Stress fracture risk factors in female football players and their clinical implications

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A stress fracture represents the inability of the skeleton to withstand repetitive bouts of mechanical loading, which results in structural fatigue, and resultant signs and symptoms of localised pain and tenderness. Reports of stress fractures in female football players are not prevalent; however, they are probably under-reported and their importance lies in the morbidity that they cause in terms of time lost from participation. By considering risk factors for stress fractures in female football players it may be possible to reduce the impact of these troublesome injuries. Risk factors for stress fractures in female football players include intrinsic risk factors such as gender, endocrine, nutritional, physical fitness and neuromusculoskeletal factors, as well as extrinsic risk factors such as training programme, equipment and environmental factors. This paper discusses these risk factors and their implications in terms of developing prevention and management strategies for stress fractures in female football players.

Football is widely considered to be the most popular sport in the world, and continues to grow in popularity and participation rates. Women account for a large proportion of this growth. Between 1981 and 2005 participation in women’s collegiate football in the USA increased over 11-fold (1039%). In comparison, participation in men’s collegiate football increased 49%, and today there are more women collegiate football players than men. As the rates of injury in collegiate women’s football have not dramatically changed since 1986, the increasing participation in football by women has resulted in a larger increase in the total number of injuries being sustained by this population.

Football is a physically demanding endeavour that requires ballistic movements often under physical duress from opposing players. This poses considerable risk for acute injury, with women’s football having the highest number of injuries per match during the 2004 Olympic Games than any other women’s team sports. Most of the injuries in women’s football are sprains, strains and contusions. The shear number of these acute injuries make them an important issue in women’s football and, thus, they have received the majority of attention. However, football also involves repetitive loading in the form of running (jogging and sprinting), direction changes and kicking. For instance, elite female football players were recently found to cover a total distance of 10.3 km during games with high-intensity running (15–25 km/h) accounting for 1.3 km. These distances and intensities have the potential to contribute to the development of chronic or overuse injuries. Overuse injuries in elite women’s football players account for approximately 12.5% of all injuries.

Stress fractures of the lower limb are important overuse injuries in female football players. A stress fracture represents the inability of a bone to withstand repetitive bouts of mechanical loading, which results in structural fatigue and resultant signs and symptoms of localised pain and tenderness. Although stress fractures have been infrequently reported in women’s football, they are an important cause of morbidity. Five (one thigh, three leg and one foot) stress fractures were reported in women’s collegiate football in the USA during the 2004–5 season. Two of these (thigh and foot) were season-ending injuries, and the three leg stress fractures accounted for the most time lost due to any acute or chronic lower leg injury. Another factor contributing to their importance is that stress fractures in female football players are probably under-reported and, therefore, under-represented in injury surveillance data. A study of 21 asymptomatic female football players found 32 focal tracer uptake changes in the femur and tibia on scintigraphy that were consistent with bone stress injuries.

To prevent stress fractures in women’s football and develop appropriate management strategies when they do occur, an appreciation of their pathophysiology and risk factors is required. This article discusses these features, and discusses their implications in terms of developing prevention and management strategies for stress fractures in women’s football.

PATHOPHYSIOLOGY OF STRESS FRACTURES

The exact pathophysiology of stress fractures is not known, although several theoretically based models have been proposed. The skeleton experiences repetitive loading in its role of providing internal support to counteract the force of gravity and in forming levers for motion. This loading results in the generation of internal bone strain. While the safety factor between usual and failure (fracture) strains in bone is large, repetitive strains below the single load failure threshold are capable of generating damage (termed microdamage). This damage is a natural phenomenon and is typically of little consequence as bone is capable of self-repair through targeted remodelling. However, imbalances can develop between damage generation and removal. The subsequent accumulation of damage is believed to be the start of a pathology continuum that results clinically in stress reactions, stress fractures and ultimately complete bone fractures.

