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Dolna 17, Warsaw, Poland 00-773 +48 226 0 227 03 editorial_office@rsglobal.pl

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BONE STRESS INJURIES IN RUNNERS - A HOLISTIC APPROACH

Alicja Toczyłowska (Corresponding Author, Email: alicja.toczylowska9@gmail.com) University Clinical Hospital in Opole, aleja Wincentego Witosa 26, 46-020 Opole, Poland ORCID ID: 0009-0007-3155-0573

Mateusz Muras

University Clinical Hospital in Opole, aleja Wincentego Witosa 26, 46-020 Opole, Poland ORCID ID: 0009-0003-4536-6006

Władysław Hryniuk

University Clinical Hospital in Opole, aleja Wincentego Witosa 26, 46-020 Opole, Poland ORCID ID: 0009-0009-8653-468X

Julia Kwiecińska

University Clinical Hospital in Opole, aleja Wincentego Witosa 26, 46-020 Opole, Poland ORCID ID: 0009-0004-0924-6063

Jacek Sitkiewicz

Silesian Centre for Heart Diseases in Zabrze, Marii Skłodowskiej-Curie 9, 41-800 Zabrze, Poland ORCID ID: 0009-0006-0889-0652

Łukasz Bialic

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warszawa, Poland ORCID ID: 0000-0003-4837-5920

Lidia Mądrzak

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warszawa, Poland ORCID ID: 0009-0005-9516-911X

Marta Korchowiec

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warszawa, Poland ORCID ID: 0009-0008-3365-4728

Wiktor Chrzanowski

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warszawa, Poland ORCID ID: 0009-0008-0820-1452

Katarzyna Krzyżanowska

Medical University of Warsaw, Żwirki i Wigury 61, 02-091 Warszawa, Poland ORCID ID: 0009-0009-3306-0804

ABSTRACT

Objective: Bone stress injuries (BSIs), ranging from periosteal edema to stress fractures, are highly prevalent in runners due to repetitive loading exceeding bone adaptation capacity. Delayed diagnosis prolongs recovery. This review synthesizes current knowledge on BSI pathophysiology, risk factors, diagnosis, management, and prevention in runners, highlighting the complexity of the problem.

Methods: A literature review was conducted using publicly available sources accessed via PubMed and Scopus.

Key Findings: BSIs arise from an imbalance between microdamage accumulation and repair, influenced by biological and biomechanical factors. Magnetic resonance imaging (MRI) is the diagnostic gold standard, recommended after initial plain radiography. Management prioritizes conservative measures; surgery is reserved for high-risk fractures (e.g., prone to nonunion) or conservative failure. Prevention strategies include individualized training programs, nutritional optimization, and preparticipation screening.

Conclusions: Early diagnosis and a multidisciplinary approach—focusing on risk factor identification, timely intervention, treatment, and prevention—are crucial for reducing BSI incidence and accelerating recovery in runners. Further research is needed to validate novel treatments and standardize risk factor detection systems for injury prevention.

KEYWORDS

Bone Stress Injuries, Stress Fractures, Runners, Risk Factors, Pathophysiology, Prevention

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Introduction.

Running is a widely practiced form of physical activity, which offers numerous health benefits but is also associated with a high rate of overuse injuries, estimated between 19% and 80% [10]. Bone stress injuries (BSIs) arise from the inability of bone to withstand repetitive mechanical loading, leading to structural fatigue and local bone pain. BSIs range in severity. Radiographic findings of early injuries show periosteal edema with varying degrees of marrow edema, while advanced cases manifest fracture lines indicative of stress fractures (SF) [2, 5, 6]. Stress fractures account for 10%-20% of all sports medicine injuries and were first described in the mid-19th century among Prussian military recruits as "march fractures", attributed to metatarsal injuries secondary to excessive training during recruitment periods [1, 12]. Delayed diagnosis may result in more serious injuries, which require protracted therapeutic interventions. This underscores the importance of early BSI detection in clinical management [2, 6].

Methodology

For this narrative review all data were collected from publicly available sources. Relevant literature published between 2015 and 2025 was identified using PubMed and Scopus in May 2025. Only studies in English were retrieved. A comprehensive literature review was conducted, analyzing peer-reviewed studies on BSIs in runners. Databases were searched for articles addressing pathophysiology, imaging modalities, risk factors, treatment protocols, and prevention strategies. Combination of keywords used to identify relevant articles included terms, such as: "bone stress injuries", "stress fractures", "risk factors", "diagnosis", "prevention", "nutrition". Emphasis was placed on clinical guidelines and epidemiological data to develop an evidence-based framework for BSI management.

