Full Title: Intermittent Lower-limb Occlusion Enhances Recovery after Strenuous Exercise

Running Title: Occlusion as a Recovery Intervention

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Abstract

Repeated cycles of vascular occlusion followed by reperfusion initiate a protective mechanism that acts to mitigate future cell injury. Such ischemic episodes are known to improve vasodilation, oxygen utilisation, muscle function, and have been demonstrated to enhance exercise performance. Thus, the use of occlusion cuffs represents a novel intervention that may improve subsequent exercise performance. Fourteen participants performed an exercise protocol that involved lower-body strength and power tests followed by repeated sprints. Occlusion cuffs were then applied unilaterally (2 x 3-min per leg) with a pressure of either 220 (intervention) or 15 mmHg (control). Participants immediately repeated the exercise protocol, and then again 24 h later. The intervention elicited delayed beneficial effects (24 h post) in the countermovement jump test with concentric (ES = 0.36) and eccentric (ES = 0.26) velocity recovering more rapidly compared with the control. There were also small beneficial effects on 10- and 40-m sprint times. In the squat jump test there were delayed beneficial effects of occlusion on eccentric power (ES = 1.38), acceleration (ES = 1.24), and an immediate positive effect on jump height (ES = 0.61). Thus, specific beneficial effects on recovery of power production and sprint performance were observed both immediately and 24 hours after intermittent unilateral occlusion was applied to each leg.

Keywords: Ischemia · Reperfusion · Muscle Function

Introduction

It has been demonstrated that brief repeated periods of occlusion followed by reperfusion can mitigate the injurious effects of prolonged ischemia in cardiac muscles, as well as attenuate other cellular damage (Eisen et al., 2004; Iliodromitis et al., 2007). The cardioprotective effect of ischemic preconditioning was first described by researchers who demonstrated that multiple brief ischemic episodes resulted in a reduction in infarct size in canine hearts subsequently exposed to prolonged ischemia (Murry et al., 1986). Subsequently, the beneficial effects of cycles of ischemia and reperfusion have been observed in a number of mammalian species, including humans, with ischemic preconditioning eliciting "robust and reproducible" protective effects (Hausenloy & Yellon, 2008). The mechanism of such protection however has not been fully elucidated.

It is known that remote application of an ischemic stimulus to the upper arm using a blood pressure cuff can produce cardioprotective effects in humans (Bøtker et al., 2010). Current knowledge suggests that cardioprotective effects of cycles of ischemia and reperfusion arise through activation of the reperfusion injury salvage kinase and survivor activating factor enhancement pathways (Gonon et al., 2010). Endogenous ligands such as adenosine, bradykinin, prostaglandins, and opioids have been implicated as initiating and mediating the protective effects of an ischemic intervention (Hausenloy & Yellon, 2008; Li et al., 2009). Importantly, elevated adenosine levels can increase oxygen delivery and blood flow due to the dilation of resistance vessels. Interestingly, increased blood flow has been purported as a mechanism behind many recovery strategies via a repletion of ATP (Connolly et al.,

2003) and metabolic waste product removal (Gill et al., 2006; Hamlin, 2007; Higgins et al., 2010; Vaile et al., 2010).

Cycles of ischemia and reperfusion have also been associated with the preservation of ATP levels in canine myocardium indicating a decreased energy demand (Jennings et al., 2001). Furthermore, it has been reported that an ischemic stimulus can improve muscle function (Lawson & Downey, 1993) as well as exercise performance and maximal oxygen uptake in humans (de Groot et al., 2010; Jean-St-Michel et al., 2011). However, the potential for cycles of ischemia and reperfusion to stimulate the recovery process following exhaustive exercise has received little research attention. As a result of the reported effects of ischemia on blood flow, oxygen extraction, and muscle function, the present study examined the effectiveness of an alternating unilateral occlusive protocol as a recovery intervention to enhance subsequent exercise performance in healthy participants.

Methods

Participants

Fourteen healthy individuals (ten males and four females, age 32 ± 7 yr, body mass 76.4 ± 12.9 kg) volunteered to participate in this study. All participants were recreationally-trained non-smokers. Based on their medical history all participants were free of contraindications that would preclude participation in strenuous exercise and gave their written informed consent. The Ethics Committee of the Waikato Institute of Technology approved the study protocol.

