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ORIGINAL ARTICLE

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Maximal accumulated oxygen deficit expressed relative to the active muscle mass for cycling in untrained male and female subjects

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Abstract The purpose of the present study was to determine if gender differences exist in the maximal accumulated oxygen deficit (MAOD) or in the blood lactate (Lac^-) and catecholamine responses to the MAOD test (120% peak oxygen uptake to exhaustion). The MAOD for cycling was measured in ten untrained male and ten untrained female subjects using the method described by Medbø et al. (Anaerobic capacity determined by maximal accumulated oxygen deficit. *J Appl Physiol* 64: 50–60, 1988). Blood Lac^- and catecholamine concentrations were measured at rest, exhaustion and for 30 min following the MAOD test. Dual-energy X-ray absorptiometry was used to measure lean body mass (LBM) and to estimate the active muscle mass (AMM) for cycling. Males achieved a significantly higher MAOD than females following correction for AMM [126.3 (5.6) versus 108.3 (6.1) ml · kg AMM^{-1}, $P = 0.04$]. The peak blood lactate concentration ([Lac^-]) in males [13.6 (0.9) mmol · l^{-1}] was significantly higher than in females [10.0 (1.0) mmol · l^{-1}]. Males obtained a 68% higher peak epinephrine concentration ([Epi]) than females, but the difference was not significant [1268 (188) pg · ml^{-1} versus 755 (179) pg · ml^{-1}, $P = 0.066$]. However, plasma [Epi] was significantly higher for males than females at 1 min [824 (116) versus 489 (116) pg · ml^{-1}, $P = 0.036$] and 3 min [330 (52) versus 179 (42) pg · ml^{-1}, $P = 0.039$] into the recovery period. No gender-dependent differences in the norepinephrine concentration were observed at any time. Peak [Lac^-] was significantly correlated with MAOD (ml · kg AMM^{-1}) in females ($r = 0.75$), but not in males ($r = 0.09$). The peak plasma [Epi] was not significantly correlated with MAOD (ml · kg AMM^{-1}) or peak [Lac^-] in either group. These findings suggest that there are gender-dependent differences in MAOD even when expressed relative to the AMM for cycling. The higher blood [Lac^-] in males compared to females obtained after supramaximal exercise was not caused by enhanced secretion of Epi. The greater MAOD in untrained males was not caused by a greater ability to produce Lac^- or by enhanced secretion of Epi.

Key words Anaerobic capacity · Blood lactate · Cycling · Epinephrine · Gender · Norepinephrine

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Introduction

The maximal amount of adenosine triphosphate (ATP) that can be produced through anaerobic metabolism during a single supramaximal exercise bout is called the anaerobic capacity (AC) (Green and Dawson 1993). Anaerobic ATP production is difficult to measure and the validity of most methods used to determine AC is questionable (Gastin 1994; Green and Dawson 1993). The maximal accumulated oxygen deficit (MAOD) method described by Medbø et al. (1988) has been used to examine differences in AC among endurance athletes, sprinters and untrained subjects (Medbø and Burgers 1990). Several researchers (Carlson and Naughton 1993; Medbø and Burgers 1990; Naughton et al. 1997; Ramsbottom et al. 1997; Weyand et al. 1993) have also compared MAOD values obtained from male and female subjects. However, these studies have not reported MAOD values expressed relative to an accurate measure of active muscle mass (AMM).

Although gender-dependent differences in AC have been reported, the magnitude of the difference remains uncertain. Part of this uncertainty may be due to differences in the method used to measure AC. Maud and Shultz (1986) reported that the higher AC of males is significantly related to their greater lean body mass (LBM). When AC values are expressed relative to LBM, gender-dependent differences are not significant (Maud and Shultz 1986). In contrast, Weyand et al.

(1993) demonstrated that trained males have a 20% higher MAOD than trained females, even after controlling for differences in the AMM for cycling. However, Weyand et al. (1993) and others (Winter et al. 1991) used fat-free leg volume as an estimate of the AMM for cycling. This method of estimating the AMM for cycling does not include the gluteal muscle group, which has been demonstrated to be important to cycling performance (Griffith 1997; Mohr et al. 1981). Dual-energy X-ray absorptiometry (DEXA) is a precise alternative to existing anthropometric methods used to estimate the AMM for cycling (Fuller et al. 1992). The AMM for cycling measured using DEXA in the present study includes the LBM of the legs and the gluteal muscle group, but it does not include bone mineral content (BMC).

In addition to discrepancies in the methods used, differences in training status and the use of both endurance- and sprint-trained subjects may also contribute to the uncertainty concerning differences in AC attributable to gender. The subjects studied by Weyand et al. (1993) were from diverse athletic backgrounds, such as running and gymnastics, and differences in their training status may have influenced the MAOD values obtained. Therefore, the greater MAOD values obtained for male athletes compared to female athletes could be partly due to differences in their training background. In the present study, we controlled for training status by including only untrained male and female subjects.