Determinants of damage formation in bone include the magnitude and rate of introduction of applied loads, and the absolute number of loading cycles. Damage is threshold related, such that strain magnitudes above a certain level result in its formation. With increasing strain there is increasing damage. Influencing the damage formation is the rate at which the strain is introduced. Strains that are introduced over shorter periods result in considerably greater damage formation. The interaction between strain magnitude and rate ultimately reduces the number of loading cycles a bone can withstand before fatigue failure. The clinical implications are...
that any activities that increase the magnitude and/or rate of bone loading may contribute to the formation of damage and its progression to stress fracture. Similarly, damage accumulation and stress fracture may also result from pure cyclic overloading. This occurs when remodelling is given insufficient time to repair damage such that more loading cycles lead to accumulation of damage. Therefore, activities that increase the number of loading cycles may also contribute to the development of stress fractures.

**RISK FACTORS FOR STRESS FRACTURES IN FEMALE FOOTBALL PLAYERS**

Stress fractures in female football players probably occur due to a range of factors with the relative contribution of each varying among individuals. There are currently no prospective or even retrospective data establishing risk factors for stress fractures in female football players. Therefore, knowledge on this topic must be translated from data on other populations.

General risk factors for stress fractures in athletes have recently been reviewed. They were divided into two categories—extrinsic and intrinsic. Extrinsic risk factors are factors in the environment or external to an individual that influence the likelihood of sustaining an injury. In terms of stress fractures in female football players, these include training programme, equipment and environmental factors. Extrinsic factors are critical in the development of stress fractures as some form of loading needs to be placed on a bone for damage to generate and accumulate. However, development of a stress fracture is also influenced by the ability of the body to respond to applied loads.

Intrinsic risk factors refer to characteristics within an individual, and how their body responds to loading and any damage it generates. The contribution of intrinsic risk factors in female football players is indicated by the fact that not all players will develop a stress fracture despite being exposed to equivalent extrinsic risk factors. Intrinsic risk factors for stress fractures in female football players include gender, endocrine, nutritional, physical fitness and neuromusculoskeletal factors.

**The gender, endocrine and nutrition connection**

Women are generally considered to be more susceptible to stress fractures than men. Military studies have shown women incur stress fractures 2–10 times more often than men performing the same activities. It is not clear whether this gender difference translates to athletic populations, with studies in male and female athletes showing either no difference or only slightly increased risk in women. However, differences are difficult to establish in athletes as gender differences in the amount and intensity of activity cannot be controlled. Despite this, female football players have been reported to have a higher incidence of asymptomatic tibial stress fractures (73%) than their male counterparts (55%), and during the 2004–5 men’s and women’s collegiate football seasons in the USA there were marginally more stress fractures in women than men (5 vs 3, respectively).

The cause for the apparent higher incidence of stress fractures in women is not known. Although it is most probably multifactorial, recent hypotheses point towards nutritional factors. Stress fractures are more likely to occur in women who restrict caloric intake, avoid high fat dairy foods, consume low calorie products, have a self-reported history of an eating disorder and have lower percentages of ideal body weight. In a cross-sectional study of female track and field athletes, those with a history of stress fractures were more likely to engage in restrictive eating patterns and dieting. Although there is no evidence that extreme restrictive dieting is prevalent in women’s football, female football players have been reported to have insufficient carbohydrate intake to meet their energy requirements and restrictive dieting has been reported in an individual female football player with a stress fracture.

Deficient caloric intake may influence the risk of stress fracture in female football players by modulating menstrual status and endocrine factors. Menstrual disturbance is a risk factor for stress fracture—the relative risk for stress fracture in amenorrhoeic athletes is between two and four times greater than in eumenorrhoeic athletes. Physically active females have a higher prevalence of menstrual disturbances compared with the general female population, and this higher prevalence appears to be related to caloric intake. Loucks and Thuma found exercise to have no disruptive effect on luteinising hormone pulsatility beyond the impact of its cost on energy availability, and that the disruption of the pulsatility of luteinising hormone in exercising women could be prevented by compensating their diet for their increased energy expenditure. Thus, it seems that athletic women acquire their menstrual disorders by not increasing dietary intake in compensation for energy expenditure during exercise. In a study wherein stress fracture susceptibility was directly associated with enhanced resistance to stress fracture in adult elite runners, showed that restricted availability of energy in young exercising women negatively altered bone turnover, as indicated by reduced concentrations of bone formation markers and raised concentration of a marker for bone resorption. This indicates that deficient caloric intake in female football players may potentiate stress fracture development by altering the skeleton’s susceptibility to developing microdamage and compromising its ability to repair such damage.