Protocol and measurements

All participants refrained from alcohol and intense physical exercise for at least 24 h prior to testing. The exercise protocols were performed at the same venue and at the same time of day to minimise any confounding effects of daily biorhythms. On the days of testing, as much as possible, all participants ensured they had achieved a previous good night's sleep and were hydrated and fed. Across the trials themselves, water was available and participants encouraged to stay well hydrated. In order to eliminate possible confounding training or familiarisation effects, the application of 220 mmHg occlusive recovery intervention and the 15 mmHg control condition were assigned in a counterbalanced, cross-over design. Participants were not informed about the rationale of the study to reduce any placebo effect.

Each participant reported to the testing venue on five occasions (Figure 1). The first visit (Session 1) allowed the participants to become familiar with the standardised warm-up and exercise protocols. The standardised warm-up that was completed before every session consisted of a 400 m jog followed by dynamic stretches targeting the muscles of the lower limbs (incorporating lunges, leg swings, and skipping) that lasted approximately 10 minutes. Session 1 was completed at least one week prior to the start of the exercise trial.

****Figure 1 near here****

In Session 2, following the warm-up, the participants performed a set of three squat jumps with a 90 degree knee angle. In a non-fatigued state the participants then performed a set of three countermovement jumps (CMJ). For the CMJ, participants

lowered themselves into a self-selected half-squat position, and utilised the stretch-shortening cycle to jump explosively in an effort to achieve maximal height. Participants were instructed to sink to absorb their mass on landing. All jumps were performed with a 6 kg bar resting on the posterior deltoids at the base of the neck. The characteristics of power production (maximum and mean values for eccentric and concentric peak power, velocity, and acceleration as well as work, jump height, time to peak power and time to peak velocity) during each squat jump and CMJ repetition were quantified with a GymAware^{TM} optical encoder (50-Hz sample period with no data smoothing or filtering; Kinetic Performance Technology, Canberra, Australia). The GymAware^{TM} system recorded displacement-time data using a signal driven sampling scheme where position points were time-stamped when a change in position was detected, with time between samples limited to a minimum of 20 ms. The first and second derivate of position with respect to time was taken to calculate instantaneous velocity and acceleration respectively. Acceleration values were multiplied by the system mass to calculate force, and the given force curve multiplied by the velocity curve to determine power. Mean values for force and power were calculated over the concentric and eccentric portions of each movement and peak values for velocity, force, power and time taken to achieve these values were derived from each of the respective curves. Jump height was determined as the highest point on the displacement-time curve. The validity and reliability of the power optical encoder to provide data regarding peak and mean values of force and power as well as jump height have been reported previously (Crewther et al., 2011; Drinkwater et al., 2007; Taylor et al., 2010). Specifically, Taylor and colleagues (2010) reported CVs of day-to-day reliability of between 0.8 and 6.2%, with smallest worthwhile changes of between 1.9 and 4.3% for power,

velocity, force, and height measurements with the GymAware^{TM} system. System mass (mass of the bar plus body mass) was used for the calculation of maximal and mean concentric and eccentric power generated in the squat jump and countermovement jump exercises (Dugan et al., 2004). Immediately following the jump tests, the participants were instructed to perform a set of six leg press repetitions on a flywheel dynamometer (Concept 2, Vermont, U.S.A.) with the intention to produce maximal velocity.

The participants then performed three sub-maximal 40 m efforts at 50, 70, and 90% intensity before performing a series of six maximal 40-m sprints departing every 30 seconds. Dual-beam timing lights (Swift Performance Equipment, Australia) were used to monitor 10- and 40-m sprint times over the six repeated sprints. A rating of perceived exertion (RPE) on a scale of 6 (no exertion) to 20 (maximal exertion) was assessed after each exercise protocol (Borg et al., 1987).

Cumulative sprint times for the six sprints are presented as, due to the variability inherent in fatigue indices, it has been suggested that total sprint time better reflects the ability of a participant to repeatedly produce maximal sprint efforts (Oliver, 2009). Repeated 40 metre sprints have been shown to be a reliable means of evaluating repeated sprint ability (Fitzsimmons et al., 1993; Glaister et al., 2009). Six repeated sprints were used as it has been suggested that less than eight sprints should be used to avoid a pacing strategy (Oliver, 2009) and greater than three sprints are required to observe a performance decrement (Balsom et al., 1992). Verbal encouragement and performance feedback was provided at each stage of the exercise protocol.

