Stress Fractures: Pathophysiology, Epidemiology, and Risk Factors

Stuart J. Warden, PhD, PT, David B. Burr, PhD, Peter D. Brukner, MBBS

Corresponding author
Stuart J. Warden, PhD
Department of Physical Therapy, Indiana University,
1140 W. Michigan St., CF-326, Indianapolis, IN, 46202, USA.
E-mail: stwarden@iupui.edu

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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 localized pain and tenderness. To prevent stress fractures, an appreciation of their risk factors is required. These are typically grouped into extrinsic and intrinsic risk factors. Extrinsic risk factors for stress fractures are those in the environment or external to the individual, including the type of activity and factors involving training, equipment, and the environment. Intrinsic risk factors for stress fractures refer to characteristics within the individual, including skeletal, muscle, joint, and biomechanical factors, as well as physical fitness and gender. This article discusses these extrinsic and intrinsic risk factors, as well as the pathophysiology and epidemiology of stress fractures.

Introduction

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 localized pain and tenderness. To prevent stress fractures and develop appropriate management strategies when they do occur, an appreciation of their pathophysiology and risk factors is required. This article discusses these features, as well as the epidemiology of stress fractures.

Pathophysiology of Stress Fractures

The precise pathophysiology of stress fractures is unknown, and current models are based on theory. One such model is outlined in Figure 1. In its role of providing internal support, the skeleton is exposed to repetitive bouts of mechanical loading, which result in bone strain. Strain refers to the change in length per unit length of a bone. It is a unitless value, but because it is very small it is often expressed in terms of microstrain (με). Although the safety factor between usual strains (400–1500 με) and strains causing failure (10,000 με) in bone is large, strains below the single load–failure threshold are capable of generating damage when introduced repetitively. As with other structural materials, repetitive strain in bone is naturally associated with the generation of damage (often termed *microdamage*). This damage is typically of little consequence, as bone is capable of self-repair through targeted remodeling. Under certain conditions, however, imbalances can develop between damage generation and its 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 strain-generated damage formation include the magnitude and rate of introduction of the applied load and the absolute number of loading cycles. Damage is a threshold-related phenomenon; it results from strain magnitudes above a certain level. 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 significantly greater damage. The interaction between strain magnitude and rate ultimately reduces the number of loading cycles a bone can withstand before fatigue failure. The clinical implication is that any activities that increase the magnitude or the rate of bone loading (or both) may contribute to damage formation with subsequent progression to a stress fracture. These activities typically involve some form of impact loading, in which high loads are introduced over short periods. Similarly, damage accumulation and stress fracture may also result from purely cyclic overloading, which occurs when remodeling is given insufficient time to repair damage and additional loading cycles enable damage to accumulate. Therefore, factors that increase the number of loading cycles may also contribute to the development of a stress fracture.

Epidemiology of Stress Fractures

As the pathophysiology of stress fractures involves some form of repetitive mechanical loading, it is not surprising

