Williams, N., Russell, M., Cook, C.J. & Kilduff, L.P. (2018). The effect of lower limn occlusion on recovery following sprint exercise in academy rugby players. *Journal of Science and Medicine in Sport.* (Published online ahead of print 3 March 2018).

Title: The effect of lower limb occlusion on recovery following sprint exercise in academy rugby players

Authors: Williams, N.[a, b] Russell, M.[c], Cook, C.J.[d,e], Kilduff, L.P.[a, d]

[a] Applied Sports Technology Exercise and Medicine Research Centre (A-STEM), Swansea University, Swansea, United Kingdom

[b] Sport Wales, Welsh Institute of Sport, Sophia Gardens, Cardiff, United Kingdom

[c] School of Social and Health Sciences, Leeds Trinity University, Leeds, United Kingdom

[d] Welsh Institute for Performance Solutions

[e] University of Canberra Research Institute for Sport and Exercise, University of Canberra, Australia

Corresponding Author: Liam P.Kilduff

L.Kilduff@swansea.ac.uk

Article type: Original investigation

Abstract word count: 249

Manuscript word count: 2936

Figur

Abstract

Objectives: The effects of vascular occlusion on recovery of physiological and neuromuscular markers over 24h, and hormonal reactivity to subsequent exercise were investigated.

Design: Counterbalanced, randomised, crossover

Methods: Academy rugby players (*n*=24) completed six 50-m sprints (five-min inter-set recovery) before occlusion cuff application (thighs) and intermittent inflation to 171-266 mmHg (Recovery) or 15 mmHg (Con) for 12-min (two sets, three-min repetitions, three-min non-occluded reperfusion). Countermovement jumps, blood (lactate, creatine kinase), saliva (testosterone, cortisol), and perceptual (soreness, recovery) responses were measured before (baseline) and after (post, +2h, +24h) sprinting. Saliva was sampled after a 30-min resistance exercise session performed 24h after sprinting.

Results: Although sprinting (total: 40.0 ± 2.8 s, p=0.238; average: 6.7 ± 0.5 s, p=0.674) influenced creatine kinase ($p<0.001$, +457.1 ± 327.3 $\mu \cdot L^{-1}$, at 24h), lactate ($p<0.001$, 6.8 ± 2.3 $mmol \cdot L^{-1}$, post), testosterone ($p<0.001$, -55.9 ± 63.2 $pg \cdot ml^{-1}$, at 2h) and cortisol ($p<0.001$, -0.3 ± 0.3 $\mu g \cdot dl^{-1}$, at 2h) concentrations, countermovement jump power output ($p<0.001$, -409.6 ± 310.1 W; -5.4 ± 3.4 cm, post), perceived recovery ($p<0.001$, -3.0 ± 2.3, post), and muscle soreness ($p<0.001$; 1.5 ± 1.1, at 24h), vascular occlusion had no effect (all $p>0.05$) on recovery. In response to subsequent exercise performed 24h after vascular occlusion, testosterone increased pre-to-post-exercise (Recovery: p=0.031, 21.6 ± 44.9 $pg \cdot ml^{-1}$; Con: p=0.178, 10.6 ± 36.6 $pg \cdot ml^{-1}$) however Δtestosterone was not significantly different (p=0.109) between conditions.

Conclusions: Vascular occlusion had no effect on physiological or neuromuscular markers 2h or 24h after sprinting or in response to a physical stress test.

Keywords: Occlusion, sprint, hormonal reactivity

Introduction

Physical and metabolic disturbances result from team sport match-play[1]. Accordingly, various measures are applied to indicate the presence of exercise-induced muscle damage (EIMD) and the efficacy of recovery interventions[2]. Elevated Creatine Kinase (CK) concentrations[3], disruption in the hormonal milieu (i.e., testosterone; T, cortisol; C)[4], and impairments in neuromuscular function (NMF) occur post-match[5]; with perturbations occurring for at least 48h[3,5]. The use of hormonal responses, such as changes in T and C indicate anabolic and catabolic balance[6] and thus readiness-to-train or competition-preparedness, is an emerging concept in the fatigue-recovery paradigm[7].

As short-term post-match fatigue impairs subsequent performance, recovery strategies (e.g. cold-water immersion and active recovery) are an integral component of weekly training practices that have been extensively investigated (for review see[8]). More recently, vascular occlusion, the use of blood pressure cuffs applied on specific limbs to restrict blood-flow, has been suggested as a recovery strategy[9,10]. While exact mechanisms are unclear, vascular occlusion is purported to elevate adenosine concentrations and activation of adenosine tri-phosphate (ATP) sensitive potassium channels (K_{ATP}), increasing blood flow[11] and benefiting oxygen and nutrient delivery via vasculature dilation; a response likely exaggerated during reperfusion, possibly improving substrate re-synthesis[11]. Alternatively, attenuated inflammatory responses[10] and reduction of muscle oedema and intramuscular pressure decrease nocioreceptor stimulation, potentially reducing muscle soreness[12].