Immediately following the completion of the exercise protocol, participants adopted a comfortable supine position in a gymnasium and were fitted with a unilateral occlusion cuff (BJ Dare Medical Equipment, China) around the proximal portion of the leg as a recovery intervention. The cuff contained a pneumatic bag along its inner surface that was connected to a pressure gauge that was manually inflated to either 15 or 220 mmHg for three minutes. The 220 mmHg occlusion protocol was selected as a stimulus likely to induce lower limb ischemia, while the 15 mmHg protocol was a control condition. The occlusion pressure was constantly monitored and the cuff was alternated to the contralateral leg for a further three minutes. This cycle was repeated twice for a total of 12 minutes, and both legs received an 'ischemic dose' of six minutes per leg. The participants then repeated the squat jump, CMJ, dynamometer leg press, and 40-m sprint exercise protocol within 5 minutes of the cuff being removed. Twenty-four hours later, each participant returned and repeated the exercise protocol (Session 3). One week later the participants returned again and repeated the two days of testing (Sessions 4 and 5) using the alternate occlusion pressure treatment during the recovery period in a cross-over manner (Figure 1).

Statistical Analyses

The dependent variables were log-transformed before analysis and no observations were excluded as outliers. Back transformation provided estimates of mean effects as percentages and errors as coefficients of variations. Standardised changes in the mean of each measure were used to assess magnitudes of effects by dividing the changes by the appropriate between-participant standard deviations in the control condition. Magnitudes of the standardised effects were interpreted using thresholds

of 0.2, 0.6, and 1.2 for small, moderate, and large, respectively (Hopkins et al., 2009). Standardised effects of between -0.19 and 0.19 were termed trivial. To make inferences about the true (large-sample) value of an effect, the uncertainty in the effect was expressed as 90% confidence limits. The effect was deemed unclear if its confidence interval overlapped the thresholds for small positive and negative effects (Batterham & Hopkins, 2006). Thresholds for assigning the qualitative terms to chances of substantial effects were: <1 %, almost certainly not; <5 %, very unlikely; <25 % unlikely; 25–75 %, possibly; >75 % likely; >95 % very likely; and >99 % almost certain. The significance level was set at $p \leq 0.05$.

Results

The occlusion intervention of 220 mmHg had a clear beneficial effect on the mean squat jump height immediately following the occlusion intervention (Effect Size = 0.63, moderate effect) compared to the 15 mmHg control condition. There were also likely detrimental effects on mean eccentric peak velocity and peak acceleration in the CMJ and mean eccentric peak power in the squat jump immediately post intervention (Table 1). Other immediate effects were generally unclear or trivial.

****Table 1 near here****

Twenty-four hours after the occlusion intervention there was a likely beneficial effect on the rate of recovery of maximal power production (W) in the squat jump compared to the control condition (ES = 0.50, small effect). This delayed beneficial effect appeared to be more pronounced in males (ES = 1.02, moderate effect). In the squat jump test there were also large, clear beneficial effects of the occlusion

intervention on the recovery of mean eccentric peak power and eccentric peak acceleration 24-h post exercise (Figure 2). In the CMJ test there were clear beneficial effects of the occlusion intervention compared to the control condition on the mean concentric (ES = 0.36, small effect) and eccentric peak velocity (ES = 0.26, small effect) 24-h post-exercise.

****Figure 2 near here****

Total power produced in the dynamometer leg press (W) test 24 h post-exercise was also clearly enhanced as a result of the occlusion intervention (ES = 0.30, small effect), and again the magnitude of this effect was greater in the male participants (ES = 0.68, moderate effect). Participants that performed the occlusion intervention also recovered at a greater rate compared to those in the control intervention when the cumulative 10- and 40-m sprint times 24-h post intervention were assessed (Figure 3). A likely detrimental effect of the occlusion intervention was observed in the change in the rate of recovery on the mean concentric work performed in the squat jump at the 24 h time point. Other delayed effects were generally unclear or trivial (Table 1).

