AMERICAN COLLEGE of SPORTS MEDICINE

The Female Athlete Triad

POSITION STAND

This pronouncement was written for the American College of Sports Medicine by Aurelia Nattiv, M.D., FACSM (Chair); Anne B. Loucks, Ph.D., FACSM; Melinda M. Manore, Ph.D., R.D., FACSM; Charlotte F. Sanborn, Ph.D., FACSM; Jorunn Sundgot-Borgen, Ph.D.; and Michelle P. Warren, M.D.

SUMMARY

The female athlete triad (Triad) refers to the interrelationships among energy availability, menstrual function, and bone mineral density, which may have clinical manifestations including eating disorders, functional hypothalamic amenorrhea, and osteoporosis. With proper nutrition, these same relationships promote robust health. Athletes are distributed along a spectrum between health and disease, and those at the pathological end may not exhibit all these clinical conditions simultaneously. Energy availability is defined as dietary energy intake minus exercise energy expenditure. Low energy availability appears to be the factor that impairs reproductive and skeletal health in the Triad, and it may be inadvertent, intentional, or psychopathological. Most effects appear to occur below an energy availability of 30 kcal·kg^{-1} of fat-free mass per day. Restrictive eating behaviors practiced by girls and women in sports or physical activities that emphasize leanness are of special concern. For prevention and early intervention, education of athletes, parents, coaches, trainers, judges, and administrators is a priority. Athletes should be assessed for the Triad at the preparticipation physical and/or annual health screening exam, and whenever an athlete presents with any of the Triad's clinical conditions. Sport administrators should also consider rule changes to discourage unhealthy weight loss practices. A multidisciplinary treatment team should include a physician or other health-care professional, a registered dietitian, and, for athletes with eating disorders, a mental health practitioner. Additional valuable team members may include a certified athletic trainer, an exercise physiologist, and the athlete's coach, parents and other family members. The first aim of treatment for any Triad component is to increase energy availability by increasing energy intake and/or reducing exercise energy expenditure. Nutrition counseling and monitoring are sufficient interventions for many athletes, but eating disorders warrant psychotherapy. Athletes with eating disorders should be required to meet established criteria to continue exercising, and their training and competition may need to be modified. No pharmacological agent adequately restores bone loss or corrects metabolic abnormalities that impair health and performance in athletes with functional hypothalamic amenorrhea.

INTRODUCTION

Because the benefits of exercise far outweigh the risks, the American College of Sports Medicine (ACSM) encourages all girls and women to participate in physical activities and sports. In 1992, however, an association of disordered eating, amenorrhea, and osteoporosis seen in activities that emphasize a lean physique was recognized as the female athlete triad (Triad) (148,215). This Position Stand replaces the 1997 ACSM Position Stand (155), updates our understanding, and makes new recommendations for screening, diagnosis, prevention, and treatment of the Triad.

EVIDENCE CLASSIFICATION

This Position Stand presents clinical recommendations for guiding primary care (Table 1). We used criteria proposed by the American Academy of Family Physicians (52) for evaluating the strength of scientific evidence supporting these clinical recommendations. These criteria categorize the strength of scientific evidence as follows: A, consistent and good-quality evidence for clinical outcomes on mortality, morbidity, symptoms, cost, and quality of life; B, inconsistent or limited quality evidence for these same clinical outcomes; and C, evidence on biochemical, histological, physiological and pathophysiological outcomes, which include hormone concentrations, bone mineral density (BMD), and asymptomatic menstrual disorders such as short luteal phase and anovulation; and evidence based on case studies, consensus, usual practice, and opinion. To avoid misunderstanding, this Position Stand differentiates between two subcategories of evidence: C-1, evidence based on biochemical, histological, physiological, and pathophysiological outcomes; and C-2, evidence based on case studies, consensus, usual practice, and opinion. This Position Stand also presents evidence statements about the current state of knowledge (Table 1). Although the clinical recommendation criteria were not developed for evaluating evidence supporting statements about the current state of knowledge (52), we used these same criteria to evaluate this evidence, as well.

THREE INTERRELATED SPECTRUMS

Low energy availability (with or without eating disorders), amenorrhea, and osteoporosis, alone or in combination, pose significant health risks to physically active girls and women. The potentially irreversible consequences of these clinical conditions emphasize the critical need for prevention, early diagnosis, and treatment. Each clinical condition is now

0195-9131/07/3910-1867/0
MEDICINE & SCIENCE IN SPORTS & EXERCISE®

DOI: 10.1249/mss.0b013e318149f111

TABLE 1. Strength of evidence taxonomy.

	Grade	Reason for C
Evidence Statements		
Severe undernutrition impairs reproductive and skeletal health.	A	
DE/ED and amenorrhea occur more frequently in sports that emphasize leanness.	A	
On average, BMD is lower in amenorrheic than in eumenorrheic athletes.	C	1
Menstrual irregularities and low BMD increase stress fracture risk.	A	
In FHA, increases in BMD are more closely associated with increases in weight than with OCP/HRT administration.	C	1
Clinical Recommendations for Screening and Diagnosis		
Screening for the Triad should occur at the preparticipation exam or annual health screening exam.	C	2
Athletes with one component of the Triad should be assessed for the others.	C	2
Athletes with disordered eating should be referred to a mental health practitioner for evaluation, diagnosis and treatment.	C	2
To diagnose FHA, other causes of amenorrhea must be excluded.	B	
BMD should be assessed after a stress or low impact fracture and after a total of 6 months of amenorrhea, oligomenorrhea, or DE/ED.	C	2
Clinical Recommendations for Treatment		
Multidisciplinary treatment for the Triad disorders should include a physician (or other health-care professional), a registered dietitian, and, for athletes with DE/ED, a mental health practitioner.	C	2
The first aim of treatment is to increase energy availability by increasing energy intake and/or reducing energy expenditure. Athletes without DE/ED should be referred for nutritional counseling.	C	1
Athletes practicing restrictive eating behaviors should be counseled that increases in body weight may be necessary to increase BMD.	C	1
Treatment for DE/ED includes nutritional counseling and individual psychotherapy. Cognitive behavioral, group therapy and/or family therapy may also be used.	B	
Athletes with DE/ED who do not comply with treatment may need to be restricted from training and competition.	C	2
OCP should be considered in an athlete with FHA over age 16 if BMD is decreasing with nonpharmacological management, despite adequate nutrition and body weight.	C	2

Evidence Categories: A—Consistent, good-quality evidence on morbidity, mortality, symptom improvement, cost reduction, and quality of life. B—Inconsistent or limited quality evidence on the same outcomes. C—Other evidence: 1—Evidence on biochemical, histological, physiological and pathophysiological outcomes, which include hormone concentrations, bone mineral density, and asymptomatic menstrual disorders such as short luteal phase and anovulation; 2—case studies, consensus, usual practice, opinion.
Definitions: DE/ED—disordered eating or eating disorders; BMD—bone mineral density; FHA—functional hypothalamic amenorrhea; OCP—oral contraceptive pills; HRT—hormone replacement therapy.

understood to comprise the pathological end of a spectrum of interrelated subclinical conditions between health and disease. Figure 1 illustrates the full range of the Triad. A glossary of terms pertaining to the Triad appears in Table 2.

The goal of ACSM is for every girl and woman's physical condition to be coincident with the upper right corner of Figure 1, which represents the healthy athlete who adjusts her dietary energy intake to compensate for exercise energy expenditure. Thick arrows in this triangle indicate that energy availability promotes bone health and development indirectly by preserving eumenorrhea (Table 2) and estrogen production that restrains bone resorption, and directly by stimulating production of hormones that promote bone formation. As a result, BMD is often above average for the athlete's age.

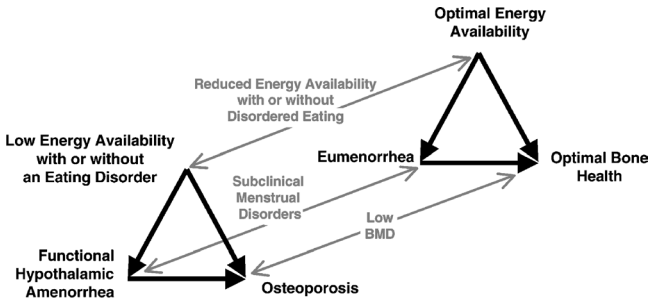

FIGURE 1—Female athlete triad. The spectrums of energy availability, menstrual function, and bone mineral density along which female athletes are distributed (narrow arrows). An athlete's condition moves along each spectrum at a different rate, in one direction or the other, according to her diet and exercise habits. Energy availability, defined as dietary energy intake minus exercise energy expenditure, affects bone mineral density both directly via metabolic hormones and indirectly via effects on menstrual function and thereby estrogen (thick arrows).

The triangle in the lower left corner of Figure 1 represents the unhealthy condition of athletes who exercise for prolonged periods without increasing dietary energy intake, who severely restrict their diet, or who have clinical eating disorders. Thick arrows in this triangle indicate that low energy availability impairs bone health and development indirectly by inducing amenorrhea and removing estrogen's restraint on bone resorption, and directly by suppressing the hormones that promote bone formation. Bone mineral accrual has slowed or reversed for so long that BMD is below average for age, and one or more stress fractures may have occurred.

The narrow arrows in Figure 1 indicate the spectrums of intermediate levels of energy availability, menstrual status, and BMD where other athletes' health status may be distributed. Moderately or recurrently reduced energy availability may induce subclinical menstrual disorders and less severely suppress estrogen and metabolic hormones, and sufficient time may not yet have passed for these athletes to fall far behind their age group in BMD. Energy availability, menstrual status, and BMD move along these spectrums in one direction or the other at different rates according to an athlete's diet and exercise habits. Energy availability can change in a day, but an effect on menstrual status may not become evident for a month or more, and an effect on BMD may not be detectable for a year.

Energy Availability

This Position Stand refers to a spectrum of energy availability from optimal energy availability to low energy availability with or without an eating disorder. Defined as dietary energy intake minus exercise energy expenditure, energy availability is the amount of dietary energy remaining

TABLE 2. Glossary of terms pertaining to the female athlete triad.

Female athlete triad	relationships among energy availability, menstrual function, and BMD that may have clinical manifestations including eating disorders, functional hypothalamic amenorrhea, and osteoporosis.
Energy availability (EA)	dietary energy intake (EI) minus exercise energy expenditure (EEE) normalized to fat-free mass (FFM), i.e., EA = (EI − EEE)/FFM, in units of kilocalories or kilojoules per kilogram of fat-free mass. For example, for a dietary energy intake of 2000 kcal·d^{-1}, an exercise energy expenditure of 600 kcal·d^{-1}, and a fat-free mass of 51 kg, EA = (2000 − 600)/51 = 27.5 kcal·kg^{-1} FFM·d^{-1}.
Exercise energy expenditure	strictly, the energy expended during exercise training in excess of the energy that would have been expended in nonexercise activity during the same time interval. Neglecting the adjustment for non-exercise activity causes EA to be underestimated by a few kcal·kg^{-1} FFM·d^{-1}, which is a negligible error for most purposes.
Disordered eating	various abnormal eating behaviors, including restrictive eating, fasting, frequently skipped meals, diet pills, laxatives, diuretics, enemas, overeating, binge-eating and then purging (vomiting).
Eating disorder	a clinical mental disorder defined by DSM-IV (8) and characterized by abnormal eating behaviors, an irrational fear of gaining weight, and false beliefs about eating, weight, and shape.
Eumenorrhea	menstrual cycles at intervals near the median interval for young adult women. In young adult women, menstrual cycles recur at a median interval of 28 d that varies with a standard deviation of 7 d.
Oligomenorrhea	menstrual cycles at intervals longer than 35 d, i.e., greater than the median plus one standard deviation.
Luteal suppression	a menstrual cycle with a luteal phase shorter than 11 d in length or with a low concentration of progesterone.
Anovulation	a menstrual cycle without ovulation.
Amenorrhea	absence of menstrual cycles for more than 90 d.
Low BMD*	bone mineral density *Z*-score** between −1.0 and −2.0.
Osteoporosis*	bone mineral density *Z*-score** ≤ −2.0 together with secondary risk factors for fracture (e.g., undernutrition, hypoestrogenism, prior fractures).

* These definitions apply to physically active and athletic premenopausal women and children.
* *A *Z*-score compares the bone mineral density of an individual to those of age, race, and sex-matched controls.

for other body functions after exercise training. When energy availability is too low, physiological mechanisms reduce the amount of energy used for cellular maintenance, thermoregulation, growth, and reproduction (199). This compensation tends to restore energy balance and promote survival, but impairs health. Stable body weight in amenorrheic athletes (44,93,127,145,150,209) suggests that energy balance can be restored while energy availability is low.

