



Original research

Profiling the time-course changes in neuromuscular function and muscle damage over two consecutive tournament stages in elite rugby sevens players



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ARTICLE INFO

Article history:

Received 16 July 2013

Received in revised form 3 October 2013

Accepted 11 November 2013

Available online 23 November 2013

Keywords:

Performance

Creatine kinase

Recovery

ABSTRACT

Objectives: Many International Rugby Board (IRB) sevens competitions require that two tournament stages are played over consecutive weekends, but the impact this has on player physical performance and recovery is lacking. We examined the influence of two consecutive tournaments on neuromuscular function (NMF) and muscle damage in rugby sevens players.

Design: Ten elite international rugby sevens players completed this observational study over 2 tournaments, separated by 5 days, during the IRB sevens series.

Methods: On the morning of day 1 and 2, of both tournament 1 (T1) and 2 (T2), players performed countermovement jumps (CMJ; jump height [JH]) and capillary blood samples (creatinase kinase [CK]) were collected. After the last match of each day, further capillary samples were collected. Additional, CMJ were performed 12 and 60 h post-T1.

Results: Player JH decreased from day 1 to day 2 during T1 (mean \pm SD; $-6.0 \pm 5.4\%$; $P = 0.016$), was reduced at 12 ($-26.1 \pm 5.0\%$; $P < 0.001$) and 60 h post-T1 ($-7.1 \pm 4.8\%$; $P = 0.003$) and remained lower, at am day 1 of T2 ($-8.0 \pm 6.0\%$; $P = 0.007$), when compared with day 1 of T1. Player JH was lower on day 1 and 2 of T2, compared with T1 ($P < 0.05$). CK concentrations were greater than baseline at all time points during each tournament ($P < 0.001$); no between tournament differences in CK responses existed ($P = 0.302$).

Conclusions: A single sevens tournament reduces NMF such that players are not fully recovered by the start of the second competition stage, however CK returns to baseline in-between and shows the same pattern across two consecutive tournaments.

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1. Introduction

Rugby union sevens is increasing in popularity, and in 2009 it was added to the 2016 Rio de Janeiro Olympic games. Rugby union sevens competitions differ dramatically from other team sports, including the traditional 15 a-side game, in that they are usually played over a 2 day tournament weekend, with matches consisting of two 7 min halves, with a 2 min half-time interval,¹ on a full dimension rugby union field. A tournament usually consists of 3 group stage matches on the first day, each separated by ~3 h, and depending on results, up to three on the second day of the

tournament. In addition, the international rugby union sevens series (the world professional competition) also requires two stages (i.e. 2 tournaments) to be played on consecutive weekends.

During a sevens match, players can spend ~75% of the game at heart rates above 80% of maximum² while also covering a total of ~1581 m, with ~9% of this distance covered through maximal sprinting, with average sprint distances of ~18 m²; while in rugby union 15-a-side games this intensity represents ~2% of the overall game demands.³ These great physiological demands, which are heavily reliant on large contributions from high-intensity, stretch-shortening cycle based movements, combined with physical collisions, may also result in an increased appearance of intramuscular protein/enzymes in the blood,⁴ which are indicative of skeletal muscle damage.⁵ For example, Takashi and colleagues⁴ reported an ~18% increase in serum creatine kinase (CK) after a

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single rugby union sevens match, and a further increase (~42% above baseline) after the second match of the day.

The induction of skeletal muscle damage is also likely to impair neuromuscular function (NMF).⁶ For example, Twist et al.,⁶ reported reductions in NMF (countermovement jump performance) and concomitant increases in CK and muscle soreness 24 and 48 h post-match in professional rugby league players. Similarly, West et al.,⁷ demonstrated that NMF may be reduced for up to 60 h post-match in professional rugby union players. Profiling the recovery time-course after intense contacts sports/training types, e.g.^{7–9}, is important for coaches, as these data can help inform post-match recovery strategies and training programme design.

