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Energy availability in athletes

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Abstract
This review updates and complements the review of energy balance and body composition in the Proceedings of the 2003 IOC Consensus Conference on Sports Nutrition. It argues that the concept of energy availability is more useful than the concept of energy balance for managing the diets of athletes. It then summarizes recent reports of the existence, aetiologies, and clinical consequences of low energy availability in athletes. This is followed by a review of recent research on the failure of appetite to increase *ad libitum* energy intake in compensation for exercise energy expenditure. The review closes by summarizing the implications of this research for managing the diets of athletes.

Keywords: *Energy availability, energy balance, diet, exercise, appetite*

Introduction

In the 2003 IOC Consensus Conference on Sports Nutrition, evidence was presented that many athletes, most often female athletes, were deficient in energy, and especially energy in the form of carbohydrates, resulting in impaired health and performance (Loucks, 2004). It was emphasized, however, that energy balance is not the objective of athletic training whenever athletes seek to modify their body size and composition to achieve performance objectives. They then need to carefully manage their diet and exercise regimens to avoid compromising their health.

Distinctions between energy availability and energy balance

In the field of bioenergetics, the concept of energy availability recognizes that dietary energy is expended in several fundamental physiological processes, including cellular maintenance, thermoregulation, growth, reproduction, immunity, and locomotion (Wade & Jones, 2004). Energy expended in one of these processes is not available for others. Therefore, bioenergeticists investigate the effects of a particular metabolic demand on physiological systems in terms of energy availability. They define energy availability as dietary energy intake minus the energy expended in the particular metabolic demand of interest. In experiments investigating effects of cold exposure, for example, energy availability would be defined, quantified, and controlled as dietary energy intake minus the energy cost of thermogenesis.

Exercise training increases, and in endurance sports may double or even quadruple, the amount of energy expended in locomotion. In exercise physiology, therefore, energy availability is defined as dietary energy intake minus the energy expended in *exercise* (EA = EI – EEE). As the amount of dietary energy remaining after exercise training for all other metabolic processes, energy availability is an *input to* the body's physiological systems.

In the field of dietetics, the concept of energy balance has been the usual basis of research and practice. Defined as dietary energy intake minus *total* energy expenditure (EB = EI – TEE), energy balance is the amount of dietary energy added to or lost from the body's energy stores after the body's physiological systems have done all their work for the day. Thus energy balance is an *output from* those systems. For healthy young adults, $EB = 0$ kcal $\cdot$ day^{-1} when $EA = 45$ kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} (where FFM = fat-free mass).

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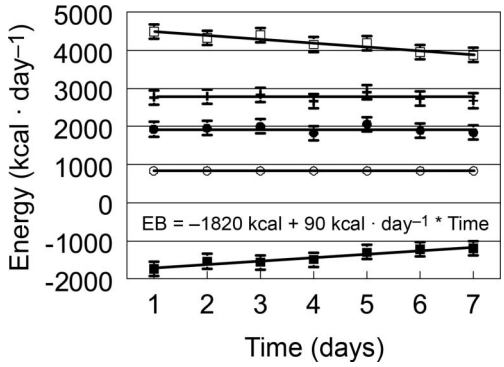

Figure 1. Negative energy balance rising at a rate of ≈90 kcal · day^{-1} as metabolic processes were suppressed while the energy intake (2770 kcal · day^{-1}), exercise energy expenditure (840 kcal · day^{-1}), and energy availability (2770 – 840 = 1930 kcal · day^{-1}) of eight lean, untrained men remained constant. EI (+) = energy intake, TEE (□) = total energy expenditure, EEE (○) = exercise energy expenditure, EA (●) = energy availability, EB (■) = energy balance. Original figure based on data in Stubbs et al. (2004).

The contrast between energy availability and energy balance is illustrated in Figure 1, which shows data collected while eight lean, untrained men lived in a room calorimeter for a week (Stubbs et al., 2004). During that week, their energy intake (2770 kcal · day^{-1}), exercise energy expenditure (840 kcal · day^{-1}), and energy availability (2770 – 840 = 1930 kcal · day^{-1} ≈ 30 kcal · kg FFM^{-1} · day^{-1}) were constant. Meanwhile, the magnitude of their negative energy balance (2770 – 4500 = –1730 kcal · day^{-1} on Day 1) decreased towards zero at a rate of ~90 kcal · day^{-1} as various physiological processes slowed down. At this rate, they would have recovered EB = 0 kcal · day^{-1} (a pathological state of energy balance achieved by suppressing physiological systems) in 3 weeks, while remaining in severely low energy availability.