Some athletes reduce energy availability by increasing exercise energy expenditure more than energy intake. Others reduce energy intake more than exercise energy expenditure. Some practice abnormal eating behaviors such as fasting, binge-eating and purging, or use diet pills, laxatives, diuretics, and enemas (5,17,18,92,182,183).

Some athletes also have eating disorders, which are clinical mental disorders often accompanied by other psychiatric illnesses (26,97). Anorexia nervosa is an eating disorder characterized by restrictive eating in which the individual views herself as overweight and is afraid of gaining weight even though she is at least 15% below expected weight for age and height. Amenorrhea is a diagnostic criterion for anorexia nervosa (5). Anorexia nervosa appears in restricting and purging subtypes. Bulimia nervosa is an eating disorder in which affected individuals, usually in the normal weight range, repeat a cycle of overeating or binge-eating and then purging or other compensatory behaviors such as fasting or excessive exercise (5). Individuals who do not meet all criteria for anorexia nervosa or bulimia nervosa are classified as having an eating disorder not otherwise specified (ED-NOS). An individual may meet all anorexia nervosa criteria except that she has regular menses, or all bulimia nervosa criteria except that she binges and purges less than twice per week (5).

Menstrual Function

This Position Stand refers to a spectrum of menstrual function ranging from eumenorrhea to amenorrhea (Fig. 1). Within this range, oligomenorrhea is recognized by menstrual cycles occurring at intervals longer than 35 d, but luteal deficiency and anovulation have no perceptible symptoms (129,197).

Because menstrual cycles longer than 90 d are rare (197), amenorrhea is defined as the absence of menstrual cycles lasting more than three months (9). Amenorrhea beginning after menarche is called *secondary amenorrhea*. *Primary amenorrhea* refers to a delay in the age of menarche. Because menarche is occurring earlier, the defining age for primary amenorrhea was recently reduced from 16 to 15 yr (9). Animal research clearly demonstrates that energy deficiency before puberty suppresses growth and delays sexual development (174). Many retrospective surveys have established that menarche often occurs later in athletes than in nonathletes, but such surveys are inherently biased (178). Only one prospective observational study has attempted to relate the age of menarche to athletic training (200). The authors found that menarche occurred at a later age in ballet dancers but at the same height and weight as in nondancers.

Bone Mineral Density

This Position Stand refers to a spectrum of BMD ranging from optimal bone health to osteoporosis (Fig. 1). Osteoporosis is defined as "a skeletal disorder characterized by compromised bone strength predisposing a person to an increased risk of fracture" (146). Bone strength and the risk of fracture depend on the density and internal structure of bone mineral and on the quality of bone protein, which may explain why one person suffers fractures while another with the same BMD does not. Although BMD is only one aspect of bone strength, this Position Stand focuses on BMD because screening and diagnosis of osteoporosis are still based on BMD. Osteoporosis is not always caused by accelerated bone mineral loss in adulthood. It may also be caused by not accumulating optimal BMD during childhood and adolescence (146).

No BMD threshold discriminates perfectly between those who will and will not experience a fracture (95). So, osteoporosis is diagnosed in terms of a BMD level at which the risk for fracture is considered unacceptable (25). The World Health Organization (WHO) criteria for diagnosing osteopenia (low BMD) and osteoporosis in postmenopausal women are based on *T*-scores that compare individuals to average peak adult BMD. These criteria derive from epidemiological data relating BMD to subsequent fractures in Caucasian postmenopausal women: BMD has a strong and independent predictive value for osteoporotic fractures in postmenopausal women (41). The fracture rate doubles for each reduction of one standard deviation in BMD (85).

The 1997 ACSM Position Stand on the Triad specified the WHO criteria for diagnosing osteopenia and osteoporosis in female athletes (96). However, epidemiologic data relating BMD to fractures in adolescents and premenopausal women are lacking. There is no agreement on standards for adjusting BMD for bone size, pubertal stage, skeletal maturity, or body composition in growing children. Therefore, fracture risk cannot be predicted for individuals in these populations on the basis of BMD alone (90,116). For the Triad, this problem is worsened by the lack of longitudinal data relating BMD to fractures in young, undernourished hypoestrogenic women exposed to elevated mechanical loads.

Recently, the International Society for Clinical Densitometry (ISCD) published official positions recommending that the WHO criteria for diagnosing osteopenia and osteoporosis not be applied to premenopausal women and children (90,115,117). Instead, the ISCD recommended that BMD in these populations be expressed as *Z*-scores to compare individuals to age and sex-matched controls, and that *Z*-scores below −2.0 be termed "low bone density below the expected range for age" in premenopausal women and as "low bone density for chronological age" in children. The ISCD also recommended that the term osteopenia not be used and that osteoporosis be diagnosed in these populations only when low BMD is present with secondary clinical risk factors that reflect an elevated short-term risk of bone mineral loss and fracture. These secondary risk factors include chronic malnutrition, eating disorders, hypogonadism, glucocorticoid exposure, and previous fractures. The ISCD recommendations have been endorsed by the American Society for Bone and Mineral Research, the International Osteoporosis Foundation, and the American Association of Clinical Endocrinologists.

Athletes in weight-bearing sports usually have 5–15% higher BMD than nonathletes (55,151,165,166). Therefore, a BMD *Z*-score < -1.0 in an athlete warrants further investigation, even in the absence of a prior fracture. ACSM defines the term "low BMD" as a history of nutritional deficiencies, hypoestrogenism, stress fractures, and/or other secondary clinical risk factors for fracture (90,100,102) together with a BMD *Z*-score between −1.0 and −2.0. To reflect an increased risk of fragility fracture (90,100), ACSM defines "osteoporosis" as secondary clinical risk factors for fracture with BMD *Z*-scores ≤ -2.0.

An athlete's BMD reflects her cumulative history of energy availability and menstrual status as well as her genetic endowment and exposure to other nutritional, behavioral, and environmental factors. Therefore, it is important to consider both where her BMD is currently and how it is moving along the BMD spectrum. The onset of amenorrhea does not cause osteoporosis immediately, but skeletal demineralization begins moving her BMD in that direction. Similarly, resuming regular menses does not immediately restore optimal bone health, but mineral accumulation begins to improve her BMD.

HEALTH CONSEQUENCES

Sustained low energy availability, with or without disordered eating, can impair health. Psychological problems associated with eating disorders include low self-esteem, depression, and anxiety disorders (167). Medical complications involve the cardiovascular, endocrine, reproductive, skeletal, gastrointestinal, renal, and central nervous systems (5,19,62,167). The prognosis for anorexia nervosa is grave with a sixfold increase in standard mortality rates compared to the general population (153). In one study, 5.4% of athletes with eating disorders reported suicide attempts (184). Although 83% of anorexia nervosa patients partially recover, the rate of sustained recovery of weight, menstrual function and eating behavior is only 33% (77).

Amenorrheic women are infertile, due to the absence of ovarian follicular development, ovulation, and luteal function. While recovering, however, they may ovulate before their menses are restored, resulting in an unexpected pregnancy if a reliable form of birth control is not utilized. Athletes with luteal deficiency may also be at risk for infertility due to poor follicular development or failure of implantation. Consequences of hypoestrogenism seen in amenorrheic athletes include impaired endothelium-dependent arterial vasodilation (78), which reduces the perfusion of working muscle, impaired skeletal muscle oxidative metabolism (75), elevated low-density lipoprotein cholesterol levels (59,154), and vaginal dryness (73).

BMD declines as the number of missed menstrual cycles accumulates (45,120), and the loss of BMD may not be fully reversible (99,201). Stress fractures occur more commonly in physically active women with menstrual irregularities and/or low BMD (21–23,112,144,149,203,211) with a relative risk for stress fracture two to four times greater in amenorrheic than eumenorrheic athletes (21). Fractures also occur in the setting of nutritional deficits and low BMD (50,111,130,169,210). Any premenopausal fracture unrelated to a motor vehicle accident is a strong predictor for postmenopausal fractures (82,213).

Evidence Statements. Severe undernutrition impairs reproductive and skeletal health. *Evidence category A.*

Menstrual irregularities and low BMD increase stress fracture risk. *Evidence category A.*

EPIDEMIOLOGY

Prevalence

In athletes, the prevalence of disordered eating, menstrual disorders, low BMD and stress fractures varies widely (31,151,161). The prevalence of inadvertent low energy availability without disordered eating or eating disorders is unknown.

Many studies of the prevalence of disordered eating and eating disorders in athletes have yielded unreliable results due to nonstandard diagnostic procedures, small sample sizes, absent or inappropriate control groups, inadequate statistics, and heterogeneity in the type and level of the athletes studied (30). Only two large, well-controlled studies have diagnosed clinical eating disorders according to the *Diagnostic and Statistical Manual of Mental Disorders* (8) to obtain unbiased and reliable estimates of the prevalence of eating disorders in elite female athletes in different types of sports (31,186). One found eating disorders in 31% of elite female athletes in "thin-build" sports compared to 5.5% of the control population (31). The other found that 25% of female elite athletes in endurance sports, aesthetic sports, and weight-class sports had clinical eating disorders compared to 9% of the general population (186). A small study of collegiate gymnasts ($N = 42$) found a prevalence of disordered eating behaviors as high as 62% (170). A larger study of collegiate gymnasts ($N = 218$) showed specific weight control behaviors included binge eating (33% $\geq$ once a week), exercise for the purpose of burning calories (57% $\geq$ 2 h·d^{-1}), and fasting or strict dieting (28% $\geq$ 4 times in the past year). However, induced vomiting and the use of diuretics and/or laxatives were rare events (158).

The prevalence of secondary amenorrhea, long known to vary widely with sport, age, training volume and body weight (161), has been reported in small studies to be as high as 69% in dancers (1) and 65% in long-distance runners (51) compared to 2–5% in large studies of the general population (11,159,175). Within distance runners, prevalence of amenorrhea increased from 3% to 60% as training mileage increased from <13 to >113 km·wk^{-1} while their body weights decreased from >60 to <50 kg (172). Prevalence of secondary amenorrhea is higher (67%) in female runners less than 15 yr of gynecological age compared to older women (9%) (13). The prevalence of primary amenorrhea is less than 1% in the general population (33) and more than 22% in cheerleading, diving and gymnastics (18). Subclinical menstrual disorders typify both highly trained (121) and recreational (42,53) eumenorrheic athletes: luteal deficiency or anovulation was found in 78% of eumenorrheic recreational runners in at least one menstrual cycle out of three (42).

Low BMD has been associated with disordered eating even in eumenorrheic athletes (34). BMD is lower in amenorrheic athletes than in eumenorrheic athletes (46,127,143,151,162). A systematic review of studies that employed WHO *T*-scores for diagnosis, found prevalence of osteopenia (*T*-score between −1.0 and −2.5) ranging from 22% to 50% and prevalence of osteoporosis (*T*-score $\leq$ −2.5) ranging from 0% to 13% in female athletes (104) compared to 12% and 2.3% expected in a normal population distribution.

Only three studies of female athletes have investigated the simultaneous occurrence of disordered eating, menstrual disorders and low BMD according to ISCD criteria (17,152,191). Only one diagnosed eating disorders (191). The prevalence of the entire Triad in elite athletes from 66 diverse sports (4.3%; 8/186) was similar to controls (3.4%; 5/145) (191), but the athletes' BMD *Z*-scores were calculated relative to the controls rather than instrument norms. Half the athletes and none of the controls had clinical eating disorders and BMD *Z*-scores < −2.0. All controls with some, but not all Triad components were overweight rather than underweight and had a history of pathogenic weight loss behavior. The other two studies referenced BMD *Z*-scores to instrument norms. One found the entire Triad in 2.7% (3/112) of collegiate athletes from seven diverse sports (17). The other found the entire Triad in 1.2% (2/170) of high school athletes (152). All these studies defined the Triad more narrowly than this Position Stand: none estimated energy availability, diagnosed subclinical menstrual disorders or the cause of amenorrhea, or assessed changes in BMD.

Future epidemiologic studies should include cases of low energy availability without disordered eating or an eating disorder, luteal deficiency and anovulation, and declining as well as low BMD *Z*-scores based upon the best available standardized normative database. Where these databases do not yet exist, they should be developed.

