Journal of Sports Sciences
Publication details, including instructions for authors and subscription information:
http://www.informaworld.com/smpp/title~content=t713721847

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Online Publication Date: 01 December 2007
To cite this Article: Manore, Melinda M., Kam, Lynn Ciadella and Loucks, Anne B. (2007) 'The female athlete triad: Components, nutrition issues, and health consequences', Journal of Sports Sciences, 25:1, S61 - S71
To link to this article: DOI: 10.1080/02640410701607320
URL: http://dx.doi.org/10.1080/02640410701607320

Journal of Sports Sciences, 2007; 25(S1): S61 – S71

Routledge
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The female athlete triad: Components, nutrition issues, and health consequences

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(Accepted 2 August 2007)

Abstract
This paper, which was part of the International Association of Athletics Federations (IAAF) 2007 Nutritional Consensus Conference, briefly reviews the components of the female athlete triad (Triad): energy availability, menstrual status, and bone health. Each component of the Triad spans a continuum from health to disease, and female athletes can have symptoms related to each component of the Triad to different degrees. Low energy availability is the primary factor that impairs menstrual dysfunction and bone health in the Triad. We discuss nutritional issues associated with the Triad, focusing on intakes of macronutrients needed for good health, and stress fractures, the most common injury associated with the Triad. Finally, we briefly discuss screening and treatment for the Triad and the occurrence of the Triad in men.

Keywords: *Energy availability, energy balance, energy intake, women, exercise, menstrual dysfunction, amenorrhoea, oligomenorrhoea, men, stress fractures, bone mineral density, eating disorders, disordered eating*

Introduction

The American College of Sports Medicine (ACSM) Position Stand on the Female Athlete Triad (Otis, Drinkwater, Johnson, Loucks, & Wilmore, 1997) identified the Triad as an association of eating disorders, amenorrhoea, and osteoporosis. The Triad is now recognized as a complex set of interrelationships between energy availability, menstrual status, and bone health, each of which occurs on a continuum between health and disease. Girls and women participating in sports that emphasize a low body weight are more likely than other active women to restrict energy availability.

Components of the Triad

Energy availability

Dietary energy is utilized in basic physiological processes such as cellular maintenance, thermogenesis, immunity, growth, reproduction, and locomotion. Any energy used in one of these processes is not available for the others. For understanding the physiology of exercise, it is useful to define energy availability (EA) as dietary energy intake (EI) minus exercise energy expenditure (EEE). This is the amount of dietary energy remaining after exercise training for all other physiological processes. Since more energy is expended in fat-free mass (FFM) than in fat mass, it is also useful to normalize energy availability to fat-free mass so that EA = (EI – EEE)/FFM.

In healthy, young adults, energy balance occurs at an energy availability of $\sim$45 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} (188 kJ $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}) (Loucks & Thuma, 2003; Loucks, Verdun, & Heath, 1998; Mulligan & Butterfield, 1990), and resting metabolic rate is $\sim$30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} (125 kJ $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}) (Beidleman, Puhl, & De Souza, 1995; Fogelholm *et al.*, 1995; Mulligan & Butterfield, 1990; Myerson *et al.*, 1991; Thompson & Manore, 1996; Wilmore *et al.*, 1992). When energy availability is reduced below 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}, the body suppresses reproductive function (Loucks & Thuma, 2003) and bone formation (Ihle & Loucks, 2004). This tends to restore energy balance and extend survival, but impairs reproductive and skeletal health.

Athletes reduce energy availability by increasing exercise energy expenditure more than energy intake,

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ISSN 0264-0414 print/ISSN 1466-447X online © 2007 Taylor & Francis
DOI: 10.1080/02640410701607320

