Effects of menstrual cycle phase on athletic performance

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ABSTRACT

LEBRUN, C. M., D. C. MCKENZIE, J. C. PRIOR, and J. E. TAUNTON. Effects of menstrual cycle phase on athletic performance. Med. Sci. Sports Exerc., Vol. 27, No. 3, pp. 437–444, 1995. The purpose of this study was to examine the effects of menstrual cycle phase on four selected indices of athletic performance: aerobic capacity, anaerobic capacity, isokinetic strength, and high intensity endurance. Sixteen eumenorrheic women (VO2max > 50 mL·kg⁻¹·min⁻¹) were tested during the early follicular (F) and midluteal (L) phases of the menstrual cycle. Cycle phases were confirmed by serum estradiol and progesterone assays. No significant differences were observed between F and L tests in weight, percent body fat, sum of skinfolds, hemoglobin concentration, hematocrit, maximum heart rate, maximum minute ventilation, maximum respiratory exchange ratio, anaerobic performance, endurance time to fatigue (at 90% of VO2max), or isokinetic strength of knee flexion and extension. Both absolute and relative VO2max, however, were slightly lower in L than in F (F = 3.19 ± 0.09·min⁻¹·L = 3.13 ± 0.08·min⁻¹, F = 0.04; and F = 53.7 ± 0.9 mL·kg⁻¹·min⁻¹, L = 52.8 ± 0.8 mL·kg⁻¹·min⁻¹, P = 0.06). These results suggest that the cyclic increases in endogenous female steroid hormones of an ovulatory menstrual cycle may have a slight, deleterious influence on aerobic capacity, with potential implications for individual athletes. Nevertheless, the cycle phase did not impact significantly on the majority of the other performance tests and cardiorespiratory variables measured in this study.

ESTRADIOL, PROGESTERONE, FOLLICULAR, LUTEAL,
VO2MAX, AEROBIC ENDURANCE, ANAEROBIC CAPACITY,
ISOKINETIC STRENGTH, PERCENT BODY FAT

The 20th century has brought ever-increasing numbers of athletic women into the competitive arena. With this has come a concomitant awareness of health issues specific to active women. A topic of relatively recent interest has been the potential effects of menstrual cycle phase and the associated variations in the female steroid hormones on athletic performance.

The first studies were largely anecdotal or retrospective surveys. Subsequently, attempts have been made to quantify performance variations using various treadmill and cycle ergometer protocols. Discrepancies in the timing of the testing, and inadequate documentation of cycle phase make these early results difficult to interpret with any validity, as detailed elsewhere (24).

A “regular” menstrual cycle (i.e., 28 d long) does not necessarily imply the presence of either ovulation or a normal luteal phase (35). Basal body temperature (BBT) patterns can be used to estimate cycle phases. A midcycle elevation of 0.2°–0.3°C reflects the thermogenic action of increased progesterone levels and strongly suggests that ovulation has occurred (41). Limitations to this method have been described (2), but accurate recording techniques and computer analysis of temperature graphs can greatly increase reliability (36). Nevertheless, hormonal levels are still the most valid criteria for absolute documentation of cycle phase (22). Most studies utilizing serum progesterone for confirmation have not found any significant menstrual cycle effects on either maximal or submaximal cardiorespiratory exercise responses (8,10,32,38), with the exception of a possible luteal phase enhancement of endurance time (18,28). One group (38) demonstrated changes in nonathletes only, suggesting that some effects on exercise performance can be overcome voluntarily and with training. In contrast, others have shown an increased oxygen consumption and decreased net efficiency for exercise of shorter duration (16), as well as a greater degree of circulatory strain (16,32,38) during the luteal phase, possibly due to the progesterone-induced increase in core body temperature and metabolic rate.