Undergraduate nutrition textbooks assert that energy requirements can be determined by measuring energy expenditure, but measures of energy expenditure contain no information about whether physiological systems are functioning in a healthy manner. Because physiological processes are suppressed by severely low energy availability, measurements of total or resting energy expenditure will underestimate a chronically undernourished athlete's energy requirements.

Therefore, because energy balance is an output from, rather than an input to, physiological systems, because it does not contain reliable information about energy requirements, and because it is not even the objective of athletic training, energy balance is not a useful concept for managing an athlete's diet.

Energy deficiency in athletes: Existence, aetiologies, and consequences

At the 2003 IOC Consensus Conference, the existence of widespread energy deficiency in athletes was still questioned. Since then, the IOC Medical Commission has published two position stands (Sangenis et al., 2005, 2006) and the American College of Sports Medicine (ACSM) has published a revised position stand (Nattiv, Loucks, Manore, Sundgot-Borgen, & Warren, 2007) on the ''female athlete triad''. In addition, a coaches' handbook on Managing the Female Athlete Triad developed by the co-chairs of the athlete interest group of the Academy of Eating Disorders has been published by the US National Collegiate Athletics Association (NCAA) (Sherman & Thompson, 2005). All four publications attribute the functional hypothalamic menstrual disorders and low bone mineral density found in many female athletes to energy deficiency, but the ACSM position stand differs from the other three in that it excludes disordered eating and eating disorders as necessary components of the triad. The ACSM emphasizes that athletes who expend large amounts of energy in prolonged exercise training can become energy deficient without eating disorders, disordered eating or even dietary restriction.

The ACSM identified three distinct origins of energy deficiency in athletes. The first is obsessive eating disorders with their attendant clinical mental illnesses. The second is intentional and rational but mismanaged efforts to reduce body size and fatness to qualify for and succeed in athletic competitions. This mismanagement may or may not include disordered eating behaviours such as fasting, diet pills, laxatives, diuretics, enemas, and vomiting that are entrenched parts of the culture and lore of some sports. The third is the inadvertent failure to increase energy intake to compensate for the energy expended in exercise. The percentages of cases of the female athlete triad originating from these three sources are unknown, but ACSM emphasized that any epidemiological study requiring the presence of an eating disorder or disordered eating for diagnosing cases of the triad (e.g. Schtscherbyna, Soares, de Oliveira, & Ribeiro, 2009) will underestimate its prevalence.

Sports vary greatly in the relative importance of various factors for competitive success. As they strive to achieve sport-specific mixes of these factors, athletes engage in different diet and exercise behaviours that impact energy availability. In endurance sports, prolonged exercise training greatly reduces energy availability, unless energy intake is increased to replace the energy expended in exercise. In sports where less energy is expended in training, dietary restriction may be a prominent part of the strategy for reducing energy availability to modify body size and composition.

Female athletes may also under-eat for reasons unrelated to sport. Around the world about twice as many young women as men *at every decile of body mass index* perceive themselves to be overweight (Wardle, Haase, & Steptoe, 2006). The disproportionate numbers actively trying to lose weight are even higher, and this disproportion *increases* as body mass index declines, so that almost nine times as many lean women as lean men are actively trying to lose weight! 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). Thus issues unrelated to sport may need to be addressed to persuade female athletes to eat appropriately.

The controversy about whether female athletes can increase glycogen stores as much as male athletes is instructive in this regard. An experiment in which participants consumed diets containing high and low percentages of carbohydrates found that women could not do so (Tarnopolsky, Atkinson, Phillips, & MacDougall, 1995). Subsequently, it was noted that the total energy intake (per kilogram of body weight) of the women in that study had been so low that the amount of carbohydrate they consumed on the high percent carbohydrate diet was no greater than the amount consumed by the men on the low percent carbohydrate diet. Later research showed that women could, indeed, load glycogen like men when they ate as much as men (per kilogram of body weight) (James et al., 2001; Tarnopolsky et al., 2001).