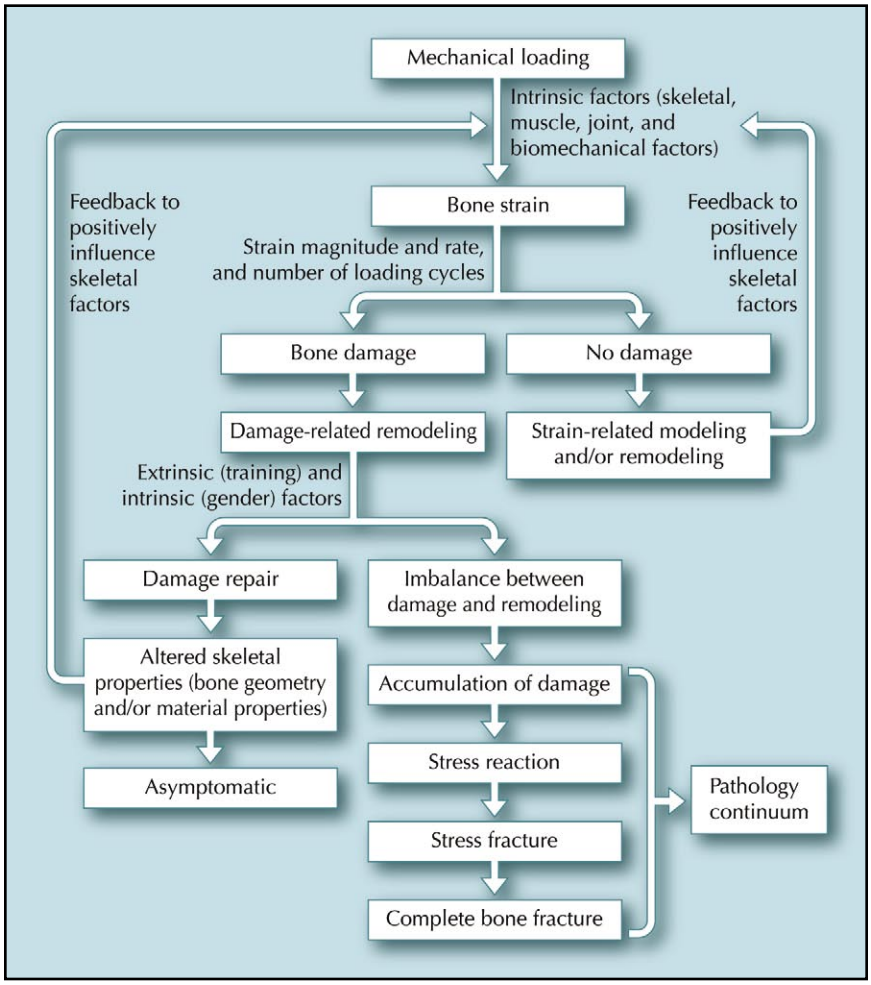

Figure 1. Proposed pathophysiology of stress fractures.

that they occur predominantly in athletes and military recruits. In both these populations, stress fractures appear to be a common overuse injury, but the precise incidence is difficult to ascertain. Retrospective studies have found that 8.3% to 52% of runners have had a history of stress fracture [1], whereas the incidence in a 12-month prospective study of track and field athletes was 21.1% [2]. When expressed as a percentage of all injuries, stress fractures have been reported to represent between 0.5% and 20.0% of all injuries sustained by athletic populations [3]. Prospective data in recruits undergoing basic training indicate a stress fracture incidence of 3.3% to 8.5% in the US military and 3.6% to 28.9% in the Israeli military [1].

Risk Factors for Stress Fractures

As with most overuse conditions, the development of a stress fracture is likely due to a range of factors, with the relative contribution of each factor varying among individuals. These factors can be grouped into two categories, extrinsic and intrinsic. Extrinsic risk factors are factors in the environment or external to the individual that influence the likelihood of sustaining an injury. In terms of stress fractures, these include the type of activity or sport, as well as factors involving training, equipment, and the environment. 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. However, stress fracture development is also influenced by the ability of the body to respond to applied loads. Intrinsic risk factors refer to characteristics within individuals themselves and how the body responds to mechanical loading and the damage that it may generate. The contribution of intrinsic risk factors is indicated by the fact that not all individuals exposed to an equivalent loading regimen will develop a stress fracture. Intrinsic risk factors include skeletal, muscle, joint, and biomechanical factors, as well as physical fitness and gender.

Extrinsic risk factors

Type of activity or sport

Participation in sport raises the risk of stress fracture, as most sports involve some form of mechanical loading. However, individuals are at varying risk based on the sport or sports in which they participate. Individuals who participate in sports that involve high-magnitude loads that are introduced over short periods (eg, sprinters) have increased risk of developing a stress fracture due to high

strain magnitudes and rates. On the other hand, individuals who participate in sports that involve high numbers of load repetitions (eg, distance runners) are at heightened risk of stress fracture due to cyclic overload.

The sport that an individual participates in influences stress fracture susceptibility at a particular skeletal site. Stress fractures are site-specific, occurring only at sites exposed to repetitive mechanical loading. Different activities result in different loading patterns within different bones. For example, power athletes such as sprinters load the bones of their feet relatively more than endurance athletes such as distance runners [4]. Subsequently, power athletes are at greater risk of tarsal and metatarsal stress fractures, whereas the endurance athletes are at greater risk of more proximal stress fractures [5].

With adequate loading, stress fractures can also occur at non–weight bearing sites. For example, rowers repetitively load their rib cage during the drive phase of the rowing stroke. Consequently, they are at heightened risk of generating rib stress fractures [6]. Similarly, baseball pitchers are at risk of midshaft humeral stress fractures, as they repetitively load this site during throwing [7].

