Eur J Appl Physiol (1999) 79: 360–366

ORIGINAL ARTICLE

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Human power output during repeated sprint cycle exercise: the influence of thermal stress

Accepted: 1 September 1998

Abstract Thermal stress is known to impair endurance capacity during moderate prolonged exercise. However, there is relatively little available information concerning the effects of thermal stress on the performance of high-intensity short-duration exercise. The present experiment examined human power output during repeated bouts of short-term maximal exercise. On two separate occasions, seven healthy males performed two 30-s bouts of sprint exercise (sprints I and II), with 4 min of passive recovery in between, on a cycle ergometer. The sprints were performed in both a normal environment [18.7 (1.5)°C, 40 (7)% relative humidity (RH; mean SD)] and a hot environment [30.1 (0.5)°C, 55 (9)% RH]. The order of exercise trials was randomised and separated by a minimum of 4 days. Mean power, peak power and decline in power output were calculated from the flywheel velocity after correction for flywheel acceleration. Peak power output was higher when exercise was performed in the heat compared to the normal environment in both sprint I [910 (172) W vs 656 (58) W; $P < 0.01$] and sprint II [907 (150) vs 646 (37) W; $P < 0.05$]. Mean power output was higher in the heat compared to the normal environment in both sprint I [634 (91) W vs 510 (59) W; $P < 0.05$] and sprint II [589 (70) W vs 482 (47) W; $P < 0.05$]. There was a faster rate of fatigue ($P < 0.05$) when exercise was performed in the heat compared to the normal environment. Arterialised-venous blood samples were taken for the determination of acid-base status and blood lactate and blood glucose before exercise, 2 min after sprint I, and at several time points after sprint II. Before exercise there was no difference in resting acid-base status or blood metabolites between environmental conditions. There was a decrease in blood pH, plasma bicarbonate and base excess after sprint I and after sprint II. The degree of post-exercise acidosis was similar when exercise was performed in either of the environmental conditions. The metabolic response to exercise was similar between environmental conditions; the concentration of blood lactate increased ($P < 0.01$) after sprint I and sprint II but there were no differences in lactate concentration when comparing the exercise bouts performed in a normal and a hot environment. These data demonstrate that when brief intense exercise is performed in the heat, peak power output increases by about 25% and mean power output increases by 15%; this was due to achieving a higher pedal cadence in the heat.

Key words Temperature · Power output · High-intensity exercise · acid-base status

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Introduction

As early as 1945, Asmussen and Boje demonstrated that performance of very brief (12 s), dynamic exercise improved if the muscles were pre-warmed by either moderate-intensity exercise or passive heating of the limbs. Based on findings from the effects of sub-maximal pre-exercise, they postulated that the improvement in performance stemmed from an increase in blood flow to the working muscle. In contrast, moderate prolonged exercise performance has been shown to be impaired if performed in the heat (Saltin et al. 1972; Wyndham et al. 1976). Similarly, maximum aerobic power is reduced when measured under conditions of thermal stress (Saltin et al. 1972).

The physiological responses to moderate sustained exercise in the heat [49°C, 20% relative humidity (RH)] compared to exercise in a normal environment (21°C, 30% RH) are well documented. These responses include an increase in heart rate, redistribution of blood flow, elevated core temperature and an increase in total body sweat rate (Rowell 1974; Young et al. 1985). When compared to exercise in the cold, the metabolic response to heat exposure during exercise at 70–85% of maximal oxygen consumption $\dot{V}O_{2max}$ leads to an increased rate of glycolysis (Fink et al. 1975). However, Young et al. (1985) reported that although muscle lactate accumulation was greater in the heat, aerobic metabolism was reduced while glycogenolytic rate had remained the same. These differences in findings could be due to the experimental protocol that was employed. More recent findings support those of Fink et al. (1975); Febbraio et al. (1994b) reported that submaximal exercise in the heat (40°C, 20% RH) resulted in higher muscle glycogenolysis, an increase in muscle lactate production, a greater decline in creatine phosphate levels and an increase ammonia and creatine accumulation. Overall, their suggestion was that heat stress results in an increase in ATP utilisation that is met by an increase in anaerobic metabolic processes (Febbraio et al. 1994b).

