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THE FEMALE ATHLETE TRIAD: A METABOLIC PHENOMENON

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Abstract

Loucks, A. B. (2014). The Female Athlete Triad: A Metabolic Phenomenon. **PENSAR EN MOVIMIENTO: Revista de Ciencias del Ejercicio y la Salud, 12 (1),** 1-23. The Female Athlete Triad (Triad) is a syndrome in which low energy availability triggers a broad range of endocrine mechanisms that conserve energy expenditure, and thereby impairs reproductive and skeletal health. Energy availability is the amount of dietary energy remaining after exercise training for all other physiological functions each day. The specific kind of reproductive dysfunctions caused by low energy availability are functional hypothalamic menstrual disorders. To ensure that affected athletes receive appropriate care, endocrine tests are required to diagnose these disorders by the exclusion of other types of menstrual disorders unrelated to the Triad. In addition, low energy availability impairs skeletal health by uncoupling bone turnover, in which the rate of bone resorption increases while the rate of bone formation declines. The result is a progressive loss or failure to accrue bone mass, which increases the risks of stress fractures and osteoporosis. Low energy availability originates in one or more of three sources: restrictive eating disorders, especially anorexia nervosa; intentional efforts to lose body weight or body fat to improve athletic performance or appearance; and the inadvertent suppression of appetite by exercise and diets containing a high percentage of carbohydrates. It is necessary to know the origin of low energy availability in a particular athlete in order to intervene effectively with her. The key behavior modification for preventing and treating the Triad is to increase energy availability, either by increasing dietary energy intake, reducing exercise energy expenditure, or both. Guidelines for doing so are provided.

Keywords: Female Athlete Triad, energy availability, menstrual function, bone mineral density, appetite, eating disorders, disordered eating, weight loss

Resumen

Loucks, A. (2014). La tríada de la atleta: un fenómeno metabólico. **PENSAR EN MOVIMIENTO: Revista de Ciencias del Ejercicio y la Salud, 12 (1),** 1-23. La tríada de la atleta (tríada) es un síndrome en el cual la baja disponibilidad de energía dispara una amplia gama de mecanismos endocrinos para disminuir el gasto energético, lo cual perjudica la salud reproductiva y esquelética. La disponibilidad energética es la cantidad de energía de la dieta que queda cada día, después del entrenamiento o el ejercicio, para todas las demás funciones fisiológicas. El tipo específico de mal funcionamiento reproductivo provocado por la baja disponibilidad energética son los trastornos menstruales hipotalámicos. Para asegurarse de que las atletas afectadas reciban la atención apropiada es necesario realizar pruebas endocrinas que sirven para diagnosticar los trastornos mencionados mediante la exclusión de otros trastornos menstruales no asociados con la tríada. La baja disponibilidad energética perjudica la salud esquelética debido al desacoplamiento del recambio óseo, en el cual la tasa de resorción aumenta mientras la tasa de formación de hueso desciende. El resultado es una pérdida progresiva de hueso o la incapacidad de acumular masa ósea, lo cual aumenta el riesgo de fracturas por estrés y de osteoporosis. La baja disponibilidad energética tiene su origen en al menos una de las siguientes tres fuentes: trastornos de la alimentación de tipo restrictivo, especialmente la anorexia nerviosa; los esfuerzos intencionales orientados a la pérdida de peso corporal o grasa corporal, para mejorar el rendimiento deportivo o la apariencia; y la supresión inadvertida del apetito causada por el ejercicio y por las dietas con un alto porcentaje de carbohidratos. Para poder realizar una intervención eficaz con cada atleta es necesario conocer el origen de su baja disponibilidad energética. La modificación clave de la conducta para la prevención y tratamiento de la tríada es el aumento de la disponibilidad energética, ya sea mediante el incremento de la ingesta energética en la dieta, la reducción en el gasto energético por ejercicio, o ambos. Por lo tanto, se ofrecen algunas pautas para lograr la disponibilidad energética correcta.

Palabras clave: Tríada de la atleta, disponibilidad energética, función menstrual, densidad mineral ósea, apetito, trastornos de la alimentación, pérdida de peso

Introduction

All girls and women are encouraged to participate in physical activities and sports, because the benefits of participation far outweigh the risks (Nattiv, Loucks, Manore, Sundgot-Borgen, & Warren, 2007). One of those risks is the Female Athlete Triad, in which reproductive and skeletal health are impaired by energy deficiency (Loucks, 2011; Loucks, Kiens, & Wright, 2011). The prevalence of the Triad varies greatly from team to team and from sport to sport, but it tends to be highest in esthetic, endurance, and weight-class sports in which a thin build confers a competitive advantage or qualifies an athlete for participation (Nattiv et al., 2007).

Each of the three components of the Triad spans a spectrum ranging from health to disease (Figure 1). The population of athletes is distributed across these spectrums, and individual athletes travel along them as changes in their diet and exercise habits affect the amount of energy they have available for reproductive and skeletal health. It should be understood that an athlete travels at different rates on each spectrum. She can change her diet and exercise behavior in a day, but changes in menstrual symptoms may not be noticed for a month, and changes in bone mineral density will not be measurable for at least 6 months. This review describes each component of the Triad, and the neuroendocrine mechanisms linking them together. It then explains how the Triad originates in unwise diet and exercise behavior, and closes with recommendations for prevention and treatment.