Subsequent work has focused on various aspects of substrate metabolism (3,21,23). Estradiol enhances glycogen uptake and storage in both liver and muscle, in animals (1,20,27) and in humans (13,28). Other metabolic actions of estradiol with the potential to improve endurance performance include an increase in lipid availability and utilization, and enhanced gluconeogenesis (for review see 4).
Researchers have also examined the influence of cycle phase on resting and exercise ventilation. Progesterone acts to stimulate respiration during the luteal phase (11), and during pregnancy. Increases in both hypoxic and hypercapnic respiratory drives (10,38) as well as in minute ventilation (11,16,18,38) also occur during the luteal phase. Nevertheless, there have been no associated alterations in VO_{2max} or endurance performance. Cyclic hormonally mediated fluctuations in plasma volume and hemoglobin concentration (10,18), and in body temperature (15,16) have also been documented, but without any corresponding impact on performance.

Little accurate information exists with regard to muscle strength and the menstrual cycle. Some investigators have measured detrimental effects during the luteal phase (31,42), potentially in response to the increase in deep muscle temperature (31), while others (9,37) have failed to demonstrate any meaningful changes across the menstrual cycle.

While some of these investigations have used moderately trained women, relatively few (8,38) have studied more highly trained athletes, who are the individuals most likely to be affected by subtle changes in performance. The complex metabolic actions of the female steroid hormones may alter various components of sports performance in different ways during the course of an ovulatory menstrual cycle. This study was therefore undertaken to test this hypothesis in a group of trained female athletes during the follicular and luteal phases, and to control for many of the variables that may have influenced the outcome of previous studies. Four common physiological measures were utilized: aerobic capacity (maximum oxygen consumption or VO_{2max}), anaerobic capacity, high intensity endurance (at 90% of VO_{2max}), and isokinetic strength. The purpose was to examine the effect of the endogenous fluctuations in steroid hormone concentrations on these tests of performance.

**METHODS AND MATERIALS**

**Subjects**

Female subjects between the ages of 18 and 40 were recruited by means of advertisement and word of mouth. Ethical approval was obtained from the Committee on Human Experimentation of the University of British Columbia, and all subjects signed a written informed consent. All of the women by history were having regular menstrual cycles (24–35 d in length) and had not taken oral contraceptives for at least 6 months before entering the study. Supportive evidence for ovulatory cycles was initially obtained by a menstrual history questionnaire determining the existence of symptoms such as breast tenderness, fluid retention, appetite change, and mood swings in the 1–2 wk preceding a normal menstrual flow. The presence of such symptoms has been shown to be an indicator of the luteal phase elevations in estrogen and progesterone (33).

All subjects were participating in some type of intensive aerobic activity on a regular basis. To more accurately document small differences in performance attributable to the experimental conditions, the population studied was limited to “trained” female athletes, defined in this study by an entrance VO_{2max} equal to or greater than 50 ml·kg^{-1}·min^{-1}. Volunteers were recruited from a variety of sports including running, cycling, triathlon, squash, cross-country skiing, ultimate Frisbee, and rowing. The level of training activities was screened at the time of entrance into the study, and fitness levels were confirmed at the time of the first testing.

A questionnaire was also administered to determine the general health of the subjects. Subjects were excluded if they were smokers, if they had any significant past medical history, or if they were taking any medication that might interfere with the exercise testing. Those who were on vitamin supplements or iron therapy were asked to maintain the exact dosage throughout the entire length of the study.

Subjects were required to maintain a steady-state level of aerobic training throughout the experimental period. They kept a daily training log, as well as a record of their basal body temperature, menstrual and ovulatory symptoms, resting heart rate, weight, and subjective sensations of performance. Basal body temperature was taken orally, before arising at the same time of day, and recorded on a standard form, with concurrent comments in a separate column. The intensity and amount of training were reviewed to ensure that there was no substantial training stimulus over the duration of the study.