or by reducing energy intake more than exercise energy expenditure. In endurance sports, this may occur inadvertently, because *ad libitum* energy intake does not compensate for exercise energy expenditure (Horvath, Eagen, Fisher, Leddy, & Pendergast, 2000; Stubbs *et al.*, 2004). The resulting energy deficiency is more extreme when endurance athletes consume a high carbohydrate diet (Horvath *et al.*, 2000; Stubbs *et al.*, 2004). Many athletes reduce energy availability intentionally to optimize body size and composition for competitive success. Some athletes practise disordered eating behaviours, including fasting, skipping meals, purging, and using diet pills, laxatives, and diuretics (Beals & Hill, 2006; Johnson, Powers, & Dick, 1999; Sundgot-Borgen & Torstveit, 2004). For some, low energy availability expresses an eating disorder, which is a life-threatening clinical mental illness that requires medical and psychiatric treatment (Rome *et al.*, 2003). Female athletes may also reduce energy availability for reasons unrelated to sports. Throughout the world, more college-age females than males *at every decile of body mass index* perceive themselves to be overweight and actively try to lose weight (Wardle, Haase, & Steptoe, 2006). This difference increases with leanness so that up to nine times as many young lean women as men are actively trying to lose weight (Wardle *et al.*, 2006).

For whatever reason, female runners report that they consume 30% less energy and carbohydrate per kilogram of body weight than male runners (Burke, Cox, Culmmings, & Desbrow, 2001). Some investigators have been sceptical of the diet records of female athletes, because estimations of their exercise energy expenditure by doubly labelled water have been 20–30% higher (Beidleman *et al.*, 1995; Edwards, Lindeman, Mikesky, & Stager, 1993; Hill & Davies, 2002; Mulligan & Butterfield, 1990; Wilmore *et al.*, 1992), but under-reporting food intake does not explain the biochemical (Laughlin & Yen, 1996), physiological (James *et al.*, 2001; Myerson *et al.*, 1991), and pathophysiological (De Souza *et al.*, 1998; De Souza, Van Heest, Demers, & Lasley, 2003; Loucks, Mortola, Girton, & Yen, 1989) evidence of chronic energy deficiency.

Athletes should aim to maintain energy availability between 30 and 45 kcal · kg $FFM^{-1} \cdot day^{-1}$ (125–188 kJ · kg $FFM^{-1} \cdot day^{-1}$) for weight loss, near 45 kcal · kg $FFM^{-1} \cdot day^{-1}$ for weight maintenance, and >45 kcal · kg $FFM^{-1} \cdot day^{-1}$ for growth and carbohydrate loading (see Table I).

Menstrual status

Clinical menstrual disorders – oligomenorrhoea (menstrual cycles $\geq$35 days) and amenorrhoea (no cycles for >90 days) – are obvious to the affected women, but sub-clinical menstrual disorders – luteal deficiency (inadequate progesterone production during the luteal phase) and anovulation (no ovulation) – are not. Amenorrhoea may be caused by a wide range of organic diseases, genetic abnormalities, energy deficiency, and perhaps stress, although an effect of stress independent of energy availability has not been demonstrated. Medical tests are required to diagnose the aetiology of amenorrhoea so that appropriate care can be offered.

The type of amenorrhoea caused by low energy availability is classified as functional hypothalamic amenorrhoea. In functional hypothalamic amenorrhoea, ovarian function is suppressed by an abnormally slow frequency of luteinizing hormone pulses in the blood. Luteinizing hormone pulsatility is regulated in part by neurological pathways originating in specialized neurons that sense the availability of oxidizable metabolic fuels (Wade & Jones, 2004).

In animal experiments, reducing energy intake by more than 33% causes infertility and delays puberty (Holehan & Merry, 1985; McShane & Wise, 1996; Merry & Holehan, 1979). In female monkeys, amenorrhoea has been induced by increasing exercise energy expenditure without changing energy intake (Williams *et al.*, 2001a), and then ovulation has been restored by increasing energy intake without reducing exercise energy expenditure (Williams, Helmreich, Parfitt, Caston-Balderrama, & Cameron, 2001b). In experiments on exercising women, luteinizing hormone pulse frequency was suppressed within 5 days when energy availability was reduced below 30 kcal · kg $FFM^{-1} \cdot day^{-1}$ (Loucks & Thuma, 2003), and the suppression of luteinizing pulse frequency was prevented by increasing energy intake in compensation for exercise energy expenditure (Loucks *et al.*, 1998).

