Effect of Hot Environment on Repetitive Sprint Performance and Maximal Accumulated O_2 Deficit of Cyclists

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Abstract. This study was undertaken with a view to compare the effect of hot environment on repeated sprint performance and maximal accumulated oxygen deficit (MAOD) during continuous sub-maximal exercise. Eight male cyclists aged 22.5 ± 2.1 yr, weight 63.4 ± 4.5 kg and VO_{2max} of 58 ± 5.2 ml/kg/min participated in this study. The method of measuring MAOD was adopted from Medbø et al., (1988). In phase 1 the VO_2max of the cyclists were measured on cycle ergometer following a graded exercise protocol. In phase 2, the cyclists did sub-maximal exercise for 10 min at 60, 70, 80, 90%VO_{2max}, on separate days. The linear regression determined from the VO_2 – power relationship was used to estimate supra-maximal power output at 120% VO_{2max}. In phase 3, the subjects performed AOD test with maximal sprint (120%VO_{2max}) until exhaustion followed by a continuous cycling for 60 min (60%VO_{2max}). The subjects performed another 3 sprints (120%VO_{2max}) after every 20 min. The exercise was conducted in thermo-neutral (25.7 ± 0.4^{o}C) condition. In phase 4 the exercise was conducted in hot condition (31.4 ± 0.1^{o}C) with the same protocol. Results indicated there was no difference in MAOD, sprint performances, core temperature, plasma lactate and plasma ammonia between the hot and thermo-neutral conditions ($p<0.05$). However, MAOD and sprint performance deteriorated in subsequent sprints ($p<0.05$) irrespective of hot and thermo-neutral conditions. This study highlighted that hot environment did not impart any significant change in the anaerobic capacity of the cyclists measured on the basis of maximal accumulated O_2 deficit, all out repeated sprint performance, plasma lactate and plasma ammonia levels.

Key Words: MAOD, cyclists, sprint performance, hot environment

1. Introduction

Endurance performance is impaired in hot environment as compared to temperate environment and the time to exhaustion is influenced by alterations of heat stress and increase in body temperature (Febbraio et al, 1994). The exposure of environmental heat and the effect of aerobic performance in the hot environment have been widely studied (Marino et al, 2001). In quantifying the effect of performance in hot environment, the anaerobic performance in hot environment has not been studied much. From our knowledge, studies on the impacts of prolonged sub-maximal cycling with intense intermittent work in temperate environment which simulate the actual condition in road cycling are scanty in the literature.

Maximal accumulated oxygen deficit (MAOD) has been described by Medbø (1991) as the total amount of adenosine triphosphate (ATP) that can be formed via the anaerobic process or metabolism under high-intensity exercise conditions. Extensive studies on the validity of MAOD as a means of measuring maximal anaerobic capacity have been conducted (Medbø et al, 1988; Medbø, 1991; Medbø and Tabata, 1993).

Finn et al. (2003) studied the effect of hot environment on anaerobic capacity of the cyclists. In this study, the duration of exposure in hot environment was very short and might not have influenced the anaerobic capacity of the cyclists. Finn et al, (2003) only estimated duration of sprint as a measure of anaerobic capacity and not the accumulated oxygen deficit. In their study, only a short time exposure for a sprint in hot environment did not exhibit any influence on sprint performance.

