
CREATINE KINASE AND ENDOCRINE RESPONSES OF ELITE PLAYERS PRE, DURING, AND POST RUGBY LEAGUE MATCH PLAY

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ABSTRACT

McLellan, CP, Lovell, DI, and Gass, GC. Creatine kinase and endocrine responses of elite players pre, during, and post rugby league match play. *J Strength Cond Res* 24(11): 2908–2919, 2010—The purpose of the present study was to (a) examine player-movement patterns to determine total distance covered during competitive Rugby League match play using global positioning systems (GPSs) and (b) examine pre, during, and postmatch creatine kinase (CK) and endocrine responses to competitive Rugby League match play. Seventeen elite rugby league players were monitored for a single game. Player movement patterns were recorded using portable GPS units (SPI-Pro, GPSports, Canberra, Australia). Saliva and blood samples were collected 24 hours prematch, 30 minutes prematch, 30 minutes postmatch, and then at 24-hour intervals for a period of 5 days postmatch to determine plasma CK and salivary testosterone, cortisol, and testosterone:cortisol ratio (T:C). The change in the dependent variables at each sample collection time was compared to 24-hour prematch measures. Backs and forwards traveled distances $5,747 \pm 1,095$ and $4,774 \pm 1,186$ m, respectively, throughout the match. Cortisol and CK increased significantly ($p < 0.05$) from 30 minutes prematch to 30 minutes postmatch. Creatine kinase increased significantly ($p < 0.05$) postmatch, with peak CK concentration measured 24 hours postmatch (889.25 ± 238.27 U·L⁻¹). Cortisol displayed a clear pattern of response with significant ($p < 0.05$) elevations up to 24 hours postmatch, compared with 24 hours prematch. The GPS was able to successfully provide data on player-movement patterns during competitive rugby league match play. The CK and endocrine profile identified acute muscle damage and a catabolic state associated with Rugby League match play. A return to normal T:C within 48

hours postmatch indicates that a minimum period of 48 hours is required for endocrine homeostasis postcompetition. Creatine kinase remained elevated despite 120 hours of recovery postmatch identifying that a prolonged period of at least 5 days modified activity is required to achieve full recovery after muscle damage during competitive Rugby League match play.

KEY WORDS muscle enzyme, salivary testosterone, cortisol ratio, GPS, monitoring

INTRODUCTION

Rugby League is a heavy contact sport played internationally and involves frequent bouts of high-intensity exercise separated by bouts of low-intensity exercise during match play (10). Traditionally, movement patterns associated with rugby league match play have been examined using time–motion analysis systems incorporating match video recordings (20,21). Varied and inconsistent categories to describe locomotor activity, however, increase the likelihood of error given the complex and varied nature of rugby league match play. Furthermore, the labor-intensive nature of video-based analysis systems and an inability of these systems to operate in real time may delay and add to error in match-play analysis (6,8).

Recent studies (15,31) have added to our understanding of the performance characteristics of professional Rugby League match play. Advances in notational analysis technologies, such as global positioning systems (GPSs) permit real-time quantitative assessment of the physiological demands of match play and player-movement patterns (4,19). The validity and reliability of GPS for assessing movement patterns in field-based sports have been reported (4,8,19,26,27); however, there are few data to identify the validity and reliability of GPS devices to measure intermittent high-intensity exercise that is characteristic of Rugby League match play (4). As more advanced technologies for performance analysis emerge, there is a need for a concomitant increase in match-play analysis methodologies to increase our understanding of the applied physiology of performance and to improve training practices to achieve desired performance outcomes.

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The combative nature of Rugby League match play combined with intermittent high-intensity activity during competition is synonymous with repeated blunt force trauma, microdamage to skeletal muscle, and postexercise muscle soreness. Exercise-induced muscle damage has been examined in humans (7,13) with plasma Creatine kinase concentration ([CK]) commonly reported as an indirect marker of skeletal-muscle damage (25,35). Elevated plasma [CK] has been reported after competitive match play in contact sports (17,34,35) suggesting that significant skeletal-muscle damage occurs during such contact sports. Takarada (35) reported a significant correlation between the number of tackles performed during Rugby Union match play with peak [CK] measured 24 hours postmatch. The CK and endocrine response to competitive Rugby League match play however is unknown.

The examination of endocrine measures in response to competitive contact sport performance (3,9,13,17) and during the postcompetition recovery period (9,17) are common practice in professional sports. Testosterone and cortisol have been identified as reliable markers of the endocrine response to competitive contact sport performance (3,9,23). Testosterone is the primary anabolic marker for protein signaling (33) and muscle glycogen synthesis (30). Cortisol is considered an important stress hormone and acts antagonistically with testosterone to mediate catabolic activity, increasing protein degradation and decreasing protein synthesis in muscle cells (16). It is possible that various hormonal and muscle enzyme measures may assist in assessing the immediate response and time course of recovery after competition.

The use of salivary testosterone concentration ([sTest]) and salivary cortisol concentration ([sCort]) assay measures provides a relatively simple, noninvasive procedure that provides a valid and reliable indication of plasma-unbound cortisol (2,37) and plasma-free testosterone (38). Testosterone and cortisol have been reported to vary in opposite directions in response to exercise, producing a decreased testosterone and cortisol ratio (T:C) when training and competitive demands are increased (9,14). Consequently, T:C has been used to examine the anabolic:catabolic endocrine profile of athletes from contact sports (3,9); however, the response of testosterone and cortisol to competitive Rugby League match play is unreported. A better understanding of the endocrine response to competitive Rugby League match play and the short-term postmatch recovery period may provide scope for improved individualized training and recovery strategies.

Uncertainty remains regarding the pattern of CK and endocrine responses to elite level contact sport, and the influence of an elite Rugby League match is unknown. It remains unclear whether the incorporation of such measures is of use to monitor performance in an applied sports setting. The aim of the present study therefore is to (a) examine player-movement patterns to determine total distance covered during competitive Rugby League match

play using GPS and (b) examine pre, during, and postmatch CK and endocrine responses to competitive Rugby League match play. We hypothesize that Rugby League match play will result in substantial skeletal-muscle damage and considerable elevation in stress hormone levels postmatch. Further, the combination of GPS performance data with CK, sCort, and sTest provides a more detailed and specific analysis of the demands of Rugby League match play than achieved previously.

