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Section: Original Investigation

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"Effects of external Counterpulsation on Post-Exercise Recovery in Elite League Players" by Roberts L et al.
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Original Investigation

Effects of external counterpulsation on post-exercise recovery in elite rugby league players

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Running title; **External counterpulsation for elite rugby players**

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ABSTRACT

Purpose: External counterpulsation (ECP) has previously been used to treat cardiac patients via compression of the lower extremities during diastole to increase venous return and coronary perfusion. However, the effects of ECP on exercise performance and markers of recovery in elite athletes is largely unknown. ***Methods***: On two separate occasions, 48 h apart, seven elite National Rugby League players performed an identical 60 min field-based conditioning session followed by a 30 min period of either regular ECP treatment or placebo. Power measures during repeated cycle bouts and countermovement jump height and contraction time derivatives were measured at rest, and 5 h post-exercise. Saliva samples and venous blood samples were taken at rest, post-exercise, and 5 h post-exercise to assess stress, inflammation and muscle damage. ***Results***: Post ECP treatment, cycling peak power output (p=0.028; 11%) and accumulated peak power (p=0.027; 14%) increased compared to the placebo condition. Post-exercise plasma Interleukin 1 receptor (IL-1ra) only increased after ECP (p=0.024; 84%) and concentrations of IL-1ra tended to be higher (p=0.093; 76%) 5 h post-exercise. Furthermore, testosterone to cortisol ratio was increased above baseline and placebo 5 hours post-exercise (p=0.017 to 0.029; 24 to 77%). Post-exercise salivary alpha amylase to Immunoglobulin A ratio decreased after treatment (p=0.013; 50%), compared with the placebo control. ***Conclusions***: Exercise performance and hormonal indicators of stress were improved, and inflammation markers were reduced following acute ECP.

Keywords: recovery, external counterpulsation, sport, pneumatic compression

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INTRODUCTION

Optimizing the post-exercise recovery windows is an invaluable aspect of athletes' physical preparation cycles. The importance of this window is highlighted by the compounding effects of successive training and/or competitive bouts on physiological and physical function, attributed to residual fatigue[1] and the subsequent requirements to strategically periodise the use of recovery in these contexts[2]. As such, numerous post-exercise recovery treatments exist to promote perceptual and/or physiological wellbeing after exercise *per se*; see Dupuy et al.[3] for a recent meta-analysis. Recovery therapies have also been reported to augment recovery after rugby specifically. For example, cold water immersion and contrast water immersion were superior than active recovery in restoring countermovement jump height, and decreasing muscle soreness and creatine kinase concentrations $\leq$ 42 h after rugby league match play[4]. Furthermore, contrast water immersion, compression garments and active recovery were efficacious in the recovery of creatine kinase activity after rugby union match play[5].

An emerging line of research relating to the post-exercise recovery period is the use of intermittent pneumatic compression (IPC) garments, which aligns with the current popularity of this therapy within the worldwide athletic community. Such IPC garments are worn on the arms and/or legs, with a methodological principle based on applying rhythmic, time-gated pressures ranging from 60-80mmHg[6-8] asynchronous to the cardiac cycle. Despite the origin of IPC stemming from clinical usage, a paucity of research exists pertaining to its efficacy in an athletic context. Few investigations have examined the use of IPC after resistance exercise[6], high intensity interval cycling[7], eccentric exercise[8], and mixed Olympic athletes training regimes[9]. However, despite the popularity of IPC and anecdotal support, contrasting data exists about perceptions of recovery[6,7], muscle soreness[6], pain[9], and performance[6-8]. Beneficial reports of IPC however, include responses such as reduced blood lactate concentrations[7], reduced elbow flexor stiffness and swelling[10], and increased transcriptional responses such as

peroxisome proliferator-activated receptor gamma coactivator 1-alpha (PGC-1α), and protein markers of vascular adaptation[11].

Another clinically-derived therapy, seen as an enhancement to IPC is external counterpulsation (ECP). ECP involves the rapid distal to proximal ECG-synchronised inflation of cuffs that surround the calves, thighs, and buttocks to supra-systolic pressures of up to 300 mmHg during diastole; and simultaneous cuff deflation during systole. Numerous patient populations have responded positively to ECP, including those suffering from type 2 diabetes[12], whilst reported benefits include increased maximal oxygen uptake[13] and peripheral flow mediated vasodilation[14].

A substantial body of literature exists pertaining to the aforementioned populations, however information on the effects of ECP on healthy, athletic populations is sparse. Furthermore, it's unknown how an acute session of ECP influences an athletic population. This is important, considering that reported approaches to ECP involve multiple sessions of ECP over time e.g. daily one-hour sessions, for 35 days[15].

