Amino Acids (2010) 38:1351–1359
DOI 10.1007/s00726-009-0343-7

ORIGINAL ARTICLE

Plasma catecholamine and nephrine responses following 7 weeks of sprint cycle training

Richard Michael Bracken · Stephen Brooks

Received: 1 July 2009 / Accepted: 25 August 2009 / Published online: 10 September 2009

Abstract The catecholamine metabolites normetanephrine (NMET) and metanephrine (MET) increase in response to acute exercise. However, changes in catecholamine 'nephrines' during sprint training are unclear. Therefore, the aim of this study was to examine the plasma nephrine and catecholamine (noradrenaline, NA; adrenaline, AD) responses to a laboratory-based cycle test before and after a 7-week period of cycle sprint training. Ten healthy men completed a 2-min cycle test at a power output equivalent to 110% of pre-training VO_2max before and after 7 weeks of laboratory based sprint cycle training, three times per week. Resting and post-sprint venous blood samples were taken. Resting plasma nephrines and catecholamines increased significantly following exercise ($P < 0.05$). Post-exercise NA and NMET were reduced after training ($P < 0.05$) and a trend for a reduction in AD ($P = 0.09$) and MET ($P = 0.07$) was observed. The results demonstrate a reduction in exercise-induced increases in plasma nephrine concentrations following sprint training. This suggests catechol-*O*-methyl transferase activity is coupled to high intensity cycle exercise. These findings may aid in the understanding of catecholamine regulation during high intensity exercise and sprint training.

R. M. Bracken (✉)
Sport and Exercise Science Research Centre,
School of Engineering, Swansea University,
Singleton Park, Swansea SA2 8PP, UK
e-mail: R.M.Bracken@swansea.ac.uk

S. Brooks
Department of Physiology and Sport Sciences,
Faculty of Health and Human Sciences,
Coventry University, Priory Street, Coventry CV1 5FB, UK

Keywords Metanephrine · Normetanephrine · Maximal exercise · Catecholamines · Training

Introduction

Changes in plasma catecholamine concentrations (noradrenaline NA, adrenaline AD) provide an important indicator of alterations to whole body sympatho-adrenal activity during physical exercise (Kjær 1999). Catecholamines play important roles in the cardiovascular, metabolic and immune systems and in determining exercise capacity by acting as both neurotransmitters and hormones (Kaiser et al. 1986; Winder et al. 1987).

Metabolism of catecholamines is important for circulatory clearance of biologically potent biogenic amines thus preventing prolonged stimulation of tissue adrenoceptors and consequent elevations in cardiorespiratory and metabolic rates. *O*-methylation of NA and AD by catechol-*O*-methyl transferase (COMT) produces the 'nephrines' normetanephrine (NMET) and metanephrine (MET), respectively. Clinically, nephrines have been shown to be good markers of both increased (Raber et al. 2000) and decreased (Merke et al. 2000) sympatho-adrenal activity. Furthermore, it has previously been demonstrated that the concentrations of urinary (Pequignot et al. 1978) and plasma (Zamecnik 1997; Raber et al. 2003; Bracken et al. 2009) nephrines increase in response to various forms of exercise below and above the maximal rate of oxygen consumption. We have previously reported that plasma nephrine and catecholamine responses to repeated cycle sprints increased with peak NMET increasing 60% and MET 230% from resting values. Furthermore, changes in plasma nephrine concentrations explained 33–36% of the variance in parent amine concentrations between

participants (Bracken et al. 2009). The above-mentioned findings suggest a role for exercise-induced increases in COMT activity that may aid in metabolising the increased catecholamine concentrations with exercise.

The changes in plasma catecholamines following various forms of exercise training have been characterised. The magnitude of the plasma catecholamine response to an acute bout of endurance exercise is dependent on the exercise duration and the relative stress of physical activity, i.e. the exercise intensity at a certain percentage of maximal oxygen consumption (Kjær 1998). Therefore, the plasma NA and AD response to sub-maximal exercise after a period of aerobic training are blunted when performed at the same absolute workload (Winder et al. 1978, 1979). The effect of endurance training on the plasma catecholamine response to sub-maximal exercise at the same relative intensity increases (Greiwe et al. 1999; Hagberg et al. 1984; Winder et al. 1978), decreases (Hartley et al. 1972) or is unaltered (Brooks et al. 1984; Peronnet et al. 1981; Winder et al. 1979). Similar results have been found with sprint training programmes where, for example, an augmented plasma NA and AD response to a single bout of cycle exercise performed at 130% VO_2max to exhaustion following 7 weeks of sprint training was demonstrated but on a separate occasion when workload and time were matched to pre-training volumes, peak plasma NA was significantly reduced post-training whereas plasma AD was not different (Harmer et al. 2000).

