

Title: Effect of the menstrual cycle on performance of intermittent, high intensity shuttle running in a hot environment

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Abstract

The present study examined the impact of the menstrual cycle and oral contraceptive use on performance of high intensity intermittent running in the heat [31.0 (0.2) °C; 23.1 (0.9) % RH]. Seven normally menstruating women (NM) and 8 oral contraceptive (OC) users participated in the study. Two trials were undertaken near the predicted mid-point of the follicular (FT) and luteal (LT) phases of the menstrual cycle and the equivalent days for the oral contraceptive users. Basal serum progesterone concentrations were higher during the LT for the NM group [FT: 2.42 (0.28) vs. LT: 25.96 (11.28) nmol.l⁻¹; P<0.05], but were not different for the OC [1-14: 2.79 (0.38) vs. 15-28: 2.61 (0.32) nmol.l⁻¹]. There were no differences in distance run between menstrual cycle phases or between the normally menstruating and oral contraceptive groups [NM FT: 6257 (1401) vs. LT: 5861 (1035) m]. However the OC ran further in the days 15-28 compared to days 1-14 [OC 1-14: 5481 (612) vs 15-28: 6615 (893) m, P<0.05]. For the NM, rectal temperature, perceived exertion, estimated SR, serum growth hormone, plasma lactate, ammonia and glucose did not differ between phases of the menstrual cycle. For the OC, heart rate, perceived exertion, sweat rate, plasma lactate and ammonia did not differ between days 1-14 of oral contraceptive use and days 15-28. However rectal temperature was higher (P<0.05) and growth hormone tended to be higher (P=0.05) during days 15-28, while plasma glucose was lower (P<0.05). These results demonstrate that for unacclimatised games players the performance of intermittent, high intensity shuttle running in the heat is unaffected by menstrual cycle phase but is influenced by oral contraceptive use.

Keywords: Women, Oral contraceptives, Intermittent exercise, Growth hormone

Introduction

Cyclic variations in deep body temperature during the menstrual cycle may alter thermoregulatory responses, and thus possibly performance, when exercising in the heat. In moderate environmental conditions the performance of continuous (90% $\dot{V}O_2$ max) and progressive high intensity running was unaffected by menstrual cycle phase (Lebrun et al. 1995; Lynch and Nimmo 1998). However, very few studies (Horvath and Drinkwater 1982; Carpenter and Nunneley 1988; Stephenson and Kolka 1988) have examined performance in the follicular and luteal phases of the menstrual cycle in the heat, despite numerous international competitions being organised in hot climates. From the studies that have been undertaken it was concluded that cycling and walking performance were unaffected by menstrual phase. In addition, it has been shown that during sub-maximal cycling in the heat for acclimatised subjects, deep body temperature may be higher during the luteal compared with the follicular phase (Carpenter and Nunneley 1988). For unacclimatised subjects reports are controversial, with deep body temperature reported to be higher (Stephenson and Kolka 1988) or unaffected by menstrual phase (Wells and Horvath 1974; Horvath and Drinkwater 1982).

Furthermore, no study has examined the metabolic and hormonal responses to exercise in the heat during the follicular and luteal phases of the cycle. In moderate environmental conditions, for sub-maximal cycling and treadmill running, blood glucose was similar in the follicular and luteal phases (Bonen et al. 1983; Kanaley et al. 1992; Zderic et al. 2001) whereas, for moderate intensity cycling and running, blood glucose has been shown to be lower (Lavoie et al.

1987) or higher during the luteal phase (Galliven et al. 1997; Zderic et al. 2001). Again in moderate environmental conditions, growth hormone responses to cycling and treadmill walking were higher during the luteal and ovulatory phases of the cycle compared with the follicular phase (Bonen et al. 1983; Hornum et al. 1997; Zderic et al. 2001). High growth hormone concentrations have been associated with the high estrogen concentrations during the luteal and ovulatory phases (Hornum et al. 1997).

