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## Physiological and metabolic responses of female games and endurance athletes to prolonged, intermittent, high-intensity running at 30°C and 16°C ambient temperatures

Accepted: 19 July 1999

**Abstract** Eight female games players (GP) and eight female endurance athletes (EA) ran intermittently at high-intensity and for prolonged periods in hot (30°C) and moderate (16°C) ambient temperatures. The subjects performed a two-part (A, B) test based on repeated 20-m shuttle runs. Part A comprised 60 m of walking, a maximal 15-m sprint, 60 m of *cruising* (90% maximal oxygen uptake,  $\dot{V}O_{2\max}$ ) and 60 m of *jogging* (45%  $\dot{V}O_{2\max}$ ) repeated for 75 min with a 3-min rest every 15 min. Part B involved an exercise and rest pattern of 60-s running at 100%  $\dot{V}O_{2\max}$  and 60-s rest which was continued until fatigue. Although the GP and EA did not respond differently in terms of distances completed, performance was 25 (SEM 4)% less (main effect trial,  $P < 0.01$ ) in the hot (HT) compared with the moderate trial (MT). Sprints of 15 m took longer to complete in the heat (main effect, trial,  $P < 0.01$ ), and sprint performance declined during HT but not MT (interaction, trial  $\times$  time,  $P < 0.01$ ). A very high correlation was found between the rate of rise in rectal temperature in HT and the distance completed [GP,  $r = -0.94$ ,  $P < 0.01$ ; EA ( $n = 7$ ),  $r = -0.93$ ,  $P < 0.01$ ]. Blood lactate  $[La^-]_b$  and plasma ammonia  $[NH_3]_{p1}$  concentrations were higher for GP than EA, but were similar in HT and MT  $[La^-]_b$ , HT: GP vs EA, 8.0 (SEM 0.9) vs 4.9 (SEM 1.1)  $mmol \cdot l^{-1}$ ; MT: GP vs EA, 8.0 (SEM 1.3) vs 4.4 (SEM 1.2)  $mmol \cdot l^{-1}$ ; interaction, group  $\times$  time,  $P < 0.01$ ;  $[NH_3]_{p1}$ , HT: GP vs EA, 70.1 (SEM 12.7) vs 43.2 (SEM 6.1)  $mmol \cdot l^{-1}$ ; MT: GP vs EA, 76.8 (SEM 8.8) vs 32.5 (SEM 3.8)  $\mu mol \cdot l^{-1}$ ; interaction, group  $\times$  time,  $P < 0.01$ . Ad libitum water consumption was higher in HT [HT: GP vs EA, 18.9 (SEM 2.9) vs 13.5 (SEM 1.7)  $ml \cdot kg^{-1} \cdot h^{-1}$ ; MT: GP vs EA, 12.7

(SEM 3.7) vs 8.5 (SEM 1.5)  $ml \cdot kg^{-1} \cdot h^{-1}$ ; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ]. These results would suggest that elevated body temperature is probably the key factor limiting performance of prolonged, intermittent, high-intensity running when the ambient temperature is high, but not because of its effect on the metabolic responses to exercise.

**Key words** Hot environment · Women · Training status · Intermittent exercise

### Introduction

The detrimental effects of high environmental temperatures on the performance of prolonged, submaximal exercise have been well established for men (Adams et al. 1975; Galloway and Maughan 1997; Suzuki 1980), but, there is a dearth of information about women. In addition, it has been shown that many sports (among them field hockey, rugby and soccer) require men and women to exercise maximally for short periods, lasting perhaps a few seconds, interspersed with longer spells of rest or lower intensity activity (Williams 1990). Recent studies have examined the physiological and metabolic responses to “maximal intermittent exercise” in temperate environments (Hamilton et al. 1991; Holmyard et al. 1988; Nicholas et al. 1995), but only one study has examined the responses to such intermittent running exercise in the heat (Morris et al. 1998).

A further area which has been little investigated is the effect of different types of training at moderate ambient temperatures on the responses to exercise in the heat. It has been shown that interval training at moderate temperatures improves the performance of a standard prolonged treadmill walking test in hot conditions in non-acclimatised, previously untrained individuals (Gisolphi and Cohen 1979). It has also been found that well-trained endurance runners show even better thermal tolerance (Gisolphi and Cohen 1979). It has been suggested that the thermal tolerance results from the

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adaptation of the body to core temperatures elevated for a sufficient duration by exercise at moderate temperatures (Gisolfo and Cohen 1979). It is not known whether intermittent activity at moderate temperatures, as typically undertaken by games players in the majority of their training sessions, can induce tolerance to exercise in the heat as effectively as the prolonged submaximal and interval training undertaken by endurance athletes.

The purpose of the present study was therefore to examine the performance, thermal and metabolic responses of women to a prolonged, intermittent, high-intensity shuttle running test (LIST), which provided a realistic representation of the physiological demands typical of the multiple sprint sports, in high (30°C) and moderate (16°C) ambient temperatures. A further aim was to investigate whether the responses differed between endurance athletes and individuals who were not exclusively endurance trained (games players).