Though there is considerable research on the catecholamine responses to various types of physical training there is a severe paucity of research examining the impact of physical training on plasma nephrine concentrations. One study examined the urine catecholamine and nephrine responses to 4 days of increased cycle training in national level road cyclists (Filaire et al. 2002). Increased urine concentrations of both nephrines were observed during the post-training recovery period for up to 48 h. Following 72 h MET returned to baseline but urine NMET concentration was lower than the control values. Another study from the same group looked at the same indices after 8 months and found significant increases in urine MET in response to a progressive increase in training load (Filaire et al. 2004). These studies provide useful information on the gross changes in urine nephrines but are limited in the absence of corresponding catecholamine data. Furthermore, given the short half-life of plasma catecholamines and nephrines (2–3 and < 4 min, respectively, Goldstein et al. 2003) the measurement of plasma concentrations would offer an advantage in better understanding the changes in catecholamine metabolism in response to a period of physical exercise and training.

It remains to be determined what effects, if any, a defined period of sprint training has on exercising plasma nephrine concentrations. Sports and exercise activities often elicit high intensity bouts and are frequently interspersed with limited recovery periods. Such 'intermittent activities' are seen in a wide variety of racquet sports (tennis, squash, badminton), court games (basketball, volleyball, netball) and field games (football, rugby, hockey). There is limited information on the effects of regular training in this type of exercise on the sympatho-adrenal system. Therefore, the aim of this study was to examine the plasma MET, NMET, NA and AD responses to a laboratory-based cycle test before and after a 7-week period of cycle sprint training.

Materials and methods

Participants

With written University Research Ethics Committee approval, ten healthy but non-specifically active male participants volunteered to take part in this study. After receiving a full explanation of the protocol, all participants completed written informed consent and medical history forms prior to participation in the study. The physical and physiological characteristics of the participants were age 20 ± 2 years, stature 177 ± 5 cm, body mass 79.9 ± 8.4 kg and estimated body fat 15.2 ± 3.0%.

Study design

Participants attended the laboratory 25 times, once for familiarisation on the cycle ergometers (Bosch ERG 551 and Monark 824e) and preliminary measurement of maximal rate of O_2 consumption, twice for the experimental trials and a further 21 occasions over 7 weeks to perform training (three sessions per week). There was at least one full day of rest between training sessions.

Preliminary testing

At least 1 week prior to the experimental trials participants reported to the laboratory for an incremental workload test on an electromagnetically braked cycle ergometer (Bosch ERG 551) to determine VO_2max. The initial workload was set at 75 W and increased by 25 W every 3 min thereafter until volitional exhaustion or when participants could not maintain a cadence of at least 40 rev min^{-1}. Expired air samples were collected in the last minute of each stage and were subsequently analysed for fractional concentration of oxygen (F_EO_2) and carbon dioxide (F_ECO_2) by sampling through a paramagnetic transducer (Servomex series 1100, Crowborough, UK) and an infrared analyser (Servomex Model 1490). The oxygen and carbon dioxide analysers

were calibrated before each test using certified gases (British Oxygen Co., London, UK). The minute ventilation (V_E) was determined using a dry gas meter (Harvard Ltd, Edenbridge, UK) previously calibrated against a precision 3 l gas syringe (Hans Rudolph, Kansas City, MO, USA). All participants fulfilled the criteria for attainment of the maximal rate of O_2 consumption (VO_2max), namely a heart rate of $\geq$ 95% of age predicted maximum before the final exercise intensity and/or respiratory exchange ratio values were $\geq$ 1.00. Exercise workloads for the experimental trials were determined by regression analysis of incremental VO_2 and workload values. Participants performed the same incremental test at least 1 day after the last training session and 48 h prior to the final experimental test.

Main exercise trial procedures

Before arriving at the laboratory for the main experimental trial, participants were instructed to abstain from strenuous activity and to avoid foods containing high biogenic amine content or caffeinated drinks for 24 h prior to testing (Bracken et al. 2005) as the influence of foodstuffs/drinks containing high concentrations of these amines such as cheese, coffee, chocolate and red wine may adversely affect resting endogenous concentrations. On arrival to the laboratory for the experimental trial stature was measured to the nearest 0.1 cm (Seca Stadiometer 208), body mass, measured to the nearest 0.1 kg (Seca Beam Balance 710) and estimated percentage body fat using skinfold calipers were determined using the equations of Durnin and Womersley (1974) with all measurements taken from the right hand side of the body using a Harpenden skinfold callipers (John Bull, England). Following this, a heart rate monitor was placed around the subject's chest (Polar Accurex *Plus* HRM, Polar Instruments Ltd, Finland). Participants were then seated whilst a 21-gauge catheter was placed in an antecubital vein. Saline was infused periodically to keep the catheter patent. Twenty minutes later resting blood samples (10 ml + 1 ml) were taken. Following this, participants mounted the cycle ergometer and sat quietly for 5 min before beginning the cycle test which was performed at a power output required to elicit 110% of VO_2max for 2 min. Following training, the post-training experimental test consisted of cycling at the same power output that elicited 110% of the pre-training VO_2max.