METHODS

Experimental Approach to the Problem

Global positioning system technology was used to examine the independent variable of player-movement characteristics to determine positional running profiles during elite Rugby League match play. Plasma CK activity was examined to reflect skeletal-muscle damage in response to the demands of match play. Cortisol and testosterone were examined to represent the primary catabolic and anabolic endocrine measures associated with metabolism and protein synthesis, respectively, pre and postmatch. To examine the acute and short-term postmatch response of the dependent variables, sCort, sTest, and [CK] were measured via saliva and blood samples, respectively. The T:C was examined to identify the balance between anabolic and catabolic metabolism. An understanding of player-movement characteristics, endocrine responses, and skeletal-muscle damage markers after competitive Rugby League match play is important to monitor recovery and effectively manage the prematch training and preparation process for subsequent matches.

Subjects

Seventeen elite male Rugby League players, age 19.0 ± 1.3 years, height 188 ± 2.3 cm, and mass 89.6 ± 15.8 kg, representing a National Rugby League (NRL) team volunteered to participate in the study. Data were collected during a single game of Rugby League with all participants completing a minimum of 30 minutes of match play in each of the 2 40-minute halves of the match. Before the commencement of the study, participants attended a presentation outlining the purpose, benefits, and procedures associated with the study. Written informed consent was obtained from all participants. The study was approved by the Bond University Human Research Ethics Committee.

Procedures

Saliva and blood samples were collected 24 hours prematch; 30 minutes prematch; within 30 minutes postmatch; and at 24, 48, 72, 96, and 120 hours postmatch. The saliva and blood collection schedule is outlined in Table 1. Subjects were asked to refrain from strenuous exercise during the 24 hours before baseline saliva and blood-sample collection (24 hours prematch). Saliva and blood samples were collected daily between 1530 and 1630 hours with the exception of the 30-minute postmatch saliva and blood samples that were collected between 1830 and 1900 hours due to the time of

TABLE 1. Saliva and blood sample collection schedule 24 hours prematch to 120 hours postmatch.

Sample	1	2 & 3	4	5	6	7	8	
Day	Pre Game	Game day			← Postgame →			
Time (h)	24	Pre/postmatch	24	48	72	96	120	
Sample	Saliva Blood	Saliva (pre & post) Blood (pre & post)	Samples collected every 24 hrs for the next 5 days.					Samples collected every 24 hrs for the next 5 days.

match play. Players provided saliva and blood samples within 30 minutes of match completion and before participation in postmatch team recovery activities. Throughout the postmatch data collection period (30 minutes postmatch to 120 hours postmatch), all subjects participated in all team recovery and training sessions (Table 2). An example of a training week during a 6-week preseason phase is outlined in Table 3.

Global Positioning System Analysis

The present study used commercially available 5-Hz GPS receivers (SPI-Pro, GPSports, Canberra, Australia) that operated in nondifferential mode and provided data in real time. The SPI-Pro units contain a triaxis accelerometer that measures accelerations in gravitational force (G force) on 3 planes, namely, forwards-backwards, up-down, and tilt left-right. The GPS model used in the current study (70 g; 45 mm × 90 mm × 34 mm) was worn in a purpose designed vest (GPSports, Australia) to ensure that range of movement of the upper limbs was not restricted. The GPS unit was worn in a padded mini-backpack in the rear of the vest and positioned in the center of the upper back slightly superior to the shoulder blades at the level of approximately thoracic vertebrae 2 (T2).

Subjects had worn GPS units during outdoor-training sessions that included Rugby League specific running, skill-related, and game simulated-contact activities. None of the players complained of any discomfort or impediment to their normal range of movement or performance from wearing the equipment during training or match participation. Data provided from the GPS units included total distance and

speed characteristics. Raw accelerometer data were available in real time via Wireless-Fidelity communication and were displayed using commercially available software (Team AMS, GPSports, Australia). The reliability of the GPS units used in the present study has previously been tested in our laboratory over distances of 10–8,000 m on a synthetic 400-m athletics track with variation <3% and the reliability of speed assessed with electronic timing gates (Smartspeed, Fusion Sports, Brisbane, Australia) from walking speed (6 km·h⁻¹) to a maximum sprint speed (>23.1 km·h⁻¹) with variation <5.5%. Our results are similar to others who have reported the reliability of the SPI-Pro GPS units (26).

Movement Classification System

Movement zones form the basis of the analysis performed by the Team AMS software, allowing 6 ranges of speed (km·h⁻¹ and m·s⁻¹) to be set and used for analysis. Zone 1 indicates the lowest effort or lowest velocity of movement with each zone progressively categorizing effort and movement intensity to zone 6 representing the highest effort and intensity of movement. The movement classification system used in the present study was based on methods reported elsewhere (5) and modified to consider forward, backward, and lateral ambulatory movement. No attempt was made to quantify movement characteristics associated with contact sport-specific movements such as tackling, wrestling, jumping, and scrimmaging in the present study. Each movement was coded as 1 of 6 speeds of locomotion (Table 4) with the frequency and duration of entries into each movement zone providing a more precise profile of activity patterns among playing position during competitive match play.

TABLE 2. Saliva and blood sample collection and training schedule 24 hours pre to 120 hours postgame.

Day	1	2	3	4	5	6	7
Sample	24 Pre	Game day Pre & post	24 Post	48 Post	72 Post	96 Post	120 Post
AM	Off	Off	Recovery 1	Off	Off	Off	Off
PM	Team training	Game	Recovery 2	Strength	Team skills	Strength	Team skills

TABLE 3. An example of a training week during a 6-week preseason phase in professional Rugby League*†.