Physiological testing was carried out in the early follicular phase (between days 3 and 8 of a normal cycle), and in the midluteal phase (between days 4 and 9 after “ovulation”). This was initially determined by a sustained rise in basal body temperature of 0.2°–0.3°C (41), and later confirmed by serum hormone measurements. By BBT charting, not all cycles appeared to be “ovulatory” in all subjects. Subjects who did not demonstrate a rise in BBT to suggest ovulation following the initial follicular phase testing were followed through the next cycle and then tested after “ovulation.” If the BBT chart still did not show a biphasic pattern during this second cycle, then the follicular phase testing was repeated again before testing during the subsequent luteal phase. The order of menstrual phase in which testing was performed was random, depending upon when subjects were enrolled in the study. Estimated cycle phases were subsequently validated by measurement of serum ovarian hormones. A serum progesterone level at rest greater than 16 nmol·L^{-1} was required for absolute confirmation of the luteal phase (22).
EXPERIMENTAL PROTOCOL

The experimental protocol was carried out on two successive days and was identical for each test period. Subjects were asked to refrain from any vigorous exercise during the previous 24 h and to report to the laboratory in a fasted, rested state. The height and weight of each subject were measured. On the first day, venous blood samples were taken before commencement of exercise, and subsequently analyzed for estradiol, progesterone, and a complete blood count. Methods for each procedure are subsequently described.

AEROBIC CAPACITY

On the first day, measurement of VO_{2max} was carried out on a Quinton 24-72 treadmill, following a 5- to 10-min warm-up at a speed between 2.2 m·s^{-1} and 2.7 m·s^{-1}. The protocol utilized a continuous progressive workload on a level grade, beginning at a speed of 2.2 m·s^{-1}, and increasing by 0.22 m·s^{-1} each minute until fatigue, as previously described (29). Heart rate was monitored with either an ECG tracing on a Burdick EK/SA electrocardiograph, or a Polar Vantage heart rate monitor; and was recorded at 45 s into each stage. Expired gases were continuously sampled and analyzed utilizing a Beckman Metabolic Measurement Cart (OM-11 oxygen analyzer and LB-2 carbon dioxide analyzer), and tabulated by a data acquisition system (Hewlett-Packard 3052A) that determined respiratory gas exchange variables every 15 s. Calibration of the volume transducer was performed utilizing a 1.0-L syringe, and both gas analyzers were calibrated with standardized calibration gases and room air before each test.

A maximal test was defined by achievement of at least two of the following three criteria: a plateau or decrease in O_{2} despite an increase in workload, a respiratory exchange ratio (RER) greater than or equal to 1.1, or attainment of at least 90% of predicted maximum heart rate. If a subject did not complete a satisfactory maximal test, the testing procedure was repeated, following a short rest (to return cardiovascular and respiratory variables to baseline), but starting at a velocity of 3.08 m·s^{-1}. The raw data were subsequently individually analyzed by two of the investigators. The maximal values for VO_{2}, VE, and RER were taken as the average of the four highest consecutive readings, discarding any outlier values.

ANAEROBIC PERFORMANCE

High intensity running performance was assessed by the anaerobic speed test (AST) of Cunningham and Faulkner (7) employing time in seconds to fatigue as the performance index. (This test was chosen for its sport specificity in these athletes, who were primarily trained as runners.) Subjects rested for at least 1.5 h following the VO_{2max} test before measurement of their anaerobic performance. Following an adequate warmup, subjects performed the treadmill run at 8 mph (3.52 m·s^{-1}) at a 20% incline until fatigue (defined as an inability of the subject to continue at the set treadmill speed). Subjects were aware of the elapsed time. The test-retest reliability of this test has been documented as r = 0.76–0.91 (25).

ENDURANCE PERFORMANCE

On the second day of testing, high intensity endurance capacity was assessed as running time in seconds to fatigue at a treadmill load calculated to require approximately 90% of maximal oxygen uptake. This definition of endurance performance has been utilized by previous investigators (17). The workload was determined by taking 90% of the treadmill speed at which the subject completed their last complete minute of running before stopping the VO_{2max} test. Once set, this workload velocity remained constant for the next two testing sessions, regardless of any subsequent variations in the actual VO_{2max} measurement.

ISOKINETIC STRENGTH

Isokinetic strength was measured as peak torque in newton-meters (N·m) generated by knee flexion and extension on a Cybex II isokinetic dynamometer at a velocity of 30°·s^{-1}. The subjects were positioned on the Cybex table so that the lateral femoral condyle was aligned with the axis of rotation of the isokinetic dynamometer. Subjects were secured to the backrest by a seat belt at the waist, and the leg to be tested was stabilized with a strap above the knee at midthigh. After a short warmup at a velocity of 240°·s^{-1}, maximal knee flexion and extension were measured. The best values of three different attempts with each leg were taken. The coefficient of variation of this test performed at this velocity has been reported as 5.9% (25).

