

# **Stress Fractures and Bone Health in Track and Field Athletes**

Aurelia Nattiv, M.D., FACSM

Department of Family Medicine, Division of Sports Medicine and Department of  
Orthopaedic Surgery, University of California, Los Angeles School of Medicine Los  
Angeles, California USA

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Nattiv, A. (2000). Stress fractures and bone health in track and field athletes. *Journal of Science and Medicine in Sport* 3 (3): 268-279.

The effect of exercise on bone health has received much attention in recent years. The problems of the female athlete triad: disordered eating, amenorrhea and osteoporosis have helped us to better understand and appreciate the important interaction of mechanical, hormonal, nutritional as well as genetic factors on bone health in the young female athlete.

The relatively high stress fracture incidence of young track and field athletes can be quite disabling for the athlete's present and future running career. A number of risk factors including low bone mineral density (BMD), menstrual irregularities, dietary factors and prior history of stress fractures have been associated with an increased risk for stress fractures in the female athlete. Few studies have found risk factors for stress fractures in the male athlete. Female gender has been found to be a risk factor for stress fractures in the military population, but this finding is less apparent in athlete studies. Caucasians have been found to have a higher risk for stress fractures than African-American military recruits, but there is very limited data assessing stress fracture risk in athletes of varying ethnicity.

Prevention of stress injury to bone involves maximizing peak bone mass in the pediatric and young adult age groups. Maintaining adequate calcium nutrition, caloric intake as well as hormonal and energy balance are important preventive measures, as are ensuring appropriate amounts of weight bearing exercise for optimizing bone health and preventing fractures. More research is needed to determine factors leading to improvements in bone density and fracture reduction in athletes at risk.

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## **Introduction**

Research in the area of bone health in the athletic woman has reached a heightened level of knowledge over the last decade. The interrelationship of the problems of the female athlete triad—disordered eating, amenorrhea and osteoporosis (ACSM Position Stand 1997; Drinkwater et al., 1984; Nattiv et al., 1994)—has helped us to better understand and appreciate the important interaction of mechanical, hormonal, nutritional as well as genetic factors on bone health in the young female athlete (Nattiv & Armsey, 1997). Less is known about the interaction of these factors in the male athlete. Despite our increased knowledge of the effects of exercise on bone, as well as potential risk factors which may lead to premature osteoporosis and stress fractures in the young athlete, there still exists a lack of prospective longitudinal data in this area. Furthermore, there are a lack of interventional studies in the young athlete which provide evidence of treatments which demonstrate an increase in bone density or reduction in stress fractures in the young athlete.

This article will review current concepts of bone stress injury and imaging, as well as review the epidemiology and risk factors for stress fractures in the young athlete, including recent findings of the author's prospective investigations assessing incidence and risk factors for stress fractures in male and female collegiate track and field athletes, and will conclude with strategies for optimizing bone density and reducing risk for stress fractures.

### **Current Concepts of Bone Stress Injury and Imaging**

Bone is a specialized dynamic tissue that is continuously renewed by the process of bone remodeling. Remodeling is comprised of cycles of bone resorption and bone formation that occur throughout skeletal life. With exercise, external mechanical forces are transmitted upon the mineral matrix of bone and resultant adaptive remodeling occurs in a predictable pattern as described by Wolff's law (Wolff, 1892). Bone stress injury results when there is a temporary disturbance in the equilibrium between bone resorption and bone formation (Arendt, 2000).

Advances in the understanding and imaging of bone stress injury have led to the current concept of stress injury to bone occurring on a continuum (ranging from the normal dynamic processes of bone modeling and remodeling occurring on one end of the continuum), progressing on to bone fatigue and, with further stress, to a frank cortical stress fracture. Terms such as bone stress injury, bone strain and stress reaction are used to reflect this progression of bone injury toward a frank stress fracture.

Earlier studies in the military assessing stress fracture incidence used radiographs only for diagnosis. Radiographs however, lack sensitivity for diagnosing bone stress injury, especially in the early stages of bone injury and remodeling. Many studies assessing stress fractures have used radionuclide bone scanning for stress fracture diagnosis. However, military studies have demonstrated that bone scans often are positive in asymptomatic people, suggesting that they may be too sensitive. Although a grading system exists with radionuclide bone scans (Chisin, 1987; Zwas et al., 1987), the scintigraphic pattern may lack specificity for bone stress injury.