Given the intense demands of rugby union sevens competition, and evidence of increases in markers of skeletal muscle damage after just 1 day of competition,⁴ sevens players may be susceptible to reduced NMF over the course of a tournament weekend. Furthermore, as there are occasions where the players may need to compete again 5 days later, there is potential that players may enter a subsequent tournament having not fully recovered from the preceding stage. However, currently there is no information available to confirm or refute these hypotheses. Therefore, the aim of this study was to examine the influence of two consecutive tournament weekends on NMF and muscle damage in elite international rugby union sevens players.

2. Methods

With approval from the Swansea University Research Ethics Committee, 10 elite International Rugby Union Sevens players (mean ± SD, age: 26 ± 5 years; height: 1.83 ± 0.08 m; body mass: 86.1 ± 10.0 kg) participated in the study. All players had at least 3 years of (monitored/recorded) training history. All were informed of the potential risks associated with the study prior to giving their informed consent. This observational study was conducted during the last two stages of the eight stage, 6 month competition period of the International Rugby Board (IRB) world seven series. The last two stages were played within the United Kingdom (London and Edinburgh) and were separated by 5 days.

The observational window and measurement protocol is presented in Table 1; this was a typical training week for this team between tournaments. A standardised warm-up was performed prior to every game, which involved jogging, running, sprinting, contact, dynamic stretching and skill based drills. This warm-up was replicated before every match, during both tournaments, allowing for the players to be in the tunnel 6 min prior to the start of each match. Capillary blood samples for the subsequent measurement of plasma creatine kinase (CK) were collected from the players upon arrival at the competition venues (~10 am) and immediately after the last match of each day's play (Table 1). Countermovement jumps (CMJ) were performed on a portable force platform (processed for peak power output [PPO] and jump height [JH]) 10 min before the start of the first match on days 1 and 2, of both tournaments, and at the time points of 12 and 60 h after

the last match at tournament 1 (Table 1). In that five day period tactical, technical and weight training sessions were carried out, of which only two sessions were optional. The only recovery modality used was a 20 min swimming pool recovery session, which involved swimming and stretching underwater on the day after T1. While in camp, all players were in a controlled setting, with prescribed meals and supplementation; players were also requested to replicate sleeping patterns.

For the measurement of CMJ PPO and JH, testing was completed on a portable force platform (Type 92866AA, Kistler, Germany). To isolate the lower limbs, participants stood with arms akimbo.¹⁰ After an initial stationary phase of at least 2 s in the upright position, for the determination of body mass, participants performed a CMJ, dipping to a self-selected depth and then exploding upwards in an attempt to achieve maximum height. Participants landed back on the force platform and kept their arms akimbo throughout the movement. Players were required to complete 3 maximum jumps with 1.5 min rest between efforts. PPO and jump height were calculated as previously described.¹⁰ The vertical component of the ground reaction force (GRF) during the CMJ was used in conjunction with the participants' body mass to determine instantaneous velocity and displacement of his centre of gravity.¹⁰ Instantaneous power was determined using the following standard relationship: Power (W) = vertical GRF (N) × Vertical velocity of centre of gravity (m.s⁻¹)

Whole blood was collected via fingertip puncture using a spring-loaded disposable lancet (Safe-T-Pro Plus, Accu-Chek, Roche Diagnostics GmbH, Germany). A 120-µL sample was collected in a capillary tube and immediately centrifuged (Labofuge 400R, Kendro Laboratories, Germany) at 3000 rpm for 10 min for the extraction of plasma, which was subsequently stored at -20 °C. The plasma samples were left to thaw before 6 µL was used in the analysis of CK (CK using a semi-automated analyzer (COBAS MIRA; ABX Diagnostics, UK). Sample testing was carried out in duplicate and the mean coefficient variation (CV) for CK assays was 1.6%.