Unfortunately, the evidence for vascular occlusion as an effective post-exercise recovery modality is currently inconsistent. Two investigations identified improved recovery[10,11] whereas others[13,14,15] disagree. Methodological differences exist when implementing vascular occlusion, for example standardised cuff pressures have been implemented despite

recommendations regarding individualised application relative to thigh girth and resting blood measurements[16]. Similarly, inconsistent timings of recovery assessments (i.e., 1-72h) post-occlusion exist between studies. Previous research has also required players to remain rested for the duration of post-exercise recovery; however, this has limited application to applied practice where football or rugby players are frequently required to play multiple games within a week (i.e., <72h separating games), and train when complete physical recovery may not be achieved[17]. Accordingly, identification of the physiological response to a subsequent physical stressor may denote if players are adequately recovered to return to training. Notably, T is a stress biomarker; consequently, the monitoring of T in response to a physical stress test could provide information on readiness to train/compete[18].

The primary aim of the study was therefore to investigate the effects of post-exercise vascular occlusion (using individualised cuff pressures) on recovery (2h and 24h) of physiological and performance markers following maximal sprint exercise whilst also considering the hormonal reactivity to a subsequent exercise challenge performed at 24h. It was hypothesised that vascular occlusion implemented post-exercise would facilitate the recovery of biochemical, neuromuscular and hormonal markers measured after 24h.

Method

Following institutional ethical approval and informed written consent, 24 male Academy rugby union players (age: 21.8 ± 3.0 y, mass: 96.9 ± 10.1 kg, stature: 1.85 ± 0.09 m) participated in the study during pre-season (1–2 sessions per day 4–5 days a week; strength, power, speed training). All participants were informed of the experimental procedures, the purpose of the study, and possible risks.

Participants attended the testing venue four times. Two main trials (Vascular occlusion: Recovery; Control: Con), seven days apart, were completed on an indoor 3G surface

100 (temperature: 20°C; humidity: 41%) in a randomised, counter-balanced, crossover design.
101 Measurement timings were consistent between trials to limit circadian variation, and
102 participants refrained from alcohol and intense physical exercise in the 24h preceding trials.
103
104 On arrival for main trials, participants rested for 10-min before recording blood pressure
105 (Omron Healthcare, Europe; systolic >140 mmHg and diastolic >90 mmHg precluded further
106 study involvement). Thigh girth, physiological (capillary blood and saliva) and perceptual
107 (soreness and recovery) assessments followed. After a 10-min standardised warm-up,
108 participants performed two maximal countermovement jumps (CMJ) separated by 90 s
109 (portable force platform: Type 92866AA, Kistler, Germany) to assess NMF[5,19]. A further 10-
110 min warm-up followed (20 m dynamic exercises and accelerations, two-50 m sprints at 80%
111 and 100% effort) with five-min of enforced rest before six-50 m (each separated by five-min
112 rest) timed sprints (Brower Timing System, Salt Lake City, Utah, USA) were performed to
113 induce muscle damage[20,21] (Figure 1). As per pre-exercise instructions, maximal effort was
114 required across all six sprints, but no encouragement was provided during exercise.
115 Participants and coaches were blinded from sprint timings and feedback was not provided
116 until all trials were completed. Average, and cumulative sprint times were recorded for the
117 six sprints.
118
119 Immediately post-exercise, baseline measures were repeated before occlusion cuffs were
120 applied to the proximal point of the thighs while participants lay supine. The cuff (11 cm;
121 Sports Rehab Tourniquet, Sportsrehab) was manually inflated to 15 mmHg (Con), reflecting
122 previous research[9], or to 60% of individually calculated pressures (171-266 mmHg;
123 Recovery), determined from thigh girth and blood pressure measurements[16]. Cuffs were
124 applied for a total of 12-min (two cycles of three-min occlusion, three-min reperfusion)[9] as
125 reports suggest that three-min cycles of occlusion fulfil the duration threshold and a total
126 ischemic stimulus of at least four-min is required to elicit a protective effect in human
127 myocardium, irrespective of the number of ischemic cycles[22]. After 2h and 24h baseline

measures were repeated; timings which are consistent with previous research[20,21] and represent the duration between competition and return to training.

To assess hormonal reactivity to a subsequent exercise stimulus performed after 24h, saliva was collected five-min before and immediately after a 30-min physical stress test (three sets of power cleans and back squats, four sets of bench press and bench pull at relative loads of 60-85% 1RM; one and three-min rest between sets and exercises, respectively). Participants were accustomed to the resistance exercise and testing procedures employed and these protocols were sufficient to elicit a stress response[18]. Session supervisors provided technical support only and were not aware of the condition that players were in. Feedback was not provided regarding within-session performance.