Hence, the present study was undertaken (i) to compare the effect of prolonged sub-maximal exercise on maximal accumulated oxygen deficit (reflecting anaerobic capacity) of the cyclists in thermo-neutral and hot
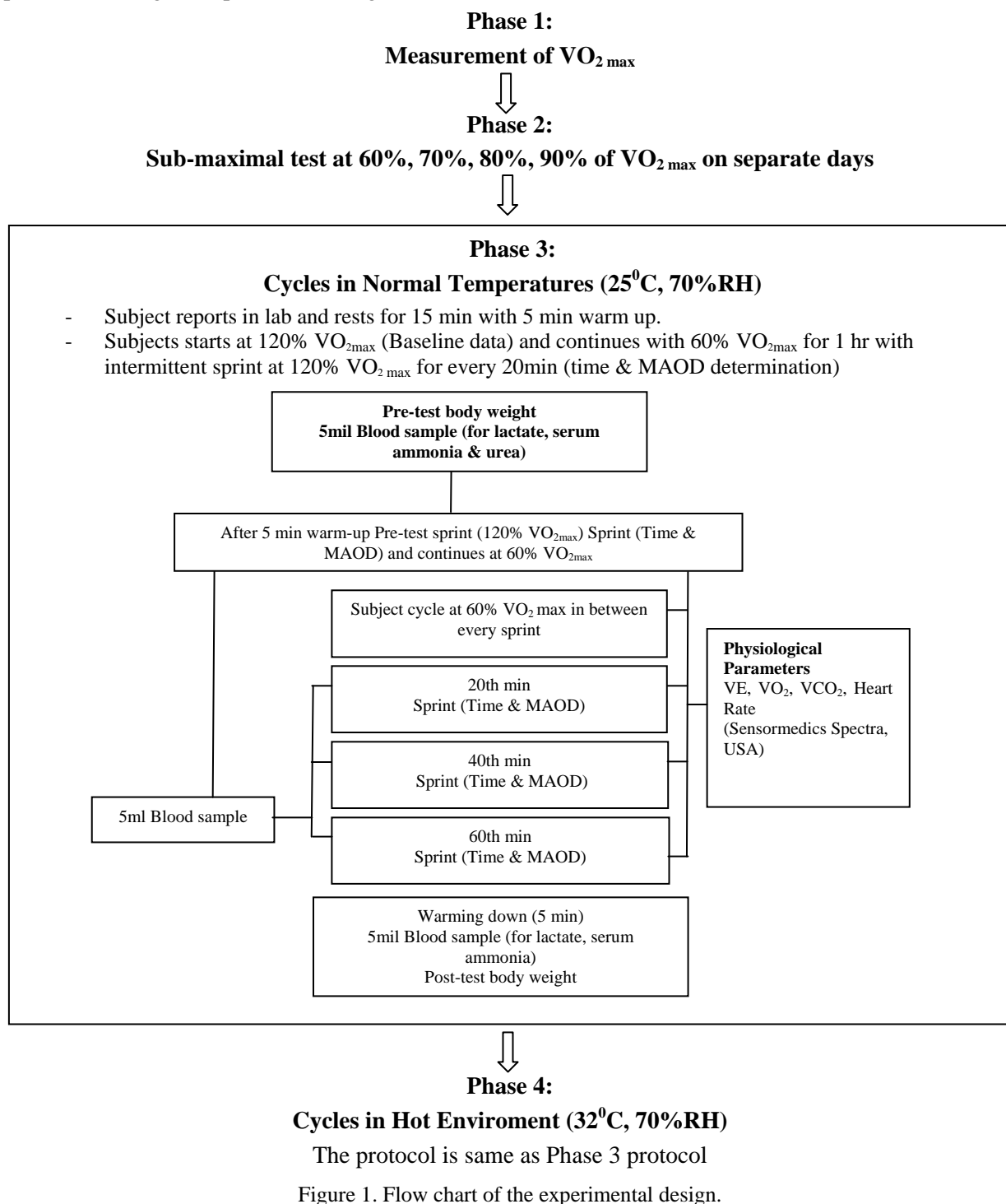
environment, (ii) to investigate the repeated sprint performance of the cyclists in thermo-neutral and hot environment and (iii) to investigate the effect of repeated sprints on blood lactate and ammonia during prolonged exercise in thermo-neutral and hot environments.

2. Methodology

Subjects: Eight male cyclists (19-28 years old) with ability to cycle at 70% VO_{2max} for 60 minutes were recruited for this study. All the subjects must have participated in a major domestic cycling competition and are undergoing regular training at the time of study. The subjects were thoroughly briefed about the purpose of the experiments and exercise procedures before signing a written consent form. The study has been approved by the Research and Ethics Committee of Universiti Sains Malaysia. A flowchart of the experimental design is represented in Figure 1.

Phase 1:

Measurement of $VO_{2\,max}$

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Phase 2:

Sub-maximal test at 60%, 70%, 80%, 90% of $VO_{2\,max}$ on separate days

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Phase 3:

Cycles in Normal Temperatures (25^0C, 70%RH)

- Subject reports in lab and rests for 15 min with 5 min warm up.
- Subjects starts at 120% VO_{2max} (Baseline data) and continues with 60% VO_{2max} for 1 hr with intermittent sprint at 120% $VO_{2\,max}$ for every 20min (time & MAOD determination)

Pre-test body weight
5mil Blood sample (for lactate, serum ammonia & urea)

After 5 min warm-up Pre-test sprint (120% VO_{2max}) Sprint (Time & MAOD) and continues at 60% VO_{2max}

Subject cycle at 60% VO_2 max in between every sprint

20th min
Sprint (Time & MAOD)

40th min
Sprint (Time & MAOD)

5ml Blood sample

60th min
Sprint (Time & MAOD)

Physiological Parameters
VE, VO_2, VCO_2, Heart Rate
(Sensormedics Spectra, USA)

Warming down (5 min)
5mil Blood sample (for lactate, serum ammonia)
Post-test body weight

⇩

Phase 4:

Cycles in Hot Enviroment (32^0C, 70%RH)

The protocol is same as Phase 3 protocol

Figure 1. Flow chart of the experimental design.

