Bone Health in Endurance Athletes: Runners, Cyclists, and Swimmers

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Abstract

Weight-bearing exercise has been recognized widely to be beneficial for long-term bone health. However inherent differences in bone-loading characteristics and energy expenditure during participation in endurance sports place many endurance athletes at a relative disadvantage with regard to bone health compared with other athletes. Adolescents and adults who participate in endurance sports, such as running, and non-weight-bearing sports, such as biking and swimming, often have lower bone mineral density (BMD) than athletes participating in ball and power sports, and sometimes their BMD is lower than their inactive peers. Low BMD increases the risk of stress and fragility fractures, both while an athlete is actively competing and later in life. This article reviews the variable effects of distance running, cycling, swimming, and triathlons on bone health; the evaluation of stress and fragility fractures; and the diagnosis, management, and prevention of low BMD in endurance athletes.

Introduction

Weight-bearing exercise has been recognized widely to be beneficial for long-term bone health (9,43). Active children and adults have been shown to have higher bone mineral density (BMD) and reduced long-term fracture risk than their inactive counterparts (43,55). Bone acquisition during childhood and adolescents is a strong determinant of peak bone mass and the eventual risk of osteoporosis and fragility fracture later in life (6). However adolescents and adults who participate in endurance sports, such as running, and non-weight-bearing sports, such as biking and swimming, often have lower BMD than athletes participating in ball and power sports, and sometimes their BMD is lower than their inactive peers (30,32,57). Low BMD increases the risk of stress and fragility fractures, both while an athlete is competing actively and later in life (44). This article will review the variable effects of endurance sports on bone health; the evaluation of stress and fragility fractures; and the diagnosis, management, and prevention of low BMD in endurance athletes.

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1537-890x/1106/328–334
Current Sports Medicine Reports

Definition of Low BMD in Athletes

The World Health Organization has defined osteoporosis in postmenopausal women as a T-score equal to or less than −2.5 SDs below peak young adult BMD as measured by dual x-ray absorptiometry (DXA) and osteopenia as a T-score between −1 and −2.5 SDs. When assessing BMD in premenopausal women and men under 50, the International Society of Clinical Densitometry recommends the use of Z-scores, which compare bone density with age- and sex-matched controls, rather than T-scores (76). They define a Z-score of −2 or below as "low bone density below the expected range for age" and recommend the diagnosis of osteoporosis be made only when additional risk factors for fracture or secondary causes are identified. In light of the fact that athletes tend to have 10% to 15% higher BMD than nonathletes, the American College of Sports Medicine defines the term "low BMD" as a history of nutritional deficiencies, hypoestrogenism, stress fractures, and/or other secondary clinical risk factors for fracture together with a Z-score between −1.0 and −2.0. The American College of Sports Medicine defines "osteoporosis" as secondary clinical risk factors for fracture with BMD Z-scores less than −2.0 (58).

Effect of Exercise on Bone Health

Wolfe's law states that bone, in a healthy subject, will respond over time to the stress it is placed under (75). In response to the strain or deformation caused by forces from muscle contraction and/or impact, osteoclasts and osteoblasts are stimulated to remodel existing bone and lay down new bone matrix in a localized and vector-appropriate fashion to improve the bone's ability to withstand future forces of similar magnitude and direction (12,63).

Studies in mice have demonstrated that high-impact, irregular, multiplanar loads and slower load-relax cycles are more effective at increasing bone mass than the lower-impact, repetitive, higher-frequency, uniplanar loads typically produced in endurance sports (12,63). Recovery periods of as little as

14 s between load cycles have been shown to improve bone formation compared with more frequent load cycling. In mice trained to jump between 5 and 100 jumps per day, there was a significant increase in bone mass with as little as five jumps per day over controls, but no difference was noted in bone mass between mice that jumped 10 versus 100 times per day (69).

Extrapolating what is known about the effects of various load characteristics on bone formation in animal studies, one can appreciate a potential disadvantage for athletes of endurance sports versus ball or power sports. In the process of jumping, cutting, starting, and stopping, high-impact forces in multiple planes are transmitted through the bones of a basketball player or gymnast at varying intervals, with brief periods of rest in between. However endurance training and racing is inherently repetitious. A marathon runner will generate repetitive forces of lower peak intensity, primarily in a single plane at a constant rate of about 80 to 110 strides per minute with little variation over the course of a long training session or race (12,63,64).

Effect of Energy Availability on Bone Health

Energy Availability

Energy availability (EA) is defined as the amount of energy consumed minus the amount of energy used during exercise divided by fat-free mass (58). Thus EA is the pool of energy that is available to fuel a wide array of normal physiological functions. Reproductive function, growth, and maturation, including bone formation, are among some of the physiologic functions supported by EA. Bone strength is affected by low EA both directly and indirectly. Direct mechanisms are through the suppression of tri-iodothyronine (T3), insulin-like growth factor 1 (IGF-1), and insulin resulting in decreased bone formation. The indirect mechanism is via reduction of reproductive hormones leading to increased bone resportion (22).

Low EA can be caused by low dietary intake, excessive caloric expenditure during exercise, or a combination of both. Studies by Loucks *et al.* have shown that 45 $kcal \cdot kg^{-1}$ Fat-free mass $(FFM) \cdot d^{-1}$ is the amount of EA needed to achieve energy balance in young women. EA levels less than approximately 30 $kcal \cdot kg^{-1}$ $(FFM) \cdot d^{-1}$ in women cause suppression of luteinizing hormone pulsatility (49). This suppression can be reversed by increasing dietary intake without decreasing exercise expenditure. Exercise is not the culprit leading to menstrual dysfunction in athletes (49). A genetic threshold may be at play causing some female athletes to be more susceptible to functional hypothalamic amenorrhea (FHA) (14).