***** INSERT FIGURE 1 NEAR HERE *****

Saliva collection required passive drooling (~2 ml) into a sterile vial (SalivaBio, Salimetrics LLC, USA) after refraining from brushing of teeth, drinking hot fluids, or eating hard foods 2h beforehand. All samples were stored (-15°C) immediately after collection and transferred to -80°C within 4h of collection. Post thawing, centrifugation (Micro Centaur, MSE, London, United Kingdom; five-min at 3000 revolutions·min^{-1}) preceded duplicate analysis of T and C concentrations due to known reliable reflections of gonadal function[23] using indirect enzyme-linked immunosorbent assay (ELISA) kits (Salimetrics Europe Ltd., Suffolk, U.K.). The lowest detection limits for T and C were 6.1 pg·ml^{-1} and 0.012 µg·dl^{-1} respectively, and inter-assay CV values were <10% in both cases.

Participants provided a 20 µL fingertip capillary blood sample (analysed retrospectively for lactate concentrations; Biosen C-Line Clinic, EKF Diagnostic GmgH, Barleben, Germany). Additionally, 120 µL sample was collected, immediately centrifuged (3000 revolutions·min^{-1}

for 10-min; Labofuge 400R, Kendro Laboratories, Germany) for the extraction of plasma, and stored (−80°C) until later analysis. Plasma samples thawed before 6 µL was used for CK analysis (automated analyser; ABX Pentra 400, Horiba ABX, Montpellier, France). Sample testing was carried out in duplicate, intra sample CV values were <2.0%.

Perceived lower body muscle soreness was assessed using a 7-point Likert scale ranging from zero (complete absence of soreness) to six (severe pain limiting movement) which is reliable and valid[24]. Perception of recovery status was assessed using a 11-point likert scale[25] from zero (very poorly recovered/extremely tired) to 10 (very well recovered) which reflects changes in total sprint time relative to prior exercise[21]. Participants were familiar with the scales and were asked to base scores on perceived soreness during normal movement and were alone when recording scores to reduce influences of peers.

Assessment of CMJ was completed on a portable force platform (Kistler instrument Ltd., Farnborough, UK) sampling at 1000 Hz. Peak power output (PPO) of the lower body was calculated as previously described[19]. The vertical component of the ground reaction force and participants' body weight were used to determine instantaneous velocity and displacement of the centre of gravity[19]. Instantaneous power output was determined using Equation 1 and the highest value produced was deemed PPO. Jump height (JH) was defined as the difference in vertical displacement of the center of gravity between take-off (toes leave the force plate) and maximum displacement[19].

Equation 1:

Power (W) = Vertical ground reaction force (N) x vertical velocity of centre of gravity ($m.s.^{-1}$)

Statistical analysis

All data is presented as mean ± standard deviation (SD). Following confirmation of parametric assumptions, multivariate analysis of variance (MANOVA) with Bonferroni

adjustment assessed between-trial differences for variables with multiple time points per trial (i.e. T, C, T:C ratio, perception muscle soreness and recovery, CK, blood lactate, PPO and JH). A one-way ANOVA was performed to assess between-trial differences in response to the physical stress test (T, C, T:C ratio and perception muscle soreness). Paired samples t-tests were performed for between-trial comparisons of data expressed over a single time point within a trial (i.e. mean and total sprint times, T and C pre-and post-stress test). Statistical analyses were carried out using SPSS (SPSS Chicago, IL) with significance being accepted at $p \leq 0.05$.

Results

There were no significant differences between conditions for total (Recovery: 39.86 ± 2.87 s; Con: 40.26 ± 2.77 s, p=0.238) or average (Recovery: 6.69 ± 0.47 s; Con: 6.71 ± 0.46 s, p=0.674) sprint times. The Δlactate concentrations from pre-to-post sprinting showed no significant difference between conditions (Recovery: 6.88 ± 2.53 $mmol \cdot l^{-1}$, 85 ± 6%; Con: 6.76 ± 2.04 $mmol \cdot l^{-1}$, 86 ± 5%, p=0.807, Figure 2).

There was a significant time effect for CK ($F_{(1,48)}$=72.928, p<0.001, Figure 2) with increases at 24h compared to pre-sprints in both Recovery (408.19 ± 291.45 $\mu \cdot L^{-1}$, 55 ± 33%, p<0.001) and Con (506.02 ± 359.14 $\mu \cdot L^{-1}$, 56 ± 34% p<0.001). However, there was no significant interaction effects between condition and time ($F_{(1, 48)}$=1.157, p=0.293).

***** INSERT FIGURE 2 NEAR HERE *****

Muscle soreness ($F_{(2, 95)}$=7.714, p<0.001) and perception of recovery ($F_{(2, 88)}$=70.931, p<0.001) were affected by sprinting, with the greatest change in muscle soreness occurring 24h post exercise (Recovery: 1.5 ± 1.0; Con: 1.6 ± 1.1). There was no significant interaction effects

between time and condition for muscle soreness ($F_{(2, 95)}$=0.009, p=0.993) or perception of recovery ($F_{(2, 88)}$=0.158, p=0.924).