BODY COMPOSITION

Anthropometric measurements included height and weight (Detecto industrial scale), measurement of skinfold thickness at six different sites (biceps, triceps, subscapular, suprailiac, anterior thigh, and medial calf) with a Harpenden skinfold caliper (John Bull, British Indicators Ltd.), and underwater densitometry using a hydrostatic weighing tank. Skinfold measurements were reported as the sum of all values. Percentage of body fat was calculated by the method of underwater densitometry using the Siri formula (39).
BLOOD SAMPLES

Venous blood samples were taken before any warm-up exercise and were kept cool (in cold water) until completion of the VO\textsubscript{2max} and AST testing (a maximum of 2 h). One tube was then immediately taken to the Laboratory at the University Hospital, U.B.C. Site, for determination of an automated blood count (Coulter S + STKR). (Variables analyzed were hemoglobin concentration, hematocrit, and mean cell volume.) The remaining blood was spun in a refrigerated centrifuge (Dammon/IEC Clinical) for 10 min at 3000 rpm. The plasma was stored in separate aliquots in Venoject plain silicone-coated glass tubes at \(-20^\circ\text{C}\) until it was subsequently analyzed (approximately 3 months later) in the Endocrine Laboratory at the Vancouver General Hospital, using commercially available no-extraction solid-phase \textsuperscript{125}I radioimmunoassays (Coat-A-Count Estradiol and Coat-A-Count Progesterone, Diagnostic Products Corporation). Over the duration of the study, blood samples were coded and analyzed in three separate batches by an independent observer. Both samples from each subject were analyzed together. The intra-assay coefficients of variation (CV) were 10.6% for estradiol and 10.3% for progesterone. Interassay CVs ranged from 4.2% to 8.1% for estradiol and from 7.2% to 10.0% for progesterone (Diagnostic Products Corporation). The sensitivities of these assays are 2.9 pmol·l\textsuperscript{-1} for estradiol and 0.16 nmol·l\textsuperscript{-1} for progesterone.

STATISTICAL ANALYSIS

Before beginning the study, power calculations were performed using predicted means and standard deviations for each test. It was determined that with \(\alpha = 0.05\) and power = 90%, an \(n\) of 16 was sufficient to detect physiologically important variations. The data from the two menstrual cycle tests were analyzed using paired Student’s \(t\)-tests for dependent means. The dependent variables were maximum aerobic consumption (VO\textsubscript{2max}), anaerobic capacity (AST), high intensity endurance (running time to exhaustion at 90% of VO\textsubscript{2max}), and isokinetic strength (Cybex II measurement of peak torque of knee flexion and extension). Cardiorespiratory variables included maximal heart rate, maximum minute ventilation, and maximal respiratory exchange ratio. Anthropometric measurements of weight, sum of skinfolds, and percentage body fat were also analyzed for changes between cycle phases, as were the results for estradiol, progesterone, hemoglobin concentration, and hematocrit.

The level of significance was set at \(P < 0.05\). The statistical package utilized was Systat version 5.01. All values are expressed as means ± SE.

RESULTS

A total of 16 subjects were determined to have ovulated on the basis of luteal phase progesterone measurements greater than 16 nmol·l\textsuperscript{-1} (22). The mean cycle length was 28.3 ± 0.4 d. Testing took place during the early follicular phase (day = 5.7 ± 0.5) and the midluteal phase (day = 23.3 ± 0.9) as determined by BBT charts.

Initial characteristics for all subjects during the follicular phase, and plasma estradiol and progesterone levels during both tests are presented in Table 1. The results of body composition measurements, blood tests, and exercise performance tests are presented in Tables 2 and 3.