Advances in imaging with magnetic resonance imaging (MRI), with its superior spatial resolution and sensitive depiction of inflammation, has shown that earlier stages of bone stress injury such as bone edema, can be diagnosed prior to frank fracture. A grading system used by Arendt and Griffiths (1997; 2000) and Fredricson and colleagues (1995) have suggested that the severity of stress related bone stress injury of the tibia can be determined with MRI findings in the periosteum and marrow, and with different findings on the T1 and T2 weighted MR images. Fredricson and colleagues (1995) found that certain physical findings, such as pain with daily ambulation and tibial tenderness with direct or indirect percussion, correlated with more severe tibial stress injury as noted on the MRI. They found that MRI more precisely defined the anatomic location and extent of injury of tibial stress reactions compared to bone scans. Yao and colleagues (1998) have found that a cortical abnormality or medullary line on MRI indicated a more severe stress injury, but that earlier grades were not prognostic of clinical outcome. More data is needed assessing the use of MRI for grading bone stress injury and correlating the pattern to clinical outcome.

## **Stress Fracture Epidemiology**

### **Incidence Rates in Athletes**

Although running is the activity most commonly associated with stress fractures (Hulkko & Orava, 1987), there are a lack of studies that compare stress fracture incidence rates in athletes participating in different sports (Bennell et al., 1997). In addition, there are a lack of studies that assess incidence in terms of athlete exposure or incidence per number of training hours. Only one published prospective study in track and field athletes exists which calculates incidence of stress fractures in terms of training hours (Bennell et al., 1996). Although the National Collegiate Athletic Association (NCAA) in the United States has injury data for a variety of sports and injuries, there is no injury data available that assesses incidence of stress fractures in the track and field athlete.

There are a number of reasons that make it difficult to determine the incidence of stress fractures in the track and field athlete and compare this incidence to athletes in various sports (Bennell & Brukner, 1997). The definition of stress fracture in studies is often different. Some studies include the number of stress fractures that have occurred over a given period of time, whereas other studies assess the number of athletes that have sustained a stress fracture. The former method would result in a higher incidence of stress fractures. The criteria for diagnosing stress fractures varies in studies (radiographs, bone scan, MRI, computed tomography), making incidence rates difficult to compare. Furthermore, unlike the elegant military studies where there is a known denominator and fairly consistent training patterns and environment, observing a finite group of runners prospectively for the same period of time who have similar training patterns and are exposed to similar conditions is difficult.

Despite these problems, there are a number of studies that have assessed stress fracture incidence rates in the track and field athlete and competitive runner (Barrow & Saha, 1988; Bennell et al., 1995; Bennell et al., 1996; Brunet et al., 1990; Cameron et al., 1992; Goldberg & Pecora, 1994; Johnson et al., 1994; Nattiv et al., 2000), as well as other professional and recreational athletes. An excellent comprehensive review of the epidemiology of stress fractures is written by Bennell and Brukner (1997). Of the studies assessing stress fracture incidence rates in competitive track and field athletes, there are only three that are prospective (Bennell et al., 1996; Johnson, 1994; and Nattiv et al., 2000). Bennell and colleagues found an incidence in the track and field athlete of 20% (expressed as number of athletes sustaining stress fractures). If expressed as number of stress fractures per 100 athletes, the incidence rate in this study would be 27%. Johnson and colleagues (1994), in their two year study assessing collegiate athletes in a variety of sports, found an annual incidence of 19% in the track and field athlete (expressed as number of stress fractures per 100 athletes). In the authors four year prospective study of collegiate track and field athletes (Nattiv et al., 2000), an average incidence rate of 11.0% (stress fractures per 100 athletes) was found. Differences in the patient population and imaging techniques used for diagnosis may explain the differences in incidence in these studies.

### **Gender Differences**

A number of studies support a greater risk for sustaining stress fractures in women. This is especially noted in military studies (Brudvig, 1983; Jones et al., 1993; Jones et al., 1989; Pester & Smith, 1992; Protzman & Griffis, 1977; Reinker

& Ozburne, 1979; & Besterman, 1982). Bennell and Brukner (1997) have pointed out that the gender differences in stress fracture rates is not as evident in athletic populations. Bennell and colleagues (1996) did not find a statistically significant difference in stress fracture incidence in track and field athletes between genders. In the authors' experience assessing risk factors in collegiate track and field athletes over four years, there was a trend for women to have a higher incidence of stress fractures, but this did not quite reach statistical significance (Nattiv et al., 2000). Bennell and Brukner (1997) suggest that the discrepancy in stress fracture rates in males and females may not be attributable to gender per se, but rather to gender-related factors such as menstrual history, nutrition and bone density. Other possible explanations include fitness level. In military studies, when recruits were stratified by fitness, low levels of running performance were associated with a higher incidence of stress fractures (Jones et al., 1993). Gender differences in stress fracture incidence were less apparent in the male and female recruits when fitness was taken into account.

