

INTRODUCTION

The female sex steroids, estrogen and progesterone have been shown to influence carbohydrate (1) and lipid (2, 3) metabolism, and respiratory function (4). The plasma progesterone level markedly increases in the luteal phase of the menstrual cycle (5). During the pregnancy (6) and the luteal phase (7) progesterone appears to increase ventilation. In this phase a decrease in resting end-tidal PCO_2 and base excess have been reported (7). Compared to follicular phase ventilatory equivalent (VE/VO_2) during progressive exercise and minute ventilation during resting (8) and exercise (9) have been observed to be higher in the luteal phase. In contrast, several studies did not find any significant difference in ventilatory responses including minute ventilation between follicular and luteal phases during exercise (10, 11).

Sleep deprivation is one of the stress sources in human (12) and most common sleep deprivation is one night or less sleep deprivation. Sleep deprivation in exercise increases the rating of perceived exertion (13, 14) and produces changes in thermoregulation (15). It has been reported that prolonged sleep deprivation (36 hours) disturbed mood but did not alter heart rate, oxygen uptake, minute ventilation and body core temperature responses to light or heavy exercise (16). The studies concerning the effects of sleep deprivation on cardiovascular and respiratory responses to exercise also showed that the sleep loss of 30 to 72 hours did not change cardiorespiratory responses to exercise (17-19), aerobic and anaerobic performance (19). However it has also been found that 30 hours sleep deprivation

decreased cardiorespiratory variables in exercise (20).

In recent years, an increase in participation of female subjects in exercise has increased the interest in physiological responses of women to exercise (21). It has been believed that enough sleep is necessary for maximum exercise performance (22). However, sleep deprivation in females during their follicular and luteal phases may occur in many situation such as travelling, night shift and over night preparing for examination. There are conflicting reports of the effect of the cycle phase and sleep deprivation on the cardiorespiratory responses to exercise. Furthermore, the effect of sleep deprivation on the cardiorespiratory and spirometric parameters in the follicular and luteal phases of healthy females has not been studied. Therefore, the present study was designed to examine the influence of one night's sleep loss on cardiorespiratory parameters during the menstrual cycle. We tested the hypothesis that sleep deprivation would have some effects on the cardiorespiratory variables during the follicular and luteal phases. We have also studied the resting spirometric parameters in the two phase of the cycle under non sleep- deprived and sleep-deprived conditions.

METHODS

Subject

In our laboratory at the altitude of 700 m (barometric pressure 699 mm Hg) nine healthy, nonsmoker, regularly menstruating females were studied for two months. All

was not in training. The subject physical characteristics are given in Table I. None of the subjects had any medication including oral contraceptives. Our protocol was approved by the Ethics Committee for protection of human subjects. Before the experiments started, the subjects gave written, informed consent and they were familiarized with the instruments and experimental procedures.

Design of the study

Our study continued two months. In the first month after a normal night sleep and the second month following one night sleep deprivation, whole blood counting, rutin biochemistry tests including the hormone analysis, spirometry and cardiopulmonary exercise testing were performed. These tests were made between days 11–13 in the follicular phase and days 22–24 in the luteal phase. Menstrual histories of the subjects provided a supportive evidence for menstrual phases and subsequently menstrual status was confirmed by low progesterone level between days 11–13 of the follicular phase and high progesterone between days 22–24 of the luteal phase.

Measurements and experimental protocol

The blood samples of the subjects were drawn in the morning of following an overnight fast and spirometric and exercise testing were applied 2 hours after the breakfast. These testings were performed at the room temperature of 20 ± 2 C° with a cardiopulmonary exercise testing instrument (Sensor Medics V_{max} 29C, Bilthoven, Netherlands). The flow sensor was calibrated

