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INFLUENCE OF A LOW-CARBOHYDRATE DIET ON THERMOREGULATORY RESPONSES TO EXERCISE IN WOMEN DURING FOLLICULAR AND LUTEAL PHASE OF THE MENSTRUAL CYCLE

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Abstract. The aim of this study was to examine the effects of a low-carbohydrate diet on thermoregulatory responses to exercise in women during follicular (F) and luteal (L) phase of the menstrual cycle. Ten subjects performed a graded bicycle exercise in a thermoneutral environment (23°C, 52-60% relative humidity). Women were tested after consuming, for 3 days, a control diet (C: 60% carbohydrates, 20% fat, 20% protein) and after that a low-carbohydrate diet (LCHO: 50% fat, 35% protein and 5% carbohydrates), in each phase of the menstrual cycle. Tympanic temperature (T_{ty}), mean skin temperature (T_{sk}), electrical skin resistance (ESR), oxygen uptake (VO_2), heart rate (HR) as well as blood β-hydroxybutyrate acid (β-HB), glucose (Glu) and lactate (LA) concentrations were measured. On the basis of ESR, dynamics of sweating was estimated. No differences in T_{ty} and T_{sk} were found between the C and LCHO during exercise tests. However, T_{ty} was significantly higher during L than F phase. Delay time for sweating was shorter after LCHO (F: 10.8 vs 9.4 min, $P<0.05$, L: 9.9 vs 9.3 N.S.), but temperature threshold for this reaction was unchanged (L: 37.22 vs 37.37 and F: 36.91 vs 36.94 °C). Sweating sensitivity was greater after LCHO during both F and L. Resting blood Glu and LA concentrations were similar in women after C and LCHO diet. Before exercise β-HB level was F: 0.45, L: 0.35 mM after LCHO and F: 0.08, L: 0.09 mM after C diet ($P<0.05$), respectively. At rest and during exercise HR was significantly higher after LCHO diet in women during F phase. In submaximal exercise loads VO_2 after LCHO diet were significantly higher than after C diet in all women. It was concluded that the low-carbohydrate diet ingested by young women in both phases of the menstrual cycle have no effect on body temperature, however, it affects heat dissipation mechanism during exercise. ***(Biol.Sport 20: 343-362, 2003)***

Key words: Carbohydrate – Thermoregulation – Sweating - Menstrual cycle – Diet – Exercise - Women

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Introduction

Several studies indicate that pre-exercise dietary manipulation aimed to decrease the availability of carbohydrate has an influence on physical performance, pattern of substrate utilization, metabolic energy production and body temperature regulation during low and moderate intensity exercise in men. [25,31,41,40]. There has been a little data available, however, concerning the influence of reduction in carbohydrate combustion and an increase of fat utilisation, which occur after consumption of low-carbohydrate diet, on thermoregulatory responses in exercising women. It is generally believed, that women have greater lipid oxidation during an endurance exercise, which may result in a sparing of muscle glycogen, and lower protein utilization at given percentage of VO_2max compared to men [24,40,50]. The mechanism responsible for the enhanced fat metabolism in women is an effect of gender differences in sex gonadal hormone levels. Female gonadal hormone concentration varies during the menstrual cycle with significant increased levels of progesterone and estradiol during the luteal phase of the menstrual cycle.

The changes in reproductive hormonal pattern during the menstrual cycle have an important role in altering thermoregulatory function as well as in substrate metabolism in women [1,5,33,40,49]. Elevation in concentration of progesterone, or rather changes in estrogens to progesterone ratio during luteal phase have been associated with an increase of internal body temperature, a higher core temperature thresholds for onset of specific thermoregulatory responses during exposure to high or low ambient temperatures or exercise [6,11,26,27,37,49].

On the other hand, the increases of circulating progesterone that occurred during the luteal phase have been associated with altered muscle glycogen and FFA utilization at rest and during submaximal exercise [17,50]. Lavoie *et al.* [32] found that plasma glucose content was decreased at 70 and 90 min of submaximal exercise (63% VO_2max) during luteal phase in women after carbohydrates (7.8%) diet applied 24 h prior to the experiment. Lynch *et al.* [35] observed similar alterations in metabolic responses to exercise in women after low-carbohydrate diet (31% vs 81% CHO) in follicular phase of the menstrual cycle. Hackney *et al.* [24] reported greater lipid utilization during luteal phase at the exercise intensities equal to 35% and 60% of VO_2max, whereas these differences between both phases of the menstrual cycle were not detectable at the level of relative exercise intensity of about 75% VO_2max.

The present experiments were designed to disturb the thermoregulatory function in exercising women by decreasing the availability of carbohydrates through a short-term consumption of a low carbohydrate diet. Although such shift in

substrate metabolism may be beneficial during prolonged low-intensity exercise a high intensity exercise, in the absence of glucose feeding, might be adversely affected [1].