### **Differences in Skeletal Site Distribution**

The tibia is consistently the most common skeletal site of stress injury in athlete studies. Other common sites of stress injury in runners include the metatarsal, fibula, navicular and femur. Of note is that a gender pattern in the skeletal site distribution of stress fractures has been observed by several authors (Bennell et al., 1996; Hulkko & Orava, 1987; Orava, 1980). These authors have found that women tend to develop more metatarsal and pelvic stress fractures and fewer fibular fractures than men. Bennell and colleagues (1996) also noted that distance runners sustained a greater number of long bone and pelvic fractures, while sprinters, hurdlers and jumpers sustained a greater number of stress fractures of the foot. The author found that distance runners sustained a significantly higher number of stress fractures than athletes in other track and field events (Nattiv et al., 2000).

### **Risk Factors for Stress Fracture**

The relatively high stress fracture incidence in young track and field athletes can be quite disabling for the athlete's present and future running career, and is often career ending. Research that assesses stress fracture risk factors in runners can therefore provide significant insight in the prevention and treatment of these potentially devastating injuries.

Although most studies on stress fracture risk factors come from the military, several risk factors for stress fractures in the young athletic population have been postulated. Studies in athletes have correlated low bone density, delayed menarche and menstrual irregularities, less use of oral contraceptive pills (OCP), lower dietary calcium, lower dietary fat, decreased calf girth, less lower extremity lean mass, a discrepancy in leg length, increased training intensity, lower hip eccentric strength, lower body weight, lower body fat, as well as prior history of stress fracture as risk factors for stress fractures. (Barrow & Saha, 1988; Bennell et al., 1996, Myburgh et al., 1990; Nattiv, et al. 2000).

In the male military population (Milgrom et al., 1989) and more recently in male runners (Crossley et al., 1999), mechanical factors including a narrower tibial diameter and a lower area moment of inertia have been correlated with an increased risk for stress fractures. The findings in Bennell's study that calf girth was a strong independent predictor of stress fractures in female track and field

athletes (Bennell et al., 1996), ie. small calf girth increasing risk, may be explained by the inability of the calf muscles to resist the loading produced at ground contact and to decrease excessive strain on bone (Scott et al., 1990). In fact, Bennell and colleagues found that for every 1 cm decrease in calf girth, the risk of sustaining a stress fracture increased fourfold (Bennell et al., 1996).

Additional risk factors for stress fractures that have been identified in the military population include external rotation of the hip (Giladi et al., 1991), low aerobic fitness (Jones et al., 1993), female gender (Brudvig et al., 1983; Jones et al., 1993; Jones et al., 1989; Pester & Smith, 1992; Protzman & Griffis, 1977; Reinker & Ozburne, 1979; Scully & Besterman, 1982), increasing age (Brudvig et al., 1983; Gardner et al., 1988), decreasing age (Milgrom et al., 1994) and Caucasian ethnicity (Brudvig et al., 1983; Gardner et al., 1988; Milgrom et al., 1994).

There have been two prospective studies assessing stress fracture risk factors in the track and field athlete (Bennell et al., 1996; Nattiv et al., 2000). Bennell and colleagues assessed Caucasian track and field athletes prospectively for one year and found that late menarche, menstrual irregularities, decreased calf girth, low bone density, reduced lower limb lean mass, lower dietary fat and a discrepancy in leg length were significant risk factors for stress fractures in women. A later age of menarche and calf girth were the best independent predictors of stress fractures in the female athlete. Gender was not a risk factor for stress fractures in this study. In men, there were no significant identifiable risk factors for stress fractures. Excessive exercise in the male endurance athlete has been associated with increased bone turnover (Hetland et al., 1993) and, in some studies, to a decrease in sex hormones (Wheeler et al., 1984) which may contribute to bone loss in this population. Bennell and colleagues (1996) however, did not find BMD to be predictive of stress fractures in the male runners.