Sprint exercise affected PPO ($F_{(2, 96)}$=42.141, p<0.001) and JH ($F_{(2, 82)}$=58.353, p<0.001) with PPO (Recovery: 417.74 ± 293.09 W, 8 ± 4%, p<0.001; Con: 401.46 ± 332.69 W, 7 ± 5%, p < 0.001) and JH (Recovery: 5.49 ± 3.24 cm, 13 ± 7%, p<0.001; Con: 5.38 ± 3.66 cm, 13 ± 8%, p<0.001) decreasing post-sprints (Figure 3). No further timing effects and no effect of Recovery on PPO ($F_{(2, 96)}$=0.304, p=0.757) or JH ($F_{(2, 82)}$=0.304, p=0.436) occurred.

Sprinting increased T ($F_{(2, 100)}$=20.127, p<0.001, Figure 2), T:C ratio ($F_{(2, 95)}$=19.200, p<0.001) and decreased C ($F_{(2, 89)}$=32.651, p<0.001, Figure 2). However, condition did not affect the recovery of T ($F_{(2, 100)}$=2.159, p=0.114), C ($F_{(2, 89)}$=0.640, p=0.531) or T:C ratio ($F_{(2, 95)}$=0.299, p=0.759).

Testosterone significantly increased in response to the physical stress test in the Recovery (+21.58 ± 44.90 pg·ml^{-1}, 7 ± 17%, p=0.031) but not the Con (+10.62 ± 36.57 pg·ml^{-1}, 4 ± 13%, p=0.178) with no differences in baseline values between conditions (p=0.232); however, ΔT was similar between conditions (p=0.109). Cortisol declined over time ($F_{(1,46)}$=7.806, p<0.001), pre-to post physical stress test (Recovery: -0.14 ± 0.23 μg·dl^{-1}, 38 ± 72%, p=0.007; Con: -0.17 ± 0.27 μg·dl^{-1}, 50 ± 94%, p=0.006), with similar results for T:C ratio ($F_{(1, 46)}$=29.836, p<0.001). Recovery had no impact on hormonal response as no differences were observed between conditions (T; p=0.226, C; p=0.679, T:C; p=0.421).

***** INSERT FIGURE 3 NEAR HERE *****

Discussion

This study aimed to investigate the effects of individualised vascular occlusion on recovery (2h and 24h) of physiological and neuromuscular indices following sprint exercise while also considering hormonal reactivity to subsequent training. Vascular occlusion did not influence the physiological or neuromuscular markers measured 2h or 24h after sprint exercise in Academy rugby players. Perception of muscle soreness was not different between conditions, sprinting, increased muscle soreness 24h post-exercise. As similar between-condition responses to a physical stress test occurred at 24h, vascular occlusion did not facilitate recovery following 2h or 24h of rest, nor change the hormonal response to a subsequent physical stress test. Likewise, vascular occlusion did not detrimentally affect any measures assessed, recovery rate was not negatively influenced in comparison to Con, thus alleviating concerns about using this strategy, acutely, within season.

Total ($p=0.238$) and average ($p=0.674$) sprint times were consistent between conditions with similar physiological responses being observed. Johnston et al.[21] highlighted CK values increased by 570 $\mu \cdot L^{-1}$ (current results +506.02 $\mu \cdot L^{-1}$). Compared to match responses, increases of 586.6 $\mu \cdot L^{-1}$ ([4]; 24h post-soccer match) and 431 $\mu \cdot L^{-1}$ ([26]; 16h post-rugby match) have been reported. However, time and distance of maximal sprint completed in a match cannot be controlled and varies depending on position, therefore comparison against match outcome is difficult as many factors may influence performance on match day. Nevertheless, previous reports suggest that six 50m sprints reflect normal training sessions[27].

The current study individualised cuff pressure (171-226 mmHg) as a standard pressure applied to different individuals may non-uniformly influence the pressure exerted on the vasculature and thus impact the degree of blood flow restriction. Loenneke et al.[28] suggested that pressures aiming to restrict blood flow of the lower body should be determined by limb

circumference; findings supported by observations that thigh circumference is the biggest predictor of arterial occlusion in the lower body (β=0.570)[16] with brachial systolic blood pressure being an additional significant predictor (β=0.231)[16]. That said, in contradiction to the findings of Beaven et al.[9] who identified improved peak power recovery 24h after occlusion, we observed no effects on recovery. Although comparable vascular occlusion timings were used, cuff pressures differed between the studies (i.e., a standardised 220 mmHg versus individualised application). Alternative methodological factors may be influencing the resulting outcome of vascular occlusion as a recovery modality; cuff pressure was determined from a regression equation[16] based on non-elite participants, although this protocol considers thigh girth, tissue type (muscle or fat) may impact the level of blood flow restriction, a variable which will vary between elite-trained and untrained participants, training status may also impact effectiveness of vascular occlusion used for recovery.