The complete methodology of the present study was divided into the following four phases.

Phase 1: VO_{2max} of each subject was measured in the laboratory following a graded incremental cycling protocol on an Excalibur, Lode cycle ergometer. The initial load was 50W and was increased by 16W every 1 minute until exhaustion.

Phase 2: In this phase four sub-maximal tests were performed on cycle ergometer at intensities of 60%, 70%, 80% and 90% VO_{2max} power output. Each subject performed a sub-maximal exercise for 10 min at each of the above power output on separate days. The steady state VO_2 at each workload between 9-10 min was recorded. Oxygen uptake values measured during min 9 and 10 from each sub-maximal test was used as the steady-state VO_2 for the corresponding power output. From the linear equation, the power output at 120% VO_{2max} was extrapolated for each subject to calculate estimated O_2 deficit (Medbø et al, 1988).

Phase 3: In this phase, the experiment was conducted in a thermo-neutral chamber (25^0C; 70% RH). The subject reported in the laboratory in the morning, after an overnight 12 hrs. fast. After a 15 min rest, the subject was fitted with gas analyzer accessories. Subjects were then allowed to warm-up for 5 minutes at 50 Watt load on the cycle ergometer followed by a sprint until exhaustion at power output of 120%VO_{2max}. Exhaustion was determined when the subjects could no longer maintain pedal cadence at 60 rpm despite verbal encouragement. The workload was then reduced to 60% of the subject's VO_{2max} and they cycled at 60% VO_{2max} continuously for 60min between the sprints. Each subject performed 3 sprints at 120%VO_{2max} after every 20 min. The pedaling frequency was kept at 60 rpm while cycling at 60% VO_{2max}, but during sprints at 120% VO_{2max} power output, the cadence was kept between 90-110 rpm. Sprint duration (min), VO_2, minute ventilation (VE), and heart rate were recorded throughout the exercise bout on a computerized gas analyzer (Sensormedics Spectra, USA). Core temperature, room temperature and relative humidity were recorded at every 5 minutes intervals throughout the trials. 5 ml of blood was withdrawn immediately after every sprint. Nude body weight was measured before and after the test. The rectal temperature was recorded using a thermistor probe (Yellow Springs Instruments, USA), 15cm beyond the anal sphincter.

Phase 4: In this phase, the same protocol of phase 3 has been conducted in a hot chamber where the temperature and humidity was controlled at 32^0C and 70% RH, respectively. Experimental trials of phase 3 and 4 were performed on two different occasions with minimum 7 days gap.

The AOD for each subject was calculated in oxygen consumption equivalents as the difference between the oxygen demand of exercise and the measured VO_2. This test procedure and calculation was adapted from Medbø et al. (1988).

Blood sample collection and analysis:

Approximately 5ml of venous blood was withdrawn during each sample collection. 2ml of blood from each sample was transferred into anticoagulant with sodium fluoride (NaF) tube and was used for the determination of plasma lactate. The plasma was separated and stored at -80°C (Thermo Forma -86 ULT Freezer, USA). The plasma lactate concentration was determined using Lactate analyzer (Yellow Springs Instrument model Sport 1500, USA). Another 3ml of the blood sample was transferred to a lithium heparin tube and the plasma was separated and was analyzed for ammonia using commercially available kits (Randox, U.K.).

2.1. Statistical Analysis

The data were analyzed using Statistical Package for Social Sciences (SPSS) version 12.0 (SPSS, 2004). Repeated measure ANOVA has been applied for

(i)   Comparing variables in two environments.
(ii)   Comparing variables in 4 sprints keeping the Sprint 1 as base.

Mean and standard deviation for continuous variable and frequency as well as percentages for categorical variable were calculated. Statistical significance was set at p value less than 0.05 ($p<0.05$).