## Methods

### Subjects

A group of 16 well-trained female athletes (8 games players, GP, and 8 endurance athletes, EA) volunteered for the study, which had the approval of Loughborough University Ethics Committee and all procedures were carried out in accordance with current UK laws. The physical characteristics of GP and EA are given in Table 1. All the subjects were fully informed of the demands and possible risks associated with participation in the study, and of their right to withdraw at any time. A signed statement of informed consent indicated each subject's understanding of this. A questionnaire completed prior to the study indicated that all the subjects fulfilled the selection criteria of having trained at least three times per week (usually 4–7 times), and if they were a GP, being involved in one match.

### Experiment design

The subjects performed a prolonged, intermittent, high-intensity shuttle running test (Loughborough Intermittent Shuttle Test, LIST) in two ambient temperatures: high (30°C) and moderate (16°C). The test was a modified version of LIST reported by Nicholas et al. (1995). All exercise took place over a marked 20-m distance in a gymnasium and comprised two parts. In part A the subjects repeated the walk, sprint, run (*cruise*) and jog pattern of exercise shown in Table 2. The pattern was repeated 11 times forming one set of exercise. Each set took 15.0 (SD 0.1) min to

**Table 1** Physical characteristics of the female games players (GP) and endurance athletes (EA) who participated in the study

	GP		EA	
	Mean	SEM	Mean	SEM
Age (years)	20.7	0.4	22.1	1.0
Height (cm)	165.3	2.0	167.1	1.8
Mass (kg)	61.6	1.6	56.4	1.7 <sup>b</sup>
Body fat (%)	24.0	1.3	21.0	1.7
Maximal O <sub>2</sub> uptake (ml · kg <sup>-1</sup> · min <sup>-1</sup> )	50.8	0.9	56.3	2.2 <sup>a</sup>

<sup>a</sup> $P < 0.05$ , <sup>b</sup> $P < 0.01$ , significantly different from GP

**Table 2** Indicates the pattern of activity followed by the subjects during part A of the prolonged, intermittent, high-intensity shuttle running test (a modified version of the Loughborough Intermittent Shuttle Test, LIST). This pattern was repeated 11 times forming one set of activity. During the 11th repetition only parts 1 and 2 of the pattern were performed; namely 3 × 20 m of walking immediately followed by a maximal 15-m sprint. The subjects then rested for 3 min during which blood samples and rectal temperature data were collected. This sequence of one set of exercise followed by 3-min rest was performed continuously five times during part A of the test.  $\dot{V}O_{2max}$  Maximal oxygen uptake

Set	Pace	Distance	Intensity
1	Walking	3 × 20 m	1.54 m s <sup>-1</sup>
2	Maximal sprint	1 × 15 m	Maximal speed
3	Recovery walk	~3 m	4-s duration
4	Running ( <i>Cruise</i> )	3 × 20 m	90% $\dot{V}O_{2max}$
5	Jogging	3 × 20 m	45% $\dot{V}O_{2max}$

complete. Five sets were completed in part A, each followed by 3-min rest. In part B the subjects ran for 60 s (at a pace equivalent to 100% of their predicted maximal oxygen uptake,  $\dot{V}O_{2max}$ ) and rested for 60 s, repeating this pattern until they were fatigued, or until rectal temperature reached 39.5°C, or until other exercise-related factors led the investigators to believe they were in distress. On these occasions the subjects were taken to a cool room. The pace at which subjects should run each 20-m distance was set using amplified audio signals generated from a microcomputer (BBC model B), to a pattern set by the exercise intensities required. The exercise intensities required were calculated from the  $\dot{V}O_{2max}$  value established during the preliminary measurements (see below).

### Preliminary measurements

A series of preliminary tests were completed prior to the main trials. A 16-min incremental speed-lactate test was used to determine the relationships between blood lactate concentration and oxygen uptake and treadmill running speed. The  $\dot{V}O_{2max}$  values were measured using an incremental, uphill treadmill test to fatigue according to Taylor et al. (1955) using the Douglas bag method. The  $\dot{V}O_{2max}$  was also estimated using a progressive multistage shuttle run test according to Ramsbottom et al. (1988). Individuals were also familiarised with LIST and were required to perform two sets of part A and at least one session of part B at 30°C which took at least 37 min to complete.

### Main trials

The subjects reported to the laboratory adjacent to the gymnasium on the morning of a main trial, 12 h after their last meal. All the experiments were carried out in the morning to minimize the influences of circadian rhythms. In the 2 days preceding their first main trial the subjects consumed their normal diets. This was recorded in terms of average food portions and they were required to consume the same diet during the 2 days prior to the second trial. The subjects were requested to refrain from intense physical activity 24 h before each trial. Having voided, nude body mass was recorded and a rectal probe (Edale Instruments Ltd., Cambridge, UK) was inserted to a depth of 10 cm beyond the anal sphincter. When rectal temperature was not being recorded the plug and lead connected to the probe was placed in a small bag, which remained around the subject's waist throughout the trial.

A 45-mm cannula (Venflon 2, BOC Ohemeda AB) was then inserted into an antecubital or forearm vein of each subject under local anaesthetic (Lignocaine hydrochloride 1% w/v, Antigen pharmaceuticals Ltd.). A similar site was used for each subject in both trials. The cannula was kept patent with saline solution (sodium chloride 0.9% w/v, Antigen Pharmaceuticals Ltd.). After

insertion of the cannula the subjects were allowed to rest for 10 min and then stood up when they felt capable of doing so. They remained standing for 15 min before a blood sample at rest was collected. The subjects remained in the upright posture for the remainder of the test and all subsequent blood samples were taken in the standing position.

