Section: Original Investigation

Article Title: The Effects of Chronic Cold Water Immersion in Elite Rugby Players

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Journal: *International Journal of Sports Physiology and Performance*

Acceptance Date: June 14, 2018

DOI: https://doi.org/10.1123/ijspp.2018-0313

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International Journal of Sports Physiology and Performance

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Submission type: Original investigation

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Running head: Cold water immersion in elite rugby athletes

Abstract word count: 229

Text-only word count: 3564

Number of figures and tables: tables = 5

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Abstract

Purpose: While the acute effects of cold water immersion (CWI) have been widely investigated, research analysing the effects of CWI over a chronic period in highly-trained athletes is scarce. The aim of this study was to investigate the effects of CWI during an intense three week pre-season phase in elite rugby athletes. **Methods:** Twenty-three elite male rugby union athletes were randomized to either CWI (10 min at 10 ºC, n = 10) or a passive recovery control (CON, n = 12) during three-weeks of high volume training. Athletes were exposed to either CWI or CON, after each training day (12 days in total). Running loads, conditioning and gym sessions were kept the same between groups. Measures of countermovement jump (CMJ), perceived muscle soreness and wellness were obtained twice a week, and saliva samples for determining cortisol and interleukin-6 (IL-6) were collected once per week. **Results:** Although no significant differences were observed between CWI and CON for any measure, CWI resulted in lower fatigue markers throughout the study, as demonstrated by the *moderate* effects on muscle soreness (d = 0.58 to 0.91) and IL-6 (d = -0.83), and the *small* effects (d = 0.23 to 0.38) on CMJ in comparison to CON. **Conclusions:** The results from this study demonstrate that CWI may provide some beneficial effect by reducing fatigue and soreness during an intense three week training phase in elite rugby athletes.

Keywords: Recovery, fatigue, chronic, acute, adaptation

INTRODUCTION

At the elite level, rugby training often occurs two or more times daily over two or more consecutive days during a week.[1,2] An imbalance between training stress and recovery can lead to an excessive level of accumulated fatigue over the training week[1] and undesirable chronic fatigue over a training phase.[3] Increased fatigue over time can lead to the athlete being unable to train at a required intensity or being unable to perform the desired training load.[4] In order to reduce the harmful effect of fatigue and allow athletes to recover faster, athletes regularly implement different recovery modalities in their routines.[1,2,5] Previous literature has identified cold-water modalities as one of the most common recovery strategies implemented by elite rugby athletes.[1,2] The exposure to cold water decreases skin, core and muscle temperature,[6] leading to vasoconstriction, and consequently, it may decrease swelling and acute inflammation from muscle damage.[7] Furthermore, the use of cold water immersion (CWI) contributes to a reduction in nerve conduction properties and to a decrease in muscle spasm and pain.[7] CWI in an acute rugby setting (<48 h post-exercise) has been effective in increasing neuromuscular function[8,9] , enhancing perceived recovery,[8] and decreasing both delayed onset muscle soreness (DOMS)[9] and creatine kinase levels.[9]

Given the beneficial effects of CWI in enhancing recovery in rugby, this modality has become commonplace following both matches and training sessions.[2,10] However, some researchers argue that the use of CWI post-exercise in a chronic setting may blunt adaptations by reducing muscle protein synthesis and therefore limiting muscle mass maintenance/growth.[11] Mechanisms involved in the hypertrophy of the muscle cell are thought to be partially associated with exercise-induced muscle damage (EIMD) and the consequent increases in the activity of satellite cells and inflammatory cells as well as the increase in the cell swelling.[11] These responses

to EIMD are proposed to mediate various anabolic signalling pathways that ultimately increase the rates of protein synthesis.[11] Roberts et al.[11] observed an acute decrease in the activity of selected components of the mammalian target of rapamycin pathway and satellite cells after 10 minutes of CWI at ~10 ºC performed post-resistance training. In the same study, the authors observed that CWI attenuates muscle mass (but not type II fibre cross sectional area or myonuclear accretion) after 12 weeks of lower body resistance training composed of two sessions per week.[11]

