

ORIGINAL ARTICLE

Neuromuscular, physiological and endocrine responses to a maximal speed training session in elite games players

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Abstract

The aim of this study was to determine the acute neuromuscular, biochemical and endocrine responses to a maximal speed training (MST) session. Eighteen male rugby players completed the protocol, which involved performing six maximal effort repetitions of 50 m running sprints with 5 minutes recovery between each sprint. Testosterone (T), cortisol (C), creatine kinase (CK), lactate (La), perceived muscle soreness (MS) and counter movement jump were collected immediately pre (PRE), immediately post (IP), 2 hours post (2P) and 24 hours post (24P) the sprint session. A bimodal recovery pattern was observed from the jump parameters with several declining significantly ($p \leq 0.05$) IP, recovering 2P and suffering a secondary decline 24P. CK and perceived MS were elevated IP and continued to rise throughout the protocol, while La was only elevated IP. T and C were unaffected IP but showed significant declines 2P. These data indicate that MST results in a bimodal recovery pattern of neuromuscular function with changes most likely being related to metabolic and biochemical responses.

Keywords: *Testosterone, cortisol, creatine kinase, neuromuscular fatigue*

Introduction

Speed is an essential component of physical fitness, which has been linked to successful performance in elite games players (Baker & Newton, 2008), with studies demonstrating that it can be developed using maximal speed training (MST; Lockie, Murphy, Schultz, Knight, & Janse de Jonge, 2012; Tønnessen, Shalfawi, Haugen, & Enoksen, 2011). For example, Tønnessen et al. (2011) reported a 0.06 second improvement in 40-m time following 10 weeks of sprint training sessions consisting of distances of up to 40 m per repetition, total volumes of up to 480 m and recoveries of between 2 and 10 minutes per repetition. There is a growing need for elite clubs to undertake specialised speed training sessions in isolation of technical, repeated sprint ability or strength training in order to ensure the specificity of the training session is maximised.

Acute post exercise responses in the neuromuscular and endocrine systems have been suggested to play an important role in the athlete's adaptation to and recovery from training (Bosco, Colli, Bonomi, von Duvillard, & Viru, 2000; Ross, Leveritt, & Riek, 2001). However, while considerable research has examined the responses induced by strength (Bosco et al., 2000; Hakkinen, 1992) and endurance (Daly, Seegers, Rubin, Dobridge, & Hackney, 2005) training sessions, little is known about the responses to an MST with only one study having profiled the acute neuromuscular and endocrine response (Pullinen, MacDonald, Pakarinen, Komi, & Mero, 2005). While this study observed declines in isometric maximal voluntary contraction alongside elevations in testosterone (T) and cortisol (C) immediately post (IP) training, it does not provide information on recovery in the hours and days that followed the

session, which is vital information for the sports scientist in order to effectively plan the training week.

The neuromuscular responses to stretch shortening cycle (SSC) exercise (Nicol, Avela, & Komi, 2006) highlight the importance of observing neuromuscular performance in the hours post training. Several studies, while reporting an initial decline immediately after the stimulus, observed a short-term recovery in neuromuscular performance followed by a second decrease in performance between 24 and 48 hours post, with the mechanisms behind this pattern potentially linked to the time frames associated with the onset of, and recovery from, metabolic disturbance and muscle damage, respectively (Kuitunen, Avela, Kyrolainen, & Komi, 2004). While previous studies into explosive SSC activities have reported a secondary declines in performance 24 hours post (24P) exercise (Dousset et al., 2007), it is unclear if a similar pattern occurs post MST, further highlighting the need for research into this area.

Therefore, the aim of this study was to profile the neuromuscular and endocrine response to an MST session over 24 hours in order to provide information to impact on effective programming.

