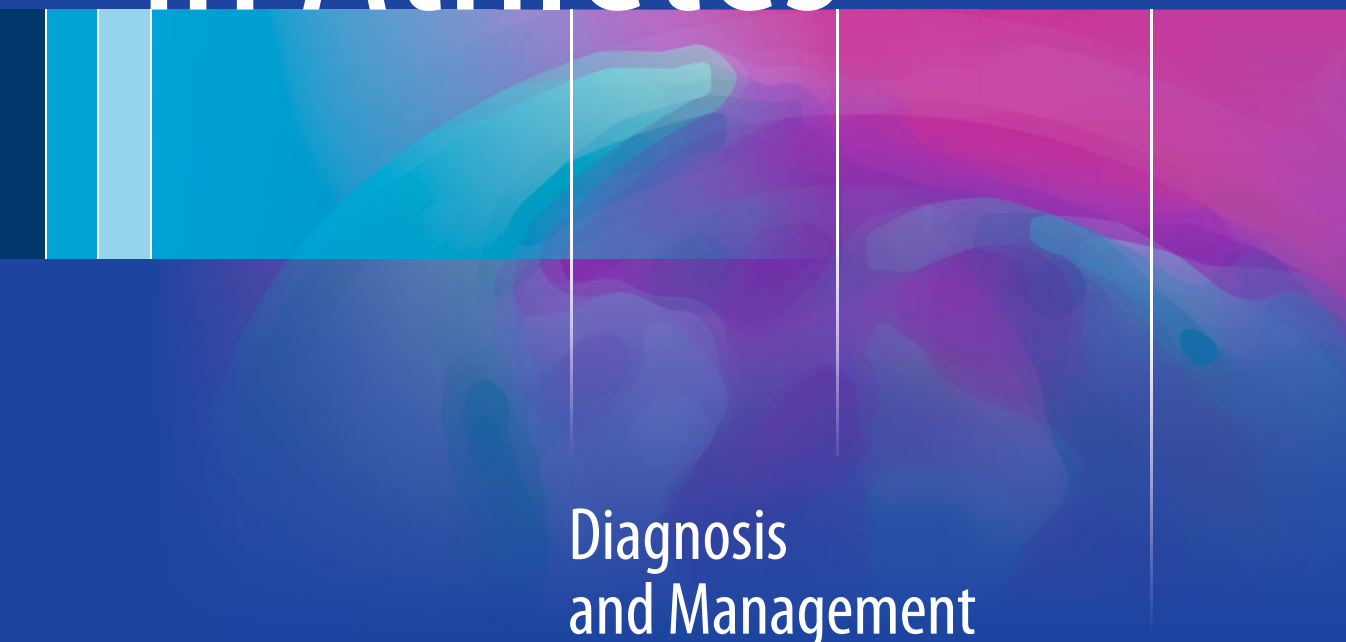
Timothy L. Miller
Christopher C. Kaeding
*Editors*

# Stress Fractures in Athletes



## Diagnosis and Management

Springer

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*To my family, in particular my parents, Tom and Kathy,*
*and my wife, Nicole, for always believing in me and always*
*showing patience and understanding when I take on*
*"yet another 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. David B. Castle, without whose coaching*
*and mentoring this journey and love affair with stress*
*fractures would have never begun.*

Timothy L. Miller, MD

*To my wife, Christine, for her bottomless source of patience*
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*and unexpected interruptions.*
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*advice, encouragement and "prodding." Without his influence*
*and mentoring, my career and this book would not have happened.*