In the 2003 IOC Consensus Conference, the disruption of reproductive function at energy availabilities < 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} was discussed in some detail and the low bone mineral density (BMD) found in amenorrhoeic athletes was represented as being mediated by oestrogen deficiency (Loucks, 2004). Since then, oestrogen-independent mechanisms by which low energy availability can reduce BMD have also been identified (Ihle & Loucks, 2004). As energy availability declines, the rate of bone protein synthesis declines along with insulin, which enhances amino acid uptake, in a linear dose–response manner. By contrast, the rate of bone mineralization declines abruptly as energy availability declines below 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}, as do concentrations of insulin-like growth factor-1 and tri-iodothyronine. These effects occurred within 5 days of the onset of energy deficiency, and without a reduction in oestrogen concentration.

In older adults, fracture risk doubles for each reduction of one standard deviation below mean peak young adult BMD. In adolescents, fracture risk can rise even as BMD increases. Because BMD normally doubles during the decade of adolescence, a child entering adolescence with a high BMD relative to others of the same age can accrue bone mineral so slowly that adulthood is entered with a relatively low BMD. Because low BMD is an aetiological factor in stress fractures, anything that impairs bone mineral accrual during adolescence is undesirable. Unfortunately, this is exactly what was found in a study of 183 interscholastic competitive female athletes, of whom 93 were endurance runners and 90 were non-runners (Barrack, Rauh, & Nichols, 2010). The BMD z-scores were similar in runners and non-runners aged 13–15 years, but were significantly lower in runners than non-runners at 16–18 years of age.

Also questioned at the 2003 IOC Consensus Conference was whether energy deficiency and its clinical consequences were a problem among elite athletes. Since then a study of 50 British national or higher standard middle- and long-distance runners found BMD to be lower in amenorrhoeic runners and higher in eumenorrhoeic runners compared with European reference data (Gibson, Mitchell, Harries, & Reeve, 2004). The duration of eumenorrhoea was positively associated with spine BMD, and the rate of bone mineralization was reduced in the amenorrhoeic runners. Alone, the Eating Attitudes Test (EAT) is not clinically diagnostic for eating disorders, but in this study scores on the EAT classified one of 24 amenorrhoeic runners and none of nine oligomenorrhoeic runners as having an eating disorder, while eight amenorrhoeic runners and three oligomenorrhoeic runners were classified as practising disordered eating behaviours. This left 63% of the cases of amenorrhoea and 67% of the cases of oligomenorrhoea unaccounted for by the EAT test (Figure 2). Similarly, a low body mass index (< 18.5 kg $\cdot$ m^{-2}) failed to account for 67% of the cases of amenorrhoea and 67% of the cases of oligomenorrhoea (Figure 3). Another study diagnosed low BMD (z-score less than –1) in the lumbar spine of 34% and osteoporosis (z-score less than –2) in the radius of 33% of 44 elite British female endurance runners (Pollock et al., 2010). Reductions in BMD over time were associated with training volume. These findings led the authors to recommend that all female endurance athletes undergo dual-energy X-ray absorptiometry screening.

Ovarian function depends critically upon the frequency with which the pituitary gland secretes luteinizing hormone (LH) into the bloodstream, and LH pulsatility in exercising women depends on energy availability, rather than energy intake or energy expenditure separately (Loucks, Verdun, & Heath, 1998). Furthermore, exercise has no suppressive effect on LH pulsatility beyond the impact of its energy cost on energy availability (Loucks et al.,

1998). Reproductive function (Loucks & Thuma, 2003) and bone formation (Ihle & Loucks, 2004) are impaired abruptly and promptly below a threshold of energy availability (< 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}), which corresponds closely to resting metabolic rate. Figure 4 shows sleeping metabolic rate (SMR) measured by indirect calorimetry in young adult men ($n = 20$) and women ($n = 17$) (Westerterp, 2003). Sleeping metabolic rate is slightly less than resting metabolic rate by the small energy expenditure associated with being awake. In Figure 4, the solid regression line relating sleeping metabolic rate to fat-free mass (SMR [MJ $\cdot$ day^{-1}] $= 2.27 + 0.091 \times$ FFM [kg]) has a significant y-intercept. The dashed line through the data and the origin has a slope of 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}. As Figure 4 shows, energy availabilities < 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} provide less energy than is required for physiological systems in young adults to function at rest.

