompass a broad spectrum of factors, including female sex, menstrual disorders, hormonal imbalances, low bone mineral density (BMD), body composition, and anatomical variations. [3, 6] In the context of female athletes, some of these biological variables manifest through two interrelated clinical frameworks: Relative Energy Deficiency in Sport (REDs) and the female athlete triad. REDs is defined as a syndrome of impaired physiological and/or psychological functioning caused by prolonged and/or severe low energy availability (LEA) [24]. It is a broader concept than the female athlete triad, which involves three components: LEA with or without disordered eating, menstrual dysfunction and low BMD [25, 26]. From a women's health perspective, these syndromes signify a profound metabolic disturbance where the body prioritizes immediate survival over non-essential functions impairing skeletal maintenance. [24]. The presence of a single component of the triad increases the risk of stress fractures to 15-20%, rising further to 30-50% if multiple aspects of the syndrome are present, highlighting its clinical significance [3].

3. Diagnosis of Stress Fractures

3.1 Medical History

Athletes, who are subject to frequent and intense training, are particularly prone to developing stress fractures. When a stress fracture is suspected, during the medical history collection, it is important to identify any external and internal risk factors that the patient has been exposed to. Next, a detailed assessment of the patient's physical activity over the past 6-8 weeks is necessary. Attention should be paid to changes in the intensity, frequency, and duration of training, as well as whether adequate time for recovery was allowed. Typical

circumstances preceding the onset of pain include an increase in training intensity, changes in the terrain or type of footwear used during training. [6] Patients usually do not report a specific injury that could clearly be linked to the onset of symptoms; the pain is typically described as dull, progressively worsening with physical exertion, intensifying during loading of the limb, and subsiding at rest. Initially, the pain appears only during physical exertion, but over time it may also occur during daily activities such as walking. [28] Pain associated with movement remains the most commonly reported symptom, occurring in approximately 81% of patients [29].

3.2 Abnormalities on Physical Examination

During a physical examination, local swelling of soft tissues and tenderness upon palpation of the bone are often observed. These symptoms may resemble muscle injuries; therefore, it is important to distinguish between soft tissue damage and a true stress fracture. Muscle tenderness indicates a strain, whereas pain upon palpating the bone suggests bone damage. [2, 6] In cases where areas are difficult to access during palpation (such as the femoral neck), pain during joint movement may indicate a stress fracture [2]. The following tests can also be helpful in making an initial diagnosis:

One-leg hop test: The patient is asked to hop on the leg suspected of having a fracture. The presence of severe pain upon landing is considered a positive result, indicating a stress fracture. This test also helps differentiate this condition from plantar fasciitis [2, 6].

Fulcrum test: The patient sits with the lower limbs hanging freely; the examiner places the forearm under the thigh and presses on the area around the knee with the other hand. By moving the hand along the thigh, the site of greatest pain is identified. An increase in pain at a specific location is considered a positive result, suggesting a stress fracture of the femoral neck [2, 6].

Stork test (lumbar extension or single-leg hyperextension): This test is used to diagnose spondylolysis, although its sensitivity and specificity are limited. The patient is asked to bend one leg and then straighten the lower back. A positive result of this test indicates a stress fracture of the femoral neck [6, 30].

Tuning fork test: The examiner applies a vibrating device to the suspected fracture site; if bone damage is present, applying the device causes localized pain. Although this test is relatively simple and non-invasive, its diagnostic value is limited. One study found a sensitivity of 75%, specificity of 67%, a positive predictive value of 77%, and a negative predictive value of 63% for diagnosing stress fractures of the metacarpal bones [29, 31].

Diagnosing a stress fracture can be challenging due to similar symptoms present in other conditions. Differential diagnoses include tendinitis, osteitis, ligament entrapment syndromes, nerve or artery compression, tumors (osteochondroma, Ewing's sarcoma), and intermittent claudication [29].

