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PRELIMINARY REPORT

Effects of the Menstrual Cycle on Excess Postexercise Oxygen Consumption in Healthy Young Women

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The effects of the menstrual cycle on excess postexercise oxygen consumption (EPOC) were studied in seven healthy young women aged 18 to 20 years. EPOC, resting metabolic rate (RMR), and energy expenditure during exercise (EEDE) in the fasting state were measured in the follicular and luteal phases. On the experimental days, subjects exercised for 60 minutes on a bicycle ergometer at an intensity of 60% maximal oxygen consumption ($\dot{V}O_2$max) followed by rest for 6 hours. The EPOC and RMR were significantly higher ($P < .05$) and the postexercise respiratory exchange ratio (RER) was significantly lower ($P < .05$) in the luteal phase versus the follicular phase, whereas differences in the EEDE and basal and exercise RER were negligible in both phases. Fat oxidation during the experimental period was significantly greater in the luteal phase ($P < .05$). These results suggest that exercise in the luteal phase results in greater postexercise energy expenditure and fat utilization than in the follicular phase.

IT IS WELL ESTABLISHED that oxygen consumption remains elevated for a period of time after exercise, and the phenomenon has recently been termed excess postexercise oxygen consumption (EPOC).[1] EPOC decreases rapidly during the first hour postexercise, but there is also a prolonged EPOC component that may persist for as long as 12 hours.[2,3] This prolonged elevation of resting oxygen consumption has been connected partly to the cost of increased triacylglycerol–fatty acid substrate cycling.[4] Bahr and Sejersted[5] showed the presence of a prolonged EPOC component in the fasting state, which must be caused by processes other than an increased rate of glycogen resynthesis or a potentiation of diet-induced thermogenesis.

On the other hand, interest in the possible variability in energy expenditure during the menstrual cycle has increased in recent years, although studies to assess the effects of the menstrual cycle on the basal metabolic rate were performed by several groups as early as the 1920s.[6-10] All such studies report an increase in the resting metabolic rate (RMR),[7] sleeping metabolic rate,[8] or total energy expenditure[9] in the luteal phase of the menstrual cycle. However, more recent studies by Piers et al[11] and Tai et al[12] found no significant variations in RMR during the different phases of the menstrual cycle.

The effect of the menstrual cycle on exercise-induced thermogenesis has not been studied as extensively as its effect on RMR. Stephenson and Kolka[13] compared the esophageal temperature during exercise (80% maximal oxygen consumption [$\dot{V}O_2$max]) in the follicular and luteal phases of the menstrual cycle, and found that the difference in core temperature was less than the difference observed at rest between menstrual cycle phases. Pivarnik et al[14] reported that during exercise (65% $\dot{V}O_2$max) in a cool environment (22°C), rectal temperature continued to increase throughout a 1-hour period during the luteal phase, whereas rectal temperature reached a steady state during the follicular phase. However, there are no reports on the effect of the menstrual cycle phase on energy expenditure during exercise (EEDE) or EPOC.

This study was performed to establish whether there are changes in exercise-induced thermogenesis, EPOC and EEDE, during the menstrual cycle in healthy young women.

SUBJECTS AND METHODS

Subjects

Seven young Japanese women aged 18 to 20 years were recruited from Sanyo Women's College (Hiroshima, Japan) for this study. All

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subjects were physically active but were not engaged in regular training at the time of study. All procedures were approved by the Institutional Review Board of Sanyo Women's College and are in accordance with the Helsinki Declaration, as revised in 1983. After a detailed explanation of the study, each subject provided informed written consent. The subjects were found to be free of disease by a medical examination before the study. Subject characteristics are as follows: height, 158.3 ± 2.4 cm; body weight, 55.5 ± 2.5 kg; body fat, 28.9% ± 2.3% (mean ± SE). The phase of the menstrual cycle was determined as described previously[12] (follicular phase, days 6 to 10; luteal phase, days 21 to 25).

Preexperimental Procedures

Before the experiments started, all subjects were familiarized with bicycle exercise at a constant pedaling rate and with breathing through a face mask. About 4 weeks before the experiments started, $\dot{V}O_2max$ was measured using criteria modified for the cycle ergometer from Taylor et al.[15] $\dot{V}O_2max$ was 49.4 ± 1.7 mL · $kg^{-1}min^{-1}$ (mean ± SE). These results were used to predict workloads corresponding to 60% of $\dot{V}O_2max$ in each subject.