using a calibrating syringe. The spirometric tests were repeated at least three times and the highest values were taken. MVV was performed asking the subject to breath as deep and as fast possible for 12 s, with a target rate of approximately 100 breaths/min. Immediately after the spirometric tests the O₂ and CO₂ analyzer was calibrated using the standart gas mixtures. Exercise testing was performed on a computer controlled, speed independent bicycle ergometer (Ergometric er900, Bitz, Germany). Subjects exercised at submaximal exercise intensities. The protocol consisted of 2 min resting, 1 min unloaded pedalling and the increments in work rate as 25 watt per minute. The subjects were strongly encouraged to cycle to the point of exhaustion. 5 min after the loaded pedalling work load and heart rate reached to 125 watt and 150–160 beats/min, respectively. Exercise testing was ended rate reached to 125 watt and 150–160 beats/min, respectively. Exercise testing was ended by supervising physican when the exhaustion was confirmed by maximum heart rate (150–160 beats/min) and by the decrease of pedalling rate. The maximal heart rate was measured during the last min of the test and this value was within 80–90% of predicted heart rate by the computer software. During the exercise testing breath-by-breath following variables were determined by the computer (Pro-2000, Inselberg); VO₂, VCO₂, VE, VT, RR, R, end-tidal PO₂ and end-tidal PCO₂. HR and ECG were continuously monitored and systolic and diastolic blood pressures were periodically measured using an automatic blood pressure system of the ergometer. SaO₂ was continuously measured with a dual wavelength finger oximeter (Sat-Trak TM Pulse oximeter, Bilthoven, Netherlands). Progesterone was analyzed by

a full automated, solid phase, two-side chemiluminescent enzyme immunometric assay using a Diagnostic Products Corporation (DPC) IMMULITE ® analyzer.

Sleep deprivation

During the period of a wakefulness night, the subjects were strictly controlled by supervisor and their activities were kept to a low level. The subjects were remained awake by reading, watching television and conversation. Drinks with caffeine or alcohol prohibited. Subjects who did not follow these restrictions were excluded from the study.

Statistical analysis

The data were analyzed using a two-way analysis of variance (ANOVA) followed by Tukey HSD test. Values are given as the mean \pm the standard error of the mean. A P value less than 0.05 was considered as statistical significant.

RESULTS

The serum progesterone values at rest of the subjects after one night sleep were 1.06 ± 0.23 and 12.93 ± 1.38 ng/ml during follicular and luteal phases, respectively. After a sleepless night progesterone levels at rest were 1.12 ± 0.24 ng/ml for follicular phase and 12.14 ± 1.87 ng/ml for luteal phase. Luteal concentrations of progesterone were significantly higher ($P < 0.05$) than follicular concentrations following normal night sleep or sleep deprivation.

The mean values of resting spirometric parameters and the comparison between the

TABLE I: Physical characteristics of the subjects.

| | Mean \pm SE | Range |
|-----------------------|-------------------|-----------|
| Age (year) | 27.83 \pm 1.66 | 24–35 |
| Height (cm) | 162.83 \pm 2.59 | 156–173 |
| Weight (kg) | 56.67 \pm 1.87 | 51–63 |
| BSA (m ²) | 1.6 \pm 0.03 | 1.51–1.69 |

BSA, body surface area.

TABLE II: Resting pulmonary function of the nine subjects.

