Role of Energy Balance in Athletic Menstrual Dysfunction

Christine A. Dueck, Melinda M. Manore, and Kathleen S. Matt

The cessation of menstrual function in the female athlete may reflect her inability to adapt to the environmental and lifestyle stressors associated with training and competition. As society’s emphasis on thinness, dieting, and exercise continues to increase, so will the incidence of menstrual dysfunction in active females. Unfortunately, some individuals view athletic menstrual dysfunction as a benign consequence of strenuous exercise. Conversely, it is most likely a strong indicator of overtraining and a marker for future decrements in performance, and it can have long-term health consequences. Thus, it is imperative that the active female be appropriately educated regarding the adverse consequences of menstrual dysfunction and the interventions available. This paper focuses on the most current information regarding athletic menstrual dysfunction and its multifactorial etiology, especially the role of energy drain. In addition, common misconceptions, adverse health and performance effects, and available treatment options are discussed.

Key Words: amenorrhea, diet, exercise, stress, female athlete

A growing body of evidence suggests that the prevalence of excessive training regimens (34) and the emphasis on thinness or leanness is increasing (74, 83). Concomitantly, there has been a dramatic increase in the number of female athletes experiencing menstrual dysfunction. The most common dysfunction reported is exercise-associated amenorrhea, in which there is a complete cessation of menstrual function and absence of menses. The prevalence of amenorrhea in the athletic population is estimated to range from 1 to 43% depending on the sport (37); however, the actual occurrence is probably greater due to underreporting. Two other common types of menstrual changes observed in athletes are luteal phase deficiency and anovulation (61). The prevalence of these disorders is even more difficult to determine. They are characterized by subtle endocrine abnormalities that can only be detected by repeated hormonal measurements or with careful monitoring of basal body temperatures (10). Thus, unless an athlete experiences the complete cessation

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of menstrual function, she is unlikely to be aware of the changes in her hormonal profile.

The female athlete, driven to excel in her chosen sport and pressured to fit a specific athletic image to attain her goals, often acquires a “win-at-any-cost” approach to training. This mentality is also held by many coaches and reinforced by parents who place incredible pressures on athletes to win. Unfortunately, this monumental pressure to attain peak performance has led to many misconceptions regarding the clinical significance of menstrual dysfunction. Frequently, athletic amenorrhea is inappropriately viewed as an indication of adequate training level, rather than an indication of overtraining that may lead to decrements in performance and poor health. This lack of recognition regarding the multiple adverse health effects associated with menstrual dysfunction perpetuates the problem.

This paper focuses on the most current information regarding athletic menstrual dysfunction and its etiology, especially the role of energy drain. In addition, common misconceptions, adverse health and performance effects, and available treatment options are discussed. A number of excellent reviews are available covering some of these topics in more detail. The reader is referred to these papers for a more in-depth review of the medical perspective (1, 53, 58, 61, 70, 80), the specific hormonal changes that occur with exercise-induced menstrual dysfunction and their underlying mechanisms (14, 30, 37, 44–46, 48, 51, 79), and the effect menstrual dysfunction has on bone health (15, 21, 58, 71). One aspect of sport-related menstrual dysfunction, the female athlete triad, has also been covered in detail in other review articles (15, 58, 76, 85).

Overview

Female athletes are faced not only with the general sociocultural demands placed on women to attain an ideal body shape but also the pressure of conforming to the specific aesthetic requirements of their sport. To achieve this goal, female athletes may adopt rigorous daily exercise programs combined with severe dietary restriction. As research data on competitive female athletes increase, it is apparent that dietary restriction and/or strenuous exercise can have a significant negative impact on numerous systems in the body (23–25, 46, 52). Thus, athletic menstrual dysfunction can no longer be viewed as a disorder of the reproductive system that only requires treatment when the individual is seeking a remedy to infertility.

We now know that the menstrual dysfunction associated with sport is multifaceted, and that for any one individual a number of factors may be involved. It has been suggested that there is a functional link between environmentally and endogenously generated stress and the cessation of menstrual function (42). Lifestyle stressors that have been identified as predisposing factors for the onset of menstrual dysfunction include a history of weight fluctuations, inadequate diet, a rigorous training regimen, and the social pressures associated with competition. Regarding endogenous stressors, the most commonly cited factor associated with amenorrhea is an inadequate level of body fat (29). Although this hypothesis of a “critical level of body fat” required for the maintenance of menstruation has been largely refuted, it cannot be entirely dismissed. Perhaps what is more important regarding the etiology of athletic menstrual dysfunction is the overall “energy reservoir.” The total energy reservoir takes into account the amount of energy stored in glycogen and body fat, and the energy consumed daily through food. This
available energy must be balanced against daily energy expenditure. Athletes with the lowest energy reserves may have less tolerance for chronic negative energy balance and may be at greatest risk for developing menstrual dysfunction.

It has been suggested that the disruption of menstrual function is an energy-conserving adaptation to energy deficiency (57, 78). Thus, luteal phase deficiency, anovulation, and amenorrhea may represent an adaptation by the body to metabolic demands that are not met due to inadequate energy intake. Evidence further suggests that energy expenditure during high-performance training produces an energy drain resembling undernutrition (78). The energy drain associated with the high energy demands of training, inadequate energy intake, and the stress of competition results in the activation of the “stress axis”—the hypothalamic–pituitary–adrenal (HPA) axis. During times of mental, physiological, or metabolic stress, the HPA axis becomes up-regulated. This results in increased secretion of corticotrophin-releasing hormone by the hypothalamus, an increase in the release of adrenocorticotropic hormone by the pituitary, and an increase in cortisol production by the adrenal cortex. Concomitant with this is a down-regulation of the “reproductive axis”—the hypothalamic–pituitary–ovarian (HPO) axis. This results in a decrease in the release of gonadotropin-releasing hormone (GnRH), a decrease in the production of luteinizing hormone (LH) and follicle-stimulating hormone (FSH), and a decrease in the production of estrogen and progesterone (50).

**Misconceptions**

Despite the extensive research available on menstrual dysfunction, a host of misconceptions continue to plague athletes of all skill levels as well as individuals who train and treat female athletes. One of the most common misconceptions among athletes and coaches is that menstruation stops when body fat levels become optimal for a given sport. Unfortunately, this misunderstanding has resulted in extreme efforts by many athletes to achieve and maintain an excessively thin body shape. These efforts, most commonly achieved by severe energy restriction, may lead to further disordered eating behaviors that, in turn, may ultimately lead to anorexia nervosa (6, 73, 74). Many sport organizations are taking steps to minimize this extreme focus on body shape and weight. For example, some sports have eliminated daily weight-ins and do not allow judges to comment on body weight and shape when scoring the athlete. Hopefully, these efforts will help to curtail the self-defeating behaviors vainly displayed by many athletes who attempt to attain an unhealthy body weight.