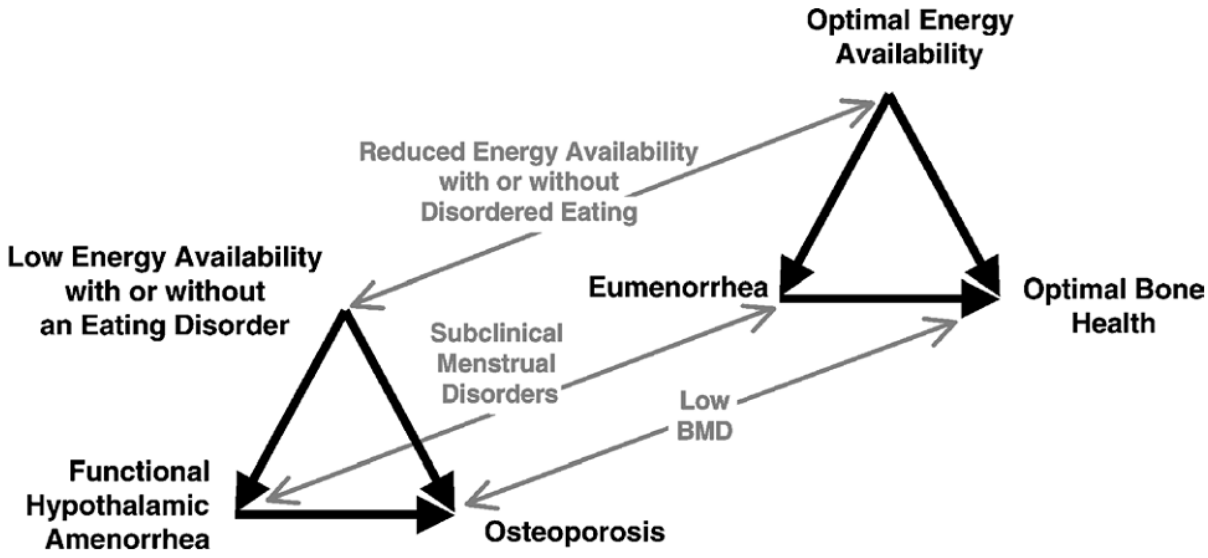

Figure 1. The spectrums of the Female Athlete Triad. (From Nattiv et al., American College of Sports Medicine Position Stand: the female athlete triad. *Medicine and Science in Sports and Exercise*, 39(10), 1867-1882, 2007. Used with permission. Promotional and commercial use of the material in print, digital or mobile device format is prohibited without the permission from the publisher Lippincott Williams & Wilkins. Please contact journalpermissions@lww.com for further information) (Nattiv et al., 2007). Each component of the Triad spans a range from health to disease, with low energy availability, hypothalamic amenorrhea and osteoporosis at the pathological end of the spectrums. The spectrums are linked by endocrine mechanisms. The effect of low energy availability on reproductive health is mediated by luteinizing hormone (LH), which regulates ovarian and thereby menstrual function. The resulting indirect effect of low energy availability on skeletal health is mediated by estrogen, which regulates the rate at which old bone is resorbed by osteoclasts. Energy availability also affects skeletal health directly by means of its influence on insulin, tri-iodothyronine (T_3) and insulin-like growth factor-1, which regulate the rate at which new bone is formed by osteoblasts. An athlete's current and history of diet and exercise behavior determine where she is located, and which direction she is moving, on each spectrum.

Components of the Triad

Energy Availability.

The concept of energy availability derives from the recognition that mammals expend dietary energy in several basic physiological processes, including thermoregulation, cellular maintenance, immunity, growth, reproduction and locomotion (Loucks, 2013). The energy expended in one of these processes is not available for the others. Therefore, energy availability can be defined for athletes as the amount of dietary energy remaining after exercise training for all of the body's other physiological processes. Energy availability determines how well those processes function.

By contrast, energy balance is the amount of energy added to or removed from the body's energy stores after all the body's physiological systems have done all of their work for the day. Energy balance determines whether you gain or lose weight. The brain responds to chronic, severe low energy availability by altering a wide spectrum of metabolic hormones that suppress diverse energy-consuming physiological processes (Laughlin & Yen, 1996; Loucks & Thuma, 2003). This tends to restore energy balance, but it is a pathological state of equilibrium in which infertility and skeletal demineralization are only part of the price paid to preserve life.