Interestingly though, when the recovery time was shorter (i.e. 6 h) the same authors report that CWI enhances recovery of muscle function and allows athletes to complete more work during subsequent training sessions.[12] Furthermore, during an intensified training phase for elite cyclists, Halson et al.[5] found likely beneficial effects of CWI in the mean power of a four minute cycling test and the one second maximum power in a sprint test. In this study the elite cyclist trained on a daily basis, therefore, the time for recovery was shorter than the typical studies investigating the effects of CWI on performance.[5] Together, these findings demonstrate that when recovery time is limited, CWI may provide a beneficial acute effect on performance that will reflect the chronic adaptations to a training regime. Research on the effects of chronic exposure to CWI (i.e. during consecutive training weeks) on rugby players, is limited to a single study performed on age-group athletes, which found no differences in performance, DOMS and perceived recovery.[13] Based on the aforementioned findings, two theories have been proposed for the response to CWI: 1) These modalities allow athletes to perform subsequent training sessions with a greater quality and/or quantity (i.e. greater training load); or 2) These modalities may blunt selected muscular adaptations to training (i.e. decrease protein synthesis).[5]

To the best of our knowledge, research investigating the training responses in elite athletes when chronically exposed to CWI are limited to endurance athletes[5] with no published studies on

elite team-sport athletes. While research on chronic CWI in non-elite team sport athletes does exist[5], we believe these findings are unlikely to apply to elite team sport athletes as the training load and training density is not comparable between settings. Therefore, the aim of the current study was to investigate the effects of chronic exposure to CWI on physiological and perceptual markers of fatigue in an elite rugby population during an intense three-week pre-season training phase.

METHODS

Participants

Twenty-nine professional male rugby athletes volunteered to participate in the current study. Athletes were members of a team that made it to the semi-finals of the Super Rugby competition in the same year as data collection took place. The Super Rugby competition is the major competition in the southern hemisphere comprising of teams from Argentina, Australia, Japan, New Zealand and South Africa. The attendance of at least 90% of the planned number of training sessions, without missing two in a row, was a requirement for inclusion in the present study. Athletes were matched by positional group and were randomly divided in one of two groups: A CWI group and a control group (CON). From the initial sample size, six subjects were excluded due to injury or illness. The 23 remaining athletes were included in the data analysis (CWI: n=10; 6 forwards [60%] 4 backs [40%]; CON: n=13; 8 forwards [61.5%] 5 backs [38.5%]) (Table 1). Written informed consent was obtained from each participant, and ethical approval was obtained from the Human Research Ethics Committee of the Institution.

Procedures

This study occurred during three weeks of the pre-season period and each week consisted of four days of training as described in table 2. Immediately after each training day, the athletes in

the CWI group were exposed to the recovery intervention. Therefore, athletes in the CWI group were exposed to CWI four times in each week, totalling 12 CWI sessions over the duration of the study. The CWI protocol consisted of athletes being immersed for 10 minutes to a level of the iliac crest in an industrial tub filled with water at a fixed temperature of 10 ºC (Hayward® EnergyLine pro ENP3M-13A, Dandenong South, VIC, Australia). The duration of the immersion and water temperature used in the current study were based on that proposed in a recent literature review on the effects of different recovery modalities in rugby which included CWI strategies for rugby players.[2] The athletes in the CON group followed their normal post-training routine and remained at the training facilities until all athletes in the CWI group completed their CWI protocol. Questionnaires and countermovement tests (CMJ) were implemented on the mornings of days one and four for each of the three weeks. Saliva samples were collected prior to any food intake on Day Four each week. The order and time of data collection were maintained throughout the study, and individual wake times were monitored through questionnaires.

Training program

The training program consisted of four resistance training sessions (two for lower body and two for upper body) designed to increase maximal strength and power (i.e. 3-5 sets of 1-6RM for core exercises and 3-5 sets of 0-70% 1-RM for power exercises),[14] seven rugby field sessions, two speed sessions and three extra-conditioning sessions per week (Table 2). Given the schedule was similar during the three weeks of the study, the duration of the sessions and the training load is presented as the average of the three weeks in Table 2.