Female Athlete Triad

The female athlete triad (Triad) encompasses a spectrum of interrelated conditions: EA, menstrual health, and bone health. Low EA, either intentional or unintentional, including disordered eating (DE) and eating disorders (EDs), represents a central feature of the Triad. Menstrual dysfunctions ranging from anovulatory cycles, luteal phase defects, oligomenorrhea, and amenorrhea have important health consequences including, but not limited to, a negative effect on bone health. BMD is 10% lower in amenorrheic athletes compared with eumenorrheic athletes, and amenorrheic athletes lose 2% to 3% of their bone mass per year if untreated. Resumption of menses by increased EA is associated with a 2% to 3% increase a year in lumbar BMD (19). Even though BMD improves with refeeding and resumption of menses, an athlete may not fully recover the bone that has been compromised (22). It also is important to note that low BMD has been associated with DE even in eumenorrheic runners (18). Athletes with oligo- or amenorrhea have increased risk of stress fractures (two to four times) compared with their eumenorrheic counterparts (22). Endurance athletes presenting with one component of the Triad should be evaluated for the other two components. Female distance runners have been identified as a high-risk group for suffering one or more Triad-related diagnoses. Pollock *et al.* (61) reported that DE, menstrual disturbance, and low BMD coexisted in 15.9% of 44 elite endurance runners.

Although data are very limited, male endurance athletes also may be at risk of low EA, either intentional or unintentional, and may experience similar suppression of reproductive hormones and growth factors important for bone health (35). Low testosterone levels, which are associated with low EA and low BMD, have been reported in male distance runners and cyclists (19,34).

BMD in Endurance Athletes

Runners

Most (23,27,69,72–74) but not all (8) cross-sectional studies on BMD and other markers of bone strength have shown an advantage for runners compared with inactive controls. Duncan *et al.* (23) compared 13- with 18-year-old female runners to inactive controls and to other endurance athletes and found the runners to have the highest average BMD (Figure). However, when endurance runners are compared with sprinters, gymnasts, or ball sports athletes, the BMD in endurance runners is consistently lower (3,23,29,56,59,67, 68). Hetland *et al.* (38) reported that higher average weekly mileage was correlated negatively with lumbar spine (LS) BMD. Wilks *et al.* (72) used peripheral qualitative computed tomography to assess multiple parameters of bone density, size, and geometry in the tibias of masters aged sprinters, middle distance runners, and long distance runners. They found that all bone strength indicators were higher in the sprinters, while cortical density was related inversely to the competitive distance run, with the lowest cortical density found in the long distance runners. Overall, runners have shown higher BMD and other bone strength indicators at primary sites of impact such as the calcaneus and tibia when these sites have been measured, compared with standard sites of DXA assessment such as femoral neck (FN) and LS (21,30,65).

Some cross-sectional studies in female adolescent and collegiate runners have demonstrated increased rates of low BMD. Barrack *et al.* (2,4) evaluated 93 high school cross-country runners aged 13 to 18 years and found Z-scores of -1 or less in 28% and -2 or less in 11.8%. Runners with menstrual irregularities, dietary restraint, low lean tissue mass, and five or greater seasons of running were more likely to have low BMD in this study.

One weakness of cross-sectional studies is selection bias, and this bias may be confounding the BMD studies in athletes. A thin body habitus is typical of athletes that excel in long distance running, and this is a known risk factor for low BMD.

However a prospective study by Taaffe *et al.* (67) confirmed reduced bone mineral accrual in runners over time.