3.3 Imaging Studies

It is of fundamental importance in the diagnostic process to compare clinical symptoms with the findings observe in imaging studies. In order to make a final diagnosis, it is often necessary to perform tests such as X-rays (RTG), computed tomography (CT), magnetic resonance imaging (MRI), or scintigraphy.

X-Rays (RTG)

The first imaging study performed in cases of suspected stress fractures is usually an X-ray. This is due to the wide availability of X-rays, their low cost, and the relatively low dose of ionizing radiation they emit. However, it should be noted that in the early stages of the disease process, X-rays have low sensitivity- in up to 70% of patients, the radiological images appear normal. It is not until 2-3 weeks after the onset of symptoms that typical changes associated with stress fractures can be observed, such as elevation of the bone cortex, thickening of the cortical bone, sclerotic changes, or a visible fracture line. [2, 30] One characteristic sign indicating a high risk of fracture, especially in the femur or tibia, is what is known as the "dreaded black line." In cases of stress fractures of the foot bones, an early symptom often is a blurred outline of the cortical bone (referred to as the "gray cortex sign"). More advanced changes include the formation of callus or a more pronounced thinning of the cortical bone. Conventional X-ray studies have little diagnostic value in cases of spondylolysis- in order to accurately assess the condition of the interarticular cartilage, it is necessary to perform as many as five different X-ray projections, and even then, there is no guarantee that early damage will be detected. If the clinical suspicion cannot be confirmed after an X-ray study, the next step is to perform further tests, most notably magnetic resonance imaging (MRI). [6, 31]

Magnetic Resonance Imaging (MRI)

Magnetic resonance imaging (MRI) is currently the gold standard for diagnosing stress fractures; it boasts the highest sensitivity (80-100%) and accuracy (up to 100%) [6, 30]. Unlike X-rays, MRI allows for early detection of changes. Thanks to its ability to visualize edema of the bone marrow, bone healing processes, the presence of a fracture line, and any involvement of soft tissues, stress fractures can be identified as early as 1-2 days after the onset of pain. Another significant advantage of MRI over other methods is its absence of exposure to ionizing radiation. To assess the severity of the changes, the Fredericson classification is used:

Grade I	No changes in the bone marrow
Grade II	Mild edema of the bone marrow visible only on T2 images
Grade III	Moderate edema on both T1 and T2 sequences
Grade IV	Visible fracture line

Despite these advantages, MRI has limitations, including limited accessibility and high cost. [6, 31]

Computed Tomography (CT)

Compared to MRI, computed tomography (CT) has high accuracy but lower sensitivity (about 40-45%). Although CT is often more readily available than MRI, it exposes patients to a higher dose of radiation and is primarily used in cases where MRI is not feasible or contraindicated (e.g., in patients with metal implants). CT is useful for distinguishing between complete and incomplete fractures, as well as for identifying stress fractures in certain bones such as the spine, pelvis, or rib cage. Due to the slower bone healing process, chronic or quiescent changes may be more evident on CT than on MRI or bone scintigraphy. Single-photon emission tomography (SPECT) is particularly useful for diagnosing stress fractures in the thoracic spine, especially in the intervertebral region of the vertebrae (spondylolysis). [6, 30]

Bone Scintigraphy (SPECT, Triple-Phase Scintigraphy)

Scintigraphy using technetium-99 is a method with high sensitivity (about 74-100%) but low accuracy. Increased uptake of the radio tracer can be observed as early as 3-5 days after the onset of symptoms, making it useful for early diagnosis, especially in cases where multiple stress fractures are suspected. However, this method has low accuracy- similar findings can also occur in cases of avascular necrosis, osteitis, or tumors. For these reasons, scintigraphy is rarely used for follow-up assessments or monitoring of the healing of stress fractures. [2, 6, [31]