Methods

Subjects

Eighteen academy players, from a professional rugby club, were recruited for this study (mean $\pm$ standard deviation: age, 20.5 ± 1.2 ; mass, 99.4 ± 8.6 kg; height, 186.4 ± 7.5 cm). Statistical power was calculated using previously published methods (Young, 1995), and a sample size of equal to or greater than 16 was deemed sufficient. Each player had been involved in a professional academy system for a minimum of 2 years, during which time they were exposed to regular strength, power and speed training and testing. This study was undertaken during the regular playing season, and subjects were performing 1–2 sessions per day 4–5 days a week and playing in one weekly competitive game. Subjects provided written informed consent and a university research ethics committee provided ethical approval.

Experimental design

The same experimental group completed the protocol over 2 days. Prior to arriving on day 1, subjects were given 2 days of training. Upon arrival [immediately pre (PRE)], subjects filled out a perceived muscle soreness (MS) questionnaire, and a blood sample was collected for subsequent analysis. Subjects then performed a 10-minute

standardised warm-up before reporting to the testing area where three counter movement jumps (CMJs) were performed with hands on hips. Subjects were provided with 30 seconds recovery between jumps.

Immediately after this initial testing, subjects reported to the indoor track to perform an MST session. Subjects performed a running-specific warm-up which concluded with 4×50 m sub-maximal sprints with 2 minutes recovery. At this point, 6×50 m maximal sprints were completed with 5 minutes recovery between each trial. These training parameters reflected their normal training sessions and are in line with the volume of maximal speed running per session suggested by elite coaches (Francis, 2008). Each sprint started 30 cm behind the start line and was timed using light gates (Brower timing system, Salt Lake City, UT, USA). Upon completing the final sprint (IP) and 2 hours post (2P), MS, neuromuscular function and blood collection were collected. Finally, on day 2 (24P), the subjects returned to complete the testing protocols. The arrival times and order on days 1 and 2 were standardised for each participant in order to minimise any potential circadian influence. Subjects also underwent a 12-hour fast prior to arriving on both days in order to control for inter- and intra-subject variations in nutritional intake, which may have, in turn, affected endocrine response (Hackney & Viru, 2008). In between IP and 2P consumed a standardised recovery drink, consisting of 70 grams of carbohydrate and 35 grams of protein, to minimise the effect of nutrition on endocrine response (Hackney & Viru, 2008). Consumption of water was allowed throughout the study.

The temperature in the testing area was maintained between 20° and 24° .

Biochemical testing

Blood samples were collected from the antecubital vein after 10 minutes of lying supine to determine the acute responses of T, C and creatine kinase (CK). Each sample was taken by trained practitioners via venipuncture. After collection, the samples were centrifuged at 3000 rpm for 10 minutes at room temperature. Plasma was analysed for T, C and CK activity using a commercially available kits (Roche Diagnostic Limited, Charles Avenue, Burgess hill) on a Cobas C8000 analyser (Roche Diagnostics, Switzerland). The inter-assay coefficients of variation (CV) for T, C and CK were 5.3%, 3.7% and 1.4%, respectively. Lactate (La) was analysed using a lactate analyser (Lactate pro, Arkray).

Neuromuscular performance

The CMJ tests were performed on a force platform (type 9287CA, Kistler Instruments Ltd., Farnborough, UK). A sample rate of 1000 Hz and a vertical force range of 20 kN were used for all trials, in accordance with previous research (Owen, Watkins, Kilduff, Bevan, & Bennett, 2013). After collection, the vertical component of the ground reaction force (GRF)-time history was exported to a custom-built excel sheet for analysis. Body mass, jump-start time and take-off were calculated using methods previously described (Street, McMillan, Board, Rasmussen, & Heneghan, 2001), and velocity and displacement were calculated using the impulse-momentum method.

In order to calculate jump height (JH), maximal vertical displacement was first calculated using the impulse-momentum method. JH (m) was then defined as the difference between vertical displacement at take-off and maximal vertical displacement. The test-retest reliability of JH using this method was found to be high (CV 3.46%). To calculate peak power (PP), instantaneous power was first calculated by multiplying vertical GRF by the vertical velocity of the centre of gravity. Relative PP (w.kg) was calculated by dividing the PP by the body weight (BW), in kg. PP and w.kg were found to have test-retest CV's of 2.49% and 2.43%, respectively.