Perceptual measures

A wellness questionnaire and a lower-body soreness (LB soreness) questionnaire previously used in rugby athletes was implemented on the morning of Day one and Day Four of

each week during the period of the study.[15] The wellness questionnaire comprised of five questions to measure perceived fatigue, general muscle soreness, sleep quality, stress levels, and mood state. The lower-body soreness questionnaire was designed to detect muscle soreness at five specific lower-body sites.[15] Both questionnaires used a 1-5 Likert-type scale with 0.5-point increments where 1 represented a high score (e.g. *no soreness*) and 5 a low score (e.g. *very sore*).[15] A total measure of each questionnaire (i.e. wellness questionnaire and lower-body soreness questionnaire) was calculated from the average of the items.[15]

Neuromuscular performance

In order to monitor neuromuscular fatigue, peak force (N) was measured during a CMJ test performed each morning on Day one and Day four during the 3-week period (Table 2). Following a standardized warm-up composed of dynamic stretches and movements (e.g. one-leg standing knee flexion, bodyweight squats, bodyweight CMJ's), athletes performed three maximal CMJ's. Two force plates (PASCO PS 2142, Roseville, CA, USA) were used to measure peak force (PF) at a sample rate of 500 Hz. The force plates were connected to an analog-to-digital converter (SPARKlink), which was then connected to a PC and the Pasco Capstone v1.4.0 software (PASCO, Roseville, California, USA) through a USB port. Each trial started with the subjects standing on the top of the force plates with their knees fully extended and their hands on hips to eliminate the influence of arm swing. Subjects were then instructed to descend to a self-selected depth and to jump as high and quickly as possible.[16] The best trial, determined by peak force, was retained for later analysis. Data obtained with 18 elite rugby athletes demonstrate that PF obtained with the same setup and protocol has an acceptable level of test-retest reliability in a similar cohort (ICC: 0.89; CV: 4.56%).[17]

Saliva samples

Whole saliva samples were collected to monitor weekly changes in cortisol and IL-6. Players expectorated a sample via passive drool into a 50-mL polyethylene tube, which was stored at −20°C until assayed. Cortisol and IL-6 concentrations were determined in duplicate using commercially available enzyme-linked immunosorbent assay kits (Salimetrics, State College, PA) as per the manufacturer's instructions. Cortisol assay sensitivity was 3.5 $pmol \cdot L^{-1}$ with intra-assay and inter-assay CV <3%. IL-6 assay sensitivity was 0.07 $pg \cdot mL^{-1}$ with intra- and inter-assay CV <10%. Saliva samples for each participant were analyzed in the same assay to eliminate inter-assay variance.

Training load

Locomotor activity of each participant was monitored during all technical-tactical training sessions with a 15-Hz GPS unit (Viper Pod, STATSport, Belfast, UK) incorporated into the players' jersey on the upper thoracic spine between the scapulae. In order to decrease the between-unit variability, the same GPS unit was used by each participant for subsequent sessions. The GPS units were turned on before the warm-up and turned off after the completion of the training sessions. After each training session, the raw data files were analyzed and individual sessions' relative distance (m/min) and high metabolic load distance (HML; distance covered >5 m/s and/or distance accelerating and decelerating over 2 m/s^2) were obtained from the company's software (Viper PSA software, STATSports, Belfast, UK). The individual training RPE of the non-running conditioning sessions and the gym training sessions were obtained between 15 and 30 minutes after the completion of the session.[18] The training load was then calculated as the product of the individual session RPE (sRPE) and the duration of the session using the following formula: Training load = sRPE (1-10) × duration of the session (min).[18] A total measure of perceived

training load was calculated from the individual sum of the non-running conditioning sessions sRPE coupled with the gym training sessions sRPE.