Christopher C. Kaeding, MD

# Preface

*Stress Fractures in Athletes* has been in many ways a labor of love for the editors. It is the culmination of many years of experience with stress fractures both as athletes and as team physicians. This textbook compiles the many concepts, experiences, and techniques required to approach and treat the complexities of stress-induced injuries to bone among the athletically active population. 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. As a developing field of Sports Medicine and Orthopaedics, Endurance Medicine continues to expand its understanding of overuse injuries as athletes continue to push the limits of running, jumping, biking, swimming, skiing, rowing, cross-fit sports, adventure sports, and many other demanding activities. Traditional treatment strategies for stress fractures such as simply stopping the causative activity or sport are no longer considered an acceptable option for many competitive athletes. Alternative training methods, including a holistic approach to the evaluation, treatment, and prevention of stress-induced injuries to bone, are now the standard of care as is evidenced throughout the 16 chapters of this book. This textbook details treatment options for bony injuries throughout the body from the spine and pelvis to the hands and feet. Even though it is too early to determine whether we can obviate the need to have athletes completely abstain from their sport of choice in response to a stress fracture, we can decrease the time lost from training and competition and allow for a more safe and predictable return to full activity. It is our hope that this textbook will be a valuable guide for sports medicine physicians, orthopaedists, athletic trainers, physical therapists, coaches, parents, and athletes in their evaluation and treatment of stress fractures.

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# Abbreviations

| | |
|---|---|
| AIIS | Anterior inferior iliac spine |
| AP | Anteroposterior |
| ASIS | Anterior superior iliac spine |
| BMD | Bone mineral density |
| BMI | Increased body mass index |
| COX | Cyclooxygenases |
| CT | Computed tomography |
| DEXA | Dual-energy X-ray absorptiometry |
| ECSW | Extracorporeal shock wave |
| EMF | Electromagnetic fields |
| EMG | Electromyography |
| ESWT | External shockwave therapy |
| FABER | Flexion, abduction, and external rotation |
| MRI | Magnetic resonance imaging |
| NSAIDs | Non-steroidal anti-inflammatories |
| pQCT | Peripheral quantitative computed tomography |
| PSIS | Posterior superior iliac spine |
| PTH | Parathyroid hormone |
| SCFE | Slipped capital femoral epiphysis |
| SI | Sacroiliac |
| SPECT | Single-photon emission computed tomography |
| STIR | Short tau inversion recovery |
| Tc-99m-MDP | Technetium-99m-methylene diphosphonate |

# Part I

# Presentation and Diagnosis of Stress Fractures

# 1 Pathophysiology and Epidemiology of Stress Fractures

David Wasserstein and Kurt P. Spindler

## Stress Fracture Pathophysiology

To understand the pathophysiology of stress fractures in bone, a review of basic bone biology, including normal bone metabolism and turnover, is necessary. From this understanding, the pathophysiology of stress fracture development will be outlined. Finally, this section will identify individual clinical parameters that have been linked to the development of stress fractures, and summarize their implication and relevance.

## Bone Biology

Bone has two forms at the microscopic level—woven and lamellar bone. Woven bone is immature with random orientation and collagen that is not stress oriented. Lamellar bone, in contrast, is mature and organized with stress-oriented collagen [1]. The mechanical properties of lamellar bone can change depending on the direction of the applied force. The macroscopic subtypes of lamellar bone include cortical and cancellous (trabecular) bone. The former is denser and has a low turnover rate. It is composed of packed osteons also called Haversian systems, which are connected by Haversian canals (Fig. 1.1). These canals contain the neurovascular supply of bone. Cancellous bone, however, has a higher turnover and is between 30 and 90 % porous, depending on the location. Cancellous bone is found more commonly in the metaphysis of long bones, compared to cortical bone, which is found in the diaphysis.

The matrix of bone is approximately 40 % organic and 60 % inorganic [1]. The organic portion of bone is primarily type-1 collagen—the component that provides tensile strength. The remaining organic portion (~10 %) consists of proteoglycans, which provide compressive strength, and matrix proteins. The function of these matrix proteins (e.g., osteocalcin) is to promote mineralization and bone formation. The inorganic component includes calcium hydroxyapatite, which is responsible for compressive strength, and osteocalcium phosphate. The inorganic component is also the mineral portion, which plays a role in calcium metabolic pathways [1].