Therefore, it's plausible that ECP may effectively promote acute post-exercise recovery in elite athletes where training demands require optimal performance twice within a day, for example training twice a day, as is typical in a pre-season period. As such, the aim of this investigation was to examine the efficacy of a single bout of post-exercise ECP in modulating acute, within-day recovery of biochemical markers, and subsequent physical performance in elite National Rugby League (NRL) players. We hypothesized that ECP would be an effective strategy to promote acute recovery characteristics.

METHODS

Participants

Seven elite NRL players (19.5 ± 0.8 yrs; 100.0 ± 7.5 kg; 186.7 ± 7.5 cm; 5 forwards and 2 backs) volunteered to participate in this investigation. All participants were screened for

musculoskeletal conditions, and experimental procedures and potential risks were explained before written informed consent was obtained. Informed consent was obtained from all participants included in the study, and the study was approved by the Human Ethics Committee of The University of Queensland.

Experimental design

A randomized, cross-over, placebo-controlled design was implemented. Two identical field-based training sessions lasting approximately 60 min were completed; separated by 48 h. One, 30-minute period of ECP or placebo treatment was undertaken by participants, beginning 30 min after the end of each training session. To minimize order and perceptional effects, participants were blinded to the recovery interventions and randomized to who (3 or 4 of the seven) undertook ECP or placebo following the first training session, before crossing-over to undertake the second intervention after the second training session. Perceptions of wellbeing and recovery, saliva and blood samples were collected periodically, and neuromuscular performance and exercise capacity was re-assessed 5 hours post-exercise. Figure 1 provides an experimental overview diagram.

Control procedures

This investigation was undertaken during a typical pre-season. Therefore, habitual practices were followed throughout. These included a request for players to consume a habitual diet throughout, ad libitum fluid consumption during and after training sessions (water and isotonic beverage), and an upper body strength training session by all ~18 hours before trial day 2.

Exercise sessions

Two, identical outdoor pre-season training sessions were performed early in the pre-season period. Both were endurance-focused involving repeated 1-3 min running intervals;

beginning at 8 am each day, under similar environmental temperatures. Session total running distance and volumes over speed thresholds (a) $VT1_{IFT}$; distance at speeds >68% V_{IFT} and (b) $VT2_{IFT}$; distance at speeds >87% V_{IFT} were examined using a portable non-differential global positioning system at 5 Hz, and interpolated to 15 Hz (SPI HPU GPSports, Canberra, Australia).

Recovery intervention

Thirty minutes after the sessions, 30 minutes of ECP or placebo were performed in separate air-conditioned rooms (~22°C) using a treatment table (NCP-4, Renew GPL, Singapore), and a 3-lead ECG attached to the torso to trigger a pneumatic inflation device (NCP-4, Renew GPL, Singapore). Inflation occurs during diastole, triggered by T-wave peak, whilst all cuffs deflate simultaneously at the peak of the P-wave. The placebo treatment was identical to ECP, but with no pneumatic pressure. Blood pressure was measured from the brachial artery with an automated sphygmometer (HEM 7320, Omron, Melbourne, AUS).

Muscle function assessment

Muscle function was measured from 5 x 20 sec maximal stationary cycling bouts (Wattbike®, Wattbike Ltd., Nottingham, UK), each separated by 90 sec passive recovery. Variables derived included; peak power (peak power from any of the 5 bouts), accumulative peak power (summation of peak over from all 5 bouts); peak work (peak work from any of the 5 bouts), accumulative peak work (summation of the peak work performed over all 5 bouts); peak mean power (highest mean power from any of the 5 bouts); and peak and mean power decrement (decrement in power derivatives between bouts 1-5).

Perceptions

Perceptions of comfort and exertion were collected during the recovery treatments via Likert scales. Comfort was measured on a bi-polar Likert scale, anchored from -10; most uncomfortable to 0; neutral, to +10; most comfortable. The 6-20 rating of perceived exertion

scale was used to collect perceptions of the perceived exertion of the heart during the treatments. Perceptions of comfort and exertion were collected immediately before, after 15 min, and immediately after the 30 min recovery treatment.

Biochemical analyses

Venous samples were collected antecubitally into EDTA vacutainers, whilst saliva was collected by swabs (#51.1534.500, Sarsedt, Germany). Both were centrifuged at 1,500 x g for 10 min at 4°C, before aliquots were formed and stored at -80°C for analysis. One player abstained from blood collections, therefore plasma results n=6.

