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Athletic Activity and Hormone Concentrations in High School Female Athletes

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Context: Physical activity may affect the concentrations of circulating endogenous hormones in female athletes. Understanding the relationship between athletic and physical activity and circulating female hormone concentrations is critical.

Objective: To test the hypotheses that (1) the estradiol-progesterone profile of high school adolescent girls participating in training, conditioning, and competition would differ from that of physically inactive, age-matched adolescent girls throughout a 3-month period; and (2) athletic training and conditioning would alter body composition (muscle, bone), leading to an increasingly greater lean–body-mass to fat–body-mass ratio with accompanying hormonal changes.

Design: Cohort study.

Settings: Laboratory and participants' homes.

Patients or Other Participants: A total of 106 adolescent girls, ages 14–18 years, who had experienced at least 3 menstrual cycles in their lifetime.

Main Outcome Measure(s): Participants were prospectively monitored throughout a 13-week period, with weekly physical activity assessments and 15 urine samples for estrogen, luteinizing hormone, creatinine, and progesterone concentrations. Each girl underwent body-composition measurements before and after the study period.

Results: Seventy-four of the 98 girls (76%) who completed the study classified themselves as athletes. Body mass index, body mass, and fat measures remained stable, and 17 teenagers had no complete menstrual cycle during the observation period. Mean concentrations of log(estrogen/creatinine) were slightly greater in nonathletes who had cycles of <24 or >35 days. Mean log(progesterone/creatinine) concentrations in nonathletes were less in the first half and greater in the second half of the cycle, but the differences were not statistically significant.

Conclusions: A moderate level of athletic or physical activity did not influence urine concentrations of estrogen, progesterone, or luteinizing hormones. However, none of the participants achieved high levels of physical activity. A significant number (17%) of girls in both activity groups were amenorrheic during the 3-month study period.

Key Words: menstrual cycle, estrogen, anterior cruciate ligament, exercise physiology

Key Points

- Urine concentrations of estrogen and progesterone in high school females were not altered by level of physical or athletic activity.
- A significant number of both athletic and nonathletic young women were amenorrheic and probably anovulatory during the 3-month study period.
- Athletic training and conditioning did not alter body composition over the course of the study.

Several studies have shown that varying levels of physical activity can alter the length of the menstrual cycle,[1,2] determine whether ovulation occurs,[3,4] and alter the pulse frequency of luteinizing hormone (LH).[1,5] Therefore, an understanding of the effect of exercise on hormone concentrations and menstrual cycle regularity may be fundamental to understanding the female athlete's physiology.

To expand on previous work,[6,7] we designed this study to investigate (1) whether estradiol-progesterone profiles of female high school athletes differed from those of inactive age-matched controls and (2) whether the relationship between body composition (lean–body-mass to fat-mass ratio) changed relative to estradiol and progesterone profiles.

ENDOCRINOLOGY OF PHYSICAL ACTIVITY AND MENSTRUAL CYCLICITY

A fundamental sex difference is the female menstrual cycle, which evolved to select and mature oocytes, prepare the uterus for pregnancy, and reset the reproductive system for the next cycle if pregnancy does not ensue. Normal ovulatory cycles demonstrate predictable patterns of hormonal variability at all secretory sites: gonadotropin-releasing hormone from the hypothalamus, follicle-stimulating hormone and LH from the pituitary; and the steroids estradiol, progesterone, and testosterone from the ovary.[8] The follicular phase of the normal cycle incorporates follicle growth, culminating in oocyte maturation and peak estradiol secretion. A brief ovulatory phase encompasses a surge in LH and initiation of the final oocyte maturation, oocyte release, and the formation of a corpus luteum.

During the luteal phase, estradiol and progesterone are secreted, and progesterone secretion peaks a week after ovulation. The onset of menses results from the decline in hormone production from the corpus luteum, a return to basal levels, and the onset of a new follicular phase.[8]

Physical Activity, Energy Balance, and Menstrual Cycle Characteristics

High and moderate levels of physical activity can alter menstrual function, although the frequency and type of menstrual disturbance varies across studies and by level and type of activity.[9] Female ballet dancers and runners, in particular, have a greater frequency of amenorrhea, anovulation, and luteal-phase defects than nonathletes do.[9–12] Results from several epidemiologic studies[2,3,10,13] suggest that women participating in moderate recreational activity have longer and more variable cycles than do sedentary women, whereas amenorrhea has been induced in previously sedentary women with the initiation of a vigorous training program.[14] Athletes who sustain regular levels of physical activity may also experience a shortened luteal phase, even when no other changes in the menstrual cycle are observed.[15,16] The hormonal changes that underlie these alterations may include lower levels of follicular-phase estradiol, lower luteal-phase progesterone, and the absence of the midcycle LH surge.[17]