**Physical fitness and neuromusculoskeletal factors**

Physical fitness seems to be a predictor of the risk for stress fracture. Studies in the military setting have consistently shown significant associations between low aerobic fitness levels and higher risk of stress fracture during basic training. Numerous studies have also shown that a history of regular participation in exercise such as ball sports considerably reduces the risk for stress fracture. The reason for this is not established, and it could possibly be due to physical activity creating beneficial changes in neuromusculoskeletal risk factors.

Neuromusculoskeletal risk factors refer to the individual and combined contributions of the skeletal, muscular and articular systems to stress fracture. It is well established that skeletal factors contribute to stress fracture risk, with the fatigue life of bone dependent on how much bone is present (mass) and its distribution (structure). This has been confirmed in prospective studies wherein stress fracture susceptibility was directly related to skeletal properties. Basically, the lower an individual’s bone mass and the smaller their skeletal structure, the greater their risk of developing a stress fracture. Fortunately, this is not a major issue in women’s football as this population generally has optimised bone mass and size. This results from football activities being conducive with progressive bone adaptation and strengthening. As a result, football playing during childhood and adolescence has been associated with enhanced resistance to stress fracture in adult elite runners.

An intimate mechanical relationship exists between muscle and bone, and muscles are protective rather than causative of stress fractures. Lower limb muscles act as active shock absorbers during weight bearing, helping to attenuate loads as they are transmitted proximally. Dysfunctional muscles (weak, fatigued or altered activation) have reduced capacity to absorb loads, leading to increased bone strain magnitudes, rates and distributions. This muscle–bone relationship seems more important in women than men, with clinical studies showing that women who develop stress fractures have smaller thigh.
and calf girth than athletes who do not develop stress fractures.27 56

Joints and their associated soft tissue structures can mediate the transmission of external loads, and thus may influence the risk of stress fracture. It is common for joints to move through a range of motion during impact loading. This aids in transmitting forces from the skeleton to muscular soft tissue structures. Restricted joint motion may disrupt this propagation resulting in increased bone strain magnitude and altered rates of bone loading.29 Given the role of these load characteristics in microdamage formation, joint restriction may potentiate the formation of stress fractures. Supporting this, a reduction in ankle dorsiflexion has been associated with stress fractures in the more proximal long bones.20

Skeletal, muscle and articular factors may independently contribute to stress fracture risk, but how these factors interact during gait (biomechanics) is important. Gait biomechanics influence the magnitude, rates and distribution of loads applied to the lower limbs. Increased leg stiffness is associated with reduced lower extremity excursions and increased peak forces acting on the lower limbs, potentially increasing the risk of stress fracture.64–67 Many factors contribute to leg stiffness including joint kinematics. McMahon et al67 found that running with exaggerated knee flexion reduces the vertical stiffness of the lower extremity, causing the runner to attenuate more shock between the shank and the head. Therefore, those who run with reduced knee flexion may have higher leg stiffness, increasing their risk for stress fracture. Supporting this, Milner et al20 found a trend towards increased knee stiffness with a moderate effect size in women with stress fracture. Thus, it seems as though gait kinematics may have an important role in the development of stress injuries in female football players.

Training programme factors
Intrinsic factors influence susceptibility to stress fracture by modifying how an individual responds to loading and the damage it generates. However, intrinsic factors are not capable of generating stress fractures in isolation. Extrinsic factors are critical in stress fracture development as some form of loading needs to be placed on a bone for damage to generate and accumulate. To this end, participating in a weight bearing athletic endeavour such as football raises the risk of stress fracture. Whether a stress fracture actually eventuates is influenced by extrinsic risk factors such as training programme, equipment and environmental factors.

Training programme factors seem critical in the development of stress fractures in female football players. Bone loading generates damage which serves as a stimulus for bone remodelling. Remodelling normally removes damage as fast as it occurs; however, the process is time dependent. The remodelling time required to reach a new equilibrium following a disturbance is in the order of one remodelling period, which is approximately three to four months.64 72 Although a remodelling reserve exists that allows increased activation of remodelling units in response to increases in damage formation, an increase in the number of active remodelling units temporarily reduces local bone mass. This occurs because resorption precedes formation in the remodelling process so that an increase in the number of remodelling units is associated with an increase in bone porosity. This reduces the elastic modulus of the bone, which in turn increases strain and, subsequently, the rate of damage formation. Thus, from a biological perspective an alteration in the local mechanical environment of a bone via a change in a football training routine has the potential to contribute to stress fracture development in female football players.