Previous studies have found that males have higher blood lactate concentrations ([Lac^-]) than females after supramaximal exercise (Gratas-Delamarche et al. 1994; Naughton et al. 1997; Ramsbottom et al. 1997). This suggests that males have a greater capacity than females to generate ATP via anaerobic glycolysis (Jacobs et al. 1983; Naughton et al. 1997). A higher rate of anaerobic glycolysis could partly account for the observation that males have a higher AC than females (Naughton et al. 1997). In addition, Brooks et al. (1990) and Gratas-Delamarche et al. (1994) found that males have significantly higher plasma epinephrine concentrations ([Epi]) than females following ten 6-s sprints and after 30 s of high-intensity exercise. The stimulation of muscle glycogenolysis by Epi may help males to sustain a higher rate of glycolytic flux compared to females during exhaustive supramaximal exercise. This could contribute to males having a greater ability than females to produce ATP from glycolysis and provide for a greater AC. No previous study has examined the relationship between MAOD and exercise-induced changes in blood Lac^- and catecholamine concentrations in untrained male and female subjects. Therefore, the purpose of the present study was to determine if gender differences exist in the MAOD for cycling or in the blood Lac^- and catecholamine responses of untrained male and female subjects to the MAOD test.

Methods

Subjects

Ten untrained male and ten untrained female subjects volunteered to participate in this study. The physical characteristics of the subjects are presented in Table 1. Subjects were considered untrained if they were not training and had not participated or competed in a sport for 24 months. Following familiarization with all testing equipment and experimental procedures, written informed consent was obtained from each subject. The Griffith University Human Research Ethics Committee approved this study.

Experimental protocol

Each subject attended four testing sessions on separate days within a 4-week period. Subjects were instructed not to engage in vigorous physical activity for 48 h prior to visiting the Exercise Science Research Laboratory. Session one involved the measurement of steady-state oxygen uptake ($\dot{V}O_2$) at three submaximal power outputs as well as the determination of the peak $\dot{V}O_2$ reached during cycling. During session two, steady-state $\dot{V}O_2$ was measured at three higher submaximal power outputs. The third session was used to measure the AC for cycling using the MAOD method described by Medbø et al. (1988). Venous blood samples were obtained prior to the MAOD test, immediately at exhaustion and at 1, 3, 5, 10, 15, 20 and 30 min of recovery. These samples were subsequently analyzed for blood Lac^- and plasma catecholamine concentrations. During the final session, body composition and the AMM for cycling were determined using DEXA.

Submaximal exercise bouts

Steady-state $\dot{V}O_2$ was measured at six submaximal power outputs using a Lode (Excalibur Sport) electronically braked cycle ergometer. Subjects performed 2 min of unloaded cycling at 70 rev · min^{-1} and then the predetermined power output was applied immediately for 10 min. The power output for the six submaximal exercise bouts varied between 20 and 75% of the peak $\dot{V}O_2$. Oxygen uptake was measured using open-circuit spirometry techniques (MedGraphics, Cardiorespiratory Diagnostic Systems). Oxygen uptake values measured at 9 and 10 min were averaged and reported as the steady-state $\dot{V}O_2$ for the corresponding power output.

Table 1 Physical characteristics of the subjects. Values presented are means (SE). (*LBM* Lean body mass, *AMM* active muscle mass for cycling, including leg and gluteal muscle groups.) LBM and AMM do not include bone mineral content

	Age (years)		Height (cm)		Body mass (kg)		LBM (kg)		Body fat (%)		AMM (kg)	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Males	23.3	1.1	175.1	1.3	77.9	2.2	58.0	1.2	17.6	1.4	28.4	0.8
Females	24.7	1.4	169.6	1.5	62.7	1.6	43.0	1.0	23.9	1.8	21.9	0.5
P value	0.441		0.008*		0.000*		0.000*		0.006*		0.000*	

* Significant difference between males and females

Thirty minutes of recovery was allowed between each submaximal exercise bout. Heart rate and rhythm were monitored continuously during the test using a CM5 electrode configuration and a Lohmeier electrocardiograph (M 607). Data collected from the six submaximal exercise bouts were used to establish the $\dot{V}O_2$–power relationship. The linear regression of the $\dot{V}O_2$–power relationship was used to calculate the power output corresponding to 120% of the peak $\dot{V}O_2$. The calculated power output was used in the subsequent MAOD test. This method of estimating the power output associated with a given level of oxygen demand is described elsewhere (Medbø et al. 1988).

