Stress Fractures in Athletes

Diagnosis and Management

Timothy L. Miller
Christopher C. Kaeding
Editors

Second Edition

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To my family, in particular my wife, Nicole, and our children, Gavin, Ashton, Avery, Sydney, and Giselle, for always believing in me and being supportive when I take on a new project.
To the many athletes who have had their seasons and careers cut short due to stress fractures and other overuse injuries, know that your hard work and efforts have not gone in vain and, in fact, were the inspiration for this book.

To Dr. Christopher C. Kaeding,
Thank you for taking a chance on a farm boy from Barnesville, Ohio. Without your guidance, support, and mentoring I would not be where I am today.

Timothy L. Miller, M.D.

To my wife, Christine, for her bottomless source of patience and support of my academic career in sports medicine with all its time demands, off hours obligations, and unexpected interruptions.
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Christopher C. Kaeding, M.D.

Preface

Stress Fractures in Athletes has been the culmination of many years of experience with overuse injuries both as athletes and as team physicians. Traditional treatment strategies for stress fractures including simply stopping the causative activity or sport are no longer considered practical or acceptable options for many competitive athletes. This textbook compiles the concepts, wisdom, and techniques required to approach and treat the complexities of stress-induced injuries to bone among the athletically active population. The editors would like our readers to understand the value we place on employing a holistic approach to treating stress fractures. A diverse treatment team that seeks to balance training, biomechanics, nutrition, hormonal status, and mental health is required to successfully prevent, treat, and recover from these troublesome injuries. We truly appreciate the contributions of the authors – many of whom are considered pioneers and leaders in the field of Sports Medicine – who have provided their invaluable insights and pearls on the evaluation and treatment of stress fractures and insufficiency fractures. As our athletes continue to break records and push the limits of running, jumping, biking, swimming, skiing, rowing, cross-fit sports, and adventure racing, the branch of Sports Medicine we refer to as *Endurance Medicine* continues to develop to serve their needs. This textbook details the pathology, assessment tools, and treatment options for bony stress injuries throughout the body from the spine and pelvis to the hands and feet. It is our hope that this textbook will be a valuable guide for Sports Medicine physicians, orthopedists, athletic trainers, physical therapists, coaches, parents, and athletes in treating stress fractures and is another step forward for the field of Endurance Medicine.

Columbus, OH, USA — Timothy L. Miller, M.D.

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Part I

General Evaluation Principles for Stress Fractures

1 Risk Factors for Developing Stress Fractures

Donald Kasitinon and Lindsay Ramey Argo

Introduction

Stress fractures are mechanical loading injuries that result from an imbalance between microdamage and bone remodeling and repair [1]. They can range in severity from mild marrow and/or periosteal edema to a visible fracture line [2]. For simplicity, all grades of bone stress injury will be referred to as a stress fracture in this chapter. Unlike acute fractures, stress fractures are typically caused by repetitive, submaximal loading of a bone [3]; like acute fractures, they can lead to significant pain, reduced performance, lost training time, and medical expense [4]. Individual athletes vary in their susceptibility to stress fractures. Risk factors for stress fractures are often categorized as intrinsic or extrinsic and modifiable or non-modifiable to aid clinicians in identifying high-risk individuals and in defining actionable ways to minimize risk [5, 6].

While the quantity of research on this topic is large, the quality of research is more limited, with few high-quality, prospective trials in the athletic population. Most studies analyzing risk factors involve military personnel rather than recreational and competitive athletes [7] and are cross-sectional in design. Further, stress fracture risk factors are often interrelated and difficult to analyze independently [5]. Despite these limitations, multiple risk factors have been established and are reviewed in this chapter (Table 1.1). In addition, proposed prediction algorithms for stress fractures based on these risk factors are outlined.

Intrinsic Risk Factors

Intrinsic risk factors are those that are directly related to the athlete's metabolic or anatomic characteristics [3, 6] and can be subcategorized into non-modifiable and modifiable factors.

Non-modifiable Intrinsic Risk Factors

Non-modifiable risk factors are those that an athlete cannot take measures to change. Non-modifiable intrinsic risk factors include specific demographics such as gender, race, and age; previous history of stress fracture; genetics; and specific anatomic alignment.