Session	Monday	Tuesday	Wednesday	Thursday	Friday	Saturday	Sunday
AM	Team training (wrestle) 60–90 min	Resistance training 2 Upper body strength & power 60 min 10–15 sets 4–8RM	Speed training 45–60 min 100 Club 45–60 min	Team training (conditioning) 60–90 min	Resistance training 3 Upper body push/upper body pull 60 min 10–15 sets 6–8RM	Resistance training 4 Lower body/upper body push 60 min 10–15 sets 6–8RM	Active rest
PM	Resistance training 1 Lower body 60 min 10–15 sets 4–8RM	Team training (skills)/recovery, 60–90 min	Active rest	Crosstraining, 45–60 min	Team training (skills)/recovery, 60–90 min	Active rest	Active rest

*Reps = repetitions and 1RM = 1 repetition maximum.

†Resistance training—typical exercises were as follows: strength exercises: squat variation, vertical push, vertical pull, horizontal push, and horizontal pull. Power exercises: bench throw, squat jump, power clean, and push press variations Team training (wrestle): individual/partner attack and defence tackling and ruck play drills. Team training (skills): attack and defensive patterns, game plans, and general skills. Recovery: pool deep-water running (15 minutes), general stretching (20 minutes), and cold water immersion (10 minutes), massage (30 minutes). Speed training: agility/footwork and reaction drills for 10 minutes, straight line and change of direction sprints 5–50 m × 4–8 reps, resisted (towing, weighted sled, and uphill sprints)/assisted (overspeed bungees, catapult sprints, and down-hill sprints) 10–40 m × 4–8 reps, plyometric drills (bounding, repeated horizontal jumps, and repeated hurdle jumps). Team training (conditioning): aerobic and anaerobic conditioning activities, repeated high-intensity running efforts. Hundred club: additional aerobic and anaerobic conditioning for nominated players. Crosstraining: boxing, squash, water polo, beach volleyball, surfing, kayaking, outriggers.

TABLE 4. Speed zone classification using team AMS software.

Zone	km·h ⁻¹	m·s ⁻¹	Movement classification	Definition
1	0–6.0	0–1.6	Standing/walking	Standing or walking at low intensity, no flight phase associated with ambulatory movement in any direction
2	6.1–12.0	1.6–2.7	Jogging (low-intensity running)	Running in any direction with minimal flight phase and minimal arm swing (1/4 pace)
3	12.1–14.0	2.7–3.8	Cruising (moderate-intensity running)	Running in any direction with progressive acceleration and elongation of stride length with moderate arm swing (1/2 pace)
4	14.1–18.0	3.8–5.0	Striding (medium-intensity running)	Running with increased velocity and arm swing (3/4 pace)
5	18.1–20.0	5.0–5.5	High-intensity running	Running at near maximum pace (>85%) with near maximum stride length, stride frequency, and arm swing
6	>20.1	>5.6	Sprinting	Running with maximum effort

Plasma Creatine Kinase Sampling and Analysis

Plasma [CK] was determined from 30- μ L capillarized whole-blood samples collected via fingertip puncture made using a spring-loaded single use disposable lancet. Blood samples were collected from subjects simultaneously at the time of saliva sample collection (Table 1). Whole-blood samples were centrifuged (Hereaus Function Line, Labofuge 400, Kendro Laboratory Products, Hanau, Germany) at 3,000 rpm for 10 minutes, and separated plasma was stored at a temperature of -30°C until analysis. Plasma samples were analyzed using a Reflotron spectrophotometer (Abbott Architect, Abbott Park, IL, USA) via an optimized UV test.

Salivary Testosterone and Cortisol Sampling and Analysis

Unstimulated saliva was collected via passive drool into a plastic tube for analysis of [sTest], [sCort] and used in the subsequent calculation of T:C. Saliva measures of [sTest] and [sCort] are independent of salivary flow rate (28). There

is a strong relationship between saliva and serum unbound cortisol concentration ($r = 0.87$), salivary-free testosterone and serum testosterone ($r = 0.96$) and the T:C in saliva is highly correlated with that in serum ($r = 0.83$) (22). All subjects were requested to avoid the ingestion of food and fluids other than water in the 60 minutes before providing each saliva sample and refrain from brushing their teeth 2 hours before each saliva sample-collection session. Subjects were instructed to wait for a period of 10 minutes after their last consumption of water before commencing the saliva sample-collection process. Saliva samples were stored at a temperature of -80°C until analysis.

Saliva cortisol ($\mu\text{g}\cdot\text{dL}^{-1}$) and Testosterone ($\text{pg}\cdot\text{mL}^{-1}$) were analyzed in duplicate via a commercially available enzyme-linked immunosorbent assay (Salimetrics LLC, State College, PA, USA) using a microplate reader (SpectraMax 190, Molecular Devices, Fullerton, CA, USA). Standard curves were constructed as per the manufacturer's instructions and

TABLE 5. Running speed and distance traveled for the first half, second half, and whole match for forwards and backs.

		Forwards ($n = 8$)		Backs ($n = 7$)	
Speed ($\text{m}\cdot\text{s}^{-1}$)	Maximum average	First half	6.8 ± 0.3	$7.5 \pm 0.8^*$	
		Second half	6.7 ± 0.1	$8.6 \pm 0.1^*$	
		Whole game	6.8 ± 0.3	$8.6 \pm 0.1^*$	
		First half	2.9 ± 0.9	3.4 ± 1.7	
		Second half	3.8 ± 1.4	4.8 ± 0.6	
		Whole game	3.2 ± 0.8	3.9 ± 1.0	
Average distance traveled (m)	First half	$2,367 \pm 620$	$3,095 \pm 510$		
	Second half	$2,463 \pm 570$	$2,936 \pm 573$		
	Whole game	$4,774 \pm 1,186$	$5,747 \pm 1,095$		

*Significant difference ($p < 0.05$) compared with forwards.

commercially available standards, and quality control samples were used for both assays (Salimetrics LLC). Assay sensitivity was $3.70 \text{ pg}\cdot\text{ml}^{-1}$ for sTest with intra-assay coefficient of variation (CV) as a percentage of 5.9%. Cortisol sensitivity was $0.007\text{ng}\cdot\text{ml}^{-1}$ with an average intra-assay CV of 2.6%. All samples were analyzed in the same series to avoid interassay variability. Testosterone to cortisol ratio was determined by dividing the concentration of sCort by the concentration of sTest at each 24-hour saliva sample-collection period.