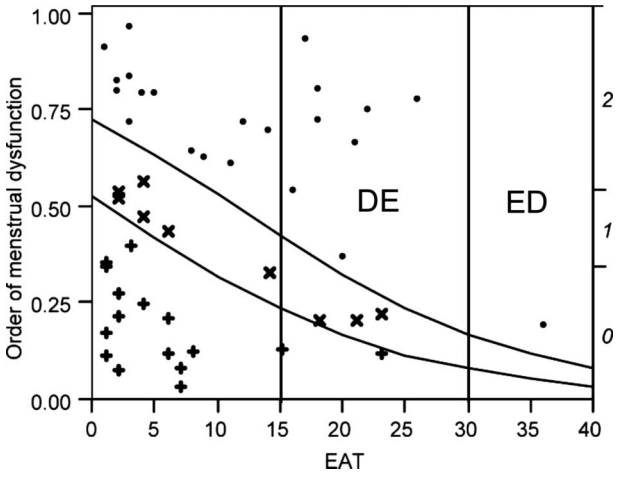

Figure 2. Logistic relationship between the order of menstrual dysfunction (right ordinate scale: 0 (+) = eumenorrhoea; 1 (×) = oligomenorrhoea; 2 (•) = amenorrhoea) and total EAT score ($P = 0.014$ for model). Left ordinate scale is a scale of probability. The right ordinate scale shows the proportions of participants in each category. DE = classified as disordered eating. ED = classified as eating disorder. Figure modified from Gibson et al. (2004).

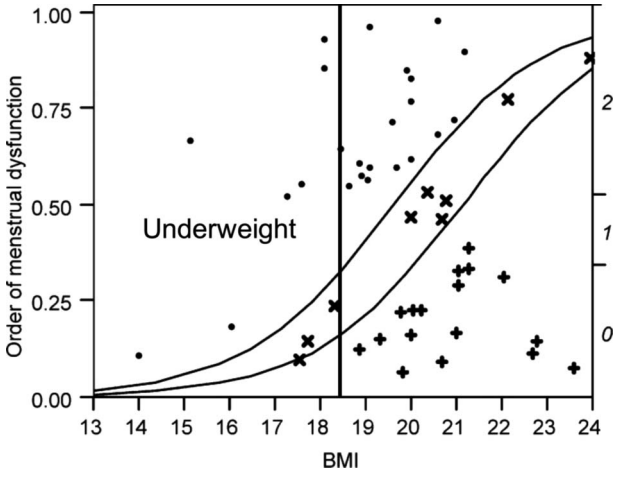

Figure 3. Logistic relationship between the order of menstrual dysfunction (right ordinate scale: 0 (+) = eumenorrhoea; 1 (×) = oligomenorrhoea; 2 (•) = amenorrhoea) and body mass index (BMI) ($P < 0.001$ for model). Left ordinate scale is a scale of probability. The right ordinate scale shows the proportions of participants in each category. BMI < 18.5 = underweight. Figure modified from Gibson et al. (2004).

Observational and experimental data indicate that low energy availability also suppresses Type 1 immunity. The immune system mounts different defences against two types of pathogens. Type 1 defences are mounted against intracellular pathogens like viruses, while Type 2 defences are mounted against extracellular pathogens like bacteria. Endurance athletes frequently suffer upper respiratory tract infections (URTI) caused by viruses. A survey of all members of Swedish teams participating in the Olympic Games of 2002 and 2004 found that those participating in disciplines emphasizing leanness made more frequent attempts to lose weight, trained longer, and reported almost twice as many illnesses, primarily URTI, during the preceding 3 months (Hagmar, Hirschberg, Berglund, & Berglund, 2008). The results of a recent experiment challenge the hypothesis that Type 1 immunity in athletes might be suppressed by exercise itself (Lancaster et al., 2005). Participants expended 2200 kcal of energy by exercising for 2½ h at 65% of maximal oxygen uptake ($\dot{V}O_{2max}$). Replacing just 23% of this energy with carbohydrate reduced the suppression of Type 1 defences by an average of 65%. Thus, ingesting sufficient energy and nutrients is vital for supporting

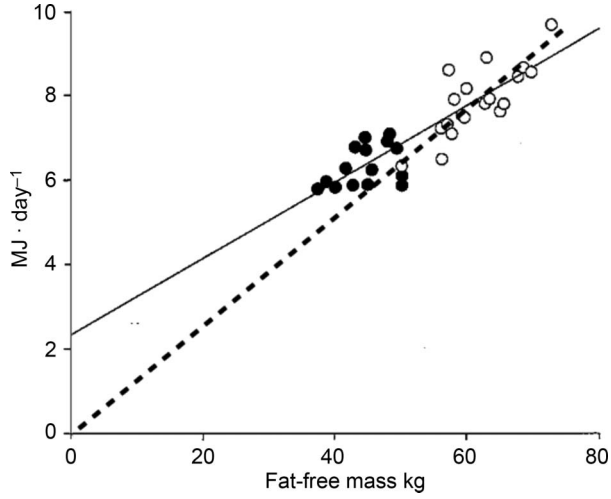

Figure 4. Sleeping metabolic rate plotted as a function of fat-free mass (FFM). • = females; (○ = males. The solid regression line has a significant non-zero intercept. The dashed line is 30 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1}. Figure modified from Westerterp (2003).

