In conclusion, current research evidence suggests that exercise associated reproductive dysfunction in women is nutritional in origin and may be attributed to a deficit of readily available energy, primarily in the form of circulating glucose, liver glycogen, and adipose tissue triacylglycerol. A low plasma insulin concentration, consequent to a low blood glucose concentration, a low body fat content, or both of these factors, is accompanied by a disturbance of leptin secretion and a reduced plasma leptin concentration. It would seem that a reduction in plasma leptin concentration below a critical threshold value, for a significant period of time, disturbs the activity of the hypothalamic GnRH pulse generator, which instigates the endocrine events that lead to ovarian stimulation. The susceptibility to ovarian suppression and FHA is therefore greatest in lean women with chronically low energy availability, as indicated by a reduced plasma triiodothyronine concentration. To avoid reproductive dysfunction, female athletes should consume adequate energy and carbohydrate to balance energy expenditure and replace glycogen. They should avoid abrupt and rapid weight loss and maintain an "adequate" body fat content, which may be individually specific, but coincides with regular menses.

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COMMENTARY

The reproductive system is extremely sensitive to nutritional restriction. One model of nutritional restriction is the thin

exercising athlete who may have low body fat reserves and exercises intensely, causing an energy output that is beyond her nutritional intake. Recent work on the neuropeptides that regulate food intake and metabolism has revealed that a peripheral signal from the fat cell in the form of the hormone leptin transmits a signal to the hypothalamus, which enables the body to determine nutritional status. In addition, leptin is sensitive to energy deficient states, indicating that it also acts as a short term metabolic signal. Interestingly, leptin expression in the fat cells depends on glucose oxidation, indicating dependence on metabolic fuels. Thus food is necessary for leptin synthesis, and fat cells are necessary as a site of production. Inadequate leptin leads to shutdown of the reproductive system. The production of this hormone appears to be compromised in the athlete who is exercising intensely and has low body fat. In her paper Dr Zanker reviews our understanding of the process, which when left unchecked can lead to bone loss. The administration of leptin in hypothalamic amenorrhoea can reverse the amenorrhoea in some people, but the effect on bone is unknown. There is much interest in bone loss at present, which appears to have a nutritional basis with possibly a hormonal component in the form of hypo-oestrogenism. However, the administration of oestrogen has not uniformly led to a reversal of bone loss, whereas refeeding has a powerful anabolic effect with significant increases in bone mass. A number of enigmas remain. Although for most athletes there is a return of menstrual function with an increase in energy intake, a decrease in exercise, or an increase in body fat, some do not have a reversal. The restraining mechanisms in this situation are as yet unknown.

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Female athlete triad

The myth of the female athlete triad

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The female athlete triad is defined as a syndrome consisting of three necessary components: (a) disordered eating; (b) amenorrhoea; (c) osteoporosis.¹ The American College of Sports Medicine (ACSM) published a Position Stand in 1997,¹ and at that time indicated a strong need for more epidemiological, laboratory, and clinical data to support the importance of this syndrome. Currently, the prevalence of regular vigorous activity among adolescent girls remains out of reach of the Year 2010 objectives,² and the problem

of overweight among young people has achieved epidemic proportions in the United States and other industrialised countries.³ Our concern is that triad related data may be misinterpreted and used as justification for setting health and social policies that may ultimately counter the US Public Health Service efforts to promote the benefits of athletic participation and an active lifestyle among children and adolescents.⁴ Moreover, there are ample historical and medical examples of iatrogenic eating and psychosexual disorders

ascribed to otherwise healthy, hard driving, and passionate women in their pursuit of social independence, political power, or athletic excellence.^{5–8} In fact, until 1972, women were banned from very challenging athletic events such as the marathon, because officials of the Amateur Athletic Union (AAU) believed that such competition would be harmful to female reproductive function.⁸ Therefore we maintain that, as girls and young women are currently striving to attain the same level of accessibility and achievement in organised sports as their male counterparts, the creation of yet another form of female specific pathology undermines this hard earned success and may have other serious implications for their health and well-being. Our purpose in writing this article is to describe the female athlete triad with regard to its epidemiology and physiology and to offer our comments and opinions which challenge many of

the causal assumptions proposed in the current literature on this topic.