Normal bone metabolism is a balanced sequence of bone turnover that includes bone breakdown, known as osteoclastogenesis, and bone formation, known as osteoblastogenesis. Osteoclasts are the cells primarily responsible for osteoclastogenesis, and osteoblasts for

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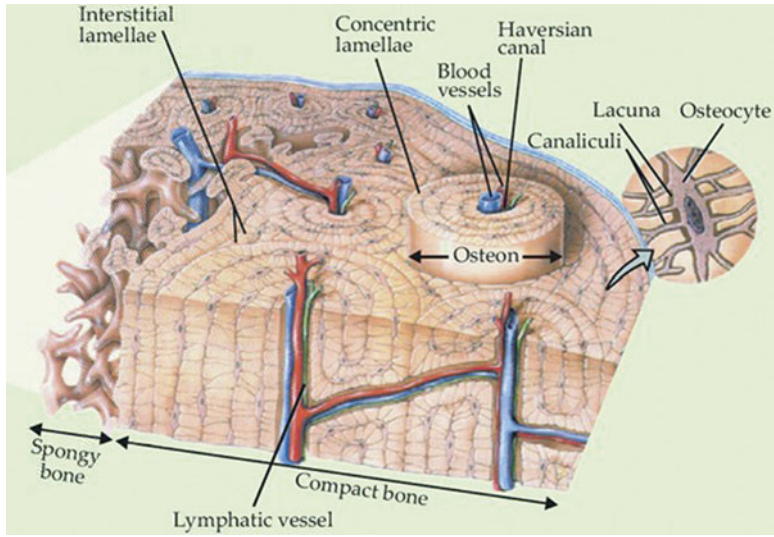


**Fig. 1.1** Illustration of the Haversian system and vascular supply in cortical bone. With permission from Springer Science + Business Media: Initiation Fracture Toughness of Human Cortical Bone as a Function of Loading Rate, 2013, C. Allan Gunnarsson

osteoblastogenesis. Many endogenous hormones regulate metabolism, including parathyroid hormone (PTH), calcitonin, growth hormone, thyroid hormone, estrogen, and testosterone. Endogenous and exogenous steroids, including vitamin D and glucocorticoids, also regulate both calcium and bone metabolism [1]. Factors that promote bone formation do so by either promoting osteoblastogenesis (e.g., PTH, vitamin D) or suppressing osteoclastogenesis (e.g., calcitonin, estrogen). Factors that promote bone breakdown typically suppress osteoblastogenesis (e.g., glucocorticoids).

When stress is applied to bone, Wolff's law dictates that bone will remodel in response to mechanical stress. The exact method by which bone remodels is not truly understood, but two theories predominate. In the piezoelectric charge theory, tensile-sided strain is said to create electropositive forces that stimulate osteoclastogenesis, while the compression side is subject to electronegative forces that stimulate osteoblastogenesis [1]. The result is the formation or remodeling of bone to increase bone mass on the compressive side in response to mechanical stress. A second theory, the Hueter–Volkmann law, states that bone remodels in small packets of cells in a process called osteoclastic tunneling. Here, there is bone resorption followed by capillaries to introduce blood supply and osteoid-producing cells to lay down new osteoid [1].

## Bone Pathophysiology in Stress Fractures

"Stress fracture" constitutes a spectrum of injury that includes bone strain, stress reaction, and stress fracture. The etiology is repetitive loading in the setting of inadequate bone remodeling. The spectrum of injury reflects to some degree the quantity of strain, although exact thresholds are not known and likely mediated by numerous individual host factors in addition to the inciting activity. In general, repetitive injury is more likely to occur in the lower extremity, which sees greater loads than the upper extremity in ambulatory athletes, and with activities that are high volume and offer repetitive loading. Running, for example, produces ground reaction forces approximately five times greater than walking. The result of excess strain is an accumulation of microdamage leading to fatigue reaction or fatigue failure. When the area of

fatigue failure is inadequately repaired, it can result in crack initiation in the bone [2] (Fig. 1.2). A simple model is illustrated in Fig. 1.3.

Stress injury may also occur with normal strain, but this is typically in the setting of depressed bone remodeling. These injuries are known as insufficiency reactions or fractures. They are more common in the setting of metabolic diseases, hormonal imbalances, and osteoporosis. In the setting of older persons with osteoporosis, both reduced remodeling and structural changes in the trabecular and cortical bone leading to reduced biomechanical strength, and contribute to the susceptibility to insufficiency fracture at physiologic loads [3]. The dichotomy of fatigue failure and insufficiency is certainly more of a continuum with respect to athletes. These individuals experience greater than physiologic strain through activity but also exhibit risk factors for insufficiency failure, putting some subpopulations of athletes at greater risk.