Experimental Design

During the study period, each subject maintained a normal life-style and ate ad libitum except for the day before the experimental period. That evening, each subject ate the same supper (50 kJ · kg^{-1} body weight) at 7:00 PM. Subjects fasted overnight and entered the experimental laboratory room at 7:00 AM, where they rested until the start of the experiment at 8:30 AM. The experiment was performed in the follicular phase and the luteal phase.

In each experiment, the subjects rested for 30 minutes, and then exercised for 60 minutes on a bicycle ergometer (Monark, Varberg, Sweden) at an intensity of 60% $\dot{V}O_2max$ at 9:00 to 10:00 AM. The subjects then rested for 6 hours. During rest and exercise, oxygen uptake and the respiratory exchange ratio (RER) were measured (8:30 AM to 4:00 PM). Urine samples were collected during the experimental period. All procedures were performed in the experimental laboratory room under the same conditions (temperature 22° ± 1°C and humidity 60%).

Measurements

Indirect calorimetry was performed using the ventilated-hood technique. For the measurement of oxygen uptake and RER, the subjects wore face masks (Takei, Tokyo, Japan) continuously. During exercise, oxygen uptake was measured from 9:15 to 9:18, 9:30 to 9:33, and 9:45 to 9:48 AM. Postexercise oxygen uptake was measured continuously for the first hour and for the last 15 minutes of every hour for the next 5 hours. All expired gas was collected into a Douglas bag (Takei) and the concentrations of oxygen and carbon dioxide were immediately measured using a gas analyzer (model RAS-30 and RAS-40; AIC, Tokyo, Japan). EPOC was calculated as the time integral of oxygen uptake above the resting baseline for the postexercise period[16] (Fig 1B). The RMR, EEDE, and postexercise total energy expenditure (PTEE) were calculated from oxygen consumption and carbon dioxide production, corrected for urinary nitrogen loss as described by Consolazio et al.[17] The RER was also calculated and used as an index of carbohydrate and fat utilization.

The statistical significance of differences between menstrual cycle phases was tested using Student's paired *t* test.

RESULTS AND DISCUSSION

Oxygen uptake, excess oxygen uptake, and the RER during rest and exercise were measured for 7.5 hours to assess the thermogenic effects of exercise (Fig 1A to C). The EPOC component in Table 1 was calculated as the time integral of the

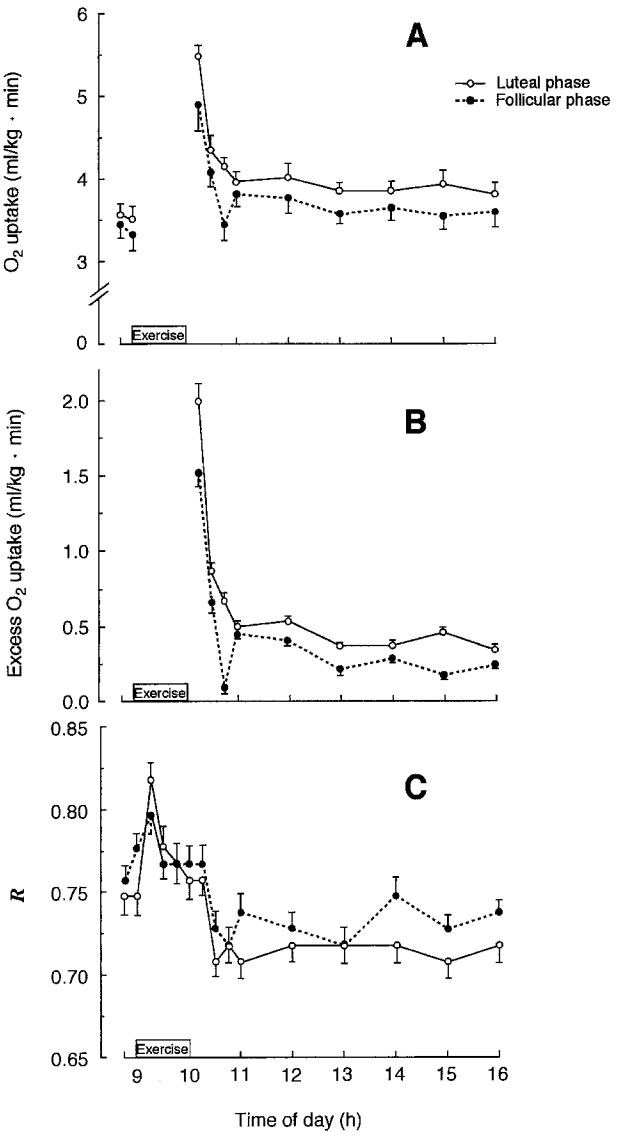

Fig 1. Oxygen uptake (A), excess oxygen uptake above the resting baseline (B), and RER (*R*,C) during rest and exercise in the follicular and luteal phases of the menstrual cycle. Oxygen consumption and carbon dioxide production were measured, and *R* was calculated from these values. Each point represents the mean ± SE for 7 subjects.