One meeting of the whole team was conducted in order to review included studies.

Results

Eventually all studies were sorted into six themes: Pathophysiology, Risk Factors, Diagnosis and Assessment, Imaging, Management, Prevention.

Pathophysiology

BSI is a broad term that includes a range of bone tissue disorders resulting from prolonged repetitive loading from periostitis, to periosteal, endosteal and bone tissue oedema, to partial or complete stress fracture. All of them can be collectively referred to as bone stress reactions or bone stress injuries. It is worth noting, however, that a stress reaction usually marks a less severe stage than a stress fracture. SF are focal structural weakness in bone occurring due to repeated stresses below the fracture threshold [22].

To understand the pathophysiology of SF, a basic knowledge of bone metabolism and structure is essential. In adults, lamellar bone predominates, consisting of cortical bone (80%) and cancellous bone (20%). Cortical bone, found primarily in the diaphyses of long bones and the outer shell of cuboid-like bones (e.g., vertebral bodies), is the most common site of stress fractures in runners [1]. Cancellous bone, located in the metaphyses of long bones and the center of cuboid-like bones, is less dense, undergoes more rapid turnover and stress remodeling, compared to cortical bone [1].

Wolff's law states that upon stress, a bone deforms through the bone's elastic range and returns to its initial conformation if the stress stops. However, if the stress continues to stay beyond the elastic range, it creates microfractures and a persistent plastic deformity. If unrepaired, these microfractures coalesce into cortical discontinuities referred to as stress fractures [1, 12].

Typically there is an equilibrium between new microdamage formation and removal of the previous ones. In cortical bone, osteoclast activation and resorption takes approximately 4 weeks, with full mineralization taking up to a year. Trabecular bone requires even longer healing periods, potentially explaining prolonged recovery times for trabecular-rich site BSIs [3].

A stress fracture, in its most comprehensive description, includes two types of fractures: both fatigue and insufficiency fractures. Fatigue fractures, sometimes equated with the term "stress fracture", are the type of fracture that occur most often in runners and other athletes, and usually involve the lower extremities. These fractures are most often caused by abnormal cyclic loading on normal bone, leading to local cortical resorption and fractures.

Insufficiency fractures, on the other hand, are common in older people. They arise secondary to osteoporosis and usually are located in and around the pelvis. They are caused by normal or traumatic loading on pathological bones [1, 12].

Risk Factors

Biological Factors:

Female sex increases susceptibility to stress fractures. A meta-analysis of distance runners revealed a 2.3 times greater SF risk in females, attributed partially to decreased lean mass and less robust bone morphology [12].

Another factor is the female athlete triad, first described in 1992 as a combination of oligomenorrhea/amenorrhea, low bone mass, and energy deficit. In 2007, the triad's definition was expanded to include a spectrum of dysfunction: menstrual irregularities, reduced bone mineral density (BMD), and low energy availability (EA), with athletes now classified even if only 1–2 features are present [15]. Menstrual dysfunction affects 12–79% of active females [15], and in runners, training duration correlates positively with its occurrence. Amenorrhea is observed in up to 60% of those exceeding 70 miles per week (about 115 km) [15]. Low EA is the triad's underlying cause, occurring with or without disordered eating. A similar pattern [low EA, low body mass index (BMI), low BMD, and elevated BSI rates] has been noted in male athletes, though diagnosis is complicated by the absence of physical signs like amenorrhea [21].

Genetic predisposition may contribute to SF development. The P2X7 receptor (P2X7R) gene, a key regulator of bone remodeling, has been linked to SF susceptibility. Studies in two distinct populations identified independent associations between SFs and functional polymorphisms (rs3751143 and rs1718119, no linkage disequilibrium was shown in either cohort) in this gene [16].

Medications or treatments that may have adverse effects on BMD include glucocorticoids, anticonvulsants, antidepressants, methotrexate, antiretrovirals and radiation therapy. However, there is no specific evidence linking any medication with BSI in adolescent athletes as no studies have been undertaken [22].

While some studies suggest oral contraceptives (OCPs) protect female runners from SFs, their relationship with bone density remains debatable [15]. It has been shown, however, that medroxyprogesterone acetate (DMPA) negatively impacts bone health. DMPA use within the last 2 years correlates with higher SF risk compared to oral contraception use or no hormonal contraception [15].