The subjects then moved into the gymnasium and, after the rectal temperature at rest was recorded, performed a standard warm-up of jogging, stretching and faster pace running which took 15 min to complete. During this period a prescribed volume of water ( $4 \text{ ml} \cdot \text{kg}^{-1}$  body mass) was drunk to encourage water intake and place an initial volume of water in the stomach to facilitate gastric emptying.

The same experimenters were responsible for conducting all the trials. During each trial investigators ensured that the subjects placed at least one foot on or over the lines delimiting the 20-m distance at each end of the gymnasium. The subjects were able to gauge their required running speeds and exercise intensities by following the audio signals. Experimenters ensured the subjects kept pace with the audio signals and reminded the subjects immediately prior to each sprint to perform it maximally and thereafter encouraged them to do so. The experimenters also continually provided the subjects with information about the type of exercise required during the next 20 m (such as "walk", "sprint", "cruise" or "jog"). The experimenters gave the same verbal cues to all subjects. Times for 15-m sprints were measured using two infra-red photo-electric cells (RS Components Ltd., Corby, UK) connected to the microcomputer.

A whirling hygrometer (Brannan Thermometers Ltd., Cumberland, UK) was used to measure ambient dry and wet bulb temperatures. In part A of LIST this was done at three set positions along the length of the gymnasium between min 1–4, 7–10 and 12–15, of each exercise set and following rest period. In part B of the test the measurements were made between min 1–4 of every 5. Heart rates were continuously monitored throughout each test using short range telemetry (Sport Tester, PE3000, Polar Electro Fitness Technology, sampling frequency 15 s). Rates of perceived exertion were recorded prior to the ninth sprint in each exercise set using the Borg scale (Borg 1962). A 10-ml blood sample was collected from each subject between the sets of exercise in part A of the test, before part B and at fatigue. Rectal temperatures were measured during the fourth and eighth cycle of each set of part A of LIST and in the 3-min blood sampling period between sets. When rectal temperatures were measured the subjects remained stationary and only walked for 20 min in that cycle (compared with 60 min when temperature was not being measured).

A period of 2 weeks on average (13–15 days) elapsed between the performance of LIST at the two ambient temperatures the order being randomly assigned. During the trials at the high temperature the temperature in the gymnasium was raised to the appropriate level using four electric fan heaters (Andrews DE65, Andrews Industrial Equipment Ltd., UK) placed in the corners of the gymnasium, and by using an externally vented gas heater (Andrews IG175, Andrews Industrial Equipment Ltd., UK) placed at a mid-point in the hall.

Sweat rates were estimated from pre- and post-exercise nude body mass measurements after adjusting for fluid intake. It was assumed that 1 l of fluid was equivalent to 1 kg.

#### Blood sampling and analysis

During the speed-lactate test duplicate 20- $\mu\text{l}$  capillary blood samples were collected from the thumb of each subject. The samples were immediately deproteinised in 200  $\mu\text{l}$  of 2.5% perchloric acid, centrifuged, stored at  $-20^\circ\text{C}$  and subsequently analysed for lactate concentration in accordance with the fluorometric method described by Maughan (1982). Lactate and glucose concentrations were also determined on deproteinised venous blood samples. The latter was analysed spectrophotometrically using a commercially available kit (Boehringer Mannheim, Perid Glucose). Further aliquots from the venous sample were used for determination of haematocrit and haemoglobin concentration (by microcentrifugation

and the cyanmethaemoglobin method, respectively). Changes in plasma volume (%) were estimated using the method of Dill and Costill (1974). A volume of 1-ml of blood was dispensed immediately into a calcium-heparin tube, centrifuged for 3-min at 12,000 rpm and the plasma frozen at  $-70^\circ\text{C}$ . Ammonia concentration was determined within 48 h using a commercially available kit (Boehringer Mannheim, MPR1 Ammonia) and the method of Da Fonseca-Wollheim (1973). The remaining blood from the 10-ml sample was dispensed into an ethylenediaminetetra-acetic acid tube and centrifuged for 15-min at 6000 rpm at approximately  $3^\circ\text{C}$ . The resulting plasma was then stored at  $-20^\circ\text{C}$  for subsequent determination of free fatty acids using a commercially available kit (NEFA-C, Wako) and fully automated colorimetric instrumentation (Cobas Bio Diagnostica, Roche Products Ltd.).

#### Statistical analysis

A three-way analysis of variance (ANOVA, group  $\times$  trial  $\times$  time) for repeated measures on two factors (trial  $\times$  time), and post-hoc Tukey tests, were used to establish if any significant differences existed between subject response (in terms of a variety of physiological and metabolic parameters) to the performance of LIST in the two different ambient temperatures. Ambient temperatures, distance covered during LIST, body mass and plasma volume responses during the main trials were analysed using a two-way ANOVA (group  $\times$  trial) with repeated measures on one factor (trial), and Tukey test where necessary. The relationship between rate of rise in rectal temperature and distance completed was investigated using a Pearson product moment correlation coefficient. Data are based on a subject population of 8 GP and 8 EA unless otherwise stated.