Nattiv and colleagues (2000) assessed track and field athletes of varying ethnicity (primarily Caucasians and African Americans) over four years and found that low bone density and prior history of stress fractures were significant risk factors for bone stress injury in both genders. Lower body fat was a significant risk factor in females, while lower body weight and lower hip eccentric strength were additional risk factors in male track and field athletes. Menstrual history and disordered eating were associated with stress injuries, but this did not reach statistical significance, although these factors were significantly related to bone density.

Most studies assessing stress fractures in athletes have been in Caucasians. There have been two studies in runners which compare stress fracture rates between races (Barrow & Saha, 1988; Nattiv et al., 2000). Barrow and Saha (1988) found that the prevalence of stress fractures in Caucasians was twice that of African Americans. However, the study was retrospective, only assessing female runners, and the number of African Americans were small. The author found that there was a trend for Caucasian track and field athletes to have a greater incidence of bone stress injury, but this did not reach statistical significance (Nattiv et al., 2000).

Few military studies have assessed individuals of African American ethnicity (Brudvig et al., 1983; Gardner et al., 1988; Milgrom et al., 1994). These studies suggest that both male and female Caucasians are at increased risk for stress fractures compared to African Americans. There is a need to assess stress injury

and stress fracture risk in athletes of varying ethnic backgrounds, especially since a large percentage of runners are African American.

A previous history of stress fractures appears to be fairly common in athletes sustaining stress injury, although not many studies in athletes have reported recurrence rates. In Bennell and colleagues' study (1996), 60% of male and female track and field athletes reported a previous history of stress fracture. A high incidence of prior stress fractures of 64% in those athletes with a stress injury was also found in our study (Nattiv et al., 2000). In other studies of runners, previous running injury (Macera et al., 1989; Marti, 1988; Walter et al., 1989), and higher mileage per week (Blair et al., 1987; Bovens et al., 1989; Jacobs & Berson, 1986; Koplan et al., 1982; Lysholm & Wiklander, 1987; Macera et al., 1989; Marti, 1988; Walter et al., 1989) have been associated with an increased risk of injury in runners. In Bennell's study, training was not found to be related to stress fracture incidence (Bennell et al., 1996), but data regarding training factors was assessed retrospectively. There is a need for prospective longitudinal studies to assess stress fracture risk in young athletes as well as effects of various exercise regimes on bone.

Early detection of risk factors for stress injury and low bone mass may help prevent these potentially devastating injuries, and may reduce the significant morbidity and, in the case of osteoporosis, even potential future mortality.

## **Preventive Strategies for Optimizing Bone Density and Reducing Stress Fractures**

### **Maximizing Peak Bone Mass**

Maximizing peak bone mass has been an important preventive strategy for decreasing future risk for significant osteopenia and osteoporotic fractures. Early detection and prevention of risk factors which are associated with stress fractures and low bone density should be a priority in the young athlete. Genetic factors are hypothesized to account for 60-80% of peak skeletal mass (Slemenda et al., 1991) with the remaining 20-40% influenced by environmental factors, including calcium nutrition, hormonal status and weight bearing exercise.

### **Optimizing Calcium Nutrition**

One of the most important preventive interventions contributing to optimal bone health in the children and young adults is adequate calcium nutrition. Bone mineral status of children ages 2 to 16 years old has been found to be positively associated with dietary calcium intake (Chan, 1991). Calcium supplementation was found to enhance the rate of increase of BMD in prepubertal white children in a 3-year double-blind, placebo-controlled trial (Johnston, 1992). In another study, Lloyd and colleagues (1993) noted an increase in BMD of the lumbar spine and total body in adolescent Caucasian girls with calcium supplementation.

In the athletic population, studies on the effects of dietary calcium have shown that a positive linear correlation exists with dietary calcium and spinal trabecular bone density in amenorrheic and eumenorrheic elite female athletes (ballet dancers, runners and rowers) in their 20's (Wolman et al., 1992). Myburgh and colleagues (1990) found that athletes with stress fractures had lower calcium intakes compared to athletes without stress fractures. It may be that diet and physical activity interact to influence BMD. The author suggests following the National Institutes of Health (NIH) guidelines for calcium needs by age group (NIH, 1994), with the added recommendation of 1500 mg/day in the female athlete with

oligomenorrhea or amenorrhea. Vitamin D in the range of 400 IU/day is also beneficial in conjunction with calcium, as the skeletal effects of vitamin D include stimulation of osteoblasts and increasing calcium absorption, as well as other effects that contribute to bone health.