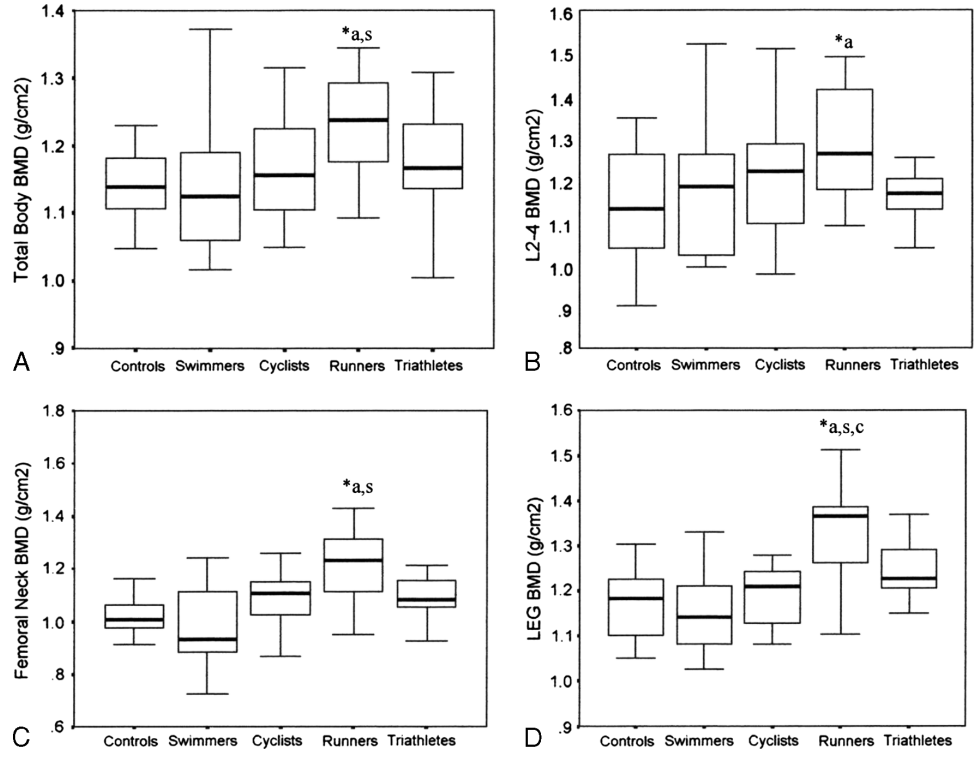

Figure: A comparison of (A) total body, (B) LS, (C) FN, and (D) leg BMD among sport groups. Data are presented as box-and-whiskers plots. The horizontal line in the box represents the group median; the upper and lower borders of the box represent the 75th and 25th percentiles, respectively; and the vertical lines represent the largest and smallest observed values that are not outliers. *a = Runners significantly greater than controls ($P < 0.05$). *s = Runners significantly greater than swimmers ($P < 0.05$.) *c = Runners significantly greater than cyclists ($P < 0.05$). BMD in adolescent female endurance athletes aged 13 to 18 years. (Reprinted with permission from Duncan CS, Blimkie CJ, Cowell CT, et al. Bone mineral density in adolescent female athletes: relationship to exercise type and muscle strength. *Med. Sci. Sports Exerc.* 2002; 34:286–94.)

They studied 26 collegiate female gymnasts, 36 runners, and 14 nonathletes, following them for an 8-month competitive season. At the end of the study period, they found a greater percentage increase in BMD among gymnasts than runners or controls, whose BMDs were unchanged over the follow-up period.

In summary, with the exception of adolescent women at risk for low EA and endocrine abnormalities, running generally has been associated with equivalent or slightly higher BMD than inactive controls, but the BMD was consistently lower than athletes of higher-impact sports. Since endurance running is a weight-bearing sport with high metabolic needs, EA may play a critical role in the bone health of long distance runners.

Cyclists

Cycling is an aerobic, low-impact, non-weight-bearing sport during which the spine is suspended evenly between the seat and the handlebars and normal weight-bearing ground reaction forces are absent. In a recent prospective study by Nichols and Rauh (60), 19 high-level masters cyclists and 18 nonathletes were followed over a 7-year period. Rates of osteopenia or osteoporosis, as classified by T-score, were high in both groups but more common in the cyclists at baseline (82% vs 50%). At the 7-year follow-up, the rates had increased to 89.5% versus 61%, respectively, and of the cyclists who were osteopenic at baseline, 31% had progressed to osteoporosis by the end of the study. Study participants who reported high-impact cross-training or weight training during the study period lost significantly less bone mass than those who did not cross-train. Nagle and Brooks (57) recently evaluated 13 of the highest quality studies on bone health in cyclists and found two prospective studies evaluating changes in BMD over time. One in male cyclists showed significant reductions in FN BMD despite taking calcium supplementation over the 12 months of follow-up. The second study, in female cyclists, showed a loss of LS BMD but no significant change at the hip over an 18-month follow-up. Four of seven cross-sectional studies showed lower LS BMD in cyclists than in runners, while the other three showed no significant difference. BMDs of cyclists were similar to that of sedentary controls in three out of four studies where they were compared directly and were lower in the fourth.

Beyond the effects of load characteristics and EA, an additional mechanism that might affect bone mineralization has been studied in cyclists. To maintain serum calcium homeostasis, calcium lost in sweat must be replaced by ingested calcium or resorption of calcium from bone. Transient elevations in parathyroid hormone (PTH) and other biomarkers for bone turnover have been noted during prolonged cycling, suggesting acute bone resorption. Limited research suggests that ingestion of 1000 mg of calcium immediately before or during cycling attenuates elevations in serum PTH and other markers for bone turnover and could play a role in the prevention of bone demineralization from this mechanism in endurance athletes (5,33).

Swimmers

Swimming is a non-weight-bearing aerobic activity that is associated with increased lean body mass and aerobic capacity but no positive effects on BMD. One prospective cohort study showed that while gymnasts had significant gains in BMD (2.3% LS and 5% FN) over an 8-month competitive season, no gain was seen in swimmers or controls (67). These differences in bone mineral accrual were noted even though the gymnasts were found to have greater menstrual dysfunction and body dissatisfaction than the swimmers or the controls. Cross-sectional studies in male and female adolescent and collegiate swimmers have uniformly shown no benefit in BMD over inactive controls and lower BMD when compared with other athletes (13,20,25,39).

Triathletes

Very limited research has been done on the topic of bone health in triathletes. In 2007, Hoch *et al.* (40) studied 15 female club triathletes with an average age of 36 years and found that despite the fact that 60% were in calorie deficit and 40% had amenorrhea, the average Z-score for the group was normal at 0.8. Two prospective studies have shown no adverse affect on BMD or bone turnover markers during a competitive season, and Z-scores were comparable with other athletes tested (51,52). Despite the fact that triathletes spend a lot of time swimming and biking, it is possible that the high amount of cross-training inherent in their sport may be healthier for bone than participating in a single discipline.