To estimate an influence of low-carbohydrate diet on body temperature regulation and the potential modifying influence sex hormone levels on the thermoregulation a low-carbohydrate diet was administered to young, eumenorrhoeic women during follicular and luteal phase of the menstrual cycle in a randomized crossover design. It was hypothesised that a short-term diet-induced changes in metabolism would modify body temperature regulation in exercising women and that the dietary-induced changes in body temperature regulation would be related to the phase of the menstrual cycle.

Material and Methods

Subjects: Ten healthy, young women, students of the Academy of Physical Education, volunteered to participate in the study after being informed about the purpose of the study and their written, informed consent. The protocol was approved by the Ethical Committee of the Medical Research Centre of the Polish Academy of Sciences. Nine of the subjects were engaged in a professional sport. Each woman had a normal menstrual cycle defined by a regular periodicity (28 to 31 day cycles) over the previous year. The women were tested twice in the follicular (F) and in the luteal (L) phase of the menstrual cycle. Testing in follicular phase was performed between 3-7 day after menstrual bleeding. Testing in luteal phase was carried out between 19-26 day of the menstrual cycle. To verify the phase of the menstrual cycle, each subject recorded daily basal body temperature (BBT), for 2 months before the experiment. The resting estradiol (E_2) and progesterone (PE) levels were measured for the both phases of the menstrual cycle. Hormonal data are presented in Table 1.

Body mass was measured with a medical scale to the nearest 50g. Body fat content was calculated according to Durning and Wormersley's formula (1974) from the skin fold measurements. Skin surface area (A_D) was calculated from subject's height and weight according to DuBois and DuBois (7). VO_2max was assessed by direct method about one week prior to the experiment, during progressive exercise testing up to volutional fatigue. The characteristic of the subjects is presented in Table 2.

Table 1
Resting blood concentrations**:** progesterone (PE), estradiol (E_2) and B-hydroxybutyric-acid (β HB)

	Control diet			Low carbohydrate diet		
	Follicular phase	Luteal phase	Level of sign	Follicular phase	Luteal phase	Level of sign
PE ($mmol \cdot l^{-1}$)	2.11±1.84	36.85±22.2	P<0.05	3.05±1.1	25.78±23.7	P<0.05
E_2 ($nmol \cdot l^{-1}$)	0.36±0.38	0.35±0.04	P<0.05	0.24±0.38	0.27±0.33	P<0.05
β-HB	0.11±0.08	0.34±0.31	P<0.05	0.15±0.14	0.42±0.29	P<0.05

The resting blood glucose and lactate concentrations were unchanged either by menstrual cycle or diet.

Table 2
Subjects characteristic

Age (years)	20.8±0.98
Weight (kg)	59.3±8.48
Hight (cm)	167.4±5.9
Body fat %	28.07±2.05
Skin surface area (m^2)	1.66±0.14
VO_2max (ml/kg/min)	36.83±4.37

Experimental protocol: Ten women participated in four experiments (twice for follicular and twice for luteal phase of the menstrual cycle). The subjects performed incremental exercise, on an electrically braked ergometer (*Elema*), at a rate of 60 rpm, with identical protocol for phases F and L. They reported to the laboratory after an overnight fasting. Before the exercise a catheter was inserted into the antecubital vein and a resting blood samples were taken for determine: blood glucose, lactate and blood β-hydroxybutyrate acid (β-HB) concentrations as well as sex hormone levels. The exercises started with 3 min unload cycling. The load was progressively increased by 30 W for each 3 min of the exercise until

voluntary cessation. The exercise was repeated after two different diets. In test I, all subjects consumed normal-mixed diet (C) for 3-days. During test II, the subjects were asked to follow a diet with a low carbohydrates and high fat content (LCHO) for 3-days.

Diet composition: A control (mixed) diet was composed of 60% carbohydrates, 20% fat, 20% protein. A low-carbohydrate (LCHO) diet consisted of 50% fat, 45% protein and less than 5% carbohydrates. Both diets had the same energy content (2400kcal /75kg /24h). The order by which the two diets were administered was randomized.

Experimental condition

All experiments were carried out at the same time of day at an ambient temperature of 23.0±1.0^{o}C, to avoid possible effects of daily variability on performance or on body temperature. Relative air humidity, ranged 50-60%, was measured by Assman's psychrometer before and after exercise tests. During the exercise tests the subjects were dressed in shorts, socks T-shirts and athletic shoes.

Thermal analysis

Assessment of thermoregulatory responses: Tympanic temperatures T_{ty} (^{o}C) were recorded at rest, before and every 3 min during experiment, with a zero-gradient aural thermometer (Muirhead type 8151,1 Great Britain).

