



The Female Athlete Triad

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SUMMARY

ACSM Position Stand on the Female Athlete Triad. *Med. Sci. Sports Exerc.*, Vol. 29, No. 5, pp. i-ix, 1997. The Female Athlete Triad is a syndrome occurring in physically active girls and women. Its interrelated components are disordered eating, amenorrhea, and osteoporosis. Pressure placed on young women to achieve or maintain unrealistically low body weight underlies development of the Triad. Adolescents and women training in sports in which low body weight is emphasized for athletic activity or appearance are at greatest risk. Girls and women with one component of the Triad should be screened for the others.

Alone or in combination, Female Athlete Triad disorders can decrease physical performance and cause morbidity and mortality. More research is needed on its causes, prevalence, treatment, and consequences. All individuals working with physically active girls and women should be educated about the Female Athlete Triad and develop plans to prevent, recognize, treat, and reduce its risks.

INTRODUCTION

The majority of girls and women derive significant health benefits from regular physical activity without incurring health risks (50,93,103). They should be encouraged to be physically active at all phases of their lives. If injuries or medical problems develop, they should be promptly identified and treated by professionals knowledgeable in the care of active women.

In 1992 the Female Athlete Triad was the focus of a consensus conference called by The Task Force on Women's Issues of the American College of Sports Medicine (141). The three components of the Triad are:

1. Disordered eating. Disordered eating refers to a wide spectrum of harmful and often ineffective eating behaviors used in attempts to lose weight or achieve a lean appearance. The spectrum of behaviors ranges in severity from restricting food intake, to bingeing and purging, to the DSM-IV defined disorders of anorexia nervosa and bulimia nervosa (1,2,44,61,110,111). Disordered eating behaviors can result in short and long term morbidity, decreased performance, amenorrhea, and even mortality (36,49,52,94).

2. Amenorrhea. Primary amenorrhea (delayed menarche) is the absence of menstruation by age 16 in a girl with secondary sex characteristics (119). Secondary amenorrhea is the absence of three or more consecutive menstrual cycles after menarche (76,82,92,118,119).

Amenorrhea associated with exercise or anorexia nervosa is hypothalamic in origin (8,77,129). Hypothalamic amenorrhea results in decreased ovarian hormone pro-

duction and hypoeestrogenemia similar to menopause. Both hypothalamic amenorrhea and menopause are associated with decreased bone mineral density (6,10,19,28,30,84,85,103,104).

3. Osteoporosis. Osteoporosis is a disease characterized by low bone mass and microarchitectural deterioration of bone tissue leading to enhanced skeletal fragility and increased risk of fracture (10). An expert panel convened by the World Health Organization has established the following diagnostic criteria (65):

a. Normal: bone mineral density (BMD) that is no more than 1 Standard Deviation (SD) below the mean of young adults.

b. Osteopenia: BMD between 1 and 2.5 SD below the mean of young adults.

c. Osteoporosis: BMD more than 2.5 SD below the mean of young adults.

d. Severe osteoporosis: BMD more than 2.5 SD below the mean of young adults plus one or more fragility fractures.

The principal cause of premenopausal osteoporosis in active women is decreased ovarian hormone production and hypoeestrogenemia as a result of hypothalamic amenorrhea (6,20,28,30,72,84,85,104).

POSITION STAND

Based upon a comprehensive literature survey, research studies, case reports, and the consensus of experts (141), it is the position of the American College of Sports Medicine that:

1. The Female Athlete Triad is a serious syndrome consisting of disordered eating, amenorrhea, and osteoporosis. The components of the Triad are interrelated in etiology, pathogenesis, and consequences. Because of the recent definition of the Triad (141), prevalence studies have not yet been completed. However, it occurs not only in elite athletes but also in physically active girls and women participating in a wide range of physical activities. The Triad can result in declining physical performance, as well as medical and psychological morbidity's and mortality.
2. Internal and external pressures placed on girls and women to achieve or maintain unrealistically low

body weight underlies the development of these disorders.

3. The Triad is often denied, not recognized, and under reported. Sports medicine professionals need to be aware of the interrelated pathogenesis and the varied presentation of components of the Triad. They should be able recognize, diagnose, and treat or refer women with any one component of the Triad.
4. Women with one component of the Triad should be screened for the other components (60,97). Screening for the Triad can be done at the time of the preparticipation examination and during clinical evaluation of the following: menstrual change, disordered eating patterns, weight change, cardiac arrhythmias including bradycardia, depression, or stress fracture (21,36,60,61,68,87,97,112).
5. All sports medicine professionals, including coaches and trainers, should learn about preventing and recognizing the symptoms and risks of the Triad. All individuals working with active girls and women should participate in athletic training that is medically and psychologically sound. They should avoid pressuring girls and women about losing weight. They should know basic nutrition information and have referral sources for nutritional counseling and medical and mental health evaluation.
6. Parents should avoid pressuring their daughters to diet and lose weight. Parents should be educated about the warning signs of the Triad and initiate medical care for their daughters if signs are present.
7. Sports medicine professionals, athletic administrators, and officials of sport governing bodies share a responsibility to prevent, recognize, and treat the Triad. The sport governing bodies should work toward offering opportunities for educational programs for coaches to educate them and to lead them toward professional certification. They should work toward developing programs to monitor coaches and others to ensure safe training practices.
8. Physically active girls and women should be educated about proper nutrition, safe training practices, and the warning signs and risks of the Triad. They should be referred for medical evaluation at the first sign of any of the components of the Triad.
9. Further research is needed into the prevalence, causes, prevention, treatment, and sequelae of the Triad.

BACKGROUND FOR THE POSITION STAND

Body Composition

There is increasing awareness of the influence of body composition on athletic performance. In some activities an increase in body weight can decrease performance (137). However, pressure to decrease body weight or body fat percentage to unrealistic levels contributes to the

development of disordered eating practices (127,140). Excessive weight loss can lead to loss of fat-free mass, dehydration, and a decrease in performance (16,136). An athlete in training inadvertently or voluntarily restricting energy intake to less than her energy expenditure can develop similar problems depending on the duration and magnitude of energy imbalance.