Determination of peak oxygen uptake

The peak $\dot{V}O_2$ for cycling was determined using a continuous, incremental exercise protocol conducted on a Lode cycle ergometer. Pedal rate was maintained at 70 rev · min^{-1} throughout the test. After 3 min of unloaded cycling, the power output was increased by 20 W · min^{-1} for females and 25 W · min^{-1} for males until exhaustion. Heart rate and rhythm were monitored continuously and the $\dot{V}O_2$ was measured breath-by-breath (MedGraphics, Cardiorespiratory Diagnostic Systems) and averaged over 30-s intervals. The two highest $\dot{V}O_2$ values were averaged and reported as the peak $\dot{V}O_2$ for cycling.

The MAOD test for cycling

All MAOD tests were performed at 7 a.m. following an overnight fast. Subjects warmed up by cycling at 50 W for 5 min followed by 2 min of unloaded cycling at 70 rev · min^{-1}. The linear regression equation of the six submaximal cycling bouts was used to calculate a power output predicted to elicit 120% of the peak $\dot{V}O_2$ for cycling. This workload was then applied immediately following the 7-min warm-up period. Heart rate and rhythm were monitored continuously and $\dot{V}O_2$ was measured breath-by-breath (MedGraphics, Cardiorespiratory Diagnostic Systems) throughout the exercise bout. The test was terminated when the subject could no longer maintain the pedalling frequency above 60 rev · min^{-1} despite verbal encouragement. The time to exhaustion for the MAOD test was recorded to the nearest second. The MAOD for each subject was calculated in oxygen consumption equivalents as the difference between the accumulated oxygen demand of exercise and the measured oxygen uptake during the MAOD test. Absolute MAOD values were reduced by 9% to correct for reductions in the O_2 stores of the body (Medbø et al. 1988). MAOD values were expressed in absolute terms and relative to body mass (BM), LBM and the estimated AMM for cycling. Previously (unpublished data), we have demonstrated that this method of determining MAOD for cycling is highly repeatable in untrained male and female subjects (intra-class correlation coefficients of 0.983 for time to exhaustion and 0.968 for MAOD values).

Determination of body composition and AMM

The same technician performed a whole-body scan on each subject using DEXA (QDR 4500 Hologic, Waltman, Mass., USA) to assess body composition. Whole-body values were presented as total mass (g) and separately for fat mass (g and % of total body mass) and LBM (g). The total LBM for both legs and the gluteal muscle group was measured and reported as the AMM (kg) for cycling. Lean body mass and AMM are reported independently of BMC.

Blood lactate and plasma catecholamine concentrations

Thirty minutes prior to the MAOD test, a 22-gauge catheter was inserted into the subject's antecubital vein. The catheter was flushed with heparinized saline to maintain patency. Five milliliters of blood was obtained at rest, immediately at exhaustion and at 1, 3, 5, 10, 15, 20 and 30 min of recovery. Whole-blood [Lac^-] was determined using an automated blood Lac^- analyzer (Yellow Spring Instruments, Model 2700) and a cell-lysing agent. In addition, plasma was separated and stored at −70°C until the samples could be analyzed for norepinephrine and Epi concentrations using high-performance liquid chromatography with electrochemical detection (Schneider et al. 1992, 2000; Waters Plasma Catecholamine Manual 1986). The between-day and within-day coefficients of variation were less than 5% and 3% for both Epi and norepinephrine, respectively.

Statistical analysis

Results are presented as means and standard error (SE) of the mean. Independent *t*-tests were used to determine gender-dependent differences in physical characteristics and the peak exercise values. Regression analysis was used to determine relationships between MAOD, peak [Lac^-] and peak [Epi]. Blood Lac^- and plasma catecholamine concentrations were compared for gender differences using analysis of variance with a repeated-measures design for time. Newman-Keuls post-hoc analysis was used where appropriate. Statistical significance was accepted at $P < 0.05$.

Results

Physical characteristics of the subjects

There was no significant difference in the mean ages of the two groups. Females had a significantly higher percentage body fat and lower mean values for BM, LBM and AMM than males (see Table 1).