Skin temperature on the chest, arm and thigh was measured with a digital thermometer (Radiometer type Cannon TB 77B). Mean skin temperature (T_{SK}) was calculated by weighting factors reflecting regional proportions at the total body surface area according to equation of Burton's [4]. The accuracy of all temperature measurements was ±0.1^{o}C.

Sweating was estimated indirectly with the changes in electrical skin resistance (ESR) and measured using disposable unpolarized ECG electrode fixed to the skin of the sternal area. Following Grucza [22] the parameters describing the dynamics of sweating were defined as follows: a delay time (t_d), time constant (τ) and inertia time (t_i) -as the sum of delay and time constant.

T_{ty} threshold for onset of sweating and the sensitivity (slope) of the thermoregulatory reactions were determined in each exercising woman by calculation of linear regression equation for T_{ty} and 1-ESR.

The threshold (Th_{ty}) for thermoregulatory sweating was defined as Tty above, which ESR progressively decreased.

Physiological measurements

Pulmonary ventilation (V_E), oxygen uptake (VO_2) and carbon dioxide output (VCO_2) was determined before and during exercise using a medical gas analyser (*Beckman* USA).

Heart rate (HR) was recorded at rest and at each exercise load with a pulse meter *(Sporttester PE 3000*, Polar Electro, Finland)

RER, the ratio of the exchange of carbon dioxide and oxygen (VCO_2/VO_2), was determined by a ventilatory system. RER was used to estimate of the energy substrate that was utilized by the body and to determine energy expenditure.

The metabolic rate (M) was calculated from VO_2 and the caloric equivalents corresponding to the actual values of the respiratory exchange ratios. From the resting and exercise VO_2 the metabolic rate (M) and mechanical efficiency of work (WE) were calculated. Mechanical work efficiency was expressed as the W/M-Mso * 100%, whereas Msp is the resting metabolic rate ($J \bullet s^{-1}$).

The net amount of heat produced (H_{NET}) was expressed as the difference between M and the heat equivalent of the mechanical work performed (**W**); **R, C** - dry heat exchange by radiation [9] and convection [15]; $\mathbf{E_{res}}$ - evaporative heat loss from the skin [9]; $\mathbf{C_{res}}$, $\mathbf{E_{res}}$ - evaporative heat loss from the skin [9].

$$H_{NET} = \mathbf{M - W \pm R \pm C \pm C_{res} \pm E_{dif} \pm E_{res} \pm K}$$

where:

K - conductive heat loss (assumed negligible).

Blood sampling and analysis

On arrival to the laboratory each subject rested on the chair for 30 min and blood samples were taken from the antecubital vein for the measurements of blood glucose, B-hydroxy-butiric acid and the sex hormones (estrogen and progesterone). Plasma progesterone and estradiol concentrations (PE and E_2) were measured by the radioimmunoassay methods (*Orion Diagnostic* Finland). Plasma level of β-HB was determined enzymatically by method described by Williamson *et al.* [53]. Blood glucose concentration was determined at rest, before exercise test, using β-glucose Hemocue spectrophotometer, (Sweden). Blood samples for determination of lactate concentrations (LA) were withdrawn from finger tip before exercise and during last minute of each work load. Blood LA concentration was determined enzymatically using commercial kits *Boehringer, Mannheim* (Germany).

Statistics

To determine differences between the phases of F and L and between the control and LCHO diet, analysis of variance for multiple comparison was used (ANOVA). When a significant F-ratio was obtained the Newman-Keuls post-hoc analysis was used to isolate differences among treatment means. For all statistical analysis the 0.05 level of significance was used.

Results

Basal estradiol concentrations for each subject were in the expected range with respect to menstrual cycle phase. Progesterone level was significantly higher in the luteal phase compared with either follicular phase. As it could be expected estradiol concentrations were similar during follicular and luteal phase (Table 1).

Blood concentrations of β-HB were significantly greater after L-CHO diet (Table 1).

Resting blood glucose and lactate concentrations were similar after LCHO and C diet. During exercise blood LA concentrations were significantly lower after L-CHO than C diet in all women (Fig. 1). Final increase in LA concentration (ΔLA) was also significantly lower after the LCHO (F: 5.2±1.4 mM, L: 5.2±1.6 mM) than after C (F: 7.1±1.5 mM, L: 6.2±1.9 mM) diet ($P<0.05$).

The initial $T_{ty,}$ measured before the exercise, was significantly greater in women during luteal than follicular phase of the menstrual cycle. The type of diet did not influence the tympanic temperature in resting women. Time courses of T_{ty} during exercises were similar during both phases of the menstrual cycle. However, T_{ty} was always higher in L than F women (Fig. 2). Increase in T_{ty} during exercise test was similar after LCHO and C diet (Table 3).