Physical exercise affects menstrual function through metabolic and psychogenic stress pathways whereby gonadotropin-releasing hormone and gonadotropin activity are inhibited, with accompanying declines in serum estrogen and anovulation.[11,12,18,19] The now well-established phenomenon of the *female athletic triad*[20,21]defines the complex relationship among low-energy availability, menstrual disruption, and bone health. Nutrition is critical to menstrual function, with caloric energy balance and food composition being relevant nutritional factors.[9,12,18,19,22,23] Dietary restraint, independent of actual weight loss and aberrant eating behaviors, also disrupts ovarian function[11,19]and menstrual cycle length.[9] Exercise coupled with inadequate caloric intake, a restricted diet, or aberrant eating behaviors can produce a state of negative energy balance, leading to a centrally mediated inhibition of gonadotropin-releasing hormone and functional hypothalamic amenorrhea. The associated estrogen deficiency may reduce bone formation, leading to low bone-mineral density, although low energy availability likely alters bone formation through additional pathways.[20,24]

Influence of Body Composition on Menstrual Cycle Function. Both body mass and body composition are associated with menstrual function; the probability of dysfunction is greater among women with either a low or high body mass index (BMI).[9,25–27] Among reproductive-aged women, those with the longest mean cycle lengths had greater BMI, lean body mass, and fat body mass, but women in the lowest decile of BMI and fat body mass also had long mean cycle lengths.[26] Studies[3,9,26,28] of athletes and the general population have consistently shown that individuals with highly variable cycles or amenorrhea tend to weigh less, to have a lower percentage of body fat, and to report more weight loss than women with normal menstrual cycles do. Weight loss that is greater than 20% to 30% of ideal weight or more than 10% of premorbid body mass is associated with amenorrhea; menstruation is likely to resume after a body-mass gain of about 4 kg or to within 5% to 15% of ideal weight.[9,19]

Heavier women report longer menstrual cycles.[13,29–31] Similarly, heavier women had significantly shorter luteal phases but longer follicular phases than did women of average body mass[32] and were more likely to experience anovulation.[33] In a recent population-based study, obese women had a 2.6-fold increase in the odds of reporting irregular cycles.[27] Women who are overweight or obese are at increased risk of having polycystic ovarian syndrome, a condition associated with oligomenorrhea and amenorrhea.[34] Nonetheless, in athletes whose body mass was within reference range, amenorrhea and oligomenorrhea have also been associated with polycystic ovarian syndrome and hyperandrogenism.[35]

With these hormone physiology factors in mind, we developed the following hypotheses:

Hypothesis 1. The estradiol-progesterone profile of high school girls participating in training, conditioning, and competition will differ from that of physically inactive, age-matched girls throughout a 3-month period.

Hypothesis 2. Athletic training and conditioning will alter body composition (muscle, bone, fat), leading to an increasingly greater lean–body-mass to fat–body-mass ratio along with accompanying hormonal changes.

METHODS

Participants

We obtained approval for the study from our institutional review board. A total of 106 adolescent girls were enrolled in the study and prospectively followed for 13 weeks. Volunteers were recruited via our institution's public clinical research page; flyers (approved by our institutional review board) posted around the community and in high schools; communication with local athletic trainers, coaches, teachers, and principals; and informal discourse from enrolled participants. Local public school board approval was also obtained to recruit directly on the school property. Both athletes and nonathletes (ie, those not active on a sports team throughout the duration of their participation in the study) were recruited, and the athletic status (athlete versus nonathlete) of participants at the time of their enrollment was determined (Figure 1). Participants were awarded small financial incentives, totaling a maximum of $100, for providing urine samples and complete questionnaires.

Inclusion Criteria. The age range for participation in this study was 14 to 18 years. Participants must have experienced at least 3 menstrual cycles in their lifetime and be willing to follow the defined protocol.

Exclusion Criteria. Girls who were premenarchal were excluded. Current hormonal therapy or inability to provide informed consent also prevented participation.