Many women mask their normal cyclical hormonal changes during the menstrual cycle by taking oral contraceptives. However, only two research groups have investigated the responses of oral contraceptive users to exercise in the heat (Martin and Buono 1997; Stachenfeld et al. 2000). During prolonged moderate cycling when taking oral contraceptives, there was a higher heart rate and rectal temperature response compared with the same exercise during a contraceptive free week (Martin and Buono 1997). Furthermore, again during cycling, when women were taking a progestin only contraceptive, oesophageal temperature and sweating threshold were higher than when the women were taking a combined estrogen and progestin contraceptive (Stachenfeld et al. 2000). In moderate environmental conditions during moderate and high intensity treadmill exercise, oral contraceptive users had higher growth hormone and lower glucose concentrations than eumennorheic women (Bonen et al. 1991; Bembien et al. 1992). However, no study has examined the performance of oral contraceptive users throughout the substitutive therapy cycle in moderate or hot environmental conditions.

A high deep body temperature has been suggested to be a limiting factor in exercise performance in the heat (Nielsen et al. 1990, 1993; Gonzalez-Alonso et al. 1999). If during the luteal phase or taking oral contraceptives, a higher deep body temperature is observed during exercise, this may decrease the time to exhaustion.

Therefore, the purpose of the present study was to test the hypothesis that the performance of high intensity, intermittent running exercise in the heat is unaffected during the menstrual cycle, or by the use of oral contraceptives. In addition, the responses of the eumenorrheic women during the normal menstrual cycle were compared with those of monophasic oral contraceptive users, across a substitutive cycle, before day 14 and after day 14, since the performance, hormonal and metabolic responses have not been studied to date.

Methods

Participants

Fifteen well-trained female games players volunteered for the study, seven of whom had normal menstrual cycles (NM) lasting between 24 and 30 days, and eight who had been taking monophasic oral contraceptives (OC) for a mean (SE) of 22 (6) months. The age, mass and height of the NM and OC groups was 20.3 (0.3) and 20.2 (0.4) years, 62.1 (2.3) and 59.8 (1.1) kg and 168.4 (2.0) and 165.6 (1.9) cm respectively. The estimated $\dot{V}O_2$ max for the NM group was 51.1 (0.7) ml.kg⁻¹.min⁻¹ and for the OC group was 50.3 (1.6) ml.kg⁻¹.min⁻¹. All subjects gave their written informed consent and the Loughborough University Ethical Committee approved the study. The oral contraceptives taken by the participants in the OC group are described in Table 1.

Table 1

Terminology

The group with normal menstrual cycles will be referred to as NM and the oral contraceptive users OC. For the normal menstruating group the follicular trial will be abbreviated to FT and the luteal trial to LT. Monophasic oral contraceptive users do not have follicular and luteal phases per se, thus first 14 days of the oral contraceptive cycle will be termed days 1-14 and the second 14 day period as days 15-28 of oral contraceptive use.

Experimental design

Subjects performed the Loughborough Intermittent Shuttle Test (LIST; Nicholas et al. 1995, 2000) in hot environmental conditions (31.0 (0.2) °C, 23.1 (0.9) %

RH). The mean difference \pm 95% limits of agreement for the LIST is -0.7 ± 5.6 min. For the NM, two main trials were completed during the follicular phase, ~ 7 days after the onset of menstruation and in the luteal phase, \sim day 21. For the OC, two trials were completed at the equivalent days. The order of trials was randomly assigned and 14 days elapsed between the follicular (FT) and luteal trials (LT) for the normal menstruating (NM) women and between trials for the oral contraceptive (OC) users. Subjects exercised over a 20 m distance and repeated a walk, sprint, cruise ($\sim 95\% \dot{V}O_2 \text{ max}$) and jog ($\sim 55\% \dot{V}O_2 \text{ max}$) pattern of exercise until 11 sprints had been completed. The 11 sprints took approximately 15 min and were followed by a 3 min rest period. This series of activities constituted 1 set of the LIST. This pattern was repeated until exhaustion, or rectal temperature reached 39.5°C .

Preliminary measurements

Maximal oxygen uptake ($\dot{V}O_2 \text{ max}$) was estimated using the progressive multistage fitness test (Ramsbottom et al. 1988). From this estimate of $\dot{V}O_2 \text{ max}$, running speeds to elicit 95 and 55% $\dot{V}O_2 \text{ max}$ were determined. Volunteers were then familiarised with the LIST at $\sim 30^\circ\text{C}$ for 2 sets or ~ 30 min.