Another special consideration in the pathophysiology of stress fractures in athletes is the influence of skeletal muscle. Muscles may protect the tibia during running by producing shear forces that counteract the joint reaction forces and result in reduced net shear stresses in the tibia. It has been hypothesized that reduced lower leg muscle strength increases the risk of stress fracture through this mechanism [4, 5], and the concept may extend to other common areas of stress fracture. This theory has only been tested in one clinical study, where a significantly lower knee extension power was observed in a case–control study of female runners with and without stress fractures [6]. Others have hypothesized that this potential protective effect of muscle may be diminished with the fatigue associated with excessive training, or be reduced in new exercisers and military recruits [7].

Finally, there is an oxidation deprivation theory of stress fracture development, which deserves some attention. In this theory, the repeated load of an activity such as running is thought to cause decreased oxygen delivery [8] and brief ischemia [9, 10] in weight-bearing bones. This ischemic environment is thought to stimulate the bone-remodeling process, specifically by increasing osteoclastogenesis [11]. The result is weakened bone that is less able to withstand subsequent loads, thereby increasing susceptibility to further stress-related injury. This theory may explain some observations that those new to activity are more at risk [12, 13].

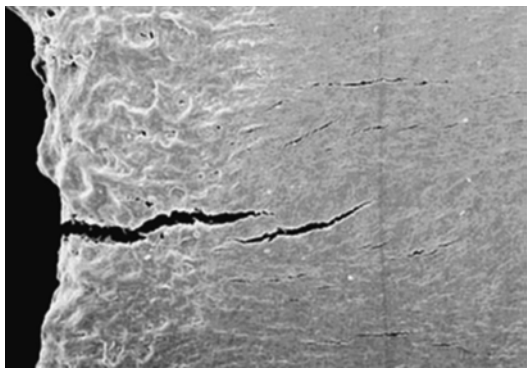

**Fig. 1.2** Crack initiation in bone. Reprinted by permission from Macmillan Publishers Ltd: Nature Materials, Nalla RK, Kiney JH, Ritchie O. Mechanistic fracture criteria for the failure of human cortical bone, 2(3). Copyright 2003

## Host Risk Factors for the Development of Bone Stress Injury

### Bone Mineral Density and Bone Thickness

Although lower bone mineral density (BMD) is likely a stronger etiological factor in insufficiency fracture development, there is evidence that BMD

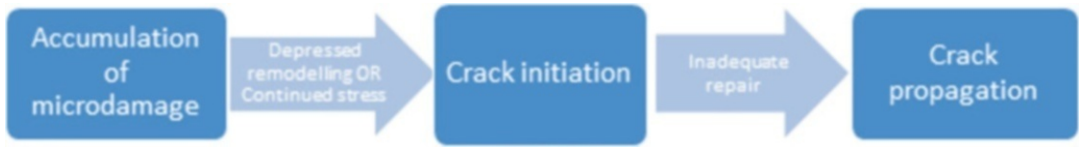


**Fig. 1.3** A simple model for the propagation of stress injury in bone

also plays a role in athletes experiencing fatigue failure-related stress fractures. Loud et al. [14] performed a case–control study of female athletes aged 13–22 years who were diagnosed with their first stress fracture. These patients were matched by age and ethnicity to two controls. The authors demonstrated that cases had lower spine BMD for their age, despite no differences in menstrual irregularity or physical activity participation. Similarly, the odds of a stress fracture were three times that for persons with a family member diagnosed with osteoporosis.

Another case–control study [6] of female athletes aged 18–45 years with and without stress fractures noted that after adjusting for body weight, those with stress fractures had thinner tibial cross-sectional area, lower trabecular BMD, and less cortical area of the posterior tibia.

These associations have been confirmed by prospective studies. The first [15] was a 12-month study of both female and male track and field athletes aged 17–26 years. At baseline, females with lower BMD in the spine were at significantly greater risk of developing a stress fracture. A study of military cadets [16] since has demonstrated that smaller tibial cortical area, lower tibial bone mineral content and smaller femoral neck diameter increased the risk of developing a stress fracture in males, and smaller femoral neck diameter was a risk factor in females.