Evidence Statement. Disordered eating, eating disorders and amenorrhea occur more frequently in sports that emphasize leanness. *Evidence category A.*

Risk Factors

Athletes at greatest risk for low energy availability are those who restrict dietary energy intake, who exercise for prolonged periods, who are vegetarian, and who limit the types of food they will eat (34,125,126). Many factors appear to contribute to disordered eating behaviors and clinical eating disorders (19,167). Dieting is a common entry point (167) and interest has focused on the contribution of environmental and social factors, psychological predisposition (34,167), low self-esteem (160,167), family dysfunction (137), abuse (168), biological factors (98), and genetics (28,179). Additional factors for athletes include early start of sport-specific training and dieting, injury, and a sudden increase in training volume (184). Surveys show more negative eating attitude scores in athletic disciplines favoring leanness (18,27). Disordered eating behaviors are

risk factors for eating disorders (167): Sundgot-Borgen found that 18% of elite female athletes and 5% of controls with disordered eating behaviors were diagnosed with clinical eating disorders (183).

Surveys of menstrual history have identified potential risk factors associated with amenorrhea, but not hormone levels. Most potential risk factors have been shown not to be causal factors in the disruption of reproductive function in athletes. For example, body weight and fatness are often low in amenorrheic athletes, but eumenorrheic and amenorrheic athletes span a common range of body weight and body fatness leaner than the general population (161,171). In addition, exercise training has no suppressive effect on luteinizing hormone (LH) pulsatility when energy intake is increased to compensate for exercise energy expenditure (123).

Risk factors for stress fracture include low BMD, menstrual disturbances, late menarche, dietary insufficiency, genetic predisposition, biomechanical abnormalities, training errors, and bone geometry (e.g., narrower tibia width, shorter tibia length) (14,23,60,144,201).

MECHANISMS

Low Energy Availability

A specific etiology for the pathogenesis of eating disorders remains unclear (167). A large, randomized, prospective study of 2000 teenage boys and girls found dieting and psychiatric morbidity to be the most sensitive, independent predictors of new, clinical eating disorders (157). Girls who were dieting at moderate and severe levels were 5 and 18 times more likely to be diagnosed with clinical eating disorders six months later, respectively, and girls in the highest and second highest of four psychiatric morbidity categories were 7 and 3 times more likely.

In athletes, dieting per se may not lead to a clinical eating disorder, but rather the situation in which the athlete is told to lose weight, the words used, and the availability of weight loss guidance (184). Nutritional counseling is also essential for preventing inadvertent low energy availability because "there is no strong biological imperative to match energy intake to activity-induced energy expenditure" (195). In experiments, dietary restriction increased hunger, but the same energy deficit produced by exercise did not (84). Resulting *ad libitum* energy deficits are more extreme when consuming high carbohydrate diets such as those recommended for endurance athletes (80,81,181). Thus, low energy availability may occur inadvertently without clinical eating disorders, disordered eating behaviors or even dietary restriction.

Menstrual Disorders

In animal experiments, reducing dietary intake by more than 30% has consistently caused infertility (79,131,133) and skeletal demineralization (94,140,173,187). In the Triad, menstrual disorders result from the pituitary gland not secreting pulses of LH at the correct frequency (114,121). Laboratory research has shown that LH pulsatility is disrupted within 5 d when the energy availability of young women is reduced by more than 33% from 45 to less than 30 $kcal \cdot kg^{-1}$ $FFM \cdot d^{-1}$ (122), which corresponds to the energy expended in resting metabolism in healthy young adults (20,57,141,145,189,209). When the energy cost of running is estimated at 90 kcal per mile, the energy availabilities of young, adult amenorrheic runners are consistently less than 30 $kcal \cdot kg^{-1}$ $FFM \cdot d^{-1}$ (44,46,93, 108,127,145,150,190,209). Some eumenorrheic runners also have energy availabilities less than 30 $kcal \cdot kg^{-1}$ $FFM \cdot d^{-1}$ (42,127,209,212) and present with subclinical menstrual disorders (42), suggesting some women may be less susceptible to low energy availability. In the only quantified intervention published to date, menses were restored in amenorrheic runners when energy availability increased from an average of 25 to 30 $kcal \cdot kg^{-1}$ $FFM \cdot d^{-1}$ (108). More studies are needed to develop effective intervention strategies for athletes.

LH pulsatility reflects the pulsatile secretion of gonadotropin-releasing hormone (GnRH) from the hypothalamus (56). Neural pathways have been traced from receptors in the brain for several metabolic hormones and substrates to the GnRH pulse generator (199). Low energy availability alters levels of metabolic hormones (e.g., insulin, cortisol, growth hormone, insulin-like growth factor-I (IGF-I), 3,3,5-triiodothyronine (T_3) and leptin) and substrates (e.g., glucose, fatty acids, and ketones). Weight loss may also lower leptin levels. One or more of these is thought to constitute a metabolic signal to GnRH-secreting neurons (198), but specific signals and pathways disrupting GnRH pulsatility in exercising women have yet to be identified.

Regardless of how low energy availability disrupts GnRH and LH pulsatility, it can occur without a diagnosable eating disorder, high scores on validated questionnaires about eating behavior indicating increased risks of developing an eating disorder, or even dietary restriction. In long-term prospective experiments, luteal deficiency and anovulation have been induced in young women by increasing exercise energy expenditure alone (29). In female monkeys amenorrhea has been induced by increasing exercise energy expenditure without reducing dietary energy intake (207). Then their ovulation was restored by increasing energy intake without moderating the exercise regimen (208). This type of amenorrhea is called *functional hypothalamic amenorrhea*.

Low BMD

The primary cause of osteoporosis in postmenopausal women is estrogen deficiency, which increases bone resorption. Osteoporosis also occurs secondary to medical disorders such as hypogonadal states, hyperthyroidism, and nutritional deficiencies (146). Estrogen deficiency probably accounts for only a small part of the abnormal bone

remodeling in athletes with functional hypothalamic amenorrhea (220). As is the case with anorexia nervosa patients (67), estrogen deficiency in athletes with functional hypothalamic amenorrhea is often accompanied by chronic undernutrition (43,114,121,190,206,220), which reduces the rate of bone formation (220) (Fig. 1). In a randomized clinical trial, the rate of bone resorption increased and the rate of bone formation declined within 5 d after energy availability was reduced below 30 kcal·kg^{-1} FFM·d^{-1} in exercising women (86). Resorption increased when energy availability was restricted enough to suppress estradiol, and bone formation was suppressed at higher energy availabilities in dose-response relationships resembling those of insulin, T_3 and IGF-I (hormones that regulate bone formation) (86). Low energy availability may also suppress bone formation via effects on other hormones, including cortisol and leptin (48,91,113,114,121,128,136,176,206,219). Further research into hormonal, metabolic, and mechanical influences on BMD in female athletes is needed (166,176,217).

SCREENING AND DIAGNOSIS

Published clinical guidelines for the diagnosis and treatment of Triad disorders are listed in Table 3. Screening for the Triad can be challenging because its health consequences are not always readily apparent. Although affected athletes are usually involved in sports where thinness is believed to be advantageous, one or more clinical consequences of the Triad can occur in individuals participating in any sport or habitual, strenuous physical activity. Screening for the Triad requires an understanding of the relationships among its components, the spectrum within each component, and rates of movement along each spectrum (Fig. 1). Optimal screening times occur at the preparticipation physical exam and annual health check ups (3,148,155). Other opportunities occur when athletes are evaluated for related problems, such as amenorrhea, stress fractures, or recurrent injury or illness. An athlete who presents with one component of the Triad should be assessed for the others (155).

Because eating disorders appear to be greatly underdiagnosed and inadequately treated (70), reference should be made to guidelines for their diagnosis and treatment in primary care (2,6,7,107,110,132,160). New screening tools for use in primary care (37,124,139) should be investigated. Failure to meet all criteria for anorexia nervosa or bulimia nervosa should not deter the health-care provider from offering early and comprehensive intervention, because early recognition and intervention with ED-NOS can prevent athletes from developing full-blown eating disorders (2,167). Even in the absence of a clinical eating disorder, restrictive and purging behaviors are of greatest concern, because they reduce energy availability. Past histories of these behaviors are of interest because their effects on bone are cumulative. Other disordered eating behaviors are also of interest, because they may indicate a tendency to restrictive and purging behaviors or alternating patterns of eating behaviors.

TABLE 3. Published clinical guidelines for the diagnosis and treatment of Triad disorders.

Eating disorders	2, 4, 5, 7, 8, 62, 107, 110, 132, 160
Functional hypothalamic amenorrhea	9, 10, 72, 177, 204
Premenopausal osteoporosis	74, 90, 100, 102

Recommendation. Screening for the Triad should occur at the preparticipation exam or annual health screening exam. *Evidence category C-2*. Athletes with one component of the Triad should be assessed for the others. *Evidence category C-2*.

Patient History

Information on energy intake, dietary practices, weight fluctuations, eating behaviors, and exercise energy expenditure should be obtained. Disturbed body image, fear of weight gain and menstrual dysfunction are common in athletes with disordered eating or eating disorders. Athletes with disordered eating should be referred to a mental health practitioner for further evaluation, diagnosis, and recommendations for treatment. Menstrual status and history and other factors associated with low BMD, such as prior stress fractures, should also be assessed.

Recommendation. Athletes with disordered eating should be referred to a mental health practitioner for evaluation, diagnosis and recommendations for treatment. *Evidence category C-2*.

Physical Exam

An athlete with a history suggestive of one or more components of the Triad should have a physical exam. The health-care provider should be alert for signs and symptoms of an eating disorder. Height, weight, and vital signs should be obtained (19,167). Bradycardia is commonly seen as well as orthostatic hypotension. Other findings include cold/discolored hands and feet, hypercarotenemia, lanugo hair, and parotid gland enlargement (19). If the athlete is diagnosed with an eating disorder by a mental health specialist, an EKG should be obtained, as the QT interval is sometimes prolonged, even in the presence of normal serum electrolytes (19). With functional hypothalamic amenorrhea, the physical exam is usually normal, but hypoestrogenism with vaginal atrophy may be present on pelvic exam.

Laboratory Tests

In the athlete with disordered eating or an eating disorder, an initial laboratory assessment should include electrolytes, a chemistry profile, a complete blood count with differential, erythrocyte sedimentation rate, thyroid function tests, and urinalysis (2,9,10,19). However, because wide ranges for normal values can be found in severely undernourished individuals, health-care providers should not be reassured

by normal test results. Since there is no blood test for functional hypothalamic amenorrhea, this condition is diagnosed by excluding other causes of amenorrhea (9,72,177,204). Initial evaluation for secondary amenorrhea includes a pregnancy test, gonadotropin measurement (follicle stimulating hormone (FSH) and LH) to rule out ovarian failure and check for the increased LH/FSH ratio seen in polycystic ovary syndrome (PCOS), a prolactin test to rule out a lactotropic secreting tumor, and a thyroid stimulating hormone test for thyroid disease. If there is evidence of androgen excess on physical exam, free testosterone and dehydroepiandrosterone sulfate may be obtained to evaluate for PCOS, an androgen-secreting tumor of the ovary or adrenal, or congenital adrenal hyperplasia. In contrast to the mild elevations of serum cortisol that can be seen in functional hypothalamic amenorrhea, extreme elevations indicate Cushing's syndrome. Serum estradiol can be obtained, or a progesterone challenge test can be done to assess estrogen indirectly by administering medroxyprogesterone acetate 10 mg once daily for 7–10 d. In functional hypothalamic amenorrhea, gonadotropins are low or normal, estradiol is low, prolactin and thyroid stimulating hormone are in the normal range. A hypoestrogenic athlete may not withdraw to a progesterone challenge, although some athletes will experience menses with this challenge as they near recovery.

Additional testing may be needed based on patient history and physical examination, and for assessment of primary amenorrhea. Consultation with a physician experienced in treating female athletes or a reproductive medicine specialist is recommended if menses are not restored after 3–6 months of treatment, especially if the athlete has suffered a fracture.

Recommendation. To diagnose functional hypothalamic amenorrhea, other causes of amenorrhea must be excluded. *Evidence category B.*

Bone Mineral Density Testing

A history of hypoestrogenism, disordered eating or eating disorders for a cumulative total of 6 months or more, and/or a history of stress fractures or fractures from minimal trauma warrants BMD assessment by dual-energy X-ray absorptiometry (DXA) (102). Reevaluation is recommended in 12 months in those with persistent Triad disorders. Serial DXA studies should be obtained on the same machine. Genetic differences may affect an individual's susceptibility to or starting point for the effects of low energy availability on BMD. Wide variability in BMD exists among amenorrheic athletes (46,166,216), and differences are seen between sports (55,166) and skeletal sites (66). Diagnosis of low BMD or osteoporosis is based on the lowest BMD Z-score of either the posterior–anterior (PA) (not lateral) spine or the hip (femoral neck or total hip, not Ward's area or greater trochanter), and both sites should be measured (74,101). In individuals less than 20 yr of age, PA spine and whole body are the preferred sites (100). Radial BMD may be normal in athletes with functional hypothalamic amenorrhea, even though lumbar BMD is often decreased (46).