Salivary αAmylase (#1-1902), Immunoglobulin A (IGA) (#1-1602) and Testosterone (#1-2402) were measured by commercial kits (Salimetrics, USA). An automated clinical analyzer (Cobas e411) and corresponding kits were used to measure plasma myoglobin and testosterone, and plasma and salivary cortisol (Roche diagnostics GmbH, Germany). Plasma Interleukin 1 receptor antagonist (IL-1ra) was measured by commercial immunoassay (#DRA00B, R&D Systems, Minneapolis, USA). Plasma Interleukin (IL) 6 (IL-6) and 10 (IL-10), and Tumor Necrosis Factor-α (TNFα) were measured by multiplex immunoassay (Luminex premixed HS, R&D Systems, USA). Mean coefficients of variation were 1.5% (αAmylase), 2.5% (IGA), 1.38% (myoglobin), 1.48 % (cortisol), 1.56 % (testosterone), 4.11% (IL-1ra), 7.3% (IL-6), 7.5% (IL-10) and, 5.1% (TNFα).

Statistical analysis

Statistical analyses were performed using the Statistical Package for Social Sciences (SPSS; v19, IBM, Armonk, NY, USA). Two-factor repeated-measures ANOVA's were used to evaluate main effects of time, condition, and time*condition interaction effects for all variables; except blood pressure and perceptions which were assessed by Student's t test. Following ANOVA, Bonferroni post hoc testing was conducted where appropriate. Data are presented as means ± SD. Significance was accepted at $p \leq 0.05$. Cohen's effect sizes (d) were

calculated; interpreted as 0.2 to ≤0.5 = small, 0.51 to ≤0.8 = moderate, and >0.8 = large. A *d* followed by an absolute range represents the range of the *d*, whilst a *d* ± represents the 90% confidence interval range.

RESULTS

Exercise sessions

No differences (p>0.05) existed between training sessions in total distance travelled (6401m Vs 6345m), mean distance above selected thresholds; $VT1_{IFT}$ (2088m Vs 2559m), $VT2_{IFT}$ (922.8 vs. 954.9), nor mean wet bulb globe temperature (28.8°C Vs. 27.4°C) when comparing session 1 with 2, respectively.

Recovery interventions

Summated average perceptions of exertion during the recovery treatments tended to differ between ECP (8.7±2.7) and placebo (6.6±0.5) treatments (p=0.083, *d* 0.93(0.00-1.86)). Average overall perceptions of comfort during the recovery treatments was higher during placebo (8.7±1.1) compared with ECP (1.4±5.1) (p=0.011, *d* 1.4(0.69-2.15)). Systolic, diastolic and mean arterial pressure did not change after undertaking the recovery therapy (all time and interaction main effects p >0.05). However, a condition main effect (p=0.003) identified lower diastolic blood pressure in placebo (58.71±5.82 Vs 70.43±8.08 mmHg) before (p=0.007, *d* 1.3(0.6-1.97)) and after (54.29±8.3 Vs 67.29±8.20 mmHg) (p=0.027, *d* 1.3(0.37-2.13)) treatment compared with ECP. Similarly, a condition main effect (p=0.004) identified lower mean arterial blood pressure (MAP) in placebo (78.05±7.09 Vs 89.14±8.06 mmHg) before (p=0.004, *d* 1.2(0.62-1.79)) treatment compared with ECP. MAP tended to remain lower after treatment in the placebo compared to ECP (77.76±5.97 Vs 87.86 ± 8.26 mmHg; p=0.060, *d* 1.2(0.16-2.15)).

Performance

Only peak power and accumulative peak power changed over the course of the trial (time main effects p=0.008 to 0.01). Peak power (p=0.028) and accumulative peak power (p=0.027) increased after ECP, with no change after placebo treatment. No significant changes occurred in peak work, accumulative peak work, or decrement in mean and peak power. See figure 2.

Blood responses

Testosterone and cortisol changed during the trial (time main effects p<0.0001) but weren't influenced by recovery treatments (no condition or interaction effects). Testosterone didn't change pre-post exercise, but decreased between from post-exercise to 5 hr in both conditions (p=0.001-0.002, d 1.59-1.68), with concentrations at 5 hr being below rest (p=0.003-0.008, d 1.34-1.53). Cortisol increased after exercise (p<0.0001-0.002, d 1-70-1.72), decreased from post-exercise to 5 hr (p < 0.0001, d 1.81-1.87), and resulted in below basal concentrations (p=0.001-0.024, d 1.37-1.52) at 5 hrs, in both conditions. The testosterone relative to cortisol ratio changed over the trial in a condition-dependent manner (interaction p=0.015). The ratio decreased pre-post exercise (p=0.007-0.015, d 1.45-1.49) and increased by 5 hrs (p=0.005-0.008, d 1.48-1.55) in both conditions. However, the ratio increased above baseline (p=0.029, d 1.2±0.81) and placebo (p=0.017, d 0.6±0.34) after ECP. See figure 3.

The muscle damage marker, myoglobin, changed equally in both conditions (time main effect p=0.002, no condition or interaction effect). Concentrations increased pre-post exercise (p=0.005-0.007, d 1-53-1.56), remaining elevated at 5 hrs (p=0.006-0.007, d 1.54-1.58).