CMJ average rate of force development (aRFD) was calculated using a published method (Thorlund, Michalsik, Madsen, & Aagaard, 2008). aRFD was defined as the change of force during the eccentric deceleration phase divided by the time of the eccentric deceleration phase. The eccentric deceleration phase was defined as the point at which the force passed through BW during the eccentric phase, through the point when displacement became positive. The test-retest CV for this method was 8.84%.

Perceived MS

MS was recorded at each data collection point, using a 7-point Likert scale designed to measure soreness in the lower body. The scale ranged from very, very good (1) to very, very sore (7). The use of this Likert scale is supported by previous research in the area (Andersson et al., 2008).

Statistical analysis

Data are expressed as the mean $\pm$ SD. After tests for normal distribution and prior to any further statistical analysis, CK recorded values were log transformed due to large inter-participant variability. Statistical analysis was carried out using a repeated measures one-way analysis of variance on the various measures. Where significant effect was observed, paired comparisons were used with a Bonferroni correction control of type I error. Effect size (ES) was determined using η^2 , with an ES of approximately 0.2 considered small, approximately 0.5 considered medium and approximately 0.8 considered large (Cohen, 1988). The level of significance was set at $p \leq 0.05$ for the present study, and all statistics were performed using SPSS 13.1 (SPSS Inc., Chicago, IL).

Results

Endocrine responses

As seen in Table I, the MST session had a significant time effect on total T (η^2 : 0.712; $F = 41.991$, $p < 0.05$) and C (η^2 : 0.778; $F = 59.555$, $p < 0.05$). At 2P, significant decreases in T and C levels were found with values returning to baseline at the 24P time point (Table I).

MS, La and a marker of muscle damage (CK)

As seen in Table II, MST had a significant time effect on CK (η^2 : 0.888; $F = 39.465$, $p < 0.05$), La (η^2 : 0.945; $F = 289.422$, $p < 0.05$) and MS (η^2 : 0.305; $F = 7.472$, $p < 0.05$). At IP, La, perceived MS and CK increased significantly (Table II) compared to baseline with values for MS and CK remaining significantly elevated at the 2P. At 24P, there was a significant increase in both CK and MS when compared to pre-training levels (Table II).

Neuromuscular response

Table III shows the time effect the MST had on neuromuscular performance. JH (η^2 : 0.557; $F = 21.376$, $p < 0.05$), PP (η^2 : 0.481; $F = 15.767$, $p < 0.05$), w.kg (η^2 : 0.463; $F = 14.678$, $p < 0.05$) and aRFD (η^2 : 0.259; $F = 5.941$, $p < 0.05$) were all

Table I. Total T and C at four different time points in team sport athletes

	PRE	IP	2P	24P
T (nmol/l)	20.1 $\pm$ 6.12	20.7 $\pm$ 6.36	15.5 $\pm$ 6.08*	20.8 $\pm$ 6.53
C (nmol/l)	543 $\pm$ 119	500 $\pm$ 104	267 $\pm$ 61.2*	531 $\pm$ 96.1

Data presented as mean $\pm$ SD.

*Significant to 0.05 vs. pre-training values.

Table II. MS, CK and La at four different time points in team sport athletes

	PRE	IP	2P	24P
MS (Likert scale)	1.00 ± 1.19	2.39 ± 1.14*	2.11 ± 1.41*	2.56 ± 1.62*
CK (u l)	420 ± 360	514 ± 406*	615 ± 437*	990 ± 703*
La (mmol/l)	1.58 ± 1.06	10.6 ± 1.58*	2.06 ± 1.07	1.16 ± 0.35

Data presented as mean ± SD.