Statistical analysis was performed using SPSS software (version 16; SPSS Inc., Chicago, IL), with significance set at $P \leq 0.05$. Time-course changes from tournament 1 to tournament 2 were assessed using one-way repeated measures ANOVA, with bonferroni adjusted pairwise comparisons. Within and between tournament responses were examined using repeated measures ANOVA on two levels (time × tournament), with bonferroni adjusted paired-samples *t*-test used to examine between tournament differences. Where significant differences have been identified, 95% confidence intervals are presented for an estimation of the population mean difference. Data are presented as mean ± SD.

3. Results

The impact of tournament 1 on the NMF recovery time course leading in to tournament 2 is presented in Fig. 1. There was a significant time effect ($P < 0.001$; Partial-eta² = 0.400) in the players CMJ PPO from AM day 1 of tournament

Table 1
Sampling time-course throughout the study period.

Sample point	Tournament 1				Recovery					Tournament 2			
	Day 1		Day 2		Day 1	Day 2	Day 3	Day 4	Day 5	Day 1		Day 2	
	AM	PM	AM	PM	Post 12	-	Post 60	-	-	AM	PM	AM	PM
Creatine kinase	10:00	20:00	10:00	16:00	-	-	-	-	-	10:00	19:00	10:00	16:00
CMJ	10:00	-	10:00	-	08:00	-	08:00	-	-	10:00	-	10:00	-
Activity AM	Match Play (x3)		Match Play (x2)		PR	-	-	WT	O-WT	Match Play (x3)		Match Play (x2)	
Activity PM					Trav	O-WT	-	FS-T	FS-T				

PR: pool recovery (20 min); Trav: travel to tournament 2, 4.5 h train journey; O-WT: optional weight training session; WT: compulsory weight training session; FS-T: field based, tactical-technical rugby session.

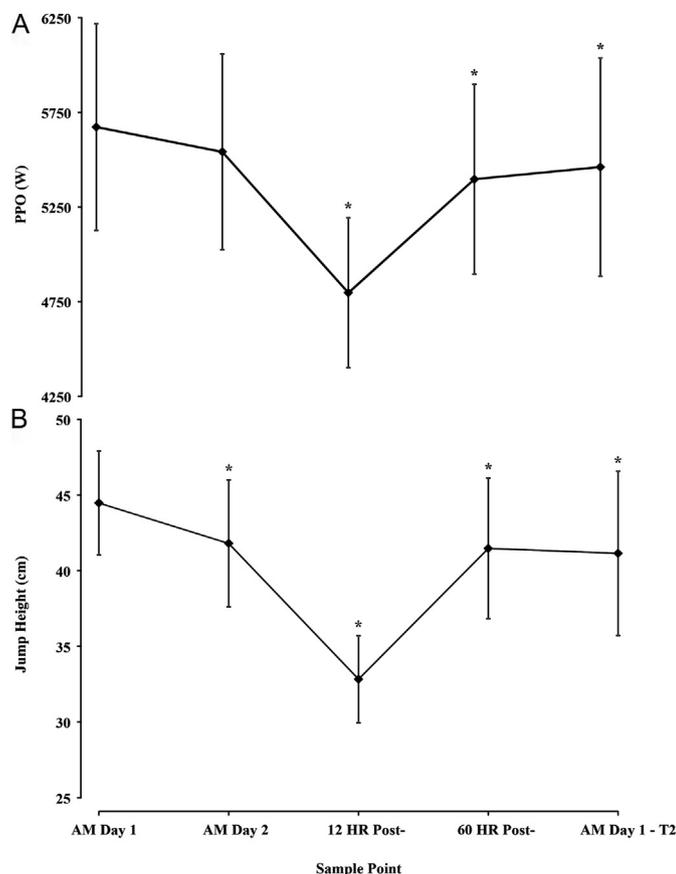


Fig. 1. Countermovement peak power output (A) and jump height time-course changes from the AM of tournament 1 to the 1 AM of tournament 2 (7 days later). *Indicates significant difference when compared with AM Day 1 ($P < 0.05$). Data presented as \pm SD ($N = 10$).