Blood samples treatment

Blood samples (10 ml) were taken at rest and immediately following exercise. An additional 1 ml of venous blood was obtained and immediately capped, kept on ice then measured within 1 h for blood pH. The rest of the blood sample was placed in 10 ml lithium-heparinised tubes. From this sample, duplicate aliquots (100 µl) of blood were immediately deproteinised in 1 ml of ice-cold perchloric acid (0.3 M) and the concentration of blood lactate was later determined using an enzymatic assay (Maughan 1982). Haemoglobin concentration was determined using the cyanomethaemoglobin technique (Sigma Diagnostics kit no. 525-A) and packed cell volume was determined using the capillary centrifuge technique from which changes in plasma volume (PV) were estimated (Dill and Costill 1974). A 1 ml aliquot of plasma was stored frozen (−70°C) until later determination of NMET and MET concentrations using a commercially available enzyme linked immunoassay kit (Labor Diagnostica Nord, Germany Code: BA 10-1400). Briefly, plasma placed in lithium-heparinised tubes was used for determination of plasma nephrines. The assay first acylated the plasma samples to their corresponding N-acyl-derivatives and based upon a competitive enzyme immunoassay used a microtiter plate format. MET and NMET were bound to the solid phase of the microtiter plate. The acylated sample nephrines and solid phase nephrines competed for a fixed number of antiserum binding sites and when the system was in equilibrium, free antigen and free antigen–antiserum complexes were removed by washing. The antibody bound to the solid phase catecholamine metabolite was detected by an anti-rabbit IgG–peroxidase conjugate using TMB as a substrate. The reaction was measured at 450 nm with the amount of antibody bound to the solid phase nephrines inversely proportional to the nephrine concentration.

To the remainder of the blood sample 200 µl of 0.1 mol l^{-1} of both ethylene glycol bis-(β-aminoethyl ether)-N',N',N',N'-tetraacetic acid as anticoagulant and glutathione as antioxidant were added. Samples were centrifuged for 10 min at 3,000 rev min^{-1} with the plasma separated and stored at −20°C for later analysis of catecholamines. Plasma NA and AD concentrations were determined according to an alumina extraction method with minor modifications using HPLC with electrochemical detection (Gilson HPLC pump, model 307: Gilson electrochemical detector, model 141; Bracken et al. 2005). One hundred microlitres of sample was injected into a HPLC column (Spherisorb S5 ODS2) and eluted with a mobile phase (monochloroacetic acid 11.14 g l^{-1}, NaOH 3.37 g l^{-1}, NaOS 0.74 g l^{-1} and EDTA 0.09 g l^{-1}, pH 3.0). Before separating plasma catecholamines in the HPLC column, a standard solution containing 100 nmol l^{-1} NA, AD, dihydroxybenzylamine; $(HO)_2C_6H_3CH_2NH_2\cdots HBr$; MW 220.1, Sigma-Aldrich) and DA was injected several times until retention times and peak heights were identical and the base line was stable. The flow rate was set at 2 ml min^{-1}. The chromatogram was analysed by computer

Table 1 Summary of 7-week sprint training programme performed by participants ($n = 10$)

	Week 1	Week 2	Week 3	Week 4	Week 5	Week 6	Week 7
Session 1							
No. of sets	2	2	2	2	2	2	2
No. of sprints per set	10	10	10	10	10	10	10
Sprint duration (s)	6	6	6	6	6	6	6
Between sprint recovery (s)	30	27	24	21	18	15	12
Between set recovery (s)	300	300	300	300	300	300	300
Session 2							
No. of sets	2	2.5	3	3.5	4	4.5	5
No. of sprints per set	10	10	10	10	10	10	10
Sprint duration (s)	6	6	6	6	6	6	6
Between sprint recovery (s)	30	30	30	30	30	30	30
Between set recovery (s)	300	300	300	300	300	300	300
Session 3							
No. of sets	2	2	2	2	2	2	2
No. of sprints	10	10	10	10	10	10	10
Sprint duration (s)	6	8	10	12	14	16	18
Between sprint recovery (s)	30	30	30	30	30	30	30
Between set recovery (s)	300	300	300	300	300	300	300

integration (Chromjet, Thermo Separation Products). The coefficients of variation for plasma catecholamines were; NA 4.8% and AD 5.9% using HPLC and for plasma nephrines: NMET 8.1% and MET 8.6% using EIA.

Training

The sprint training programme is outlined in Table 1 and was based on several principles of training, i.e. overload, specificity (frequency, intensity, duration and mode) and reversibility (Bompa 1999). Participants completed 21 supervised sprint sessions performed on a cycle ergometer (Monark 824e) and over the course of the training period were asked to continue with normal diet, activity and training commitments. In session 2 participants' power was determined according to Lakomy (1986) and workload determined for the first ten sprints each week as an index of the changes in performance over the 7 weeks training. Participants completed each training session separated by at least 1 day with the first session 3 days after the pre-training experimental trial. Prior to participating in any sprint training session, all participants performed a warm-up consisting of leg stretching and 5 min sub-maximal intensity cycling ($\sim$ 60 W). All sprint sessions were performed on a cycle ergometer against a loading of 7.5% body mass, which was individually determined before each session. *Session 1* was designed to progressively reduce the inter-sprint recovery each week. Therefore, in week 1 participants performed twenty 6-s sprints with 30 s recovery between sprints with a reduction in recovery time of 3 s each week to a final inter-sprint recovery period of 12 s by week 7. *Session 2* was designed to increase the number of sprints each week from twenty 6-s sprints in week 1 to fifty such sprints in week 7, an increase of five sprints per week. Recovery period was constant at 30 s between each sprint in this session. *Session 3*: The weekly third session was designed to increase sprint duration from twenty 6-s sprints in week 1 to twenty 18-s sprints by week 7, an increase in sprint duration by 2 s per sprint each week. Recovery time between each sprint was kept constant at 30 s. In all sessions there was a 5-min recovery period between each set of ten sprints. All participants were verbally encouraged to maximally exert themselves in every session. On successful completion of the training programme participants performed the final experimental trial 1 week after completing the final supervised training session to allow sufficient recovery time from the programme but not enough time for deconditioning to occur.

