Journal of Sports Sciences

ISSN: 0264-0414 (Print) 1466-447X (Online) Journal homepage: https://www.tandfonline.com/loi/rjsp20

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To cite this article: Colin Evans , Steven Vance & Maggie Brown (2010) Short-term resistance training with blood flow restriction enhances microvascular filtration capacity of human calf muscles, Journal of Sports Sciences, 28:9, 999-1007, DOI: 10.1080/02640414.2010.485647

To link to this article: https://doi.org/10.1080/02640414.2010.485647

Published online: 11 Jun 2010.

Journal of Sports Sciences, July 2010; 28(9): 999–1007

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Short-term resistance training with blood flow restriction enhances microvascular filtration capacity of human calf muscles

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(Accepted 12 April 2010)

Abstract
Resistance training increases muscle strength and endurance but may require high intensity and long duration to enhance capillarity. Vascular occlusion during low-load resistance training augments the strength and endurance gains compared with low-load resistance training alone, but in this study we investigated whether it also promotes microvascular filtration capacity, an index of capillarity. Nine healthy males performed short-term low-intensity resistance training of the calf muscles (four sets of 50 heel raises, three times a week for 4 weeks) under restricted (thigh cuff inflated to 150 mmHg on the non-dominant leg) or unrestricted (dominant leg without thigh cuff) blood flow conditions. Before and after resistance training, calf filtration capacity and resting blood flow were assessed by strain gauge plethysmography, and calf muscle strength and fatigue were assessed respectively by maximal voluntary contraction and force decline during electrically evoked ischaemic contractions in both legs. Calf filtration capacity increased by 26% in the restricted leg but did not increase significantly in the unrestricted leg. Calf muscle strength was 18% greater in the restricted leg but unchanged in the unrestricted leg. Calf muscle fatigue and resting blood flow did not change in either leg. Resistance training promoted microvascular filtration capacity, an effect that was somewhat enhanced by blood flow restriction, and could be due to increased capillarization.

Keywords: *Microvascular filtration, ischaemia, capillarization, angiogenesis*

Introduction

It is well documented that high-intensity resistance training [$\sim$70–100% of one-repetition maximum (1-RM)] leads to gains in muscle strength (Fry, 2004). When high-intensity resistance training is contra-indicated, such as in cases of injury or disease, low-intensity resistance training in combination with vascular occlusion has been proposed as an alternative. Several studies have shown that low-intensity resistance training ($\leq$50% 1-RM) combined with vascular occlusion leads to similar increases in muscle strength and size compared with high-intensity resistance training alone (reviewed by Wernbom, Augustsson, & Raastad, 2008).

Most studies demonstrating that vascular occlusion during low-intensity resistance training enhances strength gains have lasted 8 weeks or longer (Madarame et al., 2008; Moore et al., 2004; Sumide, Sakuraba, Sawaki, Ohmura, & Tamura, 2009; Takarada et al., 2000; Takarada, Sato, & Ishii, 2002; Takarada, Tsuruta, & Ishii, 2004), although improvements have been shown after as little as 2 weeks (Shinohara, Kouzaki, Yoshihisa, & Fukunaga, 1998). However, in the study of Shinohara et al. (1998), the gains in maximal strength after 4 weeks (26% increase) appeared to be greater than after 2 weeks of training (9% increase). Taken together, these findings suggest that both duration of the training programme and the number of training sessions completed are important in determining the magnitude of adaptation.

The mechanisms responsible for training-induced increases in muscle strength with blood-flow-restricted resistance exercise remain unclear, but muscle hypertrophy and neural adaptations are the most likely pathways. For example, vascular occlusion during low-intensity resistance training led to increases in muscle cross-sectional area (Madarame et al., 2008; Takarada et al., 2000, 2002, 2004) and electromyography (EMG) activity (Moore et al., 2004).

There is also evidence that vascular occlusion combined with low-intensity resistance training leads to greater muscular endurance than low-intensity resistance training alone (Sumide et al., 2009; Takarada et al., 2000, 2002). Several metabolic changes that could act as a stimulus for the vascular occlusion-induced increases in muscle strength and endurance following low-intensity resistance training

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ISSN 0264-0414 print/ISSN 1466-447X online © 2010 Taylor & Francis
DOI: 10.1080/02640414.2010.485647

have been suggested. They include increased concentrations of lactate (Reeves et al., 2006), growth hormones (Reeves et al., 2006; Takarada et al., 2000), and inorganic phosphate (Greiner et al., 2007), and decreased concentrations of phosphocreatine and pH (Greiner et al., 2007).