3. Results

The mean age, height, weight and VO_{2max} are 22.5 ± 2.1 yrs, 169.8 ± 4.8 cm, 63.4 ± 4.5 kg and 58 ± 5.2 ml/kg/min, respectively. The mean room temperature was 25.7 ± 0.4˚C in thermo-neutral and 31.4 ± 0.1˚C in hot condition. Mean room relative humidity was 69.7 ± 0.4 % in thermo-neutral condition and 70.4 ± 0.5 % in hot condition.

MAOD (Mean ± SD) of the cyclists at different sprints in thermo-neutral and hot environment is presented in Table 1. There was no significant difference in MAOD between the hot and thermo-neutral

condition at base line level and during all the sprints. However, MAOD of the cyclists reduced significantly from the base line level ($p < 0.05$) in both the environments. The mean MAOD decreased in Sprint 2, 3, and 4 from the Sprint 1 in both the hot and thermo-neutral condition. No significant difference existed among sprint 2, 3 and 4.
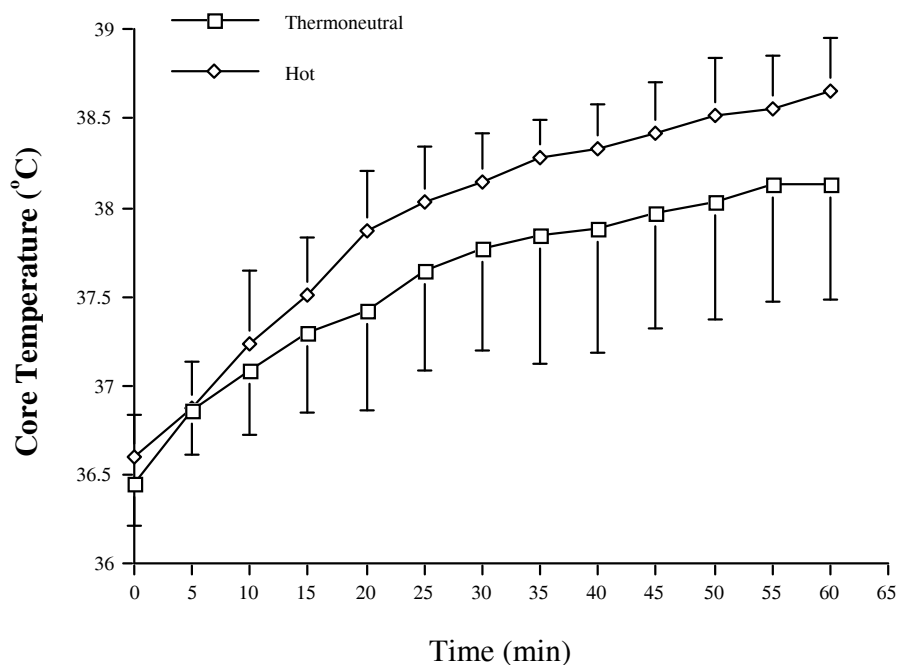

Figure 2**.** Core body temperature (˚C) changes of the subjects during sub-maximal exercise in hot and thermo-neutral condition.

Table 1. The maximal accumulated oxygen deficit (MAOD) during exercise in thermo-neutral and hot environment.

Environment	Spint 1		Sprint 2		Sprint 3		Sprint 4	
	(L)	(ml/kg)	(L)	(ml/kg)	(L)	(ml/kg)	(L)	(ml/kg)
Thermo-Neutral	3.8	60.8	2.9	47.9 *	2.7	43.9*	2.6	40.9 *
	± 1.2	± 21.9	± 1.1	± 21.3	± 0.4	± 8.4	± 0.5	± 10.4
Hot	3.5	55.7	2.9	47.2 *	2.8	45.3*	2.5	39.9*
	± 1.5	± 25.6	± 1.2	± 22.4	± 0.6	± 11.8	± 0.6	± 9.6

Values are means ± SD. (Sprint 1= Baseline MAOD; Sprint 2, 3, and 4 are subsequent MAOD after every 20 min.)
*$p<0.05$ (from sprint 1) in thermoneutral environment. *$p<0.05$ (from sprint 1) in hot environment.

The mean ± SD values for repeated sprint performances are illustrated in Table 2. No significant difference in sprint performance was observed between the sprints in hot and thermo-neutral environments. However, sprints 2, 3, and 4 timings were significantly ($p<0.05$) lower from the sprint 1 (baseline sprint) in both the thermo-neutral and hot environments. The time tom exhaustion at sprints 2, 3 and 4 did not differ among themselves.