immune function, and even more so for immune-compromised individuals, such as those infected with HIV (Fenton & Silverman, 2008), whose resting energy expenditure is elevated (Mangili, Murman, Zampini, & Wanke, 2006). Therefore, HIV-infected athletes should take special care to reach their energy and nutrient needs.

Seventeen years have now passed since the first IOC Consensus Conference on Nutrition. Yet studies of energy intake and total energy expenditure continue to report that elite American figure skaters (Ziegler, Nelson, Barratt-Fornell, Fiveash, & Drewnowski, 2001), elite Kenyan runners (Fudge et al., 2006), and high-performance Canadian athletes in several sports (Lun, Erdman, & Reimer, 2009) train in substantial negative energy balance. Depending on the level of energy availability, such negative energy balance may either impair or benefit health and performance. In the current review period, two studies of elite athletes reported energy intake and exercise energy expenditure so that their average energy availability could be estimated. In the week before a race, the high percent carbohydrate diet of male Kenyan runners provided an energy availability of 34 kcal · kg FFM^{-1} · day^{-1} (Onywera, Kiplamai, Boit, & Pitsiladis, 2004). This energy availability may or may not have been appropriate depending on their athletic objectives at the time. If their performance in that particular race was less important than losing weight to improve performance in a later race, it was fine. However, no athletic objective would appear to justify professional male cyclists training for the Tour de France 6 months later at an energy availability of only 8 kcal · kg FFM^{-1} · day^{-1} (Vogt et al., 2005). Such observations indicate that the diet and exercise regimens of elite athletes range widely and are sometimes dangerously energy deficient.

Effects of prolonged exercise on hunger and energy intake

Eating disorders may be intractable, and weight and fat loss programmes may be challenging to manage effectively and safely, but these two origins of low energy availability in athletes are at least familiar to sports dietitians. The third origin, the suppression of appetite by prolonged exercise, appears to be less familiar. Neglect of appetite as an important factor in sports nutrition is indicated by the appearance of the word ''appetite'' only once (and then only in a discussion of fluid losses at high altitude) in the recently revised position stand on nutrition and athletic performance jointly adopted by the American Dietetic Association, the Dietitians of Canada, and the American College of Sports Medicine (Rodriguez, DiMarco, & Langley, 2009).

Some of the then available evidence that appetite is not a reliable indicator of energy needs in athletes was reviewed at the 2003 IOC Consensus Conference (Loucks, 2004). Shortly afterwards, the suppressive effect of prolonged exercise on *ad libitum* energy intake was clearly demonstrated by the experiment in which eight lean, untrained men expended ~840 kcal · day^{-1} by cycle ergometry as they lived in a room calorimeter for 7 days (Stubbs et al., 2004). During that week, their *ad libitum* energy intake was similar to another week in the room calorimeter when they did not exercise. This inadvertent failure to increase energy intake in compensation for exercise energy expenditure reduced their *ad libitum* energy availability by ~10 kcal · kg FFM^{-1} · day^{-1}.

The participants in that experiment actually performed the experiment four times, with and without exercise while consuming equally palatable 62% and 37% carbohydrate diets in a 2 × 2 cross-over design. Compared with weeks when the participants ate the 37% carbohydrate diet, their *ad libitum* energy intake declined by ~1000 kcal · day^{-1} on the 62% carbohydrate diet, reducing their *ad libitum* energy availability by ~16 kcal · kg FFM^{-1} · day^{-1}. Moreover, the suppressive effects of prolonged exercise and the high percent carbohydrate diet were additive. *Ad libitum* energy availability declined from ~47 kcal · kg FFM^{-1} · day^{-1} when the participants were sedentary on the 37% carbohydrate diet to ~21 kcal · kg FFM^{-1} · day^{-1} when they exercised on the 62% carbohydrate diet (Figure 5).

