The Influence of Exercise Load and Blood Flow Restriction on the Recovery of Neuromuscular Strength following Resistance Exercise

Charlie Davids[1,5], Truls Raastad[3], Glen Lichtwark[1], Jeff Coombes[1], Jonathan Peake[4,5] and Llion Roberts[2,5]

[1]*School of Human Movement and Nutrition Sciences, The University of Queensland, Brisbane, Australia*
[2]*Griffith Sports Physiology and Performance, School of Allied Health Sciences, Griffith University, Southport, Australia*
[3]*Department of Physical Performance, Norwegian School of Sport Sciences, Oslo, Norway*
[4]*School of Biomedical Sciences, Queensland University of Technology, Brisbane, Australia*
[5]*Queensland Academy of Sport, Nathan, Queensland, Australia*

1 OBJECTIVES

Due to the diverse demands in many sports, athletes are required to simultaneously develop multiple facets of physical fitness. This often requires multiple training sessions within short timeframes, meaning recovery between sessions is of fundamental importance to promote optimal performance during training. Such effects are only exacerbated in the competitive season when performance during games becomes the key priority. However, traditional high load resistance exercise (HL-RE), which is the current gold standard for enhancing skeletal muscle adaptations, is accompanied by high levels of mechanical stress (Schoenfeld, 2010). Mechanical stress can impair muscular performance in the hours and days following training, impacting subsequent training sessions, and competitive performance (Doma, 2017). Consequently, many coaches reduce volume and intensity during the season to mitigate these mechanical stresses, but this approach may lead to suboptimal stimuli for skeletal muscle adaptation.

A solution to this issue is the combination of blood flow restriction with low load resistance exercise (LL-BFR), which has been demonstrated to produce significant increases in skeletal muscle hypertrophy, strength and endurance (Clark, 2011; Kacin and Strazar, 2011). This is achieved with reduced mechanical stress, as often loads ranging from 20-30% of one-repetition maximum (1RM) are used. Restriction of blood, and ultimately oxygen, to the exercising muscle results in greater metabolic stress, which appears to compensate for the lack of mechanical stress. Despite this change in stimulus from mechanical to predominantly metabolic, it appears that LL-BFR is still capable of producing robust hypertrophic and strength gains that are comparable to HL-RE, even in athletic populations (Luebbers et al., 2017). Importantly, these adaptations seem to occur with much less training volume load (load x sets x reps), enhancing training efficiency and minimising stress to connective tissues. However, less is known of the acute recovery from LL-BFR, and whether the shift in stimulus (from mechanical to metabolic) observed with this type of exercise leads to a hastened recovery of muscle performance. This possibility is supported by the absence of muscle damage that has been reported with LL-BFR (Loenneke, 2014). Knowledge of the timeline of recovery from LL-BFR is necessary to understand the exercise-adaptation cycle of this innovative mode of exercise, so an optimal balance of maximising adaptations while still allowing sufficient recovery periods, can be achieved.

The majority of previous studies have assessed neuromuscular performance immediately following LL-BFR, which appears to be impaired to a similar extent as HL-RE (Cook, 2013; Loenneke, 2015). However, neuromuscular performance needs to be evaluated further into the post-exercise period to establish an acute timeline of fatigue and recovery. Husmann (2017) demonstrated that neuromuscular performance is significantly impaired immediately following LL-BFR. However, performance improves drastically within 8 minutes upon reperfusion of the exercising muscles, perhaps indicating that acute strength impairment is a result of peripheral fatigue caused by metabolite accumulation, as opposed to central factors. It is important to acknowledge that strength did not completely recover to pre-exercise levels. Indeed, Loenneke (2013) reported these levels were still not regained with LL-BFR at 60 minutes post-exercise. However, it is not clear how these effects compare to the use of HL-RE, and whether there is a difference in the origin of fatigue (be it

Davids, C., Raastad, T., Lichtwark, G., Coombes, J., Peake, J. and Roberts, L.
The Influence of Exercise Load and Blood Flow Restriction on the Recovery of Neuromuscular Strength following Resistance Exercise.
In *Extended Abstracts (icSPORTS 2018)*, pages 19-23

central or peripheral) observed after both exercise protocols.