## Genetics

There appears to be some genetic susceptibility to stress fracture. Early investigation concluded that ethnicity was a risk factor for the development of stress fracture, with lower rates seen in African-American compared to Caucasian and Asian women. Much of this difference, however, may be related to inherited differences in bone metabolism through bone mineralization. One study has demonstrated an inherited difference in calcium excretion [17].

The association between a family history of osteoporosis in first degree relatives and increased risk of developing a stress fracture among athletes [6] also suggests there is a genetic role in bone turnover as a risk factor.

## Nutritional Factors

Dietary and nutritional factors may play a role in the pathophysiology of stress fracture. Calcium and vitamin D are important components of normal bone metabolism and contribute to BMD, with the former being a mineral building block and the latter playing a role in both calcium homeostasis and bone turnover. One randomized trial of female military recruits found a 20 % reduction in fracture injuries with supplementation of 2,000 mg elemental calcium and 800 IU vitamin D compared to no supplementation [18]. Other research has been inconclusive as to whether dietary intake of calcium is important in the development of stress fractures [19, 20].

Other macronutrients may play a role in susceptibility to stress fractures, although the potential pathophysiologic mechanisms are unclear. Merkel et al. [21] demonstrated that among asymptomatic female military recruits, only those females with low iron anemia developed a stress fracture.

## Menstrual Irregularity

Late-onset menarche appears to be a risk factor for stress fracture development [15, 16]. It is unclear whether this is due to low peak bone mass attainment, or whether it is a marker of another influence such as excessive training, or low body weight/body fat. The association is further confounded by the fact that under normal circumstances female athletes appear to reach menarche later than their non-athlete counterparts [22].

Disordered menstruation has also been linked to stress fracture risk. Estrogen functions to increase bone mass by inhibiting osteoclastogenesis. It may also function by reducing the adaptation to stress [23]. As such, numerous studies have demonstrated that female athletes who are amenorrheic [19, 24, 25] or oligomenorrheic [19, 20, 26] are at increased risk of stress fracture. Authors have hypothesized about the combined role of menstrual irregularities and low BMD in some female athletes with the so-called “female athlete triad” (disordered eating, amenorrhea, and decreased BMD).

## Summary

Bone stress injury occurs via an imbalance of repetitive stress and normal bone remodeling/recovery in response to that stress. Although the paradigms of fatigue failure (high stress overwhelming normal turnover) and insufficiency failure (normal stress overwhelming disordered turnover) are a simple means of conceptualizing this disorder. In reality components of both will contribute to stress injury in any one individual. This is further complicated when one considers that many of the host factors that influence the pathophysiology of bone stress injury are also interrelated. The findings from a study such as that performed by Cosman et al. [16] illustrate that even with the current state of knowledge, we can explain only a small proportion of the risk for stress fracture development. More research is warranted.

From a practical standpoint, the clinician who will diagnose and treat patients with bone stress injuries must understand the basics of bone biology, including stress remodeling. Once a diagnosis has been made, further probing into the potential role of etiologic factors is recommended. This may include diet and nutritional deficiencies, menstrual irregularity, family history, and training volume. Some of these factors may be modifiable and useful in both the treatment of the current stress injury, as well as the prevention of future injury.

## Stress Fracture Epidemiology

The epidemiology of stress fractures is described as the occurrence of stress fractures in athletic populations, and is typically expressed on the basis of exposure (e.g., number of stress fractures per athlete-years or per athlete-exposures). One of the challenges in defining the incidence of stress fractures lies in accurately determining the exposure component. Stress fracture cases are comparatively easy to identify, typically through chart records or physician visits. The challenge of a retrospectively designed study is that while it may identify most or all stress fractures over a given time period, accurate information regarding athletic exposure is comparatively lacking. Consistent and accurate injury reporting data is important to identify risk factors, at-risk subpopulations, and monitor the effectiveness of interventions.