Statistical analysis

Data were analysed using SPSS software (version 13; SPSS Inc., Chicago, IL, USA). Data were presented as the mean $\pm$ SD, with significance level set at $P < 0.05$. All data were assessed for normality (Shapiro–Wilk's test). Data were analysed using a repeated measures ANOVA

with a Tukey post hoc test where appropriate. Mauchly's test was consulted and Greenhouse–Geisser correction applied if the assumption of sphericity was violated.

Results

Performance parameters

The body mass of participants increased from a pre-training value of 80.1 ± 8.4 to 80.9 ± 9.1 kg following training. The maximal power output elicited in the incremental cycle test to exhaustion increased by 9.9 ± 0.8% following sprint training (pre-train 262.5 ± 17.7 vs. post-train 288.4 ± 19.4 W, $P < 0.05$). Interestingly, the absolute maximal rate of oxygen consumption increased by 2.9 ± 0.3% (pre-train 2.78 ± 0.44 vs. post-train 2.86 ± 0.41 l min^{-1}, $P < 0.05$) but not when scaled to the relative increase in body mass (pre-train 34.7 ± 10.5 vs. post-train 35.4 ± 11.25 ml kg^{-1} min^{-1}, *NS*). Peak heart rate increased from 198 ± 7 to 200 ± 9 beats per minute ($P < 0.05$). The performance responses to sprint training are shown in Fig. 1. The total work (kJ) over the first ten sprints in session 2 significantly increased from week 2 onwards with the greatest amount of work achieved over ten 6-s sprints in week 7 ($P < 0.05$).

Plasma normetanephrine and metanephrine

The plasma NMET and MET responses to exercise and training are outlined in Fig. 2a, b, respectively. Prior to exercise, resting plasma NMET concentrations of 0.49 ± 0.07 nmol l^{-1} increased following exercise to 0.97 ± 0.33 nmol l^{-1} ($P < 0.05$). After training, resting plasma

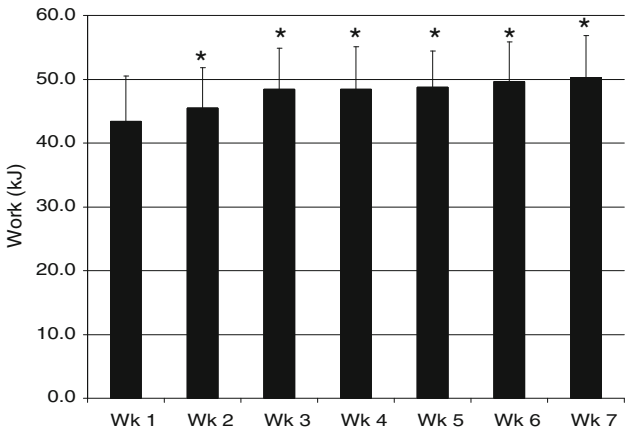

Fig. 1 Weekly amount of total work completed over ten 6-s cycle sprints (mean ± SD). The data was recorded from the first ten sprints of session 2 per week. *Asterisk* indicates a significant increase from week 1 values ($n = 10$, $P < 0.05$)