Therefore, the aim of the present study was to compare how low body neuromuscular performance was influenced 60 minutes after lower body resistance exercise is influenced by blood flow restriction (BFR) protocol type, and exercise load. It was hypothesised that although the addition of BFR to low load exercise would enhance strength decrements following exercise, such effects would still be reduced in comparison to high load exercise.

2 METHODS

Participants.
Twelve healthy resistance-trained males (mean ± standard deviation; age: 22.3 ± 3.2 years; height: 182.1 ± 6.3cm; body mass: 84.1 ± 9.0kg) volunteered to participate in the study. All participants had been resistance training continuously for a minimum of two years leading up to the trials as an attempt to translate the findings to athletic cohorts. This study has been approved by the Human Research Ethics Committee at The University of Queensland.

Experimental Design.
A randomised within-participants repeated measures experimental design was used to assess the neuromuscular responses to different BFR protocols and exercise intensities. After baseline and familiarisation visits, participants attended the laboratory on four occasions, separated by a minimum of 5 days to complete four experimental trials in a randomised manner. The conditions were: (a) low load resistance exercise (LL, 30%1RM); (b) LL with continuous blood flow restriction (LL-CBFR); (c) LL with intermittent blood flow restriction (LL-IBFR); (d) high load resistance exercise (HL, 70%1RM).

Baseline Visits.
Participants first had their arterial occlusion pressure (AOP) determined via Doppler ultrasound (uSmart3300, Terason, USA) of the posterior tibial artery, as previously advocated by Loenneke et al., (2015). Participants then completed the baseline strength testing on the isokinetic dynamometer as described below. On a separate visit, participants performed their 1RM squat, and completed a familiarisation of the LL-CBFR condition, as this has been demonstrated to be the most challenging (Brandner and Warmington, 2017).

Experimental Trials.
Each experimental visit began with a lower body exercise session. The session consisted of 4 sets of barbell squat exercise, with 2 minutes of seated inter-set rest in between. For all LL conditions, the first set consisted of 30 repetitions, following by three sets of 15 repetitions. For the HL condition, 4 sets of 10 repetitions were completed. Following the final set of exercise, participants remained seated for 60 minutes.

Blood Flow Restriction Protocol.
For both CBFR and IBFR trials, participants had a pair of 8cm-wide nylon pneumatic cuffs placed around the proximal thigh. The cuffs were inflated to 60% of the ultrasound determined AOP immediately prior to the first set of exercise using a rapid cuff inflator (E20, Hokanson, Bellevue, WA). In the CBFR trial, the cuffs remained inflated until the final set of exercise was completed, whereas during the IBFR trial, the cuffs were deflated following each set, and re-inflated immediately prior to beginning the next set.

Neuromuscular Strength Assessment.
During the baseline visit, and 60 minutes following exercise during each of the experimental trials, participants completed a series of maximal isometric contractions on an isokinetic dynamometer (Biodex 3, Biodex Medical Systems, USA). Prior to being seated in the dynamometer, participants had reusable stimulation electrodes (50mm x 90mm; Metron, Patterson, UK) placed over the femoral nerve. The cathode electrode was placed just below the inguinal fold on the anterior groin, with the anode electrode placed underneath the gluteal fold on the posterior thigh. Participants were then seated with a 55-degree hip angle, with their dominant leg strapped to the lever arm of the machine. The lever arm was fixed at an angle corresponding to 70 degrees of knee flexion (full knee extension defined as 0 degrees of flexion). Following three warm-up submaximal voluntary contractions of the knee extensors, participants performed three 5 second maximal voluntary contractions, each separated by 120 seconds. Participants were instructed to apply force as rapid and as hard as possible for the entire 5 seconds. The peak torque value generated during the best contraction was recorded as the maximal voluntary torque (MVT). The rate of torque development was also calculated by determining the time taken to reach 50% (TPT50) and 90% (TPT90) this peak torque value.

Following these voluntary contractions, involuntary activation of the knee extensors was

achieved via supramaximal stimulation of the femoral nerve through the stimulation electrodes, connected to a Digitimer DS7AH (Digitimer Ltd, Welwyn Garden City, Hertfordshire, UK). Participants performed an additional three maximal voluntary contractions, during which the knee extensors were maximally stimulated, with another maximal stimulation to the resting muscle following approximately 3 seconds after the contraction. Utilising the interpolated twitch technique, voluntary activation of the quadriceps was determined, as well as evoked twitch torque from the resting stimulation.

Statistical Analysis.