The misconceptions regarding athletic menstrual dysfunction are further perpetuated by a lack of adequate coach and athlete education. Many coaches are simply unaware of the adverse health and performance ramifications associated with menstrual dysfunction. Concomitantly, many female athletes welcome the cessation of menstruation without recognizing the detrimental effects it may have on both performance and overall health. Furthermore, athletic amenorrhea has become a status symbol among some female athletes who falsely believe that it signifies appropriate training volume and intensity. In actuality, athletes with an exercise-induced altered menstrual cycle are most likely overtrained (37), and, thus, are performing at less than optimal levels. Coaches need to recognize that a change in menstrual status may warrant a modification in an athlete’s training program so that her exercise energy expenditure is slightly reduced. Such modifications will not
only improve performance but will also protect the athlete from the multiple adverse effects of menstrual dysfunction.

In the medical community at large, some are still unaware of the far-ranging negative consequences associated with athletic menstrual dysfunction. Consequently, little attention may be given to the recognition, treatment, and prevention of this disorder. Frequently, a female athlete with a stress fracture is not questioned about her menstrual function, even though her injury may result in part from her compromised hormonal status. Athletic amenorrhea represents more than just the cessation of menstrual function; the associated hormonal aberrations have a negative impact on multiple systems in the body. Thus, it is imperative that this disorder be treated not only in those individuals desiring fertility but in any active woman displaying an altered menstrual cycle. Athletes may display a variety of symptoms such as soft-tissue injuries, low energy, fatigue, hypothyroidism, and anemia that may not be clearly associated with the endocrine aberrations of menstrual dysfunction. Furthermore, these symptoms may represent an athlete’s overtrained state and her energy and hormonal imbalance. Thus, as with other endocrine diseases, the cause rather than the symptoms should be treated.

Finally, many physicians still assume that the only way to treat the hormonal aberrations associated with menstrual dysfunction is by prescribing hormonal therapy or oral contraceptives (33, 34). Unfortunately, athletes often associate the use of exogenous hormones with performance hindering side effects such as fatigue, nausea, headaches, and mood swings. As a result of these fears, many amenorrheic athletes avoid seeking appropriate medical attention, thus placing themselves at an increased risk for both short- and long-term health repercussions. An alternative approach for treating athletes who are unwilling to adhere to hormonal therapy is through modifications in lifestyle factors (26).

**Etiology of Menstrual Dysfunction in Sport**

As mentioned earlier, a variety of factors have been identified as contributing to the development of athletic amenorrhea and the menstrual dysfunction associated with exercise. Many of these factors have been associated or could be associated with changes in energy balance and/or the body’s energy reserves. The following section outlines how negative energy balance may encompass many of the factors contributing to menstrual dysfunction in sport and indeed may be one initiator of this disorder.

**Energy Drain Hypothesis**

There is a growing body of evidence suggesting that athletic amenorrhea, and other reproductive hormone abnormalities seen in active women, may be due in part to periods of energy deficiency. Figure 1 outlines a model of the role negative energy balance may play in the initiation of menstrual dysfunction in some active women. Negative energy balance (energy drain) in the athlete is most likely due to three factors: high energy expenditure, low energy intake, and high psychological and physical stress. High energy expenditure is due to increases in exercise energy expenditures brought on by strenuous training routines and elevated stress levels. High stress levels in the athlete are associated with the demands of competition and training, the social stresses of family and friends, and the stresses of work and
Figure 1 — Model to illustrate the influence of energy drain on the development of menstrual dysfunction in active women, and the potential health and performance outcomes due to low reproductive hormones and high cortisol levels. hGH = human growth hormone, GnRH = gonadotropin-releasing hormone, LH = luteinizing hormone, FSH = follicle-stimulating hormone, RMR = resting metabolic rate, TEF = thermic effect of food, EEE = exercise energy expenditure, SPA = spontaneous physical activity.
Concurrently, energy intake may not keep up with demand due to fatigue, limited opportunities to eat, restrictive budgets, concerns about body weight, and the suppression of hunger that frequently follows strenuous exercise. Researchers have examined the effect of negative energy balance on the menstrual cycle and reproductive hormones by investigating the role of diet (energy restriction) alone, exercise alone, diet plus exercise, or diet plus stress. The following section will review the research done in each of these four areas.

Human studies examining the effect of energy deprivation on reproductive abnormalities report that energy restriction can alter the hormonal profiles and the menstrual cycles of healthy women. The magnitude of energy restriction and the body’s level of energy reserves before dieting begins may both influence the degree of menstrual dysfunction that occurs with energy restriction. At present, we do not know if these factors influence menstrual function independently, jointly, or synergistically. For example, Kurzer and Calloway (38) fed 6 healthy women (104–130% of ideal body weight) two diets differing only in energy intake. All subjects reported normal menstrual cycles during the 9 months before participating in the study. The first diet provided subjects with a typical energy intake (40 kcal/kg body weight), while the second diet provided 41% of the first diet (17 kcal/kg body weight). Both diets lasted the length of the menstrual cycle plus 1 week, and all subjects received their food daily from a metabolic unit. Weight loss on the low-energy diet ranged from 3.2 to 6.7 kg during the study. The two leanest women lost the most weight and became anovulatory and amenorrheic during this period. Thus, in this study, the leanest subjects displayed the greatest menstrual dysfunction when placed on an energy-restrictive diet. Their bodies appeared to respond to low energy reserves in the presence of energy restriction by suppressing the ability for reproduction. This is supported by data showing that amenorrhea is increased in women during times of starvation. For example, amenorrhea is one of the criteria used to classify an individual as having anorexia nervosa (2), a condition where starvation is self-inflicted. Thus, it appears that energy restriction alone can trigger abnormal menstrual function in some otherwise healthy women. No criteria are currently available for identifying those individuals who are most susceptible to the impact of energy restriction on menstrual function. However, these criteria may be related to levels of energy reserves when dieting starts and to the degree of energy restriction imposed.