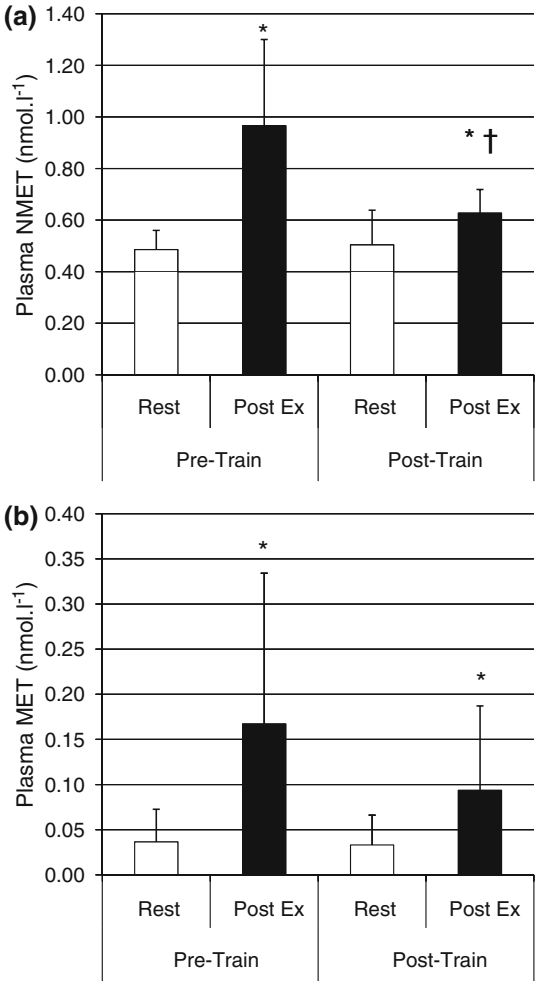

Fig. 2 Plasma **a** normetanephrine and **b** metanephrine concentrations of participants ($n = 10$) at rest and after exercise, before (pre-train) and after (post-train) 7 weeks of sprint training (mean ± SD). *Asterisk* indicates a significant difference from rest values ($P < 0.05$). *Dagger* indicates a significant reduction from pre-train values ($P < 0.05$)

NMET concentrations were unaltered but peak NMET concentrations were lower than the pre-training equivalent value (post-train 0.63 ± 0.09 nmol l^{-1}, $P < 0.05$), representing a 34% reduction in peak values. Plasma MET concentrations at rest were also unaltered with training and although not reaching significance there was a lower peak MET concentration following exercise in response to sprint training (pre-train 0.27 ± 0.17 vs. post-train 0.18 ± 0.09 nmol l^{-1}, $P = 0.07$).

Plasma catecholamines

The plasma catecholamine responses to high intensity exercise and sprint training are shown in Fig. 3a, b. Plasma NA increased from a resting value of 1.7 ± 0.5 to 18.4 ± 5.3 nmol l^{-1} following high intensity exercise before training ($P < 0.05$), representing a 10.8-fold

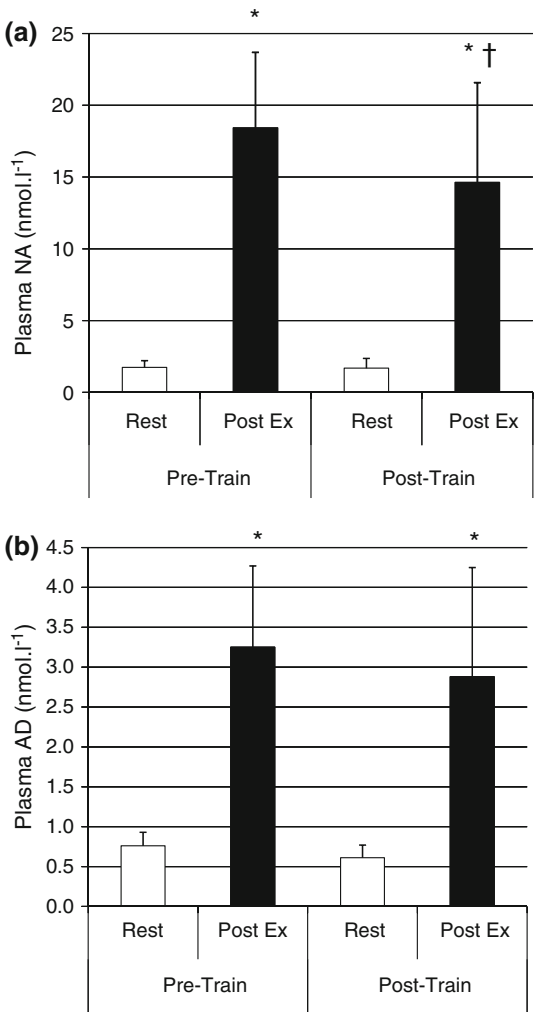

Fig. 3 Plasma **a** noradrenaline (NA) and **b** adrenaline (AD) concentrations of participants ($n = 10$) at rest and after exercise, before (pre-train) and after (post-train) 7 weeks of sprint training (mean ± SD). *Asterisk* indicates a significant difference from rest values ($P < 0.05$). *Dagger* indicates a significant reduction from pre-train values ($P < 0.05$)

increase. Sprint training did not alter resting values but peak values were lower compared to the equivalent pre-training value (post-train 14.6 ± 7.0 nmol l^{-1}, $P < 0.05$). Pre-training resting plasma AD concentrations of 0.8 ± 0.2 nmol l^{-1} increased 4.3-fold following exercise to 3.3 ± 1.0 nmol l^{-1} ($P < 0.05$) and similar to plasma MET there was a lower peak value after sprint training (post-train 2.9 ± 1.4 nmol l^{-1}, $P = 0.09$).

Blood acid-base status

The responses of blood acid-base variables are shown in Table 2. There was a significant decrease in resting blood pH after exercise before or after training ($P < 0.05$) but with less of a decline observed following post-training cycle exercise ($P < 0.05$). Resting blood lactate concentration was unaltered with training but the increase in post-exercise value was attenuated by 31 ± 8% following sprint training ($P < 0.05$).