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Table 1.1 Risk factors for developing stress fractures

	Intrinsic	Extrinsic
Non-modifiable	Demographics Female gender Caucasian race Age: Undetermined History of stress fracture Genetic predisposition Anatomic characteristics Pes cavus foot morphology Leg length discrepancy Altered knee alignment	Preseason, start of season
Modifiable	Relative energy deficiency syndrome Low energy availability with or without disordered eating Functional hypothalamic amenorrhea Osteoporosis Calcium and/or vitamin D deficiency Low body weight/BMI Poor biomechanics and strength imbalance Medication use Contraceptives Corticosteroids Other Substance abuse Tobacco Alcohol	Training variables High volume Noncompliant or uneven surface Poor pre-training physical condition Equipment variables Old or non-supportive footwear Type of sport Leanness sports: track & field, cross country, gymnastics

Demographics

Gender

The majority of studies have found that females have a higher incidence of stress fractures than males. This is likely multifactorial in nature and due, at least in part, to gender-associated risk factors that include dietary deficiencies, menstrual irregularities, hormonal differences, lower bone mineral densities, and narrower bone width. Slower rates of force development in muscle physiology, especially neuromuscular control, also seem to play a role in the increased incidence of stress fractures among females [5, 8, 9].

The risk of stress fractures in female recruits in the United States military was found to be up to 10 times higher than that in their male counterparts while undergoing the same training program [10]. This increased risk in females has also been noted in multiple studies involving high school and collegiate athletes [11–14]. While some studies have reported no difference in incidence between genders [15–17], these studies did not account for total training volume and should be interpreted with caution [18, 19]. In a recent meta-analysis, female gender was one of two risk factors identified with strong evidence to support an association with stress fractures (OR 2.31) [20].

Race

Military studies have shown the highest incidence of stress fractures among white recruits as compared to other races. Among females, incidence rates are the highest in white followed by Asian and Hispanic females, with the lowest incidence rates in African Americans [21–25]. Similar trends by race have been identified in male military recruits [21–25]. This is thought to be due to differences in bone turnover and peak bone density and not due to race independently [5].

Studies of athletes outside the military have shown mixed results. Two studies involving United States collegiate athletes reported no significant differences in stress fracture incidences between white and African American athletes [17, 26]. However, studies from Japan and Korea display slight differences in stress fracture incidence when compared with white populations. Of note, these studies were not sport specific and differences may be related to variation in activity type between groups rather than race [15, 27, 28]. Evidence within the military suggests that white individuals are at higher risk of stress fracture in comparison to other races, particularly African

Americans; however, evidence outside of the military is limited.

Age

The association between age and incidence of stress fracture is currently unclear. Studies in athletes have not found a consistent correlation between age and risk of stress fracture. Within the military, studies have found similar inconsistent results across age groups [21, 22, 29]. Few studies have reported stress fracture frequency by age group [18]. Within the female athlete population, some studies have found an increased risk of stress fractures with increasing age [22, 23], while others have found decreased risk [21, 24, 29, 30] or no effect [31, 32]. Confounding factors associated with aging, particular in the female population, including changes in hormone levels, bone mineral density (BMD), activity level and activity type, may contribute to the inconsistency presented in the current literature [5]. Studies controlling for these confounding variables are needed to determine whether age is an independent risk factor for stress fractures.

Previous History of Stress Fracture

Previous history of stress fracture has been consistently associated with increased risk of future stress fractures across studies, with odds ratios ranging from 2.90 to 6.36 [16, 19, 33]. These findings were supported by a recent exploratory meta-analysis noting that athletes with a previous history of stress fracture have a five time higher risk of developing a new stress fracture as compared to individuals with no prior history [20].

Genetics

Several observations suggest that genetic factors contribute to stress fracture susceptibility. Case reports have described multiple stress fractures at the same anatomic sites in monozygotic twins after the sixth week of basic training in the army [34] as well as multiple lower limb fractures in the same individual [35]. Findings between twins and their families indicate that differences in traits, such as bone size, shape, and BMD, are largely due to genetic differences and not environmental differences [36]. Additionally, mutations or allelic variants in genes can lead to a variety of bone pathologies such as osteoporosis or osteogenesis imperfecta that can result in increased bone fragility and increased risk of stress fracture. In a Finnish military study, eight genes involved in bone metabolism and pathology were examined in subjects with femoral neck stress fractures versus controls. While details are beyond the scope of this chapter, specific genetic patterns were associated with increased risk of stress fractures, suggesting that genetic factors do play a role in the development of stress fractures in individuals who undergo heavy exercise and mechanical loading [37]. Of note, in the absences of specific mutations that identify a specific bone disorders, it is unclear how this can be used clinically for risk stratification or modification at this time.

Lower Extremity Alignment

Specific anatomic variants have been theorized to increase the risk for stress fractures in the lower extremity, including abnormal foot morphology, leg length discrepancy, and knee alignment. While alignment and biomechanical changes associated with these variants can be minimized with bracing, orthotics, and/or appropriate rehabilitation efforts, here we consider these factors to be non-modifiable as the anatomic variant itself requires surgery for correction.