“non-athletes have been observed to be at greater risk of the triad than elite athletes”

EPIDEMIOLOGY OF THE FEMALE ATHLETE TRIAD

It is quite difficult to describe the epidemiology of this syndrome because of a number of methodological problems. Thus far, aetiological study of the triad has relied on cross sectional data that describe the prevalence of each of its individual components and then compares these proportions between athletes and a non-athletic control group. The only published study of the triad in toto describes the simultaneous occurrence of all three components in elite Norwegian athletes as both rare (4.3%) and no different from that of the non-athletic control group (3.4%).⁹ Indeed, several studies have also indicated that, although individual components of the triad can occur more often in athletes (especially those participating in “leanness sports”),^{10–11} very few athletes possess all three components simultaneously.^{9–10–12–13} More importantly, non-athletes have been observed to be at greater risk of the triad than elite athletes.¹³ Other studies suggest that individual components of the triad are found in similar proportions among athletes and non-athletes.^{9–12–13} Interpreting prevalence data on the female athlete triad is made more difficult by the continued reliance on self reported data and by the following differences in methodology among the studies: (a) differences in the classification or definition of the three components; (b) differences in the study groups with regard to age, level of competition, or level of physical activity; (c) the mixing of various terms that have very different behavioural and clinical significance—for example, disordered eating versus eating disorder. Even if studies were to rely entirely on clinically obtained biomarkers of the triad components, the substantial amount of variability both within and among adolescent girls and young women with regard to body weight, oestrogen concentrations, and bone mineral density¹⁴ makes it quite difficult to establish clinical cut-off points that have any real significance to altered function.

“Unfortunately, this new definition of the triad may have extended the classification so broadly that almost

any woman (athlete or not) could be considered ‘at risk’.”

More recent descriptions of the triad have suggested that its individual components may actually develop on separate continua.^{9–15} For example, the definition of disordered eating has been expanded to include “abnormal eating behaviours”, amenorrhoea has been expanded to include menstrual cycle alterations such as luteal and follicular phase dysfunctions, oligomenorrhoea, and anovulation, and osteoporosis includes a continuum of osteopenia, low bone mineral density, and other measures of bone metabolism. Therefore a single athlete need not have progressed to a clinically diagnosed eating disorder, amenorrhoea, and/or osteoporosis in order to be classified as having met the criteria for the female athlete triad.^{9–11} Unfortunately, this new definition of the triad may have extended the classification so broadly that almost any woman (athlete or not) could be considered “at risk.” In fact, a young woman needs to meet the definition of only one component and at an early stage on its continuum to be considered at risk of a myriad of purportedly life threatening medical sequelae.^{9–11}

Even the more salient triad related health risks are difficult to determine because, as stated previously, most studies of the triad rely on cross sectional data. Data from cross sectional studies must be viewed with caution, as they often do not account for some pre-existing and confounding factors that, in fact, influence the proposed physiological relation of interest. Indeed, heredity plays a strong influential role in athleticism, oestrogen/androgen status, body composition, and bone mineral density,¹⁴ and the temporal sequencing for the development of these factors cannot be determined without studying them longitudinally. For instance, a low oestrogen/high androgen status can determine leanness and consequent success in sport, just as participation in elite competition can alter body composition in such a way that alters concentrations of reproductive hormone.¹⁶ A gene-environment—that is, sport—interaction may also influence the aetiology of the triad, such that very lean and weight conscious athletes who participate in an impact sport such as gymnastics (also referred to as a leanness sport) often have greater bone mineral density than heavier athletes participating in, say, swimming.^{17–18} Furthermore, we do not know what the long term health consequences of the triad are, or even if their early prognostic factors (disordered eating or

alterations in menstrual function) are, in fact, irreversible, because there continue to be no prospective data available that link conditions such as amenorrhoea to clinical disorders such as infertility or osteoporosis in later years. Taken together, the data on the female athlete triad thus far provide little evidence of either a triad or its specificity to athletes.^{9–12–13}