Relatively little attention has since been given to the effect of thermal stress on the capacity to perform short-term high-intensity exercise. However, in a recent study hot-water immersion was shown to have a significant effect on improving the power output of type I (oxidative, fatigue-resistant) fibres (Sargeant and Rademaker 1996). The contribution to power output from the type II (glycolytic, fatigue-sensitive) fibres was found not to change following passive heating within the locomotory range used during isokinetic cycling.

The experiment described here was designed to measure human power output during two 30-s bouts of maximum sprint exercise on a cycle ergometer under normal and hot environmental conditions. The metabolic responses to exercise under conditions of thermal stress were compared to those found during exercise in a normal environment.

Materials and methods

Subjects

Seven healthy males volunteered for the present study, which had received Ethics committee approval. The subjects' characteristics were [mean (SD)], age 25 (5) years, height 1.80 (0.50) m and body mass 70.0 (5.5) kg. After each subject had been given a verbal and written description of the experiment and any possible risks associated with the experiment they provided written informed consent. All subjects participated in some form of regular physical exercise but none was highly trained.

Experimental protocol

On two separate occasions, 4 days apart, subjects reported to the laboratory following an overnight fast. Each subject's body weight was measured, while they were wearing only shorts. After dressing they then sat for 15 min. One hand was then immersed in hot water (42°C), and after 10 min an arterialised-venous blood sample was obtained from a superficial vein on the dorsal surface of the heated hand via a 21-gauge butterfly cannula.

Each subject then performed two 30-s maximum sprints on a friction-loaded cycle ergometer (Monark 814 E, Varburg, Sweden) in either a normal environment [18.7 (1.5)°C; 40 (7)% RH] or in a hot environment [30.1 (0.5)°C; 55 (9) % RH]. The order of the experimental trials in the different environmental conditions was randomised. Each experimental trial consisted of a 30-s sprint, from a static start (sprint I), followed by 4 min of seated passive recovery and then a further 30-s sprint (sprint II). The test load was body-mass dependent and was adjusted to equal 0.075 $kg \cdot kg^{-1}$ body mass. Power output was computed from friction load and flywheel velocity. Instantaneous flywheel velocity was measured using a d.c. generator, and data was logged using an analogue-to-digital converter on a PC. Power output was corrected for flywheel acceleration and calculated every second (Lakomy 1986). All subjects completed a minimum of three habituation sessions prior to completing either experimental trial. The habituation sessions were identical to the experimental trials but were conducted without blood sampling.

When performing the exercise trial in the heat, after providing the resting blood sample subjects entered an environmental chamber for 30 min to thermally equilibrate them with the increased environmental temperature. For the last 10 min of this period subjects were seated with their hand immersed in water at 42°C. The same protocol was then followed for the trials in the normal environment.

Blood sampling

Before exercise, arterialised-venous blood samples (2.5 ml) were collected, without coming into contact with air, capped, kept on ice and then measured within 2 h for blood pH, partial pressure of carbon dioxide and partial pressure of oxygen (Forster et al. 1972). Plasma bicarbonate and blood base excess were calculated according to Siggaard-Andersen (1963). Further blood samples (2.5 ml) were collected and mixed with tri-potassium ethylene diaminetetraacetic acid (1 $mg \cdot ml^{-1}$). From this, duplicate aliquots (100 µl) of blood were immediately deproteinised in 1 ml of ice-cold 2.5% perchloric acid. These samples were later analysed for concentration of glucose using the glucose peroxidase method (Boehringer Chemicals, Mannheim, Germany). The concentration of blood lactate was determined according to the method of Maughan (1982). The remainder of the whole blood sample was used to determine haemoglobin concentration, using the cyanmethaemoglobin technique, and packed cell volume, using the microcapillary centrifuge technique. Changes in plasma volume were estimated according to the method of Dill and Costill (1974) but the blood data were not corrected for these changes in plasma volume. Further blood samples for the determination of acid-base status were taken 2 min after sprint I, and at 2, 5 min after sprint II. Blood samples for the determination of metabolites were also taken 2 min after sprint I, and at 2, 5 and 15 min after sprint II.

Statistical analysis

All data were checked for normality of distribution prior to performing any inferential statistics. Data were analysed using a two-way analysis of variance with repeated measures; where a difference

was found this was located by post-hoc Tukey test. Significance is declared at the 5% probability level. Data are presented as the mean (SD) in the text and tables, and for reasons of clarity as mean (SEM) in the figures.