Foot morphology has long been theorized to play a role in lower extremity stress fractures, as the structure of the foot can affect the absorption and distribution of the ground reaction force during impact exercise. Pes cavus refers to a rigid, high-arched foot and results in more force absorption in the leg (femur, tibia, and fibula) and less force absorption in the foot. Pes planus refers to a flexible, low-arched foot and results in less force absorption in the leg and more force

absorption in the foot [5]. A study among the Israeli military population found that those with the highest arches sustained 3.9 times as many stress fractures as those with the lowest arches [38]. A prospective military study found that those with pes cavus morphology were at increased risk of femoral and tibial stress fractures, while those with pes planus were at increased risk of metatarsal fractures [39]. Studies outside of the military have found similar associations with pes cavus morphology and overuse injuries, particularly among runners [40–42]. However, pes planus morphology has shown inconsistent results outside of the military [43, 44]. In a study of 31 runners with recurrent stress fractures, pes cavus was one of three anatomical factors associated with stress fracture recurrence [45].

Leg length discrepancy has also shown a weak association with stress fractures, particularly in female athletes [43, 44]. In the aforementioned study of 31 runners with recurrent stress fractures, leg length discrepancy was one of three anatomical factors associated with stress fracture recurrence [45]. The degree of leg length discrepancy is thought to correlate with increased risk for stress fracture [46] although a study with male military recruits did not confirm this correlation [5, 47].

Valgus knee alignment and increased quadriceps angle (Q angle >15°) have been proposed as potential risk factors for lower extremity overuse injuries, including stress fractures. However, data to support this is limited. Research in this area is dated and largely focused on males in the military [47, 48]. In a 1996 prospective study of 294 male infantry trainees, those with knee valgus alignment had a significantly higher risk for lower extremity overuse injuries (RR = 1.9) and those with an elevated Q angle had a significantly higher risk for lower extremity stress fractures (RR = 5.4) [47]. However, more recent studies have not reproduced these results in athletes of various sports and mixed gender activities outside of the military [49–51]. While theorized, evidence is lacking to define a clear association between knee alignment and risk for stress fracture outside of the military, and further research is needed to better define this relationship.

Limited evidence suggests that orthotics to support pes cavus or correct leg length discrepancies decreases risk of stress fracture, but no clear evidence has been shown [52].

Modifiable Intrinsic Risk Factors

Modifiable risk factors are those that an athlete can take measures to change. Identified modifiable intrinsic risk factors include relative energy deficiency syndrome (RED-S) encompassing low energy availability, functional hypothalamic amenorrhea, and osteoporosis; calcium and/or vitamin D deficiency; low body weight or body mass index (BMI); suboptimal biomechanics and strength; and exposure to certain medications or substances.

Relative Energy Deficiency Syndrome

The term “female athlete triad” was initially used to describe a medical condition observed in physically active females involving one or more of the following: low energy availability, menstrual dysfunction, and low BMD [53]. The Task Force on Women’s Issues of the American College of Sports Medicine published the first triad position statement in 1997 describing a syndrome of three distinct but interrelated conditions: disordered eating, amenorrhea, and osteoporosis [54]. Later studies found that athletes were developing negative health consequences of the triad associated with less severe conditions than these clinical endpoints, so the triad was redefined in 2007 as relative energy deficiency syndrome (RED-S) including low energy availability with or without disordered eating, functional hypothalamic amenorrhea, and osteoporosis with a spectra between optimal health and these endpoints (Fig. 1.1) [55]. The goal of presenting the triad symptoms along a spectrum is to emphasize the importance of identifying subclinical abnormalities among athletes to allow for early intervention