Some investigators have been skeptical of the diet records of female athletes, because comparisons of such records to estimations or measurements of energy expenditure have found apparently huge negative energy balances, some exceeding 4 MJ/d, in athletes with stable body weights (Edwards, Lindeman, Mikesky, & Stager, 1993; Wilmore et al., 1992). Such large discrepancies have been interpreted as indicating that female athletes grossly under-report their dietary intake, but few of these studies have included biochemical measurements to validate this interpretation. Under-reporting would not account for biochemical evidence of energy deficiency, and several studies characterizing reproductive disorders in female athletes (Laughlin & Yen, 1996; Laughlin & Yen, 1997; Loucks et al., 1992) have found metabolic substrates and hormones telling a consistent story of a decline in glucose utilization, mobilization of fat stores, and a slow metabolic rate, with more extreme abnormalities in amenorrheic athletes than in regularly menstruating athletes. So the available biochemical data clearly demonstrate that some female athletes are, indeed, energy deficient.

Menstrual Function.

Low energy availability impairs reproductive health by disrupting the pulsatile secretion of gonadotropin-releasing hormone by certain neurons in the hypothalamus. This, in turn, disrupts the pulsatile secretion of luteinizing hormone (LH) by the pituitary gland. Ovarian function critically depends not on the concentration of LH, but rather on the frequency of LH pulses (Tsutsumi & Webster, 2009). Athletes with menstrual disorders should not be assumed to have such functional hypothalamic menstrual disorders, because menstrual disorders can be symptoms of many medical conditions. Moreover, functional hypothalamic menstrual disorders can be caused by insufficient intestinal absorption (as in Celiac disease) as well as by insufficient dietary intake, and they can conceal underlying polycystic ovary disease (Sum & Warren, 2009). Therefore, to ensure that they receive appropriate care, athletes with menstrual

disorders should be properly diagnosed through a series of hormone measurements and neuroendocrine stimulation tests (American Society of Reproductive Medicine Practice Committee, 2008). Obviously, menstrual disorders due to other causes are not effectively treated by increasing energy availability.

Clinical menstrual disorders such as amenorrhea (no cycles for 3 or more months) and oligomenorrhea (cycles longer than 35 days) can be identified by interviewing athletes, but athletes with subclinical menstrual disorders such as anovulation (no ovulation) and luteal phase deficiency (inadequate progesterone secretion) menstruate regularly and are unaware that they have a menstrual disorder. One study of regularly menstruating runners found that almost 80% of them had subclinical menstrual disorders in one or more of three consecutive menstrual cycles (De Souza et al., 1998). Subclinical menstrual disorders can only be detected by measuring sex steroid hormones during the luteal phase of the menstrual cycle.

Reproductive function has been disrupted in rodents by dietary restriction; by administering drugs that block the oxidation of glucose and fatty acids; by insulin administration, which diverts blood glucose into storage while inhibiting the mobilization of fat stores; and by cold exposure, which consumes large quantities of metabolic fuels in thermogenesis; as well as by physical activity, which consumes metabolic fuels in muscular contractions (Wade & Jones, 2004; Wade & Schneider, 1992). The energy costs of systemic infections and major trauma probably have similar effects. In monkeys, amenorrhea has been induced by increasing their exercise energy expenditure without restricting their dietary energy intake (Williams et al., 2001), and then their menstrual cycles have been restored by increasing their dietary energy intake without moderating their exercise regimen (Williams, Helmreich, Parfitt, Caston-Balderrama, & Cameron, 2001). In healthy young women, LH pulsatility has been disrupted by extreme dietary restriction alone (Loucks & Heath, 1994), by extreme exercise energy expenditure alone (Loucks, Verdun, & Heath, 1998), and by the combination of moderate amounts of both (Loucks & Thuma, 2003). LH pulsatility has also been preserved in strenuously exercising women by increasing their dietary energy intake in compensation for their exercise energy expenditure (Loucks et al., 1998). Thus, exercise has no suppressive effect on reproductive function apart from the impact of its energy cost on energy availability.

The dose-response effects of energy availability on LH pulsatility have also been determined in a prospective experiment (Loucks & Thuma, 2003). Healthy, regularly menstruating, habitually sedentary, young women expended 15 $kcal*kgFFM^{-1}*d^{-1}$ walking on a motorized treadmill at 70% VO2max. They completed this exercise in about 100 min each day for 5 days in 2 trials, separated by at least 2 months to allow for the effects of the first trial to be washed out before the second trial began. One trial was performed with an energy intake of 60 $kcal*kgFFM^{-1}*d^{-1}$ (250 $kJ*kgFFM^{-1}*d^{-1}$) and one with an energy intake of 45, 35 or 25 $kcal*kgFFM^{-1}*d^{-1}$ (188, 146 or 105 $kJ*kgFFM^{-1}*d^{-1}$) for net energy availabilities of 45 and either 30, 20 or 10 $kcal*kgFFM^{-1}*d^{-1}$ (125, 96 or 48 $kJ*kgFFM^{-1}*d^{-1}$). LH pulsatility was disrupted below a threshold of energy availability at 30 $kcal*kgFFM^{-1}*d^{-1}$ (125 $kJ*kgFFM^{-1}*d^{-1}$). This threshold of

energy availability is similar to resting metabolic rate, whereas energy balance in a healthy adult occurs at 45 kcal∗kgFFM^{-1}∗d^{-1} (188 kJ∗kgFFM^{-1}∗d^{-1}).