Study Protocol

Once a volunteer contacted the study coordinator and expressed interest in enrolling in the study, she (and her parent or legal guardian, if she was younger than 18 years) was interviewed to determine her eligibility to participate in the study as well as to review procedures and address

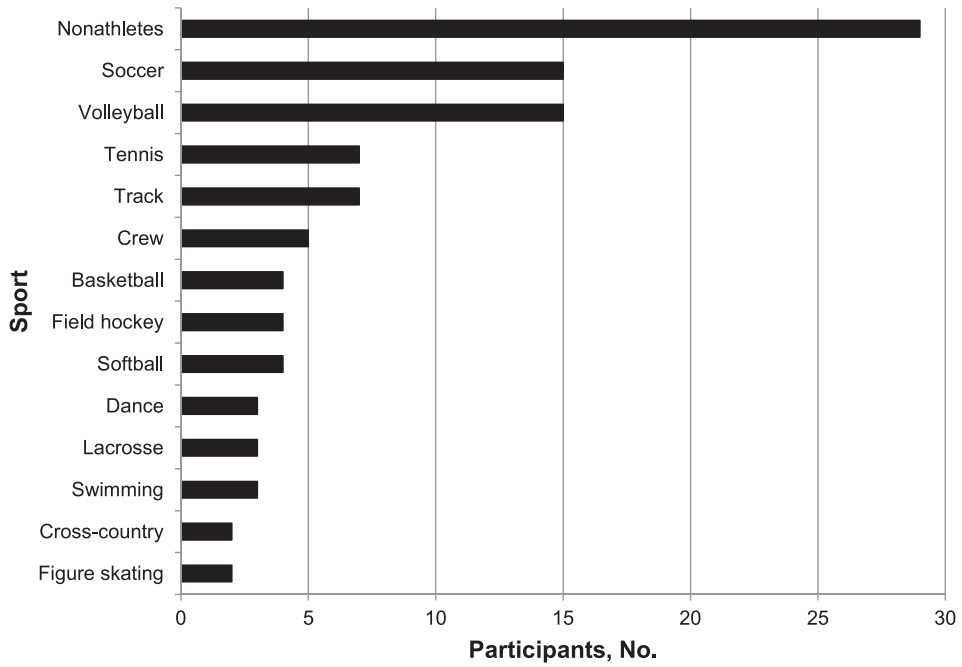

Figure 1. Summary of participants' sports activity during the study period (withdrawn participants were excluded). Nonathletes were not involved in a sport while participating in the study.

questions. Informed consent approved by the institutional review board was obtained from eligible participants or from their parents or legal guardians (if the participant was a minor). The rest of the scheduled interview consisted of pretests for body composition. Throughout the study, participants collected weekly urine specimens and completed weekly questionnaires regarding their physical activity levels. Time between consecutive menstrual cycles and day of current cycle were derived from reported menses start dates for each participant. Once the weekly specimen was collected, the participant's body composition and leg-strength posttests were performed.

Body-Composition Measurement

Body-composition measurements were made twice, once during enrollment and again after all 15 urine samples had been collected. Participant height and body mass were determined, and BMI was calculated. A bioelectrical impedance analysis (BIA) device (Quantum X; RJL Systems, Clinton Township, MI), which is a 4-terminal impedance plethysmograph, was used to assess body composition.[36] Measurement allows for a 2-compartment system comprising lean tissue and adipose tissue. Participants were asked to remove the shoe and sock from the left foot and lie supine on an examination table with the arms 30° from the body and legs not in contact with each other. The 4 electrode sites were cleaned with alcohol, particularly if the skin was dry or covered in lotion. Four single-use, resting electrodes were placed in the following locations on the left side of the body: between the metacarpophalangeal and proximal interphalangeal joints of the middle finger, on the wrist next to the ulnar styloid, at the base of the second toe, and on the ankle between the malleoli. Cable leads were attached to each of the electrodes, and all were connected to the handheld BIA instrument. Resistance and reactance values were determined as the participant refrained from movement for approximately 1 minute. The entire testing time, including setup, was less than 5 minutes. The resistance and reactance values were used to estimate body-composition data (fat, fat-free mass, and total body water) with the manufacturer's NHANES-III equation set.[37] Results of the BIA test were recorded and analyzed using body-composition software (RJL Systems).