It is critical to recognize the limitations of present techniques to measure body composition and the problems associated with establishing standards for relative body fat. Densitometry, using the hydrostatic weighing technique, is considered the gold standard for assessing body composition (58,67). However, there is an error range of at least 3–4% in a normal, healthy population with this technique (73,83). New techniques are available such as bioelectric impedance analysis and dual-energy x-ray absorptiometry (DXA) (62), but their accuracy, particularly for lean athletic populations, is not adequately established (67,83,138). The precipitation of disordered eating in active girls and women already highly sensitized to their body weight by overemphasizing or overestimating body fat is of particular concern.

Published standards of mean values for relative body fat for small numbers of athletes (137) are arbitrary. They do not recognize measurement error, the wide variation in body fat percentage associated with successful performance, and the genetic variation within a given somatotype (137). If standards must be established, a range of values that recognize both individual variability and methodological error should be set (137). The upper limit of the range should be justified for the individual and attainable with the least risk of precipitating disordered eating. The lower limit of the range should represent the lowest value achieved by elite performers who are healthy and exhibit no signs or symptoms of disordered eating. More research must document the influence of excessive weight loss and caloric deprivation on both health and performance (14,16,47,56).

Disordered Eating

During puberty boys gain muscle mass and are encouraged to gain weight to improve physical performance. In contrast, girls gain body fat during puberty and are encouraged to lose weight to improve performance and/or appearance. Most girls and women try to achieve unrealistic thinness because of societal standards and peer pressure. They may attempt to lose weight or body fat by using caloric restriction or a wide range of disordered eating practices (34,107,110,111,125–127). These practices occur across a wide spectrum and include fasting, diet pills, laxatives, diuretics, and vomiting.

Restrictive eating behaviors include inadvertently failing to balance energy expenditures with adequate energy intake, episodic fasting, and chronic voluntary starvation. Significant caloric restriction reduces metabolic rate and causes changes in the musculoskeletal (6,104,113,133),

cardiovascular (3,56,57,68,94), endocrine (55,97,130,131), thermoregulatory (94), and other systems (11,36,43,49,52,97). Anorexia nervosa is the extreme of restrictive eating behavior in which the individual views herself as overweight and is afraid of gaining weight even though she is 15% below ideal body weight. Amenorrhea is one of the DSM-IV diagnostic criteria for anorexia nervosa (2).

Bulimic behavior is a cycle of food restriction or fasting leading to overeating or bingeing (because of physiological hunger) followed by purging. Purging behavior includes vomiting, the use of laxatives, diuretics, or enemas, and excessive exercise (2). Fluid and electrolyte losses during purging are the major causes of short-term morbidity, including dehydration, acid-base and electrolyte imbalances, and cardiac arrhythmia (49,52,68). Purging can result in chronic physical problems including gastrointestinal disorders, parotid gland enlargement (69), and erosion of tooth enamel (52,97,106). Acute and chronic psychological problems associated with binge-purge cycle include low self esteem, anxiety, depression, and suicide (1,2,43,44).

At the severe end of the spectrum of disordered eating practices are the disorders of anorexia nervosa and bulimia nervosa (2). Between ½ and 1% of young women have anorexia nervosa (1,2), and 1–4% have bulimia nervosa as defined by the DSM-IV psychiatric diagnostic criteria (1,2). Many women who chronically use disordered eating practices do not fit these strict criteria. However, these women who have significant weight, eating, and body image concerns, are at risk for morbidity and mortality, and need evaluation and treatment (14,44,120).

Are athletes and physically active girls and women at greater risk for disordered eating practices than the general population? Conducting accurate prevalence studies with athletic populations is difficult. Disordered eating behaviors are often denied by athletes on standard questionnaires (15). The reported prevalence of disordered eating practices in some college and elite athletes is equal to or greater than the general population (15,34,91,110,111,125,126).

Many factors contribute to the development of disordered eating behavior. These include societal pressures to be thin (107,120,124), chronic dieting (14,15), low self-esteem and depression (43), family dysfunction (86,108), physical or sexual abuse (109), and biological factors (66). Additional factors for athletes include a sport-related emphasis on body weight and body fat, perfectionism, lack of nutrition knowledge, drive to excel and win at any cost, the impact of injury, and pressure to lose weight from parents, coaches, judges, and others (74,107,127,136,140,143).

When surveyed, many athletes think disordered eating practices are harmless (110,143). They are erroneously told or believe that losing weight by whatever method enhances performance (110). In reality, inadequate ca-

loric intake and disordered eating practices impair physical performance and health (16,47,56). Problems result from depletion of muscle glycogen stores, dehydration, loss of muscle mass, hypoglycemia, electrolyte abnormalities, anemia, amenorrhea, and osteoporosis (9,16,36,49,52,97).

Eating disorders are chronic illnesses with serious medical and psychological sequelae. These include depression, substance abuse, and death owing to suicide or cardiac arrhythmia (1,44,49,52,57,68,94,99,140). Treatment requires an individualized, long-term, multidisciplinary approach. It usually involves a physician, mental health practitioner, and registered dietitian, all supported by the family and significant others (43,60,108,112,128). The prognosis for recovery depends upon early intervention, the duration and severity of the disorder, and the individual's willingness to seek care and remain in treatment.

Amenorrhea

The monthly menstrual cycle is a complex interaction of the endocrine and reproductive systems. External stimuli affect the system through hormonal signals to the hypothalamus. The cessation of menses coincident with physical training has long been recognized (8,17,22,38,39,76,78,80,82,98,116,142). However, the etiology, prevalence, sequelae, and treatment of exercise associated amenorrhea are not completely known. Between 2 and 5% of women of reproductive age who are not pregnant or lactating experience some form of secondary amenorrhea (5,95,121). Studies report between 1 and 44% of athletic women experience amenorrhea at any given time (22,38,39,76,98).