Data were initially checked for normality using a Shapiro-Wilk test. Repeated measures two-way ANOVAs were then used to compare differences between trials and time points (baseline vs 1-hour post-exercise). Significant main effects of time, condition, or interaction were followed by post-hoc repeated measures t-tests, with Bonferroni's multiple comparisons correction. Effect sizes (Cohen's d) were also calculated to provide magnitude-based inferences. Effect sizes were assessed as 0.2 = small effect, 0.5 = moderate effect, and ≥0.8 = large effect. Statistical significance levels were accepted at $p<0.05$.

3 RESULTS

There was a significant time x trial interaction for MVT ($p=0.03$). Post hoc analyses revealed significant time interactions for HL ($p<0.01$; -8.77%; ES=0.56) and CBFR conditions ($p<0.01$; -5.90%; ES=0.36), while both LL and IBFR showed no significant change from baseline (Figure 1). Significant condition interactions were also found between HL and LL ($p<0.01$; ES=0.40), and HL and IBFR conditions ($p=0.02$; ES=0.23) with no other interactions between conditions reported (Figure 1A).

There was a significant time x trial interaction for evoked twitch torque ($p<0.01$). Post hoc analyses revealed significant time interactions for HL ($p<0.01$; -18.75%; ES=1.51), CBFR ($p=0.01$; -5.96%; ES=0.47) and LL ($p<0.01$; -6.79%; ES=0.53) conditions, while no change from baseline was reported for IBFR. Significant trial interactions were also found between HL and each of the other conditions ($p<0.01$ for all, Figure 1B).

No significant change in voluntary activation of the knee extensors was observed in any of the conditions ($p=0.40$; Table 1). There were significant time interactions for TPT50 with LL, CBFR and IBFR conditions being higher than baseline, but no between condition interactions were found (Table 1). For TPT90, there were significant time interactions for HL and CBFR, and a significant condition interaction, with CBFR being significantly different from LL and IBFR conditions (Table 1).

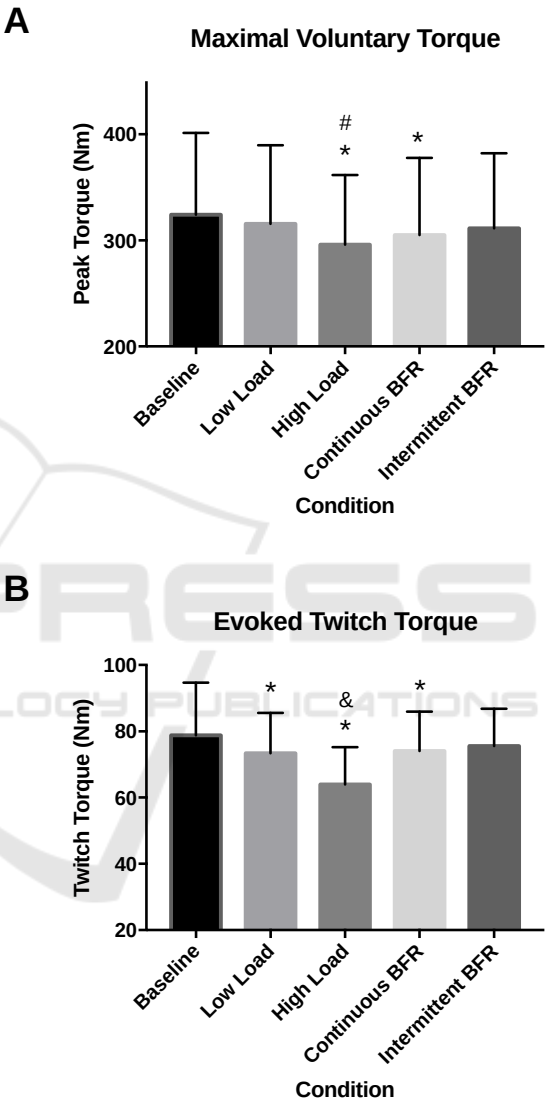

Figure 1: (A) maximal voluntary torque and (B) evoked twitch torque of the knee extensors. Black bars represent baseline values. *indicates a significant difference from baseline ($p<0.05$). #indicates a significant difference from the low load (LL) and intermittent BFR (IBFR) condition ($p<0.05$). &indicates significant different all other conditions ($p<0.05$).