Dietary contributors, such as low-fat diets, insufficient calcium and vitamin D, elevate SF risk. Low EA, excessive exercise and disordered eating disrupt menstrual function which results in low BMD [5, 13].

Biomechanical Factors:

Training patterns significantly influence SF risk. Short stance phases and high peak loads are potential risk factors for BSI [19]. A change in the nature and/or intensity of mechanical loading can result in the accumulation of bone microdamage and may progress to

a BSI [23]. It has also been reported that runners averaging more than 40 miles (about 67 km) per week face greater risk [15].

Bone morphology is another biomechanical factor. Studies have shown that narrower bones and a thinner cortex are risk factors for fractures [5, 11]. Small tibial width was shown to be a risk factor which partially explains gender differences in SF risk [15]. While studies of correlation of low BMD with higher rates of SF remain controversial, oligo/amenorrheic athletes with SFs had lower whole body and spine BMD detected [12].

Anatomic considerations include leg length discrepancy, lean mass, smaller calf cross-sectional area and foot type [5]. For example, a cavus foot can cause SF of the femur and metatarsal bones, while a flat foot increases pronation and SF of the tibia, the fibula and the tarsal bones. Varus alignment in the lower limb is a SF risk factor of the femur and the tarsal bones, and cavovarus feet predispose to SF because of a rigid foot shape that does not attenuate the impact [12]. Also certain features of footstrike biomechanics should be considered, such as increased forefoot valgus, a shift toward forefoot loading in general, and higher regional foot forces that may contribute to BSI. [18]

Body composition also matters: low body fat reduces estrogen levels, leading to osteoporosis and SF susceptibility [13]. BMI exhibits an indirect relationship with SFs, with risk peaks at $<19 \text{ kg/m}^2$ and $>30 \text{ kg/m}^2$ [12].

Other factors:

Other factors include the quality of footwear and equipment as well as type of the running surface [12]. Over the last two decades running shoe technologies were significantly modified not only to improve performance but also to reduce injury risk [8].

Some research suggests a link between sleep deprivation and suppressed bone formation, however, it has not been intensively studied. For example, 72 days of sleep restriction in rats resulted in a 45-fold reduction in osteoid-lined bone and reduced osteoid thickness compared to controls, indicating inhibited bone formation. In a cross-sectional analysis of sleep-deprived men and women (<6.5 h per night), sleep-deprived women had lower cortical volumetric bone mineral density, while sleep-deprived men had lower polar strength-strain indices than sleep-replete controls [11].

Psychological stress is another potential factor that may suppress bone formation. Elevated levels of circulating inflammatory cytokines and cortisol have been associated with low bone density, altered bone metabolism, and greater fracture risk [11].

Having a past SF increases the chances of a new one - in female runners by 5-6 times [12].

Early sport specialization (defined as intensive prepubertal participation in a single sport for more than 8 months per year at the expense of other sports) has been linked to an increased risk of overuse injuries, including BSIs. The period from birth to puberty represents a critical window for bone mass accrual, which may be compromised by such specialization [3].

Diagnosis and Assessment

When establishing a diagnosis of SF, a comprehensive medical history should be collected, including details about the patient's activity patterns, as individuals with SFs often present a prolonged, predictable history of pain [2]. The evaluation should also address underlying risk factors, such as prior SFs, dietary habits (particularly calcium and vitamin D intake), medications, coexisting conditions (e.g., eating disorders, depression, autoimmune diseases, endocrinopathies, malabsorption, bariatric surgery), and treatment history [12]. For female patients, assessing menstrual status, including past or present periods of amenorrhea, is critical [12]. Differential diagnoses, such as infections, periostitis, tendinitis, neoplasms, osteoid osteoma, exertional

compartment syndrome, nerve entrapment, avulsion injury, and intermittent claudication, should be carefully considered to avoid misdiagnosis [12, 28].

A thorough physical examination is essential. Stress fractures typically manifest as activity-related pain that resolves with rest, but worsens with continued exertion [1]. Key features of BSI include localized bone tenderness and pain on percussion. In deeper or less accessible areas to palpate (e.g., femoral neck), it is important to evaluate pain with range of motion of the joint. Pain may be elicited using functional tests, such as the single-leg hop test (which can be used to distinguish tibial SFs from medial tibial stress syndrome), or fulcrum test (useful in the diagnosis of femoral and tibial SFs). Severe cases may present swelling or skin discoloration [2, 12].