3.4 Classification of Low- and High-Risk Fractures

The classification of stress fractures into low-risk and high-risk categories is based on differences in bone blood supply, the nature of mechanical stresses, and the potential for complications such as delayed healing or fracture displacement. The biomechanical properties of the affected area, as well as the type of forces applied during stress, also play a significant role. High-risk fractures occur in areas subjected to maximum tensile stress and poor blood circulation, which increases the likelihood of avascular necrosis (dead bone tissue) or delayed healing. Low-risk fractures are associated with lower mechanical stress and a better potential for healing- in these cases, conservative treatment is usually sufficient. Regions classified as low-risk include the posterior surface of the femur, the second to fourth metatarsal bones, the tibia, the pelvis, and the sacrum. In these cases, the prognosis is good, and treatment is typically conservative. High-risk fractures occur in areas with limited blood supply and subjected to high levels of tensile stress. Typical locations include the femoral neck, the anterior surface of the femur, the navicular bone of the foot, the talus, and the first and fifth metatarsal bones. Due to the high risk of progression to complete fracture, delayed healing, or displacement of the fragments, these fractures require more stringent diagnostic attention and modified treatment approaches. Fractures of the femoral neck are particularly critical, as they carry a risk of developing avascular necrosis of the femoral head if the fragments displace. Magnetic resonance imaging (MRI) is recommended for diagnosis in these cases, given the limited sensitivity of conventional X-ray images. Injuries to the upper (tensile) part of the femur require immediate orthopedic consultation and are usually treated surgically. Fractures involving less than 50% of the femoral neck width may be treated conservatively, provided that they are closely monitored clinically and radiographically. If pain worsens or if there is an increase in the fracture line, surgical intervention is indicated. Fractures located in other high-risk areas (e.g., the anterior surface of the femur, navicular bone of the foot) tend to have poor healing prospects, often necessitating surgical treatment or prolonged immobilization of the affected limb. [2, 28, 30]

4. Treatment

Therapeutic management of stress fractures in athletes should be individualized, taking into account the specific type of injury, its anatomical location, the stage of progression and the associated risk of potential complications along with the patient's goals and expectations [32, 33]. Evaluating the regenerative potential of bone tissue microcracks and implementing effective prevention strategies requires a comprehensive assessment of the athlete's overall health, including nutritional habits, training schedule, medications [34]. Effective recovery is contingent upon an interdisciplinary treatment model incorporating physiotherapy, appropriate nutritional support, advanced regenerative therapies, and psychological interventions. Such an approach aims to ensure both the physical and psychological readiness of the patient for a safe return to sport [33].

An important component of clinical management is the classification of fractures into low and high risk categories, which allows determining their gradation, prognosis and treatment method. [28]. Stress fractures characterized by a low probability of fracture propagation, delayed union or non-union, and therefore belonging to the low-risk group, occur primarily in places where compression forces predominate [2, 35]. These include stress fractures of the posteromedial aspect of the tibial shaft, the metatarsal shafts, the fibula, the medial aspect of the femoral neck, the femoral shaft, and the calcaneus, as well as the pelvis, ribs, and the ulnar shaft [2, 35, 36]. They can usually be effectively treated conservatively, incorporating rest, activity modification, and rehabilitation [35].

A two-phase therapeutic approach is commonly recommended. The first stage of treatment involves limiting physical activity and administering pain medication, excluding nonsteroidal anti-inflammatory drugs [28]. During this time, athletes may be encouraged to engage in alternative, lower-impact exercises, such as hydrotherapy or swimming, anti-gravity treadmill use, and elliptical training, to maintain strength and fitness while minimising muscle atrophy. If pain occurs during movement, temporary immobilization may be indicated. The next step is a gradual return to physical activity, supported by appropriate physiotherapy, taking into account the patient's individual tolerance, pain intensity, and monitoring of the injury's healing progress [2, 37]. Assessment of bone union formation and disappearance of fracture lines visible on X-ray, MRI or CT scans may be helpful in assessing the progress of injury healing [2].