Statistical Analyses

Endocrine and muscle enzyme variables analyzed prematch and postmatch included sTest, sCort, and CK. Before statistical analysis, log transformation was applied to the endocrine data to normalize the distribution and reduce nonuniformity bias. The data for each of the dependent variables are represented as mean ($\pm SEM$) using standard statistical methodology. Changes in hormonal concentrations were analyzed using a one-way repeated measures analysis of variance. Significant differences were identified via a Bonferroni post hoc test. The criterion level for statistical significance was set at $p \leq 0.05$. The correlation between peak changes in endocrine measures and GPS variables was analyzed using the Pearson product-moment correlation coefficient. The mean CV for CK assays was 6.1%. All statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS for Windows, version 14.0; SPSS, Inc., Chicago, IL).

RESULTS

Global Positioning Systems Movement Analysis

There was no significant difference in the total distance traveled during the match between backs ($5,747 \pm 1,095 \text{ m}$) and forwards ($4,774 \pm 1,186 \text{ m}$; Table 5). Backs traveled greater distance at high-intensity running ($5.0\text{--}5.5 \text{ m}\cdot\text{s}^{-1}$) ($135 \pm 49 \text{ m}$; $p < 0.05$) and sprinting ($>5.6 \text{ m}\cdot\text{s}^{-1}$) ($290 \pm 69 \text{ m}$; $p < 0.05$) compared with the forwards (82 ± 21 and $149 \pm 32 \text{ m}$, respectively; Table 6) during the whole match. In the first half, there was no significant difference in the total distance traveled between the backs and forwards (Table 5); however, backs traveled greater distance at sprinting speeds ($110 \pm 32 \text{ m}$; $p < 0.05$) compared with the forwards ($68 \pm 13 \text{ m}$; Table 6). In the second half, there was no significant difference in the

TABLE 6. Distance traveled in different speed zones for the first and second halves and whole match for forwards and backs.

	Speed ($\text{m}\cdot\text{s}^{-1}$)	Forwards (m) ($n = 8$)	Backs (m) ($n = 7$)
First half	0–1.6	$1,037 \pm 378$	$1,200 \pm 379$
	1.6–3.3	841 ± 258	870 ± 272
	3.3–3.9	156 ± 49	263 ± 77
	3.9–5.0	158 ± 54	195 ± 59
	5.0–5.6	35 ± 12	57 ± 21
Second half	>5.6	68 ± 13	$110 \pm 32^*$
	0–1.6	923 ± 338	$1,195 \pm 263$
	1.6–3.3	804 ± 287	930 ± 265
	3.3–3.9	230 ± 70	244 ± 76
	3.9–5.0	168 ± 56	$267 \pm 68^*$
Whole game	5.0–5.6	39 ± 13	$87 \pm 30^*$
	>5.6	82 ± 14	$177 \pm 38^*$
	0–1.6	$2,021 \pm 496$	$2,407 \pm 541$
	1.6–3.3	$1,739 \pm 456$	$1,605 \pm 424$
	3.3–3.9	419 ± 115	410 ± 134
3.9–5.0	368 ± 101	440 ± 145	
5.0–5.6	82 ± 21	$135 \pm 49^*$	
>5.6	149 ± 32	$290 \pm 69^*$	

*Significant difference ($p < 0.05$) compared with forwards.

total distance traveled between the backs and forwards. The backs traveled greater distance at moderate-intensity running ($267 \pm 68 \text{ m}$; $p < 0.05$), high-intensity running ($87 \pm 30 \text{ m}$; $p < 0.05$), and sprinting speeds ($177 \pm 38 \text{ m}$; $p < 0.05$)

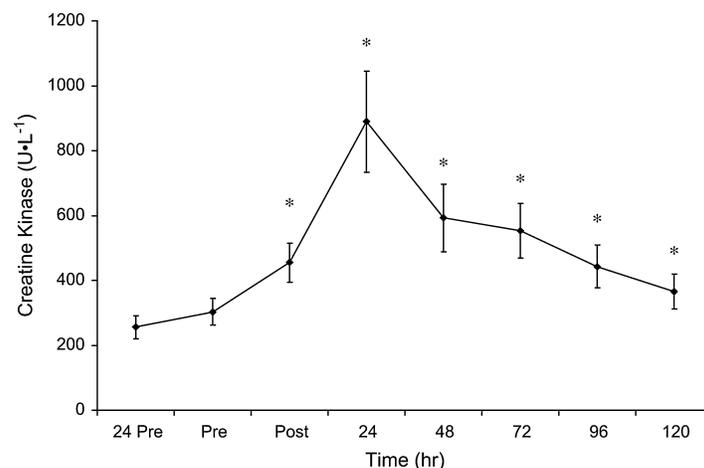


Figure 1. Serum creatine kinase concentration pre and post rugby league match play. All data log transformed and are reported as mean $\pm SEM$. *Significantly ($p < 0.05$) different from 24 hours prematch.

compared with the forwards (168 ± 56 , 39 ± 13 , and 82 ± 14 m, respectively; Table 6).

Creatine Kinase

Plasma CK concentrations ([CK]) measured from 24 hours prematch to 120 hours postmatch are displayed in Figure 1. Plasma [CK] was not significantly correlated ($p > 0.05$) with total distance traveled ($r = 0.28$) during the match. In comparison to 30 minutes prematch, a significant increase in [CK] was established immediately postmatch ($p < 0.05$) with a further significant increase and peak measure at 24 hours postmatch ($p < 0.05$). Substantial increases in [CK] were identified immediately postmatch (+56%) and 24 hours postmatch (+91%) with progressive decreases in [CK] from 48 hours postmatch (-32%), 72 hours postmatch (-3%), 96 hours postmatch (-18%), and 120 hours postmatch (-12%). In comparison with 24 hours prematch, significant increases in [CK] were identified at all subsequent sample-collection points. Despite 120 hours of recovery postmatch plasma, CK did not return to 24-hour prematch baseline levels.