Main trials

Subjects reported to the laboratory at least 12 h after their last meal. In the 2 days prior to each trial subjects were encouraged to consume the same diet. All experiments were arranged so that each individual ran at the same time of day for

both trials to control for circadian influences. Nude body mass was recorded and a 45 mm cannula was then inserted into a forearm vein of the subject under local anaesthetic (Lignocaine hydrochloride 1% w/v). The cannula was kept patent with saline solution (Sodium chloride 0.9%). A rectal probe (Edale Instruments Ltd.) was inserted to a depth of 10 cm beyond the anal sphincter.

Fifteen minutes after cannulation, during which time subjects remained standing, a resting blood sample was collected. Subjects then moved into the gymnasium and a resting rectal temperature was recorded. A standardised warm-up of ~15 min was performed which consisted of jogging, stretching and faster pace running. During the warm up and throughout the exercise period subjects were encouraged to drink water to ensure adequate hydration levels.

During the sprints subjects were verbally encouraged to perform maximally. Sprint times over 15 m were measured using 2 infra-red photo electric cells connected to the microcomputer. Heart rate was continuously monitored throughout each test using short range telemetry (Sport Tester™, Polar Electro Fitness Technology). Rating of perceived exertion was recorded prior to the 11th sprint in each exercise set using the Borg scale (1962). A 10 ml blood sample was collected from each subject between the sets of exercise and at exhaustion. Rectal temperatures were measured during the 4th and 8th cycle of each set and in the 3 min blood sampling period between sets of LIST. When rectal temperatures were measured subjects were stationary for 40 m of the 60 m walk in that cycle.

At exhaustion, after the participant had stopped sweating and wiped any excess

sweat off the skin, nude body mass was again recorded. Sweat rates were estimated from pre- and post-exercise nude body mass measurements correcting for fluid intake. It was assumed that 1 litre of water was equivalent to 1kg

Blood sampling and analyses

Five ml of blood was dispensed into an EDTA tube and aliquots from the venous sample were used for determination of haematocrit and haemoglobin concentration (by microcentrifugation and the cyanmethaemoglobin method [Boehringer Mannheim UK] respectively). Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974). One ml of blood was dispensed immediately into a calcium-heparin tube, centrifuged for 3 min at 13,000 rev.min⁻¹ (11,337 g) and the plasma frozen at -70°C. Ammonia concentration was determined within 24 h using a commercially available kit (Sigma Diagnostics). The remaining blood was centrifuged for 15 min at 6000 rev.min⁻¹ (2415 g) at ~3°C. The resulting plasma was then stored at -20°C for subsequent determination of lactate and glucose using fully automated colorimetric instrumentation (Cobas Mira, Roche Products Ltd.).

Five ml of blood were also dispensed into a serum tube for determination of progesterone and growth hormone concentration by using a commercially available radioimmunoassay kits (Diagnostic Products Corporation). The progesterone assay has a sensitivity of 0.06 nmol.l⁻¹, an intra-assay coefficient of variation (cv) of 2.7-8.8% and an inter-assay cv of 3.9-9.7%. The growth hormone assay has a sensitivity of 1.8 mIU.l⁻¹, an intra-assay cv of 1.5-5.9% and an inter-assay cv of 3.4-8.3%.

Statistical analyses

Statistical analyses were undertaken on the NM and OC groups independently to allow comparisons between trials. The physiological and blood responses to the performance of the LIST were analysed using a two-way analysis of variance (ANOVA; trial x time) with repeated measures on one factor (time). Significant differences between means were identified using a Tukey post-hoc test. Environmental temperatures, distance covered during the LIST, body mass and plasma volume responses during the main trials were analysed using a students t-test. Comparisons between groups and trials were also undertaken. The physiological and blood responses to the performance of the LIST were analysed using a three-way analysis of variance (ANOVA; group x trial x time) with repeated measures on two factors (trial x time). Environmental temperatures, distance covered during the LIST, body mass and plasma volume responses during the main trials were analysed using a two-way ANOVA (group x trial) with repeated measures on one factor (trial). Significant differences between means were identified using a Scheffè post-hoc test. Data are presented as means (SE) and are based on a subject population of 7 NM and 8 OC unless otherwise stated.