Diagnostic imaging and laboratory tests include MRI (the gold standard for confirming SFs), basic metabolic panels, thyroid function tests, vitamin D3 levels, 24-hour urinary calcium excretion, and BMD evaluation in cases of recurrent fractures [15].

It is essential to classify fractions by location based on the likelihood of uncomplicated healing with conservative management alone. Low-risk sites (e.g., the second through fourth metatarsal shafts, fibula/lateral malleolus, calcaneus, cuneiforms, cuboid and medial femoral neck compression fractures) often heal with conservative management, whereas high-risk sites (e.g., the pars interarticularis of the lumbar spine, femoral head, lateral femoral neck tension fractures, transverse fractures of patella, anterior cortex of the tibia, talar body, navicular, proximal second metatarsal, and great toe, or hallux sesamoids) require orthopedic consultation due risks of delayed union, nonunion, or progression to complete fractures [1, 2].

Imaging

Radiographs

Plain radiographs remain the first-line imaging modality for evaluating musculoskeletal injuries, pain, or suspected stress fractures [1]. However, their sensitivity is very low, due to planar characteristics and low resolution [2]. Therefore, it is difficult to diagnose early stress fractures and the setting of osteopenia. When visible, radiographic findings of a stress fracture may include subtle linear sclerosis, focal periosteal or endosteal reaction, or a cortical fracture with superimposed periosteal reaction [1].

Computed tomography (CT)

Computed tomography (CT) can be used in detecting longitudinal fracture lines and has the added benefit of orthogonal reformations. It is particularly useful for differentiating lesions seen on a bone scan that can mimic stress fracture, including osteomyelitis, osteoid osteoma and malignancy. However, CT lacks sensitivity for transverse fractures which can cause diagnostic limitations [1, 25].

Magnetic resonance imaging (MRI)

Magnetic resonance imaging (MRI) is the gold standard for diagnosing BSIs, offering exceptional sensitivity (100%) and specificity (85%) [1]. However it is assumed as a second-line modality. It is typically employed when radiographs are inconclusive, pain etiology is unclear, or athletes require definitive diagnosis [1]. MRI stands out with visualizing edema of both bone tissue and soft tissue adjacent areas, which are early indicators of stress injury, detectable within 1–2 days of symptom onset [15]. Among all modalities, MRI provides the most comprehensive assessment of BSIs, providing both functional and morphologic information about the bone with no radiation needed [1], [22].

Injury severity can be classified with The Fredericson MRI grading system: **Grade 1** BSI shows periosteal edema on T2 weighted imaging, with normal bone marrow on T1 and T2. **Grade 2** BSI shows moderate to severe periosteal edema on T2 and bone marrow edema on T2. **Grade 3** BSI shows moderate to severe periosteal edema on T2 and marrow edema on T2 and T1 **Grade 4** shows visible fracture line, commonly referred to as a stress fracture [5, 7].

Imaging findings allow also another BSIs grading. In this system, grades 1 and 2 are low-grade BSIs, whereas grades 3 and 4 are classified as high-grade BSIs. After low- and high-grade BSIs, the time allowing athletes to return to sports was reported to be 13.1 and 23.6 weeks, respectively [2].

MRI also plays an important role in diagnosing subchondral insufficiency fractures which cannot be visualized on radiographs, unless there is a linear subchondral lucency or collapse [1].

Ultrasound

Ultrasound is emerging as a portable and efficient tool, particularly for superficial bones. It can detect cortical irregularities, such as cortical buckling and surrounding hypoechoic callus. [1]. While not yet validated, power Doppler may help assess vascularity, an increase in which may be connected with acuity of bone injury [1]. Its accessibility makes it valuable for on-field evaluations, enabling early detection of BSIs, and follow-up in sports medicine [2].

Bone scintigraphy

Three-phase bone scintigraphy with Technetium-99m-methylene diphosphonate demonstrates high sensitivity for metabolic bone activity but poor specificity, with up to 40 % of increased tracer uptake detected at asymptomatic sites [1]. Increased uptake can also be caused by other pathologies (e.g., avascular necrosis, osteomyelitis, neoplasm), which contributes to low specificity [12]. Bone scintigraphy provides diagnostic benefits when patients are suspected of having multiple SFs simultaneously [12]. However, it has lost its diagnostic value, preferring the use of non-ionising radiation sources of MRI [22].

PET/CT

PET/CT also detects stress fractures. Some patterns of uptake like the "Honda sign" in sacral insufficiency fractures, achieving 96% sensitivity and 92% positive predictive value, can be useful in a diagnostic process [1].