Physical Activity Questionnaires

The Physical Activity Questionnaire for Adolescents (PAQ-A)[38] was administered to each participant. Weekly questionnaires concerning physical activity levels and menstrual cycle information were completed once during the first week and each week thereafter (n = 13). The questionnaires were the same each week and included the PAQ-A[38] and the International Physical Activity Questionnaire.[39] The PAQ-A scores range from 1 to 5, with low scores indicating low levels of physical activity and high scores indicating high levels of physical activity. In our study, the PAQ-A scores ranged from 1 to 3.3 and were divided into tertiles of less than 1.76 (the lowest 33%), 1.76 to 2.22 (the middle 33%), and greater than 2.22 (the uppermost 33%). The questionnaires took approximately 5 to 10 minutes to complete each week and pertained to the previous 7 days.

Specimen Collections

On enrollment, each participant was provided with specimen-collection kits. The study coordinator reviewed the collection procedure and schedule with each participant. Urine specimen collections started within 7 days of the enrollment session, beginning with 3 baseline specimens collected on 3 consecutive days. The participants then proceeded to collect 1 specimen per week (on the same day

each week or as close to every 7 days as possible) for the next 12 weeks, thereby collecting 15 specimens in the 13 weeks. Once the volunteers completed their participation in the study, the samples were delivered to our institution's clinical ligand assay service satellite laboratory, where all of the assays were performed. The clinical ligand assay service satellite laboratory is accredited by the Commission on Laboratory Accreditation of the College of American Pathologists and is certified under the Clinical Laboratory Improvement Act.

Urinary Assay Analysis

The urinary estrone-conjugate (E1c) assay is a semiautomated, competitive immunoassay with offline incubation. The reporting range for the E1c assay is 5.10 to 408.0 ng/mL. The assay is standardized against a dual standard prepared with estrone-glucuronide and estrone-sulfate obtained from Sigma Chemical Company (St Louis, MO). The interassay coefficient of variation (CV) = 11.00% and the intraassay CV = 7.77%. The urinary pregnanediol-glucuronide (PdG) assay is also a semiautomated, competitive immunoassay with offline incubation. The assay requires 50 μL of urine in addition to sufficient dead volume for aspiration, repeating, and creatinine correction. The reporting range for the urine PdG assay is 0.005 to 25.5 μg/mL. The assay is standardized against PdG obtained from Sigma Chemical Company. The interassay CV = 16.31% and the intraassay CV = 7.65%. The urinary LH assay is a 2-site, or sandwich, immunoassay run on a Bayer Diagnostic ACS-180 automated analyzer (LH2 MCM; Siemens Healthcare Global, Tarrytown, NY) using chemiluminescent technology. The assay uses 2 monoclonal antibodies. One is directed against the α subunit, 1 against the β subunit, 1 labeled with dimethylaminoethanol, and the other covalently coupled to peripheral membrane protein. The Study of Women's Health Across the Nation reporting range for the urinary LH assay is 0.1 to 30.7 mIU/mL (actual assay range = 0.1–53.1 mIU/mL). The LH2 MCM is standardized against the World Health Organization's Second International Reference Preparation 80/552 (http://www.who.int/bloodproducts/catalogue/EndoMay2011.pdf). We report the interassay CV = 10.67% and the intraassay CV = 4.78%. Creatinine analysis uses a spectrophotometric assay. The assay is standardized against creatinine obtained from the Sigma Chemical Company. The reporting range is 0.05 to 1.15 mg/mL (assay range = 0.05–1.4 mg/mL). The interassay CV = 11.4% and the intraassay CV = 4.3%. Urinary assays for E1c and PdG correlate well with estradiol and progesterone (serum estradiol with E1c, $r = 0.93$; progesterone with PdG, $r = 0.98$).[40]

Data Analysis

We evaluated distributions via frequencies and univariate statistics for normality and outliers, and 3 of 474 observations (0.6%) were excluded from analysis because of unrealistically high creatinine-adjusted E1c concentrations. Body mass, BMI, E1c, and PdG were log transformed before analysis to satisfy normality assumptions, and all urinary hormone concentrations were creatinine adjusted before log transformation. We tabulated frequencies and, where relevant, tested for differences via log-likelihood χ^2. Penalized B-splines[41] were used to characterize changes in creatinine-adjusted E1c and PdG across the observation period, and binary logistic regression models[42] were used to relate the odds of cycle duration given athlete or nonathlete status or tertile of physical activity. All analyses and data management were conducted in SAS (version 9.2; SAS Institute Inc, Cary, NC).