Consequences of Poor Bone Health

Stress Fractures

Estimates of stress fracture incidence in athletes has ranged from 1% to 2.6% in National Collegiate Athletic Association athletes to up to 15% in runners presenting to a sports medicine clinic (1,7,10,66). The relative risk in women is 1.5 to 3.5 compared with their male counterparts and is more than three times more common in oligo- or amenorrheic athletes compared with eumenorrheic athletes (22,49).

Stress fractures are more common in runners than other endurance athletes due to the repetitive impact forces inherent in the sport. In a 10-year retrospective review of division I college athletes, distance runners suffered the most bone stress injuries of all the male and female athletes. The most common stress fracture sites in runners are tibia, metatarsals, navicular, fibula, and calcaneous (1) but stress fractures also are seen in sesamoids, FN, distal femur, ischium, and sacrum (36,31).

Stress fractures occur when the normal remodeling and reparative properties are overwhelmed by increased load and strain leading to osteoclastic activity (bone resorption) that outpaces osteoblastic activity (new bone formation). For an athlete, this is usually in the setting of increased training frequency, intensity, or duration. The process initially leads to trabecular microfracture and resulting marrow edema, which can be seen on magnetic resonance imaging and commonly is termed *stress reaction*. Continued repetitive stress will eventually lead to fatigue of the weakened cortex and result in a stress fracture (31).

Both intrinsic and extrinsic factors can place a bone at risk for stress injury. The most important extrinsic factor is a rapid increase in activity or training intensity. For runners, this includes a sudden change in frequency, mileage, pace, or terrain. Lack of rest after long runs, running shoes greater than 6 months old or with more than 300 to 500 miles, and running on hard or cambered surfaces are additional extrinsic factors that have been implicated in the development of a bony stress injury (36). Aspects of gait that lead to higher load rates are likely to put a runner at risk as well, including excessive hip adduction, rear foot eversion, and stride length (24,53,77).

Intrinsic factors are related to bone health and muscle mass and include low body mass index (BMI), low body fat percentage, low muscle mass or endurance, oligo- or amenorrhea, and low BMD. In a recent study by Burgi et al. (11), there were twice as many tibial stress fractures in women with serum 25(OH) vitamin D concentrations of less than 20 $ng \cdot mL^{-1}$ compared with those with concentrations of 40 $ng \cdot mL^{-1}$ or greater.

Though any stress fracture may be a sign of poor bone health, some clinical factors should raise suspicion and prompt a bone health evaluation (Table 1) (36).

Osteoporosis

Not all endurance athletes with poor bone health will experience a stress fracture. Due to their lack of weight-bearing impact, swimmers and bikers are at low risk for stress fracture despite their higher risk for low BMD. In these athletes, low bone mineral accrual and/or increased bone resorption can occur silently over time, leading to osteoporosis without developing a stress fracture during their competitive years. Because 60% of osteoporosis risk is related to the amount of bone mass accrued by early adulthood, a high index of suspicion needs to be maintained in order to identify and screen athletes at risk for low BMD (6).

Evaluation and Treatment of Low BMD

Evaluation

When low BMD is found in an athlete, a clinical history, physical examination, and a directed laboratory evaluation to uncover secondary causes of low BMD (Table 2) should be undertaken (28). Further discussion of secondary causes of poor bone health is beyond the scope of this review, but at a minimum, a screening serum vitamin D and calcium levels should be ordered. Other laboratory testing could include complete blood count with differential, metabolic panel, thyroid-stimulating hormone, PTH, bone-specific alkaline

Table 1.
Factors prompting BMD evaluation in athletes with stress fracture.

Low BMI (<18.5 $kg \cdot m^{-2}$)
Recurrent stress fractures
Oligo- or amenorrhea ≥6 months
A history of an ED, DE, or low EA
Chronic medical conditions associated with bone loss
Medications associated with adverse affects on bone health
Cancellous versus cortical bone fractures, particularly proximal femur, tibial plateau, and calcaneus
Cyclists, swimmers
No recent change in activity level or training intensity

Table 2.
Secondary causes and contributors to bone health compromise.

Endocrine/Metabolic	*Inflammatory*
Low EA	Juvenile inflammatory arthritis
ED	Rheumatoid arthritis
DE	Other chronic inflammatory conditions
Excessive energy expenditure	
Low vitamin D	*Medications*
Hypogonadism	Glucocorticoids
Hyperthyroidism	PPIs
Hyperparathyroidism	SSRIs
Cushing's syndrome	Anticonvulsants
Diabetes type 1	Progestin-only contraceptives
Chronic kidney disease	GnRH inhibitors
Familial hypercalciuria	High dose thyroxine
	Cyclosporine
Gastrointestinal	Chemo and radiation therapy
Inflammatory bowel disease	
Celiac disease	*Other*
Status postgastric bypass	Connective tissue disease
Cystic fibrosis	HIV/HAART
Chronic liver disease	Thalassemia
Other malabsorption syndromes	Hemochromatosis
	Systemic mastocytosis
	Alcoholism
	Tobacco use

PPI, proton pump inhibitors; SSRI, selective serotonin reuptake inhibitor; GnRH, gonadotropin-releasing hormone; HAART, highly active antiretroviral therapy.

phosphatase, 24-h urine for calcium, screening for cortisol excess, celiac sprue antibodies, and markers of bone formation and resorption such as serum osteocalcin and urine N-telopeptide. Evaluation of reproductive hormone levels, including testosterone levels in male athletes, can uncover an endocrine disease or indicate low EA (47).