Cortisol

No significant correlation ($p > 0.05$) was found for sCort and sTest and total distance traveled ($r = 0.09$ and $r = -0.07$, respectively) or during the whole match. The [sCort] response from 24 hours match to 120 hours postmatch is shown in Figure 2. Before the start of the match, a significant increase ($p < 0.05$; +28%) in [sCort] was found between 24 hours prematch and 30 minutes prematch. The [sCort] continued to increase significantly ($p < 0.05$; +68%) from 30 minutes prematch to 30 minutes postmatch resulting in the peak [sCort]. A significant decrease in [sCort] was found at 24 hours postmatch ($p < 0.01$; -32%). A return of [sCort] to baseline measures was evident 48 hours postmatch (-37%),

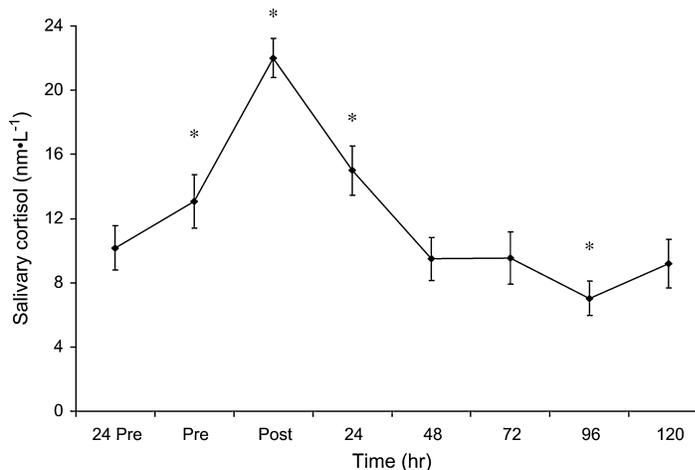


Figure 2. Saliva cortisol concentration pre and postrugby league match play. All data are log transformed and are reported as mean \pm SEM. *Significantly ($p < 0.05$) different from 24 hours prematch.

which remained below baseline measures for the remainder of the study.

Testosterone

The [sTest] response to competitive match play can be found in Figure 3. There was a significant decrease in [sTest] from 24 hours prematch to 30 minutes prematch ($p < 0.01$; -47%). Despite a small increase (+14%) in [sTest] 30 minutes postmatch, [sTest] remained significantly reduced ($p < 0.05$) in comparison to 24 hours prematch. The [sTest] increased (+33%) 24 hours postmatch resulting in a return to the

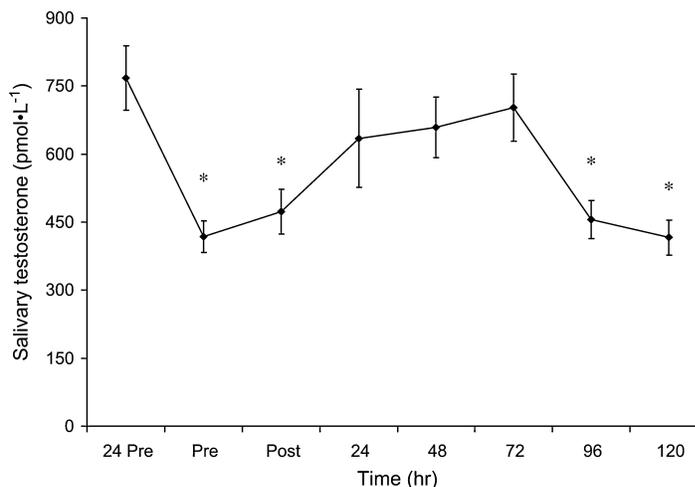
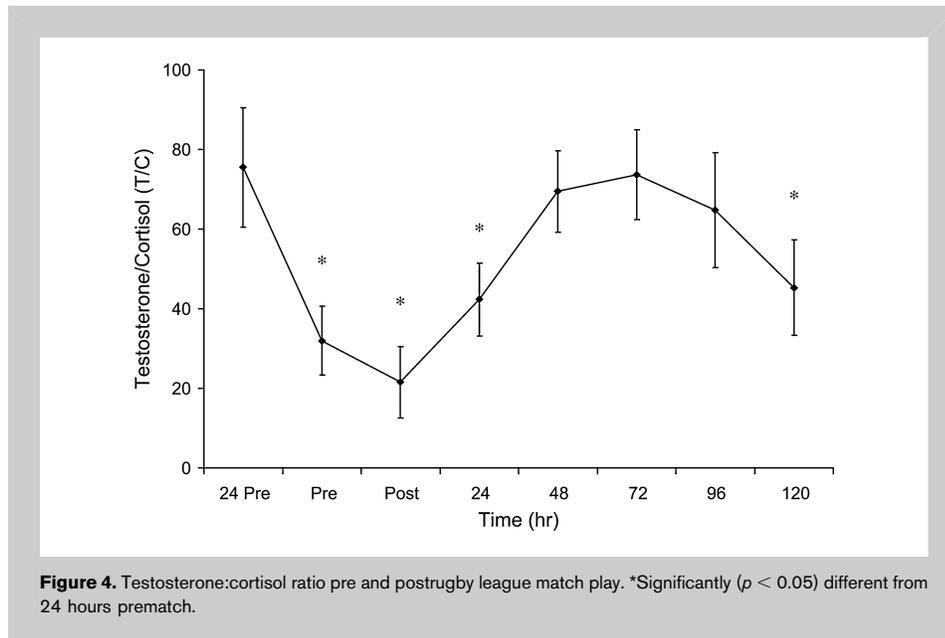


Figure 3. Saliva testosterone concentration pre and postrugby league match play. All data are log transformed and are reported as mean \pm SEM. *Significantly ($p < 0.05$) different from 24 hours prematch.