High-risk stress fractures, in contrast, are characterized by an increased rate of fracture propagation, displacement, delayed union, and nonunion [35]. They are located within the cortical surface of the bone subjected to tensile stress or in areas with limited vascularization, which may significantly impair the healing process [34].

The most common high-risk stress fractures include those of the anterior tibial shaft, femoral head, femoral neck, fifth metatarsal, sesamoid of the first metatarsal, navicular bone, medial malleolus, and patella [35, 38]. Treatment consists primarily of immobilization with complete restriction of weight-bearing of the limb and a prolonged period of rest from practicing sports compared to low-risk fractures [38]. Evaluating the need for surgical intervention is also crucial, and should always be considered for high-risk fractures. This is particularly important if there is no improvement despite conservative therapy, symptoms worsen, or there is radiological evidence of fracture progression [34].

Available data indicate that in selected cases, surgical treatment may be associated with shorter healing times and a faster return to sports compared to conservative treatment [39]. Studies that investigate stress fractures of the navicular and fifth metatarsal have shown a significantly faster return to physical activity when surgical methods using intramedullary screws or other forms of internal fixation are used. However, it should be noted that the quality of evidence is limited due to the retrospective nature of the studies and their limited number [40].

Modifying intrinsic and extrinsic risk factors also is crucial in the treatment and prevention of stress fractures. Particular attention is paid to optimizing nutrition and maintaining an appropriate energy balance. Available research indicates that adequate calcium and vitamin D intake may play a role in preventing stress fractures. However, the decision to supplement should be made individually, based on an assessment of nutritional status, laboratory test results, and the athlete's overall risk profile [41, 42].

The use of bisphosphonates in the treatment of stress fractures is not routinely recommended due to limited evidence of their effectiveness and a potentially negative impact on bone remodeling [43].

Prevention

The primary strategy for preventing stress fractures and other forms of BSI involves the systematic management of training workloads to avoid large increases in the acute:chronic workload ratio (ACWR). It is crucial to personalise training volume, intensity and frequency to account for individual tolerance to changing loads [27, 44]. For prepubertal athletes, prevention should focus on multidirectional loading instead of a single sport specialisation in order to optimise bone growth and structural stability [27]. Maintaining a balanced diet with sufficient intake of micronutrients and the mitigation of Relative Energy Deficiency in Sport are critical biological safeguards against BSI. It is recommended to consume at least 45 kcal/kg of lean body mass per day to support normal bone formation and metabolic health [45]. Additionally, prophylactic supplementation with vitamin D (800-2000 IU/day) and calcium (1500-2000 mg/day) significantly reduces stress fracture incidence, particularly in athletes with baseline deficiencies [8, 11]. Sport-specific biomechanical interventions may also play a role; for instance, increasing stride rate in runners reduces vertical excursion and internal tibial loading, which is associated with a reduced incidence of BSI [11, 27]. Furthermore, although evidence is limited, shock-absorbing footwear and foot orthoses could potentially help prevent stress injuries [46].

Conclusions

Stress fractures (SF) account for 10–20% of all injuries in sports medicine and occur when repetitive submaximal loading exceeds the bone’s intrinsic repair capacity. The risk of these type of trauma is heightened by abrupt transitions in training load and metabolic factors, such as Relative Energy Deficiency in Sport (REDs) or the Female Athlete Triad. While initial X-rays (RTG) often appear normal in the early stages, Magnetic Resonance Imaging (MRI) is the gold standard for achieving an early and precise diagnosis. Management is determined by the fracture's category: low-risk fractures are typically treated conservatively through activity modification, whereas high-risk cases may require surgical intervention or prolonged immobilisation. Systematic workload management and the optimisation of calcium and vitamin D intake play a crucial role in preventing these injuries.