When the energy cost of running is estimated to be 90 kcal · $mile^{-1}$ (235 kJ · km^{-1}), the energy availability of amenorrhoeic runners described in published reports is consistently <30 kcal · kg $FFM^{-1} \cdot day^{-1}$ (Deuster *et al.*, 1986; Drinkwater *et al.*, 1984; Kaiserauer, Snyder, Sleeper, & Zierath, 1989; Kopp-Woodroffe, Manore, Dueck, Skinner, & Matt, 1999; Marcus *et al.*, 1985; Myerson *et al.*, 1991; Nelson *et al.*, 1986; Thong, McLean, & Graham, 2000; Wilmore *et al.*, 1992). The same is true of many eumenorrhoeic runners (De Souza *et al.*, 1998; Marcus *et al.*, 1985; Wilmore *et al.*, 1992; Winters-Stone & Snow, 2004), 78% of whom have sub-clinical menstrual disorders at least one month out of three (De Souza *et al.*, 1998). In the few case studies of nutritional interventions published to date, menstrual cycles were restored in amenorrhoeic runners by increasing their energy availability to $\geq$30 kcal · kg $FFM^{-1} \cdot day^{-1}$ (Kopp-Woodroffe *et al.*, 1999).

Table I. Examples of energy availability calculations.

Example	Body weight (kg)	Body fat (%)	FFM (kg)	EI (kcal · day^{-1})	EEE (kcal · day^{-1})	EA = (EI – EEE)/FFM (kcal · kg FFM^{-1} · day^{-1})
Low energy availability	61.5	13.5	53.2	1422	520	17
Weight loss	61.5	13.5	53.2	2382	520	35
Weight maintenance	61.5	13.5	53.2	2914	520	45
Carbohydrate loading	61.5	13.5	53.2	3192	0	60

Abbreviations: FFM = fat-free mass; EI = energy intake; EEE = exercise energy expenditure.

The prevalence of amenorrhoea varies widely with sport, age, training volume, and body weight (Redman & Loucks, 2005). Among collegiate runners, the prevalence was found to increase from 3% to 65% as training mileage increased from less than 8 miles · week^{-1} (13 km · week^{-1}) to greater than 70 miles · week^{-1} (113 km · week^{-1}), while body weight decreased from more than 60 kg to less than 50 kg (Sanborn, Martin, & Wagner, 1982).

The prevalence of menstrual disorders in the general population declines dramatically during the first decade after menarche (Vollman, 1977), and the dependence of luteinizing hormone pulsatility on energy availability also declines dramatically during adolescence (Loucks, 2006). Accordingly, the prevalence of amenorrhoea was found to be 9% among marathon runners who were older than 15 years of gynecological age (years since menarche) and 67% among those who were younger (Baker, Mathur, Kirk, & Williamson, 1981).

Bone health

Osteoporosis is ''a skeletal disorder characterized by compromised bone strength predisposing a person to an increased risk of fracture'' that develops not only through bone loss during adulthood, but also through the failure to accumulate sufficient bone mass during adolescence (NIH Consensus Development Panel on Osteoporosis Prevention, Diagnosis and Therapy, 2001). Bone strength and the risk of fracture depend not only on bone mineral but also on bone protein (Burr, 2002). Each constitutes about 40% of bone volume. Mineral gives bone its stiffness (i.e. the amount of force required to bend it), while protein gives bone its toughness (i.e. the amount of energy required to break it) (Burr, 2002).

Nevertheless, osteoporosis is diagnosed in post-menopausal women on the basis of bone mineral density alone, because the fracture rate in post-menopausal women doubles for each reduction of 1 standard deviation in bone mineral density (Hui, Slemenda, & Johnston, 1989). Unfortunately, epidemiological data relating bone mineral density to fractures are lacking in pre-menopausal women, and there is no agreement on standards for adjusting bone mineral density for bone size, pubertal stage, skeletal maturity or body composition in growing adolescents. Therefore, fracture risk cannot be predicted in these populations on the basis of bone mineral density alone (International Society for Clinical Densitometry, 2004).

Therefore, the International Society for Clinical Densitometry (ISCD) recently recommended that bone mineral density be quantified in children and pre-menopausal women in terms of *Z*-scores, which compare individuals to age-, race-, and sex-matched controls, and that osteoporosis be diagnosed in pre-menopausal women and children by *the combination* of a bone mineral density *Z*-score below -2.0 *together with* the presence of secondary risk factors for fracture, including under-nutrition, hypogonadism, and a history of fractures (International Society for Clinical Densitometry, 2004).