Peak exercise values for incremental cycling

The peak exercise values obtained during incremental cycling are given in Table 2. Peak $\dot{V}O_2$ expressed in absolute terms and relative to BM (ml · kg^{-1} · min^{-1}) was significantly higher for males than for females. However, when peak $\dot{V}O_2$ was expressed relative to LBM (ml · kg LBM^{-1} · min^{-1}), no difference was found between the two groups. Males achieved a significantly higher peak power output during incremental cycling than females. There were no significant gender-dependent

Table 2 Peak values obtained during incremental cycling. Values presented are means (SE). (*LBM* Lean body mass, *Peak* $\dot{V}O_2$ peak oxygen uptake)

	Peak $\dot{V}O_2$			Peak power (W)
	(l · min^{-1})	(ml · kg^{-1} ·min^{-1})	(ml · kg LBM^{-1} · min^{-1})	
Males	3.43 (0.16)	43.4 (1.5)	59.2 (2.6)	315 (14)
Females	2.41 (0.11)	38.5 (1.8)	55.9 (1.6)	237 (10)
P value	0.000*	0.036*	0.301	0.000*

* Males significantly higher than females

differences in peak HR [males 195 (4) versus females 193 (3) beats · min^{-1}) or respiratory exchange ratio (RER) [males 1.25 (0.02) versus females 1.24 (0.02)] values.

MAOD test results

Males [331 (16.7) W] cycled at a higher power output than females [247 (11.6) W] and performed significantly more total work [51.5 (3.8) versus 34.5 (1.9) kJ]. However, when expressed relative to LBM (W · kg LBM^{-1}), males and females cycled at similar relative power outputs [5.7 (0.3) versus 5.7 (0.2) W · kg LBM^{-1}]. The time to exhaustion for the MAOD test was not significantly different between the two groups. Males obtained a significantly greater absolute MAOD for cycling than females (see Table 3). In addition, when MAOD was expressed relative to AMM (ml · kg AMM^{-1}), males obtained a significantly higher MAOD than females.

Blood lactate and plasma catecholamine responses to supramaximal cycling

The change in blood [Lac^-] following the MAOD test for male and female subjects is presented in Fig. 1. While the resting blood [Lac^-] was not significantly different for the two groups, the mean peak blood [Lac^-] of 13.6 (0.9) mmol · l^{-1} for males was significantly higher than the value of 10.0 (1.0) mmol · l^{-1} obtained for females. Blood [Lac^-] remained higher for males than for females throughout recovery. However, gender-dependent differences were not found to be significant at 3 and 5 min.

No significant difference between males and females was found for resting plasma Epi or norepinephrine concentrations. The mean peak plasma [Epi] obtained after exercise was 68% higher for males than females, but the difference was not statistically significant ($P = 0.066$). However, plasma [Epi] was significantly higher for males than females at 1 and 3 min of the recovery period (see Fig. 2). No gender-dependent differences in [Epi] were observed after 3 min of recovery. The mean peak plasma norepinephrine concentration of 4192 (548) pg · ml^{-1} obtained for males was *not* significantly different from the value of 4392 (491) pg · ml^{-1} obtained for females ($P = 0.789$). In addition, there were no gender-dependent differences in plasma norepinephrine concentration measured at any time after exercise. Plasma Epi and norepinephrine concentrations returned to resting levels by 10 min of the recovery period for both groups.

Table 3 Time to exhaustion and maximal accumulated oxygen deficit values. Values presented are means (SE). (*AMM* Active muscle mass for cycling, *BM* body mass, *LBM* lean body mass)

	Time to exhaustion (s)	Maximal accumulated oxygen deficit			
		(l)	(ml · kg BM^{-1})	(ml · kg LBM^{-1})	(ml · kg AMM^{-1})
Males	161 (16)	3.6 (0.2)	46.3 (2.4)	61.8 (2.7)	126.3 (5.6)
Females	140 (5)	2.4 (0.2)	38.2 (2.6)	55.2 (3.1)	108.3 (6.1)
P value	0.257	0.000*	0.035*	0.126	0.043*