Treatment: EA

Increasing EA to avoid suppression of reproductive and other important hormones and growth factors is critical to improving bone health that is already in jeopardy. Amenorrheic athletes have been reported to increase their BMD more by resumption of menses and weight gain compared with other strategies (70). It has been suggested that endurance athletes, either independently or with the expert guidance of a nutritionist, monitor their energy intake and expenditure daily, along with knowing their fat-free mass in order to maintain EA in the range of 45 kcal·kg^{-1} FFM·d^{-1} or above. It is important to remember that dietary caloric requirements will be underestimated in the face of exceedingly low fat-free mass status, and this should be accounted for when striving to improve EA into a range that promotes healthy bones (50).

Horvath *et al.* (41,42) has shown that the body does not appropriately match appetite to energy requirements in male and female runners, resulting in many endurance athletes who are chronically undernourished. This mismatch is more pronounced when consuming high-carbohydrate diets. EA was improved by approximately 30% by increasing fat intake from 17% to 31%, which resulted in improved running performance without increasing body weight or body fat.

Endurance athletes attempting to improve their EA also may benefit from working with a nutritionist, preferably one with knowledge of endurance sports, to help them determine ways to find the time to fit in all of the calories that they need to consume. Training for endurance sports coupled with going to school or work can occupy a large portion of the day, leaving little time to eat. Simple suggestions such as frequent snaking along with carrying portable nutritious snacks to eat when time allows can make a positive impact on EA status.

Low EA may be a simple matter of "underfueling" due to a lack of understanding the high caloric needs associated with endurance sport training or may be associated with pathological weight management behaviors, disturbed body image, and at times a full-blown ED. In these circumstances, improving EA will require addressing the underlying psychological issues in conjunction with a psychologist and/or psychiatrist in addition to a nutritionist and a primary care physician (58).

Another strategy to increased EA is to reduce caloric expenditure by decreasing the amount of exercise. This can be accomplished by adding an additional rest day per week or substituting a running day with an alternative form of exercise that is less calorically demanding, such as weight lifting. Shortening workouts overall is another way to reduce caloric output (22).

Adequate amounts of calcium, vitamin D, and vitamin K are important for improving bone health. The optimal calcium intake for an athlete with bone loss is unknown, and age, gender, and sport may affect the amount required. The Female Athlete Triad Position Stand recommends calcium 1000 to 1300 mg·d^{-1}, vitamin D 400 to 800 IU·d^{-1}, and vitamin K 60 to 90 μg·d^{-1} (58). Vitamin K, particularly K_2, is a cofactor for enzymes critical to bone formation.

Treatment: Medications

Currently there are no medications approved by the Food and Drug Administration (FDA) to treat bone loss in premenopausal women and men younger than 50 years old. Thus young athletes with significant bone loss have limited pharmacologic options.

Oral contraceptive pills (OCPs) are frequently prescribed for athletes with FHA despite not being the recommended first line treatment (58). While monthly withdrawal bleeds may result from taking OCPs, it is important to realize that this is not equivalent to restoring natural menstrual cycles through improved EA. OCPs do not treat the underlying hormone and growth factor suppression seen with FHA. The studies on the effect of OCPs and bone health remain mixed and are frequently confounded by weight gain, which is known to improve BMD. OCPs may help prevent further loss, but given that they suppress bone formation markers in addition to bone resportion markers, they may be harmful to the bone overall (18,37,70).

Bisphosphonates, which are routinely used to treat postmenopausal osteoporosis, are not approved by the FDA for treatment of bone loss in young athletes. Evidence is lacking regarding positive bone health benefits in this population. Another concern surrounding the use of bisphosphonates in women of childbearing age is the potential teratogenic affect, since almost all of these medications have long half lives and are bound to the bone for years (58).

Leptin, a hormone produced predominately by fat cells, may play a future role in the treatment of low BMD associated with FHA in athletes. Leptin stimulates osteoblast development and proliferation along with decreasing the genesis of osteoclasts. Leptin plays an important role in energy balance, and low leptin levels are seen in conjunction with low fat mass, energy deficiency, and increased physical activity (16,29). A randomized, placebo-controlled, 36-wk trial of recombinant human leptin in 20 (13 completed the study) women with hypothalamic amenorrhea found that 7/10 women treated with leptin resumed menstruation compared with 2/9 in the placebo arm. The treatment group showed a statistically significant increase in markers of bone formation and stabilization of bone resorption, although no differences were found in BMD at the completion of the study. The authors surmise that 36 wk was not a long enough time to find changes in BMD (15).

Treatment: Exercise Modification

Endurance athletes with low BMD may be counseled to modify their exercise to include weight training and/or cutting and jumping sports. Please note that in athletes with low EA, these alternative activities should not just be added to their current training program but rather substituted for some of the high energy utilizing endurance exercise. This training modification suggestion has not been studied specifically as a treatment intervention in athletes with low BMD but appears to be a logical extension of the current scientific literature. Future research is needed to support this recommendation.

Prevention

Recommendations for the prevention of poor bone health in endurance athletes are similar to those outlined for the treatment above and include optimizing loading forces on bone with cross-training as well as a diet with sufficient calories to maintain adequate EA (59). Women of childbearing age should try to maintain regular menses. Age-appropriate intake of calcium and vitamin D are recommended. Supplements can be used if dietary intake from food sources is inadequate to achieve those amounts. Two studies in female military recruits showed reductions in stress fractures with increasing amounts of calcium and vitamin D, either as a supplement or through consumption of dairy products (45,46). There is limited evidence to suggest that cyclists ingest 500 to 1000 mg of calcium immediately before or during a ride to prevent calcium resorption from bone that may occur in the setting of calcium loss in sweat (33).