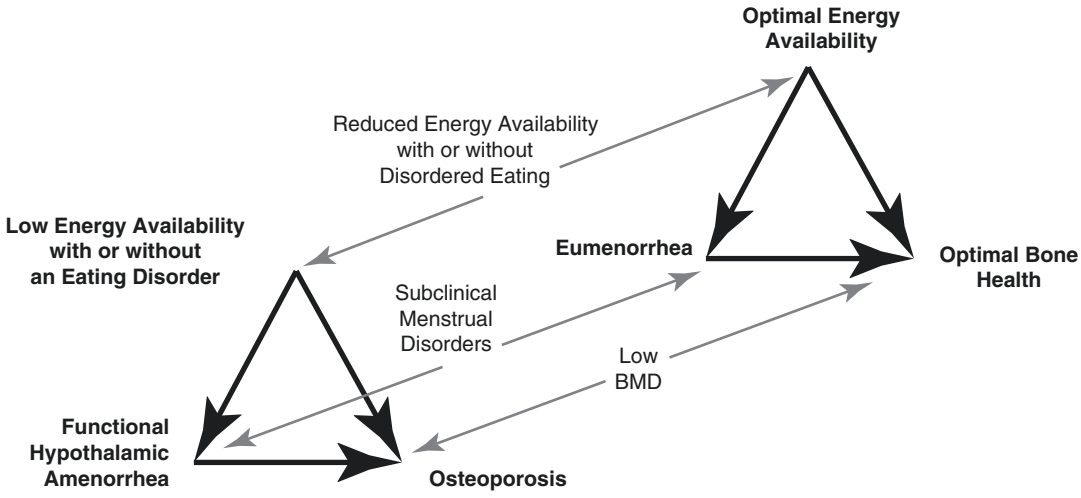

Fig. 1.1 Spectra of the female athlete triad. Spectra along which female athletes are distributed, including energy availability, menstrual function, and BMD. An athlete's condition moves along each spectrum at a different rate based on diet and exercise habits. (Reprinted with permission from Eguiguren, M. L., & Ackerman, K. E. (2016). The female athlete triad. In C. Stein, A. Stracciolini, & K. Ackerman (Eds.), *The young female athlete* (p. 64). Cham, Switzerland: Springer International Publishing. Originally from De Souza et al. [53])

[53]. RED-S is the broader, more comprehensive name as components of the triad are also reported in males [56].

Low Energy Availability with or Without Disordered Eating

Low energy availability is defined as inadequate caloric intake relative to the energy expenditure required for physical activity level [5]. In the absence of disordered eating, it can be difficult to diagnose. Signs of low energy availability include low body weight (a BMI < 17.5 or <85% of expected body weight in adolescents), reduced resting metabolic rate, menstrual dysfunction, low triiodothyronine (T3), excessive fatigue, and impaired immunity/frequent infections [57, 58]. When RED-S is suspected, detailed information regarding food intake and energy expenditure should be evaluated [53].

Low energy availability seems to be the primary mechanism by which female athletes are predisposed to menstrual dysfunction and negative effects on bone health. Anorexia nervosa has been associated with a significantly decreased BMD [59, 60] with nearly 75% of adolescent girls with anorexia having a BMD more than two standard deviation below the normal value [61]. Females with anorexia are at an increased risk of stress fracture development [62, 63]. Similarly, in a group of competitive female track and field athletes, those with stress fractures had significantly higher scores on the EAT-40, an eating attitude test, and tended to weigh themselves more often, indicating a greater preoccupation with weight control among those with stress fractures [64].

Functional Hypothalamic Amenorrhea

Functional hypothalamic amenorrhea (FHA) is a form of hypogonadotropic hypogonadism caused by a disturbance in the normal pulsatile release of gonadotropin-releasing hormone (GnRH) from the hypothalamus [65]. It is a diagnosis of exclusion as the term "functional" indicates that there is no organic disease identified [66, 67]. FHA can result from a combination of low energy availability (weight loss-related, stress-related, and/or exercise-related) and genetics, and it predisposes athletes to osteoporosis [5, 59]. Previous studies have indicated that menstrual dysfunction is

associated with the development of stress fractures [24, 26, 30, 44, 68, 69]. Track and field athletes with a history of oligomenorrhea (defined as four to eight menses per year) were found to be six times more likely to have sustained a prior stress fracture [64]. Female military recruits who reported no menses during the 12 months prior to training were more than five times likely to suffer a stress fracture and more than eight times likely to suffer a pelvic or femoral stress fracture than their eumenorrheic counterparts. Those with secondary amenorrhea, defined as six or more consecutively missed menses during the past year, were more than twice as likely to suffer a pelvic or femoral stress fracture [70]. In a group of female high school competitive runners, multiple menstrual variables were associated with increased risk for stress fracture, as follows: fourfold increase with late menarche at or after 15 years of age, twofold increase with current amenorrhea, and successive decrease in risk with increasing number of periods in the past year—each menstrual period was associated with an 11% decreased risk of suffering a stress fracture [16].

Osteoporosis

Osteoporosis is a condition marked by decreased BMD, resulting in fragile bone and is defined by the World Health Organization as a T score less than −2.5 in post-menopausal females and males aged 50 and over on dual-energy X-ray absorptiometry (DEXA) scans [71]. Those with a T score ranging from −1.0 to −2.5 are considered to be osteopenic. The T score represents the number of standard deviations a person is above or below a reference database of a healthy 30-year-old adult. For pre-menopausal females, males younger than 50, and children, the Z score should be used [72]. The Z score is the number of standard deviations a person is above or below a reference database of similar age, gender, and body size. A Z-score of −2.0 or below is considered outside of the expected range based on the International Society of Clinic Densitometry. This suggests that something other than aging is causing abnormal bone loss, which should prompt an evaluation for secondary causes of osteoporosis [73]. However, the American College of Sports Medicine notes that, as athletes in weight bearing sports typically have higher than normal BMD, a Z-score of −1.0 or below should be considered low BMD in this population and is worthy of further investigation. DEXA alone should not be used to diagnose osteoporosis, but Z-score combined with secondary clinical risk factors for fracture would support a diagnosis of osteoporosis in this younger age group.