An alternative methodological discrepancy existing between previous research is the training status of participants. Research completed by Beaven et al.[9] investigated healthy, active non-sport participating males and found vascular occlusion improved recovery. However, Northey et al. [14] and the current study investigated the use of vascular occlusion in well-trained individuals, identifying no effect on recovery. This potentially suggests that training status may mediate the efficacy of vascular occlusion as a recovery strategy. However, even with a group of active or well-trained participants, high inter-individual variability in EIMD marker responses has been found even when participants perform the same exercise[29]. Research suggests that the large inter-individual variability including non-modifiable factors (ethnicity, age, and gender) could be responsible for most of the equivocal findings and uncertainties regarding EIMD etiology[29]; another variable potentially explaining variation between the current study and previous work. Therefore, it may be important to examine the use of vascular occlusion on an individual basis as it may offer a practical recovery option for some athletes/players and at some levels of performance.

A novel aspect of this research was the response of a physical stress test as an indicator of recovery status and readiness-to-train following sprint exercise at 24h. Within the literature, previous recovery studies have assessed response to a recovery strategy when the athlete is rested, however players are frequently required to return to training 24h after a match. Therefore, hormonal response to a physical stress test may indicate if players are recovered and able to return to training. In the current study, T increased pre-to post stress test in both conditions (Recovery: 21.58 ± 44.90 $pg \cdot ml^{-1}$, 7 ± 17%, p=0.031; Con: 10.62 ± 36.57 $pg \cdot ml^{-1}$, 4 ± 13 %, p=0.178), but ΔT was not significantly different between conditions, suggesting Recovery had no impact on hormonal response. Furthermore, C showed a significant decline from pre-to post-stress test in both the Recovery (0.14 ± 0.23 $\mu g \cdot dl^{-1}$, 38 ± 72%, p=0.007) and Con (0.17 ± 0.27 $\mu g \cdot dl^{-1}$, 50 ± 94%, p=0.006). Therefore, consistent with rested results, there was no difference between conditions regarding rate of recovery following a physical stress test.

Limitations of the research should be acknowledged; the placebo effect is not accounted for, which may be important to consider. Within Con, cuffs were still applied and therefore is difficult to blind participants from the conditions due to the obvious difference in cuff pressure. Therefore, a further condition would be optimal to identify the impact of vascular occlusion, determining whether there is a placebo or physiological effect on performance when compared against a control in which cuffs are absent as completed by Marocolo et al. [30]. Similarly, it is difficult to blind testers to conditions due to variation in cuff pressure, however, all testers and coaches were informed that no verbal encouragement was to be given during exercise (sprints, jumps, stress test).

Conclusions

The data presented in this investigation highlight application of two cycles of vascular occlusion administered intermittently after sprinting did not influence, either positively or

negatively, physiological or neuromuscular markers of recovery assessed after 2h or 24h of rest or after a subsequent physical stress test.

Practical Implications

- Individualised vascular occlusion applied post-exercise didn't influence recovery or readiness-to-train after 24h
- An absence of negative effects for physiological and performance markers alleviates concerns about the use of vascular occlusion within season
- Methodological variations (such as exercise protocol, cuff pressure, duration of occlusion and training status) may be modulating the efficacy of vascular occlusion as a recovery strategy

Acknowledgements

None to declare. There was no financial support for this study. This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

345 **References**

346 1. Bangsbo, J., Mohr, M. & Krustrup, P. Physical and metabolic demands of training and
347 match-play in the elite football player. *J Sports Sci*, 2006, 24(7), pp.665–674.
348 2. Ferguson, R.A., Dodd, M.J. & Paley, V.R. Neuromuscular electrical stimulation via the
349 peroneal nerve is superior to graduated compression socks in reducing perceived muscle
350 soreness following intense intermittent endurance exercise. *Eur J Appl Physiol*, 2014,
351 pp.2223–2232.
352 3. Russell, M. et al. Relationships between match activities and peak power output and
353 Creatine Kinase responses to professional reserve team soccer match-play. *Hum Mov*
354 *Sci*, 2016, 45, pp.96–101.
355 4. Shearer, D.A. et al. Measuring Recovery in Elite Rugby Players: The Brief Assessment
356 of Mood, Endocrine Changes, and Power. *Res Q Exerc Sport*, 2015, 86(4), pp.379–86.
357 5. West, D.J. et al. Neuromuscular function, hormonal, and mood responses to a
358 professional rugby union match. *J Strength Cond Res*, 2014, 28(1), pp.194–200.
359 6. Fry, R.W. et al., 1991. Monitoring exercise stress by changes in metabolic and hormonal
360 responses over a 24-h period. *Eur J Appl Physiol Occup Physiol*, 63(3–4), pp.228–234.
361 7. Crewther, B.T. et al. Can salivary testosterone and cortisol reactivity to a mid-week
362 stress test discriminate a match outcome during international rugby union competition? *J*
363 *Sci Med Sport,* 2017.
364 8. Nédélec, M. et al. Recovery in soccer: Part II-recovery strategies. *Sports Med*, 2013,
365 43(1), pp.9–22.
366 9. Beaven, C.M. et al. Intermittent lower-limb occlusion enhances recovery after strenuous
367 exercise. *Appl Physiol Nutr Metab*, 2012, 37(6), pp.1132–9.
368 10. Page, W., Swan, R. & Patterson, S.D., 2017. The effect of intermittent lower limb
369 occlusion on recovery following exercise-induced muscle damage: A randomized
370 controlled trial. *J Sci Med Sport*, 2017.
371 11. Liu, G.S. et al., 1994. Evidence that the adenosine A3 receptor may mediate the
372 protection afforded by preconditioning in the isolated rabbit heart. *Cardio Res*, 28(7),

pp.1057–1061.