## Results

#### Training status

The mean blood lactate concentration at a reference speed of  $3.6 \text{ m} \cdot \text{s}^{-1}$  was lower for EA [GP ( $n = 7$ ) vs EA ( $n = 8$ ),  $5.8$  (SEM 1.1) vs  $2.9$  (SEM 0.5)  $\text{mmol} \cdot \text{l}^{-1}$ ,  $P = 0.01$ ] but this was largely due to differences in  $\dot{V}\text{O}_{2\text{max}}$  (Table 1,  $P < 0.05$ ), as the percentage of  $\dot{V}\text{O}_{2\text{max}}$  corresponding to a concentration of  $4 \text{ mmol} \cdot \text{l}^{-1}$  was no different [GP ( $n = 7$ ) vs EA ( $n = 6$ ),  $78.1$  (SEM 1.1) vs  $81.2$  (SEM 2.1)%, n.s.].

#### Ambient temperatures

The ambient temperatures given here are the averages of the three measurements made along the length of the gymnasium. Dry bulb temperatures were successfully manipulated to average  $30^\circ$  and  $16^\circ\text{C}$  in the hot (HT) and moderate trials (MT) respectively [HT: GP vs EA,  $30.4$  (SEM 0.1) vs  $30.5$  (SEM 0.1) $^\circ\text{C}$ ; MT: GP vs EA,  $16.0$  (SEM 0.3) vs  $15.9$  (SEM 0.3) $^\circ\text{C}$ ; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ]. Similarly, mean wet bulb temperature was  $6^\circ\text{C}$  higher in the heat [HT: GP vs EA,  $17.1$  (SEM 0.2) vs  $17.1$  (SEM 0.4) $^\circ\text{C}$ ; MT: GP vs EA,  $10.5$  (SEM 0.7) vs  $10.7$  (SEM 0.5) $^\circ\text{C}$ ; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ]. However, relative humidity was greater in MT [HT: GP vs EA,  $24.4$  (SEM 0.8) vs  $24.3$  (SEM 1.6)%; MT: GP vs EA,  $49.5$  (SEM 3.6) vs  $52.1$  (SEM 3.1)%; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ].

**Table 3** The distance completed by the female games players (*GP*) and endurance athletes (*EA*), in part A and in total, while performing the LIST (for explanation see Table 2) in the hot (*HT*) and moderate (*MT*) trials. The numbers in brackets refer to the number of subjects who had to be removed from the particular trial because their rectal temperature had exceeded 39.5°C

	Distance (m)							
	HT				MT			
	Part A		Total		Part A		Total	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
<i>c, d</i>								
GP	7194	981	7876	1270 (1)	8464	826	10995	1586 (3)
EA	8819	627	9140	712 (2)	9725	0	11928	863 (1)

*c* Main effect, trial,  $P < 0.01$ , Total; *d* main effect, trial,  $P < 0.05$ , Part A

**Table 4** Mean 15-m sprint time of the female games players (*GP*) and endurance athletes (*EA*) during the first 15-min set and end set of the LIST (for explanation see Table 2) in the hot trial (*HT*), and at the same time (but not at fatigue) in the moderate trial (*MT*)

	Time (s)							
	HT				MT			
	Set 1		End set		Set 1		HT fatigue set	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
<i>c, i</i>								
GP	2.69	0.05	2.75	0.04	2.69	0.04	2.72	0.03
EA	2.77	0.05	2.84	0.05	2.75	0.05	2.74	0.05

*c* Main effect, trial,  $P < 0.01$ ; main effect, time,  $P = 0.054$ ; *i* interaction, trial  $\times$  time,  $P < 0.01$

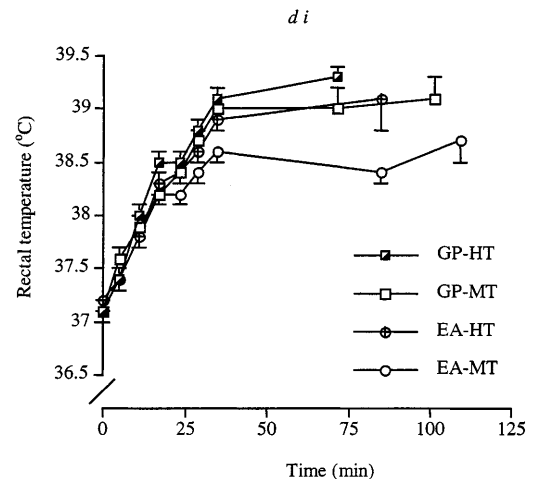
## Performance

The mean distance completed in HT was 2953 (SEM 550) m or 25 (SEM 4)% less than that completed in MT (main effect trial,  $P < 0.01$ ). The distances completed by EA and GP were similar (Table 3). Exercise time was approximately 78 min in HT and approximately 105 min in MT [HT: GP vs EA, mean 71.0 (SEM 11.9) vs 84.4 (SEM 5.2) min; MT: GP vs EA, mean 100.9 (SEM 14.9) vs 109.1 (SEM 8.5) min; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ].

Sprints of 15 m took longer to complete during HT than during MT (main effect, trial,  $P < 0.01$ ; Table 4). Sprint performance declined during HT (set 1 vs end set, interaction trials  $\times$  time,  $P < 0.01$ ) but did not decline during MT (set 1 vs end set, interaction, trial  $\times$  time, n.s.). Sprints of 15-m appeared to be completed faster by GP than by EA (n.s.).