By contrast, reproductive function does not depend on the amount of energy stored in body fat, as was also once believed. Almost all observational studies of amenorrheic and eumenorrheic athletes have found them to span a common range of body size and composition (Redman & Loucks, 2005). Moreover, after gastric bypass surgery women become amenorrheic while they are still greatly overweight (e.g., body weight = 97 kg, BMI = 35 kg/m^2) (Di Carlo et al., 1999).

Bone Mineral Density.

Low energy availability impairs skeletal health by uncoupling the turnover of bone in a manner that lowers bone mineral density over time (Christo et al., 2008; De Souza et al., 2008; Pollock et al., 2010). Like other tissues, bone is constantly turning over as osteoclast cells resorb old bone and osteoblast cells form new bone. By disrupting ovarian function, low energy availability lowers estrogen levels. This increases the rate of bone resorption by osteoclasts. Low energy availability also lowers the levels of insulin and tri-iodothyronine (T_3), and thereby insulin-like growth factor-1 (IGF-1). This suppresses the rate of bone formation by osteoblasts. Peripheral signals of low energy availability, such as falling insulin, rising ghrelin, rising peptide YY (PYY) and falling leptin levels, also act centrally to activate neuropeptide Y (NPY) secreting neurons in the hypothalamus, which act via sympathetic pathways on osteoblastic Y1 receptors to repress osteoblast activity and bone formation (Shi & Baldock, 2012). Amenorrheic athletes display elevated ghrelin and PYY, also known as peptide tyrosine tyrosine, as well as lower leptin levels (Ackerman et al., 2012; Scheid, Williams, West, VanHeest, & De Souza, 2009). Conversely, when energy availability increases, NPY expression is reduced, osteoblast activity is increased, bone formation is stimulated, and the mass of both cortical and cancellous bone increases in a generalized manner throughout the skeleton (Shi & Baldock, 2012). In adults, a greater rate of bone resorption than formation causes a progressive loss of bone mass. In adolescence, such uncoupling of the rates of bone resorption and formation prevents girls from accruing as much bone mass as other girls (Barrack, Rauh, & Nichols, 2010). The resulting bone loss predisposes women to stress fractures (Goolsby, Barrack, & Nattiv, 2012; Okamoto, Arai, Hara, Tsuzihara, & Kubo, 2010; Popp et al., 2009) in the near term and to the premature onset of osteoporosis later in life.

The dose-response effects of energy availability on bone turnover have also been determined (Ihle & Loucks, 2004). The rate of bone resorption increased when energy availability was low enough to reduce estradiol levels. The rate of bone protein synthesis, indicated by the plasma concentration of the bone formation marker Type I procollagen carboxy-terminal propeptide (PICP), was found to decline linearly with energy availability, as did insulin. Insulin stimulates osteoblast differentiation (Lu, Kraut, Gerstenfeld, & Graves, 2003). Meanwhile, the rate of bone mineralization, indicated by the plasma concentration of the bone formation marker osteocalcin, was found to decline non-linearly with energy availability, with most of the decline occurring between 20 and 30 kcal∗kgFFM^{-1}∗d^{-1} (96 and 125 kJ∗kgFFM^{-1}∗d^{-1}), in parallel with the non-linear responses of IGF-1 and T_3. Osteocalcin is the glue that binds

bone mineral to bone protein. Its secretion is mediated by IGF-1, the hepatic production of which is stimulated by growth hormone and modulated by T_3 (Wolf, Ingbar, & Moses, 1989).

Origins of Low Energy Availability

In athletes, low energy availability originates from three sources that we might describe as obsessive, intentional and inadvertent. The obsessive source is anorexia nervosa and other restrictive eating disorders; the intentional source is purposeful effort to reduce weight or body fat; and the inadvertent source is the suppression of appetite by diet and exercise. It is important to identify the origin of low energy availability in an athlete in order to understand how to develop a strategy for modifying her diet and exercise behavior to correct it.

Obsessive Low Energy Availability.

The prevalence of anorexia nervosa has been reported to be higher in "thin build" sports (dance, gymnastics, light-weight rowing, long distance running, diving and swimming) than other sports (5% vs 0%) (Byrne & McLean, 2002), and even higher in "aesthetic" sports (i.e., gymnastics, dance, figure skating, aerobics, and diving) than other sports (12% vs 1%) (Sundgot-Borgen & Torstveit, 2004). The highest prevalence has been found in dance (17%-33%) (Brooks-Gunn, Warren, & Hamilton, 1987; Evers, 1987; Gadpaille, Sanborn, & Wagner, 1987; Holderness, Brooks-Gunn, & Warren, 1994) and ice skating (48%) (Rucinski, 1989).

Anorexia nervosa is a clinical mental illness, often accompanied by other mental illnesses (Klump, Bulik, Kaye, Treasure, & Tyson, 2009), requiring psychiatric treatment and sometimes even unwilling inpatient treatment with forced feeding (Carney, Tait, Richardson, & Touyz, 2008). Anorexia nervosa also has one of the highest risks of premature death of any mental illness (Harris & Barraclough, 1998) with a mortality 10 times higher than that of age and sex matched peers (Birmingham, Su, Hlynsky, Goldner, & Gao, 2005). Sixty percent of deaths in anorexia nervosa are due to medical consequences of the disease, for which the mortality risk is increased four times (Harris & Barraclough, 1998). The other 40% of deaths due to accident, misadventure, homicide, and suicide are increased 11 times, and the specific risk of suicide is increased 32 times (Harris & Barraclough, 1998).