Author Contributions
Conceptualization: Wiktoria Sobieraj, Anna Kamosińska, Olga Sławatyniec, Julia Pająk
Methodology: Olga Sławatyniec, Wiktoria Sobieraj
Software: Julia Pająk, Olga Sławatyniec
Check: Anna Kamosińska, Wiktoria Sobieraj
Formal analysis: Julia Pająk, Olga Sławatyniec
Investigation: Wiktoria Sobieraj, Anna Kamosińska, Olga Sławatyniec, Julia Pająk
Resources: Julia Pająk, Wiktoria Sobieraj, Anna Kamosińska, Olga Sławatyniec
Data curation: Anna Kamosińska, Olga Sławatyniec, Julia Pająk, Wiktoria Sobieraj
Writing - rough preparation: Anna Kamosińska, Julia Pająk
Writing - review and editing: Olga Sławatyniec, Wiktoria Sobieraj
Visualization: Olga Sławatyniec
Supervision: Julia Pająk
Project administration: Wiktoria Sobieraj

All authors have read and agreed with the published version of the manuscript.

Conflicts of Interest: The authors declare no conflict of interest

Funding: The study received no funding and incurred no expenses unrelated to the publication costs for the author.

Declaration on the use of AI: In preparing this work, the authors used Gemini for the purpose of improving language and readability, text formatting, and verification of bibliographic styles. After using this tool/service, the authors have reviewed and edited the content as needed and accept full responsibility for the substantive content of the publication.

REFERENCES

1. Welck, M. J., Hayes, T., Pastides, P., Khan, W., & Rudge, B. (2017). Stress fractures of the foot and ankle. *Injury, 48*(8), 1722–1726. https://doi.org/10.1016/j.injury.2015.06.015
2. da Rocha Lemos Costa, T. M., Borba, V. Z. C., Correa, R. G. P., & Moreira, C. A. (2022). Stress fractures. *Archives of Endocrinology and Metabolism, 66*(5), 765–773. https://doi.org/10.20945/2359-3997000000562
3. Abbott, A., Bird, M. L., Wild, E., Brown, S. M., Stewart, G., & Mulcahey, M. K. (2020). Part I: Epidemiology and risk factors for stress fractures in female athletes. *The Physician and Sportsmedicine, 48*(1), 17–24. https://doi.org/10.1080/00913847.2019.1632158
4. Changstrom, B. G., Brou, L., Khodaee, M., Braund, C., & Comstock, R. D. (2015). Epidemiology of stress fracture injuries among US high school athletes, 2005–2006 through 2012–2013. *The American Journal of Sports Medicine, 43*(1), 26–33. https://doi.org/10.1177/0363546514562739
5. Lund, S. D., Garoussian, J., Bjerre-Bastos, J. J., Fredberg, U., & Barfod, K. W. (2025). Stressfrakturer [Stress fractures]. *Ugeskrift for Laeger, 187*(52), V06250495. https://doi.org/10.61409/V06250495
6. Bergman, R., & Kaiser, K. (2025). Stress reaction and fractures. In *StatPearls*. StatPearls Publishing.
7. Barrack, M. T., Gibbs, J. C., De Souza, M. J., Williams, N. I., Nichols, J. F., Rauh, M. J., & Nattiv, A. (2014). Higher incidence of bone stress injuries with increasing female athlete triad-related risk factors: A prospective multisite study of exercising girls and women. *The American Journal of Sports Medicine, 42*(4), 949–958. https://doi.org/10.1177/0363546513520295
8. Knechtle, B., Jastrzębski, Z., Hill, L., & Nikolaidis, P. T. (2021). Vitamin D and stress fractures in sport: Preventive and therapeutic measures—A narrative review. *Medicina, 57*(3), 223. https://doi.org/10.3390/medicina57030223
9. Rizzone, K. H., Ackerman, K. E., Roos, K. G., Dompier, T. P., & Kerr, Z. Y. (2017). The epidemiology of stress fractures in collegiate student-athletes, 2004–2005 through 2013–2014 academic years. *Journal of Athletic Training, 52*(10), 966–975. https://doi.org/10.4085/1062-6050-52.8.01
10. Daponte, V., Henke, K., & Drissi, H. (2024). Current perspectives on the multiple roles of osteoclasts: Mechanisms of osteoclast-osteoblast communication and potential clinical implications. *eLife, 13*, e95083. https://doi.org/10.7554/eLife.95083
11. Kini, U., & Nandeesh, B. N. (2012). Physiology of bone formation, remodeling, and metabolism. In I. Fogelman, G. Gnanasegaran, & H. van der Wall (Eds.), *Radionuclide and hybrid bone imaging* (pp. 29–57). Springer. https://doi.org/10.1007/978-3-642-02400-9_2