When comparing pre-menopausal female athletes and military recruits with and without stress fractures, those with a stress fracture have demonstrated lower BMD by DEXA [74]. The first study to prospectively examine this link found that lower BMD in the lumbar spine and foot were significant predictors of future stress fracture development in female track and field athletes [64]. Studies in male athletes have shown similar, but inconsistent, trends [68, 75, 76]. Cancellous, as opposed to cortical, bone stress fractures have more consistently correlated with lower BMD in both the female [77] and male [78] athletic population.

DEXA testing should be considered in athletes with a stress fracture and any of the following: stress fracture of cancellous (rather than cortical) bone, recurrent stress fracture of any site, low BMI (<18.5 kg/m^2), oligo- or amenorrhea for 6 months or more, history of disordered eating or low energy availability, chronic medical condition associated with bone loss, chronic medication use associated with low BMD and non-weightbearing athletes (cyclists, swimmers).

Calcium and/or Vitamin D Deficiency

Calcium and vitamin D are two nutritional components widely recognized as necessary to maintain optimal bone health and have been shown to improve BMD in the general population [79–81]. This increased BMD has been shown to be somewhat protective against stress fractures as described above in the Osteoporosis section.

However, among athletes of ages 18–35 years, the role of calcium and vitamin D in both bone development and the prevention of stress fractures is not as well-established. Some studies have found an association between calcium/vitamin D and BMD/stress fracture risk in athletes [68, 82], while others have not [23, 31, 44, 83]. Nieves et al. utilized dietary data collected during the course of a randomized trial of the effect of oral contraceptive pills (OCPs) on bone health in female competitive distance runners. Dietary variables were assessed with a food frequency questionnaire and found that higher dietary intake of both calcium and vitamin D were associated with a gain in hip BMD and that higher dietary intake of calcium was associated with a gain in whole-body BMD and lower rates of stress fractures [82]. Lappe et al. performed a double-blind, randomized clinical trial among female navy recruits with one group taking 2000 mg of calcium and 8000 international units (IU) of vitamin D supplementation a day while the other group was given a placebo. They found a 21% lower incidence of stress fractures in the supplemented group [84]. Tenforde et al. performed a prospective study among competitive high school runners and found that female athletes who used a calcium supplement were at three times higher risk of developing a stress fracture, though this was not true in males [16]. However, the results of this study have been brought into question as athletes were asked about regarding calcium supplementation late in the study, meaning that injured athletes may have been prescribed calcium supplementation as part of their treatment plan rather than primary prevention [16, 20]. There is limited data focused on male athletes. More prospective studies are needed to evaluate the relationship between calcium and vitamin D with the risk for stress fracture, particularly studies including males [85].

Based on the current evidence linking calcium and vitamin D to increased BMD and an association with stress fracture incidence in females, dietary intake of both nutrients should be discussed and optimized in all athletes, particularly those at high risk [86, 87]. The National Osteoporosis Foundations recommends at least 1000 mg of calcium daily in women 50 and under and men 70 and under, and 1200 mg daily above these age groups in in the general population. Recommended vitamin D daily intake ranges between 600 and 1000 international units (IU) daily, with an upper limit of 4000 IU daily set by the Institute of Medicine [88]. Dietary intake is preferred to supplementation due to both absorption and potential side effects of long-term supplementation.

Low Body Weight/BMI

Low body weight and/or low BMI is often seen in RED-S and has been associated with stress fracture incidence, particularly among female athletes. In one study conducted on a group of competitive high school runners including both males and females, a BMI < 19 was found to be one of the strongest independent predictors for developing a stress fracture with a two- to threefold increase in the rate of stress fracture [16]. Lower adult weight was also noted to increase the likelihood of stress fracture in a study of female army recruits [5, 23]. On the contrary, in a study conducted among a group of competitive collegiate runners including only males, BMI risk scores were not associated with increased risk of stress fracture. This may be due to the need for different criteria required to define low BMI among male athletes [75].

Biomechanics and Strengthening

The amount of force a bone can withstand is proportional to its cross-sectional area and moment of inertia, and military studies have found these parameters to be significantly lower among those who develop stress fractures [5, 89–91]. Faulty biomechanics can contribute to stress fracture risk. These can either be due to abnormal forces or abnormal motions. Increased forces on a normally aligned lower extremity can result in abnormal bone loading, as can normal forces on a malaligned lower limb. Increased forces on a malaligned lower limb are thought to further

increase the risk of stress fracture. Runners with abnormal loading are thought to be at higher risk of stress fracture; individuals with a history of stress fracture have been found to have greater vertical ground reaction force loading rates and peak accelerations [92–94]. Torsional loads have been associated with similar risks [95], and increases specifically in peak hip adduction, absolute free movement, and peak rearfoot eversion have been associated with an increased risk of prior tibial stress fracture [93].