****Figure 3 near here****

Discussion

Cuff occlusion has previously been suggested as improving acute exercise performance however; to our knowledge these benefits have not been proposed as a recovery strategy. We report that the treatment of both legs with intermittent unilateral cycles of occlusion at 220 mmHg and reperfusion were effective at

eliciting substantial beneficial effects on specific aspects of subsequent maximal exercise performed both immediately following exposure and 24 hours later. Previously, acute beneficial effects of ischemic preconditioning on exercise performance have been observed and speculatively attributed to vasodilation and improved oxygen delivery associated with increases in adenosine, activation of ATP-sensitive potassium channels (de Groot, et al., 2010) and inhibition of afferent fatigue signalling increasing neural drive (Crisafulli et al., 2011). The preconditioning effects of adenosine are mediated via interaction A_3 adenosine receptors (G. S. Liu et al., 1994). Interestingly, stimulation of adenosine A_3 receptors has been reported to decrease creatine kinase responses to a muscle damaging eccentric exercise protocol (Wang et al., 2010).

Our study showed small but meaningful improvements in cumulative 10- and 40-m sprint times 24 h after the occlusion intervention. Ischemic preconditioning has been associated with a reduced rate of anaerobic glycolysis, elevated glucose levels, and ATP preservation (Jennings, et al., 2001; Pang et al., 1995). Enhanced muscle oxygenation (Saito et al., 2004), vasodilation, and oxygen delivery have also been attributed to cycles of ischemia and reperfusion which could have contributed to the observed results (de Groot, et al., 2010). Functional sympatholysis associated with activation of ATP-sensitive potassium channels (Joyner & Thomas, 2003) and changes in teleoanticipation (Noakes, 2011) may have also contributed to the observed recovery of maximal sprint performance.

The occlusion intervention was also associated with specific delayed beneficial effects on both concentric and eccentric force produced during CMJ and squat

jumps. Previous research has indicated that ischemia can enhance measures of skeletal muscle contractile function such as maximal isometric force production, Ca^{2+} handling, and EMG amplitude in animal models (Kohin et al., 2001; Lawson & Downey, 1993; Phillips et al., 1997). It should however be noted that a number of measures of lower limb function were impaired immediately after the occlusion intervention. These impairments were particularly apparent in the eccentric measures. The mechanism behind this impairment is not obvious, however hypoxia has also been reported to impair muscle spindle reactivity and such alterations of sensorimotor control may have contributed to the observed effects on eccentric measures (Delliaux & Jammes, 2006; Hoshikawa et al., 2010). It is also possible that the occlusive pressure was not well tolerated and a greater degree of familiarisation or incremental application of this pressure may have alleviated the detrimental effects to some degree. It should also be noted that due to the large number of variables measured in non-highly trained participants, the possibility that type I errors are apparent cannot be discounted despite rigorous statistical attempts to present reliable and worthwhile effects.

In the current study, the majority of the beneficial effects of the occlusion intervention were observed after 24 h. This observation suggests that the increased blood flow due to the reperfusion phenomena and improved muscular oxygen utilisation led to a more rapid return of muscle function. Alternatively, the observed results may have been partially mediated by a bimodal time course of ischemic preconditioning that has been described previously (Kuzuya et al., 1993). However, the physiological mechanism responsible for the effects on exercise performance

were not examined in the current study as it was the intention to evaluate functional benefits of the occlusive intervention.

The occlusion pressure of 220 mmHg was selected for the ischemic stimulus as this pressure has been suggested to restrict venous blood flow, cause pooling of blood in capacitance vessels distal to the belt, restrict arterial blood flow, and can elicit meaningful physiological responses in strength (Abe et al., 2005), sprint (Jean-St-Michel, et al., 2011), and endurance exercises (de Groot, et al., 2010). The cycle of three minutes occlusion and three minutes of reperfusion that comprised the occlusion intervention was repeated twice by both legs to give a total of a six minute 'ischemic dose' per leg. Unilateral occlusion has previously been demonstrated to elicit systemic cardioprotective effects (Bøtker, et al., 2010), although our occlusion intervention was designed to specifically target the musculature of the lower limbs. The three minute cycles of occlusion and reperfusion were also selected to fulfil the duration threshold criterion that has been reported previously (Van Winkle et al., 1991). This threshold has been suggested to reflect the period of ischemia required to accumulate sufficient localised adenosine concentrations where the adenosine receptors are sufficiently populated to elicit a preconditioning effect. Importantly, a total ischemic stimulus of 4-6 minutes has been shown to be most effective at eliciting a protective effect in human myocardium, regardless of the number of cycles of ischemia and reperfusion (Ghosh et al., 2000). Although the occlusion intervention used in the current study was effective in improving specific aspects of subsequent exercise performance, it is worth noting that other protocols may be equally, or more, effective in eliciting effects of intermittent ischemia. Also, due to