We also know that the impact of energy restriction on menstrual function may depend on an individual’s initial hormonal status before dieting begins. In other words, if an individual already has luteal phase deficiency or is anovulatory, dieting may cause amenorrhea more quickly than it would in subjects who begin the diet with normal menstrual function. Unfortunately, most individuals do not know if they have these more subtle menstrual abnormalities before beginning a diet. This point is illustrated in a study by Pirke et al. (62), who examined the effect of dieting on menstrual cycles. They fed 9 young healthy women (95–112% of ideal body weight) a very low calorie diet (800–1,000 kcal/day) for 6 weeks. All subjects self-reported normal menstrual cycles at baseline; however, it was determined through hormonal analysis that 3 of the 9 women were anovulatory before starting the diet. Thus, the response of these 3 anovulatory individuals to the diet was a further suppression of plasma estradiol concentrations. In addition, 2 of these 3 women became amenorrheic during the diet period. Of the remaining 6 subjects, 3 became anovulatory during the dieting period and displayed luteal phase insuffi-
Researchers have also examined the effect of diet (energy restriction) and exercise or exercise alone on reproductive hormones and menstrual cycles in active women. Increasing energy expenditure while decreasing energy intake places the body in a state of negative energy balance and requires that the body draw on energy reserves to cover the cost of exercise. We now know that this combination has a more negative effect on menstrual status than just exercise alone. This may be due in part to the greater negative energy balance and resulting weight loss that occurs when dieting is combined with high exercise energy expenditure. This point is demonstrated by the following recent research.

Williams et al. (82) examined the effect of exercise with and without energy restriction on LH secretion. They studied 4 moderately trained eumenorrheic women over three consecutive menstrual cycles during the follicular phase. To examine the effect of exercise and diet on pulsatile changes in LH levels, serial blood samples were taken over a 5-hr period before and after 90 min of running at 74% \( \text{VO}_{2\text{max}} \). Before each exercise test, both diet and exercise were controlled for 7 days. During the control period, subjects were eucaloric on Days 1–7 and performed no exercise on Days 5–7. During the high exercise period, subjects were eucaloric and completed 90 min of running on Days 5–7. During the diet plus high exercise period, subjects consumed 60% of the calories necessary to maintain weight on Days 1–7 of the control period and completed 90 min of running on Days 5–7. Subjects experienced a significant decrease in LH pulse frequency during the diet plus high exercise period compared to the other two periods. No changes were observed in mean LH levels or peak LH concentrations. These results suggest that abrupt increases in training volume only disrupt LH secretion when energy restriction is present and subjects are in negative energy balance. Furthermore, only the diet plus high exercise period produced significant weight loss, while weight was maintained in the high exercise only period. It has been suggested by Loucks et al. (50) that reduced LH pulse frequency is associated with athletic training; however, Williams et al. (82) found that only the high exercise plus diet restriction period reduced LH pulse frequency. The results of Williams et al. (82) support the earlier work of Bullen et al. (13), who reported a higher incidence of menstrual abnormalities following abrupt increases in physical activity that resulted in weight loss (i.e., negative energy balance) as compared to exercise accompanied by weight maintenance. They found that in 85% of their subjects (\( n = 28 \)), exercise disrupted menstrual cyclicity; however, the weight-loss group had a significantly higher incidence of delayed menses and loss of the preovulatory LH surge. These changes occurred even though the weight-loss group did not have body fat levels below “critical” levels.

The effect of diet (energy intake) and psychological stress on menstrual function in athletes has also been examined. Increased stress, whether psychological or physiological, can increase total daily energy expenditure by increasing metabolic rate. Thus, the combination of high stress and energy restriction can result in a greater energy deficit than just dieting alone. This in turn can influence the secretion of reproductive hormones, as demonstrated by Schweiger et al. (69), who found a positive correlation (\( P < .01 \)) between energy intake and progesterone levels (area under the curve) during the luteal phase of the menstrual cycle. They also found that ratings of subjective stress (partners, family, and friends) correlated
negatively with luteal phase progesterone levels \( (P < .01) \). In addition, subjective stress correlated negatively with estrogen production during the luteal phase (area under the curve). Their data support the hypothesis that nutrition (energy intake) and stress play a critical role in the etiology of menstrual disturbances in athletes.

Other evidence supporting the role of energy drain in the initiation of menstrual dysfunction comes from the measurement of thyroid hormones and metabolic rate in amenorrheic athletes. In general, negative energy balance decreases thyroid hormones and subsequently decreases resting metabolic rate (RMR). If amenorrheic athletes are in a state of energy drain, one would expect them to have reduced thyroid hormones and RMR. Two recent studies by Loucks et al. (49) and Wilmore et al. (84) reported lower thyroid hormones in amenorrheic versus eumenorrheic runners. Loucks et al. (49) found lower free \( T_4 \), free \( T_3 \), and reverse \( T_3 \), while Wilmore et al. (84) found lower \( T_3 \) uptake. This research supports the earlier work of Marcus et al. (52) and Myerson et al. (57). Of these studies, all but Wilmore et al. (84) reported lower energy intakes in amenorrheic compared to eumenorrheic athletes (130–440 kcal/day lower). Thus, lower thyroid hormones in amenorrheic athletes may be another indication that these women are in a state of energy drain. Both Myerson et al. (57) and Wilmore et al. (84) also compared the RMR of their eumenorrheic and amenorrheic runners to determine if amenorrheic athletes had greater energy conservation at rest. Myerson et al. (57) reported significantly lower RMR values in amenorrheic athletes, while Wilmore et al. (84) found no differences between the groups. Three factors may contribute to the differences in these studies: the presence of eating disorders in the amenorrheic athletes, undetected menstrual dysfunction in the eumenorrheic athletes, and not controlling for when RMR was measured in the eumenorrheic athletes. Myerson et al. (57) reported that at least three of their eumenorrheic athletes may have been anovulatory. No measure of ovulation was done by Wilmore et al. (84). Thus, some of these athletes could have been experiencing menstrual dysfunction despite the presence of regular menstrual bleeding. In addition, the amenorrheic runners in the Myerson et al. (57) study reported significantly higher aberrant eating patterns than eumenorrheic runners. Wilmore et al. (84) did not screen for eating disorders. Finally, only Myerson et al. (57) controlled for the time point during the menstrual cycle at which RMR was measured in their eumenorrheic athletes. We now know that RMR changes over the menstrual cycle and is lowest in the follicular phase and highest in the luteal phase (9, 72). Certainly more research needs to be done in this area to determine the exact effect of menstrual dysfunction on metabolic rate and thyroid hormones.