Management

Critical to recovery is addressing modifiable risk factors (e.g., biomechanical imbalances, nutrition) and ensuring early diagnosis, as delayed treatment prolongs healing [22].

It is crucial for management of injuries to correlate the severity of stress injury with recovery time, as having a reliable expectation of time to return to sports after these injuries can alleviate anxiety of athletes, coaches and sports medicine providers [17].

The initial management of most BSIs is non-surgical, focusing on offloading the affected bone, physical therapy, and a gradual return to activity, provided exercises remain pain-free [7]. Surgical intervention is typically reserved for fractures prone to delayed union or nonunion, or when conservative treatment fails [13].

Biopsy is generally avoided due to the risk of histopathological confusion with aggressive tumors like parosteal osteosarcoma, due to the presence of osteoblastic reparative callus. Additionally, biopsies may further weaken the bone, increasing fracture risk [1].

Phase 1 in management begins with emphasizing pain control through cold massage, physiotherapy, and analgesics, while avoiding nonsteroidal anti-inflammatory drugs (NSAIDs) due to their potential negative effect on bone healing [2]. In the initial management of low-risk BSIs, it is recommended to temporarily discontinue activities, such as running, and modify the exercises performed. However, weight-bearing is permitted for daily activities if tolerable. [2, 15]. Walking boots, canes, or crutches are recommended for athletes who have difficulty walking pain-free. Different types of specialized shoes can also be helpful. Rigid-soled shoes are recommended for forefoot and midfoot BSI, while cushioned shoes or insoles - for rearfoot and leg BSI [2].

Maintaining cardiovascular fitness during recovery is critical. Conditioning exercises, such as swimming, cycling, deep-water running, or antigravity treadmill are recommended, with the latter two probably the most beneficial for runners, since endurance training athletes experience cardiovascular insufficiency within two weeks after training discontinuation. Such programs are implemented to maintain physical activity but are unlikely to enhance healing of the BSI [2, 22].

Phase 2 is followed by 10–14 pain-free days, introducing a progressive running program at half the original speed and distance one week after the loss of bony tenderness. It is then gradually increased to pre-injury levels over 3–6 weeks under supervision. Such treatment, however, is reserved for low-risk BSIs [2, 15].

High-risk BSIs may require prolonged activity modification, non-weight-bearing protocols, or surgical fixation, with confirmation of complete healing before returning to sport [2].

To maintain physical activity an antigravity treadmill (ATT) may prove useful. It enables progressive impact loading to maintain fitness while allowing healing of lower extremity BSIs. Antigravity treadmills consist of a treadmill equipped with a pressure-controlled chamber that surrounds the lower body and is filled with air. They enable adjustable body weight support and enhance fitness during exercise while protecting the BSI site [2, 5].

Foot

Foot stress fractures may occur after repeated strain on any bone of the foot. However, the most common anatomic sites involved are the metatarsal and tarsal bones, as well as the navicular and sesamoid bones [4]. If foot SF is suspected, it is also essential to examine the entire lower limb to search for any anatomic features of malalignment in all planes [27]. Typically, foot SFs respond well to conservative treatment. Conservative options include rest, the use of assistive devices for weight-bearing, immobilization with a cast, or a walking boot, activity modification and physical therapy interventions [4].

Fibula

Stress fractures of the fibula account for 6.6% of lower limb stress fractures [26]. Fibular SFs may involve the entire length of the bone, but most commonly occur in the middle and distal third [4]. Treatment and rehabilitation typically follow a conservative approach due to reduced mechanical loads and lower frequency of SFs in this bone. Conservative management may involve rest, the use of assistive devices and partial weight-bearing [4].

Femur

The femur accounts for approximately 5 to 7% of all stress fractures [29]. Femoral SFs are categorized by anatomical location: the femoral neck, femoral shaft, or condyles [4]. The most common symptom is anterior groin pain, usually with an onset related to intensive exercise [4]. Treatment and rehabilitation of femoral SFs can vary depending on the specific case.

Although not always consistent, most studies indicate the femoral neck is the most common part of the femur at risk of fracture [29], with both conservative and operative treatments described in the literature. Conservative management includes rest, partial weight-bearing, and physical therapy to promote healing [4]. Operative options, such as internal fixation with a dynamic hip screw, may be used in certain cases for stability and faster healing [4]. Early diagnosis of femoral neck SFs is crucial to prevent displacement, which is associated with complications like avascular necrosis and malunion, potentially impacting hip function and athletic performance [14].