Table 1: Voluntary activation, time to 50% peak torque (TPT50), and time to 90% peak torque (TPT90). *indicates a significant difference from baseline ($p<0.05$). **indicates a significant difference between LL and IBFR conditions.

	BAS	LL	HL	CBFR	IBFR
Voluntary Activation (%)	94.42±5.7	96.63±3.7	97.94±2.2	95.84±4.5	97.21±3.1
TPT50 (ms)	0.09±0.01	0.13±0.06*	0.12±0.03	0.14±0.07*	0.15±0.09*
TPT90 (ms)	0.71±0.35	0.85±0.31	0.96±0.32*	1.10±0.36**	0.86±0.27

4 DISCUSSION

The present study compared the decrements in neuromuscular performance between different lower body exercise protocols, varying in exercise intensity, and blood flow restriction application. The primary findings of the present study indicated that at 60 minutes post-exercise: (i) compared to baseline levels, MVT was significantly impaired following only HL and CBFR conditions, whereas there were no differences from baseline following LL or IBFR conditions; (ii) compared to baseline levels, evoked twitch torque was significantly impaired following HL, CBFR and LL conditions, with no change after IBFR; and (iii) there were no changes in central activation of the knee extensors in any of the conditions compared to baseline levels. These findings partially supported the hypothesis. Although both CBFR and HL exercise resulted in significant neuromuscular performance impairment at 60 minutes post-exercise, there were no significant differences between conditions.

Previous studies examining the influence of blood flow restricted exercise on neuromuscular performance have often assessed this effect immediately post-exercise. It remains unclear how performance is recovered acutely in the hours following exercise. Immediately post-exercise, it appears that the combination of BFR with low-load exercise tends to exacerbate the magnitude of fatigue, and that this effect occurs due to contractile perturbations caused my metabolite accumulation (Husmann, 2017). This effect tends to remain at 1 hour following exercise, with strength performance recovering to baseline levels in the unrestricted condition (Loenneke, 2015). This outcome aligns with the results of the present study. Maximal voluntary torque remained significantly reduced at 60 minutes post-exercise following CBFR, whereas strength recovered to baseline levels in both LL and IBFR conditions. It is likely that the larger degree of metabolic stress experienced in the CBFR condition caused greater perturbations within the skeletal muscle, impairing the contractile function. Interestingly, despite the additional exercise volume completed in the HL condition (volume-load = load x sets x reps; HL: 3941.6 ± 485kg; LL, IBFR, CBFR: 3171.9 ± 370kg), MVT remained impaired at 60 minutes post-exercise in both HL and CBFR conditions, with no differences between them. This finding suggests that the restriction of oxygen to the working muscles during exercise and rest periods that occurs with CBFR, leads to metabolic perturbations within the skeletal muscle that match those of higher loads and higher volumes of exercise.

This explanation is further supported by the reduction in evoked twitch torque, and increase is TPT90 that was observed in the present study for HL and CBFR conditions. Evoked twitches consisted of supramaximal stimulations being delivered to the knee extensors, meaning the reduction in torque after exercise is due to factors distal to the neuromuscular junction. This observation adds weight to the claim that the fatigue observed in the present study is of peripheral origin and is related to metabolite accumulation. Further support for this idea was provided by Suga (2012), who observed metabolic stress (indicated by inorganic phosphate accumulation and pH decline) to increase over the course of four sets of exercise to match levels seen with HL exercise. While acute impairments in neuromuscular performance are not a valid indicator of chronic hypertrophy, they do tend to align with the results of chronic studies which report similar hypertrophy between CBFR and HL conditions, with inferior hypertrophy in load-matched unrestricted conditions. This possibility could suggest that CBFR may be used as a tool to achieve similar hypertrophy as HL training, despite a marked reduction in training volume, although chronic training studies are required for confirmation.

The lack of change in voluntary activation of the knee extensors found in the present study aligns with previous findings. While Husmann (2017) found central activation to be reduced immediately after the

fourth set of LL-BFR, this effect rapidly recovered upon reperfusion at 2 minutes post-exercise. Further, Cook (2013) found no change in central activation post-exercise between HL, LL-BFR or LL conditions. This outcome would explain the lack of change seen at 60 minutes post-exercise in the present study. Together, with the results mentioned previously, evidence suggests that the decrement in neuromuscular performance observed in the present study is due to peripheral fatigue, as opposed to central factors.