Mean skin temperature (T_{sk}) was slightly higher at rest and at the beginning of exercise tests during L than F phase, but no differences in T_{sk} were detected between LCHO and C diet (Fig. 2). After LCHO diet T_{sk} was significantly higher in women during phase L than F. T_{sk} begun to fall at the very beginning of exercise in both tests. The fall in skin temperature (ΔT_{sk}) was greater in L than in F, independently on the diet applied (Table 3). The decrease in mean skin temperature following the increase in intensity of exercise coincided well with an increase in evaporation of sweat.

Minute ventilation and respiratory exchange ratio (RER) before and during exercise tests did not differ significantly between F and L phase after control diet. However, RER was significantly lower during exercise after LCHO diet in women during the luteal phase of the menstrual cycle. VO_2 and V_E increased during exercise in both trials but no significant differences were observed in between F and L in each exercise protocol. In submaximal exercise loads VO_2 was significantly greater after LCHO than C diet in all subjects (Fig. 1).

There were no significant diet-related differences between the metabolic heat and net heat production after LCHO when compared with C diet. However a greater values of H_{NET} was observed after the LCHO diet during phase L. Resting values of M did not differ between C and LCHO diet nor between phase F and L.

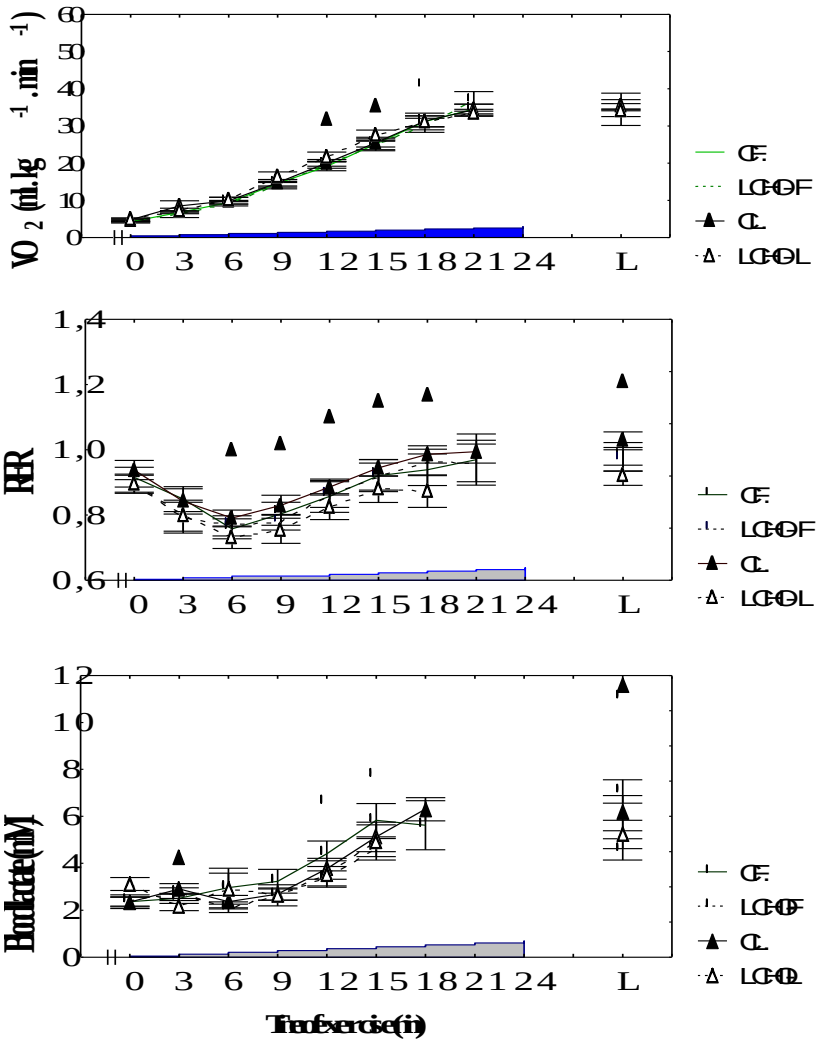

Fig. 1
Oxygen uptake, respiratory exchange ratios and blood lactate concentrations, in women exercising after control (C) and low carbohydrate (LCHO) diet during follicular and luteal phase of the menstrual cycle