Training program factors

Training program factors appear critically important in the development of stress fractures. Depending upon characteristics of the loading stimulus, bone loading can generate damage. Damage serves as a stimulus that activates bone remodeling, a sequential process involving cellular activation, osteoclastic bone resorption, and finally osteoblastic bone formation. Remodeling ensures relative homeostasis between damage formation and its repair, and maintains skeletal mechanical competence. It also enables a bone to adapt over time to its mechanical environment so that it can meet increasing levels of loading. Modification in a training routine has the potential to disturb this homeostasis.

Remodeling normally removes damage approximately as fast as it occurs. However, the remodeling time required to reach a new equilibrium following a disturbance is in the order of one remodeling period (approximately 3 to 4 months). If insufficient time is given to adapt to a change in training routine, additional damage may occur. Although a remodeling reserve exists, which allows increased activation of remodeling units in response to increases in damage formation, an increase in the number of active remodeling units removes bone temporarily, reducing bone mass and potentially increasing the chance for damage initiation when loading is continued. This feed forward results from the fact that resorption precedes formation during remodeling, so that an increase in the number of currently active remodeling units is associated with increased bone porosity. This porosity reduces the elastic modulus, which in turn increases strain and subsequently increases the rate of damage formation. Therefore, from a biologic perspective, an alteration in the local mechanical environment of a bone via a change in training routine has the potential to contribute to stress fracture development.

Changes in a training routine that may disturb the balance between damage formation and remodeling and contribute to stress fracture development include 1) changes that alter strain magnitude or rate at a particular site, such as a change in training intensity (eg, increasing speed), and 2) a change in the number of bone loading cycles (ie, an increase in the duration or number of training sessions). Surveys report that up to 86% of injured athletes could identify some change in their training prior to a stress fracture [8,9]. However, it has not been established how many athletes do not develop a stress fracture following a change in their training program. This knowledge is important, as athletes frequently alter characteristics of their training program without pathologic consequences.

Equipment factors

Equipment factors can influence stress fracture risk by altering the loading environment of the skeleton, whether by increasing bone strain or redistributing strain such that it occurs at less-accustomed locations within a bone. The most commonly implicated pieces of equipment in stress fracture development are shoes and inserts (insoles and orthotics). Located at the foot-ground interface, shoes and inserts act as filters that theoretically attenuate ground impact forces. In addition, they have the potential to influence the motion of the foot and ankle and the subsequent mechanics proximally in the kinetic chain. Via these two mechanisms, shoes and inserts may potentially influence bone strain and subsequently have an effect on stress fracture risk.

There is contradictory evidence for the role of shoes and inserts in stress fractures, with most data being generated in military populations. Initial studies failed to find any effect on stress fractures of a shock-absorbing insole placed within a standard military boot [10,11]. A more recent prospective, randomized controlled trial demonstrated a statistically significant difference in the incidence of stress fractures between recruits who trained with custom orthoses and those who trained without such devices, with the incidence double among nonusers of the orthoses [12]. However, this finding was not confirmed in a subsequent study by the same research group [13]. In terms of shoes, there is similar contradictory evidence. Although recruits who trained in a modified basketball shoe had a lower incidence of metatarsal stress fractures than those who trained in a standard infantry boot, there were no differences in the incidence of tibial or femoral stress fractures [14]. Also, there were no differences in the total number of stress fractures between the two groups. In contrast, Gardner et al. [10] 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. Overall, the precise contribution of shoes and insoles to stress fracture risk remains unclear.

Environmental factors

The primary environmental factor considered to be involved in the etiology of stress fractures is training surface. Running on cambered or uneven surfaces may accentuate biomechanical problems, and running on less compliant surfaces can increase impact forces and the subsequent magnitude and rate of bone loading. Confirming the influence of training surface compliance on bone loading, Milgrom et al. [15] demonstrated that running on a treadmill resulted in significantly lower tibial bone strain than running overground. Although this finding suggests that running on softer, more compliant surfaces may reduce the risk of stress fracture, this result may be influenced by muscle fatigue. Running on compliant surfaces (eg, sand, dirt, gravel) requires greater energy expenditure and may hasten muscle fatigue, which has the potential to influence bone loading.