*Significant to 0.05 vs. pre-training values.

significantly affected. JH, PP and w.kg were significantly decreased IP (Table III). JH and aRFD were significant decreased 24P, while PP had a very strong tendency towards depression at 24P ($p = 0.051$; Table III). None of the parameters were significantly different to PRE at 2P.

Discussion

The current study is the first to document the neuromuscular, endocrine and biochemical responses over a period of 24 hours to an MST session. The main finding of this study was that several neuromuscular markers followed complex recovery patterns post training and were still declined 24P.

Neuromuscular response to MST

Initially, it was found that the majority of variables declined IP. While the lack of consistency in terms of variables used to assess changes in neuromuscular performance in different studies makes it difficult to draw definitive conclusions, the initial decrements in w.kg observed were lower than those reported after a marathon (Armstrong, 1990; 4.4% vs. 11%). However, the 10% decline in JH found IP was similar to the 8% decrease in JH reported after a strength training session (Walker, Ahtiainen, & Hakkinen, 2010) and the 8% decrease found after a drop jump session (Skurvydas, Dudoniene, Kalvenas, & Zuoza, 2002). While more research is required, it seems possible that the greater immediate post exercise losses in performance experienced by the marathon runners was related to the high volume of running,

and it is possible that initial depressions in performance are more sensitive to volume than intensity.

At 2P, none of the jump parameters differed from PRE levels, suggesting that the neuromuscular performance had recovered. However, when observed 24P, the majority of markers of neuromuscular performance were again significantly depressed compared to PRE levels. This would suggest that MST effects neuromuscular performance in a bimodal pattern similar to that described by Nicol et al. (2006).

In previous studies, both decreased levels of T (Bosco et al., 2000) and increased afferent feedback in response to muscle damage (Nicol et al., 2006) have been suggested to be involved in the immediate post exercise loss of neuromuscular performance. However, in the current study, no depression in T was found IP exercise, while both CK and MS were significantly elevated; both were also found to be still rising 2P. It seems unlikely that performance would have improved in the face of further increases in markers of muscle damage if these had been a major contributor to the initial declines in performance observed.

A more likely explanation for the initial decreases observed might be a decreased functioning of the contractile mechanisms of the muscle fibre (Skurvydas et al., 2007) in the presence of the metabolites produced during exercise (H^+ , ADP, Pi). Blood La was significantly elevated upon completion of the sprints and, while not a direct marker of metabolic fatigue; significant increases in La may be viewed as an indicator of metabolite accumulation (Skurvydas et al., 2006). Indeed, our results suggest that an MST session consisting of 6×50 m with 5 minutes recoveries between repetitions results in significant

Table III. CMJ parameters at four different time points in team sport athletes

	PRE	IP	2P	24P
CMJ JH (m)	0.40 ± 0.05	0.36 ± 0.06*	0.39 ± 0.05	0.38 ± 0.06*
CMJ PP (w)	5193 ± 461	4963 ± 562*	5154 ± 503	5106 ± 508
CMJ Rel. Power (w/kg)	54.3 ± 6.37	51.8 ± 6.34*	53.9 ± 6.7	53.4 ± 6.41
CMJ aRFD (n/s)	4557 ± 1014	4333 ± 1282	4579 ± 1077	3891 ± 936*

Data presented as mean ± SD.

*Significant to 0.05 vs. pre-training values.

metabolic stress (Table II) and produces La levels higher than would be expected from strength training protocols using similar recoveries (Skurvydas et al., 2002). Therefore, the metabolic stress associated with MST should be considered when planning volumes and recoveries.