Recommendation. BMD should be assessed after a stress or low impact fracture and after a total of 6 months of amenorrhea, oligomenorrhea, disordered eating or an eating disorder. *Evidence category C-2.*

PREVENTION AND TREATMENT RECOMMENDATIONS

The discovery of suppressed bone formation and the failure of antiresorptive therapy to fully restore BMD in athletes with functional hypothalamic amenorrhea changed our understanding and recommendations for management of the Triad. Uncoupling of bone turnover with a reduction of bone formation and an increase in bone resorption can cause irreversible reductions in BMD (35). Suppression of bone formation by moderate restrictions of energy availability also suggests that large numbers of only moderately energy-deficient adolescents without clinical hypoestrogenic menstrual disorders may fail to achieve their genetic potential for peak BMD.

Prevention and treatment of the Triad should employ a team approach including a physician or other health-care provider (physician's assistant or nurse practitioner), a registered dietitian, and for athletes with disordered eating or an eating disorder, a mental-health practitioner (3,16,148,155,215). Health-care personnel with knowledge of disordered eating behavior and eating disorders in particular sports will be better able to understand the demands of those sports. Additional valuable team members may include a certified athletic trainer, an exercise physiologist, and the athlete's coach, parents and other family members. Barriers to the treatment of eating disorders in the U.S. should be recognized (167).

Recommendation. Multidisciplinary treatment for the Triad disorders should include a physician (or other health-care professional), a registered dietitian, and, for athletes with disordered eating or an eating disorder, a mental health practitioner. *Evidence category C-2.*

Prevention

Athletic administrators and the entire health-care team should aim to prevent the Triad through education (148,155,215). Emphasis should be placed on optimizing energy availability for prevention (86,122,123). Special attention should also be given to maximizing bone mineral accrual in pediatric and adolescent athletes (103) and to maintaining bone health throughout life (106). Children, adolescents, and young adults should be counseled on nutritional requirements for their age, including calcium and vitamin D (88,196), and on the benefits of regular weight-bearing exercise for bone health (106). Athletes with menstrual disorders and/or low energy availability with or without disordered eating or eating disorders should be

educated about the risk of impaired bone mineral accrual, declining BMD, osteoporosis, and stress fractures.

Like other organizations (4,89), ACSM recommends that national and international governing bodies of sports and athletic organizations put procedures and policies in place to eliminate potentially harmful weight loss practices of female athletes. Procedures and policies are not specified, because best practices may be sport-specific.

Treatment

Nonpharmacological therapy. Increases in BMD of 5% per year have accompanied increases in body weight in cohort and case studies of amenorrheic athletes (47,58,118, 201,202,218). In anorexia nervosa, increases in BMD of 2–3% per year have been seen with weight gain in most (12,15,24,64,83,87,135) but not all (36,164) studies. Therefore, the first aim of therapy to restore menstrual cycling and increase BMD is to modify diet and exercise behavior to increase energy availability by increasing energy intake, reducing energy expenditure, or a combination (49,108) according to the athlete's compliance with recommendations. Menstrual cycles may be restored by increasing energy availability to more than 30 $kcal \cdot kg^{-1}$ $FFM \cdot d^{-1}$ (108), but the strong association between increases in BMD and increases in body weight (47,58,118,201,202,218) implies that increasing BMD may require more than 45 $kcal \cdot kg^{-1}$ $FFM \cdot d^{-1}$. This value corresponds to energy balance in healthy young women (122,123,141). Athletes practicing restrictive eating behaviors should be counseled that increases in body weight may be necessary to increase BMD. More research is needed to determine whether this is true.

Affected athletes should be referred to a dietitian for nutrition counseling and to have their energy availability estimated. Exercise, diet, and low/fluctuating weight should all be discussed. Adequate amounts of bone-building nutrients such as calcium (1000–1300 $mg \cdot d^{-1}$), vitamin D (400–800 $IU \cdot d^{-1}$), and vitamin K (60–90 $\mu g \cdot d^{-1}$) are needed (65,88,146,147,194). Supplements for calcium and vitamin D may be necessary. More research is needed to determine if higher intakes of calcium and vitamin D increase BMD and reduce fractures in female athletes with the Triad disorders. Protein needs for female athletes engaged in intense exercise training may also be higher (1.2–1.6 $g \cdot kg^{-1} \cdot d^{-1}$) (126,188) than those recommended for the population at large (0.8 $g \cdot kg^{-1} \cdot d^{-1}$) (193). Increased energy availability should continue until menses resume and be maintained while training and competing.

The treatment goal for athletes with disordered eating or eating disorders is to optimize overall nutritional status, normalize eating behavior, modify unhealthy thought processes that maintain the disorder, and treat possible emotional issues that for some athletes create a need for the disorder. Treatment success is based on a trusting relationship between the athlete and the care providers. The younger the athlete, the more the family's involvement is recommended. In addition to nutrition counseling and individual psychotherapy, treatment includes cognitive behavioral, group and family therapy (6,7,16,19,62,110,167).

An athlete in treatment for disordered eating or eating disorders should meet minimal criteria to continue training and competition. The athlete must agree (i) to comply with all treatment strategies; (ii) to be closely monitored by health-care professionals; (iii) to place precedence on treatment over training and competition; and depending on her medical status (iv) to modify the type, duration, and intensity of training and competition (16,185). A written contract may be used to specify these agreements. Close follow-up of progress and ongoing communication with the health-care team are essential. If the athlete does not accept treatment, breaks her contract, or her eating behavior and weight do not improve, she may need to be excluded from training and competition, but follow-up should continue.

Recommendations. The first aim of treatment is to increase energy availability by increasing energy intake and/or reducing energy expenditure. Athletes without disordered eating or eating disorders should be referred for nutritional counseling. *Evidence category C-1.* Athletes practicing restrictive eating behaviors should be counseled that increases in body weight may be necessary to increase BMD. *Evidence category C-1.* Treatment for disordered eating and eating disorders includes nutritional counseling and individual psychotherapy. Cognitive behavioral, group therapy, and/or family therapy may also be used. *Evidence category B.* Athletes with disordered eating and eating disorders who do not comply with treatment may need to be restricted from training and competition. *Evidence category C-2.*

Pharmacological therapy. Antidepressants are often utilized for bulimia nervosa, anorexia nervosa following weight restoration, ED-NOS, and for concomitant depression and anxiety disorders (2,7,62,110,167,214), but no pharmaceutical agent approved for use in this population has been shown to fully restore BMD in women with functional hypothalamic amenorrhea. When 93 women diagnosed with functional hypothalamic amenorrhea (without anorexia nervosa) chose between two pharmacological treatments or no treatment, 30% had still not recovered menstrual cycles after 8 yr (54). There was no benefit from hormone replacement therapy (HRT) and the oral contraceptive pill (OCP) delayed and reduced the likelihood of restoring menstrual cycles. No woman recovered whose body mass index (BMI) declined, but all those whose BMI increased did recover (54).

Bone mineral density increased by less than 4% per year in two cohort studies of women with functional hypothalamic amenorrhea who were treated with HRT (38,69), but not in a third (202). In a cohort study of anorexia nervosa patients, adjustment for weight gain cancelled out apparent effects of HRT (64). Evidence of the effectiveness of OCP for increasing BMD in athletes and other women with functional hypothalamic amenorrhea without eating

disorders is also mixed (119), with some randomized clinical trials and cohort studies finding partial recovery (32,40,69,71,76,163,205) and others not (61,66), but concurrent changes in body weight were often not reported. One study reported that the increase in BMD was accompanied by an increase in weight (163) and another reported that the effect of weight gain exceeded the effect of OCP (69). Neither HRT nor OCP has increased BMD in any prospective study of women with anorexia nervosa (63,64,68,105,109,142,180).

It must be emphasized that pharmacological restoration of regular menstrual cycles with OCP will not normalize metabolic factors that impair bone formation, health and performance. Thus it is unlikely to fully reverse the low BMD in this population (39,138,202). Bone mineral density should be monitored annually in women with persistent functional hypothalamic amenorrhea, disordered eating, and/or low BMD. If BMD declines in an athlete greater than 16 yr of age with persistent functional hypothalamic amenorrhea despite adequate nutritional intake and weight, then OCP may be considered with the hope of minimizing further bone loss. There are no established guidelines as to when or if to administer OCP to the adolescent athlete less than 16 yr of age with functional hypothalamic amenorrhea (3), due to concern about premature closure of growth plates and lack of research to support this therapy in this age group.

Bisphosphonates approved for the treatment of postmenopausal osteoporosis should not be used in the young athlete with functional hypothalamic amenorrhea for two reasons. The first is because of their unproven efficacy in women of child-bearing age (134). The second reason is that the bisphosphonates may reside in a woman's bone for many years, potentially causing harm to a developing fetus during pregnancy (156).

If a further aim of therapy is to restore fertility in the athlete who wishes to become pregnant, induction of ovulation with agents such as clomiphene citrate and exogenous gonadotropins is indicated, although the athlete should be warned about the risks and hazards of having a low birth weight infant when an undernourished mother does not reform her dietary habits (192).

Clearly, more research is needed to resolve whether any currently approved or new form of hormone therapy is effective for increasing BMD in athletes with functional hypothalamic amenorrhea. In this research, BMD and other factors should be monitored carefully to distinguish pharmacological and nonpharmacological effects. Research is also needed on other types of pharmacologic therapies. Pending further research, increased energy availability and restoration of gonadal function are the cornerstones of treatment for the Triad.

Evidence statement. In functional hypothalamic amenorrhea, increases in BMD are more closely associated with increases in weight than with OCP/HRT administration. *Evidence category C-1*. OCP should be considered in an athlete with functional hypothalamic amenorrhea over age 16, if BMD is decreasing with nonpharmacological management, despite adequate nutrition and body weight. *Evidence category C-2*.

CONCLUSION

Low energy availability with or without eating disorders, functional hypothalamic amenorrhea, and osteoporosis, alone or in combination, pose significant health risks to physically active girls and women. Prevention, recognition, and treatment of these clinical conditions should be a priority of those who work with female athletes to ensure that they maximize the benefits of regular exercise.

This pronouncement was reviewed for the American College of Sports Medicine by members-at-large; the Pronouncements Committee; and by Loretta DiPietro, Ph.D., FACSM; Mimi D. Johnson, M.D., FACSM; Constance M. Lebrun, M.D., FACSM; Richard D. Lewis, Ph.D.; Carol L. Otis, M.D.; Hugh S. Taylor, M.D.; B. Timothy Walsh, M.D.; and Nancy I. Williams, Sc.D., FACSM. Additional acknowledgments are given to Barbara L. Drinkwater, Ph.D.; James S. Skinner, Ph.D., FACSM; and Lisa Schnirring for their invaluable assistance with reviewing and editing this Position Stand.

This Position Stand replaces the 1997 ACSM Position Stand, "The Female Athlete Triad," *Med. Sci. Sports Exerc.* 29(5):i–ix, 1997.

REFERENCES

1. Abraham, S. F., P. J. Beumont, I. S. Fraser, and D. Llewellyn-Jones. Body weight, exercise and menstrual status among ballet dancers in training. *Br. J. Obstet. Gynaecol.* 89:507–510, 1982.
2. American Academy of Pediatrics Committee on Adolescence. Identifying and treating eating disorders. *Pediatrics* 111: 204–211, 2003.
3. American Academy of Pediatrics Committee on Sports Medicine and Fitness. Medical concerns in the female athlete. *Pediatrics* 106:610–613, 2000.
4. American Academy of Pediatrics Committee on Sports Medicine and Fitness. Promotion of healthy weight-control practices in young athletes. *Pediatrics* 116:1557–1564, 2005.
5. American Psychiatric Association Work Group on Eating Disorders. Practice guideline for the treatment of patients with eating disorders (revision). *Am. J. Psychiatry* 157:1–39, 2000.
6. American Psychiatric Association Working Group on Eating Disorders. Treating Eating Disorders: A Quick Reference Guide. Available at: http://www.psych.org/psych_pract/treatg/quick_ref_guide/EDs_QRG.pdf. Accessed December 23, 2006.
7. American Psychiatric Association Working Group on Eating Disorders. Treatment of patients with eating disorders, third edition. *Am. J. Psychiatry* 163:4–54, 2006.
8. American Psychiatric Association Task Force on DSM-IV. *Diagnostic and Statistical Manual of Mental Disorders: DSM-IV-TR*., 4th, text revision. ed, Washington, DC: American Psychiatric Association, 2000.
9. American Society of Reproductive Medicine Practice Committee. Current evaluation of amenorrhea. *Fertil. Steril.* 82:266–272, 2004.
10. American Society of Reproductive Medicine Practice Committee. Current evaluation of amenorrhea. *Fertil. Steril.* 86:S148–S155, 2006.