BODY COMPOSITION

There were no significant differences between follicular and luteal phases of the cycle in the same subject in weight (\(P = 0.92\)), percent body fat (\(P = 0.80\)), or sum of skinfolds (\(P = 0.61\)).
TABLE 2. Effect of menstrual cycle phase on anthropometric, hormonal and hematological variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Follicular</th>
<th>Luteal</th>
<th>Paired t-Test (df = 15)</th>
<th>Mean Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>Weight (kg)</td>
<td>59.5 ± 1.7</td>
<td>59.5 ± 1.8</td>
<td>P = 0.92</td>
<td>-0.02 ± 0.24</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>17.4 ± 1.2</td>
<td>17.3 ± 0.9</td>
<td>P = 0.89</td>
<td>+0.11 ± 0.44</td>
</tr>
<tr>
<td>Sum of skinfolds</td>
<td>75.2 ± 3.9</td>
<td>76.1 ± 3.8</td>
<td>P = 0.51</td>
<td>-0.88 ± 1.71</td>
</tr>
<tr>
<td>Estradiol (pmol·L⁻¹)</td>
<td>141.4 ± 15.8</td>
<td>461.4 ± 36.9</td>
<td>P &lt; 0.0001</td>
<td>-319.94 ± 37.29*</td>
</tr>
<tr>
<td>Progestrone (nmol·L⁻¹)</td>
<td>1.22 ± 0.09</td>
<td>40.6 ± 3.7</td>
<td>P &lt; 0.0001</td>
<td>-39.38 ± 3.73*</td>
</tr>
<tr>
<td>Hemoglobin (g·L⁻¹)</td>
<td>131.4 ± 1.5</td>
<td>133.2 ± 1.2</td>
<td>P = 0.11</td>
<td>-1.87 ± 1.05</td>
</tr>
<tr>
<td>Hematocrit (%)</td>
<td>38.5 ± 0.4</td>
<td>39.2 ± 0.3</td>
<td>P = 0.07</td>
<td>-0.69 ± 0.35†</td>
</tr>
<tr>
<td>MCV (fl)</td>
<td>91.8 ± 0.9</td>
<td>91.8 ± 0.9</td>
<td>P = 1.00</td>
<td>0.00 ± 0.35</td>
</tr>
</tbody>
</table>

Values are means ± SE (N = 16).
Sum of skinfolds = total skinfold; MCV, mean cell volume.
*P < 0.05; †P < 0.10.
No other significant differences observed.

TABLE 3. Effect of menstrual cycle phase on performance variables.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Follicular</th>
<th>Luteal</th>
<th>Paired t-Test (df = 15)</th>
<th>Mean Difference</th>
</tr>
</thead>
<tbody>
<tr>
<td>VO₂max (L·min⁻¹)</td>
<td>3.19 ± 0.08</td>
<td>3.13 ± 0.08</td>
<td>P = 0.04</td>
<td>+0.06 ± 0.03*</td>
</tr>
<tr>
<td>V̇E (mL·kg⁻¹·min⁻¹)</td>
<td>53.7 ± 0.9</td>
<td>52.6 ± 0.8</td>
<td>P = 0.08</td>
<td>+0.93 ± 0.46†</td>
</tr>
<tr>
<td>HR(max) (bpm)</td>
<td>189.4 ± 2.3</td>
<td>186.5 ± 2.6</td>
<td>P = 0.92</td>
<td>-0.13 ± 1.28</td>
</tr>
<tr>
<td>RER(max)</td>
<td>1.17 ± 0.01</td>
<td>1.15 ± 0.01</td>
<td>P = 0.56</td>
<td>+0.01 ± 0.01</td>
</tr>
<tr>
<td>V̇E(max)</td>
<td>164.4 ± 2.3</td>
<td>163.8 ± 2.4</td>
<td>P = 0.32</td>
<td>-0.89 ± 0.88</td>
</tr>
<tr>
<td>(L·min⁻¹·BTTPS)</td>
<td>80.8 ± 3.2</td>
<td>78.4 ± 2.3</td>
<td>P = 0.27</td>
<td>-2.69 ± 2.37</td>
</tr>
<tr>
<td>Endurance (s)</td>
<td>723.8 ± 2.6</td>
<td>769.3 ± 1.4</td>
<td>P = 0.72</td>
<td>15.50 ± 4.44</td>
</tr>
<tr>
<td>R Quadriceps</td>
<td>143.9 ± 2.3</td>
<td>142.5 ± 2.6</td>
<td>P = 0.78</td>
<td>+1.44 ± 5.09</td>
</tr>
<tr>
<td>(N·m)</td>
<td>80.8 ± 4.4</td>
<td>83.3 ± 4.9</td>
<td>P = 0.27</td>
<td>-2.69 ± 2.37</td>
</tr>
<tr>
<td>L Quadriceps</td>
<td>141.9 ± 8.4</td>
<td>141.8 ± 7.2</td>
<td>P = 0.98</td>
<td>+0.13 ± 5.94</td>
</tr>
<tr>
<td>(N·m)</td>
<td>82.5 ± 6.6</td>
<td>83.8 ± 6.4</td>
<td>P = 0.66</td>
<td>-1.19 ± 2.62</td>
</tr>
</tbody>
</table>