Results

Power output

Peak power $\dot{W}_{peak}$ output was not different between sprint I and sprint II in either environmental condition (Fig. 1). When subjects performed the sprints in the hot environment the average $\dot{W}_{peak}$ output was higher [909 (80) W; $P < 0.01$] than that achieved in a normal environment [650 (45) W]. Mean power $\bar{W}$ output followed a similar trend to $\dot{W}_{peak}$ output. There were no differences in $\bar{W}$ output between sprint I and II in either environmental condition, but the $\bar{W}$ output was higher when exercise was performed in the heat. On average, $\bar{W}$ output increased ($P < 0.05$) from 496 (53) W in the normal environment to 612 (81) W in the heat (Fig. 2). The decline in power output did not differ between sprint I and II in both environments. The decline in power output was significantly greater ($P < 0.01$) in the heat during sprint I [48 (5)%] than under normal conditions [34 (3)%]. However, the decline in power output that occurred during sprint II was similar in both conditions (Fig. 3a). The greater $\dot{W}_{peak}$ output and decline in power were mirrored by the initial difference and subsequent change in mean pedal frequency over the 30-s period. Overall, the pedal frequency was significantly higher [111 (13) rev · min^{-1}; $P < 0.01$] when exercise was performed in the heat compared to the normal environment [90 (7) rev · min^{-1}], but over the final 5 s of exercise the pedal frequency attained in the heat was not different from that attained in the normal environment (Fig. 3b).

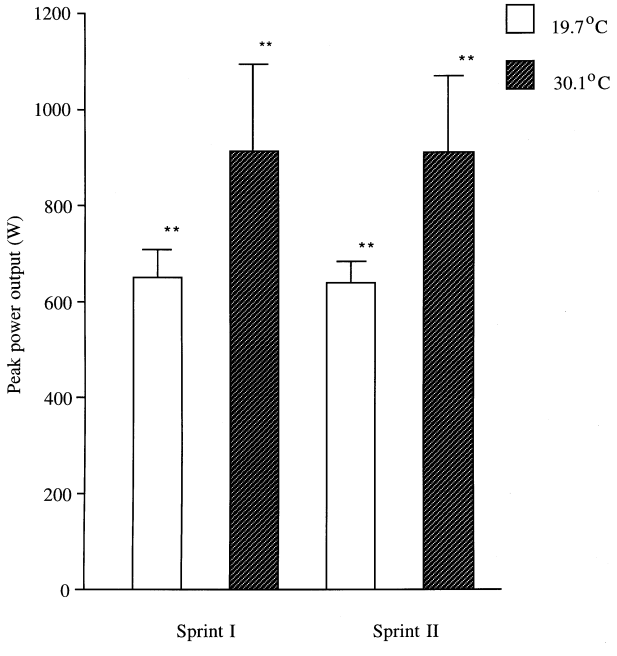

Fig. 1 Peak power output (W) [mean (SE)] during sprint I and sprint II performed in either a normal [18.7°C, 40% relative humidity (RH); *open bar*] or a hot (30°C, 55% RH; *closed bar*) environment. Significant differences between environmental conditions are denoted by: $*P < 0.05$, $**P < 0.01$

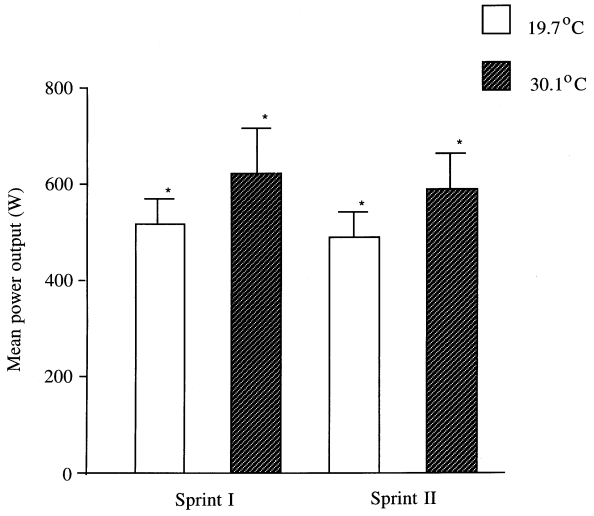

Fig. 2 Mean power output (W) [mean (SE)] during sprint I and sprint II performed in either a normal (*open bar*) or a hot (*closed bar*) environment. Significant differences between environmental conditions are denoted by: $*P < 0.05$, $**P < 0.01$