Table 2. The sprint performance (time to exhaustion) during exercise in thermoneutral and hot condition

Environment	Sprint 1	Sprint 2	Sprint 3	Sprint 4
Thermo-Neutral	84.5 ± 23.5	66.4 * ± 11.8	66.6 * ± 14.3	65.6 * ± 10.1
Hot	85.4 ± 21.5	63.9 * ± 9.1	57.9 * ± 14.9	55.5 * ± 13.8

* $p < 0.05$ from sprint 1.

No significant difference was observed in the body weight of the cyclists after 60 min cycling (Table 3).

Table 3. Change of body weight (kg) and percentage change in body weight (%) of subjects during thermoneutral and hot condition (Mean ± SD)

Exercise Condition	Pre-exercise weight (kg)	Post-exercise weight (kg)	Change in body weight (kg)	Percentage change in body weight
Thermo-neutral	63.4 ± 7.9	62.8 ± 7.7	0.59 ± 0.3	0.8 ± 0.4
Hot	63.0 ± 7.9	62.4 ± 7.6	0.63 ± 0.5	0.9 ± 0.7

The change in core body temperature is presented in Figure 2. There was a significant (p<0.05) rise in core body temperature during cycling in thermo-neutral and hot environments over time (p<0.05). But the core temperature during repeated sprints did not differ significantly between hot and thermo-neutral environments.

The mean plasma lactate and plasma ammonia concentrations are presented in Table 4. No significant differences existed either in plasma lactate concentration or in plasma ammonia concentrations between thermo-neutral and hot environments at any sprint bouts. Both the plasma lactate concentration and the plasma ammonia concentrations increased significantly (p<0.05) from resting to all sprints bout in thermo-neutral and in the hot environments.

Table 4. The plasma lactate [La] and ammonia [NH$_3$] during exercise in thermo-neutral and hot environments.

Environment	Resting		Spint 1		Sprint 2		Sprint 3		Sprint 4	
	(La)	(NH$_3$)	(La)	(NH$_3$)	(La)	(NH$_3$)	(La)	(NH$_3$)	(La)	(NH$_3$)
Thermo-Neutral	3.3 ±1.1	86.7 ±27.4	14.6[*] ±4.6	204.4[†] ±59.9	15.8* ±4.8	223.9[†] ±62.4	15.5* ±4.6	218.1[†] ±72.7	15.9* ± 4.1	232.4[†] ±72.7
Hot	3.9 ±1.5	97.1 ±25.6	14.4* ±4.0	198.6[†] ± 95.1	16.1* ±4.8	210[†] ±73.3	14.4* ±5.2	175.6[†] ±29.4	13.8* ±5.4	206[†] ±57.3

Values are means ± SD. [La: mmol/L] [NH$_3$: µg/dL].   * = La Significant higher from resting. †=NH$_3$ significantly higher from resting. No significant difference existed between sprints in hot and thermo-neutral environments.

4. Discussion

MAOD is a valid measure of the anaerobic energy release during exercise (Medbø and Tabata, 1993). The present study indicated there was neither any significant difference in MAOD (anaerobic capacity) nor in sprint performance between thermoneutral and hot condition. This study also confirmed the previous observation during a single sprint that hot environment did not exert any significant effects on anaerobic capacity (Finn et al, 2003). Maximal accumulated oxygen deficit is a quantity and is not a rate (Finn et al, 2003). Since both the stores of creatine phosphate and the extent to which lactate can accumulate are limited, the anaerobic capacity is a finite and a separate entity from the aerobic energy system (Medbø et al., 1998). The concept of a finite entity independent of aerobic influence has also been supported with no change in magnitude of MAOD in the hypoxic conditions (Linnarsson et al., 1974), in which the anaerobic contribution to performance is also isolated. Since there are not many studies to support these findings, evidence in human studies however support the notion that fatigue in hot environment appears to coincide with a critically high internal body temperature (Fuller et al., 1998; Galloway and Maughan, 1997; MacDougal et al., 1974) and reduction in muscle glycogen which is substrate independent (Febbraio, 2000). The MAOD and sprint performances in this study deteriorated over time. It is expected the degree of deterioration in MAOD and sprint performances in between prolonged submaximal exercise were reflecting the subject's inability to maintain the supramaximal power output during repeated sprint bouts. Studies on the relationship between the oxygen deficit and the time to exhaustion during prolonged submaximal exercise are scanty in the literature. Hence, we conclude that the anaerobic capacity performance, measured on the basis of maximal accumulated oxygen deficit and time to exhaustion deteriorated in subsequent sprints as an effect of prolonged submaximal exercise and not as an effect of hot environment.