Pelvis

Pelvic SFs are uncommon in long-distance runners, typically occurring in the pubic ramus near the symphysis, though cases in the sacrum, the acetabulum, and the iliac bone have been reported [4]. The symptoms are mainly lower back or groin pain. Pelvic SF are at highest risk of poor or delayed healing, however, most of them are treated conservatively due to their low-morbidity classification. Studies show positive outcomes with this kind of treatment, allowing runners to regain activity within four weeks to one year [4, 22].

Patella

Patellar SFs can be longitudinal or transverse. They are rare and challenging to diagnose due to similar symptoms of conditions, such as patellar tendonitis. Accurate differentiation from other causes of anterior knee pain is critical. Treatment varies based on severity. Conservative approaches are often effective, but surgery may be needed if conservative measures fail [4]. Reported surgical treatment options involve internal fixation with the tension band technique, the cannulated screw technique, curettage, bone grafting, or drilling [30].

There are therapies that show promise in accelerating fracture healing: adjunctive therapies like lowintensity pulsed ultrasound (LIPUS) and extracorporeal shockwave therapy (ESWT). LIPUS has been proven to accelerate bone formation in fresh fractures, delayed unions, nonunions, and distraction osteogenesis through various molecular, biological and biomechanical interactions in fracture areas [24]. ESWT, on the other hand, generates forces that create periosteal detachment and trabecular microfractures with hemorrhage, thus promoting mesenchymal stem cells into osteoblasts and stimulating osteogenesis [7]. However, there is sparse literature associated with ESWT efficiency in treating stress fractures [13].

There are also medications that show promise in bone healing. Denosumab (a RANKL inhibitor used in the treatment of postmenopausal osteoporosis) may enhance cortical bone healing. Promising drugs also include abaloparatide (an analog of PTHrP) and romosozumab (an antibody against sclerostin). There was no study that has specifically been conducted to evaluate the effect of teriparatide (a parathyroid hormone analog) on stress fractures, however, it might be relevant to the hypothesis that fracture healing is accelerated when bone remodeling is enhanced [12, 15].

Prevention

Appropriate training regime

Training progression should be carefully managed. Beginners are advised to limit weekly distance increases to no more than 10%, and to avoid exceeding 160 km total volume within a three-month period [14].

It is suggested that emphasizing low velocity, high volume training (e.g., "train slow to race fast") may be safer for bone health. In a probabilistic model it has been shown that reducing running speed from 3.5 to 2.5 m/s, given the same distance, can halve tibial stress fracture likelihood [3].

Periodization strategies, which alternate loading phases with rest blocks involving low-impact activities, such as cycling or swimming, can restore bone mechanosensitivity and enhance adaptation. In animal studies, periodized loading protocols achieved superior bone adaptation despite delivering one-third less cumulative load compared to continuous training [3]. Running or activity apps can be useful for athletes to monitor their loading in order to avoid spikes in their training activities [9].

In seasonal sports (e.g., outdoor track), the period that is most dangerous in terms of BSI is the preseason and the period when training intensity increases from preseason to competition. It is suggested that progressive pre-season conditioning and workload reductions post-season are crucial to mitigating injury risk spikes during transitions to competition [3].

To improve lower limb bone health, a jumping program can be used to exceed one-way loads associated with running (which are insufficient to improve bone health and reduce the risk of later BSIs). It is recommended for a jumping program to apply progressively higher loads, include several repetitions of the load per session, and take place several times a day for at least 3 days a week [6]. It is also believed that bones respond well to unusual or novel forms of loading [22].

It is suggested that improving muscular endurance and strength may benefit runners with BSI risk. Therefore, implementing heavy resistance training into a training program should be considered and encouraged at all stages of life to either maximise peak bone mass or slow its decline in later years. Higher bone density was observed in runners who followed such a plan instead of solely doing their sport. However, much of the evidence supporting resistance training in reducing BSI risk is retrospective [3, 22].

Pretraining may prove to be an important part of a training plan. In Israeli elite infantry recruits who played ball sports immediately prior to basic training a 50% reduction in stress fracture incidence was noted. Such findings highlight the value of pretraining conditioning to promote adaptive bone formation [11].

Before adolescent pubertal growth period, it is recommended for athletes to implement optimal bone workload that consists of low-repetitions of fast, high-magnitude, multidirectional, novel loads introduced a few times per day. Such workload promotes beneficial adaptation to enhance function and reduces the risk of an injury. It is worth noting, however, that optimal bone workload should be considered individually. [3].