curve in Fig 1B. The mean values for RMR, EEDE, PTEE, and RER calculated from oxygen consumption (data in Fig 1), carbon dioxide production, and urinary nitrogen loss are shown in Table 1. The RMR, EPOC, and PTEE were significantly higher in the luteal phase versus the follicular phase ($P < .05$). The difference in EEDE was negligible in both phases. On the other hand, whereas the basal and exercise RER did not differ between the follicular phase and luteal phase, the postexercise RER was significantly lower in the luteal phase versus the follicular phase ($P < .05$). Substrate oxidation during the experimental period (450 minutes) in the follicular phase was 41.5 g (36.5% of energy), 26.5 g (52.5%), and 12.5 g (11.0%) for carbohydrate, fat, and protein, respectively. In the luteal phase, these figures were 44.9 g (35.2%), 31.2 g (55.1%), and

Table 1. Metabolic Indices Measured During a Single Menstrual Cycle

Metabolic Index	Follicular Phase	Luteal Phase
RMR (J/kg/min)	67.3 ± 3.1	70.2 ± 2.9*
EPOC (L/6 h)	8.40 ± 1.18	12.3 ± 1.91*
EEDE (kJ/kg/h)	6.17 ± 0.44	6.08 ± 0.36
PTEE (kJ/kg/6 h)	27.2 ± 1.1	29.6 ± 1.1*
RER		
Basal	0.76 ± 0.02	0.75 ± 0.02
Exercise	0.78 ± 0.02	0.80 ± 0.02
Postexercise	0.74 ± 0.01	0.72 ± 0.01*

NOTE. Values are the mean ± SE for 7 subjects.
*$P < .05$ *v* follicular phase (Student's paired *t* test).

12.4 g (9.7%), respectively. Fat oxidation during the experimental period was significantly greater in the luteal phase ($P < .05$).

EPOC was higher in the luteal phase. Because subjects in both menstrual phases were treated identically throughout the experimental period, the difference in EPOC was ascribed to the different menstrual phases.

It should be noted that fat metabolism may be of importance for EPOC observed in the fasting state, through the energy cost of increased triacylglycerol–fatty acid substrate cycling after exercise.[4,5,18] In the present study, the RER was significantly lower in the luteal phase after exercise compared with the follicular phase. Because there appears to be a relationship between the rate of triacylglycerol–fatty acid cycling and a reduction in the RER after exercise,[4] it is possible that the observed substrate shift in the luteal phase is caused by an increased rate of cycling. Triacylglycerol–fatty acid cycling is affected by the sympathetic nervous system and circulating catecholamines.[18] The higher blood estradiol concentrations generally observed in the luteal phase[11] are thought to be related to the elevation of catecholamines. Neuronal reuptake of catecholamines is an important mechanism for quickly terminating neurotransmitter activity.[19] Catecholamines secreted from the adrenal medulla are rapidly metabolized by catechol-*O*-methyltransferase to form inactive *O*-methylated metabolites in the liver or skeletal muscle.[19] Salt[20] reported that catecholamine reuptake was inhibited by estradiol in isolated rat hearts. Davidson et al[21] demonstrated that estradiol competitively inhibited catechol-*O*-methyltransferase.

The EEDE and exercise RER were not different between the follicular and luteal phases. Carpenter and Nunneley[22] reported that during 2 hours of low-intensity exercise, core temperature differences observed at rest during the follicular and luteal phases were increased by approximately the same magnitude. Because energy metabolism is related to core temperature, our finding would support their data.

Exercise training has useful effects on diet therapy for obese individuals because of the increase in energy expenditure and fat metabolism.[23] In the present study, energy expenditure and fat utilization during the experimental period were greater in the luteal compared with the follicular phase of the menstrual cycle. Although further studies are required to clarify the details, the findings suggest that the menstrual cycle affects postexercise energy metabolism in women.

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