12. Matcuk, G. R., Jr., Mahanty, S. R., Skalski, M. R., Patel, D. B., White, E. A., & Gottsegen, C. J. (2016). Stress fractures: Pathophysiology, clinical presentation, imaging features, and treatment options. *Emergency Radiology, 23*(4), 365–375. https://doi.org/10.1007/s10140-016-1390-5
13. Burr, D. B. (2002). Targeted and nontargeted remodeling. *Bone, 30*(1), 2–4. https://doi.org/10.1016/S8756-3282(01)00619-6
14. Parfitt, A. M. (2002). Targeted and nontargeted bone remodeling: Relationship to basic multicellular unit origination and progression. *Bone, 30*(1), 5–7. https://doi.org/10.1016/S8756-3282(01)00642-1
15. Beck, B., & Drysdale, L. (2021). Risk factors, diagnosis and management of bone stress injuries in adolescent athletes: A narrative review. *Sports, 9*(4), 52. https://doi.org/10.3390/sports9040052
16. Hughes, J. M., Castellani, C. M., Popp, K. L., Guerriere, K. I., Matheny, R. W., Jr., Nindl, B. C., & Bouxsein, M. L. (2020). The central role of osteocytes in the four adaptive pathways of bone's mechanostat. *Exercise and Sport Sciences Reviews, 48*(3), 140–148. https://doi.org/10.1249/JES.0000000000000225
17. Frost, H. M. (2003). Bone's mechanostat: A 2003 update. *The Anatomical Record Part A: Discoveries in Molecular, Cellular, and Evolutionary Biology, 275A*(2), 1081–1101. https://doi.org/10.1002/ar.a.10119
18. Warden, S. J., Burr, D. B., & Brukner, P. D. (2006). Stress fractures: Pathophysiology, epidemiology, and risk factors. *Current Osteoporosis Reports, 4*(3), 103–109. https://doi.org/10.1007/s11914-996-0029-y
19. Warden, S. J., Davis, I. S., & Fredericson, M. (2014). Management and prevention of bone stress injuries in long-distance runners. *Journal of Orthopaedic & Sports Physical Therapy, 44*(10), 749–765. https://doi.org/10.2519/jospt.2014.5334
20. McCormick, F., Nwachukwu, B. U., & Provencher, M. T. (2012). Stress fractures in runners. *Clinics in Sports Medicine, 31*(2), 291–306. https://doi.org/10.1016/j.csm.2011.09.012
21. Hoenig, T., Hollander, K., Popp, K. L., Fredericson, M., Kraus, E. A., Warden, S. J., Tenforde, A. S., & Bone Stress Injury Authorship Group. (2025). International Delphi consensus on bone stress injuries in athletes. *British Journal of Sports Medicine, 59*(2), 78–90. https://doi.org/10.1136/bjsports-2024-108616
22. Tenforde, A. S., Kraus, E., & Fredericson, M. (2016). Bone stress injuries in runners. *Physical Medicine and Rehabilitation Clinics of North America, 27*(1), 139–149. https://doi.org/10.1016/j.pmr.2015.08.008
23. Nieves, J. W., Melsop, K., Curtis, M., Kelsey, J. L., Bachrach, L. K., Greendale, G., Sowers, M. F., & Sainani, K. L. (2010). Nutritional factors that influence change in bone density and stress fracture risk among young female cross-country runners. *PM&R, 2*(8), 740–794. https://doi.org/10.1016/j.pmrj.2010.04.020