329 the study design we were unable to identify the specific time course and duration of
330 the effects observed.
331
332 Interestingly, although the small female sample size (n = 4) however makes it
333 difficult to draw any firm conclusions regarding gender differences associated with
334 the intervention, our data suggested that the observed beneficial effects of the
335 occlusion protocol on exercise appeared to be more pronounced in the male subjects.
336 While our participants would not be considered trained athletes, they were all
337 currently participating in competitive or semi-competitive sports (e.g. football, touch
338 rugby & volleyball) and the females were of a similar training level to the males. In
339 rats, it has been demonstrated that preconditioning required testosterone to increase
340 heat shock protein 70 synthesis, which mediated delayed onset cardioprotection in
341 the male via an androgen receptor-mediated mechanism (J. Liu et al., 2006). More
342 recently, testosterone has been shown to confer cardioprotection by upregulating the
343 α_1-adrenoceptors (Tsang et al., 2008). Thus, the greater effect of the occlusion
344 intervention on specific aspects of performance in males in our study could
345 speculatively be due to the presumably higher levels of testosterone.
346

347 **Conclusion**

348 The unilateral occlusive recovery intervention applied in the current study elicited
349 both positive and negative effects on specific aspects of neuromuscular function.
350 Importantly though, beneficial effects on functional measures of athletic
351 performance, including repeated sprint ability and jump height were observed 24
352 hours after the intervention. Although the mechanism is yet to be defined, improved
353 blood flow and enhanced efficiency of muscular oxygen utilisation associated with

cycles of lower limb occlusion and reperfusion, may have contributed to a more rapid return of muscle function.