11. Bachmann, G. A., and E. Kemmann. Prevalence of oligomenorrhea and amenorrhea in a college population. *Am. J. Obstet. Gynecol.* 144:98–102, 1982.
12. Bachrach, L. K., D. K. Katzman, I. F. Litt, D. Guido, and R. Marcus. Recovery from osteopenia in adolescent girls with anorexia nervosa. *J. Clin. Endocrinol. Metab.* 72:602–606, 1991.
13. Baker, E. R., R. S. Mathur, R. F. Kirk, and H. O. Williamson. Female runners and secondary amenorrhea: correlation with age, parity, mileage, and plasma hormonal and sex-hormone-binding globulin concentrations. *Fertil. Steril.* 36:183–187, 1981.
14. Barrow, G. W., and S. Saha. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am. J. Sports Med.* 16:209–216, 1988.
15. Bass, S. L., L. Saxon, A. M. Corral, C. P. Rodda, B. J. Strauss, D. Reidpath, and C. Clarke. Near normalisation of lumbar spine bone density in young women with osteopenia recovered from adolescent onset anorexia nervosa: a longitudinal study. *J. Pediatr. Endocrinol. Metab.* 18:897–907, 2005.
16. Beals, K. A. *Disordered Eating among Athletes: A Comprehensive Guide for Health Professionals*, Champaign, IL: Human Kinetics, 2004.
17. Beals, K. A., and A. K. Hill. The prevalence of disordered eating, menstrual dysfunction, and low bone mineral density among US collegiate athletes. *Int. J. Sport Nutr. Exerc. Metab.* 16:1–23, 2006.
18. Beals, K. A., and M. M. Manore. Disorders of the female athlete triad among collegiate athletes. *Int. J. Sport Nutr. Exerc. Metab.* 12:281–293, 2002.
19. Becker, A. E., S. K. Grinspoon, A. Klibanski, and D. B. Herzog. Eating disorders. *N. Engl. J. Med.* 340:1092–1098, 1999.
20. Beidleman, B. A., J. L. Puhl, and M. J. De Souza. Energy balance in female distance runners. *Am. J. Clin. Nutr.* 61: 303–311, 1995.
21. Bennell, K., G. Matheson, W. Meeuwisse, and P. Brukner. Risk factors for stress fractures. *Sports Med.* 28:91–122, 1999.
22. Bennell, K. L., and P. D. Brukner. Epidemiology and site specificity of stress fractures. *Clin. Sports Med.* 16:179–196, 1997.
23. Bennell, K. L., S. A. Malcolm, S. A. Thomas, S. J. Reid, P. D. Brukner, P. R. Ebeling, and J. D. Wark. Risk factors for stress fractures in track and field athletes. A twelve-month prospective study. *Am. J. Sports Med.* 24:810–818, 1996.
24. Bolton, J. G., S. Patel, J. H. Lacey, and S. White. A prospective study of changes in bone turnover and bone density associated with regaining weight in women with anorexia nervosa. *Osteoporos. Int.* 16:1955–1962, 2005.
25. Bonnick, S. L. Current controversies in bone densitometry. *Curr. Opin. Rheumatol.* 14:416–420, 2002.
26. Braun, D. L., S. R. Sunday, and K. A. Halmi. Psychiatric comorbidity in patients with eating disorders. *Psychol. Med.* 24:859–867, 1994.
27. Brooks-Gunn, J., C. Burrow, and M. P. Warren. Attitudes toward eating and body weight in different groups of female adolescent athletes. *Int. J. Eat Disorder* 7:749–757, 1988.
28. Bulik, C. M., P. F. Sullivan, F. Tozzi, H. Furberg, P. Lichtenstein, and N. L. Pedersen. Prevalence, heritability, and prospective risk factors for anorexia nervosa. *Arch. Gen. Psychiatry* 63:305–312, 2006.
29. Bullen, B. A., G. S. Skrinar, I. Z. Beitins, G. von Mering, B. A. Turnbull, and J. W. McArthur. Induction of menstrual disorders by strenuous exercise in untrained women. *N. Engl. J. Med.* 312:1349–1353, 1985.
30. Byrne, S., and N. McLean. Eating disorders in athletes: a review of the literature. *J. Sci. Med. Sport* 4:145–159, 2001.
31. Byrne, S., and N. McLean. Elite athletes: effects of the pressure to be thin. *J. Sci. Med. Sport* 5:80–94, 2002.
32. Castelo-Branco, C., J. J. Vicente, F. Pons, M. J. Martinez de Osaba, E. Casals, and J. A. Vanrell. Bone mineral density in young, hypothalamic oligoamenorrheic women treated with oral contraceptives. *J. Reprod. Med.* 46:875–879, 2001.
33. Chumlea, W. C., C. M. Schubert, A. F. Roche, H. E. Kulin, P. A. Lee, J. H. Himes, and S. S. Sun. Age at menarche and racial comparisons in US girls. *Pediatrics* 111:110–113, 2003.
34. Cobb, K. L., L. K. Bachrach, G. Greendale, R. Marcus, R. M. Neer, J. Nieves, M. F. Sowers, B. W. Brown Jr, G. Gopalakrishnan, C. Luetters, H. K. Tanner, B. Ward, and J. L. Kelsey. Disordered eating, menstrual irregularity, and bone mineral density in female runners. *Med. Sci. Sports Exerc.* 35:711–719, 2003.
35. Compston, J. E. Sex steroids and bone. *Physiol. Rev.* 81: 419–447, 2001.
36. Compston, J. E., C. McConachie, C. Stott, R. A. Hannon, S. Kaptoge, I. Debiram, S. Love, and A. Jaffa. Changes in bone mineral density, body composition and biochemical markers of bone turnover during weight gain in adolescents with severe anorexia nervosa: a 1-year prospective study. *Osteoporos. Int.* 17:77–84, 2006.
37. Cotton, M. A., C. Ball, and P. Robinson. Four simple questions can help screen for eating disorders. *J. Gen. Intern. Med.* 18: 53–56, 2003.
38. Cumming, D. C. Exercise-associated amenorrhea, low bone density, and estrogen replacement therapy. *Arch. Intern. Med.* 156:2193–2195, 1996.
39. Cumming, D. C., and C. E. Cumming. Estrogen replacement therapy and female athletes: current issues. *Sports Med.* 31:1025–1031, 2001.
40. De Cree, C., R. Lewin, and M. Ostyn. Suitability of cyproterone acetate in the treatment of osteoporosis associated with athletic amenorrhea. *Int. J. Sports Med.* 9:187–192, 1988.
41. De Laet, C. E., B. A. Van Hout, H. Burger, A. E. Weel, A. Hofman, and H. A. Pols. Hip fracture prediction in elderly men and women: validation in the Rotterdam study. *J. Bone Miner. Res.* 13:1587–1593, 1998.
42. De Souza, M. J., B. E. Miller, A. B. Loucks, A. A. Luciano, L. S. Pescatello, C. G. Campbell, and B. L. Lasley. High frequency of luteal phase deficiency and anovulation in recreational women runners: blunted elevation in follicle-stimulating hormone observed during luteal-follicular transition. *J. Clin. Endocrinol. Metab.* 83:4220–4232, 1998.
43. De Souza, M. J., and N. I. Williams. Physiological aspects and clinical sequelae of energy deficiency and hypoestrogenism in exercising women. *Hum. Reprod. Update* 10:433–448, 2004.
44. Deuster, P. A., S. B. Kyle, P. B. Moser, R. A. Vigersky, A. Singh, and E. B. Schoomaker. Nutritional intakes and status of highly trained amenorrheic and eumenorrheic women runners. *Fertil. Steril.* 46:636–643, 1986.
45. Drinkwater, B. L., B. Bruemner, and C. H. Chesnut 3rd. Menstrual history as a determinant of current bone density in young athletes. *JAMA* 263:545–548, 1990.
46. Drinkwater, B. L., K. Nilson, C. H. Chesnut 3rd, W. J. Bremner, S. Shainholtz, and M. B. Southworth. Bone mineral content of amenorrheic and eumenorrheic athletes. *N. Engl. J. Med.* 311:277–281, 1984.
47. Drinkwater, B. L., K. Nilson, S. Ott, and C. H. Chesnut 3rd. Bone mineral density after resumption of menses in amenorrheic athletes. *JAMA* 256:380–382, 1986.
48. Ducy, P., T. Schinke, and G. Karsenty. The osteoblast: a sophisticated fibroblast under central surveillance. *Science* 289:1501–1504, 2000.
49. Dueck, C. A., K. S. Matt, M. M. Manore, and J. S. Skinner. Treatment of athletic amenorrhea with a diet and training intervention program. *Int. J. Sport Nutr.* 6:24–40, 1996.