A second complicating factor in deciphering the literature defining the occurrence of stress fractures in athletes is the method of diagnosis. Older studies used modalities such as X-ray, which can have poor sensitivity in identifying changes [27]. Many newer studies utilized bone scan or MRI techniques, which offer greater sensitivity and will identify stress fractures at an earlier stage. The MRI is so sensitive that it can detect stress reaction, a precursor to stress fractures, and thus studies utilizing this method of detection will report a greater incidence/occurrence but for a broader spectrum of clinical disorder. Many of these topics are explored in further detail in the remaining chapters of this text.

This heterogeneity in diagnosis, study design, and accuracy of exposure precludes the pooling of data to formulate incidence rates by sport or activity, at the current time. Therefore, this chapter will focus on a descriptive review of the literature, the most robust of which originates from military populations. Studies from various sports will also be reviewed and interpreted. A preference towards higher level of evidence studies published in the last 10 years is given.

## Stress Fracture Epidemiology: Military

Military populations are a unique group that facilitates epidemiological research on stress fractures. Patient follow-up and activity exposure can be well controlled and documented, which allows for more homogeneous comparisons and higher level of evidence designs such as prospective cohorts. Additionally, large numbers of patients can be recruited for study, which is helpful when investigating a condition that typically occurs infrequently or when performing multivariate analyses to identify risk factors.

Most important, however, is that military personnel appear to have a higher incidence of stress fractures than the general population due to the suddenly increased and extensive exercise associated with training. Accordingly, military studies on stress fractures have been performed all over the world, including the USA [16, 24, 27–29], Finland [30], and Israel [31].

A common theme in this population is a higher reported occurrence or incidence of stress fractures among females compared to males. In one study of cadets, 19.1 % of females and 5.7 % of males reported at least one stress fracture [16]. Similarly, in the largest studies of US Army recruits [28], the incidence of stress fractures was 79.9/1,000 female and 19.3/1,000 male recruits. This pattern holds true internationally. An Israeli military study [31] identified a similar discrepancy (ratio 2.13) of bone scan positive stress fractures in females (23.9 %) to males (11.2 %). A similar pattern was seen among a prospective cohort of 152,095 Finnish conscripts [30], where the ratio of female to male bone stress injury on MRI was 9:2. The overall incidence rate of stress fractures in this population was 311/100,000 person-years (95 % confidence interval: 277–345).

There also appears to be a difference in the distribution of stress fracture location between male and female military personnel. Compared to males, females have higher reported rates of stress fracture for the pelvis [30, 31], sacrum [30], and tibia [30, 31].

These sex differences have prompted many researchers to specifically study female recruits. Shaffer et al. [24] identified a stress fracture rate of 5.1 % in a cohort ($N$=2,962) of female US marine recruits. All stress fractures occurred in the lower extremity, most commonly in the tibia, followed by the metatarsal bones, pelvis and femur. In regression analysis the odds of developing a stress fracture were more than five times higher among recruits who were amenorrheic during the prior year (odds ratio 5.64, 95 % confidence interval 2.8–25.8). Lower aerobic performance on a timed run also increased the odds of developing stress fractures in the pelvis and femur.

In a separate study of female US Marine Corps recruits [25], the same authors reported on all overuse injuries of the lower extremity [24]. They determined an incidence rate of lower extremity stress fractures of 1.0/1,000 days of training exposure. Having multiple overuse injuries was common, and in multivariate regression analysis, again lower aerobic fitness and amenorrhea predicted increased odds of stress fracture.

Among lower extremity stress fractures in military populations, the tibia and metatarsals appear most common [16, 29]. A rarer occurrence is the calcaneal stress fracture. One study identified calcaneal stress fractures from MRI in recruits who had undergone ankle MRI for exercise induced heel or ankle pain [27]. The incidence rate of stress fractures among all recruits during the study period was 2.6/10,000 person-years (95 % confidence interval 1.6–3.4). Most calcaneal stress fractures were found in the posterior aspect of the bone, and 22/34 (65 %) were associated with stress fractures in other tarsal bones. A comparison to plain radiographs in the same patients revealed only 15 % had abnormal films, attesting to the higher sensitivity and ability to detect stress changes at an earlier stage by MRI.