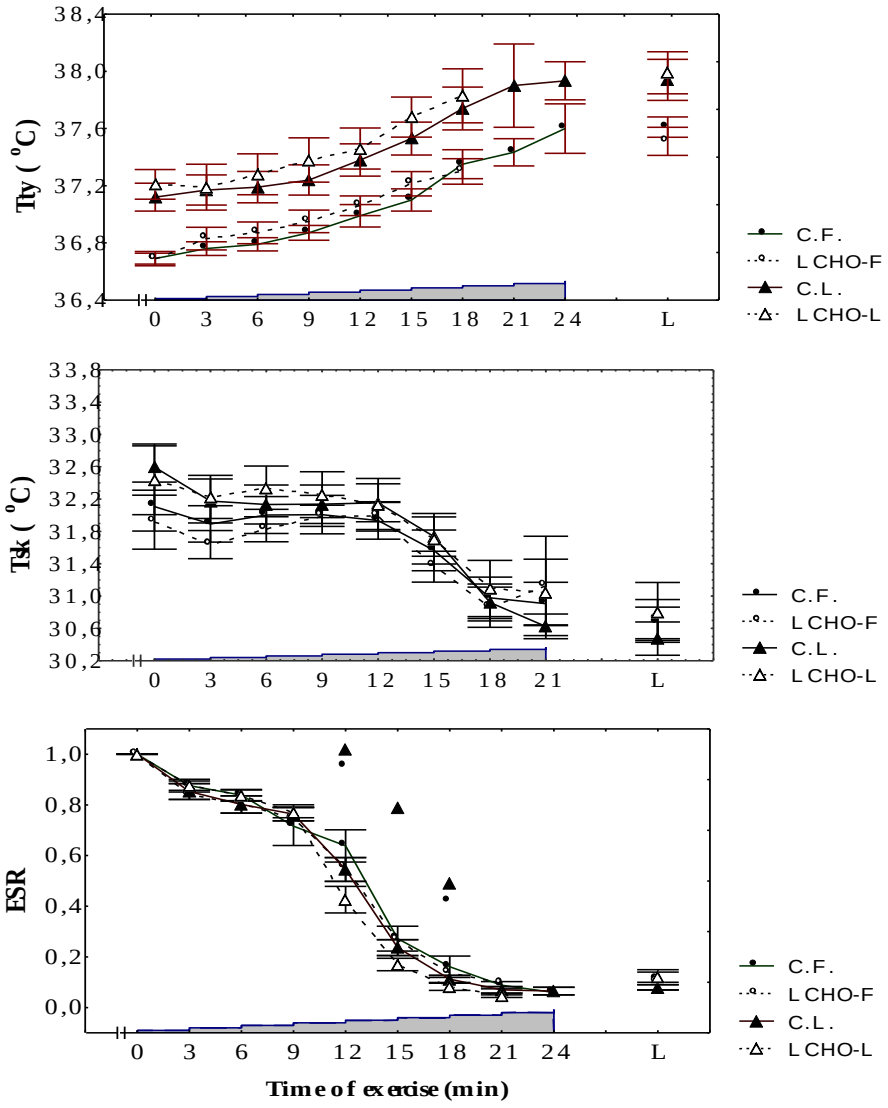

Fig. 2
Changes in tympanic temperature, mean skin temperature and electrical skin resistance (ESR) after (C) and (LCHO) diet in women exercising during follicular (F) and luteal phase of the menstrual cycle

Metabolic heat production increased with the work load in both groups of subjects. However, during last minutes of exercise the metabolic heat production was greater in subjects during the phase L after LCHO diet. No diet related differences were observed between the rates of dry (R+C), respiratory (C_{res} +E_{res}) and wet (E_{dif}) heat loss.

Mechanical work efficiency (WE%) dropped significantly faster in L than in F, although the overall level did not significantly differ between L and F after C and LCHO diet. WE% was significantly lower during exercise test after LCHO diet but only in women during the luteal phase of the menstrual cycle (Fig. 3).