For adults with a mature skeleton, workload tracking is recommended in order to avoid acute spikes. Rest periods are an important part of a training program and are recommended at least 1 day/week as well as 1 week every 3 months. This optimal workload enables gains in running performance while minimizing the accumulation of bone damage [3].

Bone workload may be altered through gait retraining. It typically involves the use of devices which allow athletes to measure the targeted biomechanical variable and provide external visual, verbal, or auditory cues to facilitate change. Currently involved techniques include increasing step rate, transitioning an athlete to a forefoot strike pattern as well as cueing a softer and quieter landing by providing feedback on peak positive tibial acceleration [3, 31].

Nutrition and supplementation

Nutritional strategies play a pivotal role in bone health. Maintaining adequate energy availability (EA) is critical, as bone formation declines at 30 kcal·kg LBM^{-1·}day⁻¹, with severe reductions to 10 kcal·kg LBM^{-1·}day⁻¹ further impairing bone health by both suppressing bone formation and increasing bone resorption. This is particularly concerning knowing that some amenorrheic athletes have been reported to have EA as low as ~16 kcal·kg LBM^{-1·}day⁻¹ [20]. While a target of 45 kcal·kg LBM^{-1·}day⁻¹ is recommended, it remains impractical for many endurance athletes (e.g., cyclists, marathon runners) due to high training energy demands and limited fueling opportunities. A calorie deficit, though often linked to enhancing endurance adaptations, necessitates identifying EA thresholds that minimize bone health risks. A 9-year case study of an elite female athlete proposed a balanced strategy: maintaining slightly above race weight with sufficient EA for most of the year, then strategically reducing

intake to achieve race weight temporarily. This approach allows periodic low EA for adaptation while avoiding year-round deficits, maintaining bone health and high quality performance [20].

Adequate supply of calcium, vitamin D, protein, magnesium, phosphorus, potassium, and fluoride is essential to support bone formation. Other key nutrients for bone health include manganese, zinc, copper, boron, iron, vitamin A, vitamin K, vitamin C, the B vitamins and silicon. These nutrients can be found in dairy, fruits, and vegetables (particularly of the green leafy kind) [20]. It is advised to meet daily calcium and vitamin D intake levels published by the Institute of Medicine to optimize bone health [5]. Supplementation with calcium (2,000 mg/day) and vitamin D (800 IU/day) has been shown to reduce stress fracture risk in military recruits [20]. The value of calcium supplementation is underscored by the fact that female runners consuming 800 mg of calcium daily are six times more likely to develop stress fractures than those who consume 1,500 mg daily [2, 5].

Recommended daily allowance of protein is $0.8 \text{ g} \cdot \text{kg BM}^{-1} \cdot \text{day}^{-1}$. However, athletes are advised to consume more protein than the recommendations state - many of them consume 2–3 times this amount [20]. Also a small-scale association study underscores the importance of adequate carbohydrate intake in injury prevention [20].

Bone health monitoring

Bone health monitoring should include screening for low BMI, low EA, prior bone stress injuries, and evaluating BMD during preparticipation evaluations [21]. Notably, 40% of female adolescent cross-country runners display reduced spine areal bone mineral density, having z-score < -1 on a dual-energy x-ray absorptiometry scan [3]. Such a procedure may help assess a risk of future bone stress injuries [21].

Preparticipation screening

Preparticipation screening is essential for an athlete's health. It enables the examination of some behavior and activity patterns, which include: smoking, drinking more than 10 bottles per week, excessive physical activity with insufficient time for rest, sudden increases in physical activity, and running on hard surfaces [2].

Identification and prevention of female athlete triad

In adolescent female athletes, identifying and correction of energy imbalance, as well as recovery of menstrual function, could be the most optimal strategy to maintain proper bone health [2]. Taking into account that low EA is the underlying cause of cascading triad conditions (menstrual dysfunction, low BMD and BSI), as a primary intervention recommendations favour restoration of EA over hormone replacement therapy [21].

Biomechanical screening

Biomechanical screening can help identify risk factors for BSI. It allows athletes to implement preventive strategies, such as changing lower extremity kinematics and sporting technique. Regular musculoskeletal screening is recommended to identify developing BSIs throughout the training season. It is possible then to implement activity modification or withdraw from training for a period of time [22].

Taking extrinsic risk factors into account

It is recommended that running shoes should be changed every 300 to 350 miles of use, depending on the type of shoe, the surface, and the individual characteristics of the athlete. Orthotics can benefit people with overly pronated or supinated feet [12].