50. Dugowson, C. E., B. L. Drinkwater, and J. M. Clark. Nontraumatic femur fracture in an oligomenorrheic athlete. *Med. Sci. Sports Exerc.* 23:1323–1325, 1991.
51. Dusek, T. Influence of high intensity training on menstrual cycle disorders in athletes. *Croat. Med. J.* 42:79–82, 2001.
52. Ebell, M. H., J. Siwek, B. D. Weiss, S. H. Woolf, J. Susman, B. Ewigman, and M. Bowman. Strength of recommendation taxonomy (SORT): a patient-centered approach to grading evidence in the medical literature. *Am. Fam. Physician* 69:548–556, 2004.
53. Ellison, P. T., and C. Lager. Moderate recreational running is associated with lowered salivary progesterone profiles in women. *Am. J. Obstet. Gynecol.* 154:1000–1003, 1986.
54. Falsetti, L., A. Gambera, L. Barbetti, and C. Specchia. Long-term follow-up of functional hypothalamic amenorrhea and prognostic factors. *J. Clin. Endocrinol. Metab.* 87:500–505, 2002.
55. Fehling, P. C., L. Alekel, J. Clasey, A. Rector, and R. J. Stillman. A comparison of bone mineral densities among female athletes in impact loading and active loading sports. *Bone* 17:205–210, 1995.
56. Filicori, M., C. Tabarelli, P. Casadio, F. Ferlini, G. Gessa, P. Pocognoli, G. Cognigni, and R. Pecorari. Interaction between menstrual cyclicity and gonadotropin pulsatility. *Horm. Res.* 49:169–172, 1998.
57. Fogelholm, G. M., T. K. Kukkonen-Harjula, S. A. Taipale, H. T. Sievanen, P. Oja, and I. M. Vuori. Resting metabolic rate and energy intake in female gymnasts, figure-skaters and soccer players. *Int. J. Sports Med.* 16:551–556, 1995.
58. Fredericson, M., and K. Kent. Normalization of bone density in a previously amenorrheic runner with osteoporosis. *Med. Sci. Sports Exerc.* 37:1481–1486, 2005.
59. Friday, K. E., B. L. Drinkwater, B. Bruemmer, C. Chesnut 3rd, and A. Chait. Elevated plasma low-density lipoprotein and high-density lipoprotein cholesterol levels in amenorrheic athletes: effects of endogenous hormone status and nutrient intake. *J. Clin. Endocrinol. Metab.* 77:1605–1609, 1993.
60. Frusztajer, N. T., S. Dhuper, M. P. Warren, J. Brooks-Gunn, and R. P. Fox. Nutrition and the incidence of stress fractures in ballet dancers. *Am. J. Clin. Nutr.* 51:779–783, 1990.
61. Gibson, J. H., A. Mitchell, J. Reeve, and M. G. Harries. Treatment of reduced bone mineral density in athletic amenorrhea: a pilot study. *Osteoporos. Int.* 10:284–289, 1999.
62. Golden, N. H., D. K. Katzman, R. E. Kreipe, S. L. Stevens, S. M. Sawyer, J. Rees, D. Nicholls, and E. S. Rome. Eating disorders in adolescents: position paper of the Society for Adolescent Medicine. *J. Adolesc. Health* 33:496–503, 2003.
63. Golden, N. H., L. Lanzkowsky, J. Schebendach, C. J. Palestro, M. S. Jacobson, and I. R. Shenker. The effect of estrogen-progestin treatment on bone mineral density in anorexia nervosa. *J. Pediatr. Adolesc. Gynecol.* 15:135–143, 2002.
64. Gordon, C. M., E. Grace, S. J. Emans, H. A. Feldman, E. Goodman, K. A. Becker, C. J. Rosen, C. M. Gundberg, and M. S. LeBoff. Effects of oral dehydroepiandrosterone on bone density in young women with anorexia nervosa: a randomized trial. *J. Clin. Endocrinol. Metab.* 87:4935–4941, 2002.
65. Greer, F. R., and N. F. Krebs. Optimizing bone health and calcium intakes of infants, children, and adolescents. *Pediatrics* 117:578–585, 2006.
66. Gremion, G., R. Rizzoli, D. Slosman, G. Theintz, and J. P. Bonjour. Oligo-amenorrheic long-distance runners may lose more bone in spine than in femur. *Med. Sci. Sports Exerc.* 33:15–21, 2001.
67. Grinspoon, S., D. Herzog, and A. Klibanski. Mechanisms and treatment options for bone loss in anorexia nervosa. *Psychopharmacol Bull.* 33:399–404, 1997.
68. Grinspoon, S., L. Thomas, K. Miller, D. Herzog, and A. Klibanski. Effects of recombinant human IGF-I and oral contraceptive administration on bone density in anorexia nervosa. *J. Clin. Endocrinol. Metab.* 87:2883–2891, 2002.
69. Gulekli, B., M. C. Davies, and H. S. Jacobs. Effect of treatment on established osteoporosis in young women with amenorrhoea. *Clin. Endocrinol. (Oxf)* 41:275–281, 1994.
70. Hach, I., U. E. Ruhl, A. Rentsch, E. S. Becker, V. Turke, J. Margraf, and W. Kirch. Recognition and therapy of eating disorders in young women in primary care. *J. Public Health (Springer-Verlag)* 13:160–165, 2005.
71. Haenggi, W., J. P. Casez, M. H. Birkhaeuser, K. Lippuner, and P. Jaeger. Bone mineral density in young women with long-standing amenorrhea: limited effect of hormone replacement therapy with ethinylestradiol and desogestrel. *Osteoporos. Int.* 4:99–103, 1994.
72. Hall, J. E., and L. Nieman. *Handbook of Diagnostic Endocrinology*, Totowa, N.J.: Humana, 2003.
73. Hammar, M. L., M. B. Hammar-Henriksson, J. Frisk, A. Rickenlund, and Y. A. Wyon. Few oligo-amenorrheic athletes have vasomotor symptoms. *Maturitas* 34:219–225, 2000.
74. Hans, D., R. W. Downs Jr, F. Duboeuf, S. Greenspan, L. G. Jankowski, G. M. Kiebzak, and S. M. Petak. Skeletal sites for osteoporosis diagnosis: the 2005 ISCD Official Positions. *J. Clin. Densitom.* 9:15–21, 2006.
75. Harber, V. J., S. R. Petersen, and P. D. Chilibeck. Thyroid hormone concentrations and muscle metabolism in amenorrheic and eumenorrheic athletes. *Can. J. Appl. Physiol.* 23: 293–306, 1998.
76. Hergenroeder, A. C., E. O. Smith, R. Shypailo, L. A. Jones, W. J. Klish, and K. Ellis. Bone mineral changes in young women with hypothalamic amenorrhea treated with oral contraceptives, medroxyprogesterone, or placebo over 12 months. *Am. J. Obstet. Gynecol.* 176:1017–1025, 1997.
77. Herzog, D. B., D. J. Dorer, P. K. Keel, S. E. Selwyn, E. R. Ekeblad, A. T. Flores, D. N. Greenwood, R. A. Burwell, and M. B. Keller. Recovery and relapse in anorexia and bulimia nervosa: a 7.5-year follow-up study. *J. Am. Acad. Child Adolesc. Psychiatry* 38:829–837, 1999.
78. Hoch, A. Z., R. L. Dempsey, G. F. Carrera, C. R. Wilson, E. H. Chen, V. M. Barnabei, P. R. Sandford, T. A. Ryan, and D. D. Gutterman. Is there an association between athletic amenorrhea and endothelial cell dysfunction? *Med. Sci. Sports Exerc.* 35:377–383, 2003.
79. Holehan, A. M., and B. J. Merry. The control of puberty in the dietary restricted female rat. *Mech. Ageing Dev.* 32:179–191, 1985.
80. Horvath, P. J., C. K. Eagen, N. M. Fisher, J. J. Leddy, and D. R. Pendergast. The effects of varying dietary fat on performance and metabolism in trained male and female runners. *J. Am. Coll. Nutr.* 19:52–60, 2000.
81. Horvath, P. J., C. K. Eagen, S. D. Ryer-Calvin, and D. R. Pendergast. The effects of varying dietary fat on the nutrient intake in male and female runners. *J. Am. Coll. Nutr.* 19:42–51, 2000.
82. Hosmer, W. D., H. K. Genant, and W. S. Browner. Fractures before menopause: a red flag for physicians. *Osteoporos. Int.* 13:337–341, 2002.
83. Hotta, M., T. Shibasaki, K. Sato, and H. Demura. The importance of body weight history in the occurrence and recovery of osteoporosis in patients with anorexia nervosa: evaluation by dual X-ray absorptiometry and bone metabolic markers. *Eur. J. Endocrinol.* 139:276–283, 1998.
84. Hubert, P., N. A. King, and J. E. Blundell. Uncoupling the effects of energy expenditure and energy intake: appetite response to short-term energy deficit induced by meal omission and physical activity. *Appetite* 31:9–19, 1998.

85. Hui, S. L., C. W. Slemenda, and C. C. Johnston Jr. Baseline measurement of bone mass predicts fracture in white women. *Ann. Intern. Med.* 111:355–361, 1989.
86. Ihle, R., and A. B. Loucks. Dose-response relationships between energy availability and bone turnover in young exercising women. *J. Bone Miner. Res.* 19:1231–1240, 2004.
87. Iketani, T., N. Kiriike, S. Nakanishi, and T. Nakasuji. Effects of weight gain and resumption of menses on reduced bone density in patients with anorexia nervosa. *Biol. Psychiatry* 37:521–527, 1995.
88. Institute of Medicine Food and Nutrition Board. *Dietary Reference Intakes. Calcium, Phosphorus, Magnesium, Vitamin D, and Fluoride*, Washington, DC: National Academy Press, 1997.
89. International Olympic Committee Medical Commission Working Group Women in Sport. Position Stand on the female athlete triad. Available at: http://multimedia.olympic.org/pdf/en_report_917.pdf Accessed December 16, 2006.
90. International Society for Clinical Densitometry Writing Group for the ISCD Position Development Conference. Diagnosis of osteoporosis in men, women, and children. *J. Clin. Densitom.* 7:17–26, 2004.
91. Jenkins, P. J., X. Ibanez-Santos, J. Holly, A. Cotterill, L. Perry, R. Wolman, M. Harries, and A. Grossman. IGFBP-1: a metabolic signal associated with exercise-induced amenorrhoea. *Neuroendocrinology* 57:600–604, 1993.
92. Johnson, C., P. S. Powers, and R. Dick. Athletes and eating disorders: the National Collegiate Athletic Association study. *Int. J. Eat Disord.* 26:179–188, 1999.
93. Kaiserauer, S., A. C. Snyder, M. Sleeper, and J. Zierath. Nutritional, physiological, and menstrual status of distance runners. *Med. Sci. Sports Exerc.* 21:120–125, 1989.
94. Kalu, D. N., E. J. Masoro, B. P. Yu, R. R. Hardin, and B. W. Hollis. Modulation of age-related hyperparathyroidism and senile bone loss in Fischer rats by soy protein and food restriction. *Endocrinology* 122:1847–1854, 1988.
95. Kanis, J. A. Diagnosis of osteoporosis and assessment of fracture risk. *Lancet* 359:1929–1936, 2002.
96. Kanis, J. A., L. J. Melton 3rd, C. Christiansen, C. C. Johnston, and N. Khaltaev. The diagnosis of osteoporosis. *J. Bone Miner. Res.* 9:1137–1141, 1994.
97. Kaye, W. H., C. M. Bulik, L. Thornton, N. Barbarich, and K. Masters. Comorbidity of anxiety disorders with anorexia and bulimia nervosa. *Am. J. Psychiatry* 161:2215–2221, 2004.
98. Kaye, W. H., and T. E. Weltzin. Neurochemistry of bulimia nervosa. *J. Clin. Psychiatry* 52 Suppl:21–28, 1991.
99. Keen, A. D., and B. L. Drinkwater. Irreversible bone loss in former amenorrheic athletes. *Osteoporos. Int.* 7:311–315, 1997.
100. Khan, A. A., L. Bachrach, J. P. Brown, D. A. Hanley, R. G. Josse, D. L. Kendler, E. S. Leib, B. C. Lentle, W. D. Leslie, E. M. Lewiecki, P. D. Miller, R. L. Nicholson, C. O'Brien, W. P. Olszynski, M. Y. Theriault, and N. B. Watts. Standards and guidelines for performing central dual-energy x-ray absorptiometry in premenopausal women, men, and children. *J. Clin. Densitom.* 7:51–64, 2004.
101. Khan, A. A., J. Brown, K. Faulkner, D. Kendler, B. Lentle, W. Leslie, P. D. Miller, L. Nicholson, W. P. Olszynski, N. B. Watts, D. Hanley, A. Hodsman, R. Josse, T. M. Murray, and K. Yuen. Standards and guidelines for performing central dual X-ray densitometry from the Canadian panel of International Society for Clinical Densitometry. *J. Clin. Densitom.* 5:247–257, 2002.
102. Khan, A. A., D. A. Hanley, J. P. Bilezikian, N. Binkley, J. P. Brown, A. B. Hodsman, R. G. Josse, D. L. Kendler, E. M. Lewiecki, P. D. Miller, W. P. Olszynski, S. M. Petak, Z. A. Syed, D. Theriault, and N. B. Watts. Standards for performing DXA in individuals with secondary causes of osteoporosis. *J. Clin. Densitom.* 9:47–57, 2006.
103. Khan, K., H. A. McKay, H. Haapasalo, K. L. Bennell, M. R. Forwood, P. Kannus, and J. D. Wark. Does childhood and adolescence provide a unique opportunity for exercise to strengthen the skeleton? *J. Sci. Med. Sport* 3:150–164, 2000.
104. Khan, K. M., T. Liu-Ambrose, M. M. Sran, M. C. Ashe, M. G. Donaldson, and J. D. Wark. New criteria for female athlete triad syndrome? As osteoporosis is rare, should osteopenia be among the criteria for defining the female athlete triad syndrome? *Br. J. Sports Med.* 36:10–13, 2002.
105. Klibanski, A., B. M. Biller, D. A. Schoenfeld, D. B. Herzog, and V. C. Saxe. The effects of estrogen administration on trabecular bone loss in young women with anorexia nervosa. *J. Clin. Endocrinol. Metab.* 80:898–904, 1995.
106. Kohrt, W. M., S. A. Bloomfield, K. D. Little, M. E. Nelson, and V. R. Yingling. American College of Sports Medicine Position Stand: physical activity and bone health. *Med. Sci. Sports Exerc.* 36:1985–1996, 2004.
107. Kondo, D. G., and M. S. Sokol. Eating disorders in primary care. A guide to identification and treatment. *Postgrad. Med.* 119:59–65, 2006.
108. Kopp-Woodroffe, S. A., M. M. Manore, C. A. Dueck, J. S. Skinner, and K. S. Matt. Energy and nutrient status of amenorrheic athletes participating in a diet and exercise training intervention program. *Int. J. Sport Nutr.* 9:70–88, 1999.
109. Kreipe, R. E., D. G. Hicks, R. N. Rosier, and J. E. Puzas. Preliminary findings on the effects of sex hormones on bone metabolism in anorexia nervosa. *J. Adolesc. Health* 14:319–324, 1993.
110. Kreipe, R. E., and S. M. Yussman. The role of the primary care practitioner in the treatment of eating disorders. *Adolesc. Med.* 14:133–147, 2003.
111. LaBan, M. M., J. C. Wilkins, A. H. Sackeyfio, and R. S. Taylor. Osteoporotic stress fractures in anorexia nervosa: etiology, diagnosis, and review of four cases. *Arch. Phys. Med. Rehabil.* 76:884–887, 1995.
112. Lauder, T. D., S. Dixit, L. E. Pezzin, M. V. Williams, C. S. Campbell, and G. D. Davis. The relation between stress fractures and bone mineral density: evidence from active-duty Army women. *Arch. Phys. Med. Rehabil.* 81:73–79, 2000.
113. Laughlin, G. A., and S. S. C. Yen. Hypoleptinemia in women athletes: absence of a diurnal rhythm with amenorrhea. *J. Clin. Endocrinol. Metab.* 82:318–321, 1997.
114. Laughlin, G. A., and S. S. C. Yen. Nutritional and endocrine-metabolic aberrations in amenorrheic athletes. *J. Clin. Endocrinol. Metab.* 81:4301–4309, 1996.
115. Leib, E. S. Treatment of low bone mass in premenopausal women: when may it be appropriate? *Curr. Osteoporos. Rep.* 3:13–18, 2005.
116. Leib, E. S., E. M. Lewiecki, N. Binkley, and R. C. Hamdy. Official positions of the International Society for Clinical Densitometry. *J. Clin. Densitom.* 7:1–6, 2004.
117. Leslie, W. D., R. A. Adler, G. El-Hajj Fuleihan, A. B. Hodsman, D. L. Kendler, M. McClung, P. D. Miller, and N. B. Watts. Application of the 1994 WHO classification to populations other than postmenopausal Caucasian women: the 2005 ISCD Official Positions. *J. Clin. Densitom.* 9:22–30, 2006.
118. Lindberg, J. S., M. R. Powell, M. M. Hunt, D. E. Ducey, and C. E. Wade. Increased vertebral bone mineral in response to reduced exercise in amenorrheic runners. *West. J. Med.* 146: 39–42, 1987.
119. Liu, S. L., and C. M. Lebrun. Effect of oral contraceptives and hormone replacement therapy on bone mineral density in