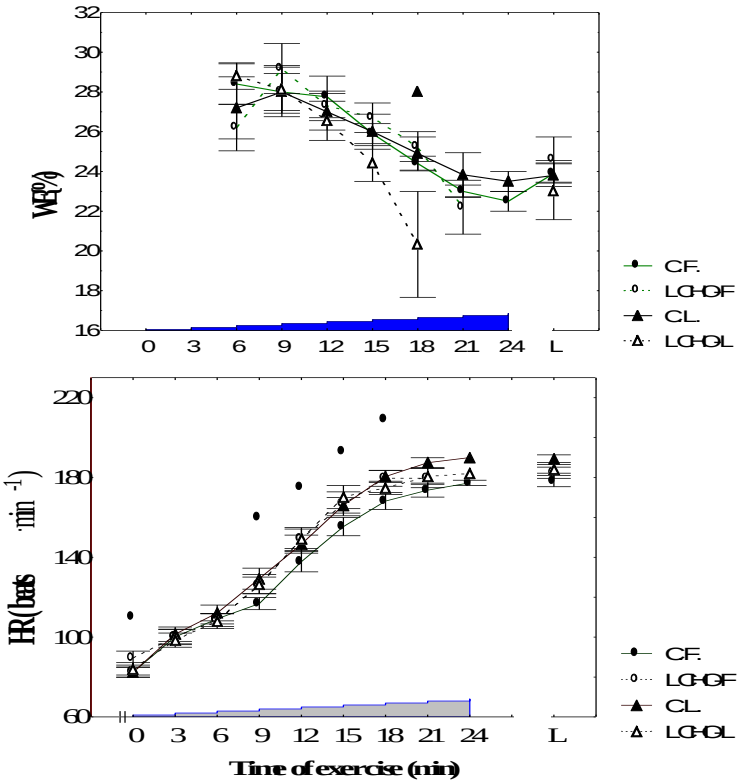

Fig. 3
Time course of heart rate (HR) and net efficiency of work (WE%) during graded exercise test in women during phase F and phase L, after control C and low-carbohydrate diet (LCHO); values presented as means ±SD; Asterisks indicate significant differences between F and L for each exercise period

Electrical skin resistance (ESR) decreased exponentially during both experimental situations (Fig. 2). The delay in the onset of ESR was markedly shorter after the LCHO than after C diet in women during both F and L (Table 3). There was no difference between the diets in the time constant (τ) of ESR. However, the inertia time (t_i) of ESR was significantly shorter after the LCHO than after the control diet. In both series of experiments the time course of changes in ESR exhibited a biphasic character with a faster time constant during the first phase ($\tau 1$) after LCHO than C diet. No diet related difference in time constant of sweating were detected in second phase ($\tau 2$) of this reaction (Table 3).

Table 3
The parameters describing the dynamics of sweating: t_a - a delay time, τ - time constant and t_1 - inertia time

	Follicular phase			Luteal phase		
	C	LCHO	Level of sign	C	LCHO	Level of sign
td (min)	10.8±1.6	9.4±1.8	P<0.05	9.9±1.4	9.3±09	NS
τ (min)	4.1±1.8	3.6±0.9	NS	3.9±1.1	3.5±1.8	NS
ti (min)	14.9±2.3	13.0±1.7	P<0.05	13.8±1.1	12.8±0.9	P<0.05
$\tau 1$ (min)	4.4±1.6	3.6±1.9	NS	3.9±2.4	3.7±1.8	NS
$\tau 2$ (min)	4.8±1.8	5.7±1.7	NS	5.1±1.6	4.6±1.5	NS
ΔT_{ty} (^oC)	0.9±0.2	0.8±0.3	NS	0.9±0.3	0.8±0.2	NS
ΔT_{sk} (^oC)	-1.5±0.7	-1.2±1.1	NS	-1.8±0.4	-1.5±1.0	NS
Thty	36.91±0.3	36.94±0.2	NS	37.22±0.3	37.37±0.4	NS
Time to exhaustion (min)	20.7±2.62	18.9±1.45	NS	20.4±2.37	18.6±3.1	P<0.05

T_{ty} threshold for initiation of sweating during exercise was shifted to a higher temperature in the luteal phase of the menstrual cycle in each exercise tests (Table 3). Type of diet did not alter the temperature threshold for sweating at any phases of the menstrual cycle. There were no differences between the phases of the menstrual cycle in slopes of the 1-ESR/T_{ty} relationships, but the slopes were greater after LCHO than C diet independently on the phase of the menstrual cycle.

Resting HR was unaffected by the diet but differed in relation to the menstrual cycle. Type of diet modified HR significantly in women for phase F. During exercise HR was about 10 beats·min^{-1} greater in women during the phase F exercising after the LCHO diet. No diet-induced differences in HR after LCHO diet were observed during the phase L (Fig. 3).

Time to exhaustion was shorter after low carbohydrate diet in comparison with control diet in women during the L phase of the menstrual cycle (Table 2).

Discussion

This study examined the combined effect of a low-carbohydrate diet and the phase of the menstrual cycle on thermoregulatory responses to exercise in women. It can be generally concluded that low-carbohydrate diet influence on the regulation of body temperature in exercising women. Phase of the menstrual cycle in women alters body temperature threshold for sweating, whereas the type of consumed diet modifies sensitivity of this reaction. The results indicate further that dietary induced changes in body temperature regulation during exercise were greater during the follicular than luteal phase. Progesterone was probably the significant factor limiting dietary induced changes in heat dissipation mechanisms activated after low-carbohydrate diet.

The sources of fuel for energy metabolism at rest and during exercise can be greatly altered by diet, phase of the menstrual cycle as well as the intensity and duration of exercise [3,24]. The present finding that the short term consumption of the low-carbohydrate diet accelerates heat loss by sweating in exercising women are in agreement with our earlier results observed in men [40,41].