Acid-base status

Resting acid-base status was similar in both environmental conditions (Fig. 4). After sprint I, blood pH declined significantly from 7.39 (0.01) to 7.21 (0.04) ($P < 0.01$). Five minutes after sprint II blood pH had declined further to 7.13 (0.05) ($P < 0.05$). The observed changes in blood pH were similar under both environmental conditions. Blood base excess can provide a better estimate of the metabolic disturbances to acid-base status. The changes in base excess followed the same pattern as that described for blood pH; following sprint I, base excess decreased from a resting value of −1.04 (1.00) mmol · l^{-1} to −11.61 (2.35) ($P < 0.01$). A further decline in blood base excess was observed after sprint II, to −18.43 (1.91) mmol · l^{-1} at 2 min, and −18.94 (2.11) mmol · l^{-1} at 5 min. The degree of post-exercise acidosis was similar under both environmental conditions (Fig. 5).

Blood metabolites

The resting concentration of blood glucose was 5.2 (0.3) mmol · l^{-1} in the normal environment. Exposure to the hot environment for 30 min had no effect on the concentration of blood glucose (Table 1). The first

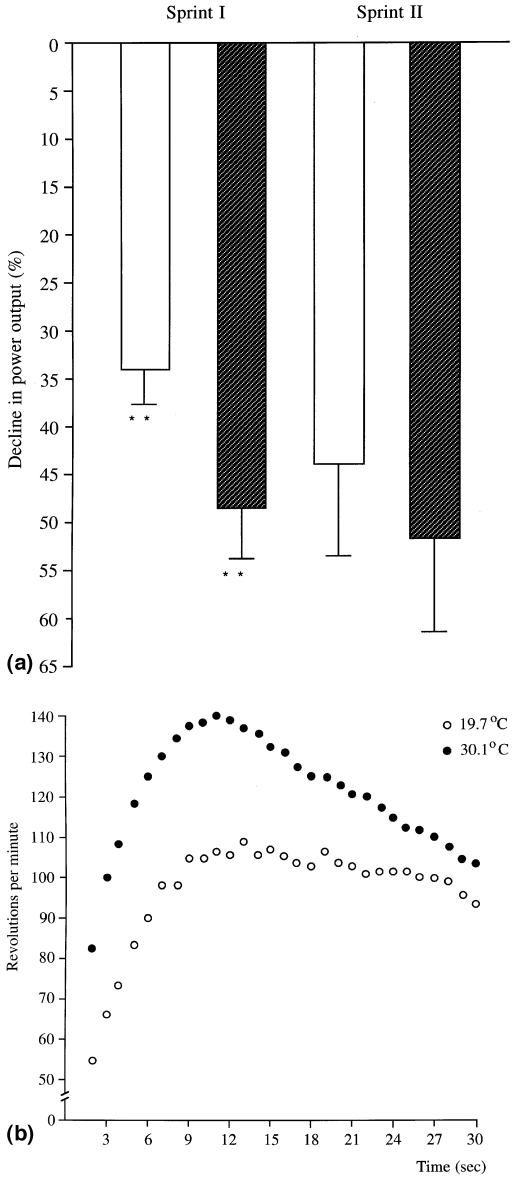

Fig. 3a, b Fatigue index and typical pedal cadence during repeated sprint exercise performed in either a normal (*open symbols*) or a hot (*closed symbols*) environment. **a** Fatigue index, represented by a decline in power output (%) [mean (SE)] **b** Pedal cadence of one subject during sprint I in a normal and a hot environment