Any changes in a football training routine that alter the magnitude or rate of bone strain at a particular site, such as a change in training intensity, may contribute to stress fracture development. Increasing the intensity of training (ie, increasing speed) has the potential to increase the magnitude or rate of bone strain at a particular site. Similarly, a change in training by way of an increase in the number or duration of training sessions may also contribute to stress fracture generation. This increases the number of bone loading cycles, a factor that decreases bone fatigue life. These training changes have the potential to disturb the balance between damage formation and remodelling. Supporting this, surveys report that up to 86% of injured athletes could identify some change in their training prior to a stress fracture.64–67 However, it has not been established how many athletes do not develop a stress fracture following a change in their training programme. This is important as female football players often alter characteristics of their training programme without pathological consequences.

Equipment factors
Equipment factors can influence the risk of stress fracture by altering the loading environment of the skeleton, with the most commonly implicated pieces of equipment in football players being shoes/boots.27–64 Shoes act as filters that theoretically attenuate ground impact forces. They also have the potential to influence foot and ankle motion, thereby altering mechanics proximally in the kinetic chain. By improving cushioning (decreasing bone strain) and modifying skeletal alignment (changing mechanics), shoes have been hypothesised as potential contributing factors in stress fracture development in football players.64 Although this contribution has logic, supportive scientific evidence is lacking.

Studies have not found appreciable differences between shoes of differing shock absorptive capacity on tibial peak strain or strain rates during running.65–71 Similarly, studies using bone pin markers to assess skeletal mechanics have shown that differences in bone movements between barefoot and shod running are small and unsystematic (mean effects being less than 2%), compared with differences between subjects (up to 10%).72–75 Thus, bone strains and skeletal kinematics during running seem individually unique, and do not appear to be substantially influenced by shoe characteristics. This is supported by a general lack of clinical evidence for a role of shoes in the development of stress fracture.

One study found that military recruits who trained in a modified basketball shoe had a lower incidence of metatarsal stress fractures compared with those who trained in a standard infantry boot; however, there were no differences in the incidence of tibial or femoral stress fractures.74 Also, there was no difference in the total number of stress fractures in the two groups. In contrast, Gardner et al77 found that military recruits who started training in shoes of advanced age (an indicator of possible reduced shock absorptive capacity) were at a greater risk of developing a stress fracture. With regard to football boots, there is some evidence to suggest that peak pressures below the metatarsals vary according to the type of stud design used, thus potentially influencing metatarsal stress fracture risk.78 However, the overall contribution of football shoes/boots to stress fracture development in female football players currently remains uncertain.

Environmental factors
The primary environmental factor in the aetiology of stress fractures is training surface. Training surface has long been considered a contributor to stress fracture development,56 and has been implicated in the development of various injuries in
Running on less compliant surfaces may increase impact forces and the subsequent magnitude and rate of bone loading.26 27 Confirming this, Milgrom et al26 showed running on a treadmill (more compliant surface) to result in considerably lower tibial bone strain than running overground (less compliant surface). Although training surface has anecdotally been associated with stress fractures, large epidemiological studies of running injuries have not shown an association between injuries and training surface after controlling for weekly running distance.83 84 However, this may be related to the difficulty in accurately quantifying running surface parameters and to sampling bias.26

### IMPLICATIONS FOR STRESS FRACTURES IN FEMALE FOOTBALL PLAYERS

Stress fractures do not appear prevalent in women’s football; however, on the basis of experience in men’s football they are probably more common than currently indicated in injury surveillance data. Also, when stress fractures do occur they are an important cause of time lost from participation. By considering risk factors for stress fractures in female football players it may be possible to reduce the impact of these troublesome injuries. These risk factors are important for management as well as prevention as a history of a stress fracture is a major risk factor for a subsequent stress fracture.26 Thus, in the management of a player with a stress fracture it is important to establish and deal with the risk factors for their injury to prevent recurrence.