In animal experiments, reducing energy intake by more than 33% lowers bone mass, bone mineral density, and bone strength (Kalu, Masoro, Yu, Hardin, & Hollis, 1988; McCay, Crowell, & Maynard, 1989; Mosekilde *et al.*, 1999; Sanderson *et al.*, 1997; Talbott, Rothkopf, & Shapses, 1998). In exercising women, the rate of bone formation and the hormones that promote bone formation are suppressed within 5 days when energy availability is reduced from 45 to less than 30 kcal · kg FFM^{-1} · day^{-1}, and the rate of bone resorption is increased when energy availability is reduced enough to suppress oestradiol (Ihle & Loucks, 2004). In amenorrhoeic runners, the rate of bone formation (Zanker & Swaine, 1998) and bone mineral density (Drinkwater *et al.*, 1984; Marcus *et al.*, 1985; Myburgh, Hutchins, Fataar, Hough, & Noakes, 1990; Nichols & Sanborn, 1998; Rencken, Chesnut, & Drinkwater, 1996) are both low.

Associated nutritional issues

When energy availability is low, macronutrient and micronutrient intakes are probably also low (Beals & Manore, 1998; Manore, 1999). Reduced intakes of macronutrients, especially the essential amino acids and essential fatty acids, can decrease the body's ability to build bone, maintain muscle mass, repair

damaged tissue, and recover from injury. Micronutrients are also necessary to build bones and muscle tissue, replace red blood cells, and provide co-factors for the energy-producing metabolic pathways.

Many researchers have examined the energy intakes of female athletes (Hill & Davies, 2002; Manore, 1999, 2002; Manore, Barr, & Butterfield, 2000), including those classified with various types of menstrual dysfunction, but few have measured energy balance or energy availability while verifying menstrual or metabolic status with biochemical measurements. Table II shows that energy balance is generally more negative (about 200–1000 kcal · day^{-1} less than estimated needs), and overall energy availability is lower, in adult athletes with menstrual dysfunction than eumenorrhoeic controls. Only Laughlin and Yen (1996) did not report lower energy availability in amenorrhoeic than eumenorrhoeic athletes, but both groups were well below 30 kcal · kg FFM^{-1} · day^{-1} and luteal function was suppressed in the eumenorrhoeic athletes. Reading *et al.* (2002) reported that energy balance was similar in adolescent (15–18 years) aesthetic athletes with a mixture of short, long, and absent menstrual cylces and athletes with menstrual cycles of normal length and non-athlete controls. These results are difficult to interpret because menstrual disorders are very common in young adolescents and the energy requirements for reproductive development and function have not been quantified. Recently, Tomten and Hostmark (2006) reported that energy intake and energy availability were higher in adult athletes they classified with ''irregular'' (mean age = 35 years) and ''regular'' (mean age = 26 years) menstrual cylces than younger adult amenorrhoeic and eumenorrhoeic athletes (see Table II). These results are also difficult to interpret because the researchers did not clearly define ''irregular'' and ''regular'' menstrual cylces, and the inclusion of older athletes may have confounded the results.

An inadequate energy intake increases the probability that intakes of protein, carbohydrate, and essential fatty acids will be below those recommended (Beals & Manore, 1998; Manore, 1999). If carbohydrate intakes are low, glycogen stores may not be fully replenished during periods of high exercise training (Burke, Millet, & Tarnopolsky, 2007). Most female athletes in training need a minimum of 5–6 g carbohydrate per kilogram of body weight (g CHO · kg BW^{-1}) to maintain glycogen stores to support moderate-duration/low-intensity training (Burke *et al.*, 2001; Burke, Kiens, & Ivy, 2004). If exercise intensity and duration are high and training occurs on a daily basis, carbohydrate needs may be 7–12 g CHO · kg BW^{-1} (Burke *et al.*, 2004). Manore (2002) found that only 3 of 12 studies of eumenorrhoeic and amenorrhoeic athletes reported carbohydrate intakes $\geq$6 g CHO · kg BW^{-1} using 7-day diet records. Finally, researchers have observed that amenorrhoeic athletes consume significantly more fibre than eumenorrhoeic athletes (Deuster *et al.*, 1986; Laughlin & Yen, 1996; Warren *et al.*, 2002), which may reduce the absorption of energy and nutrients.