Improved endurance in a trained muscle could be related to expansion of the capillary network, which facilitates oxygen delivery. High-intensity resistance training without vascular occlusion led to increases in the levels of mRNA for vascular endothelial growth factor (VEGF) (Gavin, Drew, Kubik, Pofahl, & Hickner, 2007), which is a potent contributor to microvascular growth. Despite this, increases in muscle capillarity after resistance training seldom match those seen in response to endurance exercise. The increase in the number of capillary contacts per muscle fibre observed after 12 weeks of high-intensity resistance training was delayed compared with strength gains and simply maintained capillary supply in line with muscle fibre growth (McCall, Byrnes, Dickinson, Pattany, & Fleck, 1996).

The main stimuli for increasing skeletal muscle capillarization are metabolic changes such as hypoxia, mechanical stretch or shear stress, and increases in the concentrations of growth factors such as VEGF (Hudlicka & Brown, 2009). Blood flow restriction during endurance training enhances several of these, including severity of hypoxia and expression of VEGF and peroxisome proliferator-activated receptor gamma coactivator (PGC) 1α (Gustafsson, Puntschart, Kaijser, Jansson, & Sundberg, 1999; Norrbom et al., 2004). These findings suggest that vascular occlusion may augment conditions for capillary growth during low-intensity resistance training and, in support of this notion, ischaemic strength training of rat skeletal muscle increased the capillary–fibre ratio more than non-ischaemic training (Suzuki, Kobayashi, Uruma, & Koyama, 2000). In human skeletal muscle, however, the effect of vascular occlusion during low-intensity resistance training on muscle capillary supply is unknown.

The primary aim of the current study was to investigate the effect of vascular occlusion on calf filtration capacity, a validated index of capillarity (Brown, Jeal, Bryant, & Gamble, 2001; Charles et al., 2006), during short-term low-intensity resistance training of the human triceps surae. A secondary aim was to investigate the effect of vascular occlusion on muscle strength and endurance during short-term low-intensity resistance training of the human triceps surae, as an indicator of training efficacy. It was hypothesized that low-intensity resistance training under vascular occlusion would lead to greater muscle capillarity, strength, and endurance compared with low-intensity resistance training alone.

Materials and methods

Experimental design

Participants performed short-term (4 weeks) low-intensity resistance training of the triceps surae under restricted (non-dominant leg) or unrestricted (dominant leg) blood flow conditions. To examine possible training-induced changes, tests of isometric plantar flexion strength and fatigue, and calf filtration capacity, were performed on both legs, before and after the training period. A subset of three participants was tested for calf filtration capacity after 2 weeks of training to monitor the time-course of changes in capillarity. The study was approved by the University of Birmingham local ethics committee.

Participants

Nine recreationally active young male participants from the student population of the University of Birmingham who were not undertaking any resistance training volunteered to participate in the study. All were free from cardiovascular disease and recent lower limb injury and provided informed written consent. The validated 7-Day Physical Activity Recall questionnaire was administered to estimate caloric expenditure as an indicator of physical activity level (Richardson, Ainsworth, Jacobs, & Leon, 2001). Participants performed a familiarization trial before the experimental protocol to accustom themselves to all testing and training procedures and testing was carried out at 09.00 h following an overnight fast.

Calf plantar flexion strength test

Participants were seated upright and barefoot in a leg dynamometer. The knee and ankle joint was positioned at 85° with the thigh horizontal, and a force transducer was clamped above the knee. Participants were instructed to perform a maximal voluntary isometric contraction of the triceps surae lasting approximately 2 s without Valsalva's manoeuvre (Davies, Mecrow, & White, 1982). Force was transmitted, amplified, and displayed on a personal computer running Spike2 software, via a CED 1401 data logger (Cambridge Electronic Design Ltd., Cambridge, UK). The best of three attempts was taken as maximal voluntary contraction force.