Results

Performance

The distance run during the Loughborough Intermittent Shuttle Test (LIST) was not different between groups, all cycle considered [NM: 6059 (896) vs. OC: 6048 (543) m]. It did not differ either when comparing the luteal and follicular phases of the menstrual cycle [FT: 6257 (1401) vs. LT: 5861 (1035) m]. In contrast, the oral contraceptive group ran further during days 15-28 [1-14: 5481 (612) vs 15-28: 6615 (893) m, $P<0.05$]. Maximal 15 m sprint performance did not differ between groups and the decline in performance was similar both between groups and between trials (main effect time $P<0.01$, Fig. 1).

Fig. 1

Rectal temperature

The resting rectal temperatures tended to be higher prior to the LT than the FT, [Rest, FT: 37.1 (0.1) vs. LT: 37.2 (0.1) °C, n.s., Table 2a] and was higher for the OC during days 15-28 than 1-14 [1-14: 37.3 (0.1) vs 15-28: 37.4 (0.0), $P<0.05$, Table 2b]. Rectal temperature increased during exercise but was not different between the FT and LT [main effect time $P<0.01$, Table 2a] and increased at a similar rate [FT: 3.36 (0.50) vs LT: 3.38 (0.54) °C.h⁻¹]. For the OC rectal temperature increased throughout the exercise period [main effect time $P<0.01$] and was higher during days 15-28 [main effect trial $P<0.01$, Table 2b]. The rate of rise of rectal temperature for the OC was higher during days 1-14 [1-14: 2.78 (0.35) vs 15-28: 2.40 (0.31) °C.h⁻¹, $P<0.05$]. Resting rectal temperature was higher for the OC than the NM being 37.4 (0.1) °C and 37.2 (0.1) °C respectively ($P<0.05$). The difference observed at rest between the OC and NM was not maintained after the onset of exercise. The rate of rise of rectal temperature

showed no differences between the two groups [NM: 3.4 (0.4) vs. 2.6 (0.2) °C.h⁻¹].

Table 2

Heart rate and rating of perceived exertion

Heart rate increased with exercise time for both the NM and OC groups (main effect time P<0.01). Heart rate was unaltered by menstrual phase or oral contraceptive status and did not differ between the groups. Table 3 shows that rating of perceived exertion (RPE) increased with exercise time (P<0.01) and was not different between menstrual phase or oral contraceptive use.

Table 3

Body mass, fluid consumption and estimated sweat rate

Body mass was well maintained by both groups during the two trials. The change in body mass as a percentage of basal body mass for the NM was -0.13 (0.24) and 0.06 (0.16) % for the FT and LT respectively and for the OC was -0.51 (0.10) and -0.25 (0.22) %. Ad libitum fluid consumption was not different between the FT and LT [NM FT: 19.6 (4.4) vs. LT: 17.1 (2.6) ml.kg⁻¹.h⁻¹] or between trials for the OC [OC 1-14: 16.1 (1.65) vs. 15-28: 16.8 (2.6) ml.kg⁻¹.h⁻¹]. Similarly there were no differences in fluid consumption between groups. Estimated sweat rate was not different between menstrual phase [NM FT:1.30 (0.07) vs LT: 0.99 (0.15) l.h⁻¹] or days 1-14 and 15-28 for the OC [OC 1-14: 1.41 (0.13) vs 15-28: 1.27 (0.09) l.h⁻¹]. There was no difference between the two groups.

Hormonal responses

Resting serum progesterone concentrations for the NM were greater for the LT than the FT [P<0.05; FT: 2.42 (0.28) vs. LT: 25.96 (11.28; range 6.7-72.8)

nmol.l⁻¹], confirming the normality of the menstrual cycle and ovulation for this group. In contrast, progesterone concentrations were the same throughout the cycle for the OC group [1-14: 2.79 (0.38) vs. 15-28: 2.61 (0.32) nmol.l⁻¹] confirming the inhibition of ovulation. Serum growth hormone concentrations for the NM increased with the onset of running and were not different between menstrual cycle phases [main effect time P<0.05; Table 4a], but tended to be higher for the OC during days 15-28 [main effect trial P=0.05, main effect time P<0.01; Table 4b]. However, the concentrations were not different between the groups.

Table 4

Metabolic Responses

For the NM both plasma lactate and ammonia responses did not differ between menstrual cycle phases, (main effect time P<0.01, Table 5). Similarly for the OC plasma lactate and ammonia did not differ between days 1-14 and 15-28 (main effect time P<0.01, Table 5). For the NM plasma glucose concentrations were unaffected by menstrual phase [NM FT End: 9.6 (1.2) vs LT End 8.3 (0.9) mmol.l⁻¹; main effect time P<0.01]. For the OC plasma glucose concentration was higher during days 1-14 than days 15-28 (main effect trial P<0.05, main effect time P<0.01; Fig. 2).