Although limited data are available on the protein (PRO) requirements of female athletes, it is recommended that they consume more protein (1.0–1.3 g PRO · kg BW^{-1}) (Phillips, 2004; Tarnopolsky, 2004) than the Recommended Dietary Allowance (RDA) (0.8 g PRO · kg BW^{-1}) (Institute of Medicine, 2005). Elite endurance athletes may have protein requirements as high as 1.6–1.7 g · kg BW^{-1} (IAAF Consensus Statement, 2007; Tarnopolsky, 2004); however, these requirements are still being debated (Millward, 2004). Protein recommendations assume that the athlete is healthy, weight stable, and in energy balance. Protein intake reported by active women varies widely (Table II) (Manore, 2002). In general, mean protein intakes are reported to be > 1.0 g · kg BW^{-1} · day^{-1}, except when energy intakes are < 1700 kcal · day^{-1} (7113 kJ · day^{-1}), but the weight stability of athletes is not always reported. When energy intakes are low and/or weight is being lost, protein requirements rise, since body protein stores are used as an energy source. This would be especially true in females who restrict their energy intake while maintaining high exercise training routines. Finally, the quality and timing of protein intake in relation to exercise has not been addressed in this population. Research suggests that the type of protein consumed differs between eumenorrhoeic and amenorrhoeic athletes (Brooks, Sanborn, Albrecht, & Wagner, 1984), with eumenorrhoeic athletes consuming more meat. In addition, recent research has suggested that high-quality protein consumed immediately after exercise may improve overall net protein balance (Koopman *et al.*, 2004).

The Institute of Medicine's Acceptable Macronutrient Distribution Range for fat is 20–35% of energy (Institute of Medicine, 2005). For the first time, the Institute also set specific recommendations (g · day^{-1}) for the essential fatty acids (linoleic acid and alpha-linoleic acid). The essential fatty acids are metabolized to important biological compounds that regulate numerous bodily processes, including vasoconstriction, inflammation, and blood clotting (Institute of Medicine, 2005). To date, no studies have compared the essential fatty acids intakes of amenorrhoeic and eumenorrhoeic athletes, but it would be expected that as dietary fat intake decreases, the intakes of these essential fatty acids would also decrease unless careful dietary planning was made. Dietary fat is almost always limited in the diets of active females who are restricting their energy intake

Table II. Mean energy intake (EI), total energy expenditure (TEE), energy balance (EB), exercise energy expenditure (EEE), energy availability (EA), and macronutrient intakes in adult female athletes with and without menstrual dysfunction[a].