Values are means ± SE (N = 16).
VO₂max = maximum oxygen consumption; V̇E(max) = maximum recorded minute ventilation; HR(max) = maximum heart rate; RER(max) = maximum respiratory exchange ratio; AST = anaerobic speed test; Endurance = at 90% VO₂max; Ṙ = right; L̇ = left (measurements of muscle strength are peak torque, measured at 30°·s⁻¹, best of three trials).
*P < 0.05; †P < 0.10.
No other significant differences were observed.

BLOOD AND SERUM RESULTS

The levels of both estradiol and progesterone were significantly different (P < 0.0001 for both hormones) between phases, as would be expected. Mean red cell volume was similar in both tests (P = 1.00). Hemoglobin and hematocrit levels in the follicular and luteal phases could not be distinguished (P = 0.11 and P = 0.07, respectively).

EXERCISE PERFORMANCE

Analysis of the exercise performance results are presented in Table 3. A slightly higher absolute VO₂max (Δ = 0.06 ± 0.03·min⁻¹, P = 0.04) was documented during the follicular as compared to the luteal phase, but a higher relative VO₂max was not detected (Δ = 0.93 ± 0.46 ml·kg⁻¹·min⁻¹, P = 0.06). These values are represented graphically in Figure 1 for all subjects. In this group of 16 athletes with ovulatory cycles, absolute VO₂max was lower during the luteal phase in 11 of 16 subjects, while relative VO₂max was lower in 10 of 16. There was a large degree of intersubject variability in this response. Exclusion of the subject with the largest phase difference in aerobic capacity reduced the achieved significant levels for absolute and relative VO₂max to P = 0.06 and P = 0.12, respectively. None of the other statistical analyses, including weight, were affected by this exclusion.

There were no significant alterations in maximum V̇E (highest recorded minute ventilation), maximal heart rate, or maximum RER (respiratory exchange ratio) attributable to phase of the cycle. The remainder of the tests of performance: the anaerobic speed test (AST), the endurance run at 90% VO₂max, and the Cybex II measurements of isokinetic strength of quadriceps and hamstrings were also not influenced by the menstrual cycle phase.

DISCUSSION

In contrast to the majority of investigations to date, in this group of 16 athletes with ovulatory cycles, both absolute and relative VO₂max appeared to be slightly lower during the luteal phase of the menstrual cycle, (P = 0.04 and 0.06, respectively). Given the limitations of the statistical analysis, and the ranges of equipment error in measurement, this change in aerobic capacity is of borderline statistical significance. Because of the multiple analyses performed, there is an increased risk of Type I error. It should be noted, however, that several of the
women had a lower $\dot{V}O_{2\text{max}}$ by almost 4 ml·kg$^{-1}$·min$^{-1}$ during the luteal phase. While aerobic capacity is not the only determinant of performance and athletic success (40), it nevertheless contributes in a substantial fashion. For the individual athlete in whom all other variables remain constant, a cyclic change in aerobic capacity of this magnitude may still have potentially meaningful implications for elite level competition, where the difference between first and second place finish is often measured in tenths of a second.