The higher the environmental temperature, the greater is the dependence on evaporative heat loss and

thus sweating. Therefore, in hot environment, a considerable amount of body water can be lost through eccrine sweat gland secretion to enable the evaporative cooling of the body (Nielsen et al, 2001). Our findings showed that the deterioration in performance, even though not significant between the trial in thermo-neutral and hot environments was related neither to the body weight loss nor to the increased core body temperature of the subjects. The mechanism by which hyperthermia cause fatigue is still not well understood but could be related to alterations in frontal brain activity (Nielsen et al, 2001). Fatigue is mainly a response to signals originating from active muscles, internal organs and/or central nervous system, secondary to the rise in temperature. Probably, these might be the causes for deterioration of repeated sprint duration.

The blood lactate response to maximal short duration exercise has been proposed as a potential marker of anaerobic capacity. In this study the results indicated that there was no significant difference in plasma lactate concentration between thermo-neutral and hot environments. However there was a significant change in plasma lactate concentration from sprint 1 to sprint 4. This increase indicated that repeated high intensity exercise has increased the anaerobic metabolism irrespective of environmental conditions. The onset of lactate concentration in the blood signifies that anaerobic energy metabolism may be the cause of muscular fatigue. As lactic acid and it's by product of hydrogen ions are formed in the muscle as a consequence of oxygen independent glycolysis. The increase in the relative contribution of lactate formation as exercise duration approaches 2 minutes suggests that the maximal rate of glycogen breakdown will increase with increased duration of the exercise (Medbø et al., 1998). It is also likely that the elevated circulating epinephrine observed during exercise in the heat will increase muscle glycolysis and lactate accumulation (Febbraio et al, 1994) irrespective of thermo-neutral and hot environments.

The major sources of ammonia during exercise are adenosine monophosphate (AMP) deaminase and catabolism of branched chain amino acids (Ogino et al, 2000). During high intensity exercise, ammonia originates mainly from the deamination of AMP (Broberg and Sahlin, 1989). However, during prolonged sub-maximal exercise, the oxidation of amino acids becomes an increasingly important source of ammonia production (MacLean et al, 1991). The present study exhibited there was no difference in plasma ammonia concentration between thermo-neutral and heat conditions similar to plasma lactate. However, there was an increase in plasma ammonia concentration during prolonged sub-maximal exercise as shown by high concentration values within the sprint bouts as similarly observed in plasma lactate response to sprints. During progressive incremental exercise, plasma ammonia concentration increases with exercise intensity in such a way that a threshold value existed in a similar way like plasma lactate, indicating anaerobic metabolism (Buono et al, 1984). Ammonia produced during exercise alters neuromuscular activity and may contribute to local muscle fatigue, and can also have detrimental effects on the central nervous system when it reaches the brain (Nielsen et al, 2001). Finding from the previous studies (Ogino et al, 2000, Yuan and Chan, 2004) have shown no significant correlation between ammonia and the measurements related to anaerobic power, anaerobic capacity and aerobic capacity. However, there is support and suggestion that there is a correlation between exercise ammonia and lactate concentration (Itoh and Ohkuwa, 1991; Ogino et al, 2000).

Present study indicated that there was no difference in O_2 deficit as well as sprint duration between the hot and thermo-neutral environments. Hence, it is concluded that the anaerobic capacity might not be influenced by the environmental temperature. Our study confirmed that even in repeated sprints, the anaerobic capacity is not influenced by hot environment. The accumulated O_2 deficit deteriorated in repeated sprints during prolonged sub-maximal exercise. There was increased stress on anaerobic metabolism which resulted from an earlier onset of fatigue reflecting the inability of the subjects to maintain the supramaximal power output during repeated sprints bouts. This was despite of the fact that there was sufficient time for anaerobic energy system to recover during prolonged sub-maximal exercise. Plasma ammonia and lactate are indicators of anaerobic metabolism. A high level of these variables reflects the anaerobic capacity utilization. Since the plasma lactate and ammonia increased during repeated sprints, it is confirmed that the cyclists utilized anaerobic metabolism during exercise. But no difference existed in both variables in hot and thermo-neutral conditions.

5. Acknowledgement

6. References

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