Reference	Menstrual Status (n size)	Athletes	Body fat (%)	BMI ($kg \cdot m^{-2}$)	FFM (kg)	EI ($kcal \cdot day^{-1}$)	TEE ($kcal \cdot day^{-1}$)	EB ($kcal \cdot day^{-1}$)	EEE ($kcal \cdot day^{-1}$)	EA ($kcal \cdot day^{-1}$)	EA ($kcal \cdot kg\ FFM^{-1} \cdot day^{-1}$)	EI ($kcal \cdot kg\ BW^{-1} \cdot day^{-1}$)	Protein intake ($g \cdot kg^{-1} \cdot day^{-1}$)	CHO intake ($g \cdot kg^{-1} \cdot day^{-1}$)	Fat intake ($g \cdot kg^{-1} \cdot day^{-1}$)
Tomten and Hostmark (2006)[a]	Irregular cycles (not classified as amenorrhoeic) ($n=10$)	runners	19.8	20.7	44.8	2318	2677	−359	526	1792	40	39.2	1.49	5.7	1.04
	Eumenorrhoeic (n = 10)		18.1	20.0	41.1	2940	2629	+311	502	2438	59	51.9	1.71	7.0	1.70
Thong *et al.* (2000)	Amenorrhoeic ($n=5$)	runners & cyclists	14.6	18.9	44.8	1672	N.A.	N.A.	970	722	16	31.8	1.0	5.5	0.73
	Eumenorrhoeic ($n=8$)		15.2	19.3	44.8	2277	N.A.	N.A.	956	1321	30	43.0	1.5	6.8	1.20
Kopp-Woodroffe *et al.* (1999)[b]	Amenorrhoeic ($n=4$)	runners & cyclists	17.7	21.0	50.4	1892	2773	−881	645	1247	25	30.9	1.23	5.5	0.67
De Souza *et al.* (1998)	Eumenorrhoeic Ovulatoary ($n=24$)	runners	N.A.	N.A.	N.A.	1837	2075	−238	N.A.	1358	30	31.6	1.16	4.9	0.93
	Eumenorrhoeic LPD ($n=21$)			N.A.	N.A.	1993	2031	−38	N.A.	1522	32	35.0	1.23	5.1	1.12
	Eumenorrhoeic Anovulatory ($n=8$)			N.A.	N.A.	1326	2405	−1079	N.A.	1167	25	20.4	0.88	3.5	0.55
Laughlin and Yen (1996)	Amenorrhoeic ($n=8$)	runners & triathletes	16.0	19.4	46.4	2106	N.A.	N.A.	1074	1032	23	38.1	1.21	6.9	0.58
	Eumenorrhoeic ($n=8$)		15.9	19.6	48.9	1739	N.A.	N.A.	906	833	19	29.9	0.98	4.4	0.86
Wilmore *et al.* (1992)	Amenorrhoeic ($n=8$)	runners	10.8	18.4	45.8	1781	2433	−651	476	1305	28	34.6	N.A.	N.A.	N.A.
	Eumenorrhoeic ($n=5$)		10.3	18.7	46.6	1690	2305	−615	402	1288	28	32.5			
Myerson *et al.* (1991)	Amenorrhoeic ($n=7$)	runners	14.6	19.4	43.8	1730	1892	−148	526	1204	28	34	1.03	4.6	1.03
	Eumenorrheic ($n=10$)		15.0	19.5	42.9	1934	2128	−193	537	1397	33	38	1.34	5.3	1.31

[a]All participants had menstrual function confirmed by blood hormones; Tomten and Hostmark (2006) did not specifically screen for amenorrhoea. To convert $kcal \cdot day^{-1}$ to $kJ \cdot day^{-1}$, multiply by 4.184. All studies used 7-day food records except for Tomten and Hostmark (2006), who used 4-day records. All studies screened for eating disorders.

[b]Data averaged over four case studies at baseline.

BMI = body mass index ($kg \cdot m^{-2}$); EB ($kcal \cdot day^{-1}$) = [EI ($kcal \cdot day^{-1}$) – TEE ($kcal \cdot day^{-1}$)]; EA = EI – EEE; LPD = luteal phase deficiency; FFM = fat-free mass; N.A. = not available

or consuming high carbohydrate diets (Beals & Manore, 1998), and amenorrhoeic athletes consistently consume less fat than eumenorrhoeic athletes (Manore, 2002; Tomten & Hostmark, 2006; Warren *et al.*, 2002) even when their energy intakes are reported to be similar.

Active women and girls who restrict their energy intake or eliminate food groups from their diet can have low micronutrient intakes, especially the energy (B-complex vitamins), blood (folate, vitamin B-12, iron) (Woolf & Manore, 2006), and bone-building (calcium, magnesium, and vitamin D) nutrients (Manore, 1999, 2000, 2002). Without these nutrients, the body is unable to use the energy consumed effectively to help maintain the health of the body and provide energy for physical activity. In addition, exercise may increase the need for some micronutrients, so active females with low dietary intakes are especially at risk for poor nutritional status (Manore, 2000, 2002).

Health consequences of the Triad

The Triad may increase the risk of cardiovascular disease through endothelial dysfunction (De Souza & Williams, 2004; Friday, Drinkwater, Bruemmer, Chesnut, & Chait, 1993; Rickenlund, Eriksson, Schenck-Gustafsson, & Hirschberg, 2005a) and may also decrease immune function (Montero, Lopez-Varela, Nova, & Marcos, 2002), but the strongest evidence for health consequences of the Triad relates to stress fractures.