* Significant difference between males and females

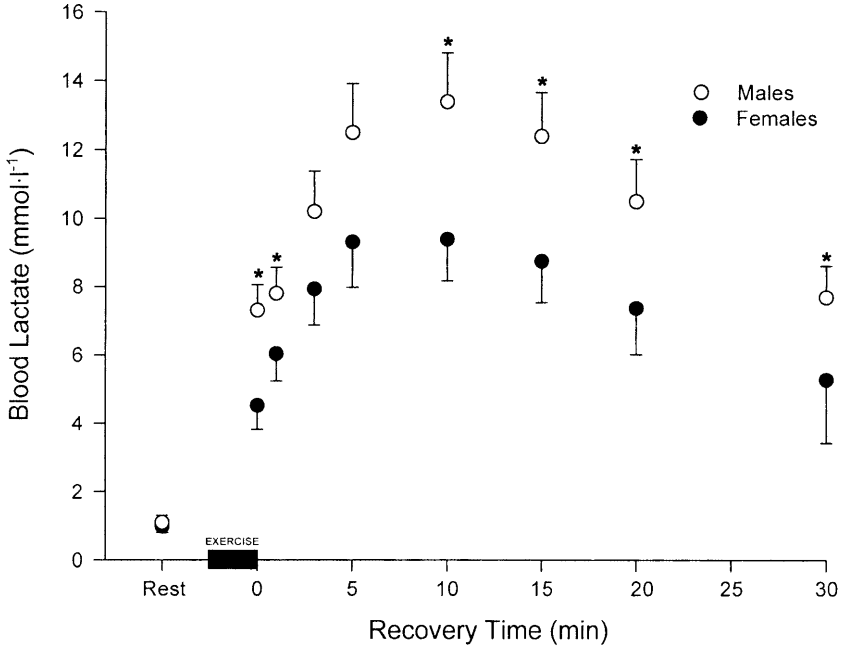

Fig. 1 Blood lactate concentration measured prior to exercise, immediately at exhaustion and at 1, 3, 5, 10, 15, 20 and 30 min of recovery. *Males significantly higher than females

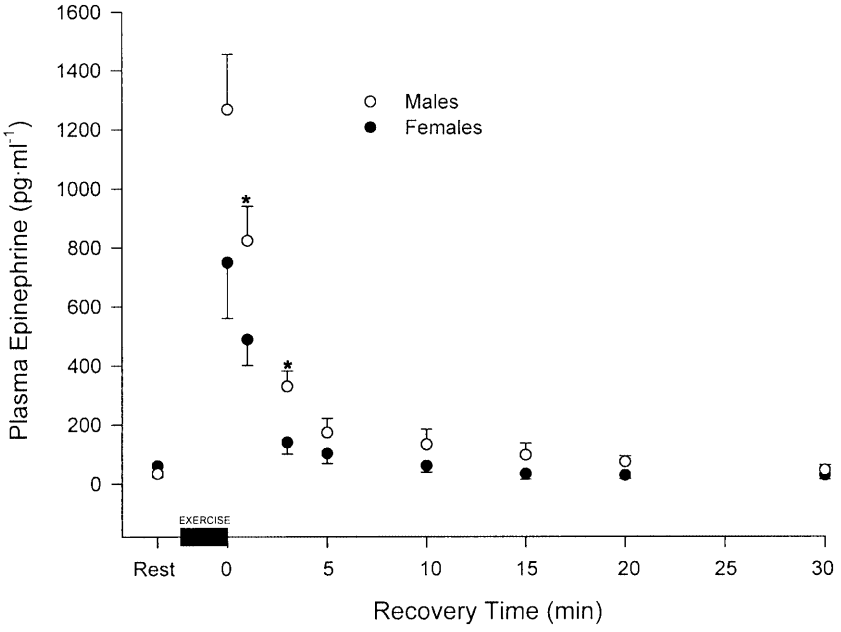

Fig. 2 Plasma epinephrine concentration measured prior to exercise, immediately at exhaustion and at 1, 3, 5, 10, 15, 20 and 30 min of recovery. *Males significantly higher than females

Relationships between MAOD and blood variables

Peak [Lac^-] was significantly correlated with MAOD expressed relative to AMM ($ml \cdot kg\ AMM^{-1}$) in females ($r = 0.75$, $P = 0.010$), but not in males ($r = 0.09$, $P = 0.412$). Peak plasma [Epi] was not significantly correlated with MAOD ($ml \cdot kg\ AMM^{-1}$) or peak [Lac^-] in either group.

Discussion

The present study demonstrates that untrained male subjects have a higher MAOD for cycling than untrained female subjects when expressed both in absolute terms and relative to the AMM for cycling. Males maintained a 17% higher MAOD even when values were expressed relative to AMM. We used DEXA in the present study to assess body composition and to provide a precise estimate of the AMM for cycling. In addition, the AMM for cycling included the LBM of the legs and the gluteal muscle group and excluded BMC. Winter et al. (1991) suggested that the gluteal muscle group is one of the major muscle groups involved in cycling and is largely ignored when traditional methods of determining the AMM for cycling are used. No previous study has used DEXA to provide an accurate estimate of the AMM for cycling.

The use of both endurance- and sprint-trained male and female subjects in previous studies (Naughton et al. 1997; Weyand et al. 1993) has contributed to some of the uncertainty surrounding gender-dependent differences in AC. Medbø and Burgers (1990) reported significantly higher MAOD values for sprint-trained athletes than for endurance-trained athletes, whereas the MAOD for running was *not* significantly different between endurance athletes and untrained subjects. These researchers (Medbø and Burgers 1990) also found that high-intensity interval training significantly increased MAOD values in males but not in females. Thus, differences in MAOD between male and female athletes may be partly caused by training adaptations. The present study eliminated the effect of training on MAOD by including only untrained subjects. A study by Medbø and Burgers (1990) also found higher MAOD values for untrained males than for untrained females, but these investigators did not express MAOD values relative to AMM. The greater MAOD expressed relative to the AMM for cycling found in males in the present study suggests that there are physiological, biochemical and/or structural differences between the muscle tissue of untrained males and females (Komi and Karlsson 1978; Sale and Spriet 1996; Simoneau and Bouchard 1989).