| Parameter | After one night's sleep | | After one night's sleep loss | |
|------------------------|-------------------------|-------------------|------------------------------|-------------------|
| | Follicular | Luteal | Follicular | Luteal |
| FVC, L | 3.93 \pm 0.22 | 3.86 \pm 0.19 | 3.9 \pm 0.3 | 3.95 \pm 0.4 |
| FEV ₁ , L | 3.13 \pm 0.14 | 2.9 \pm 0.02 | 3.06 \pm 0.15 | 2.93 \pm 0.33 |
| FEV ₁ /FVC% | 80.8 \pm 4.65 | 76 \pm 3.46 | 79.83 \pm 4.23 | 74.16 \pm 5.18 |
| FEV ₃ , L | 3.72 \pm 0.18 | 3.58 \pm 0.16 | 3.68 \pm 0.25 | 3.51 \pm 0.03 |
| FEF 25-75%, L/s | 3.9 \pm 0.26 | 2.94 \pm 0.17 | 3.47 \pm 0.32 | 2.98 \pm 0.36 |
| FEF 25%, L/s | 3.95 \pm 0.35 | 3.34 \pm 0.22 | 4.51 \pm 0.49 | 3.58 \pm 0.44 |
| FEF 50%, L/s | 3.81 \pm 0.34 | 3.18 \pm 0.22 | 3.89 \pm 0.42 | 3.11 \pm 0.4 |
| FEF 75%, L/s | 1.97 \pm 0.18 | 1.81 \pm 0.19 | 1.96 \pm 0.27 | 2.03 \pm 0.3 |
| FEF 75-85%, L/s | 1.47 \pm 0.19 | 1.26 \pm 0.13 | 1.46 \pm 0.3 | 1.56 \pm 0.34 |
| PEF, L/s | 4.57 \pm 0.39 | 3.65 \pm 0.23 | 4.65 \pm 0.51 | 4.02 \pm 0.46 |
| ERV, L | 1.29 \pm 0.09 | 1.05 \pm 0.1 | 1.07 \pm 0.14 | 1.45 \pm 0.2 |
| IC, L | 2.58 \pm 0.15 | 2.53 \pm 0.11 | 2.46 \pm 0.12 | 2.58 \pm 0.25 |
| MVV, L/min | 116 \pm 9.78 | 115.83 \pm 7.73 | 123.5 \pm 7.23 | 121.16 \pm 5.83 |

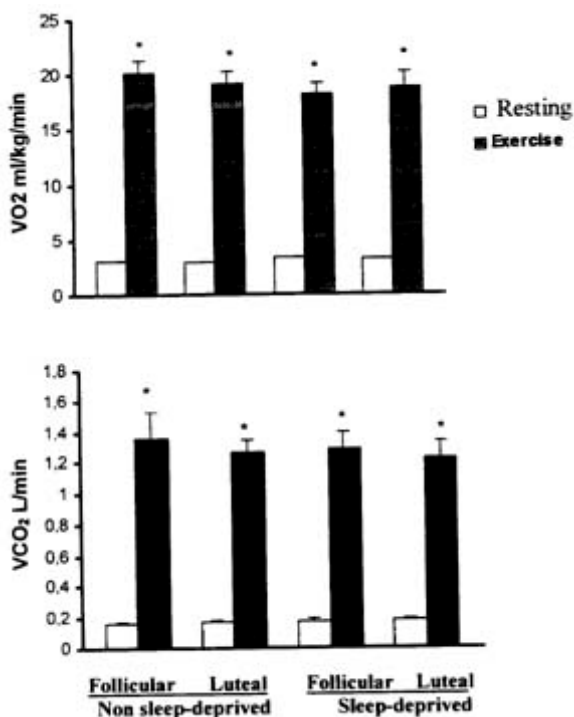


Fig. 1: Resting and exercise values of VO_2 and VCO_2 in follicular and luteal phases following one night's sleep or sleep deprivation. * $P < 0.001$ significantly different from the respective resting value (n = 9).