Stress fractures are a common injury suffered by athletes, especially long-distance runners (Bennell, Malcolm, Thomas, Wark, & Brukner, 1996b; Brunet, Cook, Brinker, & Dickinson, 1990; Hulkko & Orava, 1987; Iwamoto & Takeda, 2003; Lloyd *et al.*, 1986). Most studies report higher rates of stress fractures in females than males (Armstrong, Rue, Wilckens, & Frassica, 2004; Bennell & Crossley, 1996; Brudvig, Gudger, & Obermeyer, 1983; Hame, LaFemina, McAllister, Schaadt, & Dorey, 2004). The most common site of stress fractures in females is the tibia, accounting for 25–63% of all stress fractures (Barrow & Saha, 1988; Bennell *et al.*, 1996b; Cline, Jansen, & Melby, 1998; Iwamoto & Takeda, 2003; Shaffer, Rauh, Brodine, Trone, & Macera, 2006).

Menstrual irregularity may increase the risk of stress fractures (Barrow & Saha, 1988; Lloyd *et al.*, 1986; Myburgh *et al.*, 1990; Shaffer *et al.*, 2006; Warren, Brooks-Gunn, Hamilton, Warren, & Hamilton, 1986), but age of menarche and oral contraceptive use are not associated with stress fractures (Armstrong *et al.*, 2004; Cline *et al.*, 1998; Lappe, Stegman, & Recker, 2001; Lloyd *et al.*, 1986; Shaffer *et al.*, 2006). Other factors that do appear to increase the risk of stress fractures include age, bone mineral density, ethnicity, prior exercise training, smoking, and alcohol (>10 drinks $\cdot$ week^{-1}) (Armstrong *et al.*, 2004; Beck *et al.*, 2000; Brudvig *et al.*, 1983; Cline *et al.*, 1998; Lappe *et al.*, 2001; Myburgh *et al.*, 1990).

Researchers have found no effect of menstrual status on exercise performance (De Souza *et al.*, 1991; Glass *et al.*, 1987; Myerson *et al.*, 1991; Rickenlund *et al.*, 2005a; Rickenlund, Eriksson, Schenck-Gustafsson, & Hirschberg, 2005b; Shangold & Levine, 1982; Wilmore *et al.*, 1992), but little is known about the effect of menstrual irregularities on skeletal muscle function and strength.

Recognition and treatment

Screening

Diagnosis and identification of the Triad depends on thorough screening of athletes by their physicians during the pre-participation physical (Rumball & Lebrun, 2004). This screening should include a menstrual history, physical activity history and current activity level, diet history and current dietary behaviours in relationship to weight and sport expectations, estimation of energy availability, biochemical screen, including endocrine and iron status assessments, and family history, especially mother's age of menarche. It is also imperative that we educate the health professionals working with athletes to be aware of the signs and symptoms of the Triad. Sport governing bodies, including the International Association of Athletics Federations (IAAF), should consider rule changes that would prevent the development of the Triad due to unhealthy weight-loss practices.

Treatment

Improving an athlete's overall energy availability may be the key to reversing menstrual dysfunction and low bone density in female athletes. Anecdotal evidence and case studies indicate that when active women are required to rest due to injury or quit their sport, menstrual status and bone mineral density can be improved (Dueck, Matt, Skinner, & Manore, 1997; Fredericson & Kent, 2005). Most active women, however, are not willing to stop training or gain excessive amounts of weight to allow menstrual status to return to normal. Thus, an intervention plan that improves energy intake, reduces exercise energy expenditure (e.g. adds 1 day rest to their weekly routine) or both, without dramatic changes in weight, is most desirable. Increasing total daily energy intake in moderate increments may be the easiest approach, but we do not know which

approach is best, which athletes will respond, and how long it takes for changes in menstrual function and bone density to occur using this approach. To date, only two small pilot studies (Dueck, Matt, Manore, & Skinner, 1996; Kopp-Woodroffe *et al.*, 1999) have addressed these questions. Dueck *et al.* (1996, 1997) and Kopp-Woodroffe *et al.* (1999) found that improving energy balance ($\sim$350 kcal $\cdot$ day^{-1} increase; 1 rest day per week) and energy availability ($\sim$5 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} increase) in female athletes with amenorrhoea resulted in the resumption of menstruation. Athletes experienced a small weight gain (1.0 – 2.7 kg) that was within the error of weight measurement. For the athletes who resumed menstruation, bone mineral density increased significantly in the femoral neck (0.031 g $\cdot$ cm^{-2} increase) and the lumbar spine (0.022 g $\cdot$ cm^{-2} increase) ($P < 0.01$), while serum cortisol decreased by 33% and reproductive hormones increased. Participants also self-reported various positive attributes, including an increase in overall energy, mental concentration, and mood state.