Summary

Current research suggests that owing to differences in loading forces and energy expenditure, bone health in distance runners, cyclists, and swimmers tends to be inferior to that seen in athletes of other sports. The consequences of reduced bone accrual in young athletes and maintenance of bone mass in masters athletes include an increased rate of stress fracture and risk for fragility fractures later in life. Clinicians need to maintain a high index of suspicion for poor bone health in endurance athletes, especially in those with a history suggestive of low EA, or a history of bone stress injury. Adequate calcium, vitamin D, and caloric intake, as well as regular high-impact cross-training, are recommended for all endurance athletes to maintain good bone health.

The authors declare no conflict of interest and do not have any financial disclosures.

References

1. Arendt E, Agel J, Heikes C, Griffiths H. Stress injuries to bone in college athletes: a retrospective review of experience at a single institution. *Am. J. Sports Med.* 2003; 31:959–68.
2. Barrack MT, Rauh MJ, Barkai HS, Nichols JF. Dietary restraint and low bone mass in female adolescent endurance runners. *Am. J. Clin. Nutr.* 2008; 87:36–43.
3. Barrack MT, Rauh MJ, Nichols JF. Cross-sectional evidence of suppressed bone mineral accrual among female adolescent runners. *J. Bone Miner. Res.* 2010; 25:1850–7.
4. Barrack MT, Rauh MJ, Nichols JF. Prevalence of and traits associated with low BMD among female adolescent runners. *Med. Sci. Sports Exerc.* 2008; 40:2015–21.
5. Barry DW, Hansen KC, van Pelt RE, *et al*. Acute calcium ingestion attenuates exercise-induced disruption of calcium homeostasis. *Med. Sci. Sports Exerc.* 2011; 43:617–23.
6. Baxter-Jones AD, Faulkner RA, Forwood MR, *et al*. Bone mineral accrual from 8 to 30 years of age: an estimation of peak bone mass. *J. Bone Miner. Res.* 2011; 26:1729–39.
7. Bennell KL, Brukner PD. Epidemiology and site specificity of stress fractures. *Clin. Sports Med.* 1997; 16:179–96.
8. Bilanin JE, Blanchard MS, Russek-Cohen E. Lower vertebral bone density in male long distance runners. *Med. Sci. Sports Exerc.* 1989; 21:66–70.
9. Boreham CA, McKay HA. Physical activity in childhood and bone health. *Br. J. Sports Med.* 2011; 45:877–9.
10. Brubaker CE, James SL. Injuries to runners. *J. Sports Med.* 1974; 2:189–98.
11. Burgi AA, Gorham ED, Garland CF, *et al*. High serum 25-hydroxyvitamin D is associated with a low incidence of stress fractures. *J. Bone Miner. Res.* 2011; 26:2371–7.
12. Burr DB, Robling AG, Turner CH. Effects of biomechanical stress on bones in animals. *Bone*. 2002; 30:781–6.
13. Carbuhn AF, Fernandez TE, Bragg AF, *et al*. Sport and training influence bone and body composition in women collegiate athletes. *J. Strength Cond. Res.* 2010; 24:1710–7.
14. Caronia LM, Martin C, Welt CK, *et al*. A genetic basis for functional hypothalamic amenorrhea. *N. Engl. J. Med.* 2011; 364:215–25.
15. Chou SH, Chamberland JP, Liu X, *et al*. Leptin is an effective treatment for hypothalamic amenorrhea. *Proc. Natl. Acad. Sci. U. S. A.* 2011; 108:6585–90.
16. Christo K, Cord J, Mendes N, *et al*. Acylated ghrelin and leptin in adolescent athletes with amenorrhea, eumenorrheic athletes and controls: a cross-sectional study. *Clin. Endocrinol. (Oxf)*. 2008; 69:628–33.
17. Christo K, Prabhakaran R, Lamparello B, *et al*. Bone metabolism in adolescent athletes with amenorrhea, athletes with eumenorrhea, and control subjects. *Pediatrics*. 2008; 121:1127–36.
18. Cobb KL, Bachrach LK, Sowers M, *et al*. The effect of oral contraceptives on bone mass and stress fractures in female runners. *Med. Sci. Sports Exerc.* 2007; 39:1464–73.
19. De Souza MJ, Arce JC, Pescatello LS, *et al*. Gonadal hormones and semen quality in male runners. A volume threshold effect of endurance training. *Int. J. Sports Med.* 1994; 15:383–91.
20. Dias Quiterio AL, Carnero EA, Baptista FM, Sardinha LB. Skeletal mass in adolescent male athletes and nonathletes: relationships with high-impact sports. *J. Strength Cond. Res.* 2011; 25:3439–47.
21. Drysdale IP, Collins AL, Walters NJ, *et al*. Potential benefits of marathon training on bone health as assessed by calcaneal broadband ultrasound attenuation. *J. Clin. Densitom.* 2007; 10:179–83.
22. Ducher G, Turner AI, Kukuljan S, *et al*. Obstacles in the optimization of bone health outcomes in the female athlete triad. *Sports Med.* 2011; 41:587–607.
23. Duncan CS, Blimkie CJ, Cowell CT, *et al*. Bone mineral density in adolescent female athletes: relationship to exercise type and muscle strength. *Med. Sci. Sports Exerc.* 2002; 34:286–94.