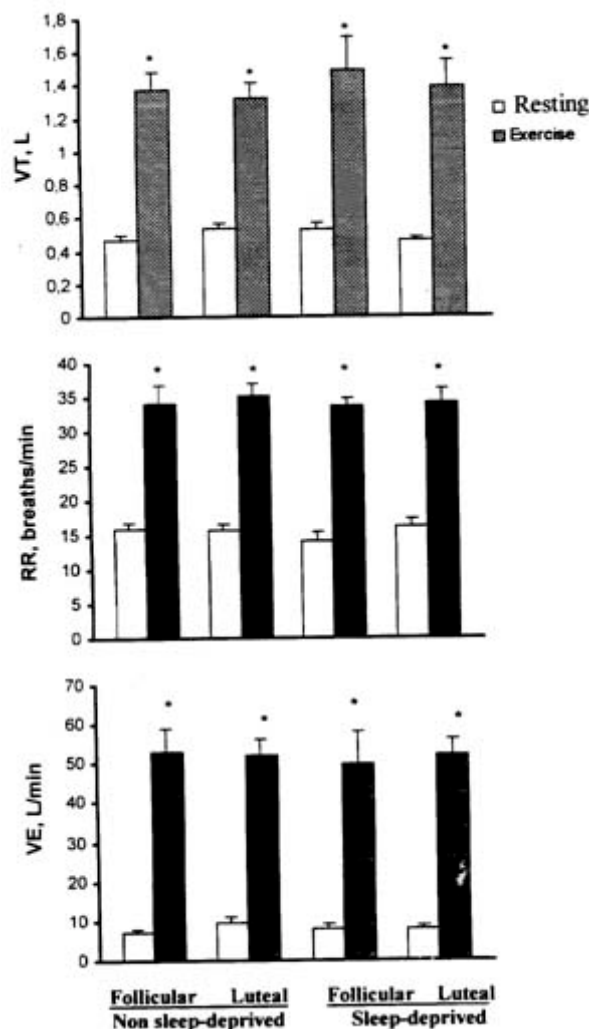


Fig. 2: Resting and exercise values of VT, RR and VE (n = 9).

menstrual phases under non sleep-deprived and sleep-deprived conditions are shown in Table II. FVC, FEV_1 , $FEV_1/FVC\%$, FEV_3 , FEF 25-75%, FEF 25%, FEF 50%, FEF 75%, FEF 75-85%, PEF, ERV, IC and MVV values showed no significant difference between the phases of the menstrual cycle following a night's sleep or a sleep-deprived night.

As shown Figs. 1, 2 and 3 VO_2 , VCO_2 , VT, RR, VE, systolic blood pressure and heart rate markedly increased with exercise both in follicular and luteal phases after a night's sleep or sleep deprivation. The mean values

of these variables at exercise were not statistically different between the cycle phases. Although under non sleep-deprived and sleep-deprived conditions of the follicular and luteal phases diastolic blood pressure and end-tidal PO_2 and end-tidal PCO_2 tended to increase with exercise, these trends did

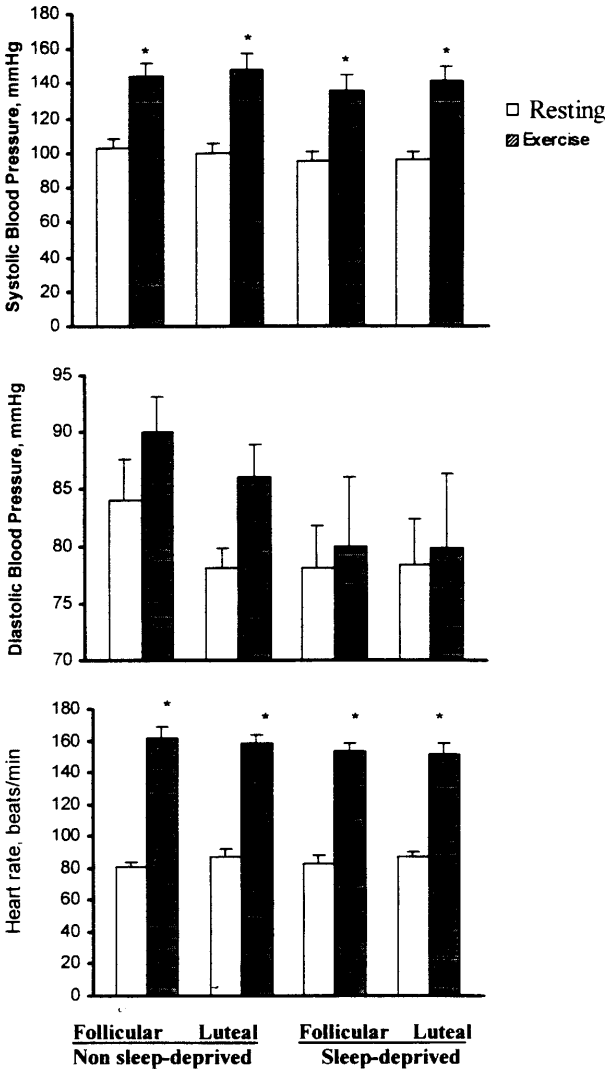


Fig. 3: Resting and exercise values of systolic and diastolic blood pressures and heart rate (n = 9).

not reach statistical significance (Fig 3 and 4). The exercise produced significant increases in R but it did not change SaO₂ (Fig 4). None of the parameters was affected significantly by menstrual cycle phase or sleep loss of one night.