Table 5

Fig. 2

Plasma volume

Estimated resting plasma volume was not different between the FT and LT for the NM [FT: 62.2 (1.1) vs LT: 61.9 (1.3) ml.100ml⁻¹]. However resting plasma volume for the OC was higher during the days 15-28 than 1-14, [1-14: 61.0 (1.1) vs. 15-28: 63.4 (1.3) ml.100ml⁻¹; P<0.05]. There were no differences in resting

plasma volume between the groups. The estimated change in plasma volume for the NM and OC groups was not different between trials. The decreases in plasma volume were not different between the two groups.

Discussion

The main finding in the present study was that in unacclimatised women, who were eumenorrheic, performance, in terms of distance run of prolonged intermittent high-intensity running was not significantly affected by the cyclic variations in hormones associated with normal menstrual function, whereas for oral contraceptive users performance was improved during days 15-28 when taking exogenous synthetic hormones compared to days 1-14. For the eumenorrheic group maximal sprint performance over 15 m, rectal temperature, plasma lactate, ammonia and glucose concentrations and serum growth hormone concentrations were also not different between the follicular and luteal phases of the menstrual cycle. However, for the oral contraceptive users plasma glucose concentration was higher and serum growth hormone concentrations were lower in days 1-14 in comparison with days 15-28.

The finding of the present study that the performance, rectal temperature, heart rate and perceived exertion of unacclimatised eumenorrheic women during intermittent running in the heat (31 ± 0.2 °C; $23.1 \pm 0.9\%$ RH) were unaffected by menstrual cycle phase is largely consistent with previous research examining other modes of exercise (Wells and Horvath 1974; Horvath and Drinkwater 1982). During walking in three different environments (28, 35 and 48°C) during the flow (follicular), ovulatory and luteal phases no differences in heart rate, deep body temperature, sweat rate or performance of the 50 min walk were observed

between the three phases (Horvath and Drinkwater 1982). In contrast in acclimatised eumenorrheic subjects cycling ($30\% \dot{V}O_2$) in the heat (48°C , 10% RH), rectal temperature was lower in the follicular in comparison with the luteal phase (Carpenter and Nunneley 1988) in good agreement with the well-documented effect of progesterone on basal body temperature. Similarly during cycling at night ($70\% \dot{V}O_2 \text{ max}$) (when the largest differences in deep body temperature between the luteal and follicular phase are observed) in moderate environmental conditions after a 4 h rest period in the heat, rectal temperature, heart rate, sweating thresholds and oxygen uptake were all lower during the follicular in comparison with the luteal phase (Hessemer and Brück 1985). Thus, in the present study, while the similar running performance in the follicular and luteal phases of the cycle is entirely consistent with previous cycling and walking studies, the lack of any effect on deep body temperature or cardiovascular responses may be explained by the unacclimatised status of the subjects and the intense nature of the exercise.

One exception to the consistency with previous literature in the present study is the lack of any difference in rectal temperature between menstrual phases for the eumenorrheic women prior to the onset of exercise. This may possibly be due to the intervening variables associated with travelling to the laboratory and the long preparation period (Wells and Horvath, 1973). The similar basal deep body temperatures between menstrual phases emphasises the need for a prolonged period of passive rest prior to the onset of exercise, to prevent the masking of the differences in deep body temperature associated with the follicular and luteal

phases of the cycle.

For the oral contraceptive users, performance was improved during days 15-28 compared to days 1-14. No previous study has examined performance per se during exercise in the heat in oral contraceptive users. However, during cycling for 1 h at 60% $\dot{V}O_2$ max in the heat (30°C, 50% RH) rectal temperature and heart rate were greater when subjects were taking oral contraceptives in comparison with a contraceptive free week, which the authors suggested might result in reduced capacity or performance in oral contraceptive users (Martin and Buono 1997). Similarly, Rogers and Baker (1997) recorded a higher rectal temperature and heart rate when taking the contraceptive pill compared with the no pill week during treadmill walking (22°C, 39% RH). In the current study rectal temperature was 0.1°C higher during days 15-28, whereas in the studies comparing pill use with the pill free week temperatures were 0.3°C higher (Martin and Buono 1997; Rogers and Baker 1997). Martin and Buono (1997) postulated that the high rectal temperature may diminish performance, however in the current study performance was actually improved. The difference of only 0.1°C clearly therefore did not have a negative effect on performance.