Finally, a number of researchers have compared the diets of eumenorrheic and amenorrheic athletes to determine if energy intake and/or nutrient intakes differ between these two groups. Some of these studies have also compared bone density between the groups. These studies are presented in Table 1. A few comments need to be made about these studies before we compare the energy and nutrient intakes of the groups. First, most of the studies examined female runners, although some included other types of athletes; thus, these data primarily reflect the diets of female runners. Second, for the most part menstrual status has been self-reported. Therefore, we do not know if the amenorrheic athletes had more subtle changes in menstrual function, such as anovulation or luteal phase deficiencies. Third, the studies varied in how carefully diet records were collected, the number of days
Table 1 Summary of Studies on Mean Energy and Nutrient Intakes of Amenorrheic and Eumenorrheic Athletes

<table>
<thead>
<tr>
<th>Age (years)</th>
<th>Weight (kg)</th>
<th>Body Fat (%)</th>
<th>Energy (kcal/day)</th>
<th>Energy (kcal/kgBW)</th>
<th>Protein (g/kgBW)</th>
<th>Fat (%)</th>
<th>CHO (g/kgBW)</th>
<th>Running (miles/week)</th>
<th>Lumbar spine BMD (gm/cm²)</th>
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</thead>
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<tr>
<td><strong>Amenorrheic athletes</strong></td>
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</tr>
<tr>
<td>Baer &amp; Taper (4) d</td>
<td>16</td>
<td>51.0</td>
<td>16.3</td>
<td>1,912</td>
<td>44.0</td>
<td>1.50</td>
<td>35</td>
<td>4.7</td>
<td>20</td>
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<td>Duester et al. (22) d</td>
<td>25</td>
<td>49.3</td>
<td>11.2</td>
<td>2,151</td>
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<td>1.50</td>
<td>28</td>
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<td>74</td>
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<td>54.4</td>
<td>15.8</td>
<td>1,623</td>
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<td>1.22</td>
<td>31</td>
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<td>42</td>
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<td>52.3</td>
<td>17.4</td>
<td>1,551</td>
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<td>1.01</td>
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<td>3.3</td>
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<td>49.3</td>
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<td>32.1</td>
<td>1.04</td>
<td>27</td>
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<td>39</td>
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<td>13.8</td>
<td>1,668</td>
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<td>1.08</td>
<td>25</td>
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<td>10.0</td>
<td>1,272</td>
<td>25.6</td>
<td>0.99</td>
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<td>30</td>
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<td>14.6</td>
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<td>1.04</td>
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<td>1,730</td>
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(continued)
Table 1 (continued)

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<th>Classification of menstrual status</th>
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<td>NS</td>
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<td>Loucks et al. (49)*</td>
<td>-137</td>
<td>NS</td>
<td>Hormones</td>
</tr>
<tr>
<td>Marcus et al. (52)*(h)</td>
<td>-443</td>
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<tr>
<td>Myerson et al. (57)*(i)</td>
<td>-204</td>
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<tr>
<td>Nelson et al. (59)*(d)</td>
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<td>=.02</td>
<td>Self-report</td>
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<tr>
<td>Wilmore et al. (84)*(j)</td>
<td>9</td>
<td>NS</td>
<td>Hormones</td>
</tr>
</tbody>
</table>

*Measured by hydrostatic weighing except for Baer and Taper (4) and Duester et al. (22), who used skinfolds. \(^*\)Asterisk indicates significant differences in bone mineral density (BMD) between groups using the L1–L4 or L2–L4 sites of the lumbar spine. \(^\#\)Used 7-day diet records. \(^\&\)Used 3-day diet records. \(^c\)Grouped oligomenorrheic and amenorrheic athletes together. \(^\text{R}\)Reported that eumenorrheic athletes had shortened luteal phase. \(^\text{R}\)Used runners, cyclists, and triathletes. \(^\text{R}\)Reported that 3 amenorrheic athletes were bulimic. \(^\text{R}\)Reported that 3 eumenorrheic athletes were anovulatory. \(^\text{R}\)Did not mention mileage, only that subjects were elite distance runners, and elite distance runners typically run 50–70 miles per week.
collected, and the accuracy of the nutrient databases used to analyze the diets. Thus, caution should be used when comparing the nutrient and energy data between studies. Fourth, we cannot discount the possibility of underreporting in these athletes. Individuals vary in their ability to accurately record dietary intake, their willingness to disclose consumption of certain foods, and their attitudes toward food. There is the possibility that athletes in both groups might be consciously restricting energy intake to lose or maintain a low body weight. Although some of the studies specifically screened for eating disorders, some of the athletes may have had subclinical or clinical eating disorders that were not evident to the researchers. Finally, these dietary intake data only reflect one side of the energy balance equation. Only Wilmore et al. (84) attempted to measure energy balance in their subjects and found all their groups to be in apparent negative energy balance (amenorrheic runners = −788 kcal/day; eumenorrheic runners = −748 kcal/day; eumenorrheic active controls = −505 kcal/day). No long-term history of weight stability was given. Thus, we do not know whether or to what degree negative energy balance may have been present in the athletes in these studies.

Dietary intake data comparing eumenorrheic and amenorrheic athletes are given in Table 1. In general, energy intake was lower in the amenorrheic athletes, but only two studies reported significant differences. This failure to find significant differences between the groups is not unusual and most likely is due to the large variance in reported energy intake within each group. Many of the researchers noted that the athletes, regardless of group, consumed diets similar to sedentary controls (35, 57). For example, Broocks et al. (12) found that sedentary controls and recreational runners with and without menstrual dysfunction all consumed similar energy intakes (within 2,120–2,200 kcal/day). They identified inadequate energy intake as one factor contributing to menstrual dysfunction in their recreational runners. In general, mean protein intakes (kcal/kg body weight) were higher in the eumenorrheic athletes and were above the Recommended Dietary Allowance (RDA) for protein (0.8 g/kg body weight) (27) in all but one study. It should be noted that in many of these studies mean protein intakes were well below the 1.2–1.4 g/kg body weight recommended for endurance athletes (41). Fat intake varied greatly among groups and ranged from 25 to 40% of energy intake. No group reported unusually low fat intakes, and there appeared to be no consistent differences between amenorrheic and eumenorrheic athletes. Finally, mean carbohydrate intake (g/kg body weight) was low in both groups but especially low for the amenorrheic athletes. Some of these studies reported mean carbohydrate intakes to be <3.5 g/kg body weight. Carbohydrate intakes less than 5 g/kg body weight will not adequately replace muscle glycogen in endurance female athletes who are training at high intensities. Thus, glycogen stores would be low in many of these athletes, increasing the risk of fatigue, injuries, and poor sport performances (19). Besides comparing dietary parameters, five studies compared bone density between the groups; all but one reported significant reductions in the amenorrheic athletes. Although it is difficult to make definitive conclusions about the dietary intakes of these two groups, it does appear that amenorrheic athletes frequently have lower energy, protein, and carbohydrate intakes than eumenorrheic athletes. These lower intakes may also be reflected in lower micronutrient intakes in the amenorrheic population, although most studies did not report these data.

The energy drain model proposes that exercise-induced menstrual dysfunction may be initiated in part by negative energy balance, which in turn produces a
variety of physiological responses. These responses may vary among individuals and may depend on a variety of factors: the degree and duration of negative energy balance; the initial level of energy reserves; the degree, composition, and distribution of weight lost; the body’s response to various physiological and psychological stressors, including training intensity; and initial menstrual status.