Although training surface has anecdotally been associated with stress fractures, large epidemiologic studies of running injuries have failed to show an association between injuries and training surface or terrain after controlling for weekly running distance [16,17]. However, this finding may be related to the difficulty of accurately quantifying running surface parameters and to sampling bias [18]. One investigation of stress fractures in female military recruits did find an increase in stress fracture incidence when training was changed from a usual flat, predictable terrain to a hilly, rocky terrain [19].

Intrinsic risk factors

Skeletal factors

The contribution of skeletal factors to stress fracture risk is well established. Seminal work by Carter and Hayes [20] demonstrated that the ability of a bone to withstand repetitive low-magnitude loads is dependent upon how much bone is present (bone mass) and the distribution of this bone (bone structure). Supporting this idea, Warden et al. [21•] demonstrated skeletal fatigue resistance to be significantly correlated to measures of both bone mass and structure. Although these findings have variable clinical support from cross-sectional studies [22–25], statistically more powerful, prospective studies have confirmed that stress fracture susceptibility is directly related to skeletal properties (bone mass and size) [2,26,27].

Muscle factors

An intimate mechanical relationship exists between muscle and bone, with muscles having the potential to influence bone loading. The general consensus is that muscle is protective rather than causative of stress fractures. During impact loading, muscle is believed to act as an active shock absorber helping to attenuate loads as they are transmitted proximally along the kinetic chain. When muscles are dysfunctional (weakened, fatigued, or altered in their activation patterns) their ability to absorb loads becomes compromised, potentially leading to increased loading on the skeleton. Laboratory-based studies show that muscle fatigue causes an increase in both bone strain magnitude and rate [28–31]. Further support for a protective role of muscle in stress fracture development comes from prospective clinical studies, which have demonstrated that stress fracture susceptibility is directly related to muscle factors [2,23,32].

Joint factors

Joints and their associated structures (capsules, ligaments, menisci) are important mediators in the transmission of applied loads, and consequently may contribute to stress fracture risk. It is typical during impact loading for joints to move through a certain range of motion. This motion primarily protects articular cartilage from excessive compressive loads, but it also functions to propagate forces away from the skeleton and to nonarticular structures such as muscle. The best example occurs in the lower extremity, where knee and ankle flexion during landing from a jump are believed to reduce the magnitude of loading [33,34]. When joint motion is restricted, the consequence may be increased magnitude of bone strain and an altered rate of bone loading [35]. Given the role of these load characteristics in microdamage formation, joint restriction may potentiate the formation of stress fractures. Hughes [36] found a decrease in ankle dorsiflexion to be associated with stress fracture development, although subsequent research has failed to validate this finding [2,37].

Biomechanical factors

Bone, muscle, and joint factors may contribute to the intrinsic risk for stress fractures, but how these factors work together as a functional unit may be of greater importance. It is difficult to establish the contribution of individual biomechanical features to stress fracture risk. This difficulty results from two major factors: 1) assessment of dynamic biomechanical features typically requires complicated measurement tools that are not often readily available and are time-consuming to use, and 2) the prospective study of risk factors for stress fracture requires large numbers of individuals to be studied over long periods in order to generate sufficient statistical power.

Reflecting the difficulty in assessing biomechanical risk factors, there are few prospective studies of the influence of specific biomechanical features on stress fracture risk. Preliminary data from a large, ongoing prospective study suggest that females who go on to develop a tibial stress fracture have altered loading (tibial shock and vertical load rates) compared with those who do not develop the injury [38]. This finding is supported by retrospective data [39•], and suggests that techniques that reduce

tibial loading may be critical to stress fracture prevention. These may include techniques to reduce the effective vertical stiffness of the leg during running, such as running with exaggerated knee flexion [39•].

Other biomechanical factors that may predispose athletes to stress fracture include a high degree of external rotation of the hip [12,40], leg-length discrepancy [2], and pes planus [9,37] and cavus [41,42]. Of these, the structure of the foot (pes planus or cavus) has received the most attention. A foot with a high longitudinal arch (pes cavus) is considered to be more rigid, with reduced shock absorbing capacity. Thus it may allow more force to be transmitted proximally. In comparison, a more flexible foot (pes planus) may allow the foot to attenuate more force, with less being propagated proximally. Supporting this hypothesis, Simkin et al. [42] demonstrated that foot arch structure influenced stress fracture location. Femoral and tibial stress fractures were more frequent in subjects with high-arched feet, whereas metatarsal stress fractures were more frequent in low-arched feet. However, studies investigating the role of these foot types frequently assess the foot statically or when it is in a nonfunctional position. What appears to be more important clinically is the amount and duration of foot motion during dynamic loading. Static measurements do not necessarily correlate with the dynamic situation. This idea was recently confirmed by Dixon et al. [43], who demonstrated that military recruits with a previous history of metatarsal stress fracture did not differ from a control group in static biomechanical measures of the foot, but dynamic biomechanical measures during running differed between the groups. Specifically, the stress fracture group demonstrated significantly earlier rearfoot eversion and a significantly more lateral peak applied resultant horizontal force [43].