There was a higher plasma glucose concentration during the high intensity intermittent running during days 1-14 in comparison with days 15-28 in the present study, for the oral contraceptive group. Previous studies examining glucose concentration, and oral contraceptive use have yielded equivocal findings with increases, decreases and no change in glucose concentrations being recorded (Bonen et al. 1991; Kanaley et al. 1992; Bemben 1993; Galliven et al.

1997; Bailey et al. 2000). These ambivalent findings might be related to the intensity and type of exercise, the type of oral contraceptive, the time during the contraceptive month and whether the subjects are fasted. When the metabolic contribution required from gluconeogenesis is high there tends to be a higher glucose concentration for oral contraceptive users during the contraceptive free week or when compared to eumennorheic women (Lavoie et al. 1987; Bonen et al. 1991; Bemben 1993). During prolonged exercise, blood glucose is initially supplied from the breakdown of glycogen and as exercise progresses gluconeogenesis becomes more crucial. In the current study, the differences in glucose concentrations became greater towards the end of the exercise period. Thus, the lower glucose concentration during days 15-28 may be related to the higher exogenous ovarian hormones that may impair hepatic gluconeogenesis (Lavoie et al. 1987). The lower glucose response could also be due to a decrease in hepatic glucose output or an enhanced glucose uptake, utilisation and glycogen storage that has been shown with estrogen treatment in animals (Bemben 1993). Furthermore, oral contraceptives have been suggested to alter the secretion of glucoregulatory hormones and to decrease insulin sensitivity that suppresses gluconeogenesis as insulin concentrations have been shown not to differ (Bonen et al. 1991; Bemben 1993).

Growth hormone was higher during second 14 day period for the oral contraceptive users. Growth hormone has been reported to be higher both at rest and during exercise when taking oral contraceptives compared to the contraceptive free week and to eumennorheic women (Bonen et al. 1991; Bemben et al. 1992; Bemben 1993). This enhanced growth hormone response

may be due to estrogen stimulation or could be due to the lower glucose concentrations stimulating hypothalamic glucoceptors (Bemben et al. 1992). Growth hormone is associated with an enhanced lipolytic effect and thus has a glucose sparing effect (Bak et al. 1991; Moller et al. 1991), which may improve performance during prolonged exercise (Bemben et al. 1992; Bemben 1993). Total carbohydrate utilisation during 90 min of treadmill exercise (50% $\dot{V}O_2$ max) was lower in women taking oral contraceptives than women in the luteal phase of the cycle and this was suggested to be related to the higher growth hormone response in this group (Bemben et al. 1992). Thus in the present study the improved performance may be related to a glycogen sparing effect, however as free fatty acids, insulin and glucagon were not measured, this clearly requires further study.

A possible explanation for the improved performance during days 15-28 for the OC users is an enhanced running economy. Giacomoni and Falgairette (2000) compared responses to a 12 min submaximal treadmill run when off OC (days 2-4), early on OC use (days 7-9) and late on OC use (days 19-21). Oxygen uptake was lower during early and late OC use compared to off OC and running economy was improved in late OC use compared to off OC. It was postulated that this may be related to biomechanical factors rather than any alteration in substrate utilisation. Furthermore, Lynch and Nimmo (1998) observed a higher blood lactate and ammonia response to high intensity intermittent running within 1 week of taking the OC compared with 1 week later. Though this did not impact upon performance, it clearly emphasises the differences in metabolism that may occur within 1 contraceptive cycle.

The only difference between the groups was a higher resting rectal temperature in the OC group than in the eumenorrheic group. The progestin concentration in the oral contraceptives was high (>150 µg) and therefore the increase in basal body temperature, in line with progestin concentration, was not of a gradual nature as seen in the menstrual cycle, where progesterone is only produced during the luteal phase of the cycle, or in multiphasic oral contraceptive preparations, where progestins are only added during the second 14 days (second phase) of the cycle. Furthermore the potency of exogenous progestins has been shown to be greater than their endogenous counterparts (Bemben 1993). Thus exogenous progestins have a significant effect upon thermoregulation (Grucza et al. 1993; Rogers and Baker 1997). Both the high progestin concentration and potency of the oral contraceptives seem the likely explanation for the higher resting rectal temperatures observed.