A reduction in the frequency of luteinizing hormone (LH) pulses from the pituitary gland is the direct cause of amenorrhea and subsequent ovarian suppression in physically active women (77,78,80,82,129). The reduction in LH pulse frequency is thought to be caused by a decrease in the frequency of gonadotrophic releasing hormone (GnRH) pulses secreted by the hypothalamus. The regulation of GnRH secreting cells is the focus of current research on amenorrhea (77,78,80).

In the 1970s, low body weight (41) and body fat (42) were postulated to cause amenorrhea. Subsequent research has refuted these hypotheses and indicated other factors are involved (12,76,114,122). One study induced reproductive disorders experimentally by abruptly imposing a high volume of intense exercise while controlling dietary intake (17). Therefore, exercise stress and energy availability are both being investigated as potential causes of the disruption of the GnRH pulse generator. The hypothesis that exercise stress disrupts the GnRH pulse generator is based on research that electroshock activates the hypothalamic-pituitary-adrenal axis and disrupts reproductive function in animals (105) and on observations of high resting cortisol levels (27,77) and

blunted cortisol responses to exercise in amenorrheic athletes (25,75). Proponents of the exercise stress hypothesis view cortisol as a stress hormone.

Energy availability can be defined as dietary energy intake minus exercise energy expenditure. However, dietary energy intake and exercise energy expenditure are both difficult to measure accurately. The hypothesis that low energy availability disrupts the GnRH pulse generator is based on widespread reports that women athletes consume less dietary energy than they would seem to need for their activity levels (139) and on observations of low T3 levels (79,89) and negative energy balance in amenorrheic athletes (18,35,89). Menstrual disorders may be caused by failure to compensate dietary energy intake for the energy cost of exercise rather than by exercise itself. Proponents of the energy availability hypothesis view cortisol as a regulator of blood glucose concentrations.

A recent experiment in eumenorrheic trained women reported that LH pulsatility was suppressed after 3 days of exercise training while dietary intake was reduced, but was not suppressed when dietary intake was supplemented (135). In another experiment, LH pulse frequency was reduced both in non-exercising women when dietary intake was restricted and in exercising women when their dietary intake was not supplemented for the energy cost of exercise (81). This study also reported that exercise stress did not reduce LH pulse frequency when non-exercising and exercising women with similar energy availabilities were compared (81).

Endurance athletes, because of their large daily exercise energy expenditure, may be energy deficient if they consume the same diets that adequately nourish sedentary women. They may not display evidence of eating disorders and may not consciously restrict their diets. However, they may inadvertently consume fewer calories than needed to sustain their high energy expenditure and may develop amenorrhea as a result of energy imbalance (35,89). To avoid alterations in their reproductive hormones and menstrual function, physically active women may have to practice eating patterns to attain energy intake that matches their energy expenditure.

Amenorrhea, once felt to be a benign, reversible condition (38,142), has been linked to premature loss of BMD since 1984 (19,28,84). Amenorrhea is neither desirable nor a "normal" result of physical training (92,118,119). It is a symptom of an underlying problem that requires medical evaluation within the first 3 months of occurrence (21,54,92,118,119). However, many physically active women and their coaches and advisors do not regard or report amenorrhea as abnormal. Women may welcome the convenience of not menstruating. Some mistakenly believe amenorrhea is an indicator of adequate training intensity rather than a symptom requiring medical attention. Amenorrhea is the most recognizable symptom of the Female Athlete Triad, and women

and their advisors need to be informed that amenorrhea requires prompt medical assessment for its cause and implications.

Ovulation and reversal of amenorrhea are unpredictable in amenorrheic women. Because ovulation precedes menstruation, all sexually active amenorrheic women should be tested for pregnancy as part of their medical evaluation and receive contraception counseling (90,21). Exercise associated amenorrhea is a diagnosis of exclusion, and all other causes of amenorrhea must be excluded by a thorough medical evaluation (92,54,118,119). Amenorrheic women should be encouraged to ingest at least 1500 mg of elemental calcium per day to assure to calcium balance (21,92).

Reversal of amenorrhea has been reported after injury, during reduced training, and with increased caloric intake (29,31,32,38,64,70). However, no epidemiological data are available on the success rate of any lifestyle change or treatment for reversing amenorrhea, improving bone mass, or fertility (114). Further prospective research needs to be done on exercise associated amenorrhea to establish its pathogenesis and accurately direct intervention and therapy.

Osteoporosis

Low concentrations of ovarian hormones in amenorrheic and oligomenorrheic athletes are associated with reduced bone mass and increased rates of bone loss. This loss is similar to bone loss in postmenopausal women or in women with a pathological hypoestrogenic condition such as premature ovarian failure, a pituitary tumor, or anorexia nervosa (37,104,113,115). In earlier studies (19,28,84), decreased BMD was reported only for the lumbar spine. Newer and more precise techniques for measuring BMD at other sites indicate the deficit appears to be generalized throughout the skeleton (87,88,101,102). Whether bone loss is observed at all regional sites may depend in part on the extent of mechanical loading at specific sites in various sports (123). The occurrence of disordered eating practices and low calcium intake combined with menstrual dysfunction may exacerbate bone loss. Not all amenorrheic athletes have low bone mass. Their skeletal status depends upon the length and severity of their menstrual irregularity, as well as factors that influence their BMD prior to the onset of amenorrhea: the type of skeletal loading during activity, their nutritional status, and a genetic component (24).

Concern for athletes with low BMD owing to hypoestrogenemia revolves around the risk for fractures during their competitive years and the future risk of premature osteoporotic fractures (132). Hip fractures and multiple compression fractures of the spine, common among elderly women, have been documented in young women with anorexia nervosa (13,104). While low weight athletes may not meet all the criteria for anorexia

nervosa or bulimia nervosa, the inadequate caloric intake and poor nutrition involved in attaining an unrealistic weight may place these women at risk for serious fractures (33,90). Studies report a higher incidence of injuries and stress fractures among amenorrheic and oligomenorrheic as compared to eumenorrheic athletes (9,33,71,132). One study (87) reports a lower BMD in women athletes who had stress fractures than among non-injured controls. This finding was not confirmed in another study (48). While the hypoestrogenic state is not a prerequisite for stress fracture, low BMD may place the athlete at higher risk.