24 hours prematch baseline measures. There was a significant decrease in [sTest] after 96 hours ($p < 0.05$; -29.35%) and 120 hours ($p < 0.05$; -7.56%) of recovery, returning [sTest] concentration to below 24 hours prematch levels.

Testosterone:Cortisol Ratio

The T:C from 24 hours prematch to 120 hours postmatch is shown in Figure 4. A significant decrease in T:C was found from 24 hours prematch to 30 minutes prematch ($p < 0.05$; -58%) followed by a further decrease 30 minutes postmatch ($p < 0.05$; -33%). During the acute recovery phase, despite a considerable increase ($+97\%$), T:C remained significantly reduced ($p < 0.05$) 24 hours postmatch in comparison to 24 hour prematch baseline measures. A substantial though not significant increase in T:C ($+64\%$) was recorded at 48 hours postmatch, returning T:C to 24-hour prematch levels. After 120 hours of the short-term recovery phase, T:C decreased ($p < 0.05$) significantly in comparison to 24-hour prematch baseline measures.

DISCUSSION

The primary findings of the present study are that participation in competitive rugby league match play results in a significant increase in muscle damage postmatch, indicated by elevated [CK] that peaks within 24 hours postmatch and remained elevated in comparison to prematch values for at least 120 hours after competition. An anticipatory rise in the concentration of sCort was found before match play followed by an acute and considerable increase in [sCort] immediately postmatch. The [sCort] was found to peak immediately postmatch followed by a rapid return to resting concentrations within 24–48 hours postmatch. Conversely, a reduction in [sTest] was found prematch followed by an

acute postmatch increase that progressively returned to baseline concentration within 24 hours postmatch.

The present study found no significant difference in the total distance traveled between backs and forwards in either half of play or over the full match. The total full-match mean distances reported for backs and forwards were $5,747 \pm 1,095$ and $4,774 \pm 1,186$ m, respectively. The maximum distances traveled by players in the present study are similar to the findings of others (15,31) using match video recordings to analyze movement characteristics in Rugby League match play. The similarity of distances covered between backs and for-

wards and the consistency of running characteristics in each half of the match indicate that match intensity was maintained and the characteristics of running performance did not deteriorate during the whole match. Further, similarity in the distances recorded by GPS and video analysis methods suggests that GPS may be a useful alternative to the measurement of distances traveled by players during competitive match play in Rugby League.

In the present study, significant differences in the running speeds used to cover the total distances traveled during match play were recorded between forwards and backs. In the first half, backs traveled a significantly greater distance during maximal sprinting in comparison to forwards (110 ± 32 and 68 ± 13 m, respectively). During the second half, backs traveled significantly greater distance during striding (267 ± 68 m), high-intensity running (87 ± 30 m), and maximal sprinting (177 ± 38 m) in comparison to forwards (168 ± 56 ; 39 ± 13 ; and 82 ± 14 m, respectively). Subsequently, on the basis of whole-match performance, backs traveled significantly greater distance during high-intensity running and sprinting (135 ± 49 and 290 ± 69 m) compared with forwards (82 ± 21 and 149 ± 32 m). To our knowledge, no previous studies have quantified distances traveled by players according to speed profile characteristics and playing position during Rugby League match play.

The significant positional differences in striding, high-intensity running, and sprinting during match play is reflective of the fundamental characteristics of positional play in Rugby League. Forwards are positioned in closer proximity to the center of play, requiring those players to run shorter distances at high speed to perform game-specific tasks. Alternatively, backs are often positioned a greater distance from their opponent and therefore are required to travel greater

distances at higher speeds (10) thereby providing greater ability to achieve higher velocity running. Backs have the additional tasks of sprinting into position over greater distances to perform kick return and kick chase activities thereby increasing their opportunity to achieve maximum sprint velocities. Overall, the data indicate that backs participate in a greater amount of high-intensity locomotor activity over similar total distances in comparison to forwards during match play.

Although eccentric muscular work has traditionally been considered the predominant contributor to increased [CK] after exercise (1), recent evidence suggests that significant increases in plasma [CK] may occur as a result of physical collisions and blunt force trauma (13,32). The present study found that participation in Rugby League match play, which is characterized by repeated eccentric muscle contractions of the lower limbs, intermittent high-intensity exercise and blunt force trauma resulting from high-speed collisions between and among players, significantly increased plasma [CK] and is consistent with the findings of others (17,35).

Creatine Kinase values were found to be elevated in players 24 hours prematch after a period of complete rest ($\sim 256 \pm 123.049 \text{ U}\cdot\text{L}^{-1}$). Other research has also reported elevated CK levels precompetition (11,34,35). Suzuki et al. (34) and Takarada (35) reported [CK] of approximately 351.6 and 400 $\text{U}\cdot\text{L}^{-1}$ 48 hours prematch and same day prematch, respectively, in Japanese college rugby players. Gill et al. (11) reported CK activity of 1,023.0 $\text{U}\cdot\text{L}^{-1}$ 3.5 hours prematch in elite rugby players. The elevated prematch [CK] found in the present study is likely to indicate residual muscle damage because of game-simulated contact-training activities or the result of cumulative muscle damage associated with the rigors of competitive game participation before the commencement of the present investigation.

Creatine kinase mean values increased from 30 minutes prematch to 30 minutes postmatch (302.83 ± 144.07 to $454.83 \pm 209.36 \text{ U}\cdot\text{L}^{-1}$) followed by a significant increase of 91% in [CK] 24 hours postmatch (454.83 ± 209.36 to $889.25 \pm 538.27 \text{ U}\cdot\text{L}^{-1}$). The increase in [CK] 30 minutes postmatch agrees with results of others (11,34,35) and indicates an acute response in CK activity to match-play trauma associated with the degree of impact during collisions. Other research has reported similar (Suzuki et al. [34] $715.4 \pm 438.3 \text{ U}\cdot\text{L}^{-1}$) and greater CK levels after competitive match play in rugby union (Takarada [35] $1,081 \pm 159 \text{ U}\cdot\text{L}^{-1}$ and Gill et al. [11] $2,194.0 \pm 833.7 \text{ U}\cdot\text{L}^{-1}$).