Because the mortality of anorexia nervosa is so high, and because coaches, sports dietitians and team physicians are not trained to care for clinical mental illnesses, they should not be expected to manage them, and they should not attempt to do so. Instead, sports organizations should establish procedures for identifying athletes who may have anorexia nervosa, referring them for psychiatric evaluation and care, and excluding them from participation until they receive psychiatric clearance. Athletes who do not comply with recommendations to modify diet and exercise behavior to increase energy availability should be referred.

Intentional Low Energy Availability.

Coaches should employ their knowledge and skills to help cooperative athletes to perfect their performance. Athletic performance is improved, in part, by acquiring an optimum sport-

specific (and, in team sports, position-specific) body size, body composition and mix of energy stores. For many female athletes, these objectives may include a reduction in fat mass. Therefore, many of these athletes will need to pursue diet and exercise regimens that reduce their energy availability. Because of the dependence of reproductive and skeletal health on energy availability, this will place their reproductive and skeletal health at risk. That risk should be acknowledged, understood, and carefully minimized through the cooperation of coaches and dietitians to manage energy availability so that athletes achieve their athletic potential without sacrificing their reproductive and skeletal health.

When athletes seek to reduce their weight or body fat, they reduce their energy availability by reducing their dietary energy intake or by increasing their exercise energy expenditure. Many female athletes do both, but athletes in aesthetic sports tend to emphasize dietary restriction, while high energy expenditure is inherent in endurance sports. In physically active women, the compounding of exercise energy expenditure with cognitive dietary restraint is associated with an increased frequency of menstrual disorders and low bone mineral density (Barrack, Rauh, Barkai, & Nichols, 2008; Vescovi, Scheid, Hontscharuk, & De Souza, 2008). Some poorly informed athletes may also practice disordered eating behaviors (e.g., skipping meals, fasting, vomiting and using laxatives) in impatient pursuit of potentially unhealthful objectives (Rauh, Nichols, & Barrack, 2010; Thein-Nissenbaum, Rauh, Carr, Loud, & McGuine, 2011). Coaches and dietitians should cooperate to help athletes to correct disordered eating behaviors, to set healthful objectives, and to pursue these objectives on a healthful schedule.

Inadvertent Low Energy Availability.

Athletes of both sexes are susceptible to unintended and unnoticed reductions in energy availability due to the suppression of appetite by diet and exercise (Loucks et al., 2011). This appetite suppression is possible even during training periods when weight or fat loss is not a training objective, and even among athletes who do not suffer from eating disorders or practice disordered eating behaviors. Despite extensive evidence that appetite is not a reliable indicator of energy requirements in either trained or untrained men and women, this problem is entirely neglected in the current joint position stand on sports nutrition of the American College of Sports Medicine, the American Dietetic Association, and Dietitians of Canada (Rodriguez, DiMarco, & Langley, 2009).

Briefly, after exercise, *ad libitum* energy intake does not increase sufficiently to compensate for exercise energy expenditure (Ballard et al., 2009; Bergouignan et al., 2010; Borer, 2010; King, Miyashita, Wasse, & Stensel, 2010; King et al., 2009; King, Hopkins, Caudwell, Stubbs, & Blundell, 2008; Ueda, Yoshikawa, Katsura, Usui, & Fujimoto, 2009): athletes who expend the most energy are susceptible to the largest deficiencies (Whybrow et al., 2008). Appetite is further suppressed by diets containing a high percentage (65%) of carbohydrates (Horvath, Eagen, Ryer-Calvin, & Pendergast, 2000; Stubbs et al., 2004). The unexpected and unintended consequence of such diets is that *ad libitum* carbohydrate intake (Horvath, Eagen, Ryer-Calvin, et al., 2000) and athletic performance (Horvath, Eagen, Fisher, Leddy, & Pendergast, 2000) can be lower than with a lower percentage carbohydrate diet. In endurance sports, appetite can be so suppressed that energy availability is reduced below 30

$kcal*kgFFM^{-1}*d^{-1}$ (125 $kJ*kgFFM^{-1}*d^{-1}$). Just as thirst is an unreliable indicator of water requirements, appetite is an unreliable indicator of energy requirements in endurance sports. Therefore, coaches should warn endurance athletes to eat not according to their appetites, but according to a plan with specified amounts of selected foods at scheduled times. Dietitians can develop eating plans to achieve target energy availabilities for specified exercise regimens.

Reasons for Focusing Attention on Female Athletes.