Heart rate

Resting heart rate was unaltered with sprint training yet the post-training mean heart rate was lower than the pre-training value ($P < 0.05$, Table 2). The post-training exercise represented a lower percentage of pre-training peak heart rate (pre-train 86.9 ± 3.8 vs. post-train 82.8 ± 3.6%, $P < 0.05$).

Estimated changes in plasma volume

Changes in PV with exercise were similar following training (Table 2, *NS*).

Discussion

This study examined the plasma nephrine and catecholamine responses to a laboratory-based cycle test before and after a 7-week period of cycle sprint training. The results demonstrated a significant increase in plasma nephrines in response to a 2-min test at 110% VO_2max. Furthermore, plasma catecholamine and NMET concentrations were reduced in response to sprint cycle training.

The sprint cycle training programme consisted of 21 sessions that progressively increased sprint duration, number of sprints and reductions in inter-sprint recovery time. The typical power outputs elicited by participants were three to four times that in excess of power output at VO_2max. The total work completed by participants over ten 6-s sprints improved by ~ 12% as a result of the sprint training programme. Indeed, significant improvements in power output were noted after as little as three exercise sessions. In the early stages of sprint training there is an increase in nerve conduction velocity along the motor axon and an improved co-ordination pattern of muscles (Ross and Leveritt 2001) which may account for some of the early increases in power during the sprint training sessions. However, cellular adaptations in the form of increases in glycolytic enzyme activity (glycogen phosphorylase, phosphofructokinase and lactate dehydrogenase) have also been shown following similar sprint training programmes (Linossier et al. 1993). Greater amounts of muscle creatine phosphate and glycogen stores in response to sprint training can also increase total energy production from adenosine triphosphate (Barnett, et al. 2004). It has been shown that muscle buffering capacity is greater in previously untrained individuals in response to sprint training (Bell and Wenger 1988) and this increased intramuscular and/or extracellular

Table 2 Heart rate, blood lactate, blood pH, haemoglobin, haematocrit and estimated changes in plasma volume (PV) of participants at rest, following exercise, before (pre-train) and after (post-train) 7 weeks of sprint training ($n = 10$, mean ± SD)

	Pre-training		Post-training	
	Rest	Post-exercise	Rest	Post-exercise
HR (bpm)	70 ± 11	172 ± 8*	68 ± 11	164 ± 8*†
Lactate (mM)	0.3 ± 0.1	10.6 ± 2.0*	0.4 ± 0.2	7.3 ± 1.1*†
pH (U)	7.36 ± 0.03	7.09 ± 0.06*	7.37 ± 0.04	7.19 ± 0.09*†
Hb (g dl^{-1})	14.8 ± 0.6	16.2 ± 0.8*	14.9 ± 0.5	16.3 ± 0.6*
Hct (%)	43 ± 2	49 ± 2*	44 ± 1	50 ± 2*
Δ PV (%)		−18.1 ± 4.0		−18.4 ± 2.8

*Significant difference from rest values within each trial ($P < 0.05$)

† Significant difference from pre-train values ($P < 0.05$)

buffering capacity could attenuate the reduction in glycogenolytic rate found during performance of intermittent exercise (Gaitanos et al. 1993). The above-mentioned adaptations all help to explain the likely lesser metabolic perturbation observed in the post-training cycle test.

Plasma nephrines (i.e. NMET and MET) provide an additional marker of sympatho-adrenal activity as they serve as a good indication of extraneuronal catecholamine metabolism (Raber et al. 2000). Therefore, they offer further information on the sympatho-adrenal response to exercise by allowing examination of the interrelationships of several amines and related metabolites. High concentrations of COMT are found in non-neuronal cells such as adrenal medulla chromaffin cells, hepatocytes, and the kidneys but interestingly, not in the sympathetic nerves. The resting NMET concentrations measured in this study illustrate the very low concentrations measured in plasma and provide an indicator of extraneuronal NA metabolism. There was a 51% increase in plasma NMET concentrations in response high intensity exercise prior to beginning the sprint training programme suggesting evidence of elevated COMT activity in extraneuronal cells following exercise. Interestingly, there was a 34% reduction in post-exercise NMET concentrations following 7 weeks of sprint training. This suggests that the rate of COMT activity (and consequent NMET formation), similar to plasma catecholamine concentrations, may be dependent on the relative workload such that the exercise test that was performed following training was performed at a lower percentage of the participants' new maximal rate of oxygen consumption. What is also interesting is that although NMET appears as a sensitive marker of NA metabolism, it is not the primary product of NA metabolism as monoamine oxidase preferentially metabolises NA to dihydroxyphenylglycol within the sympathetic nerve terminal (Esler et al. 1990). Therefore, differences in NMET with sprint training may be due to altered rates of distribution to organs and sites containing COMT. On the other hand, MET is a primary route for AD metabolism, and chromaffin cells possess high quantities of COMT (Goldstein et al. 2003). Therefore, the adrenal medulla represents not only the primary site of AD secretion but also a major site for AD metabolism and as such changes in plasma MET concentrations represent alterations in the metabolic rate of AD (Goldstein et al. 2003). Prior to beginning the sprint training programme resting concentrations of plasma MET significantly increased by 200–300% following exercise demonstrative of an increased rate of AD metabolism. Following sprint training, peak plasma MET concentrations exhibited a trend for a lower circulating concentration ($P = 0.07$). This trend suggests evidence of a lower rate of COMT activity within adrenal medulla chromaffin cells following sprint training and might be indicative of COMT activity being coupled to the relative workload of high intensity cycle exercise.