Researchers (20,30,45,72,85) report that prior menstrual history is the best predictor of current BMD. Women with a history of amenorrhea have a lower BMD than those who have always been cyclic. As the rate of bone loss in hypoestrogenic women is greatest in the 5–6 years following the decrease in levels of endogenous estrogen, there may be only a brief window of opportunity to initiate intervention therapy to prevent irreversible bone loss. In the adolescent athlete, poor nutrition and the hypoestrogenic condition may result in a lack of bone accretion during the critical years of skeletal consolidation (6,26,46,63,100,134).

An important question is whether the hypoestrogenic low BMD condition can be treated or reversed and normal bone mass attained. Newer techniques of measuring bone density, specifically dual energy x-ray absorptiometry (DXA) (62), can identify individuals with low BMD and, if done serially, can assess those who are rapidly losing bone and monitor their response to therapy. Three studies (29,64,70) report an increase in BMD in amenorrheic athletes resuming normal menses, but it appears these gains may be limited. Amenorrheic athletes using hormone replacement therapy in doses used for menopausal women have shown maintenance of BMD but no gains (51). Women with amenorrhea and less severe bone loss may prefer to adjust training and nutritional patterns to resume their normal reproductive cycle (7,30,31,32,92,119).

Who is at Risk?

Potentially all physically active girls and women could be at risk for developing one or more components of the Triad. The biological changes, peer pressure, societal drive for thinness, and body image preoccupation that occur during puberty make adolescence the most vulnerable time. Participation in sports that emphasize low body weight can also be a risk factor. Those sports include:

1. Sports in which performance is subjectively scored (dance, figure skating, diving, gymnastics, aerobics).
2. Endurance sports emphasizing a low body weight (distance running, cycling, cross-country skiing).

3. Sports requiring body contour-revealing clothing for competition (volleyball, swimming, diving, cross-country running, cross-country skiing, and track, cheerleading).
4. Sports using weight categories for participation (horse racing, some martial arts, wrestling, rowing).
5. Sports emphasizing a prepubertal body habitus for performance success (figure skating, gymnastics, diving).

Male athletes, particularly those in weight regulated and endurance sports, are also at risk for disordered eating (4,23) and anorexia nervosa. Extensive exercise and anorexia nervosa in men have been associated with hypogonadism and osteoporosis (4,40,53,59,104). The Female Athlete Triad also occurs in nonathletes and in physically active girls and women who are not training or competing in a specific sport.

CONCLUSION

Based on the existing evidence of the magnitude and seriousness of the problems associated with the Female Athlete Triad, the American College of Sports Medicine strongly advises that specific strategies be developed to prevent, recognize, and treat this syndrome. Strategies specific to prevention, surveillance, research, health consequences, medical care, and public and professional education need to be developed, implemented, and monitored. Target groups for education include coaches, trainers, parents, athletes, peers, athletic administrators, officials of sport governing bodies, and health care providers who work with physically active individuals.

More research is needed in many areas related to the Triad: body composition, disordered eating, amenorrhea, osteoporosis, psychological factors, prevalence, warning signs, treatment, and outcomes. Prospective longitudinal studies should include athletes and active women and men from diverse activities, ages, sports, and backgrounds. Healthy, positive role models who are sport and ethnically relevant for physically active girls and women should be developed. Further guidelines for the recognition, evaluation, treatment, and clearance for athletic participation need to be developed and disseminated to target audiences. Sport administrators and officials of sport governing bodies need to develop standards and encourage certification for coaching and training practices and the monitoring of young women to prevent and treat the Female Athlete Triad.