24. Mountjoy, M., Ackerman, K. E., Bailey, D. M., Burke, L. M., Constantini, N., Hackney, A. C., Heikura, I. A., Melin, A., Pensgaard, A. M., Stellingwerff, T., Sundgot-Borgen, J. K., Torstveit, M. K., Jacobsen, A. U., Verhagen, E., Budgett, R., Engebretsen, L., & Erdener, U. (2023). 2023 International Olympic Committee's (IOC) consensus statement on relative energy deficiency in sport (REDs). *British Journal of Sports Medicine, 57*(17), 1073–1097. https://doi.org/10.1136/bjsports-2023-106994
25. De Souza, M. J., Nattiv, A., Joy, E., Misra, M., Williams, N. I., Mallinson, R. J., Gibbs, J. C., Olmsted, M., Goolsby, M., Matheson, G., & Expert Panel. (2014). 2014 Female Athlete Triad Coalition consensus statement on treatment and return to play of the female athlete triad: 1st international conference held in San Francisco, California, May 2012 and 2nd international conference held in Indianapolis, Indiana, May 2013. *British Journal of Sports Medicine, 48*(4), 289. https://doi.org/10.1136/bjsports-2013-093218
26. Cabre, H. E., Moore, S. R., Smith-Ryan, A. E., & Hackney, A. C. (2022). Relative energy deficiency in sport (RED-S): Scientific, clinical, and practical implications for the female athlete. *Deutsche Zeitschrift für Sportmedizin, 73*(7), 225–234. https://doi.org/10.5960/dzsm.2022.546
27. Warden, S. J., Edwards, W. B., & Willy, R. W. (2021). Preventing bone stress injuries in runners with optimal workload. *Current Osteoporosis Reports, 19*(3), 298–307. https://doi.org/10.1007/s11914-021-00666-y
28. Kahanov, L., Eberman, L. E., Games, K. E., & Wasik, M. (2015). Diagnosis, treatment, and rehabilitation of stress fractures in the lower extremity in runners. *Open Access Journal of Sports Medicine, 6*, 87–95. https://doi.org/10.2147/OAJSM.S39512
29. Patel, D. S., Roth, M., & Kapil, N. (2011). Stress fractures: Diagnosis, treatment, and prevention. *American Family Physician, 83*(1), 39–46.
30. May, T., & Marappa-Ganeshan, R. (2023). Stress fractures. In *StatPearls*. StatPearls Publishing.
31. Astur, D. C., Zanatta, F., Arliani, G. G., Moraes, E. R., Pochini, A. de C., & Ejnisman, B. (2015). Stress fractures: Definition, diagnosis and treatment. *Revista Brasileira de Ortopedia, 51*(1), 3–10. https://doi.org/10.1016/j.rboe.2015.12.008
32. Mayer, S. W., Joyner, P. W., Almekinders, L. C., & Parekh, S. G. (2014). Stress fractures of the foot and ankle in athletes. *Sports Health, 6*(6), 481–491. https://doi.org/10.1177/1941738113486588
33. Pietrzak, K., Wasilewska, M., & Polok, S. (2025). Fatigue fractures in recreational and professional athletes: Risk factors, prevention, and recovery strategies. *International Journal of Innovative Technology and Social Science, 1*, Article 3466. https://doi.org/10.31435/ijitss.3(47).2025.3466
34. Miller, T. L., & Best, T. M. (2016). Taking a holistic approach to managing difficult stress fractures. *Journal of Orthopaedic Surgery and Research, 11*(1), 98. https://doi.org/10.1186/s13018-016-0431-9