premenopausal and perimenopausal women: a systematic review. *Br. J. Sports Med*. 40:11–24, 2006.
120. Lloyd, T., C. Myers, J. R. Buchanan, and L. M. Demers. Collegiate women athletes with irregular menses during adolescence have decreased bone density. *Obstet. Gynecol*. 72:639–642, 1988.
121. Loucks, A. B., J. F. Mortola, L. Girton, and S. S. C. Yen. Alterations in the hypothalamic-pituitary-ovarian and the hypothalamic-pituitary-adrenal axes in athletic women. *J. Clin. Endocrinol. Metab*. 68:402–411, 1989.
122. Loucks, A. B., and J. R. Thuma. Luteinizing hormone pulsatility is disrupted at a threshold of energy availability in regularly menstruating women. *J. Clin. Endocrinol. Metab*. 88:297–311, 2003.
123. Loucks, A. B., M. Verdun, and E. M. Heath. Low energy availability, not stress of exercise, alters LH pulsatility in exercising women. *J. Appl. Physiol*. 84:37–46, 1998.
124. Luck, A. J., J. F. Morgan, F. Reid, A. O'Brien, J. Brunton, C. Price, L. Perry, and J. H. Lacey. The SCOFF questionnaire and clinical interview for eating disorders in general practice: comparative study. *BMJ* 325:755–756, 2002.
125. Manore, M. M. Nutritional needs of the female athlete. *Clin. Sports Med*. 18:549–563, 1999.
126. Manore, M. M., S. I. Barr, and G. A. Butterfield. Nutrition and athletic performance. A joint position statement of the American Dietetic Association, the Dietitians of Canada, and the American College of Sports Medicine. *Med. Sci. Sports Exerc*. 32:2130–2145, 2000.
127. Marcus, R., C. Cann, P. Madvig, J. Minkoff, M. Goddard, M. Bayer, M. Martin, L. Gaudiani, W. Haskell, and H. Genant. Menstrual function and bone mass in elite women distance runners. Endocrine and metabolic features. *Ann. Intern. Med*. 102:158–163, 1985.
128. Matejek, N., E. Weimann, C. Witzel, G. Molenkamp, S. Schwidergall, and H. Bohles. Hypoleptinaemia in patients with anorexia nervosa and in elite gymnasts with anorexia athletica. *Int. J. Sports Med*. 20:451–456, 1999.
129. Matsumoto, S., Y. Nogami, and S. Ohkuri. Statistical studies of menstruation: a criticism on the definition of normal menstruation. *Gunma. J. Med. Sci*. 11:294–318, 1962.
130. Maugars, Y., J. M. Berthelot, S. Lalande, C. Charlier, and A. Prost. Osteoporotic fractures revealing anorexia nervosa in five females. *Rev. Rhum. Engl. Ed*. 63:201–206, 1996.
131. McShane, T. M., and P. M. Wise. Life-long moderate caloric restriction prolongs reproductive life span in rats without interrupting estrous cyclicity: effects on the gonadotropin-releasing hormone/luteinizing hormone axis. *Biol. Reprod*. 54:70–75, 1996.
132. Mehler, P. S. Diagnosis and care of patients with anorexia nervosa in primary care settings. *Ann. Intern. Med*. 134:1048–1059, 2001.
133. Merry, B. J., and A. M. Holehan. Onset of puberty and duration of fertility in rats fed a restricted diet. *J. Reprod. Fertil*. 57:253–259, 1979.
134. Miller, K. K., and A. Klibanski. Clinical review 106: Amenorrheic bone loss. *J. Clin. Endocrinol. Metab*. 84:1775–1783, 1999.
135. Miller, K. K., E. E. Lee, E. A. Lawson, M. Misra, J. Minihan, S. K. Grinspoon, S. Gleysteen, D. Mickley, D. Herzog, and A. Klibanski. Determinants of skeletal loss and recovery in anorexia nervosa. *J. Clin. Endocrinol. Metab*. 91:2931–2937, 2006.
136. Miller, K. K., M. S. Parulekar, E. Schoenfeld, E. Anderson, J. Hubbard, A. Klibanski, and S. K. Grinspoon. Decreased leptin levels in normal weight women with hypothalamic amenorrhea: the effects of body composition and nutritional intake. *J. Clin. Endocrinol. Metab*. 83:2309–2312, 1998.
137. Minuchin, S., L. Baker, and B. L. Rosman. *Psychosomatic Families: Anorexia Nervosa in Context*, Cambridge, Mass: Harvard University Press, 1978.
138. Misra, M., and A. Klibanski. Anorexia nervosa and osteoporosis. *Rev Endocr Metab Disord*, 2006.
139. Morgan, J. F., F. Reid, and J. H. Lacey. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. *BMJ* 319:1467–1468, 1999.
140. Mosekilde, L., J. S. Thomsen, P. B. Orhii, R. J. McCarter, W. Mejia, and D. N. Kalu. Additive effect of voluntary exercise and growth hormone treatment on bone strength assessed at four different skeletal sites in an aged rat model. *Bone* 24:71–80, 1999.
141. Mulligan, K., and G. E. Butterfield. Discrepancies between energy intake and expenditure in physically active women. *Br. J. Nutr*. 64:23–36, 1990.
142. Munoz, M. T., G. Morande, J. A. Garcia-Centenera, F. Hervas, J. Pozo, and J. Argente. The effects of estrogen administration on bone mineral density in adolescents with anorexia nervosa. *Eur. J. Endocrinol*. 146:45–50, 2002.
143. Myburgh, K. H., L. K. Bachrach, B. Lewis, K. Kent, and R. Marcus. Low bone mineral density at axial and appendicular sites in amenorrheic athletes. *Med. Sci. Sports Exerc*. 25:1197–1202, 1993.
144. Myburgh, K. H., J. Hutchins, A. B. Fataar, S. F. Hough, and T. D. Noakes. Low bone density is an etiologic factor for stress fractures in athletes. *Ann. Intern. Med*. 113:754–759, 1990.
145. Myerson, M., B. Gutin, M. P. Warren, M. T. May, I. Contento, M. Lee, F. X. PiSunyer, R. N. Pierson Jr., and J. Brooks-Gunn. Resting metabolic rate and energy balance in amenorrheic and eumenorrheic runners. *Med. Sci. Sports Exerc*. 23:15–22, 1991.
146. National Institutes of Health Consensus Development Panel. Osteoporosis prevention, diagnosis, and therapy. *JAMA* 285: 785–795, 2001.
147. National Osteoporosis Foundation. *Physician's Guide to Prevention and Treatment of Osteoporosis*, Washington, DC: National Osteoporosis Foundation, 2003.
148. Nattiv, A., R. Agostini, B. Drinkwater, and K. K. Yeager. The female athlete triad. The inter-relatedness of disordered eating, amenorrhea, and osteoporosis. *Clin. Sports Med*. 13:405–418, 1994.
149. Nattiv, A., J. C. Puffer, J. Casper, F. Dorey, J. M. Kabo, S. Hame, K. Fulton, E. Moore, and G. A. Finerman. Stress fracture risk factors, incidence and distribution: a 3-year prospective study in collegiate runners. *Med. Sci. Sports Exerc*. 32:S347, 2000.
150. Nelson, M. E., E. C. Fisher, P. D. Catsos, C. N. Meredith, R. N. Turksoy, and W. J. Evans. Diet and bone status in amenorrheic runners. *Am. J. Clin. Nutr*. 43:910–916, 1986.
151. Nichols, D. L., and C. F. Sanborn. Female Athlete and Bone. In: *Nutrition for Sport and Exercise*, J. R. Berning and S. N. Steen. Gaithersburg, Md.: Aspen Publishers, pp. 205–215, 1998.
152. Nichols, J. F., M. J. Rauh, M. J. Lawson, M. Ji, and H. S. Barkai. Prevalence of the female athlete triad syndrome among high school athletes. *Arch. Pediatr. Adolesc. Med*. 160:137–142, 2006.
153. Nielsen, S., S. Moller-Madsen, T. Isager, J. Jorgensen, K. Pagsberg, and S. Theander. Standardized mortality in eating disorders—a quantitative summary of previously published and new evidence. *J. Psychosom. Res*. 44:413–434, 1998.
154. O'Donnell, E., and M. J. De Souza. The cardiovascular effects of chronic hypoestrogenism in amenorrhoeic athletes: a critical review. *Sports Med*. 34:601–627, 2004.
155. Otis, C. L., B. Drinkwater, M. Johnson, A. Loucks, and J. Wilmore. American College of Sports Medicine Position Stand: The female athlete triad. *Med. Sci. Sports Exerc*. 29:i–ix, 1997.
156. Patlas, N., G. Golomb, P. Yaffe, T. Pinto, E. Breuer, and A. Ornoy. Transplacental effects of bisphosphonates on fetal skeletal ossification and mineralization in rats. *Teratology* 60: 68–73, 1999.