Fatigue test

Fatigue resistance of the triceps surae was tested using an electrically evoked plantar flexion fatigue test performed under ischaemic conditions as described previously (Fisher & White, 1999). Ischaemic endurance was used as an indirect evaluation of

muscle metabolic properties and as an indicator of training efficacy. Participants were positioned as in the strength test with two foil-in-tissue electrodes attached above and below the belly of the calf muscle. Maximal tetanic tension was achieved by stimulating the muscle at 20 Hz, 50-μs pulse width, in 300-ms trains, using incremental increases in stimulus current intensity until contraction force reached a plateau. The stimulus current was then lowered to the level that produced 60% of maximal tetanic tension. The fatigue protocol consisted of trains of stimuli at 20 Hz lasting 300 ms repeated once every second for 2 min. Blood flow was occluded throughout the test by inflating a thigh cuff (15 cm wide) to 200 mmHg, sufficient to prevent flow to the calf in the seated position (Cole & Brown, 2000). Fatigue index was calculated as the relative decline in force at 30, 60, 90, and 120 s from the initial force at time 0.

Measurement of calf filtration capacity and resting blood flow

Calf filtration capacity, resting blood flow, venous pressure, and isovolumetric venous pressure were assessed using strain gauge plethysmography as previously described (Christ et al., 2000; Gamble, Gartside, & Christ, 1993). Plethysmography traces were analysed in a blinded fashion. Participants lay supine for at least 10 min before the test protocol commenced and ambient temperature was recorded continuously throughout (23–25°C). Both legs were supported on a vacuum mattress with the mid-calf level with respect to the right atrium (i.e. one-third of the distance from sternal angle to the surface of the supporting bench). Mean arterial pressure was recorded at the brachial artery before and after the protocol for calf filtration capacity and resting blood flow assessment using an automated sphygmomanometer (Vital Signs Monitor 8100, Critikon Dinamap, FL, USA). Calf filtration capacity and resting blood flow were assessed bilaterally using a validated mercury-free strain gauge with an integrated automatic calibration device (Filtrass 2001, Medizintechnik, Germany) (Christ et al., 2000). Electromagnetic strain gauges were placed around the belly of each calf and sensors that detect changes in calf circumference were attached to the anterior side of both legs. Congestion cuffs were fitted bilaterally at mid-thigh level and pressure was applied by the Filtrass in-built pressure module. Signals from the strain gauges and pressure cuffs were amplified onto a computer monitor running Filtrass software. Resting blood flow was estimated by measuring the first 3–5 s of the calf volume response to a rapid inflation of the thigh cuffs to 50 mmHg. Cuffs were inflated three times with 20 s rest between inflations and a mean resting blood flow was taken as the average of these ($ml \cdot 100\ ml\ tissue^{-1} \cdot min^{-1}$).

Calf filtration capacity was assessed when calf volume had returned to baseline by applying cumulative pressure steps of 8 mmHg, maintained for 4 min each without exceeding diastolic pressure. Once isovolumetric venous pressure (the balance between hydrostatic and oncotic pressure at the microvascular interface) was exceeded, there was a two-phase increase in calf volume (Figure 1). The initial rapid increase is due to vascular filling and the slower gradual increase in volume from minute 2 onwards is caused by fluid filtration (Christ et al., 2000). Estimated venous pressure was calculated by extrapolating the vascular filling volume against cuff pressure relationship to zero cuff pressure, a method validated against actual venous pressure from catheter measurements (Christ, Gamble, Baschnegger, & Gartside, 1997). Calf filtration capacity ($ml \cdot min^{-1} \cdot mmHg^{-1} \cdot 100\ ml\ \ tissue^{-1} \times 10^{-3}$) was calculated from the linear relationship between the rate of the slow calf volume increment and thigh cuff pressure (Gamble et al., 1993).