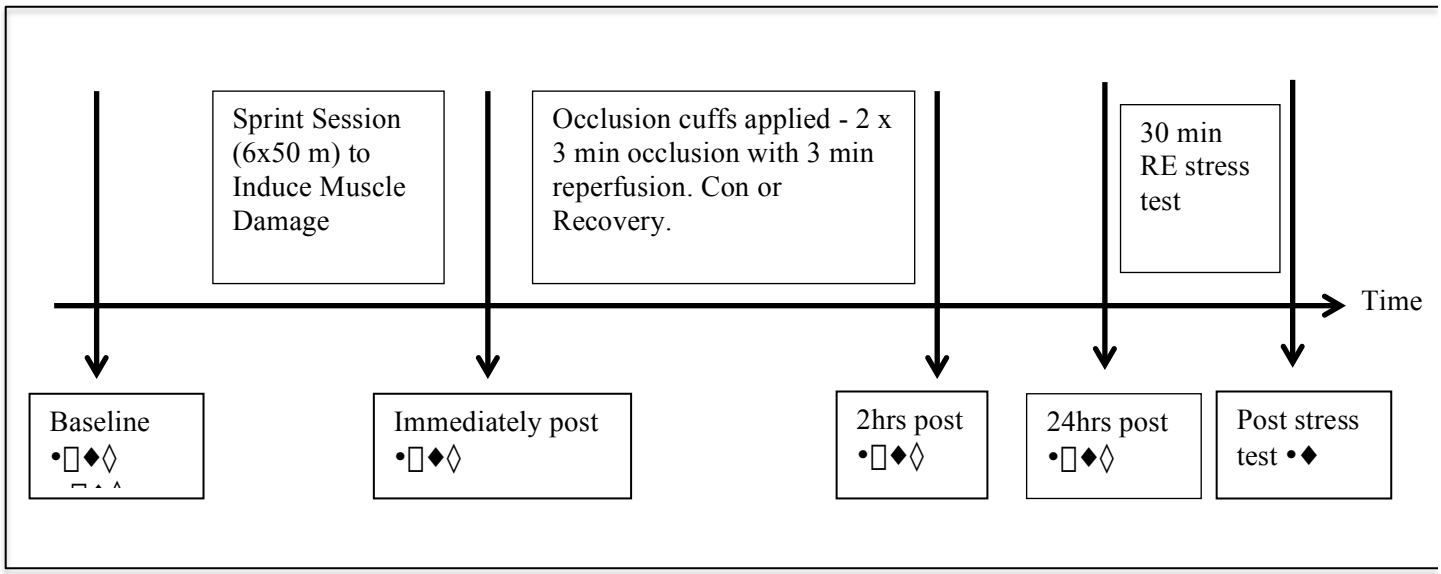
12. Howatson, G. & van Someren, K.A., 2008. The prevention and treatment of exercise-induced muscle damage. *Sports Med (Auckland, N.Z.)*, 38(6), pp.483–503.
13. Garcia, C.A. et al. Ischemic Preconditioning and Acute Recovery of Performance in Rugby Union Players. *Sports Med Int Open,* 2017, 1, pp.107–112.
14. Northey, M. et al. Vascular Occlusion and Sequential Compression for Recovery Post Resistance Exercise. *J Strength Cond Res*, 2016, 30(2), pp.533–539.
15. Borne, R., Hausswirth, C. & Bieuzen, F. Relationship Between Blood Flow and Performance Recovery: A Randomized, Placebo-Controlled Study. *I J Sports Physiol Perform*, 2016, (May), pp.1–44.
16. Loenneke, J.P. et al. Blood flow restriction in the upper and lower limbs is predicted by limb circumference and systolic blood pressure. *Eur J Appl Physiol*, 2015, 115(2), pp.397–405.
17. Nédélec, M. et al., 2012. Recovery in soccer: Part 1. *Sports Med*, 43(1), pp.9–22.
18. Crewther, B.T. & Cook, C.J. Effects of different post-match recovery interventions on subsequent athlete hormonal state and game performance. *Physiol Behav*, 106(4), 2012, pp.471–475.
19. West, D.J. et al. Relationships between force-time characteristics of the isometric midthigh pull and dynamic performance in professional rugby league players. *J Strength Cond Res*, 2011, 2(12), pp.3070–3075.
20. Taylor, T. et al. The impact of neuromuscular electrical stimulation on recovery after intensive, muscle damaging, maximal speed training in professional team sports players. *J Sci Med Sport*, 2014, 18(3), pp.328–332.
21. Johnston, M. et al., 2015. Neuromuscular, physiological and endocrine responses to a maximal speed training session in elite games players. *Eur J Sport Sci*, pp.1–7.
22. Ghosh, S., Standen, N. B. Galiñanes, M., 2000. Preconditioning the human myocardium by simulated ischemia: Studies on the early and delayed protection. *Cardio Res,* 45(2), 339-350.