Other important factors affecting performance in distance running are running efficiency or economy, as well as the ability to sustain a high fractional utilization of $\dot{V}O_{2\text{max}}$ for a prolonged period of time (30). This “endurance capacity” has been linked to a high percentage of Type I muscle fibers, the capacity to store large amounts of muscle and/or liver glycogen; the capacity to spare carbohydrate reserves by preferential use of free fatty acids; and the capacity to efficiently dissipate heat (30). As already discussed, the last three variables may be affected by changing hormonal status throughout the menstrual cycle. During the luteal phase there is at least a theoretical metabolic advantage, as well as a potential thermoregulatory disadvantage.

In the present study, high intensity endurance performance was not significantly increased during the luteal phase ($\Delta = 15.5 \pm 44.4$ s, $P = 0.72$). It should be noted, however, that the observed standard error for this test was higher than predicted. In contrast, one group (18) found a luteal phase doubling in endurance time, in association with lower lactate levels. Luteal phase decreases in blood lactate and respiratory exchange ratio during maximal (10) and submaximal (23) exercise have also been demonstrated by other investigators. However, earlier studies (3,15,16,21) and more recent work (8,19,28) have not substantiated these findings.

Initial muscle fuel stores are also important for optimal endurance performance. Studies with (28) and without hormonal documentation (13) have shown higher muscle glycogen levels during the luteal phase. One group (28) also found a trend ($P < 0.07$) toward an associated increase in muscle endurance, but no difference in actual glycogen utilization during exercise in the two phases. Other authors have found either no phase difference in endurance performance (10), or even a longer work time during the follicular phase, albeit in nonathletes only (38).

This inconsistency in results can possibly be explained by differences in the testing procedures used. One study (18) used a continuous progressive incremental protocol on a bicycle ergometer (20 min successively at workloads of 33% and 66% of $\dot{V}O_{2\text{max}}$ followed by a test to exhaustion at 90% $\dot{V}O_{2\text{max}}$), while another employed steady state cycling at 70% of $\dot{V}O_{2\text{max}}$ (28). In the present protocol, the endurance run was performed on the treadmill on the second day of testing, after completion of the strength tests and a short warm-up run. At an intensity of 90% of $\dot{V}O_{2\text{max}}$, the subjects likely all exceeded their anaerobic threshold; however, they were able to maintain an output of at least 90% of $\dot{V}O_{2\text{max}}$ for anywhere from 4.5 to 22 min, independent of menstrual cycle phase. The ability to sustain performance at such a high intensity may be a more important overall determinant of athletic prowess than absolute $\dot{V}O_{2\text{max}}$ (30).

In the current study, aerobic capacity was not measured at midcycle when estradiol levels are very high and progesterone levels are still low. Some investigators have shown an increased perception of effort, and a higher fat utilization during the time of ovulation (14). Aerobic capacity and/or endurance performance might be altered in a different fashion during this time. None of the other physiologic or performance measurements taken during exercise in the present study showed any significant variation between the follicular and luteal phases. Again, these findings are in accordance with the conclusions reached by the majority of previous researchers. Both early studies without hormonal measurements (see 24 for review) and more recent work utilizing progesterone and estradiol levels for confirmation of cycle phase have failed to demonstrate any significant alterations in maximum heart rate (8,10,18,28) or respiratory exchange ratio (8,16,19,21,28) in athletes related to phase of the cycle. A few authors (15,32,38) have documented a higher heart rate during submaximal and maximal exercise in the luteal phase, and postulated (32) adverse implications for prolonged exercise at high ambient temperatures.

The lack of change in maximum minute ventilation in this study is also in agreement with most previous results (8,10,21,38), although some studies (16,18) have found a luteal phase increase in minute ventilation. One group (38) found changes in normally menstruating nonathletes, but not in normally menstruating athletes or amenorrheic controls, suggesting a possible effect of a subjective sensation of dyspnea in limiting exercise performance. The lack of a significant progesterone effect on ventilation in athletes, however, is consistent with the observation that outstanding endurance athletes have decreased ventilatory drives in response to hypoxia and hypercapnia (6,26) with beneficial effects for performance. Although the increased sensitivity of these respiratory drives seen during the luteal phase could potentially cause a performance decrement, this has not been substantiated to date (10,38).