In conclusion, HL and CBFR squat exercise appears to impair neuromuscular performance to a similar extent at 1-hour post-exercise despite the reduced mechanical stress and total training volume completed in the CBFR condition. The impairment in performance was due to peripheral factors as voluntary activation of the knee extensors remained unchanged following exercise. Further research should seek to extend the timeline of neuromuscular performance recovery past 60 minutes to determine if differences exist between HL and CBFR. Furthermore, whether the equivalent acute neuromuscular responses between HL and CBFR exercise translate to similar chronic hypertrophic changes should be evaluated, as LL-BFR training may serve as a strategy to manage total training stress and chronic fatigue during busy periods of training and competition.

ACKNOWLEDGEMENTS

This work is supported by the Queensland Academy of Sport's Sport Performance Innovation and Knowledge Excellence unit.

REFERENCES

Brandner, C. R., & Warmington, S. A. (2017). Delayed onset muscle soreness and perceived exertion following blood flow restriction exercise. *J Strength Cond Res*. doi:10.1519/jsc.0000000000001779.

Clark, B. C., Manini, T. M., Hoffman, R. L., Williams, P. S., Guiler, M. K., Knutson, M. J., Kushnick, M. R. (2011). Relative safety of 4 weeks of blood flow-restricted resistance exercise in young, healthy adults. *Scand J Med Sci Sports, 21*(5), 653-662. doi:10.1111/j.1600-0838.2010.01100.x.

Cook, S. B., Murphy, B. G., & Labarbera, K. E. (2013). Neuromuscular function after a bout of low-load blood flow-restricted exercise. *Med Sci Sports Exerc, 45*(1), 67-74. doi:10.1249/MSS.0b013e31826c6fa8.

Doma, K., Deakin, G. B., & Bentley, D. J. (2017). Implications of Impaired Endurance Performance following Single Bouts of Resistance Training: An Alternate Concurrent Training Perspective. *Sports Med, 47*(11), 2187-2200. doi:10.1007/s40279-017-0758-3.

Husmann, F., Mittlmeier, T., Bruhn, S., Zschorlich, V., & Behrens, M. (2017). Impact of Blood Flow Restriction Exercise on Muscle Fatigue Development and Recovery. *Med Sci Sports Exerc*. doi:10.1249/mss.0000000000001475.

Kacin, A., & Strazar, K. (2011). Frequent low-load ischemic resistance exercise to failure enhances muscle oxygen delivery and endurance capacity. *Scand J Med Sci Sports, 21*(6), e231-241. doi:10.1111/j.1600-0838.2010.01260.x.

Loenneke, J. P., Allen, K. M., Mouser, J. G., Thiebaud, R. S., Kim, D., Abe, T., & Bemben, M. G. (2015). Blood flow restriction in the upper and lower limbs is predicted by limb circumference and systolic blood pressure. *Eur J Appl Physiol, 115*(2), 397-405. doi:10.1007/s00421-014-3030.

Loenneke, J. P., Thiebaud, R. S., & Abe, T. (2014). Does blood flow restriction result in skeletal muscle damage? A critical review of available evidence. *Scand J Med Sci Sports, 24*(6), e415-422. doi:10.1111/sms.12210.

Loenneke, J. P., Thiebaud, R. S., Fahs, C. A., Rossow, L. M., Abe, T., & Bemben, M. G. (2013). Blood flow restriction does not result in prolonged decrements in torque. *Eur J Appl Physiol, 113*(4), 923-931. doi:10.1007/s00421-012-2502.

Luebbers, P. E., Witte, E. V., & Oshel, J. Q. (2017). The Effects Of Practical Blood Flow Restriction Training On Adolescent Lower Body Strength. *J Strength Cond Res*. doi:10.1519/jsc.0000000000002302.

Schoenfeld, B. J. (2010). The mechanisms of muscle hypertrophy and their application to resistance training. *J Strength Cond Res, 24*(10), 2857-2872. doi:10.1519/JSC.0b013e3181e840f3.

Suga, T., Okita, K., Takada, S., Omokawa, M., Kadoguchi, T., Yokota, T., Tsutsui, H. (2012). Effect of multiple set on intramuscular metabolic stress during low-intensity resistance exercise with blood flow restriction. *Eur J Appl Physiol, 112*(11), 3915-3920. doi:10.1007/s00421-012-2377-x.