From the results of the present study, the lower plasma NA response during the cycle exercise test was coupled to the relative intensity of the work. As such, the pre-training power output that equated to 110% of participants' maximal rate of oxygen consumption represented a lower percentage of the participants' maximum values attained following sprint training, as both maximum workload and rate of oxygen consumption values increased following sprint training. Plasma NA and AD responses to sub-maximal exercise are dependent on both exercise intensity and duration. Above a relative threshold intensity (~ 40–50% VO_2max), plasma concentrations increase exponentially with exercise intensity (Kjær et al. 1985) and at a constant sub-maximal exercise intensity increase linearly with exercise duration (Kjær 1998). Therefore, the relative stress of the exercise, in large part, determines the magnitude of the circulatory increase in catecholamines. The peak plasma NA concentration was 21% lower after training, results which mirror the findings of Harmer et al. (2000) that demonstrated lower plasma NA responses of

participants completing the same high intensity exercise test (130% VO_2max) before training following 7 weeks of sprint training. Plasma AD increased three- to fourfold in response to exercise and there was a trend for a smaller increase following exercise in post-train ($P = 0.09$). Harmer et al. (2000) found no differences in the plasma AD response to cycle exercise performed at the pre-training intensity (130% VO_2max) following 7 weeks of sprint training yet when the exercise was performed to exhaustion (new 130% VO_2max) on another occasion post-training plasma AD was significantly higher (Harmer et al. 2000). The mechanisms responsible for the reduction in catecholamines and nephrines with sprint training in our study are not clear but it is possible that high intensity training led to a greater disinhibition of motor neurons and lesser overall recruitment of the available muscle mass (Ross and Leveritt 2001). The lower plasma catecholamine concentrations following sprint training might also be due to lower sympathetic nerve activity caused by parallel reductions in the activation of motor and sympathetic nerve centres in the brainstem with exercise and/or via mechanoreceptor feedback from group III and IV afferent nerve fibres resulting in less post-ganglionic/adrenal medulla chromaffin cell catecholamine release (Vissing 2000).

In conclusion, this study examined the plasma nephrine and catecholamine responses to a laboratory-based cycle test before and after a 7-week period of cycle sprint training and found a significant increase in plasma nephrines in response to a 2-min test at 110% VO_2max. Furthermore, plasma NMET and NA concentrations were reduced in response to sprint cycle training, with a trend for a decrease in plasma MET and AD. The results suggest COMT activity may be coupled to the relative workload of high intensity cycle exercise.

References

Barnett C, Carey M, Prioetto J, Cerin E, Febbraio MA, Jenkins D (2004) Muscle metabolism during sprint exercise in man: influence of sprint training. J Sci Med Sport 7(3):314–322

Bell GJ, Wenger HA (1988) The effect of one-legged sprint training on intramuscular pH and non-bicarbonate buffering capacity. Eur J Appl Physiol Occup Physiol 58(1–2):158–164

Bompa TO (1999) Periodisation: theory and methodology of training, 4th edn. Human Kinetics, Champaign

Bracken RM, Linnane DM, Brooks S (2005) Alkalosis and the plasma catecholamine response to high intensity exercise in man. Med Sci Sports Exerc 37(2):227–233

Bracken RM, Linnane DM, Brooks S (2009) Plasma catecholamine and nephrine responses to brief intermittent maximum exercise. Amino Acids 36(2):209–217

Brooks S, Cheetham M, Williams C (1984) Endurance training and the catecholamine response to brief maximum exercise in man. J Physiol 361:81

Dill DB, Costill DL (1974) Calculation of percentage changes in volumes of blood, plasma, and red cells in dehydration. J App Physiol 37(2):247–248

Durnin JVGA, Womersley J (1974) Body fat assessed from total body density and its estimation from skinfold thickness: measurements on 481 men and women aged 16 to 72 years. Br J Nutr 32:77–97

Esler M, Jennings G, Lambert G, Meredith I, Horne M, Eisenhofer G (1990) Overflow of catecholamine neurotransmitters to the circulation: source, fate, and functions. Physiol Rev 70(4): 963–985

Filaire E, Legrand B, Bret K, Sagnol M, Cottet-Emard JM, Pequignot JM (2002) Psychobiologic responses to 4 days of increased training and recovery in cyclists. Int J Sports Med 23:588–594

Filaire E, Legrand B, Lac G, Pequignot JM (2004) Training of elite cyclists: effects on mood state and selected hormonal responses. J Sports Sci 22:1025–1033

Gaitanos GC, Williams C, Boobis LH, Brooks S (1993) Human muscle metabolism during intermittent maximal exercise. J App Physiol 75(2):712–719