## Stress Fracture Epidemiology: Running

Runners are at higher risk of developing stress fractures. In many cases, however, athletes may compete in multiple sports, and attributing stress fractures solely to running can be challenging. A survey study of 1,505 runners performed in 1990 [32] identified female long-distance runners at highest risk for stress fracture.

Since that survey, two prospective cohort studies have attempted to better define the epidemiology of stress fractures in runners. One study of 748 competitive high school cross-country and track and field runners identified a 5.4 % and 4.0 % rate of stress fractures in girls and boys, respectively [33]. The tibia and metatarsal bones were among the most commonly affected. Multivariate models identified late menarche, low BMI and a prior history of stress fracture as significant contributors to increased risk of new onset stress fracture. In a second, smaller cohort study [34] of competitive high school runners followed for 3 years, stress fractures were identified in 21/230

**Table 1.1** Stress fracture epidemiology by miscellaneous sports

| Reference | Sport | Study design | *N* | Incidence | Notes |
|---|---|---|---|---|---|
| Pearce et al. [40] | Rugby | Prospective cohort | 12/899 (8 %) | – | Navicular SF associated with longest time away |
| Ekstrand et al. [41] | Football | Prospective cohort | 51/2379 | 0.04/1,000 h | 78 % fifth metatarsal; 29 % re-injury; 3–5 months absence |
| McCarthy et al. [42] | Women's basketball | Case series | 506 (7.3 %) | – | WNBA player injury reports at draft |
| Frost et al. [43] | Cricket | Prospective cohort | 248 | 51.6/10,000 player-h | Professional; SF to low back had longest return to play |
| Ekegren et al. [44] | Ballet | Prospective cohort | 266 | Not stated | SF had longest return to participation |

(9.1 %) athletes, representing an incidence of 0.06 stress fractures per athlete exposure.

## Stress Fracture Epidemiology: Tennis

The nature of tennis lends the potential for stress fracture development in both the racket hand and lower extremity from running and sudden stops. Abrams et al. [35] reviewed the literature for case reports on uncommon stress fracture locations in tennis players, and identified them in the ischium, first rib, humerus, sacrum, patella, hook of hamate, ulna, and distal radius. Another study [36] examined a case series of high level junior tennis players, noting seven cases of second metatarsal stress fractures postulated to be related to racket grip.

The largest tennis study followed 139 elite tennis players of a median age 20 years, 65 % male and 57 % professional status over the course of 2 years [37]. In total, 15 players had 18 stress fractures for a rate of 12.9 %. The most common location was the navicular (5/18), pars interarticularis (3/18), metatarsals (2/18), tibia (2/18) and lunate (2/18). There were also more stress fractures among juniors (20.3 %) compared to professionals (7.5 %). Unfortunately, none of these studies provided a metric for exposure to calculate an incidence rate.

## Stress Fracture Epidemiology: Pediatric/Adolescent Athletes

Particular attention has been directed towards pediatric/adolescents with respect to describing stress fractures. This is an important subpopulation due to potentially open physes and associated metabolic changes that accompany menarche. A national survey study of adolescent girls [38] has followed 6,831 girls aged 9–15 years for 7 years. Among them, 267 (3.9 %) developed a stress fracture. Multivariate modeling demonstrated that running, basketball, cheerleading, and gymnastics were all significant predictors of developing a stress fracture.

In a retrospective case series of pediatric athletes with open physes, Niemeyer et al. [39] followed 19 children with 21 stress fractures over a mean 4.8 years. The mean age at diagnosis was 14 years, and most fractures were found in the lower extremity. They noted tibial stress fractures were more likely to accompany sports with sudden stops, and were also associated with a longer course of treatment.

## Stress Fracture Epidemiology: Other Sports

Individual case reports and series have been published, documenting the occurrence and incidence of stress fractures in various sports. These are reviewed in Table 1.1.

## Summary

The reported incidence and occurrence of stress fractures in the literature is variable. The most robust data from the military suggests that new activity (i.e., recruits) and females have the highest

incidence of stress injury. Among athletes, the pattern of injury and incidence/occurrence varies by sport and level of competition.

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