The protocol employed in the present study was designed to allow comparison between groups primarily and as such the lack of specificity of the OC cycle is a major limitation. For example, some of the participants would have been taking OC for 1-2 days after the week off during the days 1-14 trial. This time has previously been associated with a feeling of nausea and may have diminished performance. However, taking account of the limitations in protocol design, there are clearly some important differences in terms of performance, and some possible mechanisms for these differences that require further investigation. Research needs to be undertaken during several specific time periods during the oral contraceptive month, which incorporates measurement of growth hormone,

glucoregulatory hormones, glucose and free fatty acids.

In summary, menstrual cycle phase did not affect performance in terms of distance run or 15 m maximal sprint time during prolonged intermittent high intensity shuttle running in the heat. However, performance was improved during days 15-28 compared to days 1-14 of the contraceptive month. This improvement in performance may be related to a glycogen sparing effect or a biomechanical improvement though this remains to be elucidated.

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Table 1 The type and active ingredients of the oral contraceptives.

Oral contraceptive	No.	Synthetic estrogens	Synthetic progesterones
Microgynon® 30	5	30 µg ethinylestradiol	150 µg levonorgestrel
Ovranette®	1	30 µg ethinylestradiol	250 µg levonorgestrel
Loestrin®	1	20 µg ethinylestradiol	1 mg norethisterone acetate
Dianette®	1	35 µg ethinylestradiol	2 mg cyproterone acetate

Table 2a Rectal temperatures ($^{\circ}\text{C}$) for the NM during the follicular and luteal trials; t = main effect time $P < 0.01$.

Subject No.	FT				LT			
	Rest	Post Warm up	Set 1	End	Rest	Post Warm up	Set 2	End
1	37.3	37.65	39.1	39.5	37.0	37.3	39.2	39.8
2	37.0	37.5	38.9	39.8	37.1	37.15	38.6	39.9
3	37.3	37.5	38.7	38.9	37.2	37.2	38.5	38.8
4	37.3	37.6	39.3	39.6	37.4	37.75	39.2	39.45
5	36.8	36.9	38	39.6	37.0	37.1	38.1	39.5
6	37.0	37.4	38.7	39.7	37.25	37.6	38.6	39.0
7	37.1	37.45	38.8	39.4	37.4	37.7	38.8	39.8
	5							
Mean	37.1	37.4	38.8	39.5	37.2	37.4	38.7	39.5
SE	0.1	0.1	0.2	0.1	0.1	0.1	0.2	0.2
					t			

Table 2b Rectal temperatures (°C) for the OC during the days 1-14 and 15-28 trials; T = main effect trial P<0.01; t = main effect time P<0.01.

Subject No.	Days 1-14				Days 15-28			
	Rest	Post Warm up	Set 1	End	Rest	Post Warm up	Set 2	End
8	37.3	37.4	38.4	39.0	37.4	37.6	38.6	39.3
9	37.25	37.4	38.95	39.7	37.5	37.7	38.9	39.7
10	37.5	37.9	39.1	39.0	37.45	37.7	39.0	39.1
11	37.2	37.2	38.0	39.4	37.4	37.4	38.4	39.0
12	37.3	37.65	38.5	38.8	37.35	37.6	38.5	39.4
13	37.25	37.6	38.9	39.4	37.3	37.7	38.9	39.4
14	37.6	37.8	38.9	39.75	37.6	37.9	39.1	39.75
15	37.1	37.1	38.6	39.3	37.3	37.3	38.45	39.2
Mean	37.3	37.5	38.7	39.3	37.4	37.6	38.7	39.4
SE	0.1	0.1	0.1	0.1	0.0	0.1	0.1	0.1
T, t								

Table 3a RPE for the NM during the first set and exhaustion sets of the LIST; t = main effect time P<0.01.

Subject No.	FT		LT	
	Set 1	End Set	Set 1	End set
1	12	20	12	15
2	10	13	11	17
3	13	19	15	19
4	13	17	14	20
5	12	15	12	19
6	16	19	17	20
7	13	17	11	19
Mean	13	17	13	18
SE	1	1	1	1
			t	

Table 3b RPE for the OC during the first set and exhaustion sets of the LIST; t = main effect time P<0.01.