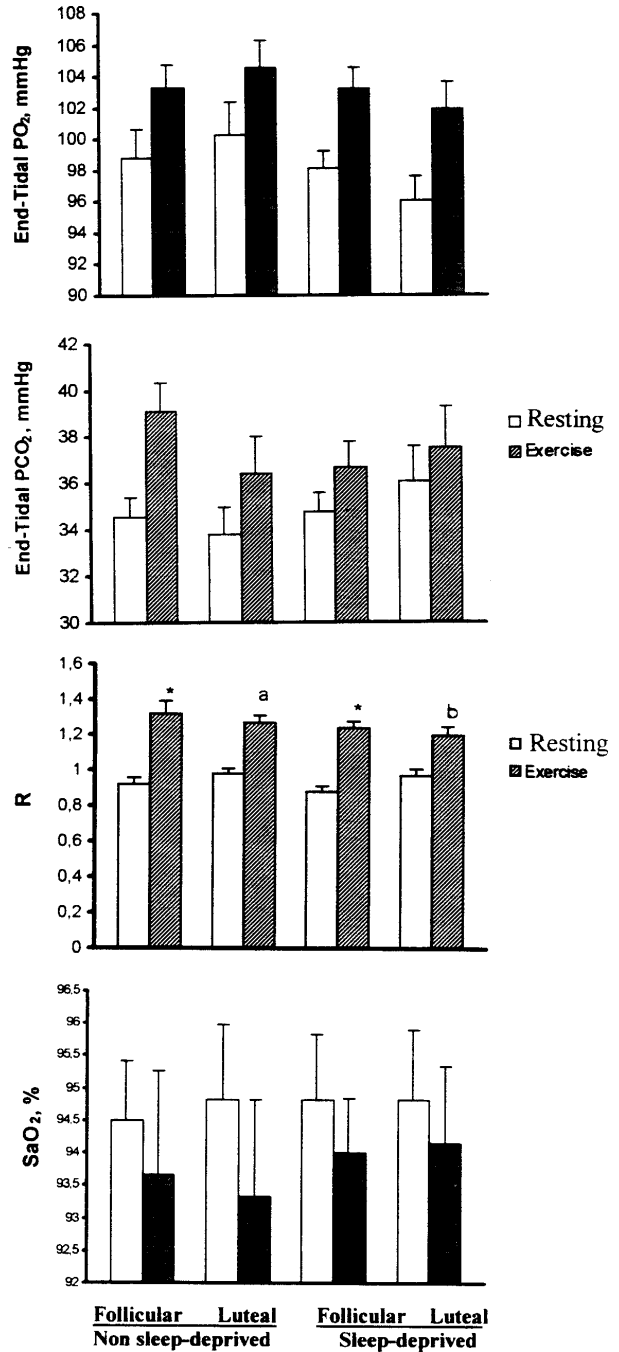


Fig. 4: End-tidal PO₂, end-tidal PCO₂, R and SaO₂ (n = 9). a; P<0.01 and b; P<0.05 significantly different from the respective resting value.

DISCUSSION

We have found that the respiratory responses to exercise were similar during the phases of the cycle following one normal sleep or one night sleep deprivation. In agreement with our results previous studies have reported that VE was not different between the follicular and luteal phases during the exercise (11, 23). Our findings are different from those of two earlier studies. Schoene et al. (8) have reported that in controls and highly trained athletes ventilation was significantly higher in the luteal phase during the progressive work-exercise using 100 kpm/ min increments carried to maximal effort. Williams and Krahenbuhl (9) have also reported that ventilation was significantly greater in the luteal phase of moderately-trained female runners during the running at speed corresponding to 55% and 80% maximal oxygen consumption (VO_2 max). It has been known that progesterone stimulates ventilation (24). In the present study exercise ventilation was not higher in the luteal phase despite increased serum progesterone level. It has been reported that exercise produces increases in estradiol and progesterone levels and the increases have been found to be higher in the luteal phase (10). Furthermore, it has been suggested that progesterone receptors are critical for resting ventilation (23). It is possible that subjects in experiments may be unresponsive to progesterone. Therefore, the increased progesterone level may not induce an increase in exercise ventilation in the luteal phase compared to follicular phase. A decrease in resting arterial partial pressure of carbon dioxide ($PaCO_2$) resulting from an increased alveolar ventilation in the luteal phase has been reported (4). Almost a 4% increase in ventilation for the decrease in