There have been few studies using serum progesterone measurements that have specifically examined the effects of cycle phase on anaerobic performance. The metabolic effects of estradiol and progesterone on substrate utilization during exercise mostly come into play in prolonged exercise, whereas short-term anaerobic metabolism relies more on ATP and creatine phosphate (7). Hormonally mediated differences in preexercise glycogen levels
could theoretically influence the proportion of energy derived from aerobic sources, but during the first 30 s of exercise, the contribution from aerobic glycolysis is so small that any potential impact would be minimal. In the current study, there were no significant phase differences found in the anaerobic speed test (AST). The variation and the range of values seen was likely due to the sport-specific training of these subjects (i.e., the squash players had the highest values at 35–40 s). These AST values are only indicative of their anaerobic capacity, and do not reflect their aerobic fitness. In actual fact, low anaerobic performance is common in aerobically or endurance-trained athletes.

Studies on strength performance throughout the menstrual cycle are sparse, conflicting, and generally lack hormonal documentation. Some have shown lower isometric strength and endurance (42) and maximal voluntary contraction of forearm muscles (31) during the luteal phase, while others have not measured any menstrual phase differences in performance in bench and leg press (37) or in knee extension and flexion strength or endurance as tested on a Cybex II dynamometer at 60, 150, and 240°·s⁻¹ (9). In the present protocol, there were no differences in isokinetic strength of knee flexion or extension between the two cycle phases.

Neither hemoglobin concentration nor hematocrit varied significantly between phases. Other investigators have noted both slight decreases (10) or increases in resting hemoglobin concentration (18) and hematocrit during the luteal phase, without any associated alteration in performance. A lower hematocrit and hemoglobin concentration may simply reflect plasma volume shifts occurring from the physiological actions of estradiol. Another author (12) has suggested that blood progesterone levels must be low in order to see this estrogenic effect. In the present study, both estradiol and progesterone levels were high during the luteal phase.

There were no significant differences in body weight, percentage body fat, or sum of skinfolds between the follicular and luteal phases in this group of 16 subjects. Many women complain of a subjective feeling of weight gain in the premenstrual phase but there is evidence (34) that regular physical exercise can ameliorate some premenstrual symptoms, including fluid retention. This factor may have accounted for the lack of variation in body weight in the athletes in the present study. Recent work (5) has also questioned the validity of underwater body densitometry in women, because menstrual cycle changes in plasma volume and total body water may lead to errors in the determination of the percentage of body fat when using conventional formulae. It may thus be more appropriate to use the sum of the obtained skinfold measurements for comparative purposes.

In conclusion, although both absolute and relative VO₂max were slightly lower during the luteal phase of the menstrual cycle in these 16 trained female athletes, there was no significant effect of menstrual cycle phase on anaerobic performance, aerobic endurance performance, or isokinetic muscle strength. Furthermore, there were no associated alterations in maximum minute ventilation, maximum heart rate, maximum respiratory exchange ratio, body composition, or any hematological variables to explain these changes in aerobic capacity.

It thus appears that the phases of an ovulatory menstrual cycle have a minimal impact on most indices of performance, with the possible exception of aerobic capacity. The magnitude of this effect varies substantially between subjects, and may be important on an individual basis. This type of study needs to be repeated with a larger number of subjects, and at different phases of the cycle, including the midcycle estrogen peak, in order to further investigate the hormonal components of this effect. Newer assays to detect the midcycle urinary peak of luteinizing hormone (LH) can further add to the accuracy of timing of testing. Further studies should focus on the potential mechanisms of any observed change in functional aerobic capacity, and also on sport-specific indicators of performance in the elite athlete. Finally, with regard to other training studies involving female athletes, it may be advisable to standardize the cycle phase during which testing is carried out to eliminate potentially confounding effects of any physiological changes that may result from endogenous variations in the female steroid hormones.

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