The calculation of MAOD in oxygen consumption equivalents partly depends on the estimation of the oxygen cost (demand) of the exercise performed at an intensity that exceeds the peak $\dot{V}O_2$. The oxygen cost of supramaximal exercise was calculated from the $\dot{V}O_2$–power relationship established from six submaximal exercise bouts. The assumption of similar mechanical efficiencies across such a wide range of exercise intensities and the assumption that the $\dot{V}O_2$–power relationship is linear have recently been questioned (Bangsbo 1996; Green and Dawson 1995). However, even if the calculation of the absolute oxygen cost during the MAOD test is not precise, the main finding of the present study would not change, because of a constant error in the calculation of MAOD for both groups. The measurements of MAOD in the present study provide an estimate of anaerobic energy production that is useful for comparative purposes where an identical methodology

has been used and the assumptions associated with this method are the same for both groups. Furthermore, the correlation coefficients for the $\dot{V}O_2$–power relationship were similar for male ($r = 0.996 \pm 0.001$) and female ($r = 0.995 \pm 0.001$) subjects, suggesting that any error associated with the regression analysis was similar for the two groups.

Although blood [Lac^-] represents the balance between Lac^- production and appearance in the blood and the rate of Lac^- removal from the blood, [Lac^-] is widely used as an index of the rate of anaerobic glycolysis during high-intensity exercise (Naughton et al. 1997; Saltin 1990). It is not likely that the higher blood Lac^- concentrations in males compared to females after the MAOD test were caused by Lac^- being removed more slowly from the blood. In the present study, the decline in [Lac^-] between 10 and 30 min of the recovery period was significantly greater in males [$\Delta 5.7$ (0.4) mmol $\cdot$ l^{-1}] than in females [$\Delta 4.1$ (0.5) mmol $\cdot$ l^{-1}]. Therefore, the difference in blood [Lac^-] between male and female subjects suggests a greater rate of Lac^- production rather than a slower rate of Lac^- removal from the blood.

The higher blood [Lac^-] obtained after exercise in males suggests that males are able to utilize anaerobic glycolysis to a greater extent than females during activities with a high anaerobic component. Early work by Jacobs et al. (1982, 1983) indicated that the maximal rate of muscle glycogenolysis during a 30-s cycle sprint was lower in females than in males. However, the peak [Lac^-] and MAOD (ml $\cdot$ kg $\cdot$ AMM^{-1}) values obtained in the present study were not significantly correlated for male subjects, whereas this relationship was significant for data from female subjects. Gratas-Delamarche et al. (1994) also demonstrated that, for males, the peak [Lac^-] is not significantly correlated with work (W $\cdot$ kg BM^{-1}) during 30 s of supramaximal cycling, while this relationship was found to be significant for the females' data. These researchers suggested that a successful performance during 30 s of supramaximal cycling is independent of the rate of anaerobic glycolysis in males. Also, Bangsbo et al. (1993) demonstrated similar findings for male soccer players, cyclists and runners, reporting that a high peak [Lac^-] was not related to a high MAOD. It may be concluded from the present study and from the results presented by other researchers that anaerobic glycolysis cannot account for the higher MAOD found in males compared to females.

The influence of catecholamines on lactate production during submaximal exercise is well established (Kindermann et al. 1982; Mazzeo 1991; Mazzeo and Marshall 1989; Schneider et al. 2000; Stainsby et al. 1984). Naughton et al. (1997) suggested that gender-related differences in the plasma catecholamine responses to supramaximal exercise might contribute to the higher blood [Lac^-] found in male compared to female subjects. Therefore, the higher [Lac^-] observed in males might be partly caused by an increased ability to stimulate muscle glycogenolysis/glycolysis via Epi secretion. The increase in [Epi] induced by the MAOD test in the present study was found to be significantly greater in males than in females. Previous research has also demonstrated a significantly greater plasma Epi response to supramaximal exercise in males than in females (Brooks et al. 1990; Gratas-Delamarche et al. 1994). Despite male subjects having a greater mean Epi response to supramaximal exercise, a significant relationship between peak [Epi] and peak [Lac^-] was *not* observed in the present study for either male or female subjects. Thus, there is no strong physiological link between peak [Lac^-] and peak [Epi] and the males' greater Lac^- response to supramaximal exercise was probably not caused by enhanced secretion of Epi. The results of the present study indicate that muscle glycogenolysis during supramaximal exercise is not essentially controlled by Epi release. Other factors, such as a rise in inorganic phosphate (Chasiotis 1988; Hirvonen et al. 1987), ADP (Bangsbo et al. 1992; Nevill et al. 1989) and cellular Ca^{2+} (Richter et al. 1982) levels, are important to the regulation of muscle glycogenolysis during exercise. The significant contribution of increased cellular Ca^{2+}, ADP and inorganic phosphate concentrations to the control of glycogenolysis might explain the poor correlation obtained between peak blood [Lac^-] and peak [Epi] in both male and female subjects in the present study.