### **Avoidance of Disordered Eating, Inadequate Calories and Low Body Weight**

Disordered eating patterns and energy deficit states can lead to profound bone loss and place athletes at risk for stress injury to bone (ACSM Position Stand on the Female Athlete Triad, 1997). Retrospective analysis assessing risk factors for stress fractures have demonstrated that young female track and field athletes with a history of stress fractures scored higher on the EAT-40 test and were more likely to engage in restrictive eating patterns and dieting (Bennell et al., 1995). Young female ballet dancers with a history of stress fractures were similarly found to have a higher incidence of disordered eating behaviors (Frusztajer et al., 1990).

Nelson and colleagues have found that amenorrheic athletes with lower lumbar spine BMD had lower daily energy intakes. Inadequate caloric intake relative to energy expenditure through exercise may result in a negative energy balance, which has been implicated as the most likely mechanism leading to athletic amenorrhea (Loucks et al., 1998) and predisposing athletes to the potential detrimental effects on bone.

The majority of studies linking disordered eating to low BMD have been in young female athletes who are also underweight and with significant energy deficits. The most profound effects on bone are found in young women with anorexia nervosa (Rigotti et al., 1984), with nearly 75% of adolescent anorectic girls having BMD of more than 2 standard deviations below normal value for age (Bachrach et al., 1990). A combination of bone loss as well as not attaining their peak bone mass probably contribute to these deficits.

### **Avoidance of a Negative Energy Balance**

The most likely mechanism involved in athletic amenorrhea that explains the suppression of reproductive function is a state of negative energy balance or low "energy availability" (Loucks et al., 1998). Loucks and colleagues (1998) have demonstrated that luteinizing hormone pulsatility depends upon carbohydrate availability. She has found that the supply of glucose may be less compromised by exercise than by dietary restriction or by a combination of dietary restriction and exercise. Prevention and treatment of energy deficits if existing in the young athlete, are the first steps to prevention of stress injury and to avoidance of bone loss.

### **Maintenance of Hormonal Balance**

There is a paucity of information regarding the long-term effects of delayed menarche on bone. There are a few studies however, that implicate osteopenia (Ulrich et al., 1995), stress fractures (Bennell et al., 1996), and scoliosis (Warren et al., 1986) as potential complications of delayed menarche.

It was not until the mid 1980's that scientists linked a decrease in BMD of the lumbar spine in premenopausal athletes with a history of athletic amenorrhea (Drinkwater et al., 1984; Lindberg et al., 1984; Marcus et al., 1985). It is now known that this bone loss in the oligomenorrheic and amenorrheic athlete is not only limited to the lumbar spine, but is more generalized (Rencken et al., 1996).

Rencken and colleagues (1996) in their study assessing the differences in BMD between amenorrheic and eumenorrheic athletes, found that amenorrheic athletes had significantly lower BMD in the lumbar spine, femoral neck, trochanter, Ward's triangle, intertrochanteric region, femoral shaft and tibia. Body weight combined with months of amenorrhea and age of menarche predicted BMD of the lumbar spine for amenorrheic athletes. Duration of amenorrhea and body weight predicted BMD at the femoral neck, trochanter, intertrochanteric region and tibia. Weight alone predicted BMD at the femoral shaft and tibia. In eumenorrheic athletes, age plus weight predicted lumbar BMD. Age of menarche predicted lumbar BMD in both the amenorrheic and eumenorrheic athletes.

### **Weight-bearing Exercise and Resistance Training**

The optimal "dose-response" relationship of various mechanical loading regimes on bone mineral density (BMD) has not as yet been determined. The biomechanical loading forces on a specific skeletal site is a function of the duration, intensity, frequency and mode of force placed on the bone. Recent research suggests that exercise with higher loads at specific sites provides a greater osteogenic stimulus than lower loads that are generally distributed (Robinson et al., 1993). A significant relationship between BMD and several measurements of muscle strength have been noted in female collegiate athletes (Emslander et al., 1998) and non-competitive young women (Snow-Harter et al., 1990). In fact, Snow-Harter and colleagues (1990) found muscle strength to be a significant independent predictor of BMD, accounting for 15-20% of the variance in BMD of young women. Witzke and Snow (1999) found that in adolescent girls, lean body mass and leg power best predicted bone mineral content and BMD of the whole body, lumbar spine, femoral shaft and hip. This study suggests an important role for muscle mass development during growth to maximize peak bone density. In a more recent study, Witzke and Snow (2000) found that plyometric jump training improved bone mass in adolescent girls. This study has important implications in the effort to maximize peak bone density in our young athletes. Data is still lacking however, on the optimal exercise program that would most effectively stimulate bone mass accretion in younger individuals who may not have reached their peak bone mass.