DISORDERED EATING AS THE CORNERSTONE OF THE TRIAD

A distorted body image, disordered—for example, restrictive—eating and underweight, in essence, have been implicated as the primary factors in the aetiology of the female athlete triad.¹ These conditions may indeed set the stage for subsequent conditions associated with hypo-oestrogenaemia, such as menstrual cycle alterations and consequent bone loss. That low energy availability and negative energy balance are associated with reproductive and metabolic changes is consistent with over a century of data from evolutionary biology and anthropology. Input from the environment regarding the availability of energy is sensed and integrated with peripheral information (such as blood ghrelin or insulin concentrations). In response to this information, neuronal pathways are activated that influence energy intake, as well as energy expenditure, in order to support reproductive function.¹⁹ Indeed, over the short term, this survival advantage during periods of famine can be considered an example of successful adaptation that exists in all species at least back to *Caenorhabditis elegans*.²⁰ Disordered eating and underweight, however, are neither necessary nor sufficient conditions for alterations in menstrual function. Amenorrhoea often occurs in the general adolescent female population, even in the absence of substantial undernutrition and underweight,^{21–22} and there are many underweight and lean athletes who menstruate regularly.¹⁴

Disordered eating and clinically diagnosed eating disorders lie at very different points along the eating continuum, but these two terms are often used interchangeably in describing the triad. Behaviours associated with disordered eating are quite prominent among our weight obsessed society and can be observed in male as well as female athletes.¹⁰ In contrast, eating disorders—for example, anorexia nervosa, bulimia nervosa, and binge eating disorder—are a class of serious psychiatric pathology (DSM-IV), and although this form of psychopathology can be observed in athletes,¹⁰ it is probably more prominent among those not engaged in competitive sport, simply

by the nature of the serious limitations these illnesses impose on physiological function. There is no question that, over time, real eating disorders have serious and life threatening physiological consequences, including cardiovascular, metabolic, and reproductive dysfunction and profound bone loss. However, to imply that disordered eating and weight control in sport are the gateway behaviours to a clinically diagnosed eating disorder^{9 10 13} is to ignore the constellation of psychopathological factors that lie in between.

We previously described a number of predisposing, enabling, and reinforcing factors that contribute to the development and exacerbation of distorted body image and eating disorders as they relate to the triad, and we propose that these factors are more characteristic of our current social, rather than athletic, environment.²³ Torsteveit and Sundgot-Borgen⁹ recently observed a greater prevalence of reported "pathogenic weight control methods" among non-athlete female control subjects than elite female athletes, and the more recent clinical data from the same group suggest a similar prevalence of clinically diagnosed eating disorders between elite athletes and controls,¹³ supporting our premise that disordered eating and eating disorders are not specific to sport. Indeed, it is quite possible that the vast majority of lean and competitive women have no psychiatric morbidity. In fact, the literature suggests that physically active people have better mental health than their sedentary peers.²⁴ Thus we challenge any suggestion that athletes are at special risk of this form of psychiatric illness as several reports on the triad have implied[1, 9, 10, 13 25]

LABORATORY STUDIES OF ENERGY AVAILABILITY AND RESERVE

Over the last decade a number of studies have been published indicating that a negative balance between energy intake and energy expenditure—that is, "energy availability"—will lead to changes in reproductive function and possible loss in bone mass in young women.^{26–29} Interestingly, however, the issues of disordered eating and/or eating disorders are not mentioned in these studies. In fact, athletes may find themselves with transient energy imbalances because of the increased demands of their sport, not because of any distortions of body image or disordered eating. It is no secret that both male and female athletes have difficulty maintaining adequate food intake during periods of intense training and competition.^{30–32} Moreover, athletes of both sexes may deliberately lose weight

before competition in order to gain a competitive advantage. Thus, over the course of a competitive season, a decrease in energy availability may occur as a byproduct of intense training, rather than from some special form of psychopathology.