There seems to be a close mechanical relationship between muscle and bone, and it has been hypothesized that muscle is protective rather than causative of stress fractures. Muscles are thought to act as a shock absorber for bones during impact loading, and when they become fatigued, this may lead to increased loads on the bones. For example, fatigue in laboratory studies have shown to lead to a decrease in shock reduction [96, 97], an increase in ground reaction force loading rates and peak acceleration [98, 99], and an increase in bone strain magnitude and rate [100, 101]. Additionally, fatigue can result in altered kinematics, which may alter the direction a bone is loaded into a direction that the bone is less accustomed to bearing force [102, 103]. Studies have shown that stress fracture risk is directly related to muscle size and strength [104, 105]. Increased muscle strength has been shown to be protective from stress fractures in numerous studies including military studies that have shown that previously inactive or less active military recruits have a higher incidence of stress fractures compared to those who are active before beginning basic training [1, 21, 30, 31]. Aerobic fitness and flexibility may also play a role in this, though this has not been as well-defined [5, 19, 29, 44, 106, 107].

Medication Use

Contraceptives

The role of OCPs in the development of stress fractures remains unclear. Some studies have reported a protective effect of OCPs against stress fractures in female athletes [26, 64, 68], while others have found no association [16, 20, 31, 33, 44, 70, 108]. To complicate things further, several small studies of amenorrheic or anorexic females found individuals on OCPs to have higher BMDs in the lumbar spine and hip than those who were not [109–111]; however, other similarly designed studies showed no difference [69, 112] or a slight decrease in BMD with OCP use [113]. Conflicting results and lack of well-controlled, prospective studies make it difficult to assess the independent effects of OCPs on skeletal health in normally menstruating females and females with a menstrual disturbance. While OCPs may help to minimize further bone loss in females with menstrual disturbances, this should be done only after appropriate nutritional counseling focused on achieving and maintaining a healthy weight and well-balanced diet. Caution must be taken to ensure that the resumption of menses following OCP use is not disguising an underlying nutritional disorder, as this will not normalize metabolic factors that impair bone formation and bone health [5]. Depo-medroxyprogesterone shots may also contribute to low BMD and should be avoided in young women [114]. More research is needed on OCPs and their role in the prevention of stress fractures [70].

Other Medications

Other medications such as corticosteroids, thyroid hormone [5], antiepileptics [115], antidepressants [116], aluminum-containing antacids (such as aluminum hydroxide) [117], and proton pump inhibitors [118] are thought to impair bone health and may increase an athlete's risk of osteoporosis. Chronic corticosteroid use has been associated with a rapid loss of BMD, with loss of up to 5–15% of bone density found during the first year of medication use. In addition, individuals on chronic corticosteroids have shown a rapid, increased risk of fractures within 2–3 months of initiation, though this is reversible when medication is discontinued [119]. Oral orticosteroids have been associated with an increased risk of stress fracture within the military, though

time-dependence and reversibility is unknown [23]. While associations with low BMD have been explored, the remaining medications have not yet been investigated as risk factors for stress fractures in athletes [5].

Substance Abuse

Tobacco and Alcohol Consumption

Cigarette smoking and alcohol consumption are known to increase the risk for osteoporosis. Among female army recruits, those with a history of current or past smoking or alcohol consumption of ≥10 drinks per week were at increased risk of a stress fracture [5, 23].

Extrinsic Risk Factors

Extrinsic risk factors are those found outside the body and include poor training habits, improper equipment, type of sport or activity, and time of season. Of these, all are modifiable except time of season.

Non-modifiable Extrinsic Risk Factors

Time of Season

Stress fracture incidence has been reported to be highest during the pre-season and first 6 weeks of training in two recent prospective studies. In a study among National Collegiate Athletic Association (NCAA) athletes of all sports, there was a 42.6% increased risk of stress fracture in the pre-season when compared to the regular season based on rates per athlete exposure [120]. Similarly, among National Basketball Association players, while most injuries occurred during the regular season, nearly half occurred within the first 6 weeks of the 6-month season [121]. These results suggest that athletes are at increased risk of stress fracture during the initiation of training for a new season. It is likely that deconditioning, decreased physical fitness levels and training volume during the off-season, followed by a rapid increase in activity at the start of the season play a role in this trend, as detailed below. Regardless of causation, training and medical staff should be aware of this trend.