The physiological mechanisms linking the components of the Triad operate in men as well as women, but more women than men engage in diet and exercise behaviors that reduce energy availability. Coaches should keep in mind that before female athletes are athletes, they are female. Worldwide, about twice as many college women as college men *at every decile of body mass index* perceive themselves to be overweight (Wardle, Haase, & Steptoe, 2006). The numbers of women and men actively trying to lose weight are even more disproportionate, and the disproportion *increases* as BMI declines. As a result, 5-9 times as many women as men in the three lowest deciles of BMI are actively trying to lose weight (Wardle et al., 2006). Indeed, more young female athletes report improvement of appearance than improvement of performance as a reason for dieting (Martinsen, Bratland-Sanda, Eriksson, & Sundgot-Borgen, 2010). This means that social issues may need to be addressed before female athletes will reform their diet and exercise behavior to increase energy availability.

Furthermore, although severe dietary restriction alone is sufficient to disrupt reproductive function, the more physically active a woman is, the less dietary restriction is required, and if she expends enough energy in exercise, she does not need to restrict her diet at all (Loucks et al., 1998). The health and longevity of experimental animals have been improved by restricting their energy intake by as much as 30% (Mattison, Lane, Roth, & Ingram, 2003), but restrictions of 40% have consistently caused both infertility (Holehan & Merry, 1985; McShane & Wise, 1996) and skeletal demineralization(Talbott, Rothkopf, & Shapses, 1998). In exercising women, reproductive function begins to be suppressed when energy availability falls more than 33% (Loucks & Thuma, 2003), and amenorrheic athletes have been reported to practice diet and exercise regimens that reduce energy availability by as much as 65% (Thong, McLean, & Graham, 2000).

Managing Energy Availability

Historically, dietitians have sought to assess the dietary energy needs of athletes by measuring their energy expenditure. In practice, an athlete's total energy expenditure would be measured, or the athlete's resting metabolic rate would be measured and multiplied by an activity factor to estimate her total energy expenditure. However, measures of energy expenditure convey no information about whether the functioning of physiological systems and, thereby, energy expenditure, have been suppressed by low energy availability. Therefore, the energy requirements of athletes cannot be determined by measuring their total energy expenditure or by measuring their resting metabolic rate and multiplying by an activity factor. In contrast, because energy availability measures only an athlete's diet and exercise behavior, it is not confounded by the athlete's physiological responses to that behavior. As the amount of

dietary energy governing the function of physiological systems, energy availability is the quantity that athletes, trainers, coaches and sports dietitians need to know and manage.

Thus, the key behavior modification for preventing and treating the Triad is to keep energy availability above 30 kcal∗kgFFM^{-1}∗d^{-1}. Energy availability (EA) is determined by measuring dietary energy intake (EI), exercise energy expenditure (EEE), and fat free mass (FFM). Various inexpensive methods are available for making these measurements. Then:

$$EA = (EI – EEE)/FFM \quad 1$$

(See the EA Calculation box.) Conversely, the EI required to achieve a target energy availability for any particular EEE can be determined by rearranging Equation 1:

$$EI = EEE + EA \times FFM \quad 2$$

EA Calculation

A runner with a body mass of 53 kg and a body fat percentage of 15% would have a fat-free mass of 45 kg. If her energy intake is 2100 kcal/day and she runs 5 miles (8 km) per day with EEE = 100 kcal∗mile^{-1}, then her energy availability would be:

$$EA = (2100 – 5*100)/45 = 35.6 \text{ kcal}*\text{kgFFM}^{-1}*\text{d}^{-1} (149 \text{ kJ}*\text{kgFFM}^{-1}*\text{d}^{-1}) \quad 1a$$

At this energy availability above 30 kcal∗kgFFM^{-1}∗d^{-1} (125 kJ∗kgFFM^{-1}∗d^{-1}), her reproductive function would likely be protected as her body fat declined. If she increased her training mileage to 8 miles per day (≈13 km) without increasing her energy intake, her energy availability would be reduced to:

$$EA = (2100 – 8*100)/45 = 28.9 \text{ kcal}*\text{kgFFM}^{-1}*\text{d}^{-1} (121 \text{ kJ}*\text{kgFFM}^{-1}*\text{d}^{-1}) \quad 1b$$

This level of energy availability is near the threshold at which the reproductive system begins to be impaired. If she further increased her training mileage to 10 miles/day (16 km per day), her energy availability would be further reduced to:

$$EA = (2100 – 10*100)/45 = 24.4 \text{ kcal}*\text{kgFFM}^{-1}*\text{d}^{-1} (102 \text{ kJ}*\text{kgFFM}^{-1}*\text{d}^{-1}) \quad 1c$$

At this low level of energy availability, not only would her reproductive function likely be impaired, but her metabolic rate might also slow enough to prevent further weight loss. The athlete would need to consume 2,250 and 2,925 kcal/day (9.4 and 12.2 MJ/day) to achieve EA = 30 and 45 kcal∗kgFFM^{-1}∗d^{-1} (125 and 188 kJ∗kgFFM^{-1}∗d^{-1}), respectively.