Since energy costs of utilisation of fat and carbohydrates are different [51] the energy expenditure during exercise could be related to different types of diet. The mean exercise VO_2 was found to be greater in the same subjects after the LCHO diet than after the control, mixed diet. Several factors might contribute to the VO_2 shift after LCHO diet e.g. elevation in circulation heat liberating hormones as. catecholamines [31] and an increased contribution of lipid substract to energy metabolism. Increases in oxygen consumption may reflect a greater reliance on FFA oxidation -increased energy from aerobic metabolism [35]. The low-carbohydrate diet applied in this study probably caused a greater contribution of fat in the oxidative metabolism. This can be concluded from lower RER and LA values observed in exercising women. Similar metabolic effects related to LCHO-diet was previously observed in men by Langfort *et al.* [31] and Hargreaves *et al.* [25].

Some studies have shown that alteration in dietary carbohydrate intake can affect high intensity exercise performance [21]. The difference in substrate utilization after LCHO diet may influence performance during exercise altering the availability of muscle glycogen [2,36]. The results of this study indicated that female's ability to perform exercise does not differ at follicular and at luteal phase when consumed control mixed diet, but after LCHO diet the time to exhaustion was shorter in all women. Bailey *et al.* [1] showed that glucose consumption in exercising women was directly related to the intensity of exercise but it was not the case for FFA use. Because in the present study intensity of exercise increased, CHO metabolism might become more important to energy production than FFA oxidation. Young and Davies [55] clearly showed that the muscle weakness produced by dynamic exercise was greater under low-CHO than normal diet. It is quite possible that under the conditions of the present study fat oxidation was increased during exercise after LCHO diet with decreased fatigue resistance and exercise performance.

The respiratory exchange ratio as well as LA blood concentrations were unchanged at rest, submaximal and maximal exercise in relation to cycle of the menstrual phase after control diet. However, after consuming the LCHO diet RER was significantly lower in women during phase luteal. A significantly lowered RER values in subjects on LCHO diet indicate that contribution of fat oxidation to energy metabolism was enhanced when carbohydrate stores were limited in presence of high blood estrogens and progesterone concentrations.

The higher VO_2 uptake observed under LCHO condition was connected with slightly elevated metabolic heat production in women exercising after the LCHO diet. The energy cost of utilizing energy substrate was higher under LCHO than in C conditions and it was reflected by lower WE% values and higher heat production in women exercising during L phase. This finding also suggests that endogenous heat load during exercise was greater after LCHO than after C diet in phase L of the menstrual cycle.

The increased heat production during exercise after LCHO diet would result mainly from activation of the β-adrenoceptor-mediated mechanisms leading to increased oxidation of free fatty acids [48]. However, the role of progesterone in the hormonal and metabolic responses to exercise in women is unclear. In the present study greater production of heat was observed after LCHO diet in women during the luteal phase but not during the follicular phase. It appears that progesterone significantly lowered efficiency of substrate energy metabolism in LCHO condition and the greater energy was released as a heat product. It might be

suggested that the rise of M and H after LCHO diet in phase L has been attributed to the putative thermogenic effect of progesterone.

Observed differences in sweating response in relation to the phase of the menstrual cycle were mainly attributed to greater body temperature threshold for sweating and slightly shorter delay time of sweating in luteal than follicular phase of the menstrual cycle in both experimental situations. These results confirm previous studies [23, 39] that increased resting body temperature during the luteal phase is related to a higher body temperature threshold for the onset of sweating during exercise. Elevation of body temperature threshold controlling the onset of sweating in this phase is mediated by high level of progesterone [5] or by it metabolites [12].

The phase of the menstrual cycle in women influenced on sweating threshold shifting it to the greater values during the luteal phase independently on the type of consumed diet. On the other hand, the type of the diet significantly modified sweating sensitivity. It would be expected that menstrual phase and diet would produce systematic changes in control of thermoregulatory responses. Thermoregulatory control is typically evaluated by examining the effector responses (e.g. sweating) to dynamic changes in body temperature [11,12,19].

It is generally accepted that sweating increases linearly with rise in the internal body temperatures. The relationship between sweating rate and the internal body temperatures shifts to the left with a rise in skin temperature. On the other hand, the sweating can increase without any changes in the internal body temperatures or skin temperature [30,54]. This response indicate that changes in sweating may be due to some non-thermal factors involving activation of mechanosensitive [19,20,29] or metabolosensitive [30] receptors in the exercising muscles, central command signals linked with volitional effort and emotional or mental stimulation.

A comparison of the time course of electrical skin resistance during exercise in both dietary conditions revealed that the LCHO diet enhanced the dynamics of sweating by shortening the delay time and inertia time of this reaction after LCHO diet in both phases of the menstrual cycle in women. These facts indicate that sweating was significantly faster in the LCHO than in C diet in all tested women.