Physical fitness

Physical fitness is a predictor of stress fracture risk. Studies in the military have consistently shown significant associations between low aerobic fitness levels and higher risk of stress fracture during basic training [44]. Milgrom et al. [45] demonstrated in three prospective epidemiologic studies that military recruits who played ball sports regularly for at least 2 years prior to basic training had less than half the risk of developing a stress fracture than recruits who did not play ball sports. Similarly, Lappe et al. [46] demonstrated that a history of regular exercise in military recruits was protective against stress fracture, and that a longer history of exercise further decreased the relative risk. The reason for these differences is not established, and could possibly be due to physical activity creating beneficial changes in other intrinsic risk factors such as skeletal, muscle, and joint factors.

Gender

Gender factors contribute to stress fracture susceptibility, with females being at a greater risk. A number of military studies have shown that women performing the same prescribed physical activities as men incur stress fractures at incidences two to 10 times higher than those for men [18]. This gender difference is not as evident in athletic populations, with studies in male and female athletes showing either no difference or only a slightly increased risk for women [5,8]. The latter studies are somewhat limited, however, as they were not able to control for differences in the amount or intensity of activity.

The cause for the higher incidence of stress fractures in females is not known. Although it is most likely multifactorial, the latest theories highlight the potential contribution of nutritional or dietary features [47••]. Stress fractures have been found to be more likely to occur in females 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 [48]. In a cross-sectional study of young adult female track and field athletes, those with a history of stress fractures scored significantly higher on the Eating Attitudes Test (EAT)-40 test (a validated test relating to dieting, bulimia, and food preoccupation) and were more likely to engage in restrictive eating patterns and dieting [22].

Deficient caloric intake may influence stress fracture risk by modulating menstrual status and endocrine factors. Menstrual disturbance is a risk factor for stress fracture, with the relative risk for stress fracture in amenorrheic female athletes being between two and four times greater than that in their eumenorrheic counterparts [18]. Physically active females have a higher prevalence of menstrual disturbances than the general female population [47••], and this higher prevalence appears to be related to caloric intake. Loucks and Thuma [49] found that exercise had no disruptive effect on luteinizing hormone pulsatility beyond the impact of its cost on energy availability, and that the disruption of luteinizing hormone pulsatility in exercising women could be prevented by supplementing their diet to compensate for their increased energy expenditure. Thus, it appears that athletic women acquire their menstrual disorders by failing to increase dietary intake in compensation for exercise energy expenditure [47••]. Furthering this work, Ihle and Loucks [50•] demonstrated that restricted energy availability in young exercising women negatively altered bone turnover, as indicated by reduced levels of bone formation markers (plasma osteocalcin and serum type I procollagen carboxy-terminal propeptide) and elevated levels of a marker for bone resorption (urinary N-terminal telopeptide). This indicates that deficient caloric intake may potentiate stress fracture development by altering the skeleton's susceptibility to developing microdamage and its ability to repair such damage.

Conclusions

Stress fractures continue to be a significant clinical problem that causes substantial morbidity and time lost from

participation. In order to prevent stress fractures, an appreciation of their risk factors is required. This article discusses potential extrinsic and intrinsic risk factors for stress fractures, based on the available literature. As with most overuse conditions, the development of a stress fracture probably requires a combination of factors. Some prospective data have indicated the potential roles of certain risk factors in various population groups, but how these translate into clinical paradigms is not clear. For example, training program factors appear critically involved in the development of stress fractures, but it has not been established how much a training program can be modified before stress fracture risk is substantially elevated. Questions of this type deserve further scrutiny. In addition, greater consideration needs to be given to the role of stress fractures in bone pain in the elderly and the occurrence of osteoporotic fractures. This area remains relatively unexplored.

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