All cytokines changed over the trial (time main effects p<0.0001), with only the change in IL-1ra dependent on the recovery treatment (interaction main effect p=0.034). TNFα concentrations increased pre-post exercise (p=0.001-0.019, d 0.84-1.28), returning to baseline by 5 hrs (p=0.006-0.012, d 0.98-1.16) in both conditions. IL-6 concentrations increased pre-

post exercise (p=0.004-0.007, d 1.59-1.68), decreased to 5 hrs (p=0.007-0.013, d 1.53-1.61) and tended to remain elevated above baseline (p=0.012-0.088, d 0.76-0.81), in both conditions. IL-10 concentrations increased pre-post exercise (p=0.004-0.021, d 1.38-1.63) before decreasing to 5 hrs (p=0.006-0.023, d 1.35-1.58). Concentrations remained above baseline after ECP (p=0.003, d 0.69±0.32), returning to baseline after placebo. IL-1ra concentrations increased pre-post exercise in both conditions (p=0.003-0.006, d 1.49-1.68), remaining unchanged and above basal levels (p=0.001, d 1.78) after placebo. However, concentrations increased further from post-exercise to 5 hr after ECP (p=0.024, d 1.08±0.70) and remained above baseline (p=0.011, d 1.46±0.75). Concentrations tended to be higher than placebo after ECP at 5 hrs (p=0.093, d 1.07±1.04).

Saliva responses

IGA activity didn't change over the trial and wasn't influenced by recovery treatments (all main effects p>0.05). Cortisol, testosterone and αAmylase concentrations increased over the trial (time main effects all p<0.0001) without condition or interaction effects (all p>0.05). Cortisol increased pre-post exercise (p=0.001-0.003, d 1.42-1.69) before decreasing by 5 hr (both p<0.0001, d 1.73-1.86), resulting in concentrations below rest (p<0.0001-0.003, d 1.50-1.64) in both conditions. Testosterone concentrations tended to increase pre-post exercise (p=0.049-0.085, d 0.81-0.83) before decreasing by 5 hr (both p=0.001, d 1.66-1.72), resulting in concentrations below rest (both p=0.001-0.002, d 1.36-1.38), in both conditions. αAmylase concentrations increased pre-post exercise in both conditions (both p<0.0001, d 1.63-1.68) before decreasing by 5 hr (both p=0.007, d 1.10-1.21); resulting in concentrations above rest after ECP (p=0.039, d 0.93), and a tendency for a similar response after placebo (p=0.082, d 0.86).

The testosterone to cortisol ratio changed over the trial (time main effect $p<0.0001$) without condition or interaction effects (both $p>0.05$). The ratio decreased pre-post exercise ($p=0.003$-0.035, d 1.15-1.56) before increasing by 5 hr ($p=0.008$-0.019, d 1.35-1.40), in both conditions. The IGA to cortisol ratio changed over the trial and differed between conditions (time main effect $p<0.0001$, condition main effect $p=0.047$), but no interaction existed ($p>0.05$). The ratio tended to decrease pre-post exercise in both conditions ($p=0.024$-0.058, d 0.98-1.26) before increasing by 5 hr ($p=0.001$-0.003, d 1.56-1.62), resulting in above basal ratios ($p=0.005$-0.008, d 1.18-1.23). The ratio tended to be higher at 5 hr after ECP compared with placebo ($p=0.098$, d 0.69±0.69). The αamylase to IGA ratio changed over the trial (time main effect $p<0.008$) without condition or interaction effects (both $p>0.05$). The ratio increased pre-post exercise in both conditions ($p=0.008$-0.012; d 1.26-1.28), but only decreased after treatment following ECP (p=0.013; d 0.92±0.56). See figure 4.

DISCUSSION

The aim of this investigation was to examine the efficacy of a single bout of post-exercise ECP in modulating acute, within-day recovery in elite football players. Exercise performance and hormonal indicators of stress were improved following acute ECP. Additionally, inflammation markers were reduced following ECP treatment. These preliminary data are favourable for the efficacy of ECP therapy to assist within-day recovery during pre-season training.

In contrast to our results herein, a single 30 min session of enhanced ECP in young, trained individuals did not enhance power output during cycling exercise, or elicit any other benefits compared with a placebo condition[16]. However, a number of factors may account for these differences. For example, a key methodological difference compared with our ECP approach was the pressure used. Valenzuela et al.[16] utilised a maximum pressure of 80 mmHg, whereas pressure was almost 4-fold higher in this investigation; potentially providing a greater

stimulus. Secondly, whilst Valenzuela and colleague's participants were classified as elite athletes, they were junior Male and Females aged 16 ± 2 yrs compared with Males aged 19.5 ± 0.8 yrs here. It is unknown whether such recovery therapy differentially effects Males compared with Females, and also juniors compared with adults. We speculate that the aforementioned factors, account for the null findings by Valenzuela and colleagues' compared with our own.