24. Edwards WB, Taylor D, Rudolphi TJ, *et al*. Effects of stride length and running mileage on a probabilistic stress fracture model. *Med. Sci. Sports Exerc.* 2009; 41:2177–84.
25. Emslander HC, Sinaki M, Muhs JM, *et al*. Bone mass and muscle strength in female college athletes (runners and swimmers). *Mayo Clin. Proc.* 1998; 73: 1151–60.
26. Fanning J, Larrick L, Weinstein L, *et al*. Findings from a 10-year follow-up of bone mineral density in competitive perimenopausal runners. *J. Reprod. Med.* 2007; 52:874–8.
27. Feldman S, Capozza RF, Mortarino PA, *et al*. Site and sex effects on tibia structure in distance runners and untrained people. *Med. Sci. Sports Exerc.* 2012.
28. Ferrari S, Bianchi ML, Eisman JA, *et al*, For the IOF Committee of Scientific Advisors Working Group on Osteoporosis Pathophysiology. Osteoporosis in young adults: pathophysiology, diagnosis, and management. *Osteoporos. Int.* 2012.
29. Franks PW, Farooqi IS, Luan J, *et al*. Does physical activity energy expenditure explain the between-individual variation in plasma leptin concentrations after adjusting for differences in body composition? *J. Clin. Endocrinol. Metab.* 2003; 88:3258–63.
30. Fredericson M, Chew K, Ngo J, *et al*. Regional bone mineral density in male athletes: a comparison of soccer players, runners and controls. *Br. J. Sports Med.* 2007; 41:664,8; discussion 668.
31. Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. *Top Magn. Reson. Imaging.* 2006; 17:309–25.
32. Fredericson M, Ngo J, Cobb K. Effects of ball sports on future risk of stress fracture in runners. *Clin. J. Sport Med.* 2005; 15:136–41.
33. Guillemant J, Accarie C, Peres G, Guillemant S. Acute effects of an oral calcium load on markers of bone metabolism during endurance cycling exercise in male athletes. *Calcif. Tissue Int.* 2004; 74:407–14.
34. Hackney AC, Moore AW, Brownlee KK. Testosterone and endurance exercise: development of the "exercise-hypogonadal male condition". *Acta Physiol. Hung.* 2005; 92:121–37.
35. Hackney AC. Effects of endurance exercise on the reproductive system of men: the "exercise-hypogonadal male condition". *J. Endocrinol. Invest.* 2008; 31: 932–8.
36. Harrast MA, Colonno D. Stress fractures in runners. *Clin. Sports Med.* 2010; 29:399–416.
37. Hartard M, Kleinmond C, Kirchbichler A, *et al*. Age at first oral contraceptive use as a major determinant of vertebral bone mass in female endurance athletes. *Bone.* 2004; 35:836–41.
38. Hetland ML, Haarbo J, Christiansen C. Low bone mass and high bone turnover in male long distance runners. *J. Clin. Endocrinol. Metab.* 1993; 77: 770–5.
39. Hind K, Gannon L, Whatley E, *et al*. Bone cross-sectional geometry in male runners, gymnasts, swimmers and non-athletic controls: a hip-structural analysis study. *Eur. J. Appl. Physiol.* 2012; 112:535–41.
40. Hoch AZ, Stavrakos JE, Schimke JE. Prevalence of female athlete triad characteristics in a club triathlon team. *Arch. Phys. Med. Rehabil.* 2007; 88:681–2.
41. Horvath PJ, Eagen CK, Fisher NM, *et al*. The effects of varying dietary fat on performance and metabolism in trained male and female runners. *J. Am. Coll. Nutr.* 2000; 19:52–60.
42. Horvath PJ, Eagen CK, Ryer-Calvin SD, Pendergast DR. The effects of varying dietary fat on the nutrient intake in male and female runners. *J. Am. Coll. Nutr.* 2000; 19:42–51.
43. Karlsson MK, Rosengren BE. Training and bone — from health to injury. *Scand. J. Med. Sci. Sports.* 2012.
44. Kelsey JL, Bachrach LK, Procter-Gray E, *et al*. Risk factors for stress fracture among young female cross-country runners. *Med. Sci. Sports Exerc.* 2007; 39:1457–63.
45. Lappe J, Cullen D, Haynatzki G, *et al*. Calcium and vitamin d supplementation decreases incidence of stress fractures in female navy recruits. *J. Bone Miner. Res.* 2008; 23:741–9.
46. Lappe JM, Stegman MR, Recker RR. The impact of lifestyle factors on stress fractures in female Army recruits. *Osteoporos. Int.* 2001; 12:35–42.
47. Lebrun CM. The female athlete triad: what's a doctor to do? *Curr. Sports Med. Rep.* 2007; 6:397–404.
48. Lips P, Bouillon R, van Schoor NM, *et al*. Reducing fracture risk with calcium and vitamin D. *Clin. Endocrinol. (Oxf).* 2010; 73:277–85.
49. Lloyd T, Triantafyllou SJ, Baker ER, *et al*. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med. Sci. Sports Exerc.* 1986; 18:374–9.
50. Loucks AB. Low energy availability in the marathon and other endurance sports. *Sports Med.* 2007; 37:348–52.
51. Maimoun L, Galy O, Manetta J, *et al*. Competitive season of triathlon does not alter bone metabolism and bone mineral status in male triathletes. *Int. J. Sports Med.* 2004; 25:230–4.