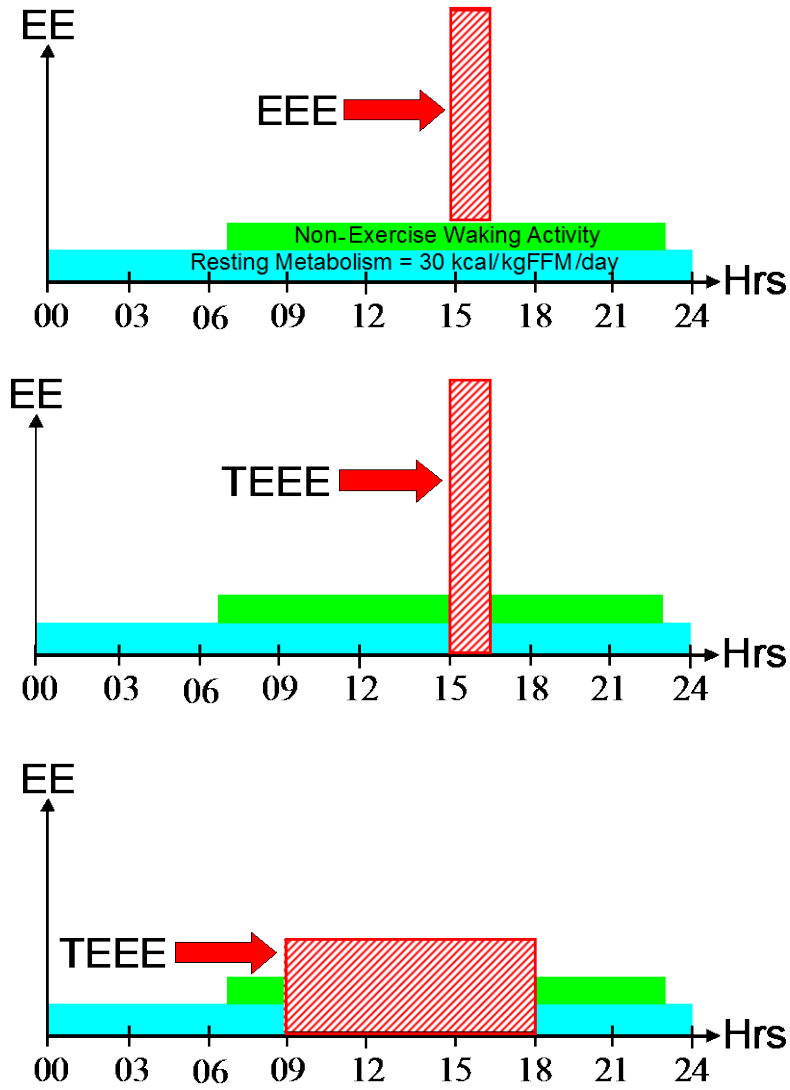

Figure 2. Calculation of exercise energy expenditure (EEE). (A. Top) EEE is the amount of energy that a woman expends because she is an athlete, and does not include the energy she expends in resting metabolism and other waking activities. (B. Middle) Ergometers measure total energy expenditure during exercise (TEEE), which overestimates EEE by ~2 kcal•kgFFM^{-1}•d^{-1} per hour of exercise. For high intensity exercise of short duration, the resulting error in calculating energy availability as EA = (EI – TEEE)/FFM is negligibly small for clinical purposes. (C. Bottom) For low intensity exercise of long duration, however, the error in EA = (EI – TEEE)/FFM is very large and will lead to unwarranted changes in diet and exercise behavior.

Having chosen EEE and EA to achieve the purposes of a particular period of training, the athlete should then consume the calculated EI. Coaches and dietitians should be aware that

when EEE is large, as it is in endurance sports, consuming the EI necessary to achieve even EA > 30 kcal$*$kgFFM$^{-1}*$d^{-1} (125 kJ$*$kgFFM$^{-1}*$d^{-1}) may require athletes to eat far beyond their appetites. Therefore, the willingness of an athlete to eat enough to protect her health may limit her EEE.

Coaches and dietitians should also be aware that EEE in Equations 1 and 2 is not the total energy expenditure during exercise that would be measured by an ergometer. Rather, it is the extra energy expenditure beyond the energy that the athlete would have expended if she had not exercised (See Figure 2 and the EEE Calculation box.). To determine EEE, energy expenditure in non-exercise activities (NEEE) during waking hours should be subtracted from total energy expenditure during exercise (TEEE):

$$EEE = TEEE - NEEE \qquad 3$$

EEE Calculation

The resting metabolism (RM) of an athlete in energy balance on a non-exercising day would be about 2/3 of her EI. For EI = 2100 kcal/day (8.8 MJ/day), RM = 1400 kcal/day (5.8 MJ/day), or 58 kcal/hour (244 kJ/hr). If she sleeps 8 hours, the rest of her EI would be expended in routine waking activities (WEE) at an average rate of WEE = 700 kcal/16 hrs = 44 kcal/hr (182 kJ/hr). If her fat-free mass (FFM) is 45 kg, then her non-exercise energy expenditure (NEEE) would be:

$$NEEE = (RM + WEE)/FFM = (58 + 44)/45 = 2.3 \text{ kcal/hr } (9.5 \text{ kJ/hr})$$

If the athlete's total energy expenditure during a 40-min run is TEEE = 500 kcal, then:

$$EEE = TEEE – NEEE = 500/45 – (2/3)*2.3 = 11.1 – 1.5 = 9.6 \text{ kcal/kgFFM} \qquad 3a$$