Again, whether low energy availability in athletes has long term negative consequences has not yet been determined. Well controlled longitudinal studies in non-human primates do suggest a causal relation between energy availability and amenorrhoea.^{33 34} However, these studies also indicate that the progression to amenorrhoea is highly variable among these primates (7–24 months in duration), that amenorrhoea occurs abruptly, and that recovery of normal menstrual function is rapid upon refeeding.

A series of well designed laboratory studies in sedentary women^{28 29 35} have also shown that low energy availability is an important factor contributing to changes in reproduction function. Not surprisingly, these studies show that low energy availability induces changes in reproductive function and metabolism in ways similar to that of starvation.^{28 29 35} These studies have shown that clinically induced and transient decreases in energy availability of both 57% (from 45 to 20 kcal/kg lean body mass) and 78% (from 45 to 10 kcal/kg lean body mass) are capable of blunting luteinising hormone pulse frequency and increasing luteinising hormone pulse amplitude in the follicular phase of the menstrual cycle.²⁹ These same changes in energy availability were also found to induce the uncoupling of bone reabsorption and formation;³⁵ however, no negative effects on reproduction function or bone metabolism were observed at a 33% decrease in energy availability (from 45 to 30 kcal/kg lean body mass). Earlier data suggest a threshold level of energy availability for such functional changes to occur at about 20–25 kcal/kg lean body mass (~49% fall).²⁸ This premise is based on previous data showing induction of low triiodothyronine syndrome at an energy availability of 19–25 kcal/kg lean body mass.²⁸

To translate these findings into more practical considerations, a 60 kg woman would have to increase recreational running time by over 54 minutes a day and elite runners by over 43 minutes a day before negative consequences to luteinising hormone pulsatility are observed.^{36 37} Although more data are required to define the precise threshold of energy availability for maintaining reproductive function, one must consider that such function appears quite robust and can be maintained even in the light of a 33% fall in energy availability.

The authors interpret the data resulting from these studies on sedentary women as relevant for athletes because some athletes purportedly function at an energy availability as low as 18 kcal/kg lean body mass.²⁶ However, a report on the Defense Women's Health Research Program (DWHRP) interpreted these same data as showing that "...moderate levels of exercise by young women did not, in fact, produce the changes that would lead to reproductive function as long as energy requirements were met".³⁸ Moreover, these same data do not pertain to athletes for the following reasons: (a) the clinically induced energy deficits were acute, rather than gradual, as they are more likely to occur with training; (b) the women in these studies were not athletes, and therefore would not have any training related substrate storage and conversion adaptations that tend to occur in muscle and the liver; (c) the method of lowering energy availability (at least in the human studies) was to restrict energy intake, rather than to increase energy expenditure as would occur with training.

Interestingly, energy availability does not always seem to be a mechanism involved in changes in reproductive function among athletes. For example, in a cross sectional study of recreational athletes, energy availability was similar between women with and without follicular phase dysfunction.²⁶ In fact, energy intake was greater in cyclic, ovulating women with menstrual dysfunction than in their sedentary counterparts (suggesting an appropriate energy balance and energy availability in these active women), while only anovulatory women in this study had significantly attenuated energy availability.²⁶

Even if all of the data on energy availability in sedentary women were to apply to athletes, there continue to be no data available that link disordered eating to the induction of low energy availability in athletes. Neither are there prospective data on the long term negative consequences of lower energy availability (e.g., infertility, osteoporosis), although its relation to longevity in several non-human species has been documented repeatedly.³⁹ We do know, however, that both reproductive³³ and cardiovascular function⁴⁰ are restored quickly when energy availability is restored to baseline levels. Thus, it is not clear how even these well designed and well controlled laboratory studies provide supporting evidence for the existence of the female athlete triad.

Finally, the female athlete triad stands in contrast with other ACSM position stands, as well as World Health

Organisation and US Public Health Services Guidelines, which state that participation in sport or in physical activity promotes the health and safety of girls and young women. Indeed, we do not want to restrict the many physical and psychosocial benefits derived from participation in sport and other activities, which for most women may ultimately translate into a lifetime of being physically active. "Clearly if participation is to be considered dangerous, the cost to both society and to sport will be very high".¹⁰

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