Enhancing bone development in childhood

Young athletes should avoid premature sports specialization. Instead, it is recommended to encourage multidirectional sports involving jumping or rapid direction changes (like soccer or basketball) in order to enhance bone density, optimize bone structure, and develop a robust skeleton capable of withstanding multidirectional loading [3].

Discussion

Bone injuries in runners require a multidisciplinary approach to management and prevention. The findings of this review align with the existing literature, which emphasizes the crucial role of early diagnosis in order to prevent severe complications, such as progression to complete fractures. MRI, as the gold standard for BSI diagnosis, offers the highest sensitivity in detecting early-stage injuries, especially when radiographs remain inconclusive. However, reliance on advanced imaging modalities such as MRI may not always be feasible in resource-limited settings, underscoring the need for accessible diagnostic tools such as ultrasound, which shows promise for superficial bone assessment.

Identification of risk factors underscores the multifactorial nature of BSIs. Biological factors, such as female sex, medications, and genetic predispositions (e.g., P2X7R polymorphisms), interact with biomechanical factors, such as training patterns, bone morphology, and anatomic considerations. A recently observed similar constellation of signs and symptoms of female athlete triad in male athletes, consisting of low EA, low BMI, low BMD and a higher observed rate of bone stress injuries, challenges clinicians to improve screening protocols, as low EA in male athletes lacks a physical sign. Also, the role of psychological stress and sleep deprivation in bone health, though understudied, introduces new possibilities in a holistic approach.

Management of most BSI is conservative. Surgical intervention is considered in cases with high risk of delayed union or nonunion, or when conservative treatment fails. There are also new treatment options that show promise (eg., ESWT, LIPUS) or drugs (eg., romosozumab). However, they require further validation in randomized, controlled trials. The differences in recovery time between various BSIs underscore the need for personalized rehabilitation plans.

Prevention is one of the most important aspects of dealing with BSI. Tailored training programs, nutritional optimization (e.g., calcium and vitamin D supplementation), bone health monitoring, and preparticipation screening are key to athletes' health. Optimal bone workload in adolescence, that consists of low-repetitions of fast, high-magnitude, multidirectional, novel loads, as well as avoiding premature sport specialization, may prove to be a key intervention to increase bone strength and prevent BSI in the future. However, there is no prospectively established workload metric which can accurately predict bone stress injuries in runners.

Limitations of this narrative analysis include heterogeneity in study designs, a limited number of publications focusing exclusively on BSI in runners, and potential under-reporting of injuries, particularly among amateur athletes.

Conclusions

BSIs in runners are common injuries that require a holistic, evidence-based approach. Early diagnosis using advanced imaging, as well as addressing underlying risk factors, is critical to treating and preventing further BSIs. Conservative management, including rest, changes in the exercise program, and physical therapy interventions, remains the basis of the treatment. Surgical methods, on the other hand, are used in cases with high risk of delayed union or nonunion, or when conservative treatment fails. Preventive strategies, including screening for female athlete triad, individualized training progression, optimized nutrition and biomechanical optimization, are essential in reducing the incidence of injury. Further research is needed to validate new treatments and to try to standardize a system for detecting risk factors to prevent injuries in runners.

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Author's contribution:

Conceptualisation: Alicja Toczyłowska

Methodology: Mateusz Muras

Software: Alicja Toczyłowska, Lidia Mądrzak

Check: Mateusz Muras, Julia Kwiecińska, Władysław Hryniuk

Formal analysis: Katarzyna Krzyżanowska, Marta Korchowiec, Jacek Sitkiewicz

Investigation: Alicja Toczyłowska, Julia Kwiecińska, Katarzyna Krzyżanowska, Władysław Hryniuk, Wiktor Chrzanowski

Resources: Alicja Toczyłowska, Marta Korchowiec, Wiktor Chrzanowski, Łukasz Bialic Data curation: Mateusz Muras

Writing-rough preparation: Alicja Toczyłowska, Julia Kwiecińska, Katarzyna Krzyżanowska, Władysław Hryniuk, Marta Korchowiec, Jacek Sitkiewicz, Łukasz Bialic, Mateusz Muras, Lidia Mądrzak, Wiktor Chrzanowski

Writing review and editing: Alicja Toczyłowska, Łukasz Bialic, Mateusz Muras, Lidia Mądrzak, Jacek Sitkiewicz

Project administration: Alicja Toczyłowska

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