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REFERENCES

1. AMERICAN PSYCHIATRIC ASSOCIATION. *Diagnostic Statistical Manual of Mental Disorders*, 3rd Ed. Revised. Washington, DC: American Psychiatric Association, 1987.
2. AMERICAN PSYCHIATRIC ASSOCIATION. *Diagnostic, and Statistical Manual of Mental Disorders*, 4th Ed. Washington, DC: American Psychiatric Association, 1994.
3. AMREIN, P. C., R. FRIEDMAN, K. KOSINSKI, and L. ELLMAN. Hematologic changes in anorexia nervosa. *JAMA* 24:2190-2191, 1979.
4. ANDERSEN, A. E. Eating Disorders in Males: a special case? In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp.172-190.
5. BACHMAN, G. A. and E. KEMMANN. Prevalence of oligomenorrhea and amenorrhea in a college population. *Am. J. Obstet. Gynecol.* 144:98-102, 1982.
6. BACHRACH, L. K., D. GUIDO, D. KATZMAN, and R. MARCUS. Decreased bone density in adolescent girls with anorexia nervosa. *Pediatrics* 86:440-7, 1990.
7. 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.
8. BAKER, E. R. Menstrual dysfunction and hormonal status in athletic women: a review. *Fertil. Steril.* 36:691-696, 1981.
9. BARROW, G. W. and S. SAHA. Menstrual irregularity and stress fractures in collegiate female distance runners. *Am. J. Sports Med.* 16:209-216, 1988.
10. BOUILLON, P., P. BURCKHARDT, C. CHRISTIANSEN, et al. Consensus development conference: Prophylaxis and treatment of osteoporosis. *Am. J. Med.* 90:107-110, 1991.
11. BOWERS, T. K. and E. ECKERT. Leukopenia in anorexia nervosa: lack of increased risk of infection. *Arch. Intern. Med.* 138:1520-1523, 1978.
12. BRONSON, F. H. and J. M. MANNING. The energetic regulation of ovulation: a realistic role for body fat. *Biol. Reprod.* 44:945-950, 1991.
13. BROTMAN, A. W. and T. A. STERN. Osteoporosis, and pathologic fractures in anorexia nervosa. *Am. J. Psychiatry* 142:495-496, 1985.
14. BROWNELL, K. D., S. N. STEEN, and J. H. WILMORE. Weight regulation practices in athletes: analysis of metabolic and health effects. *Med. Sci. Sports Exerc.* 19:546-556, 1987.
15. BROWNELL, K. D. and J. RODIN. Prevalence of eating disorders in athletes. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992a, pp.128-145.
16. BROWNELL, K. D. and S. N. STEEN. Weight cycling in athletes: effects on behavior, physiology and health. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp.159-171.
17. 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.
18. CAMERON, J. L., C. NOSBISCH, D. L. HELMREICH, and D. B. PARFITT. Reversal of exercise-induced amenorrhea in female cynomolgus monkeys (*Macaca fascicularis*) by increasing food intake (Abstract). *Proceedings of the Endocrine Society 72nd Annual Meeting*, 1990, p. 285.
19. CANN, C. E., M. C. MARTIN, H. K. GENANT, and R. B. JAFFE. Decreased spinal mineral content in amenorrheic women. *JAMA* 251:626-629, 1984.
20. CANN, C. E., D. J. CAVANAUGH, K. SCHNURPFEL, and M. C. MARTIN. Menstrual history is the primary determinant of trabecular bone density in women runners (Abstract) *Med. Sci. Sports Exerc.* 20(Suppl. 5):354, 1988.
21. COMMITTEE ON SPORTS MEDICINE. Amenorrhea in adolescent athletes. *Pediatrics* 84:394-395, 1989.
22. DALE, E., D. H. GERLACH, and A. L. WILHITE. Menstrual dysfunction in distance runners. *Obstet. Gynecol.* 54:47-53, 1979.
23. DEPALMA, M. T., W. M. KOSZEWSKI, J. G. CASE, R. J. BARILE, B. F. DEPALMA, and S. M. OLIARO. Weight control practices of lightweight football players. *Med. Sci. Sports Exerc.* 25:694-701, 1993.
24. DEQUEKER, J., J. NUS, A. VERSTRAETEN, P. GUESSENS, and G. GEVERS. Genetic determination of bone mineral content at the spine and radius: a twin study. *Bone* 8:207-209, 1987.
25. DE SOUZA, M. J., M. S. MAGUIRE, C. M. MARESH, W. J. KRAEMER, K. R. RUBIN, and A. B. LOUCKS. Adrenal activation and the prolactin response to exercise in eumenorrheic and amenorrheic runners. *J. Appl. Physiol.* 70:2378-2387, 1991.
26. DHUPER, S., M. P. WARREN, J. BROOKS-GUNN, and R. FOX. Effects of hormonal status on bone density in adolescent girls. *J. Clin. Endocrinol. Metab.* 71:1083-1088, 1990.
27. DING, J.-H., C. B. SHECKTER, B. L. DRINKWATER, M. R. SOULES, and W. J. BREMNER. High serum cortisol levels in exercise-associated amenorrhea. *Ann. Intern. Med.* 108:530-534, 1988.
28. DRINKWATER, B. L., K. NILSON, C. H. CHESNUT, III, 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.
29. DRINKWATER, B. L., K. NILSON, S. OTT, and C. H. CHESNUT, III. Bone mineral density after resumption of menses in amenorrheic athletes. *JAMA* 256:380-382, 1986.
30. DRINKWATER, B. L., B. BRUEMNER, and C. H. CHESNUT, III. Menstrual history as a determinant of current bone density in young athletes. *JAMA* 263:545-548, 1990.
31. 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.
32. DUECK, C. A., M. M. MANORE, and K. S. MATT. Role of energy balance in athletic menstrual dysfunction. *Int. J. Sport Nutr.* 6:165-190, 1996b.
33. 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.
34. DUMMER, G. M., L. W. ROSEN, W. W. HEUSNER, ET AL. Pathogenic weight-control behaviors of young competitive swimmers. *Physician Sportsmed.* 15:75-86, 1987.
35. EDWARDS, J. E., A. K. LINDEMAN, A. E. MIKESKY, and J. M. STAGER. Energy balance in highly trained female endurance runners. *Med. Sci. Sports Exerc.* 25:1398-1404, 1993.
36. EICHNER, E. R. General health issues of low body weight and undereating in athletes. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp. 191-201.
37. EMANS, S. J., E. GRACE, F. A. HOFFER, C. GUNDBERG, V. RAVNIKAR, and E. R. WOODS. Estrogen deficiency in adolescents and young adults: impact on bone mineral content and effects of estrogen replacement therapy. *Obstet. Gynecol.* 76:585-592, 1990.
38. ERDELYI, G. J. Gynecological survey of female athletes. *J. Sports Med.* 2:174-179, 1962.
39. FEICHT, C. B., T. S. JOHNSON, B. J. MARTIN, K. E. SPARKS, and W. W. WAGNER JR. Secondary amenorrhea in athletes. *Lancet* 2:1145-1146, 1978.
40. FINKELSTEIN, J. S., A. KLIBANSKI, R. M. NEER, S. L. GREENSPAN, D. I. ROSENTHAL, and W. F. CROWLEY JR. Osteoporosis in men with idiopathic hypogonadotropic hypogonadism. *Ann. Intern. Med.* 106:354-61, 1987.
41. FRISCH, R. E. and R. REVELLE. Height and weight at menarche and a hypothesis of menarche. *Arch. Dis. Child.* 46:695-701, 1971.
42. FRISCH, R. E. and J. W. MCARTHUR. Menstrual cycles: Fatness as a determinant of minimum weight for height necessary for their maintenance or onset. *Science* 185:949-951, 1974.
43. GARFINKEL, P. E. and D. M. GARNER. *Anorexia Nervosa: A Multidimensional Perspective*. New York: Brunner/Mazel, 1982.