Training protocol

Participants continued with their normal recreational activity throughout the strength training protocol, which was similar to that described by Fisher and White (1999). Participants trained three times per week for a period of 4 weeks. Training sessions consisted of four sets of 50 heel raises with 1 min rest between sets. Heel raises were performed at a rate of one heel rise and lower per second by following a digital metronome set at 60 beats $\cdot$ min^{-1}. When the rate could not be sustained or participants could not heel raise maximally (as judged by failure of the top of the head to reach a previously determined maximal height), the exercise was stopped and the number of repetitions noted. Once four sets of 50 heel raises could be completed, participants exercised carrying a load of 10% body mass. Subsequent additions of 5% body mass were made on successful completion of four sets of 50 heel raises. During training, both legs performed the same number of repetitions at the same load but blood flow to one leg was restricted in all participants with a thigh cuff (15 cm wide) inflated to 150 mmHg, which was deflated between sets. This pressure induced a level of ischaemia similar to patients with mild peripheral arterial disease – that is, an ankle-brachial pressure index of between 0.51 and 0.95 (classified as Fontaine grade II; Ubbink, Jacobs, Tangelder, Slaaf, & Reneman, 1994). Brachial systolic blood pressure in our participants ranged between 111 and 133 mmHg. For ankle brachial pressure index to fall between 0.51 and 0.95 in all participants when

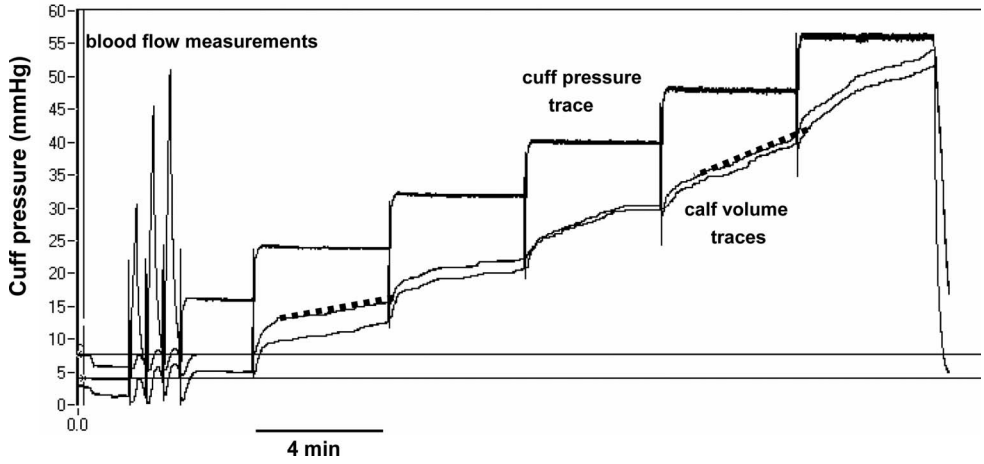

Figure 1. Representative recording showing cuff pressure and bilateral calf volume increases during the protocol for assessment of resting blood flow and calf filtration capacity. Three resting blood flow measurements at the start of the recording are followed by six stepped increases in cuff pressure of 8 mmHg. Dotted lines illustrate rate of calf volume increase due to filtration (ml · 100 ml^{-1} · min^{-1}) for two of these steps. Calf filtration capacity is derived from the slope of these plotted against cuff pressure for all steps where filtration occurs.

supine, a thigh occlusion pressure of 67–105 mmHg was required (Cole & Brown, 2000). To correct for the effects of gravity when standing, 0.776 mmHg (hydrostatic load; Gamble, Christ, & Garside, 1997) multiplied by the vertical distance from heart to thigh mid-point (approximately 60 cm) was added. The resulting thigh cuff pressure range required was 113–151 mmHg.

Statistical analysis

Data were analysed using SPSS for Windows release 13 (SPSS Inc., Chicago, IL, USA). Pre- and post-training maximal voluntary contraction, initial force produced in the fatigue test, force decline during the fatigue test at 120 s, calf filtration capacity, resting blood flow, venous pressure, and isovolumetric venous pressure were compared using two-way analysis of variance (ANOVA) (leg: restricted or unrestricted × test: before or after training) with repeated measures. Changes to calf filtration capacity, resting blood flow, venous pressure, and isovolumetric venous pressure after 2 and 4 weeks training were analysed using a two-way (leg: restricted or unrestricted × test: before, 2 weeks into, or after training) ANOVA with repeated measures for a subset of three participants. Significant *F*-ratios ($P < 0.05$) were followed by LSD pairwise comparisons to identify changes in legs and/or tests. Mauchly's test of sphericity was applied to the maximal voluntary contraction, initial force produced in the fatigue test, fatigue index at 120 s, calf filtration capacity, resting blood flow, venous pressure, and isovolumetric venous pressure data. Statistical significance was s et at $P < 0.05$. Data are presented as means ± standard errors unless otherwise stated.