Peak [CK] in the present study was found 24 hours postmatch ($889.25 \pm 538.27 \text{ U}\cdot\text{L}^{-1}$) and is consistent with the findings of others (35). Our findings contrast the results reported by Gill et al. (11), however, who observed peak [CK] immediately after rugby match play. The findings of the present study therefore are consistent with the concept that peak [CK] is delayed 24–96 hours postcompetition (13,17,25,35) and support the practice of prolonging the sample collection period postcompetition to accurately

assess muscle damage and provide direction with respect to the postmatch recovery process.

Methodological differences may explain the discrepancy between [CK] observed in the present study and the studies of Takarada (35), Suzuki et al. (34), and Gill et al. (11). The present study reported [CK] at each time point prematch and postmatch as mean values, whereas Takarada (35), Suzuki et al. (34), and Gill et al. (11) only reported peak CK activity. The rationale for reporting mean [CK] in the present study at each time point prematch and postmatch was to identify the overall adaptation of players from all positions during competitive match play. Peak [CK] in response to competition highlights the response of a single player to match participation and the [CK] response of any single player may therefore be determined by playing position or skill level leading to error in match-play analysis.

Although no significant positional difference was evident between [CK] and total distances traveled during the match, the backs covered greater distance at high-intensity running ($135 \pm 49 \text{ m}$; $p = 0.03$) and sprinting speeds ($290 \pm 69 \text{ m}$; $p < 0.01$) compared with the forwards (82 ± 21 and $149 \pm 32 \text{ m}$, respectively). The repeated high-intensity acceleration and deceleration associated with sprinting efforts seen in backs, requires considerable eccentric muscle activity in the hamstring muscles. An increased likelihood of structural damage associated with eccentric muscle activity may contribute to the CK response of backs. Alternatively, the exposure of forwards to repetitive high-intensity collisions may contribute to acute soft-tissue trauma and structural damage to muscle tissue.

Sampling differences may also offer an alternative explanation for variation between the present study and others regarding greater [CK] in response to match play in contact sports reported previously (11,35). The present study examined capillary blood samples, whereas Takarada (35) examined venous blood samples and Gill et al. (11) sampled interstitial fluid. On the basis that muscle-damage results in CK leakage from the muscle cells into the interstitial fluid before entering the blood through the lymphatic system, it is conceivable that [CK] in the interstitial fluid is greater than in blood because of partitioning effects (11).

The present study examined 2 hormones that represent the major catabolic and anabolic profile in response to contact-sport participation. Testosterone is the dominant anabolic marker for protein signaling and glycogen synthesis (33). Cortisol was also examined in the present study on the basis that it is dependent on the type, intensity, and duration of exercise (24) and is influenced by psychological stress (29), whereas the T:C was used to monitor the balance between anabolism and catabolism in players throughout the game preparation and recovery process.

The pattern of increased cortisol measured precompetition in contact sport is well documented (9,29). An increase in [sCort] from 24 hours prematch to 30 minutes prematch in the present study is consistent with others (29). Increased

prematch cortisol is thought to reflect a psychophysiological mechanism influenced in part by cognitive anticipation and anxiety used by athletes as a precompetitive arousal and coping mechanism used to manage pregame stress (12).

Elevated postmatch [sCort] found in the present study is consistent with other studies (3,9,17) after competitive performance. During match play, [sCort] increased 69% from 30 minutes prematch to 30 minutes postmatch and is consistent with the results of others (3,9,23) who have reported increases in sCort during exercise and competition involving high-intensity collision between opposing competitors. The mean [sCort] 30 minutes postmatch was more than double baseline measures recorded 24 hours prematch. Several factors associated with Rugby League match play provide an explanation for the sharp increase in sCort found 30 minutes postmatch.

Rugby League is a form of high-intensity, intermittent exercise of 80-minute duration involving frequent collisions with opponents and is influenced by psychological factors associated with anxiety and perceived stress. Passelergue et al. (24) identified that raised levels of anxiety and stress associated with competition contribute to elevated cortisol concentration in simulated Olympic weight-lifting competition. Lac and Berthon (18) reported that the higher the intensity and the longer the duration, the greater the cortisol response to such exercise, whereas Vanhelder et al. (36) highlighted a stronger adrenal response to intermittent anaerobic exercise in comparison to aerobic exercise. The sharp postmatch increase in [sCort] found in the present study may be explained by the interplay of psychological, exercise type, and the duration of exercise experienced during Rugby League match play.

After the peak in [sCort] measured 30 minutes postmatch, there was a significant reduction in [sCort] 24 hours postgame (-32%) and 48 hours postmatch (-37%), decreasing [sCort] to below 24 hours prematch concentrations. The return of [sCort] toward 24-hour prematch levels within 24–48 hours postmatch is consistent with others (9,17,23) who have reported a progressive decrease in cortisol postcompetition.

During the postmatch recovery phase, sCort sampling took place at 4 PM on a daily basis at 24-hour intervals for 5 days postmatch. There were further reductions in sCort at 48, 72, and 96 hours postmatch compared with 24 hours prematch. Elloumi et al. (9) reported a similar pattern of progressively reduced cortisol levels in rugby players from the first to fourth days postmatch. The progressive decline in [sCort] identified during the recovery phase in the present study is in agreement with previous reports describing the sCort response after competition (3,23) and is reflective of a return to hormonal homeostasis and removal of the psychological and physical stress associated with match play.

A nonsignificant increase in [sCort] was found 120 hours postmatch; however, [sCort] remained below 24 hours prematch levels. The trend for cortisol to become elevated toward the end of the training week in preparation for the

next game is consistent with other research (9) and in the present study is indicative of a return to high-intensity precompetition sport-specific training and the accompanying stress associated with team selection and performance expectations.