$PaCO_2$ is necessary. When the increase of ventilation was continued during the exercise it may be responsible for approximately 1 or 2 liters ventilation difference between the follicular and luteal phases during the light or heavy exercise, respectively (10). It appears that ventilation difference may not be large enough for significant difference in VE between the cycle phases. It has been demonstrated that prolonged sleep deprivation (60 hours) did not alter the peak ventilatory responses of females to maximal aerobic exercise using a cycle ergometer protocol (25). However, in this study menstrual cycle phase was not considered. Our study indicates that one night sleep deprivation does not affect VT, RR and VE responses to exercise in the different phases of the menstrual cycle.

We have observed that VO_2 , VCO_2 , R, systolic blood pressure and heart rate increased during the progressive exercise tests. Furthermore, no difference in these responses following either normal sleep or sleep deprivation has been found. Our results suggest that the phase of the menstrual cycle or sleep deprivation does not modify these variables. Similarly previous studies on healthy women exercised on a treadmill or cycle ergometer reported no effect of the menstrual cycle phase on submaximal and maximal exercise VO_2 (10, 11, 23, 26, 27), VCO_2 (10, 11, 27) and heart rate (10, 11, 23, 26, 27). In young females the sleep deprivation (60 hours) remained unchanged VO_2 max and the other responses such as peak exercise ventilation, peak heart rate and peak R to maximal exercise (25) In contrast Chen (20) reported that sleep deprivation (30 hours) decreased exercise maximal oxygen consumption, peak VCO_2 and maximal heart rate. Plyley et al. (28) also reported that sleep deprivation (64

hours) decreased VO_2 max during submaximal treadmill exercise at 28% VO_2 max. It is possible that the male-female difference may be responsible for the different findings concerning the effects of sleep deprivation, because Chen (20) and Plyley et al. (28) studied in men.

In our study SaO_2 , diastolic blood pressure, end-tidal PO_2 and end-tidal PCO_2 were unaffected by exercise, menstrual cycle phase or sleep deprivation. Similarly, it has been reported that submaximal and peak SaO_2 (23) and end-tidal CO_2 tension (29) were unchanged by menstrual cycle phase.

Our results indicate that one night sleep deprivation does not change the responses to submaximal exercise during the follicular and luteal phases. It is possible that the methodological differences involving the duration of sleep loss or exercise protocols utilized may modify the effect of sleep deprivation on exercise cardiorespiratory responses in the follicular and luteal phases. Further studies are necessary to investigate the possible influence of sleep loss on exercise cardiorespiratory parameters in the different phases of the menstrual cycle.

In the present study, we have observed that spirometric variables following a night sleep or sleep deprivation did not show any difference between the follicular and luteal phase. These findings indicate that cycle phase or sleep deprivation does not affect spirometric parameters. Strinic et al. (30) similarly found no difference in lung volumes and flows between the follicular and luteal phases either in patients with genital descensus or in controls. Pauli et al. (31) also found no significant changes in spirometric parameters between the follicular and luteal phases in asthmatic and normal subjects. Our results suggest that hormonal changes associated with the menstrual cycle phases do not affect spirometric variables.

In conclusion, the resting spirometric and the cardiorespiratory responses to submaximal exercise appear to be independent of menstrual cycle phase. Sleep deprivation of one night does not affect the responses during the follicular and luteal phases of the menstruation. Therefore, our finding is considerable important for healthy women who perform physical work during their follicular and luteal phases after one night sleep loss.

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