**Mechanisms**

This section will briefly highlight some of the proposed mechanisms of athletic menstrual dysfunction. We will focus on the underlying disruption of homeostasis, the hormonal dysregulation, and the diverse symptoms associated with athletic menstrual dysfunction.

**Normal Menstrual Cycle**

To illustrate the complexity of the menstrual cycle and its intricately timed series of hormonal events, we have briefly described the “normal” menstrual cycle; for more extensive details see Goldfein and Monroe (31). The menstrual cycle is typically characterized by two phases that are separated by ovulation at midcycle (Figure 2). The beginning or follicular phase is marked by the onset of menstruation. During this phase of the cycle FSH plays an integral role in the recruitment of a single follicle. As the follicle grows, it begins to secrete estradiol, which acts in a positive feedback loop to stimulate the release of LH. The end of the follicular phase is marked by ovulation, which typically occurs on Days 15–18, and is characterized

![Normal Menstrual Cycle](image)

**Figure 2** — A simplified diagram of the “normal menstrual cycle” showing changes in plasma levels of hormones, including follicle-stimulating hormone (FSH), luteinizing hormone (LH), estradiol (E2), and progesterone (P). Days 1–15 represent the follicular phase and Days 15–28 the luteal phase; they are divided by ovulation at the midpoint of the cycle.
by a large increase in LH. The hormonal events associated with the follicular phase ensure that ovulation and fertilization take place.

After ovulation, the second phase of the cycle begins. This stage, called the luteal phase, is characterized by increases in ovarian production of estradiol and progesterone, which act in a negative feedback loop to inhibit LH and FSH release. These ovarian steroids play an integral role in preparing the uterus for implantation, if fertilization occurs. Without fertilization, there is a rapid decrease in estradiol and progesterone as the corpus luteum becomes atrophied. The intricate series of hormonal events associated with normal menstrual function can be easily disrupted by physiological, metabolic, and/or physiological stress. Furthermore, the intensity of the stressor can have variable effects on the level and magnitude of disruption.

**Athletic Menstrual Dysfunction**

The impact of diet and exercise on reproductive physiology is variable and ranges from no recognizable effect on the reproductive hormones to the severely dampened hormonal profile associated with athletic amenorrhea. Between these two extremes are various categories of reproductive dysregulation that are difficult to discern without sensitive assays to detect the subtle hormonal changes (50). Furthermore, comprehensive statistical packages are needed to analyze the often complicated variations in the pulsatile pattern of pituitary hormone release (77). The categories of these intermediate stages are subject to multiple interpretations and are often defined differently by physicians (according to clinical symptoms) (31) and researchers (regarding underlying mechanisms) (45). The hormonal changes associated with the cessation of menstrual function are reflected by the complete inhibition of the reproductive axis. These changes include decreases in the activity of the pulse generator regulating GnRH release, pituitary release of LH and FSH, and ovarian steroid hormone production of estradiol and progesterone. The intermediate stages of disruption include more subtle changes in pulsatile release of LH and FSH and ovarian insufficiency, as reflected by decreased production of estradiol and progesterone. These subtle hormonal changes are manifested as anovulatory cycles and shortened luteal phases. It is unclear whether these various reproductive profiles represent multiple adaptations to the same relative training stimulus or whether they reflect the impact of different training intensities and degrees of energy deficits.

This alteration of the HPO axis in the female athlete is believed to be an adaptive response to the stress of training and/or the metabolic stress of chronic energy deficit. This is supported by research showing that reduced energy intake and dietary restraint are associated with dampened hormonal profiles and menstrual dysfunction (5, 69). The effect of this stress on the reproductive system is reflected endocrinologically by increased levels of plasma cortisol from the HPA axis. It has been shown in both animal and human models that activation of the HPA axis results in a compensatory down-regulation of the HPO axis (67). Furthermore, this activation of the HPA axis has many ramifications on other neuroendocrine systems including the thyroid axis and growth hormone axis. The effect of elevated plasma cortisol on numerous physiological systems (17) can help explain the impact of stress in the overtrained athlete (see Figure 3).
The hormonal profile characteristic of the amenorrheic athlete includes elevated levels of glucocorticoids and suppression of estradiol and progesterone. These hormonal aberrations can have multiple adverse effects, including damage and inadequate repair of soft tissue, inhibition of bone formation and enhancement of bone resorption, inhibition of immune and thyroid function, and adverse effects on cardiovascular and renal function (3). In the short term, these responses to elevated levels of cortisol are adaptive and have a positive impact on initiating such responses as the fight or flight response. Chronic activation of the HPA axis, however, and inhibition of ovarian steroid production can be seen as a long-term health risk. Clearly, the amenorrheic individual is at risk for these long-term negative effects on health.

Unfortunately, it has not yet been clearly established at what level the alteration in hormonal profile becomes associated with increased health risks. However, some evidence is beginning to emerge. For example, Prior et al. (64) reported lower spinal BMD in premenopausal women with asymptomatic disturbances of ovulation (without amenorrhea). These individuals lost BMD at twice the rate of controls (4.2%/year vs. 2.0%/year, respectively). At the present it is not known where on the menstrual function continuum the positive effects of ovarian
steroids become overshadowed by the detrimental effects of chronic elevations in cortisol. Perhaps these detrimental effects will be associated with a ratio rather than an absolute hormonal level. In the male athlete, the ratio of testosterone to cortisol has been extensively examined as a factor limiting muscle hypertrophy and performance (20, 75). Thus, it may be more appropriate to look at the ratio of estradiol to cortisol in the active female to determine the impact of hormone dysregulation on long-term health risks. Clearly more information is necessary and more longitudinal studies on females are desperately needed.

Women with amenorrhea typically present with multiple endocrine abnormalities and symptoms (34), including low estrogen and progesterone, elevated cortisol, low thyroid hormones, high prolactin, fatigue, depression, and decreased performance. Exogenous estrogen replacement may be treating only one symptom of the disorder without reducing the risk of chronic disease. Additionally, supplementation of estrogen with or without progesterone may be ineffective in improving bone density in the presence of high cortisol, due to cortisol’s counter-regulatory effects. Thus, it is extremely important to understand the physiological alterations of this disorder to better assist in its diagnosis and treatment. Furthermore, a better understanding of the factors that lead to menstrual dysfunction will help health professionals identify the disorder before it develops into multiple abnormalities.

Adverse Effects

The potential health problems associated with athletic menstrual dysfunction extend far beyond the reproductive system. This can be clearly demonstrated when one considers all the bodily systems affected by the hormonal milieu involved with normal menstrual function. Of particular concern are the reduced levels of estrogen and progesterone characteristically seen in active females displaying menstrual dysfunction. Elevated levels of the “stress hormone” cortisol have also been suggested to have numerous adverse effects on a variety of tissues. The following sections will outline the health risks associated with menstrual dysfunction, the underlying mechanisms of those risks, and the possible effects they may have on athletic performance.