157. Patton, G. C., R. Selzer, C. Coffey, J. B. Carlin, and R. Wolfe. Onset of adolescent eating disorders: population based cohort study over 3 years. *BMJ* 318:765–768, 1999.
158. Petrie, T. A., and S. Stoever. The incidence of bulimia nervosa and pathogenic weight control behaviors in female collegiate gymnasts. *Res. Q. Exerc. Sport* 64:238–241, 1993.
159. Pettersson, F., H. Fries, and S. J. Nillius. Epidemiology of secondary amenorrhea. I. Incidence and prevalence rates. *Am. J. Obstet. Gynecol.* 117:80–86, 1973.
160. Pritts, S. D., and J. Susman. Diagnosis of eating disorders in primary care. *Am. Fam. Physician* 67:297–304, 2003.
161. Redman, L. M., and A. B. Loucks. Menstrual disorders in athletes. *Sports Med.* 35:747–755, 2005.
162. Rencken, M. L., C. H. Chesnut 3rd, and B. L. Drinkwater. Bone density at multiple skeletal sites in amenorrheic athletes. *JAMA* 276:238–240, 1996.
163. Rickenlund, A., K. Carlstrom, B. Ekblom, T. B. Brismar, B. Von Schoultz, and A. L. Hirschberg. Effects of oral contraceptives on body composition and physical performance in female athletes. *J. Clin. Endocrinol. Metab.* 89:4364–4370, 2004.
164. Rigotti, N. A., R. M. Neer, S. J. Skates, D. B. Herzog, and S. R. Nussbaum. The clinical course of osteoporosis in anorexia nervosa. A longitudinal study of cortical bone mass. *JAMA* 265:1133–1138, 1991.
165. Risser, W. L., E. J. Lee, A. LeBlanc, H. B. Poindexter, J. M. Risser, and V. Schneider. Bone density in eumenorrheic female college athletes. *Med. Sci. Sports Exerc.* 22:570–574, 1990.
166. Robinson, T. L., C. Snow-Harter, D. R. Taaffe, D. Gillis, J. Shaw, and R. Marcus. Gymnasts exhibit higher bone mass than runners despite similar prevalence of amenorrhea and oligomenorrhea. *J. Bone Miner. Res.* 10:26–35, 1995.
167. Rome, E. S., S. Ammerman, D. S. Rosen, R. J. Keller, J. Lock, K. A. Mammel, J. O'Toole, J. M. Rees, M. J. Sanders, S. M. Sawyer, M. Schneider, E. Sigel, and T. J. Silber. Children and adolescents with eating disorders: the state of the art. *Pediatrics* 111:e98–e108, 2003.
168. Root, M. P. P. Persistent, disordered eating as a gender-specific, post-traumatic stress response to sexual assault. *Psychother.-Theor. Res.* 28:96–102, 1991.
169. Rose, M., M. Hildebrandt, F. Schoeneich, G. Danzer, and B. F. Klapp. Severe anorexia nervosa associated with osteoporotic-linked femural neck fracture and pulmonary tuberculosis: a case report. *Int. J. Eat Disord.* 25:463–467, 1999.
170. Rosen, D. S., and D. O. Hough. Pathogenic weight-control behavior of female college gymnasts. *Phys Sportsmed.* 16: 141–144, 1988.
171. Sanborn, C. F., B. H. Albrecht, and W. W. Wagner Jr. Athletic amenorrhea: lack of association with body fat. *Med. Sci. Sports Exerc.* 19:207–212, 1987.
172. Sanborn, C. F., B. J. Martin, and W. W. Wagner Jr. Is athletic amenorrhea specific to runners? *Am. J. Obstet. Gynecol.* 143:859–861, 1982.
173. Sanderson, J. P., N. Binkley, E. B. Roecker, J. E. Champ, T. D. Pugh, L. Aspnes, and R. Weindruch. Influence of fat intake and caloric restriction on bone in aging male rats. *J. Gerontol. A Biol. Sci. Med. Sci.* 52:B20–B25, 1997.
174. Schneider, J. E., and G. N. Wade. Inhibition of reproduction in service of energy balance. In: *Reproduction in Context: Social and Environmental Influences on Reproductive Physiology and Behavior*, K. Wallen and J. E. Schneider eds. Cambridge: The MIT Press, pp. 35–82, 2000.
175. Singh, K. B. Menstrual disorders in college students. *Am. J. Obstet. Gynecol.* 140:299–302, 1981.
176. Snow, C. M., C. J. Rosen, and T. L. Robinson. Serum IGF-I is higher in gymnasts than runners and predicts bone and lean mass. *Med. Sci. Sports Exerc.* 32:1902–1907, 2000.
177. Speroff, L., and M. A. Fritz. *Clinical Gynecologic Endocrinology and Infertility*, 7th ed, Philadelphia: Lippincott Williams & Wilkins, 2005.
178. Stager, J. M., J. K. Wigglesworth, and L. K. Hatler. Interpreting the relationship between age of menarche and prepubertal training. *Med. Sci. Sports Exerc.* 22:54–58, 1990.
179. Strober, M., R. Freeman, C. Lampert, J. Diamond, and W. Kaye. Controlled family study of anorexia nervosa and bulimia nervosa: evidence of shared liability and transmission of partial syndromes. *Am. J. Psychiatry* 157:393–401, 2000.
180. Strokosch, G. R., A. J. Friedman, S. C. Wu, and M. Kamin. Effects of an oral contraceptive (norgestimate/ethinyl estradiol) on bone mineral density in adolescent females with anorexia nervosa: a double-blind, placebo-controlled study. *J. Adolesc. Health* 39:819–827, 2006.
181. Stubbs, R. J., D. A. Hughes, A. M. Johnstone, S. Whybrow, G. W. Horgan, N. King, and J. Blundell. Rate and extent of compensatory changes in energy intake and expenditure in response to altered exercise and diet composition in humans. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 286:R350–R358, 2004.
182. Sundgot-Borgen, J. Nutrient intake of female elite athletes suffering from eating disorders. *Int. J. Sport Nutr.* 3:431–442, 1993.
183. Sundgot-Borgen, J. Prevalence of eating disorders in elite female athletes. *Int. J. Sport Nutr.* 3:29–40, 1993.
184. Sundgot-Borgen, J. Risk and trigger factors for the development of eating disorders in female elite athletes. *Med. Sci. Sports Exerc.* 26:414–419, 1994.
185. Sundgot-Borgen, J. Weight and eating disorders in elite athletes. *Scand. J. Med. Sci. Sports* 12:259–260, 2002.
186. Sundgot-Borgen, J., and M. K. Torstveit. Prevalence of eating disorders in elite athletes is higher than in the general population. *Clin. J. Sport Med.* 14:25–32, 2004.
187. Talbott, S. M., M. M. Rothkopf, and S. A. Shapses. Dietary restriction of energy and calcium alters bone turnover and density in younger and older female rats. *J. Nutr.* 128:640–645, 1998.
188. Tarnopolsky, M. Protein requirements for endurance athletes. *Nutrition* 20:662–668, 2004.
189. Thompson, J., and M. M. Manore. Predicted and measured resting metabolic rate of male and female endurance athletes. *J. Am. Diet. Assoc.* 96:30–34, 1996.
190. Thong, F. S., C. McLean, and T. E. Graham. Plasma leptin in female athletes: relationship with body fat, reproductive, nutritional, and endocrine factors. *J. Appl. Physiol.* 88:2037–2044, 2000.
191. Torstveit, M. K., and J. Sundgot-Borgen. The female athlete triad exists in both elite athletes and controls. *Med. Sci. Sports Exerc.* 37:1449–1459, 2005.
192. Treasure, J. L., and G. F. Russell. Intrauterine growth and neonatal weight gain in babies of women with anorexia nervosa. *Br. Med. J. (Clin. Res. Ed.)* 296:1038, 1988.
193. Trumbo, P., S. Schlicker, A. A. Yates, and M. Poos. Dietary reference intakes for energy, carbohydrate, fiber, fat, fatty acids, cholesterol, protein and amino acids. *J. Am. Diet. Assoc.* 102:1621–1630, 2002.
194. Trumbo, P., A. A. Yates, S. Schlicker, and M. Poos. Dietary reference intakes: vitamin A, vitamin K, arsenic, boron, chromium, copper, iodine, iron, manganese, molybdenum, nickel, silicon, vanadium, and zinc. *J. Am. Diet. Assoc.* 101:294–301, 2001.
195. Truswell, A. S. Energy balance, food and exercise. *World Rev. Nutr. Diet* 90:13–25, 2001.
196. United States Dept. of Health and Human Services, United States Dept. of Agriculture, and United States Dietary Guidelines Advisory Committee. *Dietary Guidelines for Americans,*

2005, 6th ed, Washington, D.C.: U.S. Dept. of Health and Human Services, U.S. Dept. of Agriculture, 2005.
197. VOLLMAN, R. F. *The Menstrual Cycle*, Philadelphia: W.B. Saunders Company, p. 193, 1977.
198. WADE, G. N., and J. E. JONES. Neuroendocrinology of nutritional infertility. *Am. J. Physiol. Regul. Integr. Comp. Physiol.* 287:R1277–R1296, 2004.
199. WADE, G. N., J. E. SCHNEIDER, and H. Y. LI. Control of fertility by metabolic cues. *Am. J. Physiol.* 270:E1–E19, 1996.
200. WARREN, M. P. The effects of exercise on pubertal progression and reproductive function in girls. *J. Clin. Endocrinol. Metab.* 51:1150–1157, 1980.
201. WARREN, M. P., J. BROOKS-GUNN, R. P. FOX, C. C. HOLDERNESS, E. P. HYLE, and W. G. HAMILTON. Osteopenia in exercise-associated amenorrhea using ballet dancers as a model: a longitudinal study. *J. Clin. Endocrinol. Metab.* 87:3162–3168, 2002.
202. WARREN, M. P., J. BROOKS-GUNN, R. P. FOX, C. C. HOLDERNESS, E. P. HYLE, W. G. HAMILTON, and L. HAMILTON. Persistent osteopenia in ballet dancers with amenorrhea and delayed menarche despite hormone therapy: a longitudinal study. *Fertil. Steril.* 80:398–404, 2003.
203. WARREN, M. P., J. BROOKS-GUNN, L. H. HAMILTON, L. F. WARREN, and W. G. HAMILTON. Scoliosis and fractures in young ballet dancers. Relation to delayed menarche and secondary amenorrhea. *N. Engl. J. Med.* 314:1348–1353, 1986.
204. WARREN, M. P., and A. R. HAGEY. The genetics, diagnosis and treatment of amenorrhea. *Minerva Ginecol.* 56:437–455, 2004.
205. WARREN, M. P., K. K. MILLER, W. H. OLSON, S. K. GRINSPOON, and A. J. FRIEDMAN. 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* 72:206–211, 2005.
206. WARREN, M. P., F. VOUSSOUGHIAN, E. B. GEER, E. P. HYLE, C. L. ADBERG, and R. H. RAMOS. Functional hypothalamic amenorrhea: hypoleptinemia and disordered eating. *J. Clin. Endocrinol. Metab.* 84:873–877, 1999.
207. WILLIAMS, N. I., A. L. CASTON-BALDERRAMA, D. L. HELMREICH, D. B. PARFITT, C. NOSBISCH, and J. L. CAMERON. Longitudinal changes in reproductive hormones and menstrual cyclicity in cynomolgus monkeys during strenuous exercise training: abrupt transition to exercise-induced amenorrhea. *Endocrinology* 142:2381–2389, 2001.
208. WILLIAMS, N. I., D. L. HELMREICH, D. B. PARFITT, A. L. CASTON-BALDERRAMA, and J. L. CAMERON. Evidence for a causal role of low energy availability in the induction of menstrual cycle disturbances during strenuous exercise training. *J. Clin. Endocrinol. Metab.* 86:5184–5193, 2001.
209. WILMORE, J. H., K. C. WAMBSGANS, M. BRENNER, C. E. BROEDER, I. PAIJMANS, J. A. VOLPE, and K. M. WILMORE. Is there energy conservation in amenorrheic compared with eumenorrheic distance runners? *J. Appl. Physiol.* 72:15–22, 1992.
210. WILSON, J. H., and R. L. WOLMAN. Osteoporosis and fracture complications in an amenorrhoeic athlete. *Br. J. Rheumatol.* 33:480–481, 1994.
211. WINFIELD, A. C., J. MOORE, M. BRACKER, and C. W. JOHNSON. Risk factors associated with stress reactions in female Marines. *Mil. Med.* 162:698–702, 1997.
212. WINTERS-STONE, K. M., and C. M. SNOW. One year of oral calcium supplementation maintains cortical bone density in young adult female distance runners. *Int. J. Sport Nutr. Exerc. Metab.* 14:7–17, 2004.
213. WU, F., B. MASON, A. HORNE, R. AMES, J. CLEARWATER, M. LIU, M. C. EVANS, G. D. GAMBLE, and I. R. REID. Fractures between the ages of 20 and 50 years increase women's risk of subsequent fractures. *Arch. Intern. Med.* 162:33–36, 2002.
214. YAGER, J., M. J. DEVLIN, K. A. HALMI, D. B. HERZOG, J. E. MITCHELL, P. S. POWERS, and K. J. ZERBE. Guideline watch: Practice guideline for the treatment of patients with eating disorders. Available at: http://www.psych.org/psych_pract/treatg/pg/prac_guide.cfm. Accessed March 28, 2007, 2005.
215. YEAGER, K. K., R. AGOSTINI, A. NATTIV, and B. DRINKWATER. The female athlete triad: disordered eating, amenorrhea, osteoporosis. *Med. Sci. Sports Exerc.* 25:775–777, 1993.
216. YOUNG, D., J. L. HOPPER, C. A. NOWSON, R. M. GREEN, A. J. SHERWIN, B. KAYMAKCI, M. SMID, C. S. GUEST, R. G. LARKINS, and J. D. WARK. Determinants of bone mass in 10- to 26-year-old females: a twin study. *J. Bone Miner. Res.* 10:558–567, 1995.
217. YOUNG, N., C. FORMICA, G. SZMUKLER, and E. SEEMAN. Bone density at weight-bearing and nonweight-bearing sites in ballet dancers: the effects of exercise, hypogonadism, and body weight. *J. Clin. Endocrinol. Metab.* 78:449–454, 1994.
218. ZANKER, C. L., C. B. COOKE, J. G. TRUSCOTT, B. OLDROYD, and H. S. JACOBS. Annual changes of bone density over 12 years in an amenorrheic athlete. *Med. Sci. Sports Exerc.* 36:137–142, 2004.
219. ZANKER, C. L., and I. L. SWAINE. Bone turnover in amenorrhoeic and eumenorrhoeic women distance runners. *Scand. J. Med. Sci. Sports* 8:20–26, 1998.
220. ZANKER, C. L., and I. L. SWAINE. Relation between bone turnover, oestradiol, and energy balance in women distance runners. *Br. J. Sports Med.* 32:167–171, 1998.