On the surface, it seems curious that neuromuscular performance had recovered by 2P while CK and MS continued to rise. Given the intensive nature of the MST, it is likely that the training resulted in a significant increase in free radical production (Vollaard, Shearman, & Cooper, 2005), which in turn may have effected membrane permeability and contributed to the elevated CK levels observed. However, it is also likely that it is the inflammatory response to muscle damage as opposed to the muscle damage itself that ultimately affects neuromuscular performance (Dousset et al., 2007). This inflammatory response in turn is not initiated until between 2 and 6 hours after the exercise (Armstrong, 1990). Taking this time frame into account, we hypothesised that the recovery in neuromuscular performance observed at 2P occurred prior to the initiation of the inflammatory response and was most likely be due to the removal of the metabolic by-products that had initially built up IP exercise (Dousset et al., 2007). This is supported by the fact that La had returned to baseline by 2P. It is further suggested that the secondary decline in muscle performance, observed 24P, was primarily related to the inflammatory response, which would be well underway at this time point.

The declines in neuromuscular performance at 24P supported previous research, where 10 m sprint performance was found to be depressed 24P intensity training (Highton, Twist, & Eston, 2009). In contrast, however, 2000 m rowing performance was shown not to decline 24 hours after a heavy strength training session, even in the presence of decreased CMJ performance and elevated levels of MS (Gee et al., 2011). When our findings are viewed in conjunction with these studies, it would seem that it is better to place explosive/maximal effort training relatively close together, and coaches may consider sequencing their training in a manner that takes advantage of this. In addition, a strategy of alternating high intensity explosive training days containing multiple sessions with days emphasising sub-maximal activities may take advantage of the observed pattern of neuromuscular recovery.

Endocrine response to MST

In the current study, MST did not cause any acute elevations in T and/or C at the IP time point. While both T and C were found to be significantly lower

than PRE values when measured at 2P, these depressions are in line with the normal circadian variations previously reported in the literature (Kraemer et al., 2001). Therefore, it seems unlikely that these depressions were a direct response to the training stimulus. However, the lack of non-exercise control data in the current study means that this cannot be confirmed.

This lack of exercise response is in contrast to previous work on MST where trained sprinted undertook 10 × 50 m sprints with 5 minutes between efforts (Pullinen et al., 2005). The authors of this study reported a 19% increase in T and a 31% increase in C IP. There appear to be several mechanisms linked to post exercise elevations in T and C. Previous research has reported a link between the secretion of both T (Walker et al., 2010) and C (Spiering et al., 2008) and metabolic accumulation. However, given that Pullinen et al. (2005) and ourselves reported similar elevations in La post exercise, this does not explain the differences between our findings. Instead, the lower sprint volume performed in the current study (6 × 50 m vs. 10 × 50 m) may have played a role. In resistance training, the T response has been linked to volume load (McCaulley et al., 2009), so it is possible that the different hormonal responses are a response to the different volume of MST performed. Indeed, studies into endurance running suggest high volumes of SSC activities can cause fluctuations in endocrine levels in the days following the training stimulus (Daly et al., 2005).

The participant population must also be considered, with the participants in the prior study being experienced sprinters, whereas academy rugby players were tested herein. Research suggests both training background (Ahtiainen, Pakarinen, Kraemer, & Hakkinen, 2004) and training experience (Kraemer et al., 1992) can influence the T response to a workout, which again could also extend to MST.

Conclusions

From our study, it appears that neuromuscular performance undergoes a bimodal recovery pattern in response to MST, with an initial loss in performance being found to occur after training. Performance was then found to have recovered by 2P, before undergoing a second decrease 24P. From these results, it appears that periods longer than 24 hours are required to allow full neuromuscular recovery from MST. However, the endocrine system was unaffected by MST, and it appears a 24 hour is sufficient for it to recover from this type of training.

The fact that several of the neuromuscular and physiological parameters had not fully recovered by

24P represents a limitation in the current study. However, it is also the reality of elite sport that a further training stimulus would be undertaken prior to full recovery. Given this, coaches should consider the timing and type of sessions undertaken after an MST session. It appears that the 2P time point would seem to represent a superior training window for explosive type activities than the 24P time point, while training undertaken at 24P should be more sub-maximal in nature.

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