Reproductive System

The three principal types of menstrual changes observed in athletes and active women are luteal phase deficiency, anovulation, and amenorrhea (61). Luteal phase deficiency is most commonly characterized by insufficient progesterone production with or without a shortened luteal phase length. Because total menstrual cycle length may remain normal, many women do not notice any change in menstrual status. Luteal inadequacy may represent a midpoint on the menstrual function continuum. It has also been suggested that luteal phase deficiency represents the endpoint of successful adaptation to athletic training in more robust women (45). Few studies have examined the adverse effects of luteal inadequacy; however, the suggested and/or documented risks include infertility, recurrent spontaneous abortion, endometrial hyperplasia, adenocarcinoma (61), and lower BMD (63).

Similar to the subtle hormonal changes associated with luteal phase deficiency, anovulatory cycles often occur despite the presence of menstrual bleeding.
There are several different patterns of irregular bleeding associated with anovulation, ranging from very short cycles (less than 21 days) to longer cycles of 35 to 150 days between bleeding. This latter pattern of bleeding is often referred to as oligomenorrhea. Anovulatory female athletes have dampened levels of estrogen and lack the spike in LH that results in ovulation. Another characteristic of this dysfunction menstrual pattern is extremely low levels of progesterone. The unopposed estrogen secretion associated with anovulation has been shown to cause proliferative endometrial overgrowth. The cyclic production of progesterone is necessary for endometrial shedding; otherwise, the endometrium becomes overstimulated in the presence of unopposed estrogen (81). The most noticeable risks associated with chronic anovulation and unopposed estrogen are endometrial hyperplasia and adenocarcinoma.

**Skeletal System**

Increasing evidence over the past decade has indicated that women with athletic amenorrhea are more likely to demonstrate diminished BMD in the axial and appendicular skeleton. The reduced levels of endogenous estrogen associated with menstrual dysfunction may attenuate the positive influence of exercise on the development of peak bone mass. Amenorrheic athletes typically display reduced levels of estradiol and progesterone and have hormonal profiles more similar to those of postmenopausal women than to those of their age-matched eumenorrheic counterparts (24, 46, 47). Thus, despite the established positive effects of exercise training on bone, training is unable to compensate wholly for the negative effects of estrogen and progesterone deficiency (21).

During estrogen deficiency, bone becomes more sensitive to the calcium-mobilizing effect of parathyroid hormone (21). As a result, a greater number of resorptive sites are established and there is a gradual loss of bone mass. Numerous studies have confirmed that axial bone mass is reduced approximately 20% in amenorrheic athletes compared to their athletic eumenorrheic counterparts but only 10% compared to sedentary women with normal menstrual function (16, 24, 35, 52, 59). In a longitudinal study by Drinkwater and colleagues (25), athletes who remained amenorrheic continued to lose bone mass, whereas athletes who resumed menstrual function showed a significant increase in bone mass. Despite this initial increase, however, bone mass levels were still lower than those in active and sedentary eumenorrheic women. Thus, despite the osteogenic stimulus of exercise on bone, hormonal aberrations associated with menstrual dysfunction compromise bone mineral status and increase the risk for fracture. This increased risk not only may jeopardize an individual’s athletic career but also increases the risk for fracture after menopause.

In the amenorrheic athlete, not only is bone density affected by lower estrogen and progesterone levels, but in many cases elevated levels of plasma cortisol may play a contributing role. Cortisol has been shown to affect bone mass by inhibiting bone formation while stimulating bone resorption. Cortisol may also contribute indirectly to lower bone density by interfering with calcium absorption in the digestive tract and increasing urinary calcium excretion. Thus, the hormonal aberrations associated with athletic menstrual dysfunction inhibit bone formation while increasing the rate of bone resorption (3).
It is important to mention that not all studies support reduced BMD in amenorrheic athletes compared to active eumenorrheic females (26, 84). Wilmore et al. (84) found no differences in lumbar spine BMD between amenorrheic and eumenorrheic runners. Furthermore, Dueck et al. (26) reported a higher BMD in the femoral neck of an endurance amenorrheic athlete compared to her eumenorrheic teammates. These discrepancies illustrate the need to consider several important issues when interpreting bone density data. The development of peak bone mass is not solely determined by the levels of estrogen and progesterone present during puberty and the ensuing 10–15 years of musculoskeletal growth and development. Bone mass can also be greatly influenced by genetics and by dietary factors such as adequate calcium and energy intake. Furthermore, the type of sport an individual has participated in and the duration of participation may also play a role in the accretion of bone mass. Thus, the actual BMD that these athletes could have achieved if menstrual dysfunction had not occurred is unknown.

**Cardiovascular System**

It has been well established that the relative high levels of ovarian steroid production that occur before menopause offer some protection against coronary heart disease. What has yet to be determined, however, is whether the hypoestrogenic state associated with athletic amenorrhea may predispose an individual to cardiovascular disease despite her active lifestyle. When compared to eumenorrheic active females, amenorrheic athletes have been shown to have higher levels of plasma cholesterol, triglyceride, and low-density lipoprotein cholesterol (LDL–C) (28, 39). Friday and colleagues (28) determined the interactive effects of hormones, exercise, and diet on plasma lipids and lipoproteins. They measured serum estrogen and progesterone levels, nutrient intake, and plasma lipid concentrations in 24 hypoestrogenic amenorrheic athletes and 44 eumenorrheic athletes. Amenorrheic athletes had significantly higher levels of plasma cholesterol ($p = .003$), triglycerides ($p = .046$), LDL–C ($p = .037$), and high-density lipoprotein cholesterol (HDL–C) ($p = .007$); however, there were no differences in the LDL–C/HDL–C ratio between the groups. Conversely, Lamon-Fava et al. (39) reported lower plasma apo A-I levels and significantly lower A-I/apo B ratios in amenorrheic versus eumenorrheic female runners. The LDL–C/HDL–C ratio was not reported in this study. Consequently, there is a concern that the adverse changes in lipids and lipoproteins associated with menstrual dysfunction may increase the risk for premature atherogenesis in amenorrheic athletes.

Based on clinical observations, several biologically plausible mechanisms for estrogen’s apparent cardioprotective effect have been suggested (32). The most widely supported role of estrogen in protection against heart disease is its positive effect on blood lipoproteins and lipoprotein composition. More specifically, physiological levels of estrogen have been shown to reduce concentrations of LDL–C, a risk factor for early cardiovascular disease, while concurrently increasing levels of HDL–C (65). Endogenous estrogen has also been shown to lower levels of the blood-clotting factor fibrinogen, which might in turn reduce the incidence of blood vessel blockages and myocardial infarctions (32). Other research has suggested that estrogen also promotes blood vessel dilation and enhances angiogenesis, resulting in a decreased risk for hypertension. Furthermore, elevations in cortisol may also contribute to the negative cardiovascular effects associated with overtraining and
menstrual dysfunction. Due to their catecholamine-enhancing effects, increased levels of glucocorticoids may increase vasoconstriction in capillary and blood vessels, thus increasing the risk of hypertension (3).