Statistical analysis

All statistical analyses were performed using SPSS 25.0 (IBM Corp., Armonk, NY, USA). Independent samples T-tests or Mann-Whitney tests were conducted to verify the differences between groups (CWI and CON) for the baseline measures (i.e. the first samples collected during the experimental period) for CMJ, LB soreness, wellness, IL-6 and cortisol. These statistical tests were also conducted to determine the differences between groups for athlete characteristics i.e. age, body mass, height, sum of 8 skinfolds, squat 1-RM, bench press 1-RM, chin-ups 1-RM, speed over 10-m, Yoyo intermittent recovery test level 1, all measured the week prior to the beginning of the experimental period.

The differences from baseline measures in CMJ, LB soreness, wellness, cortisol and IL-6 were calculated. A repeated measures ANOVA was performed to determine the effect of different treatments (CWI or CON) over time (day/week) on all measured variables. Analysis of the studentized residuals was verified visually with histograms and also by the Shapiro-Wilk test of normality.

In addition, effect-size statistics were performed for differences between groups from baseline (Day one of Week one) for CMJ, IL-6, cortisol, LB soreness, and total wellness. For these measures, the standardised change in the mean from baseline for each day was determined and expressed as standardised (Cohen's *d*) effects.[19] Effect sizes were also determined to compare differences between groups for the training load markers in each week. Magnitudes of the standardised effects were interpreted using thresholds of 0.2, 0.6, 1.2 and 2.0 for *small, moderate,*

large, and very large, respectively.[20] An effect size <0.2 was considered *trivial.*[20] Where the 90% confidence limits overlapped *small* positive and negative values, the effect was deemed *unclear*.[21]

RESULTS

Subjects characteristics for age, bdy mass, height, sum of 8 skinfolds and performance markers (i.e. maximum strength, speed and aerobic power) are included in Table 1 as means ± SD. No significant differences ($p < 0.05$) were found between groups for athletes' characteristics or measures of training load. (Table 3).

Although results from ANOVA found no interaction for Time × Group for any of the measured variables (Table 4), the analysis of the effect sizes demonstrates a *small* effect in favour of CWI on CMJ performance and a *moderate* effect on LB soreness (Table 5).

Moreover, a *moderate* effect of CWI attenuating increases in IL-6 in comparison to CON was observed in the comparison from Week Three to baseline (Table 5). No differences between or within groups were observed for Cortisol over the duration of the study (Table 5). Further analysis of the effect sizes revealed that on Day Four of each week, the athletes in the CON group demonstrated *small* reductions in CMJ performance and wellness scores, and *moderate* increases in soreness, while the athletes in the CWI were able to maintain scores in comparison to baseline (Table 5).

DISCUSSION

The main finding from the current study is that the chronic exposure to cold water immersion over a pre-season phase in elite rugby athletes showed no detrimental effect to performance or recovery. In fact, there was a *moderate* effect in favour of CWI for attenuating increases in muscle soreness when compared to CON (Table 5). The beneficial effects of CWI were also extended to performance in the CMJ and IL-6 values, as demonstrated by the *small* and

moderate effects, respectively (Table 5). While the acute effects of CWI have been previously investigated in elite rugby athletes,[9] this study is the first to show the positive effects of chronic exposure to CWI in elite rugby athletes in terms of neuromuscular performance and immune function.[2]

Delayed onset of muscle soreness (DOMS) is a well-documented outcome occurring from exercise-induced muscle damage, with a recent meta-analysis demonstrating that CWI is beneficial for reducing DOMS after exercise strenuous enough to induce damage.[22] Particularly in rugby, previous research observed that CWI decreases muscle soreness and markers of muscle damage, such as creatine kinase clearance, measured 1, 18 and 42 h post-match when compared to active recovery.[9] Although the ANOVA revealed no significant treatment interaction, our findings support the beneficial effect of CWI reducing muscle soreness with *moderate* effects sizes observed between groups (Table 5) over a three-week pre-season training phase. While athletes in the CON reported higher scores of lower body soreness (i.e. *small* to *moderate* effect sizes) than athletes in CWI (i.e. *small* negative effect sizes). It is important to mention that statistical significance ($p < 0.05$) does not necessarily demonstrate that there is no worthwhile effects in the athletic field, specifically at the elite level where sample sizes may be limited, and where small changes in performance from training interventions can still yield meaningful results.[21]