23. Dabbs, J.M. Salivary testerosterone measurements: reliability across hours, days, and weeks. *Physiol Behav,* 1990, pp.83-86.
24. Impellizzeri, F.M. & Maffiuletti, N. A. Convergent evidence for construct validity of a 7-point likert scale of lower limb muscle soreness. *Clinical J Sports Med*, 2007, 17(6), pp.494–496.
25. Laurent, C.M. et al. A practical approach to monitoring recovery: development of a perceived recovery status scale. *J Strength Cond Res*, 2011, 25(3), pp.620–628.
26. Jones, M.R. et al., 2014. Match play performance characteristics that predict post-match creatine kinase responses in professional rugby union players. *BMC Sports Sci Med Rehab*, 6(1), p.38.
27. Francis, C. (2008). The structure of training for speed. Retrieved from www.Charliefrancis.com
28. Loenneke, J.P. et al. Effects of cuff width on arterial occlusion: implications for blood flow restricted exercise. *Eur J Appl Physiol*, 2012, 112(8), pp.2903–2912.
29. Damas, F. et al. Susceptibility to Exercise-Induced Muscle Damage : a Cluster Analysis with a Large Sample. *Int J Sports Med,* 2016, 37(8), 633-640.
30. Marocolo, M. et al., 2015. Are the Beneficial Effects of Ischemic Preconditioning on Performance Partly a Placebo Effect? *Int J Sports Med*, 36(10), pp.822–825.

Figure 1: Timeline of data collection. Con; control trial, Recovery; occlusion trial, RE; resistance exercise. Measurements: salivary testosterone, salivary cortisol (•), blood sampling for blood lactate and Creatine Kinase (□), perception muscle soreness questionnaires (♦), countermovement jump (◊)

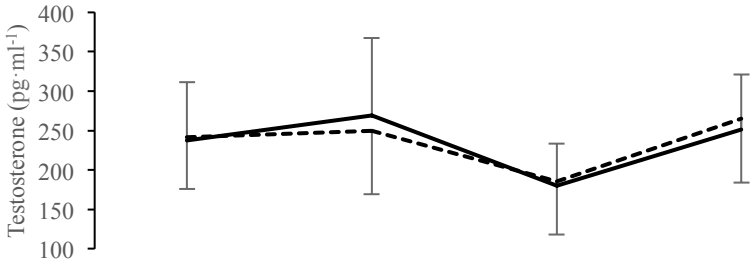
Figure 2: Physiological responses (a) Testosterone (b) cortisol (c) Creatine Kinase (d) Blood lactate collected pre, post, 2 h and 24 h post sprint protocol (* $p<0.05$)

Figure 3: (a) Peak power output and (b) jump height, determined from countermovement jump collected pre, post, 2 h and 24 h post sprint


Sprint Session (6x50 m) to Induce Muscle Damage
Occlusion cuffs applied - 2 x 3 min occlusion with 3 min reperfusion. Con or Recovery.
30 min RE stress test
Time
Baseline
Immediately post
2hrs post
24hrs post
Post stress test