35. Robertson, G. A., & Wood, A. M. (2017). Lower limb stress fractures in sport: Optimising their management and outcome. *World Journal of Orthopedics, 8*(3), 242–255. https://doi.org/10.5312/wjo.v8.i3.242
36. Komatsu, J., Mogami, A., Iwase, H., Obayashi, O., & Kaneko, K. (2019). A complete posterior tibial stress fracture that occurred during a middle-distance running race: A case report. *Archives of Orthopaedic and Trauma Surgery, 139*(1), 25–33. https://doi.org/10.1007/s00402-018-3035-5
37. Hadjispyrou, S., Hadjimichael, A. C., Kaspiris, A., Leptos, P., & Georgoulis, J. D. (2023). Treatment and rehabilitation approaches for stress fractures in long-distance runners: A literature review. *Cureus, 15*(11), e49397. https://doi.org/10.7759/cureus.49397
38. McInnis, K. C., & Ramey, L. N. (2016). High-risk stress fractures: Diagnosis and management. *PM&R, 8*(3 Suppl.), S113–S124. https://doi.org/10.1016/j.pmrj.2015.09.019
39. Hollander, J. J., Rikken, Q. G. H., Dahmen, J., Stufkens, S. A. S., & Kerkhoffs, G. M. M. J. (2021). High union rates following surgical treatment of proximal fifth metatarsal stress fractures. *Knee Surgery, Sports Traumatology, Arthroscopy, 29*(8), 2495–2503. https://doi.org/10.1007/s00167-021-06490-2
40. Mallee, W. H., Weel, H., van Dijk, C. N., van Tulder, M. W., Kerkhoffs, G. M., & Lin, C. W. C. (2015). Surgical versus conservative treatment for high-risk stress fractures of the lower leg (anterior tibial cortex, navicular and fifth metatarsal base): A systematic review. *British Journal of Sports Medicine, 49*(6), 370–376. https://doi.org/10.1136/bjsports-2013-093246
41. Lappe, J., Cullen, D., Haynatzki, G., Recker, R., Ahlf, R., & Thompson, K. (2008). Calcium and vitamin D supplementation decreases incidence of stress fractures in female navy recruits. *Journal of Bone and Mineral Research, 23*(5), 741–749. https://doi.org/10.1359/jbmr.080102
42. Beck, B., & Drysdale, L. (2021). Risk factors, diagnosis and management of bone stress injuries in adolescent athletes: A narrative review. *Sports, 9*(4), 52. https://doi.org/10.3390/sports9040052
43. Gao, Y., Liu, X., Gu, Y., Song, D., Ding, M., Liao, L., Wang, J., Ni, J., & He, G. (2021). The effect of bisphosphonates on fracture healing time and changes in bone mass density: A meta-analysis. *Frontiers in Endocrinology, 12*, 688269. https://doi.org/10.3389/fendo.2021.688269
44. Jones, B. H., Thacker, S. B., Gilchrist, J., Kimsey, C. D., Jr., & Sosin, D. M. (2002). Prevention of lower extremity stress fractures in athletes and soldiers: A systematic review. *Epidemiologic Reviews, 24*(2), 228–247. https://doi.org/10.1093/epirev/mxf011
45. Close, G. L., Sale, C., Baar, K., & Bermon, S. (2019). Nutrition for the prevention and treatment of injuries in track and field athletes. *International Journal of Sport Nutrition and Exercise Metabolism, 29*(2), 189–197. https://doi.org/10.1123/ijsnem.2018-0290
46. Lavigne, A., Chicoine, D., Esculier, J. F., Desmeules, F., Frémont, P., & Dubois, B. (2023). The role of footwear, foot orthosis, and training-related strategies in the prevention of bone stress injuries: A systematic review and meta-analysis. *International Journal of Exercise Science, 16*(3), 721–743. https://doi.org/10.70252/ZNRS2138