Epinephrine, as well as having regulatory effects on metabolism within muscle, must also be considered for its effects on active muscle blood flow. Despite extensive investigation, the control of blood flow during dynamic exercise is not fully understood. There is limited evidence to suggest that sympathetic beta-adrenergic receptors play a major role in increasing active muscle blood flow (Buckwalter et al. 1998). However, it is possible that male subjects, with their increased secretion of Epi, were able to increase muscle blood flow to a greater extent compared to female subjects.

Increased muscle blood flow could improve muscle function through an oxygen-independent mechanism. This is thought to involve the removal of metabolites such as hydrogen ions and delay the onset of muscle fatigue. Nevertheless, peak plasma [Epi] was not significantly correlated with MAOD or peak [Lac^-] in either group.

The norepinephrine concentration measured in plasma has previously been used as an index of sympathetic nervous system (SNS) activity (Mazzeo 1991). The norepinephrine responses to the MAOD test were similar for males and females in the present study and suggest that the level of SNS activity during exercise was the same for both groups. This was also supported by the similar peak heart rates obtained during the MAOD test for male [184(5) beats $\cdot$ min^{-1}] and female [181(4) beats $\cdot$ min^{-1}] subjects in the present study. The catecholamine response of male and female subjects to supramaximal exercise reported in the present study is similar to that reported elsewhere. Brooks et al. (1990) and Gratas-Delamarche et al. (1994) found that males had higher Epi concentrations in plasma after supramaximal

exercise than females, but there were no such gender-dependent differences for norepinephrine. The greater Epi response to supramaximal cycling at the same level of SNS activity suggests that males have a greater capacity for adrenomedullary secretion compared to females. However, the mechanism responsible for the greater secretion of Epi in males than in females has not been determined.

In summary, males had a greater absolute MAOD than females and maintained a 17% greater MAOD than females, even when values were expressed relative to the AMM for cycling. The exercise-induced blood Lac^- and plasma Epi responses to supramaximal cycling were greater in males than in females. The greater MAOD demonstrated by males in the present stydy was not due to a greater ability to produce Lac^- or to enhanced secretion of Epi.

References

Bangsbo J (1996) Bangsbo responds to Medbø's paper. Can J Appl Physiol 21: 384–388

Bangsbo J, Graham T, Johansen L, Strange S, Christensen C, Saltin B (1992) Elevated muscle acidity and energy production during exhaustive exercise in humans. Am J Physiol 263: R891–R899

Bangsbo J, Michalsik L, Petersen A (1993) Accumulated O_2 deficit during intense exercise and muscle characteristics of elite athletes. Int J Sports Med 14: 207–213

Brooks S, Nevill ME, Meleagros L, Lakomy HKA, Hall GM, Bloom SR, Williams C (1990) The hormonal responses to repetitive brief maximal exercise in humans. Eur J Appl Physiol 60: 144–148

Buckwalter JB, Ruble SB, Mueller PJ, Clifford PS (1998) Skeletal muscle vasodilation at the onset of exercise. J Appl Physiol 85: 1649–1654

Carlson J, Naughton GA (1993) An examination of the anaerobic capacity of children using maximal accumulated oxygen deficit. Pediatr Exerc Sci 5: 60–71

Chasiotis D (1988) Role of cyclic AMP and inorganic phosphate in the regulation of muscle glycogenolysis during exercise. Med Sci Sports Exerc 20: 545–550

Fuller NJ, Laskey MA, Elia M (1992) Assessment of the composition of major body regions by dual-energy x-ray absorptiometry (DEXA) with special reference to limb muscle mass. Clin Physiol 12: 253–266

Gastin PB (1994) Quantification of anaerobic capacity. Scand J Med Sci Sports 4: 91–112

Gratas-Delamarche A, LeCam R, Delamarche P, Monnier M, Koubi H (1994) Lactate and catecholamine responses in male and female sprinters during a Wingate test. Eur J Appl Physiol 68: 362–366

Green S, Dawson BT (1993) Measurement of anaerobic capacities in humans: definitions, limitations and unsolved problems. Sports Med 15: 312–327

Green S, Dawson BT (1995) The oxygen uptake-power regression in cyclists and untrained men: implications for the accumulated oxygen deficit. Eur J Appl Physiol 70: 351–359

Griffith GE (1997) An electromyographic comparison of seated and standing up-hill cycling. Microform Publications, International Institution for Sport and Human Performance, University of Oregon, Eugene, Ore.