These findings in lean, untrained men exercising in a laboratory confirmed a previous report of a high

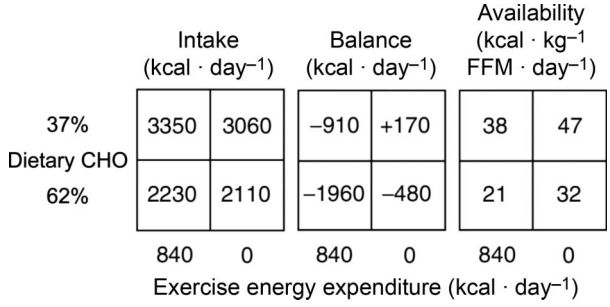

Figure 5. The *ad libitum* energy intake, energy balance, and energy availability of eight lean men living in a laboratory for 7 days during an experiment contrasting two diets (50% fat, 32% carbohydrate [CHO]; and 25% fat, 67% CHO) and two levels of exercise (840 and 0 kcal · day^{-1}) (data from Stubbs et al., 2004). Energy availability was estimated assuming 16% body fat. Appetite failed to match energy intake to activity-induced energy expenditure on either diet. Appetite also failed to drive energy intake on a low fat, high carbohydrate diet to match energy expenditure at either activity level. These effects were additive. Reproduced from Loucks (2007) with permission from Adis, a Wolters Kluwer business (© Adis Data Information BV 2007. All rights reserved).

percent carbohydrate diet suppressing *ad libitum* energy intake in 12 male and 13 female trained runners living at home (Horvath, Eagen, Fisher, Leddy, & Pendergast, 2000a; Horvath, Eagen, Ryer-Calvin, & Pendergast, 2000b). These runners ran 42 miles a week, expending $\sim$600 kcal $\cdot$ day^{-1} for 31 days. They repeated this regimen three times while consuming equally palatable diets containing 42%, 55%, and 67% carbohydrate. As the percent carbohydrate content of the diet decreased, *ad libitum* energy availability increased from 27 to 34 and 39 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} in the women and similarly from 27 to 37 and 42 kcal $\cdot$ kg FFM^{-1} $\cdot$ day^{-1} in the men. Endurance time at 80% $\dot{V}O_{2max}$ on a treadmill improved by 18% as the percent carbohydrate content of the diet was reduced from 67% to 55%. Interestingly, this reduction in percent carbohydrate content did not reduce the amount of carbohydrate consumed, because of the associated increase in *ad libitum* energy intake. The differences in *ad libitum* energy intake on the three diets had no effects on body weight or body fat (Figure 6). The mechanism by which a high percent carbohydrate diet suppresses appetite has yet to be investigated, but plausible factors include the greater bulk and fibre content (Mann et al., 2007) of carbohydrate-rich foods.

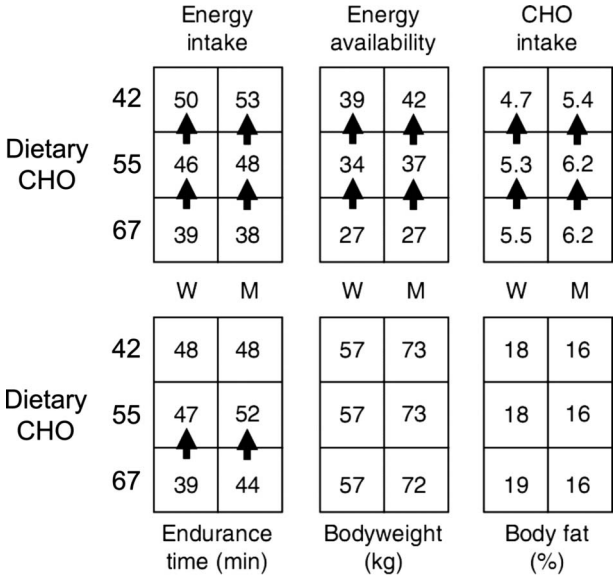

Figure 6. The *ad libitum* energy intake (kcal $\cdot$ kg FFM^{-1} $\cdot$ day,$^{-1}$), energy availability (kcal$\cdot$kg FFM^{-1} $\cdot$ day^{-1}), carbohydrate (CHO) intake (g $\cdot$ kg body weight^{-}1 $\cdot$ day^{-1}), 80% maximal oxygen uptake endurance time, body weight and percent body fat of 12 female (W) and 13 male (M) endurance-trained runners living at home for 31 days during an experiment contrasting three diets (17% fat, 67% CHO; 31% fat, 55% CHO; and 44% fat, 43% CHO) (data from Horvath et al., 2000a, 2000b). Arrows indicate statistically significant differences. Increasing dietary fat from 17% to 31% (reducing dietary CHO from 67% to 55%) increased *ad libitum* energy intake enough to preserve carbohydrate intake and increased endurance performance by 18% without affecting body weight or body fat. Reproduced from Loucks (2007) with permission from Adis, a Wolters Kluwer business (© Adis Data Information BV 2007. All rights reserved).