## Rectal temperature

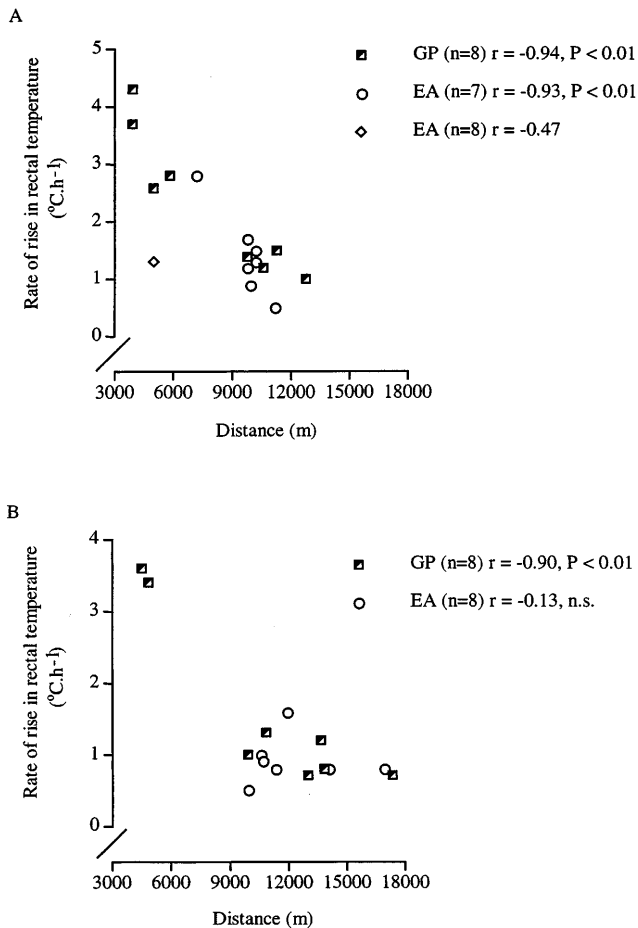
At fatigue in HT rectal temperature was greater than at a similar time (but not at the time of fatigue) in MT (interaction, trial  $\times$  time,  $P < 0.01$ ; Fig. 1). When rest and actual exhaustion were analysed mean rectal temperature was higher in HT than MT but was no different for GP and EA [HT rest: GP vs EA, 37.1 (SEM 0.1) vs 37.2 (SEM 0.1)°C; MT rest: GP vs EA, 37.1 (SEM 0.1) vs 37.1 (SEM 0.1)°C; HT end: GP vs EA, 39.3 (SEM 0.1) vs 39.1 (SEM 0.2)°C; MT end: GP vs EA, 39.1 (SEM 0.2) vs 38.8 (SEM 0.2)°C; main effect, group,



**Fig. 1** Rectal temperature of the games players (*GP*) and endurance athletes (*EA*) during the LIST (for explanation see Table 2) in the hot trial (*HT*) and the moderate trial (*MT*) *d* main effect, trial  $P < 0.05$ ; main effect, time,  $P < 0.01$ ; *i* interaction, trial  $\times$  time,  $P < 0.01$

n.s.; main effect trial,  $P < 0.01$ ; main effect, time,  $P < 0.01$ ].

The relationship between the rate of rise in rectal temperature in HT and the distance completed was very high in GP but not in EA (Fig. 2A). However, the relationship for EA was heavily influenced by one outlier (outlying diamond, Fig. 2A). This particular subject's HT responses in terms of rate of rise in rectal temperature and distance completed were at least two standard deviations from the respective means. When only seven



**Fig. 2** The relationship between rate of rise in rectal temperature and distance completed during the LIST (for explanation see Table 2), by the games players (GP) and endurance athletes (EA) during the hot trial (A) and moderate trial (B)

EA were considered, the relationship between rate of rise in rectal temperature and distance completed was very strong. In MT the rate of rise in rectal temperature was poorly related with distance completed in EA but this was not the case for GP (Fig. 2B).

### Heart rate

Although heart rates were higher in HT compared with MT during the same period there were no differences

**Table 5** Mean heart rate of the female games players (GP) and endurance athletes (EA) during the first 15-min set and end set of the LIST (for explanation see Table 2) in the hot trial (HT), and at the same time (but not at fatigue) in the moderate trial (MT)

	Heart rate (beats · min <sup>-1</sup> )							
	HT				MT			
	Set 1		End set		Set 1		HT fatigue set	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
<i>d</i>								
GP	168	2	180	2	164	3	176	3
EA	165	4	178	3	160	5	174	4

*d* Main effect trial,  $P < 0.05$ ; main effect, time,  $P < 0.01$

between GP and EA (Table 5; main effect, group, n.s.; main effect, trial,  $P < 0.05$ ). At exhaustion mean heart rate was similar in HT and MT [HT end: GP vs EA, 180 (SEM 2) vs 178 (SEM 3) beats · min<sup>-1</sup>; MT end: GP vs EA, 178 (SEM 3) vs 180 (SEM 5) beats · min<sup>-1</sup>; main effect, group, n.s.; main effect, trial, n.s.; main effect, time,  $P < 0.01$ ; interaction, trial × time,  $P < 0.01$ ].