52. McClanahan BS, Ward KD, Vukadinovich C, *et al*. Bone mineral density in triathletes over a competitive season. *J. Sports Sci.* 2002; 20:463–9.
53. Milner CE, Hamill J, Davis IS. Distinct hip and rearfoot kinematics in female runners with a history of tibial stress fracture. *J. Orthop. Sports Phys. Ther.* 2010; 40:59–66.
54. Mirsa M, Le Clair M, Mendes N. Use of SSRIs may impact bone density in adolescent and young women with anorexia nervosa. *CNS Spectr.* 2010; 15:579–86.
55. Moayyeri A. The association between physical activity and osteoporotic fractures: a review of the evidence and implications for future research. *Ann. Epidemiol.* 2008; 18:827–35.
56. Mudd LM, Fornetti W, Pivarnik JM. Bone mineral density in collegiate female athletes: comparisons among sports. *J. Athl. Train.* 2007; 42:403–8.
57. Nagle KB, Brooks MA. A systematic review of bone health in cyclists. *Sports Health.* 2011; 3:235–43.
58. Nattiv A, Loucks AB, Manore MM, *et al*, American College of Sports Medicine. American College of Sports Medicine position stand. The female athlete triad. *Med. Sci. Sports Exerc.* 2007; 39:1867–82.
59. Nichols JF, Rauh MJ, Barrack MT, Barkai HS. Bone mineral density in female high school athletes: interactions of menstrual function and type of mechanical loading. *Bone.* 2007; 41:371–7.
60. Nichols JF, Rauh MJ. Longitudinal changes in bone mineral density in male master cyclists and nonathletes. *J. Strength Cond. Res.* 2011; 25:727–34.
61. Pollock N, Grogan C, Perry M, *et al*. Bone-mineral density and other features of the female athlete triad in elite endurance runners: a longitudinal and cross-sectional observational study. *Int. J. Sport Nutr. Exerc. Metab.* 2010; 20:418–26.
62. Popp KL, Hughes JM, Smock AJ, *et al*. Bone geometry, strength, and muscle size in runners with a history of stress fracture. *Med. Sci. Sports Exerc.* 2009; 41:2145–50.
63. Robling AG, Turner CH. Mechanical signaling for bone modeling and remodeling. *Crit. Rev. Eukaryot. Gene. Expr.* 2009; 19:319–38.
64. Rubin CT, Lanyon LE. Regulation of bone formation by applied dynamic loads. *J. Bone Joint Surg. Am.* 1984; 66:397–402.
65. Smock AJ, Hughes JM, Popp KL, *et al*. Bone volumetric density, geometry, and strength in female and male collegiate runners. *Med. Sci. Sports Exerc.* 2009; 41:2026–32.
66. Snyder RA, Koester MC, Dunn WR. Epidemiology of stress fractures. *Clin Sports Med.* 2006; 25:37,52, viii.
67. Taaffe DR, Robinson TL, Snow CM, Marcus R. High-impact exercise promotes bone gain in well-trained female athletes. *J. Bone Miner. Res.* 1997; 12:255–60.
68. Tenforde AS, Fredericson M. Influence of sports participation on bone health in the young athlete: a review of the literature. *PM R.* 2011; 3:861–7.
69. Umemura Y, Ishiko T, Yamauchi T, *et al*. Five jumps per day increase bone mass and breaking force in rats. *J. Bone Miner. Res.* 1997; 12:1480–5.
70. Vescovi JD, Jamal SA, De Souza MJ. Strategies to reverse bone loss in women with functional hypothalamic amenorrhea: a systematic review of the literature. *Osteoporos. Int.* 2008; 19:465–78.
71. Warren MP, Miller KK, Olson WH, *et al*. Effects of an oral contraceptive (norgestimate/ethinyl estradiol) on bone mineral density in women with hypothalamic amenorrhea and osteopenia: an open-label extension of a double-blind, placebo-controlled study. *Contraception.* 2005; 72:206–11.
72. Wilks DC, Winwood K, Gilliver SF, *et al*. Bone mass and geometry of the tibia and the radius of master sprinters, middle and long distance runners, race-walkers and sedentary control participants: a pQCT study. *Bone.* 2009; 45:91–7.
73. Wilks DC, Winwood K, Gilliver SF, *et al*. Age-dependency in bone mass and geometry: a pQCT study on male and female master sprinters, middle and long distance runners, race-walkers and sedentary people. *J. Musculoskelet. Neuronal Interact.* 2009; 9:236–46.
74. Wiswell RA, Hawkins SA, Dreyer HC, Jaque SV. Maintenance of BMD in older male runners is independent of changes in training volume or VO(2)peak. *J Gerontol. A Biol. Sci. Med. Sci.* 2002; 57:M203–8.
75. Wolff J. *The Law of Bone Transformation.* Berlin: A. Hirschwald; 1892.
76. Writing Group for the ISCD Position Development Conference. Diagnosis of osteoporosis in men, premenopausal women, and children. *J. Clin. Densitom.* 2004; 7:17–26.
77. Zadpoor AA, Nikooyan AA. The relationship between lower-extremity stress fractures and the ground reaction force: a systematic review. *Clin. Biomech. (Bristol, Avon).* 2011; 26:23–8.