In contact sports, changes in testosterone concentration typically do not occur immediately postcompetition; however, increases have been identified during a subsequent period of recovery (9,23). The expected pattern of response of sTest during competitive Rugby League match play is unclear. In the present study, [sTest] decreased significantly ($p < 0.05$; -47%) 30-minutes prematch in comparison to 24-hour prematch baseline levels. Although the match itself produced a small increase in sTest (+14%), [sTest] remained significantly reduced compared with 24 hours prematch, supporting the results of others that have reported a reduction (3,9) in [sTest] postcontact sport participation. The present results disagree with the findings of others (13,17) who reported no change in testosterone in American Football players after match play. The inconsistency between our results and others (13,17) is likely because of considerably greater metabolic requirements of Rugby League match play in comparison to a game of American Football.

After the match a return to normalized [sTest] was evident with no significant difference between [sTest] at 24 hours postmatch in comparison to 24 hours prematch. The return to 24-hourprematch [sTest] within 24 hours of competitive match play are in contrast to the work of Elloumi et al. (9) who reported higher testosterone levels in Rugby Union players in the presence of reduced cortisol during a 6-day postcompetition period in comparison to rested values. Considerable variation in the positional play requirements, match-play intensity and postmatch recovery protocols may have contributed to inconsistency between our results and those reported after Rugby Union match play (9).

The results of the present study clearly identify a precompetition anticipatory decrease in T:C influenced by prematch anxiety and perceived stress in elite Rugby League players. The combination of substantially increased [sCort] and reduced [sTest] identified 30 minutes prematch in comparison to 24-hour prematch baseline measures resulted in a low T:C and predominant catabolic hormonal environment. The subsequent catabolic environment associated with a low T:C before match play in the present study is likely to be a reflection of the diversity of [sTest] and [sCort] prematch. Our results are in contrast to the findings of Elloumi et al. (9) who reported game day [sCort] and [sTest] was unchanged in comparison to rest. Subsequently, Elloumi et al. (9) found similar game day T:C in comparison to resting levels in rugby union players. Our results are consistent with Cormack et al. (3) who identified a similar pregame pattern in Australian Rules Football players and reported a substantial decrease in T:C immediately prematch in comparison to 48 hours prematch.

A substantial drop in T:C 30 minutes postmatch in comparison with 24 hours prematch produced the lowest T:C found during the prematch or postmatch data collection period in the present study. Despite a 97% increase in T:C 24 hours postmatch in comparison to the 30-minute postmatch level, T:C remained significantly lower than baseline levels, representing a persistent catabolic hormonal profile. The prolonged catabolic hormonal profile of players after Rugby League match play has implications for postmatch recovery and subsequent match preparation on the basis that matches may be scheduled with as few as 4 days separating match play in the NRL.

A return to baseline measures of both [sTest] and [sCort] at 48 hours postmatch is reflected in a reciprocal recovery of T:C to baseline levels within the same time period. The normalization of T:C remained evident in the present study at 72 and 96 hours postmatch and preceded a drop in T:C 120 hours postmatch in comparison to baseline levels. The reduction in T:C that occurred 5 days postmatch may reflect a return to higher intensity precompetition sport-specific training and the associated increased demand on the endocrine system.

The acute and short-term recovery phase findings with respect to T:C in the present study reflect the findings of others (3,9) that have examined the recovery status of contact-sport athletes after competitive performance. With respect to the acute 30 minute postmatch T:C response, Elloumi et al. (9) reported a substantial reduction in T:C at the end of an international level Rugby Union match. Conversely, however, during the short-term recovery phase, our findings contrast those of Elloumi et al. (9) who reported a high T:C from day 1 to day 5 postmatch in excess of baseline measures. Our results reflect the findings of Cormack et al. (3) who reported a 36% decrease in T:C from prematch to postmatch. The acute decrease in T:C is likely a function of significantly increased [sCort] and little or no change in [sTest] immediately after match play. During the short-term postmatch recovery phase, however, our results are inconsistent with those of Cormack et al. (3) who found substantially reduced T:C in all comparisons from 48 hours pregame to 120 hours postgame following Australian Rules Football performance suggesting a prolonged catabolic hormonal profile despite 5 days of recovery.

The explanation for variation in short-term recovery rates in athletes from collision sports such as Rugby League is multifactorial. The influence of individual biological responses, specialized team recovery protocols including nutrition and hydration regimes, travel commitments, and weekly team training schedules all contribute to a player's ability to recover from match play in an optimal time frame. The use of T:C to represent the anabolic:catabolic hormonal profile of athletes after competition has implications for the design and implementation of training programs, particularly in a team sport environment competing in a prolonged regular season period such as 24 matches in a 26-week period as seen in the

NRL. The return of the postmatch T:C to baseline measures identified in the present study within 48 hours is indicative of a successful recovery of [sTest] and [sCort] and thereby represents an restoration of resting anabolic:catabolic hormone profile in elite rugby league players.

PRACTICAL APPLICATIONS

The present study provides an insight into player movement patterns during elite Rugby League match play using contemporary GPS performance analysis methods that have not been reported previously. Our findings indicate that the rigors of elite Rugby League matchplay result in skeletal-muscle damage and is reflected by peak [CK] measured 24 hours postmatch. Elevated [CK] persisted in comparison to prematch levels despite 120 hours of modified activity postmatch suggesting that a prolonged recovery phase of at least 5 days is required to achieve full recovery of muscle damage after match play.

The endocrine profile depicted in the present study identified a substantial acute sCort and small sTest response to Rugby League match play followed by a return to homeostasis within 48 hours. A minimum period of 48 hours is therefore recommended to enable hormonal homeostasis to be achieved postmatch. The evolution of real-time data acquisition with respect to player-movement characteristics in team sports will continue to facilitate a more robust analysis approach and enable sports scientists and coaches to further quantify the requirements of performance. By comparing endocrine and CK responses to performance, coaches are able to establish a more tangible identification of individual responses and adaptation to performance will be achieved in team sports such as Rugby League.

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