Change in Urinary Hormones Over Time

Because many participants in both groups had multiple cycles of different lengths, the behavior of creatinine-adjusted E1c and PdG was restricted to cycle lengths between 24 and 35 days. Cycles longer than 35 days (15 of 146 [10.2%]) and those shorter than 24 days (16 of 146 [10.9%]) were excluded from analysis because of their small cell size and atypical menstrual pattern. Cycles shorter than 24 days were included in the longitudinal plots of the hormone data, as indicated in the subtitle of the spline plots. The cycle durations between 24 and 35 days were characterized by athlete or nonathlete status and tertiles of physical activity.

RESULTS

Adolescent girls enrolled in the study represented the local community based on age, race, socioeconomic status, and geographic location. The age (mean ± SD) of the athlete group was 15.7 ± 1.2 years (95% confidence interval [CI] = 13.4, 18.1). The age of the nonathlete group was 16.3 ± 1.2 years (95% CI = 13.9, 18.6).

Of the 106 high school students enrolled in the study, 4 (3.8%) dropped out before completing the study, and 4 (3.8%) were excluded because of invalid questionnaire data. The remaining 98 participants (92.4%) completed the sample-collection protocol. All 98 participants (100%) provided 13 of the 15 required specimens (87%) (including 3 samples the first week to establish baseline). Seventy-four of the 98 participants (75.5%) classified themselves as athletes and 24 (24.5%) classified themselves as nonathletes. Of interest were the various activity levels of those who classified themselves as athletes, some of whom were not very active. This made the physical activity tertiles more relevant in the analysis. The observation time ranged from 77 to 92 days and averaged 84.9 days, with no difference according to athlete or nonathlete status.

Body Composition

Measures of BMI, body mass, and fat remained stable for athletes and nonathletes throughout the study (Table 1). The preobservation BMI measures (mean ± SD) were 23.0 ± 3.4 kg/m^2 and 21.7 ± 3.1 kg/m^2 for the nonathletes and athletes, respectively (Table 1). After the urine-collection period, BMI measures were 23.1 ± 3.4 kg/m^2 and 21.9 ± 3.1 kg/m^2 for nonathletes and athletes, respectively. Neither preobservation nor postobservation BMI levels were different between athletes and nonathletes. Among athletes, BIA fat mass at the precollection and postcollection time points were 17.0 ± 5.8 kg and 17.7 ± 5.5 kg, respectively. Although BIA fat mass was, on average, 13.5% to 14.5% higher in nonathletes, the difference was not significant.

Table 1. High School Athletes and Nonathletes: Body Composition Before and After Sample Collection

Body Composition	Group, Mean ± SD		*P* Value
	Athletes (n = 74)	Nonathletes (n = 24)	
Height, cm	165.3 ± 7.4	166.9 ± 7.4	NS
Body mass, kg			
At start	59.2 ± 10.2	63.9 ± 11.1	NS
At end	59.8 ± 10.1	64.4 ± 10.9	NS
Body mass index, kg/m^2			
At start	21.7 ± 3.1	23.0 ± 3.4	NS
At end	21.9 ± 3.1	23.1 ± 3.4	NS
Bioelectrical impedance analysis, fat, kg			
At start	17.0 ± 5.8	19.9 ± 6.8	.054
At end	17.7 ± 5.5	20.5 ± 6.3	.051

Abbreviation: NS, not significant.

Menstrual Cycle Patterns

Seventeen participants (14 athletes [82%] and 3 nonathletes [18%]) had no complete menstrual cycles within the observation period; for each of those girls, only 1 menstrual start date was reported. Among the 81 remaining participants (83%), 146 completed cycles were observed and classified into 4 groups according to duration between consecutive menses start dates: less than 24 days (n = 17, 11.6%), 24 to 35 days (n = 114, 78.1%), 36 to 39 days (n = 3, 1.4%), and longer than 39 days (n = 12, 8.2%) within the observation period (Table 2). Among participants with complete menstrual cycles, athletes and nonathletes did not differ in relative frequency of having 1, 2, or 3 observed cycles (Table 3).

Urinary Hormone Values

Athletes Versus Nonathletes. In cycle durations shorter than 24 days or longer than 35 days, mean log(E1c/Cr) concentrations were not different in nonathletes compared with athletes (Figure 2). Although nonathletes' mean log(PdG/Cr) concentrations were less in the first half and greater in the second half of the cycle, the difference was not significant. Too few cycles of more than 35 days' duration in the nonathlete participants (6 of 35, 17.1%) prevented meaningful comparison of their log(E1c/Cr) and log(PdG/Cr) concentrations with those of athletes.