### **Role of Medications**

There are a lack of prospective studies that demonstrate an increase in BMD with oral contraceptive pill (OCP) use in athletic women with hypoestrogenic amenorrhea or oligomenorrhea. Similarly, there are a lack of studies that demonstrate bone gain or prevention of fractures in the young oligomenorrheic or amenorrheic athlete using postmenopausal doses of premarin and provera.

There has been conflicting data regarding whether or not there is a protective effect of OCP use on stress fracture development in the female athlete. Most of the studies assessing the effect of OCP use on stress fractures have been in runners and have been cross-sectional or retrospective. In the prospective studies of track and field athletes by Bennell et al. (1996) and the author (Nattiv et al., 2000), there was no significant effect of OCP use on stress fractures. It is possible that the influence of OCP on stress fracture reduction differs depending upon the athlete's menstrual history and current menstrual status. More research is needed in this area.

Drinkwater and colleagues (1993) have found that use of nasal calcitonin in



young amenorrheic athletes significantly increased BMD in the spine and proximal femur. No data exists on prevention of stress fractures in this population. More research is needed using nasal calcitonin in this population.

Other medications approved for the treatment of postmenopausal osteoporosis include the bisphosphonates. Although some physicians have been using these medications in the osteopenic young athlete, they are not recommended in women of childbearing age. The half life for some of the bisphosphonates, such as alendronate are very long (several years) and use in the young athlete who may bear children is not recommended, since teratogenicity has been noted in animals. It can not be assumed that these drugs will be effective or safe in the young athlete.

The selective estrogen receptor modulators (SERMS), approved for the prevention and treatment of postmenopausal osteoporosis should not be used for osteopenia or osteoporosis treatment in the young premenopausal athlete. Studies have shown some SERMS to cause bone loss in premenopausal women.

Medications which assist with bone formation are promising for future use in the young osteopenic or osteoporotic athlete. Although none of these medications have been approved as yet, parathyroid hormone (PTH) has been studied in conjunction with estrogen, and looks promising.

There are a lack of studies assessing interventions in the male athlete that may lead to an increase in bone density or stress fracture reduction. Although some studies have demonstrated reduced serum testosterone levels in male distance runners (Wheeler et al., 1984), these findings have not been consistent. It is possible that the hormonal and nutritional environment of male endurance athletes parallels that found in the female athlete, including an energy deficit state, although the effects of an energy deficit on bone health in the male athlete are unknown.

### Summary

Stress injury to bone exists on a continuum, involving mechanical, hormonal and nutritional factors. Advances in imaging have helped us appreciate the progression of bone stress injury and fatigue, and have helped us to identify and diagnose these injuries earlier, prior to the development of a frank cortical fracture. The epidemiology of stress fracture incidence and risk factors in the athletic population is still in its infancy. There have been a few prospective studies in track and field athletes which have found an incidence of bone stress injury between 11% and 21%. Risk factors for stress fractures vary in studies and include delayed menarche, menstrual dysfunction, less use of OCP, decreased calf girth, low bone mineral density, low calcium intake, low dietary fat, a discrepancy in leg length, less lower extremity lean mass, low body weight, low body fat, decreased calf girth, decreased hip eccentric strength, increased training intensity and a prior history of stress fractures. Gender differences in stress fracture incidence are less apparent in athlete studies than in the military. The tibia is the most common skeletal site fractured in both sexes. Different patterns of bone stress injury are seen in the female athlete, as well as athletes in different track events.

Prevention of stress injury to bone involves maximizing peak bone mass in the pediatric, adolescent and young adult age groups. Maintaining adequate calcium nutrition, caloric intake as well as hormonal and energy balance are important

preventive measures, as are ensuring appropriate amounts of weight bearing exercise for optimizing skeletal integrity and preventing fractures.

There are no studies that demonstrate a treatment that will consistently increase bone density or prevent fractures in the female athlete with disordered eating or menstrual dysfunction with low bone density or in the male athlete. More research is needed to determine additional factors that may be contributing to bone loss, as well as to assess other preventive and treatment options leading to improvements in bone density, skeletal integrity and fracture reduction.

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