Hormone replacement therapy (HRT) offers no benefit and oral contraceptives delay and reduce the likelihood of restoring menstrual cycles in women with functional hypothalamic amenorrhoea (Falsetti, Gambera, Barbetti, & Specchia, 2002). No pharmaceutical agent has been shown to fully restore bone mineral density in women with functional hypothalamic amenorrhoea (Cumming & Cumming, 2001; Misra & Klibanski, 2006; Warren *et al.*, 2003). Increases in bone mineral density have been more closely associated with increases in weight. Because these agents do not normalize the metabolic factors that impair reproductive and skeletal health in the Triad, modifying diet and exercise behaviour to increase energy availability is the first aim of treatment for the Triad.

Men

Cases of the Triad in male athletes rarely come to the attention of physicians because reproductive effects are not perceived by men. Its prevalence is also lower than in female athletes, because fewer male athletes practise diet and exercise regimens that severely reduce energy availability. In men, a low energy availability occurs most often in weight-class sports such as wrestling (Roemmich & Sinning, 1997a, 1997b), in endurance sports such as long-distance running (De Souza, Arce, Pescatello, Scherzer, & Luciano, 1994), and in soldiers during military training (Friedl *et al.*, 2000; Opstad, 1992a, 1992b).

In runners, most reproductive effects occur beyond a training volume threshold of $\sim$100 km $\cdot$ week^{-1} (De Souza *et al.*, 1994). When energy availability is severely reduced, alterations in metabolic hormones are similar to those seen in female athletes; testosterone is suppressed (Arce, De Souza, Pescatello, & Luciano, 1993; Booth, Mazur, & Dabbs, 1993), and sperm count, mobility, and morphology are reduced but usually not below the normal range (Arce *et al.*, 1993; De Souza *et al.*, 1994; Jensen, Wiswedel, McLoughlin, & van der Spuy, 1995). Increased feeding restores hormone levels to normal within one week while training continues (Friedl *et al.*, 2000).

Most studies of bone in exercising men have been observational in design making their results subject to selection and other sources of bias. Consequently, bone mineral density in male athletes has been found to be both higher (Kemmler *et al.*, 2006; MacKelvie, Taunton, McKay, & Khan, 2000; Wittich *et al.*, 1998) and lower (Bennell, Brukner, & Malcolm, 1996a; Hind, Truscott, & Evans, 2006) than in controls. Low bone mineral density may occur with or without low testosterone levels, suggesting the existence of nutritional, steroid-independent mechanisms of skeletal demineralization (Bennell *et al.*, 1996a).

Summary of nutritional guidelines

Consensus for:

- Monitor energy intake and exercise energy expenditure routinely to prevent low energy availability.
- Diagnose functional hypothalamic amenorrhoea by excluding other potential causes of amenorrhoea.
- Primary amenorrhoea in adolescents requires special medical care before and during nutritional intervention to avoid short stature.
- Screen athletes with menstrual dysfunction for disordered eating and/or an eating disorder.
- Athletes with menstrual dysfunction have increased risk of stress fractures and number of injury days.
- Sport governing bodies, including the IAAF, should consider rule changes to discourage unhealthy weight loss practices.

Consensus against:

- Energy availability $<$ 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} impairs reproductive and skeletal health in both males and females within 5 days.

Issues that are equivocal:

- Conceptual and pragmatic differences between energy availability and energy balance are not yet well appreciated.

- Confidence in estimates of the energy intake of female athletes needs to be improved. Methods for identifying under-reports need to be utilized, where appropriate.
- Effective and accepted nutritional intervention strategies need to be developed.

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