### Body mass, fluid consumption and estimated sweat rate

Body mass was well maintained by both GP and EA during HT and MT as shown by the actual mean body mass losses (pre-trial mass minus post-trial mass) experienced, which were always less than 1% of pre-trial body mass [HT: GP vs EA, 0.05 (SEM 0.20) vs -0.20 (SEM 0.10) kg; MT: GP vs EA, -0.15 (SEM 0.15) vs -0.30 (SEM 0.15) kg; main effect, group, n.s.; main effect, trial, n.s.]. Ad libitum fluid consumption during HT was considerably greater than during MT, but there were no differences in consumption between the two groups [HT: GP vs EA, mean 18.9 (SEM 2.9) vs 13.5 (SEM 1.7) ml · kg<sup>-1</sup> · h<sup>-1</sup>; MT: GP vs EA, mean 12.7 (SEM 3.7) vs 8.5 (SEM 1.5) ml · kg<sup>-1</sup> · h<sup>-1</sup>; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ]. Estimated mean sweat rate in HT was higher than that in MT, but no differences in rate were seen between GP and EA [HT: GP vs EA, 1.46 (SEM 0.21) vs 1.06 (SEM 0.10) l · h<sup>-1</sup>; MT: GP vs EA, 1.07 (SEM 0.22) vs 0.76 (SEM 0.10) l · h<sup>-1</sup>; main effect, group, n.s.; main effect, trial,  $P < 0.01$ ].

### Metabolic responses

Blood lactate concentrations were similar in HT and MT (main effect, trial, n.s.) but were higher for GP than EA at the HT end point (interaction, group × time,  $P < 0.01$ ; Table 6). Similarly plasma ammonia concentrations were unaffected by the heat (main effect, trial, n.s.) but were higher for GP than EA (main effect, group,  $P < 0.05$ ). In contrast blood glucose concentrations were higher in HT than MT (main effect, trial,  $P < 0.01$ ) but were similar in GP and EA (main effect, group, n.s.). Plasma free fatty acid concentrations at the end of exercise in HT and at the same times in MT tended to be

**Table 6** Blood metabolites of the female games players (GP) and endurance athletes (EA) at rest and at the end of the LIST (for explanation see Table 2) in the hot trial (HT), and at the same time (but not at fatigue) in the moderate trial (MT). FFA Free fatty acid

	Concentration (mmol · l <sup>-1</sup> )							
	HT				MT			
	Rest		End		Rest		HT End	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
<i>e, h</i>								
Blood lactate								
GP	0.8	0.1	8.0	0.9	0.9	0.1	8.0	1.3
EA	0.9	0.1	4.9	1.1	0.9	1.1	4.4	1.2
<i>f, h</i>								
Plasma ammonia								
GP	18.5	2.4	70.1	12.7	18.5	2.3	76.8	8.8
EA	14.6	1.9	43.2	6.1	15.9	2.2	32.5	3.8
<i>c, i</i>								
Blood glucose								
GP	4.3	0.1	6.7	0.3	4.3	0.1	6.2	0.5
EA	4.2	0.1	6.9	0.8	4.0	0.1	5.3	0.4
<i>g</i>								
Plasma FFA								
GP	0.46	0.05	0.48	0.07	0.52	0.04	0.62	0.13
EA	0.50	0.05	0.89	0.09	0.48	0.06	0.72	0.10

*c* Main effect, trial,  $P < 0.01$ ; *e* main effect, group,  $P = 0.056$ ; *f* main effect, group,  $P < 0.05$ ; *g* interaction, group  $\times$  time,  $P = 0.051$ ; *h* interaction, group  $\times$  time,  $P < 0.01$ ; *i* interaction, trial  $\times$  time,  $P < 0.01$ ; all main effects with time were statistically significant,  $P < 0.01$

**Table 7** Rate of perceived exertion of the female games players (GP) and endurance athletes (EA) during the first 15-min set and end set of the LIST (for explanation see Table 2) in the hot trial (HT), and at the same time (but not at fatigue) in the moderate trial (MT). RPE Rate of perceived exertion (Borg 1962)

	RPE (Borg scale)							
	HT				MT			
	Set 1		End set		Set 1		HT fatigue set	
	Mean	SEM	Mean	SEM	Mean	SEM	Mean	SEM
<i>c</i>								
GP	14	1	17	1	12	1	15	1
EA	12	0	16	0	11	0	14	1

*c* Main effect, trial,  $P < 0.01$ ; main effect, time,  $P = 0.01$

higher for EA than for GP (interaction, group  $\times$  time,  $P = 0.051$ ).

#### Plasma volume

The estimated change in mean plasma volume during HT was no different from that seen over the same time in MT [HT: GP vs EA,  $-2.6$  (SEM 1.9) vs  $2.7$  (SEM 2.8)%; MT: GP vs EA,  $-2.9$  (SEM 1.8) vs  $0.6$  (SEM 2.5)%; main effect, group, n.s.; main effect, trial, n.s.].

#### Rate of perceived exertion

As Table 7 shows while there were no differences in the pattern of response between the GP and EA both groups perceived exercise to be harder in HT than in MT (main effect, trial,  $P < 0.01$ ).

## Discussion

The main finding in the present study was that exercise at an ambient temperature of 30°C resulted in a 25% decrease in distance run by non-heat-acclimatised GP and EA during LIST. The subjects were also unable to maintain 15-m sprint times in HT, while they were able to do so in MT. Rectal temperature, heart rate and blood glucose concentrations were greater in HT than MT but plasma ammonia and blood lactate concentrations were similar. The GP had higher plasma ammonia and blood lactate concentrations than EA.