Similar to the findings of the current investigation, Borne et al.[17] showed improvements in peak and mean power output during repeated all out 30s cycling sprints following a recovery intervention that increased blood flow. They used neuromuscular electrical stimulation as a means of increasing blood flow to the muscles through low-frequency stimulation, specifically increasing calf arterial inflow. When considering ECP as an aid to assist recovery between repeated high-intensity exercise sessions, a number of mechanisms could potentially explain the improvement in performance. Firstly, an increase in either local and/or systemic blood flow may enhance the removal of metabolic waste products from the exercised muscle. Maintaining adequate blood flow to the lower limbs could have possibly assisted with the clearance of the accumulated lactic acid that could otherwise adversely affect muscle function[18]. This has been shown previously, where blood lactate levels have been reduced following ECP treatment, although being in a canine model[19].

A second possible explanation of beneficial improvement from the increase in blood flow induced by ECP during recovery could be related to the muscle-pump theory as suggested by Borne et al.[17] During ECP treatment, the contraction of the exercised muscle could reduce venous pressure thus increasing the arteriovenous pressure gradient. The enhanced arteriovenous pressure gradient would result in an increase in arterial blood reaching the skeletal muscles[20]. The ECP treatment could enhance arterial blood flow resulting in an increased supply of oxygen supply to the trained muscles[21]. Following high intensity exercise,

oxygen demand is increased to replenish myoglobin and hemoglobin stores and re-synthesise ATP and PCr and muscle oxygen uptake remains above resting levels for over one hour[22]. The increased blood flow caused by ECP during recovery may also play a role in the resynthesis of glycogen from lactate by assisting in glucose delivery and uptake by the muscle cells[23]. Therefore, ECP may be particularly useful to assist in recovery from high intensity exercise where blood glucose peak level are much greater than following prolonged exercise and more glycogen resynthesis is required[24].

The findings of the current study align with research involving skeletal muscle ischemic preconditioning. Patterson et al.[25] showed likely increases both mean and peak power output in the first three sprints by 2-4% during the early stages of repeated sprint cycling. They suggested ischemic preconditioning may have potentiated performance by improving muscle force production following which has previously be shown[26]. Whilst speculative, it's possible that the similarities between ECP and ischemic preconditioning, involving periods of ischemia followed by reperfusion, may provide similar aid for improving sprint-based performance during subsequent within-day exercise sessions.

The testosterone to cortisol ratio may reflect the body's overall anabolic-catabolic balance. In the current investigation the plasma T:C ratio only increased after ECP treatment, reflecting that the body is in a positive state of anabolism. This finding is in contrast to another blood flow enhancing recovery method, neuromuscular electrical stimulation, where no changes in T:C ratio were found following treatment in elite rugby and football players[27]. However, the finding is consistent with the increase in T:C ratio found following cryotherapy[28]. The increases in T:C at 5-hours indicate a potentially favourable hormonal profile following a single exposure to ECP after exercise. This finding may have important implications for practitioners needing to implement recovery strategies during tournaments when congested periods of competition on the same day exist.

Interleukin (IL)-1 is a pro-inflammatory cytokine involved in both normal homeostasis and has a key role in the body's response to tissue injury and inflammation following exercise[29]. Interleukin 1 receptor agonist (IL-1ra) is a distinct form of IL-1 that binds to mediate these responses following exercise. In the current investigation post-exercise plasma IL-1ra increased after ECP (p=0.024; 84%) and this is a similar response to that shown previously following repeated bouts of whole body cryotherapy. Whilst this is in contrast to the findings of Peake et al.[30] who showed no difference in plasma IL-1ra concentrations following other recovery modalities (either cold water immersion or active recovery) following resistance exercise. Therefore, the role of IL-1ra following recovery interventions warrants further investigation.

This investigation examined the efficacy of ECP to assist in recovery of elite athletes, to act as a 'proof of concept' to determine whether further research into ECP is warranted. Whilst initial findings are promising, limitations must be considered. Athletes only received one familiarisation with ECP, therefore further sessions may have reduced the magnitude of the reported diastolic blood pressure and heart rate responses. A 5-hr testing time-course was used to determine the value of ECP when a morning and afternoon training program was implemented. A longer recovery period of 24-hrs would have greater application to post-competition ECP, however was beyond the scope of this investigation. The current findings now provide evidence to support research to examine effects at 24 or 48-hrs post-exercise. Finally, while the conditioning session was performed at high intensity, specific game demands such as contacts, body movement off the ground and changes of direction were not included. Therefore, the efficacy for ECP to be effective following a competition match at this stage can only be speculative.