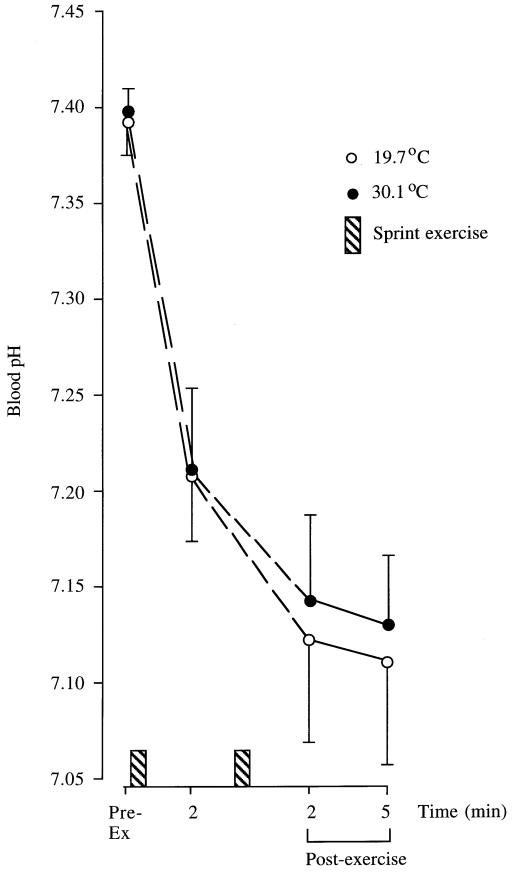

Fig. 4 Blood pH [mean (SE)] before exercise (Pre-Ex), 2 min after sprint I, and at 2 and 5 min after sprint II, performed in either a normal (*open circles*) or a hot environment (*closed circles*)

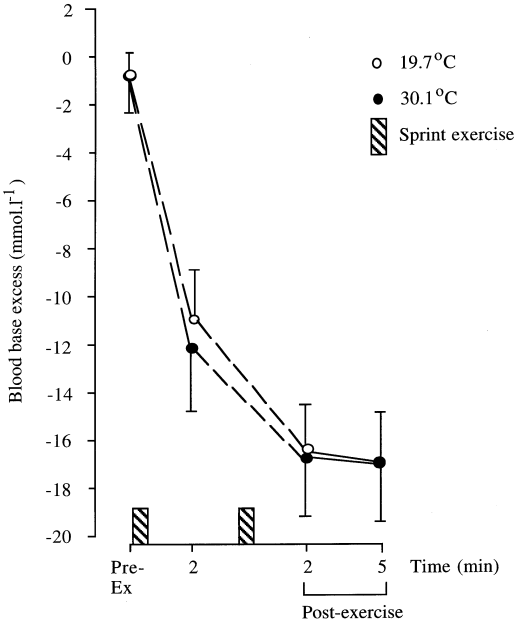

Fig. 5 Blood base excess [mmol · l^{-1} mean (SE)] before exercise (Pre-Ex), 2 min after sprint I, and at 2 and 5 min after sprint II, performed in either a normal (*open circles*) or a hot environment (*closed circles*)

bout of sprint exercise did not result in a significant increase in the concentration of blood glucose. However, following sprint II the concentration of blood glucose increased ($P < 0.01$) to 6.1 (0.5) mmol · l^{-1} in the normal environment, and to 6.1 (0.4) mmol · l^{-1} in the hot environment. Blood glucose was still higher ($P < 0.05$) 15 min after sprint II than the resting concentration in both environmental conditions (Table 1).

Exposure to heat for 30 min had no effect on the concentration of blood lactate at rest (Table 1). Following sprint I, blood lactate had increased from 0.3 (0.2) to 5.0 (0.9) mmol · l^{-1} in the normal condition, and to 5.1 (0.7) mmol · l^{-1} in the hot trial; there was no

Table 1 The effect of thermal stress (normal environment, 19°C, vs hot environment, 30°C) on the concentration of blood glucose ($mmol \cdot l^{-1}$) and blood lactate ($mmol \cdot l^{-1}$) before and after two bouts of sprint exercise (sprint I and sprint II) separated by a 4-min rest period. Data are presented as the mean (SD)

Time (min)	Blood glucose ($mmol \cdot l^{-1}$)		Blood lactate ($mmol \cdot l^{-1}$)	
	Normal (19°C)	Hot (30°C)	Normal (19°C)	Hot (30°C)
Pre-heat stress	–	5.3 (0.3)	–	0.28 (0.14)
Pre-exercise	5.2 (0.3)	5.2 (0.1)	0.27 (0.19)	0.26 (0.14)
2 post-sprint I	5.3 (0.4)	5.4 (0.2)	5.00 (0.93)**	5.10 (0.73)**
2 post-sprint II	6.1 (0.5)*	6.1 (0.4)*	8.00 (1.32)**	7.78 (1.07)**
5 post-sprint II	5.9 (0.4)*	6.2 (0.4)*	9.00 (2.06)**	7.60 (1.12)**
15 post-sprint II	5.9 (0.5)*	5.9 (0.3)*	8.96 (2.16)**	6.91 (0.75)**