The decrease in the distance run during LIST in HT was of a similar magnitude (25% vs 21%) to that reported for men performing the same LIST at 20° and 30°C (Morris et al. 1998). The finding that mean rectal temperature was higher at fatigue in HT compared with the same time (but not at fatigue) in MT [HT: GP vs EA,  $39.3$  (SEM 0.1) vs  $39.1$  (SEM 0.2)°C; MT: GP vs EA,

39.0 (SEM 0.2) vs 38.4 (SEM 0.1)°C; interaction, trial  $\times$  time,  $P < 0.01$ ], and the very strong relationship found between the rate of rise in rectal temperature and distance completed in HT was also consistent with the findings in the study of men (Morris et al. 1998). These results support the assertion that high body temperature, as indicated by rectal temperature in this study, was probably the key factor in the decreased exercise performance seen in HT. While it is unclear precisely how this elevated body temperature curtailed the performance of exercise, possible mechanisms have been suggested to include alterations in the metabolic response to exercise (Febbraio et al. 1994a), reductions in cardiac output (MacDougall et al. 1974), a reduced drive to exercise (Nielsen et al. 1990), or dehydration (Mountain and Coyle 1992).

In the present study the high ambient temperatures resulted in slower 15 m sprints. This contrasts with a number of other studies which have found substantial increases in power outputs when individuals have performed maximal intensity cycle ergometer sprints in high as against more moderate ambient temperatures, or after immersion in a water bath at various temperatures (Ball et al. 1999; Falk et al. 1998; Sargeant 1987). However, in these previously published studies the sprints were of longer duration ( $\geq 15$  s) and a smaller number were performed (maximum 5). Also, unlike the present study, the subjects in these investigations were not, or were unlikely to have been, hyperthermic during exercise. Where some measure of deep body temperature has been reported (Falk et al. 1998) the subjects were clearly not hyperthermic (peak rectal temperature 37.5°C). Therefore, while it is far from clear exactly why sprint performance declined in the heat in the present study, this finding may well be related to elevated body temperature.

If heat stress alters the metabolic response to activity it might provide an explanation for the earlier onset of fatigue seen when individuals exercise to exhaustion in high environmental temperatures. In the present study blood lactate and plasma ammonia concentrations were similar when HT and MT were compared, suggesting that the metabolic responses to exercising in the different environmental conditions were alike. However, even where changes in metabolism in muscle have been found as a result of exercising in hot environments (Febbraio et al. 1994a, b), when the exercise is exhausting it has seemed that the muscle glycogen concentrations were not sufficiently low to provide a likely explanation of the earlier onset of fatigue in the heat (Febbraio et al. 1996).

The blood lactate and plasma ammonia concentrations of GP were almost twice as great as those of EA at the end of exercise in HT and at the same time (but not at fatigue) in MT. There are a number of possible explanations for these differences, including sprint trained athletes having higher proportions of type II fibres compared with greater proportions of type I fibres in EA (see Costill et al. 1976). It is also possible that EA had a greater capacity for lactate clearance. However, what-

ever were the precise mechanisms for the greater blood lactate and plasma ammonia concentrations seen in GP, the performance of this group was similar to that of EA. These findings would suggest that differences in the metabolic responses to the environmental temperature or between the two groups were not crucial in producing the performances seen in this study.

In the present study, the decreased performance may have occurred because of thermal strain induced changes in blood flow around the body. It has been suggested that the diversion of blood flow to the body's periphery for heat dissipation may result in a reduction in central blood volume as blood accumulates in compliant cutaneous veins (Werner 1993). This may produce a reduced stroke volume and therefore decreased cardiac output. The greater heart rate in HT compared with MT was a good indication that the cardiovascular system was attempting to maintain cardiac output. However, it has been shown that when exercise-heat stress is too great even large increases in heart rate cannot compensate for declines in stroke volume and so cardiac output may fall (Werner 1993). Several authors have noted the inability of cardiac output to meet the demands placed upon it during exercise in the heat, resulting in an early onset of fatigue (MacDougall et al. 1974; Suzuki 1980). In the present study, none of the subjects complained of, or displayed any obvious signs of heat syncope, which suggests that inadequate cardiac output was not a major factor in the earlier onset of fatigue in LIST during HT.

It has been suggested (although not directly measured) that high body temperature may curtail performance by affecting central nervous system functioning which results in a reduced drive to continue exercising (Nielsen et al. 1990; Savard et al. 1988). The higher rate of perceived exertion values seen in HT in the present study may give some indication that central drive was reduced and may provide an explanation for the curtailment of performance seen in HT.

It is also possible that dehydration may have contributed to reduced performance in HT as the extent of heart rate increase, stroke volume decrease and degree of hyperthermia experienced has been found to be linearly related to the magnitude of dehydration that accrues during 2 h of cycling (Mountain and Coyle 1992). In addition, rectal temperature and heart rate increases have seemed to be directly related to the level of hypohydration at which subjects began a heat stress test (Sawka et al. 1985). However, pre-trial body mass measurements and haemoglobin concentrations suggest that our subjects started LIST in the two environments in similar states of hydration. During exercise the fluid consumption of the subjects was approximately 50% greater in HT compared to MT. This seemed to offset the higher estimated sweat rates in HT as plasma volume changes were similar for the subjects in the two trials, and body mass was well maintained in both HT and MT. Consequently, these results suggest that the changes in fluid status in the subjects were similar in HT and MT. They also support the assertion that a difference in

hydration either prior to or during exercise was not a major factor in the decrement in performance seen in HT.