The results obtained in the present study indicated that the slope of relationships 1-ESR to tympanic temperature was not affected by the phase of the menstrual cycle but it was affected by the type of consumed diet. Sweating sensitivity as well as the time course of this reaction was slightly greater during LCHO than during C diet. Differences in sweating response in both experimental situations support the view, that the set-point temperature for activation of sweating was unchanged by the type of consumed diet in women.

It is possible that sweat glands and cutaneus vessels are influenced by increased symphatetic activity, different in both examining diets and probably dependent on the phase of the menstrual cycle [18,28]. Most probably the same neurohormonal effects during given type of diet might modify efferent outflow from the hypothalamus to thermoregulatory effectors, resulting in changes in dynamics of sweating and in the changes in skin blood flow.

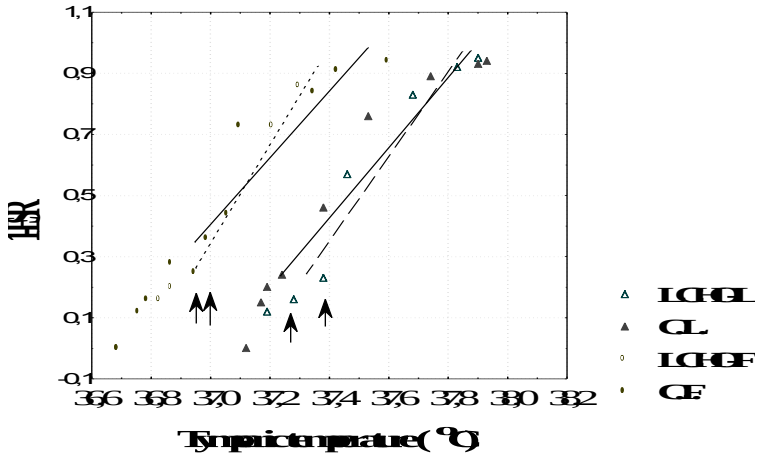

Fig. 4
The relationships of changes in Tty vs 1-ESR during exercises in F and L phase of the menstrual cycle in women after C and LCHO diet (T_{Ty} - body temperature threshold for sweating)

LCHO diet had a significant effect on cardiovascular system, increasing HR at rest and during exercise in women during follicular phase of the menstrual cycle. Similar results were observed in men [40,41] and after 2-deoxy-D-glucose infusion [8] as well as after insulin induced hypoglycaemia [16]. Subject's HR values increased gradually over time of exercise in both phases of the menstrual cycle. HR averaged about 10 beat·min^{-1} greater during exercise performed in luteal than in follicular phase but only under control diet. These elevated HR values found both before and during exercise in the L phase may be related to decreased plasma volume or to increased capillary permeability or other cycle differences [37]. It is known that plasma volume varies during the menstrual cycle with a peak around ovulation. Many previous studies support the concept that estrogens alter body

fluid balance due to a lower body water content and more restricted water movement in the F women. Friedlander *et al.* [13] reported a 25% reduction in blood volume associated with falls in E_2 concentration in ovariactomized women. Vellar *et al.* [53] reported significantly lower hematocrit and haemoglobin concentration in women during the postovulatory (high estrogens) than during the early preovulatory (low estrogens) phase of the menstrual cycle. This estrogenic effect on changes in plasma volume may be detected with, and without, elevation in blood progesterone. Stachenfeld *et al.* [44,45] suggested that progestins administration may down-regulate estrogen receptors. High level of estrogens leads to expand of plasma volume and it interacts with systems that regulated the volume and osmotic pressure of the extracellular fluid [3,10,49]. During the menstrual cycle, after the control diet, high estrogen level in the blood was associated with plasma volume expansion and lowered HR in women during follicular phase of the menstrual cycle.

Table 4
Threshold for thermoregulatory sweating (ThTty) and the sensitivity (slope) of sweating reactions (Tty/1-ESR)

	Control diet		Low carbohydrate diet	
	Follicular phase	Luteal phase	Follicular phase	Luteal phase
Tty/1-ESR	1.1	1.07	1.52	1.3
ThTty	36.91±0.25	37.22±0.33	36.94±0.24	37.37±0.49

These differences in HR were not present after LCHO diet. Stephenson and Kolka [46,47] showed that in exercising women plasma volume declined slightly faster in the follicular than in luteal phase. It seems likely that after ovulation resting plasma volume and dynamics of water movement was reduced in comparison to the follicular phase [14]. Results of the previous works suggest that after LCHO diet, in presence of high level of progesterone, more water were conserved in the body [38,42]. In consequence dietary induced hypovolemia in women would be greater in F women [42].

Finally, it might be concluded that reduced availability of carbohydrates as an energy source for working muscles accelerates heat loss by sweating in exercising women. This mechanism may be profitable for an organism preventing exercise hyperthermia, which might have developed as a result of the enhanced heat production caused by greater utilization of lipids.

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