*$P < 0.05$
**$P < 0.01$

difference in blood lactate between conditions. After sprint II, the concentration of blood lactate was similar between conditions and in both cases had increased to 8.0 (2.1) $mmol \cdot l^{-1}$ and 7.8 (1.1) $mmol \cdot l^{-1}$ in the normal and hot trials, respectively. The highest concentration of blood lactate occurred 2 min after sprint II in the heat and 5 min after sprint II in the normal condition (Table 1).

Discussion

The results of the present experiment demonstrate a large and unexpected increase in power output during short-term sprint exercise with acute exposure to a warm (30°C) environment when compared to a normal environment. $\dot{W}_{peak}$ and $\bar{W}$ output were found to increase by 25% and 15%, respectively, when exercise was performed in the heat compared to a normal environment, although there was a greater decline in power output. This improvement in $\bar{W}_{peak}$ and $\bar{W}$ was achieved without any apparent difference in the metabolic response to this type of exercise, as determined by acid-base status and blood metabolite concentration.

In a recent study examining sprint exercise in the heat, Falk et al. (1998) reported that both $\dot{W}_{peak}$ and $\bar{W}$ increased by at least 8% compared to that measured in a normal environment. Their reported increase in power output could, however, be underestimated since $\dot{W}_{peak}$ and $\bar{W}$ were not corrected for flywheel acceleration, and the values for these parameters were averaged over 5 s and 15 s, respectively. In this study the increase in power output achieved when exercise was completed in the heat is substantially higher than that observed by Falk et al. (1998); it also appears to be somewhat higher than expected. However, it is known that direct heating and cooling of the limbs can significantly affect power output during dynamic exercise (Sargeant 1987). During 20 s of maximal cycling at optimal pedal rates for maximum power output ($\dot{W}_{max}$), Sargeant (1987) reported that for every 1°C increase in muscle temperature there was a concomitant 4% increase in power output ($\dot{W}_{max}$). This effect, however, was velocity dependent; at 54 $rev \cdot min^{-1}$ there was a 2% per 1°C improvement in $\dot{W}_{max}$, and at 140 $rev \cdot min^{-1}$ there was a 10% increase per 1°C. With an improvement in power output under the present experimental conditions this would be measured as an increase in pedal velocity since the resistive load remains fixed. An increase in pedal cadence towards optimal velocity (110 $rev \cdot min^{-1}$, Sargeant et al. 1981) will result in achieving an even greater power output because of the power/velocity relationship for skeletal muscle. In the study described here, during exercise in the heat the mean pedal cadence was 111 (13) $rev \cdot min^{-1}$ and in the normal environment the average cadence was 90 (7) $rev \cdot min^{-1}$. It should be borne in mind that increases in muscle temperature, as well as changing the power/velocity relationship, will also simultaneously affect the mechanical efficiency/velocity relationship (see, e.g., Sargeant and Jones 1996). Thus, the increased power attained at a higher velocity under hot conditions may have been delivered at a similar efficiency as in the control. The precise impact of these changes on the metabolic demands of our experiment is, however, difficult to predict.

The effect of muscle temperature on isometric force production in humans is somewhat equivocal; force production is reported to be relatively insensitive to the effects of temperature change (Binkhorst et al. 1977; Davies and Young 1983). Although peak isometric force is insensitive to temperature change, endurance capacity during isometric contractions is significantly affected by temperature (Edwards et al. 1972). After cold-water immersion (12°C) endurance capacity at 66% of maximum voluntary contraction was longer than after hot-water immersion (44°C). The earlier onset of fatigue following heating was due to the increased crossbridge cycling from the onset of contraction and an inability to match this ATP utilisation rate during subsequent contractions. The results of the present experiment would appear to support this suggestion; although subjects generated significantly more power under thermal stress they also exhibited a faster rate of fatigue. Sargeant (1987), studying isokinetic cycling at 95 $rev \cdot min^{-1}$, reported a decline in force production of between 12% and 21% as muscle temperature decreased by 4.7°C and 7.6°C, respectively. Following hot-water immersion

(44°C), force production, at the same cadence, was found to be 11% higher than in the normal rested condition. The power output measured during the present experiment is a product of flywheel velocity and both the resistive load and the excess load (Lakomy 1986). Since the excess load will increase as flywheel acceleration increases, the force necessary to achieve a higher flywheel velocity will be greater. It is therefore presumed that force production in the hot environment was greater than in the normal environment.