In binary logistic regression, when we compared cycle-length category by athlete or nonathlete status, we found that cycles shorter than 24 days were no more likely in athletes than they were in nonathletes (odds ratio [OR] = 1.49, 95% CI = 0.40, 5.56).

Table 2. Number of Complete Menstrual Cycles by Cycle Length

Participants, No. of Cycle(s)	No. (%) of Cycles by Length		
	<24 d	24–35 d	>35 d
Athletes (n = 74, 111 total cycles)			
1	1 (0.9)	13 (11.7)	6 (5.4)
2	4 (3.5)	51 (45.9)	3 (2.7)
3	8 (7.2)	25 (22.5)	0 (0.0)
Nonathletes (n = 24, 35 total cycles)			
1	0 (0.0)	2 (5.0)	5 (14.0)
2	1 (3.0)	24 (67.0)	1 (3.0)
3	2 (5.0)	1 (3.0)	0 (0.0)

Table 3. Number of Complete Menstrual Cycles[a]

Cycle(s)	Participants, No. (%)
Athletes, n = 74	
0[b]	14 (19)
1	20 (27)
2	29 (39)
3	11 (15)
Nonathletes, n = 24	
0[b]	3 (12)
1	7 (29)
2	13 (54)
3	1 (4)

[a] *P* value for group × cycle comparison was not significant.
[b] No complete cycle was observed.

Tertiles of Physical Activity. The log(E1c/Cr) mean concentrations did not differ by tertile of activity (Figure 3). Although participants in the least-active tertile had slightly

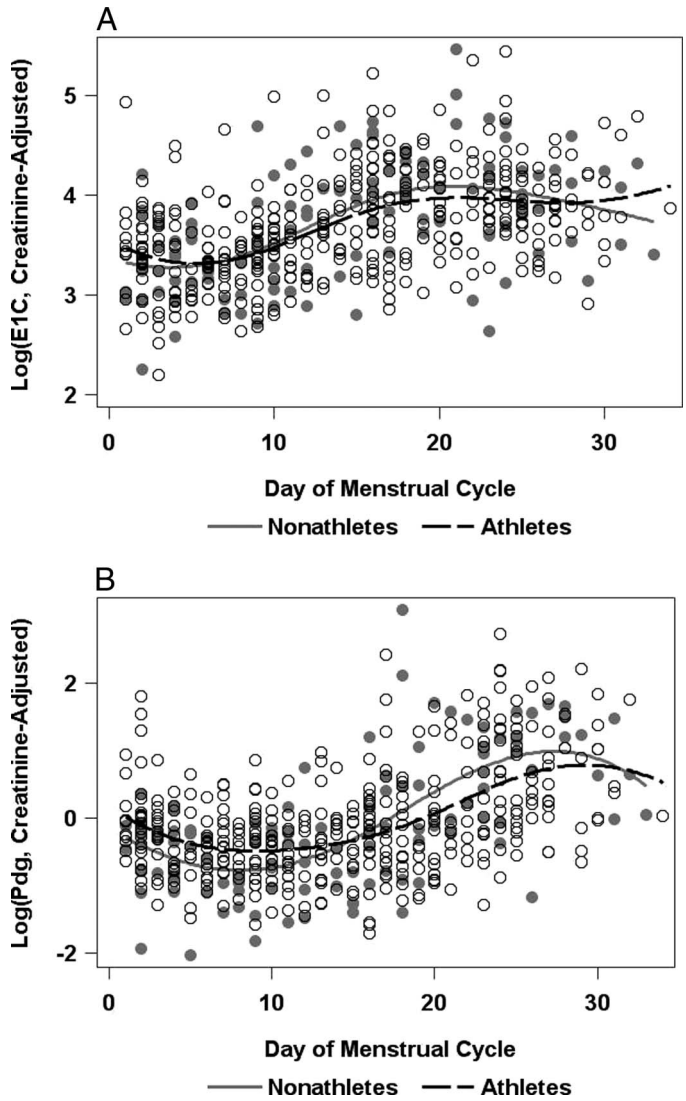

Figure 2. Concentrations of A, log(urinary estrone-conjugate [E1C]), and B, log(urinary pregnanediol-glucuronide [PdG]) by day of cycle, for athletes and nonathletes. Clear circles represent athletes; filled circles, nonathletes.