Muscle soreness has been demonstrated to be related to reductions in neuromuscular performance in rugby athletes,[15,23] with skeletal muscle damage proposed as a causative factor.[24] Exposure to cold, and CWI in particular, has been demonstrated to enhance recovery in neuromuscular function in rugby.[2] In our study, the beneficial effects of CWI attenuating decrements in neuromuscular function (i.e. CMJ peak force) were demonstrated by a *small* effect in favour of the CWI group (Table 5). These results are in concordance with previous literature

demonstrating a beneficial effect of CWI enhancing neuromuscular performance for up to 48 h after rugby training or competition.[8,9] Moreover, our findings are consistent with findings from Roberts et al.[12] that demonstrated that CWI enhances the recovery in muscle function as demonstrated by the capability to perform more volitional work in the squat exercise.

One of the key questions this study aimed to answer was whether CWI would attenuate the fatigue accumulated in response to the high week-to-week training load experienced by elite rugby athletes during a pre-season phase of training. Neuromuscular performance decreased for the CON group on the first day of Week Three (i.e. post ~65h of no training) from baseline (-6.9%). In contrast, for the CWI group, CMJ was only slightly decreased on the first day of Week Three (-1.32%), with *small* effect being observed for the differences between groups (Table 5), suggesting the athletes on Week Three were better recovered and able to maintain training intensity. Moreover, a trend towards lower levels of IL-6, associated with a *moderate* effect size in favour of CWI, demonstrates that CWI may attenuate IL-6 over the longer term. It has been suggested that if high training loads occur without adequate recovery, a chronic increase of circulating cytokines (e.g. IL-6) can occur, increasing the risk of maladaptation.[25] For example, Anderson et al.[26] found a chronic elevation in IL-6 in collegiate American football athletes following a high intensity 6-week period, demonstrating that when training loads are high, IL-6 can be chronically increased. Together, with the decrement in CMJ performance observed in the CON group, the *moderate* increase in IL-6 in the comparison between groups (i.e. CON > CWI) may suggest that athletes were not able to positively respond to training load.[25]

Our findings are supported by those from Halson et al.[5] whom observed a likely beneficial effect of CWI on cycling performance in highly-trained endurance cyclists during a 21-day intensification phase, followed by an 11-day taper period. In rugby, the investigation of the chronic

effects of CWI is limited to the one study that observed no beneficial effects of CWI.[13] However, as previously discussed, the results from that study are not necessarily transferable to elite team-sport athletes as the training load was relatively low. The participants of our study are professional athletes that were exposed to a very dense training schedule (e.g. two or more training sessions occurring every training day) (Tables 2 and 3). Therefore, CWI may aid in the maintenance of high mechanical outputs (e.g. high values of force, power and speed) during periods with increased training volume. Particularly in this study, on Day Four of each week, the athletes in CON had a decrease in neuromuscular performance. This may suggest that athletes underperformed during the speed session performed on Day Four, due to an increased level of fatigue. Moreover, when CWI is implemented with athletes exposed to a high-density training schedule, it may prevent maladaptive responses from training.

It is important to note some limitations of this study. Similarly to other research investigating the effects of CWI in rugby, the small sample size (10 and 13 athletes in CWI and CON, respectively) may be a limiting factor in the present study. The inference-based analysis method used in this study is often used in research with small samples of elite athletes to overcome this limitation.[21] Given the limited access to a greater sample size, it was not possible to include a placebo group in our study. Previous research has demonstrated that a thermoneutral water immersion placebo is as effective as CWI on the improvement of acute muscle function and perceptual measures. Therefore, the potential for placebo effect in the current study cannot be discounted [27]. Another limitation of the current study is that saliva was collected on day four of each week where athletes had ~36 hours to recover from the previous training session. Previous research has demonstrated that cortisol and IL-6 increase significantly after an elite rugby match, but values decrease to baseline values 14 hours post-match.[28] While the IL-6 and cortisol were