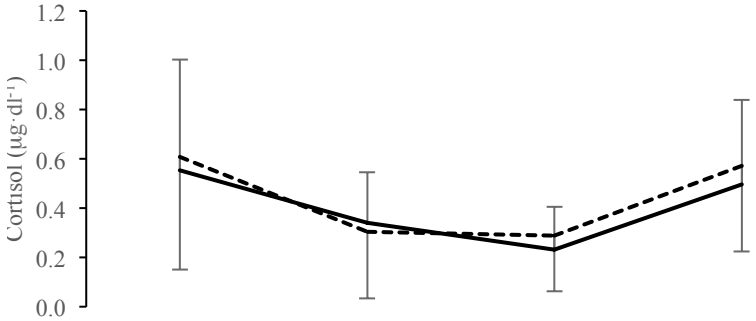
473

a



474
475
476
477
478
479
480
481
482

483

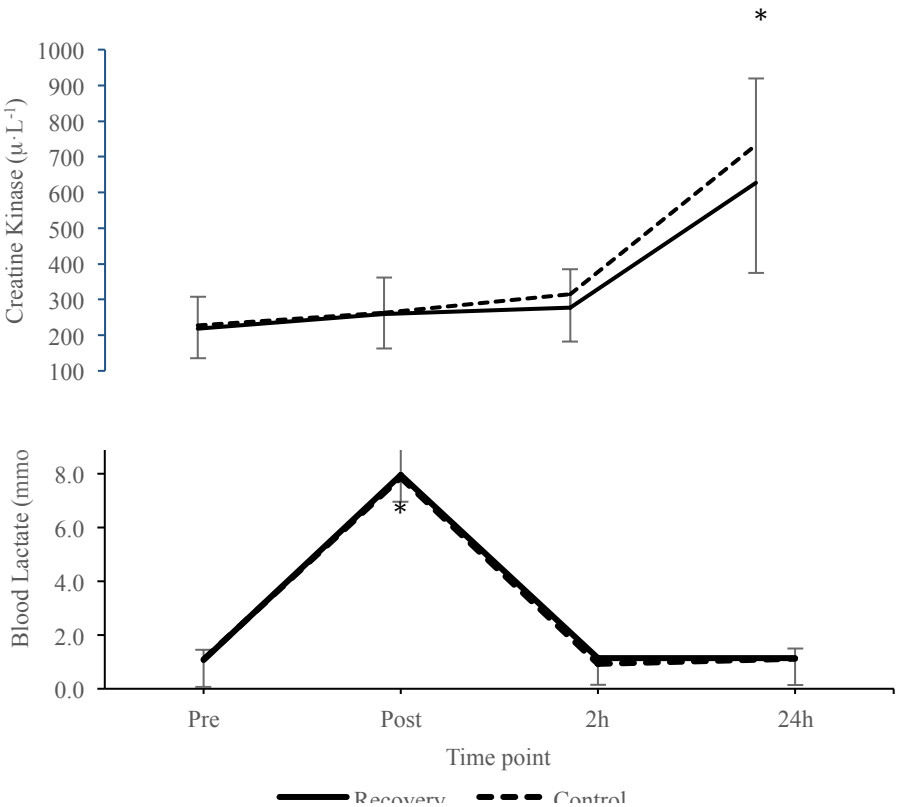


494
495
496
497
498
499
500
501
502
503
504
505
506
507
508

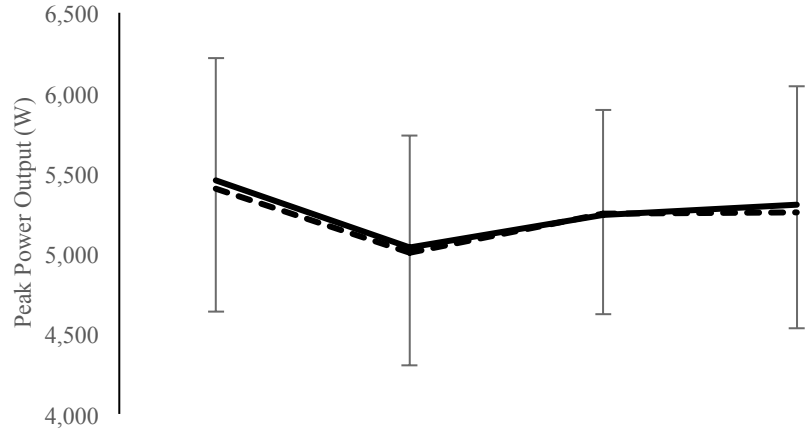
c

d

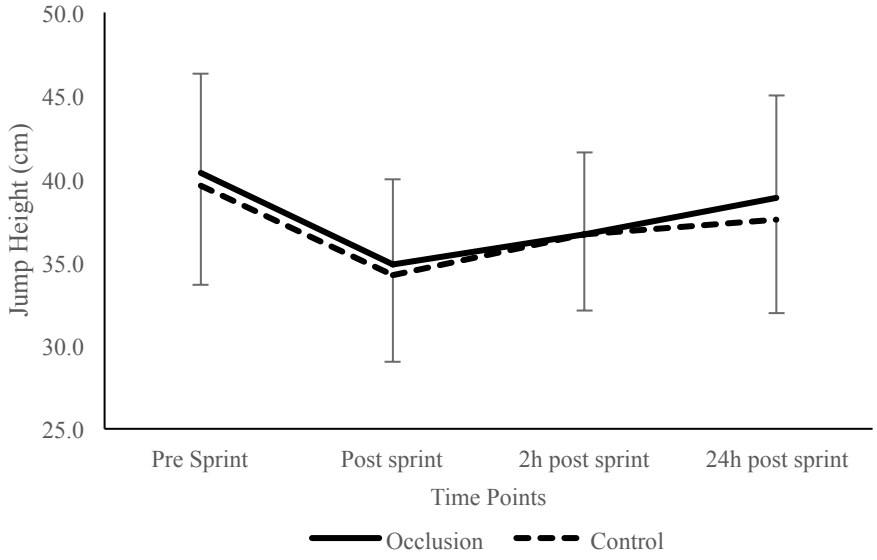
a



509



510
511
512 b



513
514
515
516
517
518
519
520