PRACTICAL APPLICATIONS

External counterpulsation could be included in typical recovery strategies, in training programs that require more than one training session in the same day, in sporting events with short recovery periods, and for tournament sports that involve successive matches or rounds on the same day with short recovery periods. In particular, ECP could be mixed with other recovery modalities such as hydration and nutritional strategies in the first moments after exercise when the time available for recovery is crucial but limited.

CONCLUSIONS

A single session of ECP between bouts of high-intensity exercise in elite athletes induced effective, positive improvements in subsequent performance. Future research examining combined modalities, and varied ECP sessions characteristics are warranted. Future investigations utilising higher sample sizes to increase the statistical power of the study are also required.

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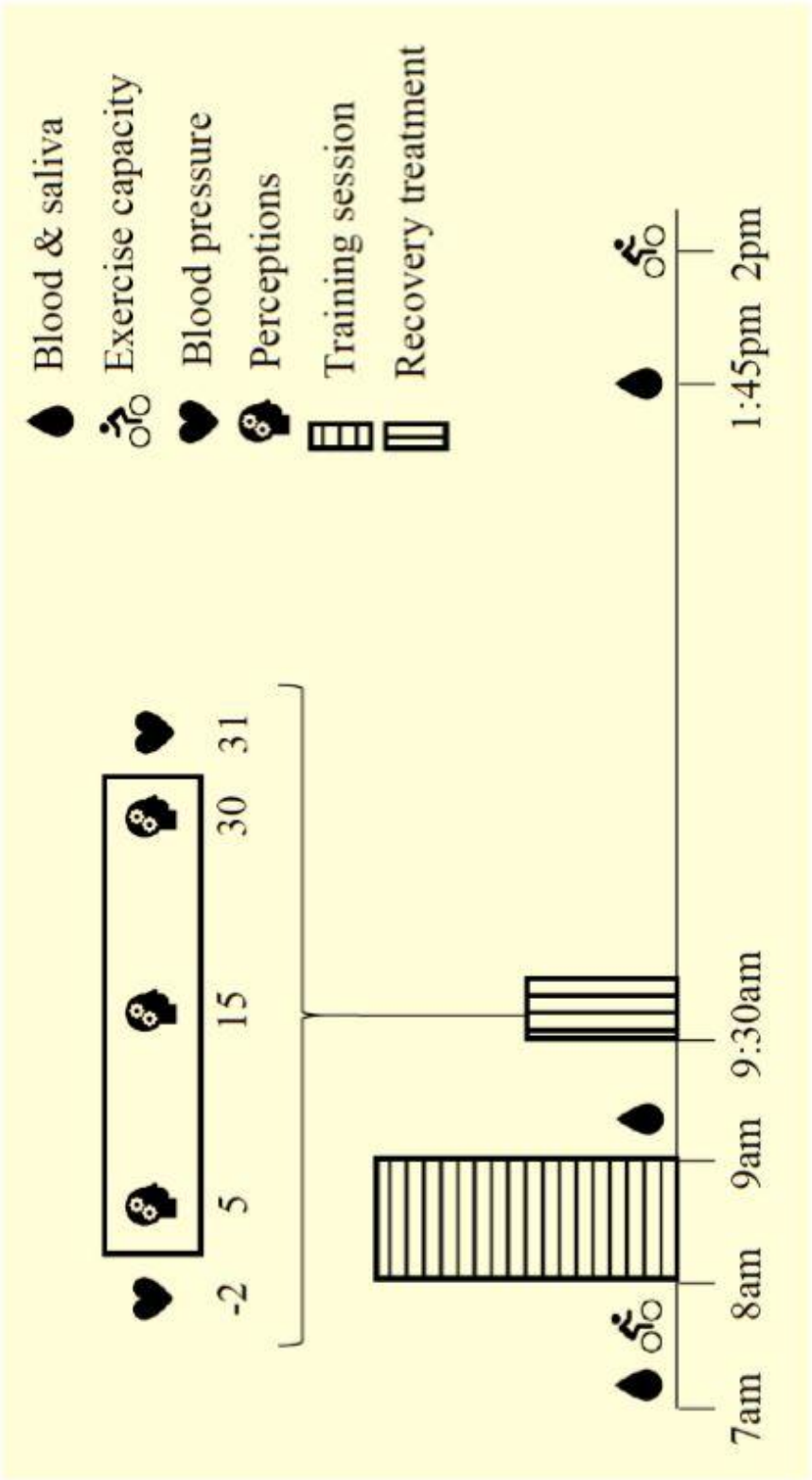

Figure 1: Experimental overview of each trial day.