likely to increase after exercise, it is unlikely that they would still be elevated 36 hours after exercise (i.e. no training occurred between Days Two and Four), therefore changes in these markers reflects a chronic (i.e. week to week) exposure to training loads.[28] Another limitation was fact that the temperature used for the CWI (10ºC) was not individualized. This approach was necessitated by the practicalities of working in the elite sport environment but should be noted as a potential limitation as differences in body composition (i.e. muscle mass and body fat) and the ratio between body surface area and body mass (BSA:BM) lead to different responses in body temperature.[29] It could be expected that if CWI temperature was individualized (i.e. lower CWI temperatures for subjects with a greater BSA:BM and fat mass), then the beneficial effects of CWI on recovery could be increased.[30]

PRACTICAL APPLICATIONS

When athletes are exposed to high volumes of training with limited time to recover between training sessions, practitioners should consider the implementation of CWI in order to speed up recovery of neuromuscular performance and improve perceptions of lower body DOMS. Furthermore, if high volume training is prolonged for several weeks (e.g. three or more weeks), CWI may prevent maladaptive responses from training.

CONCLUSIONS

Our study is the first to demonstrate that CWI may provide a beneficial effect to recovery in both the acute and chronic setting when elite team-sport athletes are exposed to high training loads. As previously mentioned, chronic exposure to cold modalities has been reported to both blunt acute anabolic pathways associated with adaptations to training,[11] and enhance recovery of submaximal muscle function.[12] Here we demonstrate that CWI may enhance recovery in elite rugby athletes, allowing the athletes to perform at greater intensities which may in turn, lead to a

greater overall adaptive stimulus.[5] Therefore, in order to further clarify this question, future research investigating the chronic effects of CWI within an athletic population should include measurements of muscle size or markers of hypertrophy simultaneously with measures of performance over a longer period of time (e.g. >4 weeks).

The results of the current study do not constitute endorsement of the product by the authors or the journal.

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TABLE 1 – Participant characteristics. Data shown as means ± SD.

	CWI group (n = 10)	Control group (n = 13)
Age (years)	22.9 ± 2.7	22.3 ± 1.9
Body mass (kg)	105.4 ± 16.3	110.2 ± 12.4
Height (cm)	185.6 ± 5.1	189.4 ± 7.3
∑ 8 skinfolds (mm)	80.2 ± 17.8	91.4 ± 28.4
Squat 1-RM (kg)	183.9 ± 30.1	184.5 ± 32.2
Bench Press 1-RM (kg)	140.5 ± 26.5	136.8 ± 28.5
Chin-ups 1-RM (kg)	144.0 ± 15.7	135.0 ± 32.2
Speed 10 m (s)	3.02 ± 0.19	2.95 ± 0.18
Yoyo IRTL1	18.2 ± 1.1	17.3 ± 1.1

One repetition maximum (1-RM), Intermittent recovery test level 1 (IRTL1)

TABLE 2 – Weekly training schedule during the three weeks of the study. Resistance training, conditioning and technical-tactical duration (minutes), and qualitative intensity or type of training are described.

	Training period						
	Day 1	Day 2	Day 3	Day 4	Day 5	Day 6	Day 7
Before	CMJ+Q			CMJ+Q+Sal			
Morning	**Speed session** (30') **Gym session** (LB: 75') **TT** (45'; Moderate)	**Gym session** (UB: 75') **TT** (45'; Moderate)	Theory sessions	**Speed session** (30') **Gym session** (LB: 75') **TT** (45'; Moderate)	**Gym session** (UB: 60') **TT** (75'; Moderate)	Day-off	Day-off
Afternoon	**TT** (90'; High) **Cond** (30'; High)	**TT** (60'; High)		**TT** (60'; High) **Cond** (30'; High)	**Cond** (50'; High)		

Jumping performance (CMJ); Wellness and soreness questionnaires (Q); Saliva Sample (Sal); Lower body resistance training (LB); Upper body resistance training (UB); Technical-tactical session (TT); Conditioning session (Cond).