Modifiable Intrinsic Risk Factors

Training Variables

Heavy training volume is a major risk factor for the development of stress fractures. Higher weekly mileage is associated with an increased incidence of stress fractures [43] and overuse injuries [122–124] in runners across multiple studies [5]. Running volumes greater than 20 miles per week significantly increase risk for stress fracture [16, 20, 125, 126]. Training more than 5 hours per day is associated with a significantly higher risk of stress fractures among ballet dancers [127]. Conversely, fewer stress fractures were seen in military recruits after reducing intensity or frequency of training [128–130]. In addition to overall volume, sudden changes in duration, frequency, and/or intensity of training also alter an athlete's risk for stress fracture [5]. Altered loading associated with large changes in training may contribute to microdamage accumulation and development of stress fracture. Scaling a running program up too rapidly or too frequently is thought to disrupt the balance between bone microdamage formation and removal [105]. In addition, those with poor physical fitness and low activity prior to starting a new training program are at increased risk of stress fracture [131]. Overall, a gradual and individualized progression in training volume and intensity, with adequate recovery periods, is recommended to minimize risk for stress fracture. No specific cutoffs have been established as "too much" or "too fast," as this varies between individuals and is related to the multiple intrinsic risk factors detailed above.

Training surface has been postulated to play a role in stress fracture risk in athletes. Conceptually, uneven training surfaces can predispose to stress fractures by increasing

muscle fatigue and redistributing bone loading. Engaging in high-impact exercise on less compliant surfaces, such as cement, has been proposed as a potential risk factor. However, studies have shown inconsistent results; some have noted correlations [122, 132] while others have displayed no effect [43, 123, 124]. Interestingly, treadmill runners have been found to be at lower risk for developing tibial stress fractures when compared to overground runners, but they demonstrated less significant improvement in tibial bone strengthening [132]. The difficulty in controlling and quantifying training surfaces is a barrier in further studying this potential association [5]. Whether the increased loading rate associated with less compliant surfaces actually leads to an increased risk for stress fracture remains unclear. What may be the key with regard to stress fracture risk is whether there has been a recent change in training surface to which the runner has not adjusted [105].

Equipment Variables (Footwear and Inserts)

Athletic footwear and inserts (orthoses and insoles) are thought to decrease ground impact forces and provide stability by controlling foot and ankle motion [133, 134]. Through these two mechanisms, shoes and inserts may affect bone loading and thus the risk for stress fracture [105]. A study conducted among United States Marines found that military recruits who trained in shoes older than 6 months were at greater risk of developing a stress fracture. Shoe cost, on the other hand, has not shown any association with stress fracture risk, implying that shoe age may be a better indicator of shock-absorbing quality than shoe cost [21]. Proper orthoses have been shown to reduce stress fracture risk in military recruits [135–137]. However, there is no clear evidence that the same benefits are observed in runners [5, 105]. To complicate matters further, potential protective factors involving adopted gait mechanics (forefoot and midfoot strike gaits) by runners who run barefoot or in minimal shoes have put the role of shoes as an injury prevention tool in question [138].

Type of Sport

Type of sport engagement plays a role in risk for stress fractures. Overall, sports that place a competitive or esthetic value on leanness, often referred to as "leanness" sports, have demonstrated higher reported rates of stress fractures. In one previous study among collegiate athletes at a major university in Australia, the percentage of athletes per season who had stress fractures were found to be as follows: softball 6.3%, track 3.7%, basketball 2.9%, tennis 2.8%, gymnastics 2.8%, lacrosse 2.7%, baseball 2.6%, volleyball 2.4%, crew 2.2%, and field hockey 2.2% [11]. A more recent study looking at NCAA athletes found similar trends with cross-country, women's gymnastics, and track athletes being at highest risk based on stress fracture per athlete exposure and ice hockey, swimming and diving, and baseball/softball athletes being at lowest risk [120]. Among track and field athletes, sprinters, hurdlers, and jumpers have been found to have more foot fractures, while middle- and long-distance runners have more long bone and pelvic fractures [19]. Increased rates of rib stress fractures have been seen in rowers and golfers [139, 140].

A prior history of playing ball sports has been proposed as a protective factor in the development of stress fractures. The jumping, cutting, and sprinting involved in ball sports are thought to provide high-impact, multidirectional loading to the skeleton that may promote peak bone mass accrual and improve geometric strength [141]. Athletes who have participated in these high-impact, multidirectional loading sports, such as basketball and soccer, consistently display greater BMD and enhanced bone geometric properties when compared to those who have participated in lower-impact sports such as running [142, 143]. In the military population, infantry recruits who had participated regularly in ball sports (primarily basketball) for 2 or more years before basic training were found to have a 46–84% reduction in stress fracture risk [144].

In an elite track and field population, those who had participated in ball sports during youth were half as likely to sustain a stress fracture later in life [145]. In a group of competitive high school runners, the boys who had participated in basketball were noted to have an 82% reduction in stress fracture risk [16]. It is important to keep in mind, however, that this potential protective benefit of ball sports for long-term bone health must be weighed against the immediate injury risks that also accompany ball sports [141].