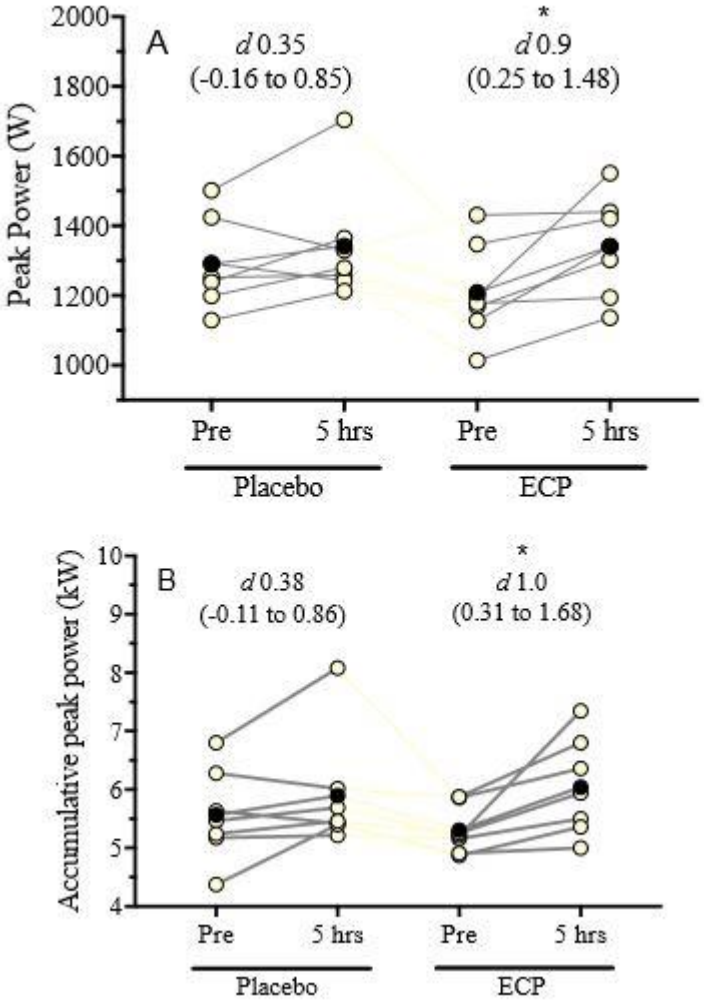

Figure 2: Individual (open circles) and group mean (closed circles) peak power (A), accumulative peak power (B), accumulative work (C), and peak power decrement (D) responses. *p<0.05 group mean change from Pre. *d* standardized Cohen changes from pre-exercise (90% confidence interval range).