1 through to AM day 1 of tournament 2, 7 days later (Fig. 1A). PPO tended to decrease from AM day 1 to AM day 2 of tournament 1 ($P = 0.072$), was decreased at 12 h (95% CI = -681 to -1071 W; $-15.5 \pm 4.3\%$; $P = 0.032$) and 60 h post-tournament 1 (95% CI = -160 to -340 W; $-5.1 \pm 3.0\%$; $P = 0.001$), and remained reduced at AM day 1 of tournament 2 (95% CI = -149 to -274 W; $-4.1 \pm 1.6\%$; $P < 0.001$; Fig. 1A).

There was a significant time effect ($P < 0.001$; Partial- $\eta^2 = 0.529$) in the players JH from AM day 1 of tournament 1 through to AM day 1 of tournament 2 (Fig. 1B). JH decreased from AM day 1 to AM day 2 at tournament 1 (95% CI = -1.1 to -4.1 cm; $-6.0 \pm 5.4\%$; $P = 0.016$), was decreased at 12 h (95% CI = -9.9 to -13.4 cm; $-26.1 \pm 5.0\%$; $P < 0.001$) and 60 h post-tournament 1 (95% CI = -1.4 to -4.2 cm; $-7.1 \pm 4.8\%$; $P = 0.003$) and remained reduced at AM day 1 of tournament 2 (95% CI = -1.3 to -4.9 cm; $-8.0 \pm 6.0\%$; $P = 0.007$).

A comparison of NMF responses between tournament 1 and 2 are presented in Fig. 2A and B. There was no time effect ($P = 0.683$) but there was a significant effect of tournament ($P = 0.006$; Partial- $\eta^2 = 0.587$) on player PPO (Fig. 2A). Player PPO was higher on day 1 of tournament 1, compared to day 1 of tournament 2 (95% CI = 149 to -274 W; $P < 0.001$; Fig. 2A). There was a significant time effect ($P < 0.001$; Partial- η^2) and a significant effect of tournament ($P = 0.012$; Partial- $\eta^2 = 0.526$) in player JH. Jump height significantly decreased from day 1 to day 2 during tournament 1 (95% CI = -1.1 to -4.1 cm; $P = 0.016$) but not during tournament 2 ($P = 0.186$). Furthermore, player JH was significantly lower on both day 1 (95% CI = -1.7 to -5.0 cm; $P = 0.003$) and day 2 (95% CI = -0.7 to -4.2 cm; $P = 0.02$) of tournament 2, when compared to tournament