Goldstein DS, Eisenhofer G, Kopin I (2003) Sources and significance of plasma levels of catechols and their metabolites in humans. J Pharmacol Exp Ther 305(3):800–811

Greiwe JS, Hickner RC, Shah SD, Cryer PE, Holloszy JO (1999) Norepinephrine response to exercise at the same relative intensity before and after endurance exercise training. J App Physiol 86(2):531–535

Hagberg JM, Goldring D, Heath GW, Ehsani AA, Hernandez A, Holloszy JO (1984) Effect of exercise training on plasma catecholamines and haemodynamics of adolescent hypertensives during rest, sub-maximal exercise and orthostatic stress. Clin Physiol 4(2):117–124

Harmer AR, McKenna MJ, Sutton JR, Snow RJ, Ruell PA, Booth J, Thompson MW, Mackay NA, Stathis CG, Crameri RM, Carey MF, Eager DM (2000) Skeletal muscle metabolic and ionic adaptations during intense exercise following sprint training in humans. J App Physiol 89(5):1793–1803

Hartley LH, Mason JW, Hogan RP, Jones LG, Kotchen TA, Mougey EH, Wherry FE, Pennington LL, Ricketts PT (1972) Multiple hormonal responses to prolonged exercise in relation to physical training. J App Physiol 33(5):607–610

Kaiser P, Tesch PA, Frisk-Holmberg M, Juhlin-Dannfelt A, Kaijser L (1986) Effect of beta 1-selective and non-selective beta-blockade on work capacity and muscle metabolism. Clin Physiol 6(2):197–207

Kjær M (1998) Adrenal medulla and exercise training. Eur J Appl Physiol 77(3):195–199

Kjær M (1999) Neuroendocrine regulation during exercise. In: Hargreaves M, Thompson M (eds) Biochemistry of exercise X. Human Kinetics, Champaign, pp 47–55

Kjær M, Christensen NJ, Sonne B, Richter EA, Galbo H (1985) Effect of exercise on epinephrine turnover in trained and untrained male subjects. J App Physiol 59(4):1061–1067

Lakomy HKA (1986) Measurement of work and power using friction loaded cycle ergometers. Ergonomics 29(4):509–517

Linossier MT, Denis C, Dormois D, Geyssant A, Lacour JR (1993) Ergometric and metabolic adaptation to a 5-s sprint training programme. Eur J Appl Physiol Occup Physiol 67(5):408–414

Maughan RJ (1982) A simple, rapid method for the determination of glucose, lactate, pyruvate, alanine, 3-hydroxybutyrate and acetoacetate on a single 20-ul blood sample. Clin Chim Acta 122(2):231–240

Merke DP, Chrousos GP, Eisenhofer G, Weise M, Keil MF, Rogol AD, Van Wyk JJ, Bornstein SR (2000) Adrenomedullary dysplasia and hypofunction in patients with classic 21-hydroxylase deficiency. N Engl J Med 343(19):1362–1368

Pequignot JM, Peyrin L, Mayet MH, Flandrois R (1978) Metabolic adrenergic changes during submaximal exercise and in the recovery period in man. J App Physiol 47:701–705

Peronnet F, Cleroux J, Perrault H, Cousineau D, de Champlain J, Nadeau R (1981) Plasma norepinephrine response to exercise before and after training in humans. J Appl Physiol 51(4): 812–815

Raber W, Raffesberg W, Bischof M, Scheuba C, Niederle B, Gasic S, Waldhäusl W, Roden M (2000) Diagnostic efficacy of unconjugated plasma metanephrines for the detection of pheochromocytoma. Arch Int Med 160:2957–2963

Raber W, Raffesberg W, Waldhausl W, Gasic S, Roden M (2003) Exercise induces excessive normetanephrine responses in hypertensive diabetic patients. Eur J Clin Invest 33(6):480–487

Ross A, Leveritt M (2001) Long-term metabolic and skeletal muscle adaptations to short-sprint training: implications for sprint training and tapering. Sports Med 31(15):1063–1082

Vissing J (2000) Muscle reflex and central motor control of neuroendocrine activity, glucose homeostasis and circulation during exercise. Acta Physiol Scand 170 Suppl 647:5–26

Winder WW, Hagberg JM, Hickson RC, Ehsani AA, McLane JA (1978) Time course of sympatho-adrenal adaptation to endurance exercise training in man. J App Physiol 45(3):370–374

Winder WW, Hickson RC, Hagberg JM, Ehsani AA, McLane JA (1979) Training-induced changes in hormonal and metabolic responses to sub-maximal exercise. J App Physiol 46(4): 766–771

Winder WW, Yang HT, Jaussi AW, Hopkins CR (1987) Epinephrine, glucose, and lactate infusion in exercising adrenodemedullated rats. J App Physiol 62(4):1442–1447

Zamecnik J (1997) Quantification of epinephrine, norepinephrine, dopamine, metanephrine and normetanephrine in human plasma using negative ion chemical ionization GC-MS. Can J Anal Sci Spectrosc 42(4):106–112