Results

All nine participants successfully completed all training sessions and test procedures without injury. Participant demographics (mean ± standard error) were as follows: age, 19.8 ± 0.7 years; weight, 76.9 ± 7.8 kg; height, 1.78 ± 0.04 m; body mass index, 24.4 ± 3.2 kg · m^{-2}; caloric expenditure, 2916.1 ± 333.5 kcal · day^{-1}; brachial mean arterial pressure, 82.1 ± 6.9 mmHg.

Muscular strength

Before training commenced there was no difference in maximal voluntary contraction between the restricted (1340 ± 93 N) and unrestricted legs (1403 ± 114 N). Training did not affect maximal voluntary contraction of the unrestricted leg but caused an 18% increase in the restricted leg (1555 ± 72 N; $P < 0.05$) (Figure 2).

Muscular fatigue

Before training, initial forces produced during the fatigue test were similar between the restricted (455 ± 35 N) and unrestricted legs (459 ± 16 N). Likewise, there was no difference prior to training in fatigue index (decline in force at 120 s) between the restricted (0.40 ± 0.04) and unrestricted legs (0.47 ± 0.05). Training did not change initial force produced during the fatigue test or fatigue index at 120 s in either leg (Figure 3a, b).

Resting blood flow and venous pressure

Before training there was no difference between the restricted and unrestricted legs in resting blood flow

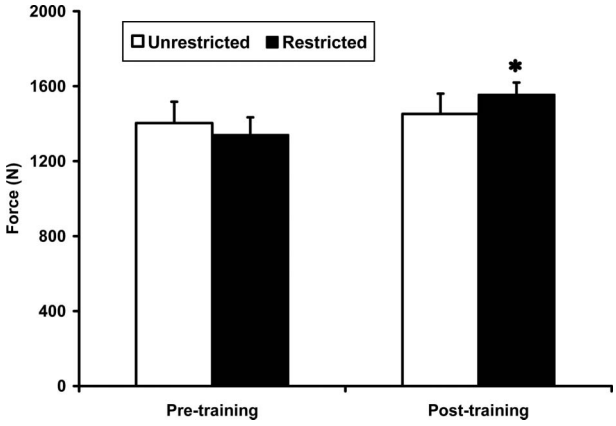

Figure 2. Maximum voluntary contraction of the triceps surae pre- and post-training under conditions of restricted (solid bars) or unrestricted (open bars) blood flow. Values shown are means ± standard errors ($n = 9$). Strength increased after training in the leg with restricted blood flow only ($^{*}P < 0.05$).

(3.2 ± 0.4 vs. 3.2 ± 0.4 ml · 100 ml tissue^{-1} · min^{-1}), venous pressure (15.2 ± 2.3 vs. 15.4 ± 2.5 mmHg), and isovolumetric venous pressure (16.0 ± 2.4 vs. 18.0 ± 2.9 mmHg). Training did not alter resting blood flow, venous pressure, or isovolumetric venous pressure in either the restricted or unrestricted legs (Table I).

Microvascular filtration capacity

Before training commenced there was no difference in calf filtration capacity between the restricted (3.64 ± 0.24 ml · min^{-1} · mmHg^{-1} · 100 ml tissue^{-1} × 10^{-3}) and unrestricted legs (4.02 ± 0.40 ml · min^{-1} · mmHg^{-1} · 100 ml tissue^{-1} × 10^{-3}). Training for 4 weeks caused a significant 26% increase in calf filtration capacity in the restricted leg (4.56 ± 0.30 ml · min^{-1} · mmHg^{-1} · 100 ml tissue^{-1} × 10^{-3}; $P < 0.05$) and a non-significant 23% increase in the unrestricted leg ($P = 0.06$) (Figure 4). Two-way ANOVA showed no significant interaction ($P > 0.05$) or difference between limbs ($P > 0.05$). In the subset of three participants, there was no difference in calf filtration capacity before training compared with after 2 weeks of training in either leg ($P > 0.05$) (Figure 5).