Subject No.	1-14		15-28	
	Set 1	End Set	Set 1	End set
8	15	18	14	19
9	15	15	13	13
10	16	20	15	20
11	12	19	11	19
12	11	15	11	17
13	12	12	15	19
14	16	19	15	19
15	13	20	13	17
Mean	14	17	13	18
SE	1	1	1	1
			t	

Table 4a Growth hormone concentration (nmol.l⁻¹) for NM during the first set, second set and exhaustion sets of the LIST; t = main effect time P<0.05.

Subject No.	FT				LT			
	Rest	Set 1	Set 2	End	Rest	Set 1	Set 2	End
1	1.1	4.2	3.0	3.0	2.5	8.0	11.0	11.0
2	10.1	21.2	17.5	10.1	21.6	31.8	25.5	24.9
3	34.8	37.4	35.0	21.9	28.9	34.5	34.1	14.3
4	5.2	27.8	25.5	19.4	8.6	41.4	32.4	32.4
5	30.5	32.7	20.1	17.3	25.8	26.1	20.7	26.1
6	0.9	26.6	28.9	26.4	2.9	44.0	36.1	36.1
Mean	13.8	25.0	21.7	16.4	15.1	31.0	26.7	24.1
SE	6.2	4.7	4.5	3.5	4.8	5.3	3.9	4.0

t

Table 4b Growth hormone concentration (nmol.l⁻¹) for OC during the first set, second set and exhaustion sets of the LIST; T = main effect trial P = 0.05; t = main effect time P<0.01.

Subject	Days 1-14				Days 15-28			
No.	Rest	Set 1	Set 2	End	Rest	Set 1	Set 2	End
8	18.5	38.3	24.1	14.2	22.0	41.5	25.8	13.8
9	34.2	44.1	37.5	35.2	41.3	54.3	43.5	39.3
10	13.4	30.4	25.7	25.7	9.5	41.3	36.7	36.7
11	6.9	24.9	20.0	8.2	17.7	49.4	22.9	4.5
12	43.5	45.0	43.7	31.2	59.4	78.6	72.6	48.9
13	4.0	32.8	37.8	31.3	15.5	68.3	58.0	61.2
14	5.6	27.4	17.5	13.2	0.4	11.6	13.5	17.3
Mean	18.0	34.7	29.5	22.7	23.7	49.3	39.0	31.7
SE	5.8	3.0	3.8	4.0	7.6	8.2	7.9	7.7
T, t								

Table 5 Plasma lactate and ammonia concentrations for NM and OC at rest and exhaustion; t = main effect time P<0.01.

	Plasma lactate (mmol.l ⁻¹)		Plasma ammonia (μmol.l ⁻¹)	
	Rest	End	Rest	End
NM				
FT	1.14 ± 0.12	5.26 ± 0.72	16.0 ± 4.0	45.6 ± 8.0
LT	0.92 ± 0.08	5.40 ± 0.92	13.5 ± 2.2	56.5 ± 10.8
		t		t
OC				
1-14	1.16 ± 0.15	5.74 ± 0.82	20.7 ± 5.1	42.0 ± 10.2
15-28	1.13 ± 0.12	5.83 ± 0.97	17.1 ± 5.9	49.9 ± 6.5
		t		t

Legends

Fig. 1 Maximal 15m sprint times during the Loughborough Intermittent Shuttle Test (LIST); t = main effect time $P < 0.05$.

Blank columns for the first set of running for the 4 different groups of subjects. Dashed columns for the results obtained during the exhaustion set for the same 4 groups of subjects. NM FT: group of women with normal menstrual cycles, during their follicular phase. NM LT: group of women with normal menstrual cycles, during their luteal phase. OC 1-14: group of women on oral contraceptives, during the first 14 days of the substitutive cycle. OC 15-28: group of women on oral contraceptives, during the second 14 days of the substitutive cycle.

Fig. 2 Plasma glucose concentrations for the OC during the days 1-14 and 15-28 trials; T = main effect trial $P < 0.05$; t = main effect time $P < 0.01$.

Solid line with triangle: group of women on oral contraceptives, during days 1-14 of the substitutive cycle. Dashed line with cross: group of women on oral contraceptives, during the days 15-28 of the substitutive cycle.