44. GARNER, D. M. and P. GARFINKEL. *Anorexia Nervosa and Bulimia*. New York: Guilford Press, 1985.
45. GEORGIU, E., K. NTALLES, A. PAPAGEORGIOU, A. KORKOTSIDIS, and C. PROUKAKIS. Bone mineral loss related to menstrual history. *Acta Orthop. Scand.* 60:192-194, 1989.
46. GILSANZ, V., D. T. GIBBENS, and T. F. ROE. Vertebral bone density in children: effect of puberty. *Radiology* 166:847-50, 1988.
47. GLEESON, M., P. L. GREENHOFF, and R. J. MAUGHAN. Influence of a 24 hour fast on high intensity exercise performance in man. *Eur. J. Appl. Physiol.* 57:653-659, 1988.
48. GRIMSTON, S. K., J. R. ENGBERG, R. KLOIBER, and D. A. HANLEY. Bone mass, external loads and stress fractures in female runners. *Int. J. Sport Biomech.* 7:293-302, 1991.
49. HARRIS, R. T. Bulimarexia. *Ann. Intern. Med.* 99:800-807, 1983.
50. HARRIS, S., C. J. CASPERSON, and G. H. DEFRIESE. Physical activity counseling for healthy adults as a primary preventative intervention in the clinical setting. *JAMA* 261:3590-8, 1989.
51. HERGENROEDER, A. C. Bone mineralization, hypothalamic amenorrhea, and sex steroid therapy in female adolescents and young adults. *J. Pediatr.* 126:683-689, 1995.
52. HERZOG, D. B. and P. M. COPELAND. Eating disorders. *N. Engl. J. Med.* 313:5:295-303, 1985.
53. HETLAND, M. L., J. HAARBO, and C. CHRISTIANSEN. Low bone mass and high bone turnover in male long distance runners. *J. Clin. Endocrinol. Metab.* 77:770-5, 1993.
54. HIGHER, R. Athletic amenorrhea: an update on aetiology, implications and management. *Sports Med.* 7:82-108, 1989.
55. HURD, H. P., P. J. PALUMBO, and H. GHARID. Hypothalamic-endocrine dysfunction in anorexia nervosa. *Mayo Clin. Proc.* 52:711-716, 1977.
56. INJER, F. and J. SUNDGOT-BORGEN. Influence of body weight reduction on maximal oxygen uptake in female elite athletes. *Scand. J. Med. Sci. Sports* 1:141-146, 1991.
57. ISNER, J. M., W. C. ROBERTS, S. B. HEYMSFIELD, and J. YAGER. Anorexia nervosa and sudden death. *Ann. Intern. Med.* 102:49-52, 1985.
58. JACKSON, A. S., M. L. POLLOCK, and A. WARD. Generalized equations for predicting body density of women. *Med. Sci. Sports Exerc.* 12:175-182, 1980.
59. JACKSON, J. A. and M. KLEEREKOPER. Osteoporosis in men: diagnosis, pathophysiology, and prevention. *Medicine* 69:8:137-152, 1990.
60. JOHNSON, M. D. Tailoring, the preparticipation exam to female athletes. *Physician Sportsmed.* 20:61-72, 1992.
61. JOHNSON, M. D. Disordered eating in active and athletic women. *Clin. Sports Med.* 13:355-369, 1994.
62. JOHNSTON, C. C. JR., C. W. SLEMENDA, and L. J. MELTON III. Clinical use of bone densitometry. *N. Engl. J. Med.* 324:104, 1991.
63. JOHNSTON, C. C., JR., J. Z. MILLER, and C. W. SLEMENDA. Calcium supplementation and increases in bone mineral density in children. *N. Engl. J. Med.* 327:82-7, 1992.
64. JONNAVITHULA, S., M. P. WARREN, R. P. FOX, and M. I. LAZARO. Bone density is compromised in amenorrheic women despite returning menses: a 2-year study. *Obstet. Gynecol.* 81:669-74, 1993.
65. KANIS, J. A., J. MELTON, III, C. CHRISTIANSEN, C. C. JOHNSTON, and N. KHALTAEV. The diagnosis of osteoporosis. *J. Bone Miner. Res.* 9:1137-1141, 1994.
66. KAYE, W. H. and T. E. WELTZIN. Neurochemistry of bulimia nervosa. *J. Clin. Psychol.* 52:21-28, 1991.
67. KOHRT, W. M. Body composition by DXA: tried and true? *Med. Sci. Sports Exerc.* 27:1349-1353, 1995.
68. KREIPE, R. E. and J. P. HARRIS. Myocardial impairment resulting from eating disorders. *Pediatr. Ann.* 21:760-768, 1992.
69. LEVIN, P. A., J. M. FALKO, K. DIXON, E. M. GALLUP, and W. SAUNDERS. Benign parotid enlargement in bulimia. *Ann. Intern. Med.* 93:827-829, 1980.
70. 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.
71. LLOYD, T., S. J. TRIANTAFYLLOU, E. R. BAKER, et al. Women athletes with menstrual irregularity have increased musculoskeletal injuries. *Med. Sci. Sports Exerc.* 18:374-379, 1986.
72. 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.
73. LOHMAN, T. G. Applicability of body composition techniques and constants for children and youths. *Exerc. Sport Sci. Rev.* 14:325-357, 1986.
74. LOPIANO, D. A. and C. ZOTOS. Modern athletics, the pressure to perform. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds). Philadelphia: Lea and Febiger, 1992, pp. 275-292.
75. LOUCKS, A. B. and S. M. HORVATH. Exercise-induced stress responses to amenorrheic and eumenorrheic runner. *J. Clin. Endocrinol. Metab.* 59:1109-1120, 1984.
76. LOUCKS, A. B. and S. M. HORVATH. Athletic amenorrhea: a review. *Med. Sci. Sports Exerc.* 17:56-72, 1985.
77. 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.
78. LOUCKS, A. B. Effects of exercise training on the menstrual cycle: existence and mechanisms. *Med. Sci. Sports Exerc.* 22:275-280, 1990.
79. LOUCKS, A. B., G. A. LAUGHLIN, J. F. MORTOLA, L. GIRTON, J. C. NELSON, and S. S. C. YEN. Hypothalamic-pituitary-thyroidal function in eumenorrheic and amenorrheic athletes. *J. Clin. Endocrinol. Metab.* 75:514-518, 1992a.
80. LOUCKS, A. B., J. VAITUKAITIS, and J. L. CAMERON. The reproductive system and exercise in women. *Med. Sci. Sports Exerc.* 24(Suppl.):S288-293, 1992.
81. LOUCKS, A. B., E. M. HEATH, K. KING, D. MORRALL, M. VERDUN, and J. R. WATTS. Low Energy availability alters luteinizing hormone pulsatility in regularly menstruating, young exercising women. (Abstract 822) *Endocrine Society Meeting*, 1994.
82. LOUCKS, A. B. The Reproductive System. In: *Perspectives in Exercise Science and Sports Medicine: Vol 9. Exercise and the Female. A Life Span Approach*, O. Bar-Or, D. R. Lamb and P. M. Clarkson (Eds.). Carmel, IN: Cooper Publishing Group, 1996, pp. 41-71.
83. LUKASKI, H. C. Methods for the assessment of human body composition: traditional and new. *Am. J. Clin. Nutr.* 46:537-556, 1987.
84. MARCUS, R., C. CANN, P. MADVIG, et al. Menstrual function and bone mass in elite women distance runners. *Ann. Intern. Med.* 102:158-163, 1985.
85. MICKLESFIELD, L. K., E. V. LAMBERT, A. B. FATAAR, T. D. NOAKES, and K. B. MYBURGH. Bone mineral density in mature, premenopausal ultramarathon runners. *Med. Sci. Sports Exerc.* 27:688-696, 1995.
86. MINUCHIN, S., B. L. ROSMAN, and L. BAKER. *Psychosomatic Families: Anorexia Nervosa in Context*. Cambridge: Harvard University Press, 1978.
87. 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.
88. MYBURGH, K. H., L. K. BACHRACH, B. LEWIS, K. KENT, and R. MARCUS. Bone mineral density at axial and appendicular sites in amenorrheic athletes. *Med. Sci. Sports Exerc.* 25:1197-1202, 1993.
89. MYERSON, M., B. GUTIN, M. P. WARREN, ET AL. Resting metabolic rate and energy balance in amenorrheic and eumenorrheic runners. *Med. Sci. Sports Exerc.* 23:15-22, 1991.
90. 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.