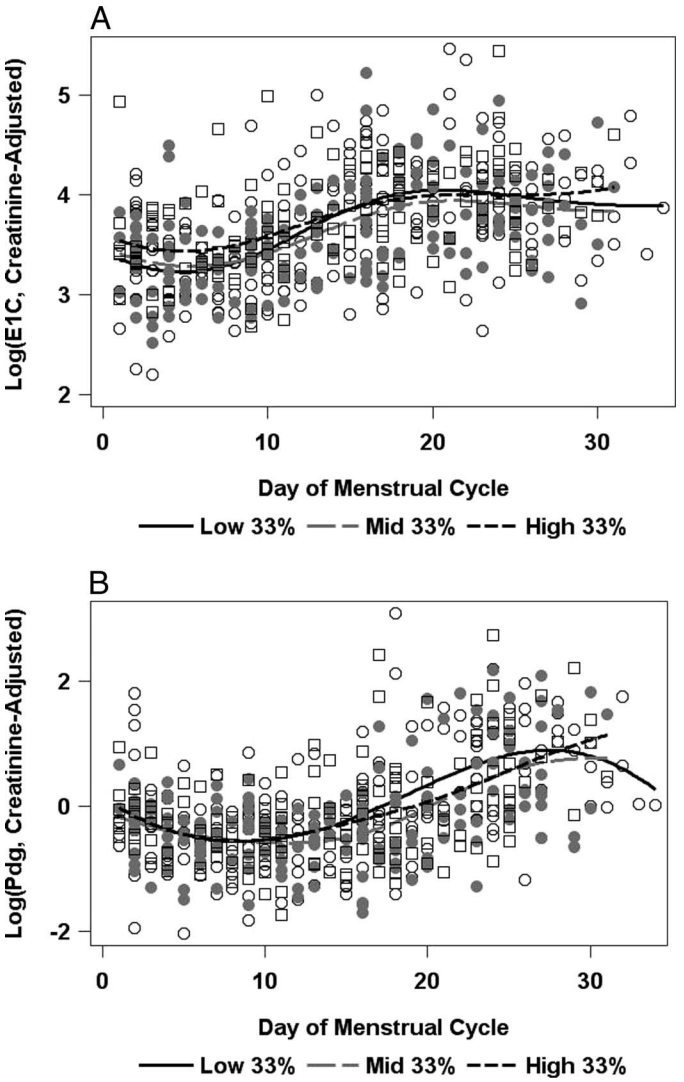

Figure 3. Concentrations of A, log(urinary estrone-conjugate [E1C]), and B, log(urinary pregnanediol-glucuronide [PdG]) by day of cycle, according to tertiles of physical activity. Open circles represent low 33%; filled circles, mid 33%; open squares, high 33%.

higher concentrations of log(PdG/Cr) at midcycle, the difference was not significant (Figure 3).

When cycle-length categories were compared by tertiles of physical activity, participants in the highest-activity tertile were no more likely (OR = 0.75, 95% CI = 0.12, 2.38) to have cycles shorter than 24 days, compared with participants in the middle tertile. Women in the lowest-activity tertile were not less likely to have cycles shorter than 24 days (OR = 0.44, 95% CI = 0.12, 1.58) relative to participants in the middle tertile.

DISCUSSION

Physical activity is an important component of health maintenance programs. However, as with most good things, too much can produce negative consequences. The threshold for turning physical activity into a negative factor in teenage female athletes is unknown, although the female athlete triad suggests that risks occur along a continuum, with energy availability being a key component of that risk. Our study was designed to examine the effects of physical activity on the hormone concentrations of teenage female athletes.

Choosing inactive controls allowed us to consider whether menstrual dysfunction occurred on a continuum and whether the athlete represented the extreme end of the continuum. The results presented here suggest that the activity levels of these female athletes did not appear to alter their concentrations of estrogen and progesterone compared with the nonathlete controls. The concern that led to the development of these hypotheses was that the physical activity level of teenage female athletes was altering their hormonal balance in a way that made them more susceptible to anterior cruciate ligament (ACL) injury. In a recent, 5-year study of 143 female collegiate athletes, Dragoo et al[43] demonstrated a relationship between circulating relaxin, a collagenolytic hormone that participates in the remodeling process of the ACL, and the risk of ACL injury. When relaxin was detectable in the serum, 14 of 46 athletes (30%) tore their ACLs during their collegiate career; however, little is known about the cyclicity of relaxin, estrogen, and progesterone in normally menstruating women. From our data, the estrogen and progesterone profiles of the athlete group for the 3-month monitoring period appeared very similar to those of the inactive (nonathlete) control group (Figure 2) and would not seem to increase injury risk.