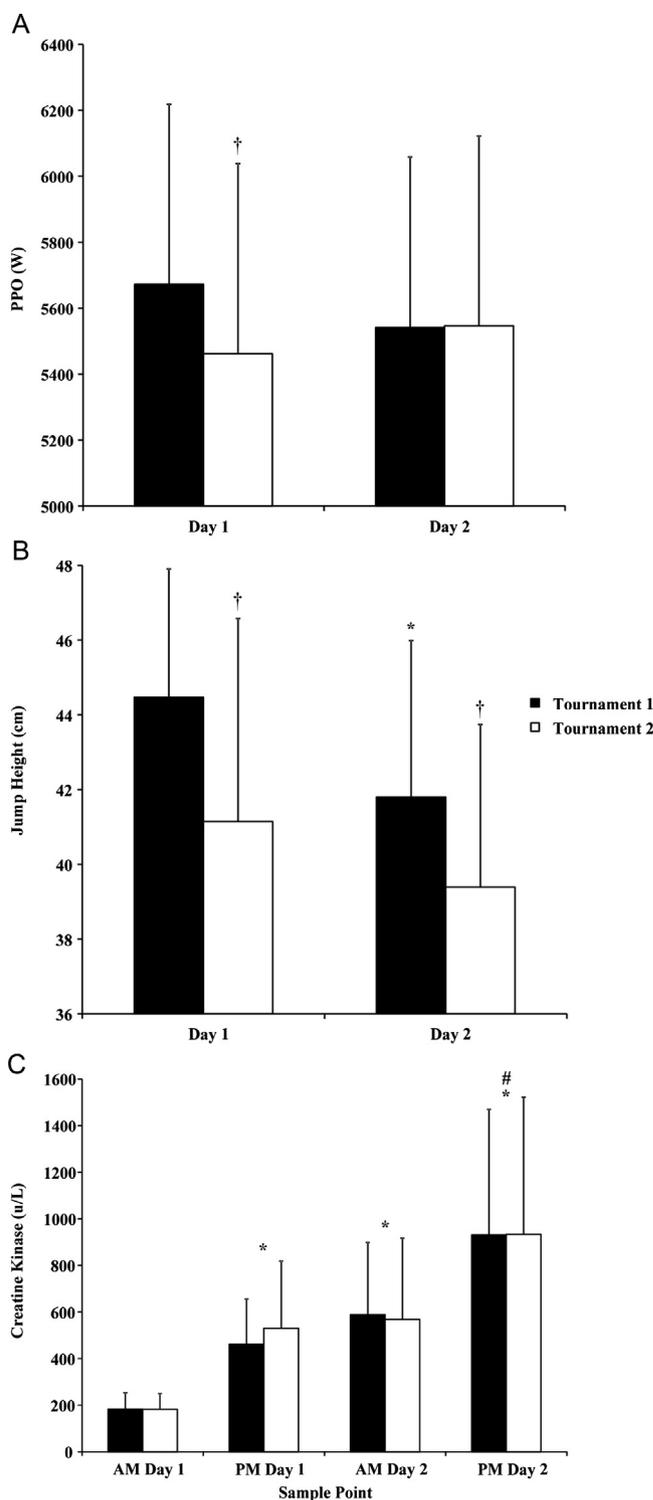


Fig. 2. Player countermovement jump peak power output (PPO; A), jump height (B) and plasma creatine kinase (C) on Day 1 and Day 2 of Tournaments 1 and 2. A and B, *indicates significantly different to Day 1 within respective tournament ($P < 0.05$); †indicates different to Tournament 1 during the same day ($P < 0.05$). C, *indicates significantly different to AM Day 1 within both tournaments ($P < 0.05$). #indicates significantly different to AM Day 2 within both tournaments ($P < 0.05$).

1 (Fig. 2B). The plasma creatine kinase responses are presented in Fig. 2C. There was a significant time effect ($P = 0.003$; Partial- $\eta^2 = 0.716$), but no effect of tournament on player CK responses ($P = 0.302$). CK concentrations increased from AM to PM on day 1, and day 2 during both tournaments 1 and 2 (Fig. 2C).

There was no significant time effect on player body mass during the observational period (AM day 1 tournament 1 89.1 ± 9.3 ; AM day 2 tournament 1 89.3 ± 9.3 ; 12 h 90.2 ± 9.8 ; 60 h 89.9 ± 9.5 ; AM day 1 tournament 2 89.9 ± 9.6 ; AM day 2 tournament 2 90.1 ± 9.0 kg; $P=0.556$).

4. Discussion

The aim of this study was to observe the time-course changes in neuromuscular function (NMF) and markers of muscle damage within and between two elite rugby union sevens tournaments played on consecutive weekends. Here we show that the first tournament resulted in a $\sim 26\%$ reduction in NMF post-tournament, and NMF remained $\sim 8\%$ reduced at the onset of the second tournament, 5 days later. In addition, our data demonstrate that plasma creatine kinase (CK) concentrations increased transiently throughout a tournament, but fully recovered between tournament weekends.