Recently, an even longer experiment simulating the microgravity in space confirmed the suppression of *ad libitum* energy intake by prolonged exercise (Bergouignan et al., 2010). In this experiment, eight healthy, lean, untrained women exercised in the prone position for 50 min at 40–80% $\dot{V}O_{2max}$ on alternate days during 60 days of bed rest while eight others did not exercise. *Ad libitum* energy balance was 0.7 MJ $\cdot$ day^{-1} lower in the women who exercised than in those who did not. Again, there were no differences in body weight between the two groups at any time during the 60-day study.

Evidence about the influence of gender on the suppression of *ad libitum* energy intake by prolonged exercise is conflicting. Some researchers have found the suppression in women to be greater (Staten, 1991) and others smaller (Stubbs et al., 2002a, 2002b) than that in men.

Investigators of the mechanisms that mediate the suppression of *ad libitum* energy intake by exercise recognize that appetite is comprised of two drives. Hunger, which urges us to begin eating, is stimulated by the orexigenic hormone ghrelin, whereas satiety, which leads us to stop eating, is stimulated by several anorexigenic hormones including peptide YY (PYY), glucagon-like peptide 1 (GLP-1), and pancreatic polypeptide (PP). Compared with placebo infusions, peripheral infusions of ghrelin and PYY at physiological concentrations alter food intake, with ghrelin increasing food intake at a single meal and cumulatively over 24 h by 28% (Wren et al., 2001) while PYY reduces it by 30% (Batterham et al., 2003).

A common experimental protocol for investigating these mechanisms has been to administer a standard dinner and breakfast followed by either a prolonged exercise bout or rest and then an *ad libitum* buffet lunch. One such study administered 60 min of exercise at 66% of maximum heart rate (Martins, Morgan, Bloom, & Robertson, 2007). The $\sim$300 kcal of exercise energy expenditure was followed by an increase of only $\sim$150 kcal in *ad libitum* energy intake. At lunchtime, hunger scores and concentrations of ghrelin were no higher after exercise than after rest, but PP was significantly increased after exercise.

Similar results have been found in one-day experiments on lean, untrained young men who performed 30 min of exercise at 50% and 75% of $\dot{V}O_{2max}$ (Ueda, Yoshikawa, Katsura, Usui, & Fujimoto, 2009), 90 min of exercise at 68% of $\dot{V}O_{2max}$ (King, Miyashita, Wasse, & Stensel, 2010), and a 450-kcal bout of resistance exercise (Ballard et al., 2009), and on healthy post-menopausal women who performed 2 h of exercise at 46% of $\dot{V}O_{2max}$ (Borer,

2010), as well as on male and female endurance trained runners who ran for 90 min at ~60% of $\dot{V}O_{2max}$ before a 10-km time-trial as fast as possible on a treadmill after 2 days of carbohydrate loading and a standardized 70% carbohydrate breakfast (Russel, Willis, Ravussin, & Larson-Meyer, 2009).

Other experiments have investigated individual differences in the effects of exercise training on *ad libitum* energy intake over a period of 12 weeks (King, Hopkins, Caudwell, Stubbs, & Blundell, 2008; King et al., 2009). These experiments on overweight and obese men and women confirmed the exercise-induced suppression of *ad libitum* energy intake mediated by suppressed hunger and increased satiety that had been found in shorter term experiments, but they also revealed a high degree of individual variability in weight and fat loss. When participants were retrospectively classified as compensators or non-compensators based on actual weight loss compared with the weight loss expected from exercise energy expenditure, *ad libitum* energy intake was found to have increased in the compensators and decreased in the non-compensators (King et al., 2008). Exercise induced a similar increase in satiety in both groups, which was even greater after 12 weeks of training than before, but compensators became progressively hungrier during the experiment (King et al., 2009).