Seventeen females (14 athletes [82%] and 3 nonathletes [18%]) in the study did not have complete menstrual cycles during the 3-month observation period and were probably anovulatory. Of the 17 individuals, 2 (12%), 4 (24%), 3 (18%), 6 (35%), and 2 (12%) were 14, 15, 16, 17, and 18 years old, respectively. Although that finding may be explained by their young ages and the data suggest that the very long cycles were anovulatory, we cannot say because the data were gathered once per week. Anovulatory cycles may simply reflect the increased likelihood of anovulation and oligomenorrhea in the perimenarcheal period,[9] yet exercise-induced amenorrhea and menstrual irregularities are also a signal of energy imbalance consistent with the female athletic triad and a consequent risk for loss of bone density.[44] Menstrual irregularities have also been associated with increased risk of musculoskeletal injuries and stress fractures in athletes.[45,46]

The percentages of those participants with no complete cycle appeared to be similar in athletes (14 of 74, 19%) and nonathletes (3 of 24, 12%), ($P = .47$). Those percentages are much higher than the 4.75% of anovulatory athletic women recently reported by Dragoo et al.[43] Of similar concern are the 29 participants (30%) with cycles shorter than 24 days or longer than 35 days. Cycles longer than 35 days are more likely to cause concern because high physical activity levels or poor nutritional states[5] (or both) can lengthen the cycle before the cycle is interrupted completely.

As for the structural makeup of these teenage girls, hypothesis 2 was disproven: athletic activity did not alter their body composition throughout the course of the study. However, not all participants began the study in the preseason; therefore, it is possible that an initial change in body composition was missed by the monitoring period. The study time (12 weeks) was short and may not have been sufficient to detect a significant change.

Additionally, had we controlled for hydration status at the time of the body-composition testing, small changes in

body composition might have been detected. Unfortunately, hydration status was not controlled. As expected, the athletes were leaner than the controls: fat mass measured by BIA was 13.5% to 14.5% higher in the nonathletes.

Traditionally, field measures of body composition have been limited to measurements such as BMI, which depends only on body-mass–adjusted height. To overcome the BMI's simplistic assumption about the distribution of lean and fat mass, bioelectrical impedance was measured by a 4-terminal impedance plethysmograph. We chose BIA because of its accuracy[36] and because the method does not require minor participants (14–18 years old) to be exposed to radiation.

Symons et al[26] determined the degree of association between body-composition measures (Quetelet Index, lean body mass, and fat body mass) and menstrual cycle length by analyzing diaries from 436 women (aged 24–45 years) encompassing 4392 menstrual cycles. Mean cycle length, variability of cycle length, and mean bleeding length were calculated from those diaries. Body-composition measures (Quetelet Index or BMI [kg/m^2], lean body mass, and fat body mass [kg]) were obtained at annual clinic visits. Using a mixed-model analysis, they[26] found a significant positive association with cycle length for each body-composition measure. The relationship between each body-composition measure and cycle length was nonlinear, with the longest mean cycle lengths occurring in participants with greater BMI, lean body mass, or fat body mass. Longer cycle length was also noted in participants at the lowest levels of BMI and fat body mass. Therefore, when examining cycle length, BMI should be considered.

This study had several limitations. The 74 athletically active participants who completed this study were involved in 12 sports. In addition, not all athletically active participants began the study before the start of their competitive season. Although this was our initial intent, because of year-round conditioning and sports participation and involvement on multiple teams and sports, that proved impractical. Another likely possibility is that some participants overestimated their activity levels. The body-composition measurements were performed without determination of hydration status. No attempt was made to control or monitor the hydration status of the participants. We relied on the BIA device to measure body water. Ensuring adequate hydration might have generated better data on body composition. Also, we did not attempt to measure serum or urine relaxin concentrations. Serum relaxin concentrations have recently been shown to predict ACL injuries in collegiate female athletes.[6] Lastly, we did not collect data on the onset of menarche or the consistency of menses thereafter. Those data could have improved the generalizability of these findings.

CONCLUSIONS

Moderate physical activity and sports participation did not appear to affect the concentrations of circulating endogenous estrogen and progesterone in 14- to 18-year-old adolescent girls. However, none of the participants reached very high levels of physical activity. The high rate of anovulation (17%: 14 athletes, 3 nonathletes) is a cause for concern; the cause in this group is unknown.

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