91. O'CONNER, P. J., R. D. LEWIS, and E. M. KIRCHNER. Eating disorder symptoms in female college gymnasts. *Med. Sci. Sports Exerc.* 27:550-555, 1995.
92. OTIS, C. L. Exercise Associated Amenorrhea. *Clin. Sports Med.* 11:2:351-362, 1992.
93. O'TOOLE, M. L. and P. S. DOUGLAS. Fitness: definition and developments. In: *Women and Exercise*, M. Shangol. and G. Mirkin (Eds.). Philadelphia: F. A. Davis Company, 1988, pp. 4-16.
94. PALLA, B. and I. F. LITT. Medical complications of eating disorders in adolescents. *Pediatrics* 81:613-23, 1988.
95. PETTERSON, F., H. FIRES, and S. J. NILLIUS. Epidemiology of secondary amenorrhea: incidence and prevalence rates. *Am. J. Obstet. Gynecol.* 117:80-86, 1973.
96. PIRKE, K. M., U. SCHWEIGER, W. LEMMEL, J. C. KRIEG, and M. BERGER. The influence of dieting on the menstrual cycle of healthy young women. *J. Clin. Endocrinol. Metab.* 60:1974-1979, 1985.
97. POMEROY, C. and J. E. MITCHELL. Medical issues in the eating disorders. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp. 202-221.
98. PUHL, J. L. and C. H. BROWN (Eds.). *THE MENSTRUAL CYCLE AND PHYSICAL ACTIVITY*. CHAMPAIGN, IL: HUMAN KINETICS PUBLISHERS, 1986.
99. RATNASURIYA, R. H., I. EISLER, and G. I. SZMUCKLER. Anorexia nervosa: outcome and prognostic factors after 20 years. *Br. J. Psych.* 158:495, 1991.
100. RECKER, R. R., K. M. DAVIES, and S. M. HINDERS. Bone gain in young adult women. *JAMA* 268:2403, 1992.
101. RENCKEN, M., B. L. DRINKWATER, and C. H. CHESTNUT III. Decreased bone density in the lower extremity of amenorrheic athletes. *J. Bone Miner. Res.* 8(Suppl.1):S254, 1993.
102. RENCKEN, M., C. H. CHESNUT, and B. L. DRINKWATER. Bone density at multiple skeletal sites in amenorrheic athletes. *JAMA* 276:238-240, 1996.
103. RIGGS, B. L. and I. J. MELTON, III. The prevention and treatment of osteoporosis. *N. Engl. J. Med.* 327:620-627, 1992.
104. RIGOTTI, N. A., S. R. NUSSBAUM, D. B. HERZOG, and R. M. NEER. Osteoporosis in women with anorexia nervosa. *N. Engl. J. Med.* 311:1601-1606, 1984.
105. RIVIER, C. and S. RIVEST. Effect of stress on the activity of the hypothalamic-pituitary-gonadal axis: peripheral and central mechanisms. *Biol. Reprod.* 45:523-532, 1991.
106. ROBERTS, M. W. and S. H. LI. Oral findings in anorexia nervosa and bulimia nervosa: a study of 47 cases. *J. Am. Dent Assoc.* 115:407, 1987.
107. RODIN, J. and L. LARSON. Social factors and the ideal body shape. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp. 146-158.
108. ROOT, M. P. P., P. FALLON, and W. N. FRIEDRICH. *Bulimia: A Systems Approach to Treatment*. New York: Norton Books, 1986.
109. ROOT, M. P. P. Persistent disordered eating as a gender-specific, post-traumatic stress response to sexual assault. *Psychotherapy* 28:96-102, 1991.
110. ROSEN, L. W., D. B. MCKEAG, and D. O. HOUGH. Pathogenic weight-control behavior in female athletes. *Physician Sportsmed.* 14:79-86, 1986.
111. ROSEN, L. W. and D. O. HOUGH. Pathogenic weight-control behaviors of female college gymnasts. *Physician Sportsmed.* 16: 141-146, 1988.
112. RYAN, R. Management of eating problems in athletic settings. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp. 344-360.
113. SALISBURY, J. J. and J. E. MITCHELL. Bone mineral density and anorexia nervosa in women. *Am. J. Psychiatry* 148:768-774, 1991.
114. SANBORN, C. F., B. H. ALBRECHT, and W. W. WAGNER, JR. Medically induced reversal of infertility in athletic amenorrhea. (Abstract). *Med. Sci. Sports Exerc.* 19(Suppl. 5):27, 1987.
115. SCHLECHTE, J. A., B. SHERMAN, and R. MARTIN. Bone density in amenorrheic women with and without hyperprolactinemia. *J. Clin. Endocrinol. Metab.* 56:1120-1123, 1983.
116. SCHWARTZ, B., D. C. CUMMING, E. RIORDAN, M. SELYE, S. S. YEN, and R. W. REBAR. Exercise-associated amenorrhea: a distinct entity? *Obstet. Gynecol.* 141:662-670, 1981.
117. SHANGOLD, M. M. and H. S. LEVINE. The effect of marathon training upon menstrual function. *Am. J. Obstet. Gynecol.* 143: 862-869, 1982.
118. SHANGOLD, M. M. Menstruation In: *Women and Exercise*, M. Shangold and G. Mirkin (Eds.). Philadelphia: F. A. Davis, 1988, pp. 129-145.
119. SHANGOLD, M. M., R. W. REBAR, A. C. WENTZ, and I. SCHIFF. Evaluation and management of menstrual dysfunction in athletes. *JAMA* 263:1665-1669, 1990.
120. SILVERSTEIN, B. and L. PERDUP. The relationship between role concern, preferences for slimness, and symptoms of eating problems among college women. *Sex Roles* 18:101-106, 1988.
121. SINGH, K. B. Menstrual disorders in college students. *Am. J. Obstet. Gynecol.* 140:299-302, 1981.
122. SINNING, W. E. and K. D. LITTLE. Body composition and menstrual function in athletes. *Sports Med.* 4:34-45, 1987.
123. SLEMENDA, C. W. and C. C. JOHNSON. High intensity activities in young women: site specific bone mass effects among female figure skaters. *Bone Miner.* 20:125-132, 1993.
124. STRIEGEL-MOORE, R. H., L. R. SILBERSTEIN, and J. RODIN. Toward an understanding of risk factors for bulimia. *Am. Psychol.* 41: 246-263, 1986.
125. SUNDOT-BORGEN, J. Prevalence of eating disorders in elite female athletes. *Int. J. Sport Nutr.* 3:29-40, 1993.
126. SUNDOT-BORGEN, J. and S. LARSEN. Pathologic weight-control methods and self-reported eating disorders in female elite athletes and control. *Scand. J. Med. Sci. Sports* 3:150-155, 1993.
127. 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.
128. TOBIN, D. L., C. L. JOHNSON, and K. FRANKE. Clinical treatment of eating disorders. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp. 330-343.
129. VELDHIJS, J. D., W. S. EVANS, L. M. DEMERS, M. O. THORNER, D. WAKAT, and A. D. ROGOL. Altered neuroendocrine regulation of gonadotropin secretion in women distance runners. *J. Clin. Endocrinol. Metab.* 61:557-563, 1985.
130. VIGORSKY, R. A., A. E. ANDERSON, R. H. THOMPSON, and D. L. LORIAUX. Hypothalamic dysfunction in secondary amenorrhea associated with simple weight loss. *N. Engl. J. Med.* 297:1141-1145, 1977.
131. WARREN, M. P. and R. L. VANDEWIELE. Clinical and metabolic features of anorexia nervosa. *Am. J. Obstet. Gynecol.* 117:435-449, 1973.
132. WARREN, M. P., J. BROOKS-GUNN, L. H. HAMILTON, L. F. WARREN, and W. G. HAMILTON. Scoliosis and fractures in young ballet dancers. *N. Engl. J. Med.* 314:1348-1353, 1986.
133. WARREN, M. P., J. BROOKS-GUNN, R. P. FOX, C. LANCELOT, D. NEWMAN, and W. G. HAMILTON. Lack of bone accretion and amenorrhea: evidence for a relative osteopenia in weight-bearing bones. *J. Clin. Endocrinol. Metab.* 72:847-853, 1991.
134. WHITE, C. M., A. C. HERGENROEDER, and W. J. KLISH. Bone mineral density in 15- to 21-year-old eumenorrheic and amenorrheic subjects. *Am. J. Dis. Child.* 146:31-35, 1992.
135. WILLIAMS, N. I., J. C. YOUNG, J. W. MCARTHUR, B. BULLEN, G. S. SKRINAR, and B. TURNBULL. Strenuous exercise with caloric restriction: effect on luteinizing hormone secretion. *Med. Sci. Sport Exerc.* 27:1390-1398, 1995.
136. WILMORE, J. H. Eating and weight disorders in the female athlete. *Int. J. Sports Nutr.* 1:104-117, 1991.