As discussed above the training background and physical make-up of GP and EA may well have affected some of their metabolic responses, yet the performance of the two groups was similar. Performance would seem to be related to body temperature and acclimatisation has been shown to reduce the rectal temperature and heart rate (and therefore the thermal strain) of individuals exercising at a set intensity in a hot environment (Lind and Bass 1963). It has been accepted that physical training "partially acclimatises" individuals to exercising in hot environmental conditions (Werner 1993). In the present study although the type of training undertaken by GP and EA was different the similarity in their speed-lactate test results would suggest that the two groups were similarly well trained. The similarity in performance of the two groups supports the assertion that if partial acclimatisation is occurring then games type activity is as effective as submaximal running in inducing it. It is of interest that EA had lower mean MT rectal temperatures than GP at the same time as fatigue occurred in HT [rest: GP vs EA, 37.1 (SEM 0.0) vs 37.1 (SEM 0.1)°C; HT fatigue: GP vs EA, 39.0 (SEM 0.2) vs 38.4 (SEM 0.1)°C; interaction, group  $\times$  trial,  $P < 0.05$ ]. This finding would seem to suggest that EA had a more sensitive thermoregulatory system as suggested by Gisolfi and Wenger (1984). Why this did not manifest itself in terms of improved performance in HT is not clear.

Elevated blood glucose concentrations in the heat, such as were found in the present study, have often been seen when comparing exercise in hot environments with that in cooler ones (Febbraio et al. 1994a; Yaspelkis et al. 1993). While higher blood glucose concentrations may be associated with elevated muscle glycogen utilization (Febbraio et al. 1994a), which is a potential source of fatigue when exercising in the heat, this has not always been found to be the case (Yaspelkis et al. 1993). The increased glucose concentrations could be the result of decreased muscle glucose uptake or greater hepatic glucose release. Where muscle glucose uptake has been measured (Nielsen et al. 1990) no significant differences between moderate and hot conditions were found. Using a primed continuous infusion method Hargreaves et al. (1996) have found that the rate of disappearance and metabolic clearance rate of glucose did not differ when cycling in hot and temperate conditions. However, the rate of appearance of glucose was significantly greater in the heat, suggesting greater hepatic release. Nonetheless, while it may be indicative of the thermal strain induced by HT, it seems unlikely that high blood glucose per se will have been responsible for the decrements in performance evident in HT in the present study.

It is recognised that a weakness of the present investigation was not allowing for differences in the phase of the menstrual cycle as it has been suggested that this affects body temperature at rest and the response to exercise (Pivarnik et al. 1992; Stephenson

and Kolka 1993), and can also influence metabolic and performance responses (Hall Jurkowski et al. 1981). Resting oesophageal or rectal temperature prior to an exercise test has been shown to be approximately 0.3°C higher in the luteal compared with the follicular phase. It is of interest that this difference has been found not only to persist during exercise but to increase as activity progresses (Pivarnik et al. 1992; Stephenson and Kolka 1993). Obviously as the rate of rise in rectal temperature seems to have been a key factor in explaining the results seen in the present study not taking account of differences in the phase of the menstrual cycle which could have affected this variable, weakens the findings of the investigation. However, while the phase of the menstrual cycle was not monitored in the present investigation the rate of rise in rectal temperature during exercise was approximately 70% greater in HT than in MT. This compares with the approximately 15% greater rate of rise in rectal temperature seen by Pivarnik et al. (1992) in the luteal phase of the menstrual cycle in comparison with the follicular phase during a relatively prolonged period of exercise.

The magnitude of the difference seen in our experiment would appear to be too great to be explained solely by possible differences in the phase of the menstrual cycle even if all the subjects who participated in the study were eumenorrhic and in different phases of their menstrual cycle, which seems unlikely given the athletic nature of EA and GP and the time between trials. It may be that in terms of exercising to exhaustion any effect of the phase of the menstrual cycle will be of lesser importance than the imposition of heat stress and the effects of the resulting heat strain. Also there are a number of studies, many using well-trained women as were used here, which have found no effect of menstrual status on performance (De Souza et al. 1990; Lebrun et al. 1995; Lynch and Nimmo 1998). Similarly in a recent study in our laboratory (Sunderland and Nevill, unpublished observations) the phase of the menstrual cycle was found to have no effect on the performance of prolonged, intermittent, high-intensity running in the heat. However, future investigations of women using this exercise protocol would benefit from taking account of menstrual status which may allow the present results to be confirmed.

In summary, in well-trained, non-acclimatised female GP and EA the performance of LIST was detrimentally affected by high ambient temperatures. While the training background of the subjects affected some metabolic responses, performances were similar in GP and EA. This suggests any inter-group metabolic differences were not major factors in the observed decrement in performance in the heat, and that the type of training being undertaken by individuals does not affect exercise performance in a hot environment as long as individuals are matched for training status. It seems clear that the performance decrements were associated with high body temperatures but the precise mechanisms of fatigue were unclear.



**Acknowledgements** The authors would like to thank Ben Brown, Nikki Gabb and Alex Poole for their help during the data collection.

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