For such brief, high intensity exercise, NEEE is too small to cause an error in judgment about the adequacy of EA. If the same TEEE had been expended in 4 hours of gymnastics training, however, NEEE would be too large to ignore:

$$EEE = TEEE – NEEE = 500/45 – 4*2.3 = 11.1 – 9.2 = 1.9 \text{ kcal/kgFFM} \qquad 3b$$

If the gymnast were to restrict her dietary intake to EI = 1575 kcal/day, ignoring NEEE would lead to excessive concern about her EA and unwarranted demands for behavior modifications:

With NEEE: EA = (EI – EEE)/FFM = 1575/45 – 1.9 = 33.1 kcal$*$kgFFM$^{-1}*$d^{-1} (138 kJ$*$kgFFM$^{-1}*$d^{-1})

Without NEEE: EA = EI – EEE)/FFM = 1575/45 – 9.6 = 25.4 kcal$*$kgFFM$^{-1}*$d^{-1} (106 kJ$*$kgFFM$^{-1}*$d^{-1})

EA Error Analysis

The errors in EA caused by errors in %Fat, EEE, and EI can be estimated from realistic values of these quantities. Consider an athlete with body mass = 60 kg, %Fat = 25%, EEE = 500 kcal/d, and EI = 2100 kcal/d (8.8 MJ/d). Her FFM is (1 – 0.25)*60 = 45 kg and her EA is

$$EA = (EI – EEE)/FFM = (2100 – 500)/45 = 35.6\ kcal*kgFFM^{-1}*d^{-1}\ (149\ kJ*kgFFM^{-1}*d^{-1})$$

2% errors in %Fat are not uncommon with body composition analyzers. A 2% overestimate of %Fat (27%) leads to an underestimate of FFM (43.8 kg) and a negligible error in EA:

$$EA = (2100 – 500)/43.8 = 36.5\ kcal*kgFFM^{-1}*d^{-1}\ (153\ kJ*kgFFM^{-1}*d^{-1})$$

A 10% error in EEE would correspond to a runner erring by half a mile in the length of a 5 mile run. A 10% underestimation of EEE leads to a similarly negligible error in EA:

$$EA = (2100 – 450)/45 = 36.7\ kcal*kgFFM^{-1}*d^{-1}\ (153\ kJ*kgFFM^{-1}*d^{-1})$$

Underestimations of EI as big as 20% have been suspected by some dietitians. A 20% underestimation of EI would lead to a large error in EA:

$$EA = (0.8*2100 – 500)/45 = 26.2\ kcal*kgFFM^{-1}*d^{-1}\ (110\ kJ*kgFFM^{-1}*d^{-1})$$

Even a 10% underestimation of EI would lead to a substantial error in EA:

$$EA = (0.9*2100 – 500)/45 = 30.9\ kcal*kgFFM^{-1}*d^{-1}\ (129\ kJ*kgFFM^{-1}*d^{-1})$$

In this case, 10% of EI (210 kcal) is similar to the energy content of 2-3 slices of bread. Therefore, accurate estimations of EA depend most importantly on complete dietary records.

Coaches and dietitians should be aware, too, that some devices measure the work done by an athlete rather than the amount of energy she expends; the latter is 4-5 times bigger due to the chemical-to-mechanical energy conversion efficiency of skeletal muscle being only 20% to 25%. Another caution is also warranted. Measurement errors in EI, EEE and FFM have different effects on the error in estimating EA. (See the Error Analysis box.) A few simple calculations with realistic values quickly reveal that the greatest efforts should be made to record EI accurately.

Besides coaches and dietitians, parents, team physicians and sport-governing bodies all have roles to play for preventing and treating the Triad. Everyone will need to experiment with

educational programs, training regimens, intervention strategies and rule changes to achieve this objective, and to publicize the results of their experiments to teach others the lessons learned. Different challenges will emerge in different sports, requiring different solutions. For example, the rule changes adopted by US collegiate men's wrestling aim to prevent harmful weight-loss practices by controlling athlete behavior (NCAA, 2013). The associated procedures are intrusive, complicated, bureaucratic, time-consuming, labor-intensive and expensive. By contrast, the rules adopted by international ski-jumping aim to achieve the same objective by reducing the competitive incentive for athletes to engage in harmful weight loss behavior, and are elegantly simple (Müller, Groschl, Müller, & Sudi, 2006). Experience will teach whether initial reforms need further refinement (Müller, 2009; Oggiano & Saetran, 2008).

Conclusion

To prevent the Triad, female athletes should seek to improve their athletic performance by managing energy availability in a periodized training program that aims to achieve interim goals on the way to long-term objectives. The goals for some periods will call for energy availability to be raised and others will call for it to be lowered, but there is a floor below which it cannot be reduced without impairing reproductive and skeletal health. The first aim of treatment for the Triad is to increase energy availability. This requires an understanding of the origins of low energy availability affecting particular athletes. Inexpensive commercial devices are available for facilitating the management of energy availability, and guidelines are offered here for achieving that objective.

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