Player CMJ jump height was reduced by $\sim 26\%$ at 12 h post-tournament 1 and remained reduced by $\sim 8\%$ by the start of tournament 2, 5 days later (Fig. 1). The initial decline in NMF is consistent with findings from research within professional rugby union⁷ and league¹¹ players; McLellan and Lovell¹¹ demonstrated reductions in peak power output of $\sim 10\%$ for up to 48 h post-match, before recovering at 72 h. This transient reduction in NMF is potentially due to an impairment of excitation-contraction coupling, which is a result of low-frequency fatigue.^{11–13} Furthermore, there is potential that muscle damage, induced by high-intensity sprinting,¹⁴ potentially inducing some selective damage of type II muscle fibres,¹² and collisions during match play,¹⁵ are also contributing factors to the reduced NMF. There was an incomplete recovery of NMF on the morning of the first day of tournament 2, despite the return of plasma CK to baseline concentrations (Figs. 1 and 2). Speculatively, the reduction in NMF could be potentially attributed to a degree of low-frequency fatigue, inflammation induced by the preceding tournament,⁵ and potentially there could be a role for player perception of soreness/recovery from tournament 1.¹⁶

Plasma CK concentrations increased within both tournaments by $\sim 250\%$ from the morning to evening of day 1, and by the end of day 2 had increased from baseline by $\sim 500\%$ (Fig. 2C). These increases in CK paralleled a reduction in NMF (Figs. 1 and 2). The increase in CK on the 1st day of the tournament is in line with previous findings⁴; Takahashi et al.,⁴ found a $\sim 42\%$ increase in serum CK after 2 games in international sevens players. The increase in CK is potentially attributed to a large percentage of game play involving high-intensity sprinting² and rapid decelerations,¹ which is characterised by repeated eccentric contractions of the lower body,¹⁴ as well as some blunt force trauma/physical collisions,¹⁷ and the high physiological stress imposed during match play. Plasma CK had returned to baseline concentrations within 5 days, with very similar concentrations on the morning of day 1 in both tournaments; however, CK concentrations have been reported to be elevated 5 days after a professional rugby league match.¹⁴ The number of contacts/tackles made during rugby match play has been shown to relate to the increase in CK and myoglobin, and thus the degree of skeletal muscle damage.¹⁷ Speculatively, the lower total accumulative maximal sprint distances covered ($\sim 142^2$ vs. ~ 321 m¹⁸), and the lower number of physical collisions during sevens match play, compared with rugby league, could potentially explain the recovery of plasma CK by the start of tournament 2. However, there is a lack of collision/contact data examining this in rugby sevens play to confirm this hypothesis.

To our knowledge we are the first to profile the time-course recovery of NMF and markers of muscle damage over two consecutive tournaments in elite rugby union sevens players. As such, we

are limited in our ability to compare our findings to data collected during different stages of the competition. Given the potential for the IRB world sevens series to involve significant travel in between tournaments, it is reasonable to speculate that factors such as climate, jet lag and travel fatigue could exacerbate the reduction in NMF experienced by the players between stages. Despite limited travelling between tournament stages, and the performance of a pool recovery session, the players involved in this study still experienced a prolonged reduction in NMF. Thus, these data may indicate the need for enhanced recovery strategies to be employed during this period, alongside optimising between tournament travelling, in order for players to perform at their peaks across two consecutive tournaments.

5. Conclusion

In conclusion, we profiled the time-course changes in NMF recovery between two rugby union sevens tournaments played on consecutive weekends. Our data demonstrate that NMF is significantly reduced following the first tournament weekend and does not fully recover for the start of the second. Creatine kinase also increases markedly during a tournament, but appears to return to baseline between tournament weekends. For peak performance across two consecutive tournaments more intensive approaches to recovery should be considered.

6. Practical implications

- A single tournament significantly reduces neuromuscular function for >120 h post-tournament.
- Markers of muscle damage may not be elevated, but practitioners should be aware that a reduced neuromuscular function may still be present.
- For the purpose of player performance and welfare, enhanced recovery strategies may need to be implemented during the (5 day) break between tournaments.
- Future research should seek to explore between tournament player recovery more closely, e.g. upper body neuromuscular function recovery, time course recovery of creatine kinase, hormonal and inflammatory responses, and the optimisation of recovery techniques. There is also a need to examine player recovery responses to different tournament structures (e.g. a single 3 day tournament).

Acknowledgements

This study was funded by UK Sport.

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