TABLE 3 – Average weekly training loads for CWI and CON and differences between training groups for weeks One, Two and Three. Data presented as means ± SD unless stated otherwise. No significant differences observed between groups for any training load parameter.

	Week One		Δ CON – Δ CWI (Mean ±90% Confidence Limit; Effect Sizes)	Week Two		Δ CON – Δ CWI (Mean ±90% Confidence Limit; Effect Sizes)	Week Three		Δ CON – Δ CWI (Mean ±90% Confidence Limit; Effect Sizes)
	CWI	CON		CWI	CON		CWI	CON	
Gym sRPE (AU)	441 ± 61	415 ± 59	-25.8 ±43.7; *unclear* (-0.39)	440 ± 60	437 ± 45	-3.0 ±39.7; *unclear* (-0.05)	596 ± 66	606 ± 71	9.3 ±49.8; *unclear* (0.13)
Conditioning sRPE (AU)	359 ± 29	355 ± 28	-3.3 ±20.7; *unclear* (-0.11)	269 ± 29	281 ± 22	-11.4 ±19.3; *unclear* (0.36)	302 ± 33	315 ± 37	13.4 ±25.2; *unclear* (0.37)
Total sRPE (AU)	800 ± 84	770 ± 79	-29.1 ±59.8; *unclear* (-0.32)	709 ± 74	718 ± 29	8.4 ±47.5; *unclear* (0.10)	898 ± 89	921 ± 97	22.7 ±67.1; *unclear* (0.23)
HML (m)	838 ± 201	839 ± 139	0.9 ±130.4; *unclear* (0.00)	844 ± 184	915 ± 231	71.8 ±149.4; *unclear* (0.36)	1130 ± 304	1066 ± 318	7.4 ±169.9; *unclear* (0.03)
Relative distance (m/min)	73.2 ± 10.8	76.6 ± 5.3	3.4 ±6.6; *unclear* (0.29)	71.9 ± 8.5	76.0 ± 11.46	4.0 ±7.2; *unclear* (0.43)	82.1 ± 12.0	80.1 ± 9.0	-2.0 ±7.9; *unclear* (-0.15)

sRPE = Session rate of perceived exertion, AU = Arbitrary unit, HML = High metabolic load

TABLE 4 – Analysis of Variance for the variables of interest with Group (CWI vs CON) as the between subjects' factor and Day (CMJ, LB soreness and wellness) or Week (IL-6 and cortisol) as within subjects' factor. * represents significant difference.

Test	Source of variation	(df, df_{error})	F	p
CMJ Performance (N)	Day	(2,42)	3.666	0.034*
	Group	(1,21)	2.647	0.119
	Day*Group	(2,42)	0.555	0.578
LB soreness (AU)	Day	(2,42)	3.357	0.044*
	Group	(1,21)	2.122	0.160
	Day*Group	(2,42)	1.393	0.260
Wellness (AU)	Day	(2,42)	0.947	0.396
	Group	(1,21)	0.178	0.677
	Day*Group	(2,42)	2.400	0.103
IL-6 (uL)	Day	(1,17)	0.055	0.055
	Group	(1,17)	2.845	0.110
	Day*Group	(1,17)	0.232	0.636
Cortisol (uL)	Day	(1,16)	0.055	0.483
	Group	(1,16)	2.845	0.882
	Day*Group	(1,16)	0.232	0.337

TABLE 5 – Changes in measures of CMJ performance, perceptual soreness and wellness and saliva markers from Baseline (Day 1) to the remaining testing days.