137. WILMORE, J. H. Body weight standards and athletic performance. *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*. K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds). Philadelphia: Lea and Febiger, 1992, pp. 315-329.
138. WILMORE, J. H. Body weight and body composition. In: *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds). Philadelphia: Lea and Febiger, 1992, pp. 77-93.
139. WILMORE, J. H., K. C. WAMBSQANS, M. BRENNER, et al. Is there energy conservation in amenorrheic compared with eumenorrheic distance runner? *J. Appl. Physiol.* 72:15-22, 1992c.
140. WILSON, G. T. and K. L. ELDREDGE. Pathology and development of eating disorders: implications for athletes. *Eating, Body Weight and Performance in Athletes: Disorders of Modern Society*, K. D. Brownell, J. Rodin, and J. H. Wilmore (Eds.). Philadelphia: Lea and Febiger, 1992, pp. 115-127.
141. YEAGER, K. K., R. AGOSTINI, A. NATTIV, and B. DRINKWATER. The female athlete triad. *Med. Sci. Sports Exerc.* 25:775-777, 1993.
142. ZAHARIEVA, E. Olympic participation by women: effects on pregnancy and childbirth. *JAMA* 221:992, 1972.
143. ZUCKER, P. Eating disorders in young athletes: a round table. *Physician Sportsmed.* 13:11:89-106, 1985.