Many stress fractures are attributed to training “errors”. While changes in training often precede stress fracture development, the relative contribution of these changes on an individual basis needs to be assessed. An important question is what training features did an individual injured player change relative to their non-injured teammates? The crux of the question is to determine why the individual player sustained a stress fracture in terms of recent bone loading history while their teammates remained symptom free. This comparative approach helps to decipher skeletally tolerable (team-wide) from intolerable (individual) training changes. Too often training errors are blamed at the expense of other risk factors as changes at the team level are assessed rather than changes at the individual player level.

The contribution of training changes to stress fracture risk is generally agreed on; however, it is important not to solely blame these at the expense of other risk factors. Nutrition needs to be considered at both the team and individual player level. While female football players are unique from other women athletes in that restrictive dieting is not prevalent, there is a need for female football players to match their caloric intake with their high energy demands. Preliminary evidence suggests that caloric intake is on the most part sufficient in female football players. Regardless, nutrition consultation is advisable for all players to ensure that caloric needs are met, and that adequate intake and balance of macronutrients and micronutrients is achieved. This is particularly advisable in players who have a current or past history of stress fracture and who develop or have unresolved menstrual disturbance. Specific nutritional and psychological intervention may be warranted in individual players who do not maintain weight due to inadequate caloric intake. Also, calcium intake and vitamin D concentrations should be assessed in female football players. A recent randomised controlled trial in female navy recruits showed calcium and vitamin D supplementation to reduce stress fracture risk by 27%.26

An important component in both the prevention and management of stress fractures in female football players is the maintenance of physical fitness and neuromusculoskeletal features. Physical fitness and neuromusculoskeletal features should be maintained both during the off-season and during injury recovery. It is important to continue some form of training during the off-season to maintain a level of physical and neuromusculoskeletal fitness. In particular, some degree of skeletal loading should be maintained to keep up football-induced bone gains and reduce potentially detrimental seasonal changes in skeletal risk factors.26 Training frequencies, durations and intensities should be progressively ramped up before the commencement of the new season to engender sufficient match conditioning and reduce the risk of injury.26 During the season and during injury recovery, individual neuromusculoskeletal risk factors for injury need to be monitored and intervened. This includes muscle strength and endurance, particularly of the calf. Calf girth is an independent risk factor for stress fracture in women27 and has been modelled to influence tibial bone loads.26 Likewise, other potential biomechanical contributors to stress fracture should be considered. This includes the gender-specific incidence of “dynamic valgus” at the knee in the female football players resulting from increased hip rotation.26 27 Besides its established connection with anterior cruciate ligament injury, this exaggerated hip rotation during jumping/landing and cutting manoeuvres likely influences the torsional loading environment of the bones within the lower extremity.26

As shoes do not seem to be able to substantially decrease bone strain or change skeletal mechanics, other mechanisms need to be considered to guide their prescription in female football players. A growing concept is the effect of shoes and inserts on muscle activity.26 27 Muscle activity in the leg is tuned in response to impact force characteristics.26 27 This activity is influenced by running in shoes of differing material characteristics.26 27 Whether this tuning influences the risk of stress fracture is not established; however, in terms of shoe and insert prescription, comfort seems to be of paramount importance for both adherence and potential benefit. If a shoe or insert is not comfortable it will not be used and any potential benefit will not be gained. Also, the comfort of inserts has been found to influence the kinematics, kinetics and muscle activity in the lower extremity during running.26 Therefore, from a practical perspective football shoes/boots should be chosen on the basis of their comfort to the individual. Typically, this is a shoe or insert that is appropriate for the individual’s foot structure and mechanics. Individual players should not be forced to wear a boot that they do not find comfortable.
CONCLUSION
Stress fractures in female football players are probably more common than reported in injury surveillance data and are an important cause of time lost from participation. To prevent stress fractures and develop appropriate intervention strategies when they do occur, an appreciation of their risk factors is required. Unfortunately, there is limited evidence regarding stress fracture risk factors in female football players. The current article addresses this void in the literature by interpreting and translating data regarding stress fracture risk when they do occur, an appreciation of their risk factors is important cause of time lost from participation. To prevent

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