	Week		Δ CWI (Mean ± SD; *Effect Size*)	Δ CON (Mean ± SD; *Effect Size*)	Δ CON – Δ CWI (Mean ±90% Confidence Limit; *Effect Size*)
CMJ Performance (N)	Week 1	Baseline	2679.6 ± 434.8	2916.8 ± 489.4	237.2
		Day 4 – baseline	11.3 ± 179.4; *trivial* (0.02)	-175.8 ± 252.7; *small* (-0.36)	-187.1 ±155.5; *small* (0.38)
	Week 2	Day 1 – baseline	-96.4 ± 228.8; *unclear* (-0.20)	-148.8 ± 210.2; *small* (-0.20)	-52.4 ±161.1; *unclear* (0.11)
		Day 4 – baseline	-77.1 ± 224.1; *trivial* (-0.16)	-191.1 ± 265.2; *small* (-0.39)	-114.0 ±176.1; *small* (0.23)
	Week 3	Day 1 – baseline	-34.9 ± 234.4; *trivial* (-0.07)	-188.7 ± 289.4; *small* (-0.39)	-153.8 ±188.4; *small* (0.31)
		Day 4 – baseline	-95.6 ± 201.2; *unclear* (-0.20)	-263.1 ± 313.3; *small* (-0.54)	-167.5 ±185.7; *small* (0.34)
LB soreness (AU)	Week 1	Baseline	3.6 ± 0.5	3.6 ± 0.6	0.0
		Day 4 – baseline	0.2 ± 0.6; *unclear* (0.30)	0.5 ± 0.6; *moderate* (0.88)	0.3 ±0.4; *small* (0.58)
	Week 2	Day 1 – baseline	-0.2 ± 0.3; *small* (-0.42)	0.1 ± 0.5; *small* (0.20)	0.4 ±0.3; *moderate* (0.62)
		Day 4 – baseline	-0.2 ± 0.6; *unclear* (-0.32)	0.3 ± 0.6; *moderate* (0.60)	0.5 ±0.4; *moderate* (0.91)
	Week 3	Day 1 – baseline	-0.3 ± 0.3; *small* (-0.58)	0.1 ± 0.5; *small* (0.23)	0.5 ±0.3; *moderate* (0.81)
		Day 4 – baseline	0.2 ± 0.6; *unclear* (0.37)	0.4 ± 0.7; *moderate* (0.66)	0.2 ±0.5; *unclear* (0.29)
Wellness (AU)	Week 1	Baseline	3.9 ± 0.7	3.8 ± 0.4	0.0
		Day 4 – baseline	0.0 ± 0.4; *unclear* (0.01)	0.3 ± 0.4; *small* (0.53)	0.3 ±0.3; *small* (0.50)
	Week 2	Day 1 – baseline	0.0 ± 0.5; *unclear* (0.03)	0.1 ± 0.4; *trivial* (0.11)	0.0 ±0.3; *unclear* (0.08)
		Day 4 – baseline	0.2 ± 0.5; *small* (0.36)	0.2 ± 0.6; *small* (0.43)	0.0 ±0.4; *unclear* (0.07)
	Week 3	Day 1 – baseline	0.0 ± 0.3; *unclear* (0.07)	0.1 ± 0.3; *trivial* (0.11)	0.0 ±0.2; *unclear* (0.04)
		Day 4 – baseline	0.3 ± 0.6; *small* (0.55)	0.2 ± 0.4; *small* (0.42)	-0.1 ±0.4; *unclear* (-0.13)
IL-6 (uL)	Week 1	Baseline	18.3 ± 6.5	19.7 ± 7.8	1.4
	Week 2	Day 4 – baseline	-1.7 ± 6.8; *unclear* (-0.23)	2.6 ± 3.0; *unclear* (0.35)	4.2 ±6.0; *unclear* (-0.58)
	Week 3	Day 4 – baseline	-3.1 ± 5.4; *small* (-0.42)	3.0 ± 10.1; *unclear* (0.41)	6.1 ±6.5; -*moderate* (-0.83)
Cortisol (uL)	Week 1	Baseline	0.84 ± 0.36	0.61 ± 0.19	-0.23
	Week 2	Day 4 – baseline	-0.01 ± 0.32; *unclear* (-0.03)	0.06 ± 0.12; *trivial* (0.19)	0.07 ±0.22; *unclear* (-0.22)
	Week 3	Day 4 – baseline	0.01 ± 0.32; *unclear* (0.02)	-0.03 ± 0.16; *trivial* (-0.10)	0.04 ±0.23; *unclear* (-0.12)