Prediction Algorithms Based on Risk Factors

The ability to utilize these risk factors to identify athletes most susceptible to stress fractures is vital in order to take early action to prevent injury. The 2014 Female Athlete Triad Coalition used six criteria to create a Female Athlete Triad Cumulative Risk Assessment Tool, scored as follows:

1. Low energy availability with or without DE/ED:
   (a) No dietary restrictions = 0 points
   (b) Some dietary restriction by self-report *or* low energy intake on diet logs *or* current/past history of disordered eating = 1 point
   (c) Meets diagnostic criteria for ED/DE = 2 points
2. BMI (absolute BMI cut-offs should not be used for adolescents):
   (a) BMI ≥ 18.5 *or* ≥90% estimated weight *or* weight stable = 0 points
   (b) BMI 17.5 to <18.5 *or* <90% estimated weight *or* 5% to <10% weight loss per month = 1 point
   (c) BMI ≤ 17.5 *or* <85% estimated weight *or* ≥10% weight loss per month = 2 points
3. BMD by z-score on DXA:
   (a) Z-score ≥ −1.0 = 0 points
   (b) Z score −1.0 to −2.0 for weight-bearing athletes = 1 point
   (c) Z score ≤ −2.0 = 2 points
4. Prior stress reaction/fracture:
   (a) No prior stress reaction/fracture = 0 points
   (b) One prior low-risk stress reaction/fracture = 1 point,
   (c) Two or more prior low-risk stress reactions/fractures OR one prior high-risk stress reaction/fracture = 2 points
5. Delayed menarche:
   (a) Onset of menarche at <15 years = 0 points
   (b) Onset of menarche at age 15 to <16 years = 1 point
   (c) Onset of menarche at ≥16 years = 2 points
6. Oligomenorrhea/amenorrhea over 12 month period (current or past):
   (a) 9 or more periods = 0 points
   (b) 6–9 periods = 1 point
   (c) <6 periods = 2 points

The summative score of these six domains is used to define an athlete as low risk (0–1 points), moderate risk (2–5 points), or high risk (≥6 points) [53]. In 2016, Tenforde and colleagues were the first to report the prevalence of stress fractures within each risk category. Athletes assigned to the higher risk categories were found to be more likely to prospectively develop a stress fracture. Nine of 169 (5.3%) low-risk athletes, 11 of 61 (18.0%) moderate-risk athletes, and 5 of 9 (55.6%) high-risk athletes sustained a stress fracture. Adjusted for cross-country participation and age, relative risk was 2.6 for moderate- versus low-risk athletes and 3.8 for high- versus low-risk athletes [146].

In 2019, Kraus et al. looked into a modified Female Athlete Triad Cumulative Risk Assessment tool to predict stress fracture risk in male athletes in a similar manner. The menstrual-related risk factors that cannot be applied to males were taken out of the assessment tool, resulting in four rather than six domains and a total possible risk score of 8 rather than 12, as follows [75]:

1. Low energy availability with or without DE/ED:
   (a) No dietary restrictions = 0 points
   (b) Some dietary restriction by self-report *or* low energy intake on diet logs *or* current/past history of disordered eating = 1 point
   (c) Meets diagnostic criteria for ED/DE = 2 points

2. BMI (absolute BMI cut-offs should not be used for adolescents):
   (a) BMI ≥ 18.5 = 0 points
   (b) BMI 17.5 to <18.5 = 1 point
   (c) BMI ≤ 17.5 = 2 points
3. BMD by z-score on DXA:
   (a) Z-score ≥ −1.0 = 0 points
   (b) Z score − 1.0 to −2.0 for weight-bearing athletes = 1 point
   (c) Z score ≤ −2.0 = 2 points
4. Prior stress reaction/fracture:
   (a) No prior stress reaction/fracture = 0 points
   (b) One prior low-risk stress reaction/fracture = 1 point,
   (c) Two or more prior low-risk stress reactions/fractures OR 1 prior high-risk stress reaction/fracture = 2 points.

Athletes were not categorized into low-, moderate-, and high-risk, but risk was instead looked at based on point increases in cumulative risk scores. Two regression analyses were performed, and both models showed an increased risk for prospective stress fracture with each point increase in cumulative risk score. Depending on the model used, each risk assessment point was associated with a 27–37% increased risk for stress fracture [75].

Such risk assessment models for female and male athletes, respectively, show potential in helping to identify athletes at higher risk of stress fractures. Ideally, athletes who fall in the moderate and high-risk groups, or those with high cumulative risk scores, should be identified early and modifiable risk factors should be assessed and optimized prior to the development of a bone stress fracture.

Conclusion

Identifying risk factors for developing stress fractures is the first step to optimum care of athletes. There are intrinsic and extrinsic risk factors, some of which can be modified to decrease risk. Recognizing these risk factors can help identify athletes who are at high risk of developing a stress fracture and can help guide the management of athletes to ensure their peak health and performance. Prediction algorithms utilizing these risk factors have been tested and show viability.

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