Hirvonen J, Rehunen S, Rusko H, Harkonen M (1987) Breakdown of high-energy phosphate compounds and lactate accumulation during short supramaximal exercise. Eur J Appl Physiol 56: 253–259

Jacobs I, Bar-Or O, Karlsson J, Dotan R, Tesch P, Kaiser P, Inbar O (1982) Changes in muscle metabolites in females with 30-s exhaustive exercise. Med Sci Sports Exerc 14: 457–460

Jacobs I, Tesch PA, Bar-Or O, Karlsson J, Dotan R (1983) Lactate in human skeletal muscle after 10 and 30 s of supramaximal exercise. J Appl Physiol 55: 365–367

Kindermann W, Schnabel A, Schmitt WM, Biro G, Cassens J, Weber F (1982) Catecholamines, growth hormone, cortisol, insulin and sex hormone in anaerobic and aerobic exercise. Eur J Appl Physiol 49: 389–399

Komi PV, Karlsson J (1978) Skeletal muscle fiber types, enzyme activities and physical performance in young males and females. Acta Physiol Scand 103: 210–218

Maud PJ, Schultz BB (1986) Gender comparisons in anaerobic power and anaerobic capacity test. Br J Sports Med 20: 51–54

Mazzeo RS (1991) Catecholamine responses to acute and chronic exercise. Med Sci Sports Exerc 23: 839–845

Mazzeo RS, Marshall P (1989) Influence of plasma catecholamines on the lactate threshold during graded exercise. J Appl Physiol 67: 1319–1322

Medbø JI, Burgers S (1990) Effects of training on the anaerobic capacity. Med Sci Sports Exerc 22: 501–507

Medbø JI, Mohn AC, Tabata I, Bahr R, Vaage O, Sejersted OM (1988) Anaerobic capacity determined by maximal accumulated oxygen deficit. J Appl Physiol 64: 50–60

Mohr TM, Allison JD, Patterson R (1981) Electromyographic analysis of the lower extremity during pedaling. J Orthop Sports Phys Therap 2: 163–170

Naughton GA, Carlson JS, Buttifant DC, Selig SE, Meldrum K, McKenna MJ, Snow RJ (1997) Accumulated oxygen deficit measurements during and after high-intensity exercise in trained male and female adolescents. Eur J Appl Physiol 76: 525–531

Nevill ME, Boobis LH, Brooks S, Williams C (1989) Effect of training on muscle metabolism during treadmill sprinting. J Appl Physiol 67: 2376–2382

Ramsbottom R, Nevill ME, Nevill AM, Hazeldine R (1997) Accumulated oxygen deficit and shuttle run performance in physically active men and women. J Sports Sci 15: 207–214

Richter EA, Ruderman NB, Gavras H, Belur ER, Galbo H (1982) Muscle glycogenolysis during exercise: dual control by epinephrine and contraction. Am J Physiol 242: E25–E32

Sale DG, Spriet LL (1996) Skeletal muscle function and energy metabolism. In: Bar-Or O, Lamb DR, Clarkson PM (eds) Perspectives in exercise science and sports medicine, Vol. 9. Cooper, Carmel, Ind., pp 289–359

Saltin B (1990) Anaerobic capacity: past, present and prospective. In: Taylor AW (ed) Biochemistry of exercise, VII. Human Kinetics, Champaign, Ill., pp 387–412

Schneider DA, McGuiggin ME, Kamimori GH (1992) A comparison of the blood lactate and plasma catecholamine thresholds in untrained male subjects. Int J Sports Med 13: 562–566

Schneider DA, McLellan TM, Gass GC (2000) Plasma catecholamine and blood lactate responses to incremental arm and leg exercise. Med Sci Sports Exerc 32: 608–613

Simoneau JA, Bouchard C (1989) Human variation in skeletal muscle fiber type proportion and enzyme activities. Am J Physiol 257: E567–E572

Stainsby WN, Summer SC, Andrew GM (1984) Plasma catecholamines and their effect on blood lactate and muscle lactate output. J Appl Physiol 57: 321–325

Waters Plasma Catecholamine Manual (1986) Manual No.10530. Waters Chromography Division, Milford Mass., USA

Weyand PG, Cureton KJ, Conley DS, Higbie EJ (1993) Peak oxygen deficit during one- and two-legged cycling in men and women. Med Sci Sports Exerc 25: 584

Winter EM, Brookes FBC, Hamley EJ (1991) Maximal exercise performance and lean leg volume in men and women. J Sports Sci 9: 3–13