**Immune Function**

It has been well established that the hormonal milieu associated with reproductive function is tightly coupled to immune response. The magnitude of this relationship, however, differs between genders. Females are immunologically superior to males, as evidenced by higher circulating levels of the major immunoglobulins and stronger cell-mediated and humoral responses. The critical hormone responsible for this gender difference is estrogen. Estrogen increases secretions of both prolactin and growth hormone, which in turn increases the production of various immune cells. The result is an increased ability in women to fight off viruses and bacteria (54).

Unfortunately, the adverse effects of high-performance training on reproductive function impair estrogen’s role in enhancing immune function. More specifically, it appears that the immunostimulatory hormones estrogen and prolactin may become suppressed because of chronic training. Concomitantly, the hormones that inhibit immune function, such as cortisol, often become chronically elevated. Cortisol inhibits the immune system by reducing circulating lymphocytes and monocytes as well as by decreasing their production and function. Furthermore, cortisol can contribute to decreased immune function by interfering with the synthesis of prostaglandins and thereby affecting inflammation (3). This hormonal profile places the active female at an increased risk for infection. Furthermore, the healing processes required for appropriate recovery from either an acute bout of strenuous exercise or an injury may become impaired.

**Cognitive Ability**

Although exercise is usually shown to be beneficial for reducing tension and increasing overall psychological well-being, this may not be the case for the amenorrheic athlete. Exercise that is sufficient to disturb the normal hormonal balance in female athletes has been shown to be psychologically self-defeating, producing a mood state profile that is less desirable than that of inactive women. More specifically, amenorrheic runners have been shown to display a higher anger score than eumenorrheic runners or inactive women (18). This finding is in direct opposition to the general contention that anxiety, anger, and hostility are subdued with exercise (55, 56).

The responses of active women to the physical stressors of training and competition are reflected in changes in menstrual function. The hormonal changes associated with menstrual dysfunction may also influence emotional behaviors and cognitive ability. It is possible that the excessive amount of training associated with athletic amenorrhea may result in reduced cognitive ability, as evidenced by alterations in the Profile of Mood State (POMS) (18, 55, 60, 66), and consequently may impair athletic performance. Furthermore, this reduced cognitive state may decrease an athlete’s sensitivity to the warning signs of an impending injury, thus increasing the risk of an injury that is more serious and threatening to performance. Elevations in cortisol may also decrease cognitive function, memory, and concentration. Additionally, elevated cortisol can affect eating behavior and sleep patterns (3).
Exercise Performance

Perhaps the most influential information that can be used to persuade an athlete to seek treatment for her menstrual dysfunction concerns its adverse effects on performance. Rather than serving as an indicator of appropriate training volume and intensity, amenorrhea should be regarded as a clinical manifestation of the over-trained state. As previously stated, one of the predisposing factors associated with menstrual dysfunction is chronic energy deficiency. When energy expenditure exceeds energy intake, the body is required to conserve energy. One way this may be achieved is through the cessation of menstrual function. Consequently, many systems in the body become adversely affected, resulting in numerous performance-hindering side effects. These adverse effects include increased risk of stress fractures and other musculoskeletal injuries, a prolonged healing process, and reduced ability to recover from acute training sessions.

Lloyd et al. (43) examined the effect of menstrual status on musculoskeletal injuries by surveying recreational athletes and reviewing the medical records of 207 collegiate female athletes. Of the 267 women who completed the survey, 39% reported an interruption of at least 3 months in their training program. The most common cause for interruption was injury. Those recreational athletes who experienced an injury were more likely to have had irregular or absent menses and less likely to have been using oral contraceptives than the other runners who experienced no interruption in their training program. Furthermore, of the 207 collegiate women athletes, x-ray documented fractures occurred in 9% of the athletes who reported regular menses and 24% of the athletes with irregular or absent menses. Benson et al. (8) found similar results in ballet dancers. Dancers with abnormal menses had significantly more bone injuries than normally menstruating dancers. These findings support the hypothesis that athletes and active females who display menstrual dysfunction while engaging in vigorous exercise programs are at an increased risk for musculoskeletal injury.

An important point to emphasize when dealing with the menstrual dysfunctional athlete is that performance will most likely improve once menstrual function resumes. In a case study conducted by Dueck et al. (26), the amenorrheic athlete’s performance consistently improved throughout the track season once she was placed on a diet and training intervention program and positive energy balance was achieved. Furthermore, half of the amenorrheic athletes who are currently in our diet and training intervention program resumed menstrual function within 3 months of improving energy balance (Dueck, unpublished data). All of these athletes have consequently reported an increase in overall energy and various performance parameters. Thus, it appears that the establishment of positive energy balance and ensuing resumption of menstrual function enhances performance and reduces the risk of injury and other performance-hindering side effects.

Treatment Options

When treating the athlete or active female diagnosed with exercise-associated amenorrhea or other sport-related menstrual dysfunctions, several important factors should be considered. It is essential to determine the value each athlete places on sport and competition regarding her personal identity and lifestyle. In addition, the anticipated time an athlete plans to be involved in intense exercise or competition
may influence recommendations regarding treatment. For example, the elite-level competitor who is planning on a long-term professional athletic career may initially be more resistant to modifying training parameters than the recreational athlete. Many athletes will also be resistant to the initiation of hormonal therapy because of the perception that the treatment will have adverse effects on body composition and performance. Consequently, it is imperative that the athlete be educated regarding the etiology of her menstrual dysfunction and its potential complications. In addition, several treatment options should be offered so that the athlete may take an active role in deciding which form of treatment will be effective considering her lifestyle and beliefs. The following section will outline the current treatment options for athletes and active females experiencing menstrual dysfunction.

**Hormonal Therapy**

The most common mode of treating athletic amenorrhea is the use of hormonal therapy. The rationale behind this form of treatment is to provide an exogenous source of estrogen and/or progesterone to prevent the adverse side effects associated with a prolonged hypoestrogenic state. Thus, hormonal therapy attempts to return the dampened hormonal profile of an amenorrheic athlete to that associated with a normal menstrual cycle. When treating the amenorrheic athlete with exogenous hormones, most physicians use doses and protocols similar to those recommended for postmenopausal women (53). Hormonal replacement, however, only prevents further bone loss and may not increase bone mass. An extensive body of research is not available concerning the use of hormone replacement therapy in amenorrheic athletes and active females. Frequently, recommendations are based solely on the dosage required to prevent further bone loss in populations of postmenopausal women (61). Some evidence is now available indicating that hormone replacement therapy (medroxyprogesterone with or without calcium supplements) can increase spinal bone density in premenopausal physically active women with amenorrhea or ovulatory dysfunction (63). However, further research is still necessary to determine the most appropriate estrogen and/or progesterone doses or forms of therapy that will be most effective for amenorrheic athletes.