Thus, findings in both trained and untrained male and female participants consistently demonstrate that a single bout of diverse forms of exercise acutely suppresses *ad libitum* energy intake and that exercise training chronically maintains the resulting energy deficiency for many weeks. This effect has been interpreted to be at least partially responsible for exercise-induced anorexia (Russel et al., 2009). Whether some female athletes become hungrier and increase *ad libitum* energy intake as their training progresses, and thereby avoid developing functional hypothalamic amenorrhoea (like overweight participants who do not lose weight on an exercise training programme), has yet to be investigated.

Implications for managing the diets of athletes

The studies of *ad libitum* energy intake cited above demonstrate that appetite is an unreliable indicator of energy requirements for athletes engaged in prolonged exercise training, just as thirst is an unreliable indicator of water requirements during a marathon race. Marathon runners are advised not to wait until they are thirsty before they begin drinking during a race. Similarly, athletes who engage in prolonged exercise training should be advised to eat by discipline, that is, to eat specific amounts of particular foods at planned times, instead of waiting until they are hungry and then eating only until they are satisfied.

The recommendation for athletes in endurance sports to consume a diet containing a high percentage of carbohydrates should also be reconsidered. The original evidentiary basis for this recommendation was the finding that high carbohydrate intake for a few days before a high-intensity endurance event increased glycogen storage and improved performance (Costill, 1988). It may be reasonable to expect endurance athletes to override their appetites to consume a high volume of a high percent carbohydrate diet for glycogen loading for a day or two as a pre-race tactic, but it may not be realistic to expect them to override the appetite-suppressive effects of both a high percent carbohydrate diet and prolonged exercise as a lifestyle. Meanwhile, research is needed to determine whether the suppression of *ad libitum* energy intake by a high percent carbohydrate diet can be ameliorated by increasing the proportion of refined carbohydrate in the diet (Mann et al., 2007).

The American Academy of Pediatrics (AAP, 2005), the IOC Medical Commission (Sangenis et al., 2005), and ACSM (Nattiv et al., 2007) have all recommended that national and international governing bodies of sports and athletic organizations put policies and procedures in place to eliminate potentially harmful weight loss practices of female athletes. Procedures and policies were not specified, because best practices may be sport-specific. These recommendations followed the establishment of such policies and procedures by the governing bodies of US men's collegiate wrestling in the late 1990s (Oppliger, Utter, Scott, Dick, & Klossner, 2006) and men's international ski jumping in 2004 (FIS, 2004). The procedures specified by NCAA Wrestling Rule 3 are expensive, intrusive for the athlete, labour-intensive for athletic trainers, and bureaucratic with local and national databases, but they seem to have been effective in reducing unhealthy weight loss behaviours and promoting competitive equity (Oppliger et al., 2006). Based on this success, a call has gone out for the International Judo Federation (IJF) to implement regulations to improve weight management behaviours among judo competitors, and for these regulations to be adopted by all National and Regional Federations (Artioli et al., 2010a).

By contrast, the International Ski Federation (FIS) employed a very different strategy for preventing excessive weight loss practices in ski jumpers (FIS, 2004). Instead of policing athlete behaviour, the FIS removed the motivation for ski jumpers to pursue excessive weight loss objectives. Ski jumpers have their height and weight measured at the top of the hill immediately before their jump, whereupon they are simply issued skis that are shorter or longer in

proportion to their body mass index. Shorter skis reduce aerodynamic lift to compensate for the advantage a lighter jumper would otherwise gain, so that a jumper's success depends on their skill rather than their body weight. Adoption of this new rule reduced the percentage of underweight ski jumpers in the next World Cup competition from 23% to 8% (Muller, Groschl, Muller, & Sudi, 2006). Replacing body mass index with mass index (MI = body mass divided by the square of sitting height) has been proposed to further improve the rule (Muller, 2009).

Periodization of training may require a periodization of energy availability to achieve training objectives. Experimental evidence indicates that athletes should follow diet and exercise regimens that provide energy availabilities of 30–45 kcal · kg FFM^{-1} · day^{-1} while training to reduce body size or fatness. However, if athletes in other sports are like judo competitors, personal counselling of athletes by sports dietitians may not be the most effective way to moderate athlete behaviour to prevent excessive energy deficiency. Among seven different types of advisors for weight management behaviour, judo competitors ranked dietitians second to last, ahead only of physicians (Artioli et al., 2010b). The most influential advisor was the coach. In that context, a more effective way to modify athlete behaviour may be for sports dietitians and nutritionists to educate coaches, using workshops and handbooks that are specifically targeted at them, about the importance of energy availability and practical techniques for managing it.

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