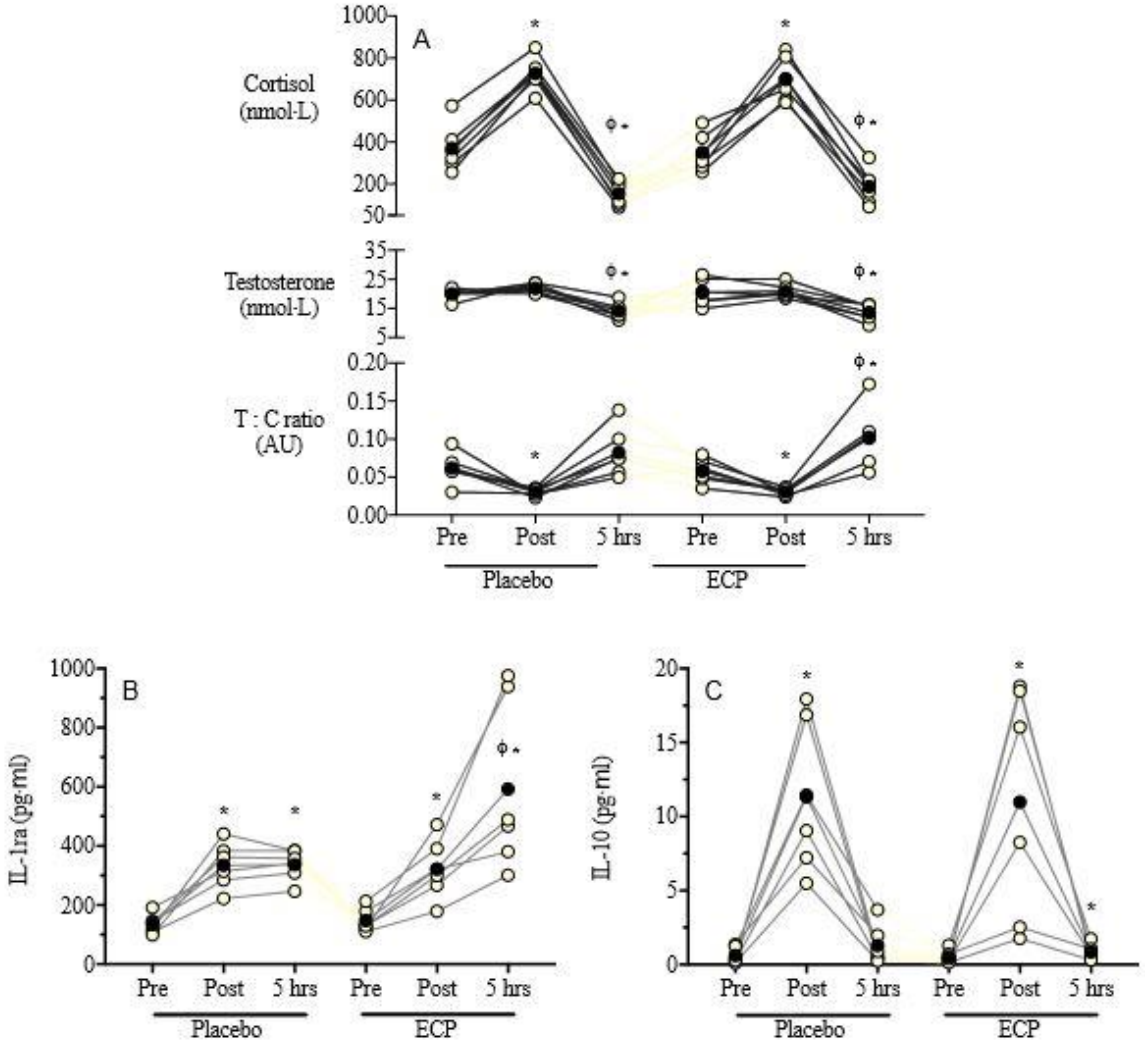

Figure 3: Individual (open circles) and group mean (closed circles) plasma cortisol (C), testosterone (T) and T:C ratio (A), IL-1ra (B), and IL-10 (C) responses. n= 6, **p*<0.05 group mean change from Pre, ϕ*p*<0.05 group mean change from Post.

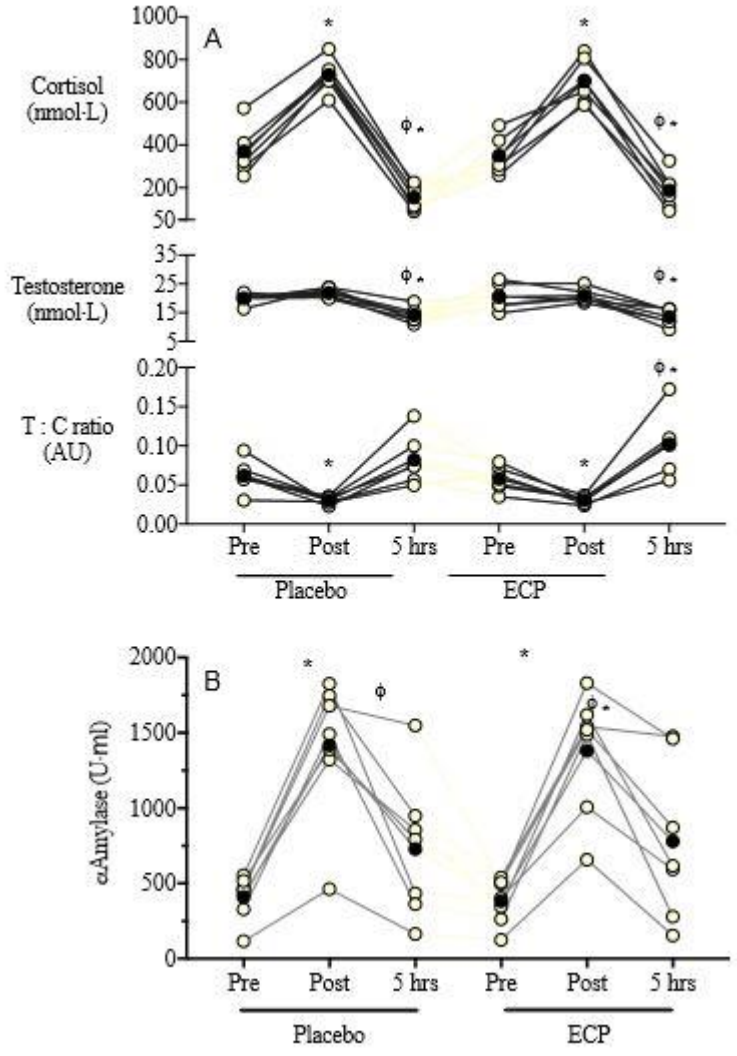

Figure 4: Individual (open circles) and group mean (closed circles) saliva cortisol (C), testosterone (T) and T:C ratio (A), and αAmylase (B) responses. n=7, *p<0.05 group mean change from Pre, $^{\phi}p$<0.05 group mean change from Post.