The two most common methods of hormonal therapy are cyclic estrogen progesterone therapy and oral contraceptives (70). A cyclical estrogen and progesterone preparation will produce regular withdrawal bleeding at predictable times but does not provide contraception. The typical dosage consists of 0.625 mg of conjugated estrogen on Days 1 to 25 and progesterone on Days 14 to 25 (61). The minimum estrogen dose that has been shown to prevent bone loss in postmenopausal women is 0.625 mg/day or 0.3 mg/day if combined with calcium supplementation (70). Side effects are less severe with this form of hormonal treatment but may include depression, lethargy, and bloating on the days of progesterone ingestion.

If the athlete is sexually active, she should be counseled about contraceptive options. Amenorrhea in itself is not an effective form of birth control. The first ovulation during the resumption of normal menstrual function may occur before the onset of menstrual bleeding. Thus, it is possible for an amenorrheic athlete to become pregnant before showing any obvious signs of resuming menstrual function. Consequently, oral contraceptives are the preferred form of hormonal treatment if contraception is an issue. The higher hormonal dosage contained in oral contraceptives, however, is associated with a greater array of adverse side effects.
Athletes may be particularly concerned about the adverse effects of weight gain, fatigue, nausea, and mood alterations on performance. Furthermore, the literature remains equivocal regarding the effect oral contraceptives may have on other factors such as substrate utilization and maximal aerobic capacity (7, 11, 40).

If hormonal therapy is used to treat amenorrhea, the athlete should be advised that this therapy does not correct the underlying problem, even though regular cyclic bleeding may resume. Furthermore, hormonal therapy may actually mask the resumption of normal menstrual function. To our knowledge, no studies have been conducted to determine when hormonal therapy should be stopped to see if menses have spontaneously resumed. If there have been no changes in lifestyle factors, however, clinical judgment indicates that menstrual function will continue to be impaired (61).

**Lifestyle Intervention**

Changes in lifestyle factors, such as dietary and training habits, may be an alternative treatment for amenorrheic athletes who are unwilling to adhere to hormonal therapy. This alternative treatment may be particularly beneficial to athletes who do not have major body weight concerns or a distorted body image. This treatment entails carefully examining each athlete’s individual diet and training regimen so that energy status can be determined and considered in conjunction with her level of body fat. Many athletes who present with menstrual dysfunction are chronically energy deficient. These athletes typically report purposefully restricting energy intake for weight loss or experiencing weight loss during high-volume training periods. It is not unusual for female athletes to participate in two exercise training sessions per day, easily expending over 1,000 kcal per day in exercise alone. In addition, the athlete may be under high emotional stress to perform well. If energy intake does not meet these increased needs, a significant energy deficit will occur and consequently prompt the body to conserve energy. One way this is accomplished is through the cessation of menstrual function. As a result, the metabolic costs associated with the menstrual cycle and maintaining a pregnancy are reduced or eliminated. By reducing exercise energy expenditure and increasing energy intake, the body can be brought back to a state of energy balance. This in turn helps the athlete to maintain body weight during periods of intense training or to increase body weight and fat slightly. Once this occurs, it is probable that normal function will resume in all the systems affected by the reproductive endocrine axis.

It is also important to identify the stressors associated with menstrual dysfunction before instituting any form of pharmacological management. Hormonal therapy does not solve the underlying problems contributing to the cessation of menstrual function but rather treats the associated symptoms. Furthermore, hormonal therapy may mask or completely override the athlete’s own endogenous endocrine function, thus making it difficult to determine if normal menstrual function truly resumes. Therefore, efforts should be made to develop an individualized intervention program that focuses on correcting the underlying etiology associated with athletic menstrual dysfunction.

Few studies have examined the use of nonpharmacological treatments for the reversal of athletic amenorrhea. Dueck et al. (26) examined the effect of a 15-week diet and exercise intervention program on energy balance, hormonal profile, body composition, and menstrual function of an amenorrheic endurance athlete. The
intervention program reduced the athlete’s training regimen 1 day per week and included the use of a sport nutrition beverage (GatorPro®) providing an additional 360 kcal per day. The intervention program was associated with a transition from negative to positive energy balance, increased body fat from 8.2 to 14.4%, increased fasting LH and decreased fasting cortisol, and the eventual resumption of menstrual function. Athletic performance also improved as evidenced by the subject continually achieving her personal record at each track meet throughout the season. In addition, she broke two school records and qualified for the National Junior Collegiate Athletic Association Track and Field Meet in several events ranging from 800 to 10,000 m. These improvements occurred in spite of a very high fiber diet (33–48 g/day) throughout the treatment period. These results suggest that nonpharmacological modes of treatment for athletic amenorrhea can contribute to the resumption of a more normal hormonal profile and menstrual status in amenorrheic athletes and improved performance.

The successful implementation of this lifestyle intervention requires a holistic treatment approach in which many individuals are involved in the total health care of the athlete. This team approach should ideally include the athlete’s primary care physician, as well as an endocrinologist, sport nutritionist, psychologist, and exercise physiologist. During this form of treatment, it is essential that the athlete remain in close contact with members of the treatment team. This will ensure that modifications can be made if concern arises over such issues as increased body fat. Furthermore, the athlete’s coach and parents should be involved in the treatment regimen, especially concerning modifications in the athlete’s training schedule. Most importantly, the athlete herself needs to be educated regarding the clinical significance of menstrual dysfunction and the long-term effects it may have on her health and ability to achieve optimal performance.

Summary

Although the female athlete comes close to resembling society’s definition of beauty and fitness, this achievement comes at high cost to both health and performance. The hormonal aberrations associated with menstrual dysfunction can have multiple long-term effects on the reproductive, skeletal, and cardiovascular systems, as well as immune function and cognitive ability. Thus, the most appropriate form of treatment for menstrual dysfunction lies in its prevention. The prevention message will be achieved through the global educational efforts of health educators, athletic associations, coaches, trainers, parents, physicians, nutritionists, and members of the scientific community. All need to be involved in a team approach toward treating the active female. It is of utmost importance, however, that the prevention message reach the athlete herself. She may then, in turn, learn to develop healthy attitudes toward training, nutrition, and body weight and composition, while realizing her full athletic potential.

References


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