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496 **Tables**

497 Table 1: Effect of Occlusion as a Recovery Intervention on Measures of Lower-limb
498 Function.

Difference in the Mean Change (%) (Intervention – Control) ± 90% CL

		Immediately Post - Pre		*24 h Post - Immediately Post*		*24 h Post - Pre*	
	Variable	%	Qualitative Outcome	%	Qualitative Outcome	%	Qualitative Outcome
Peak Values	CMJ (W)	-0.1 ± 8.1	unclear	-0.9 ± 7.4	unclear	-1.0 ± 11.2	unclear
	Squat Jump (W)	*-5.2 ± 8.5*	*possibly -ve*	***9.8 ± 12.6***	***likely +ve***	4.1 ± 13.9	unclear
	LP Max. Str. (kg)	0.7 ± 3.7	trivial	-1.8 ± 4.1	likely trivial	-1.1 ± 3.6	likely trivial
	LP Av. Str. (kg)	-0.6 ± 3.1	trivial	-1.0 ± 3.6	likely trivial	-1.6 ± 3.5	likely trivial
	LP Total Power (W)	-5.8 ± 8.9	possibly –ve	*10.4 ± 11.7*	*possibly +ve*	4.0 ± 7.9	possibly +ve
	LP Work (J)	1.3 ± 3.8	likely trivial	0.7 ± 2.8	trivial	2.0 ± 3.8	likely trivial
	LP Velocity ($m \cdot s^{-1}$)	0.0 ± 2.2	unclear	1.2 ± 1.8	likely trivial	1.1 ± 2.0	likely trivial
Mean Values CMJ	Con PP (W)	-1.9 ± 15.1	unclear	*4.9 ± 8.8*	*possibly +ve*	2.9 ± 17.7	unclear
	Ecc PP (W)	*-7.0 ± 12.8*	*possibly -ve*	11.6 ± 20.8	unclear	3.7 ± 16.2	unclear
	Con PV ($m \cdot s^{-1}$)	-1.0 ± 5.2	unclear	***4.2 ± 4.2***	***likely +ve***	3.2 ± 7.6	unclear
	Ecc PV ($m \cdot s^{-1}$)	***-32.4 ± 54.5***	***likely -ve***	***16.4 ± 11.4****	*possibly +ve*	-21.3 ± 47.0	unclear
	Con PA ($m \cdot s^{-1} \cdot s^{-1}$)	5.6 ± 23.9	unclear	1.6 ± 27.0	unclear	7.2 ± 12.3	unclear
	Ecc PA ($m \cdot s^{-1} \cdot s^{-1}$)	***-11.6 ± 11.6****	***likely -ve***	6.4 ± 10.4	unclear	-5.9 ± 17.3	unclear
	Con Work (J)	-3.9 ± 7.8	possibly -ve	2.3 ± 4.9	likely trivial	-1.7 ± 10.6	unclear
	Jump Height (cm)	0.7 ± 12.2	unclear	2.7 ± 12.2	unclear	3.5 ± 9.0	unclear
	Time to PP (s)	*-9.5 ± 15.1*	*possibly -ve*	8.8 ± 42.0	unclear	-1.5 ± 35.7	unclear
	Time to PV (s)	-4.7 ± 12.9	unclear	2.2 ± 15.3	unclear	-2.6 ± 21.2	unclear
Mean Values Squat Jump	Con PP (W)	-2.9 ± 11.6	unclear	8.0 ± 16.7	unclear	2.2 ± 18.1	unclear
	Ecc PP (W)	***-14.2 ± 17.6***	***likely -ve***	***38.5 ± 14.7****	***almost certainly +ve***	14.5 ± 24.9	unclear
	Con PV ($m \cdot s^{-1}$)	1.7 ± 3.9	unclear	-2.9 ± 5.5	unclear	-1.3 ± 4.5	unclear
	Ecc PV ($m \cdot s^{-1}$)	-8.1 ± 31.9	unclear	-2.7 ± 43.7	unclear	-10.6 ± 48	unclear
	Con PA ($m \cdot s^{-1} \cdot s^{-1}$)	5.7 ± 16.9	unclear	-0.4 ± 28.5	unclear	5.3 ± 30.8	unclear
	Ecc PA ($m \cdot s^{-1} \cdot s^{-1}$)	-4.6 ± 13.1	unclear	***28.1 ± 19.4****	***very likely +ve***	***22.1 ± 20.5***	***likely +ve***
	Con Work (J)	3.6 ± 4.8	possibly +ve	***-5.4 ± 2.9****	***likely -ve***	-2.0 ± 3.1	likely trivial
	Jump Height (cm)	***9.0 ± 9.1***	***likely +ve***	-2.5 ± 9.7	unclear	***6.9 ± 8.5***	***likely +ve***
	Time to PP (s)	5.6 ± 22.3	unclear	-2.6 ± 15.5	unclear	2.8 ± 18.2	unclear
	Time to PV (s)	0.8 ± 7.9	unclear	-2.5 ± 8.4	unclear	-1.6 ± 9.6	unclear

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501 PP: Peak Power; PV: Peak Velocity; PA: Peak Acceleration; LP: Leg Press; CMJ: Countermovement
502 Jump; Con: Concentric; Ecc: Eccentric *: $p < 0.05$; **: $p < 0.01$. Italics represent effects that are non-
503 trivial; Bold type indicates effects that are both likely and non-trivial.

Figure Captions

Figure One: Experimental Design.
*: Squat jump test, Δ: Counter movement jump test, ●: Dynamometer leg press test, □: 40 m sprint, Treatment: Occlusion cuff fitted unilaterally and inflated to 220 or 15 mmHg for two, three minute periods per leg in a cross-over experimental design, Arrows represent the recording of perceived exertion (Borg, 1997).

Figure Two: Comparison of the eccentric peak power and peak acceleration before, after, and 24 hours following the occlusion and control interventions when performing a squat jump test.
ES: Effect Size difference between the post & 24 h-post squat jump tests; *: $p = 0.033$; **: $p = 0.004$.

Figure Three: Comparison of the cumulative 10- and 40-m sprint times before, after, and 24 h following the occlusion and control interventions.
ES: Effect Size difference between the change in mean post & 24 h post sprint times. #: $p = 0.06$ difference in the change in the mean post to 24 h post sprint time. Inset A: Individual change in 40 m sprint times difference between occlusion intervention and control. Inset B: Individual change in 10 m sprint times difference between occlusion intervention and control. Dashed lines in inserts represent female participants.