The concentration of blood lactate and glucose observed after sprint exercise in the heat was similar to that measured under the normal condition. This is somewhat surprising considering the reported effects of thermal stress on the metabolic response during moderate sustained exercise. During three bouts of cycle exercise at approximately 70–85% of aerobic capacity, Fink et al. (1975) reported that muscle glycogen utilisation was approximately 1.8 times higher when exercise was performed in the heat (41°C) compared to a cold environment (9°C). In the same study they also reported that the measured concentration of blood lactate was almost twice as high in the heat compared to the cold. An increase in the rate of glycogenolysis with exposure to thermal stress has been reported to occur during moderate-intensity prolonged exercise (Febbraio et al. 1994a, b; Kozlowski et al. 1985; Young et al. 1985). In these studies the increased glycogenolytic rate was partially explained as a function of a decrease in oxygen delivery to the working muscle. In all of these studies the exercise intensity was submaximal, which should therefore permit an increase in the glycolytic rate. In the study described here, sprint exercise was employed, and from the onset of exercise the glycogenolytic rate is presumed to be maximal, while the duration of exercise was only 30 s. It is, perhaps, not surprising therefore that there was little observable effect of thermal stress on the concentration of blood lactate and glucose following exercise.

In the present experiment all subjects were thermally equilibrated in the environmental chamber for 30 min prior to starting exercise; this may have induced the release of catecholamines. Powers et al. (1982) reported that exposure to heat at rest resulted in a significant rise in noradrenaline and a small but significant rise in adrenaline concentration. Moderate exercise produced a marked increase in catecholamine concentration but there were no differences between a normal condition and under conditions of thermal stress. In a recent study (Brenner et al. 1997) 60 min of exposure to 40°C at rest was shown to increase the concentration of adrenaline compared with exposure to a thermoneutral environment (23°C). Increases in circulating levels of catecholamines are known to stimulate a higher rate of glycolysis; this effect is fibre-type specific. Greenhaff et al. (1991) demonstrated that adrenaline infusion increased the glycogenolytic rate in type I fibres by ten fold without any measurable effect on the type II fibres. A higher glycogenolytic rate may be expected to induce a greater acid load due to the increased production of H^+ ions, and also a concomitant increase in lactate production. In the present study there were no differences in blood acid-base status either before or after exercise between environmental conditions.

Exposure to the warm environment could have led to a favourable shift in the force/velocity relationship of the type I fibres, resulting in an improvement in both $\bar{W}$ and $\dot{W}_{peak}$ output without a difference in the metabolic response to exercise. This suggestion is based on evidence from human experiments. In a study in which two groups with different proportions of muscle fibre types were used, Rademaker (1997) investigated the effect of thermal stress on power output during brief (5 s) sprint exercise on a cycle ergometer at three different pedal speeds. The group that had a greater percentage of type I fibres (73%) generated less power than those with a lower percentage (45%) of type I fibres. However, those subjects with 73% of their fibres as slow fibres were, on average, 2–3 times more sensitive (in terms of power production) to changes in temperature than the group in whom 45% of their fibre population comprised type I muscle fibres. Whatever the mechanism, a small increase in velocity will result in a marked increase in power output because of the power/velocity relationship; this is more so with the type I fibres because at this locomotory range they will be operating at around their optimal velocity.

In conclusion, the study described here has demonstrated that power output during two bouts of sprint exercise (30 s) is improved when completed in a hot (30°C) environment compared to a normal (19°C) environment. The metabolic response to the sprint exercise appeared to be relatively similar when exercise was performed in either environment since there was no difference in either blood lactate, blood glucose or blood acid-base status. The improvement in power output that occurred during exposure to thermal stress could be attributed to a direct effect on the force/velocity and power/velocity characteristics of the slow type I muscle fibres.

Acknowledgement The authors wish to thank R.A. Ferguson for technical assistance during the completion of this study.

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