Discussion

This study is the first to show that vascular occlusion modestly enhances microvascular filtration capacity in response to short-term resistance training in human skeletal muscle. Calf filtration capacity increased by 26% in the triceps surae trained under conditions of restricted blood flow and by 23% in the leg trained under normal blood flow, although this change did not reach statistical significance. Calf filtration capacity is determined by Starling forces – that is, the surface area available for filtration and permeability per unit of surface area (Brown et al., 2001). There is a significant correlation between increases in Calf filtration capacity and the length of contact between capillaries and muscle fibres in older men following endurance training (Charles et al., 2006). This strong relationship between capillary surface area and calf filtration capacity therefore allows the latter to be used as an indirect measure of capillarization (Brown et al., 2001; Gamble et al., 2000), which has been augmented by vascular occlusion in resistance-trained rat calf muscle (Suzuki et al., 2000).

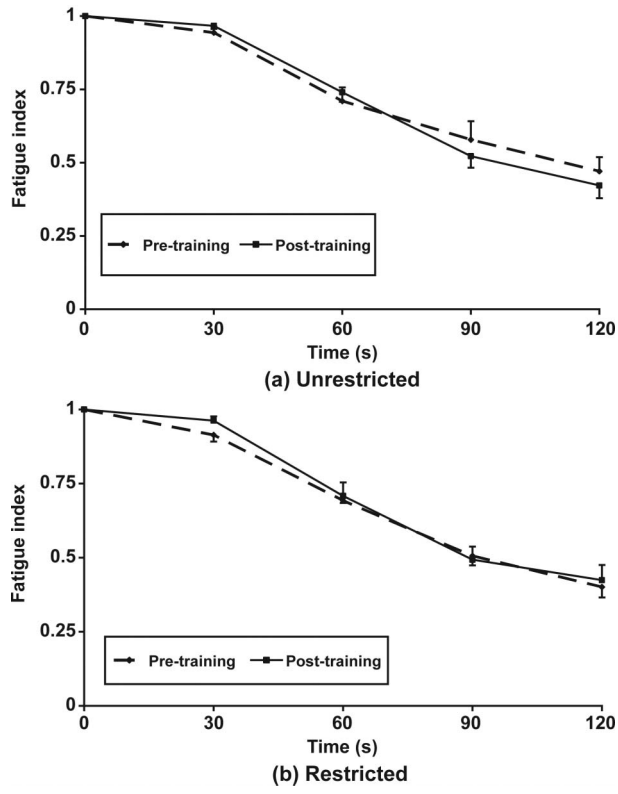

Figure 3. Fatigue test indices expressed as relative decline in force of the triceps surae during 2 min of electrically evoked contractions (60% of maximum tetanic force) performed under ischaemic conditions (thigh cuff occlusion). Data are means ± standard errors from legs trained under conditions of (a) unrestricted or (b) restricted blood flow ($n = 9$). Fatigue index at 120 s did not change after training in either leg. – –, pre-training; —, post-training.

Calf filtration capacity values before training in the current study were similar to those reported previously in healthy untrained young participants (Brown et al., 2001; Christ et al., 2000; Gamble et al., 1993, 1997). The increases observed (~20–25%) were smaller than that found in the study by Charles et al. (2006), but their participants were elderly men with a lower initial calf filtration capacity and were endurance trained for 14 weeks. They are, however, comparable with the difference in calf filtration capacity between control and strength-trained athletes (~20%) seen in a cross-sectional

Table I. Resting blood flow, venous pressure, and isovolumetric venous pressure before and after training in the restricted and unrestricted legs (mean ± standard error).

	Unrestricted		Restricted	
	Pre-training	Post-training	Pre-training	Post-training
Resting blood flow (ml · 100 ml tissue^{-1} · min^{-1})	3.2 ± 1.2	3.1 ± 1.7	3.2 ± 1.3	3.0 ± 1.1
Isovolumetric venous pressure (mmHg)	18.0 ± 8.6	20.9 ± 6.4	16.0 ± 7.1	20.1 ± 6.1
Venous pressure (mmHg)	15.4 ± 7.5	18.0 ± 5.1	15.2 ± 6.8	13.1 ± 6.0

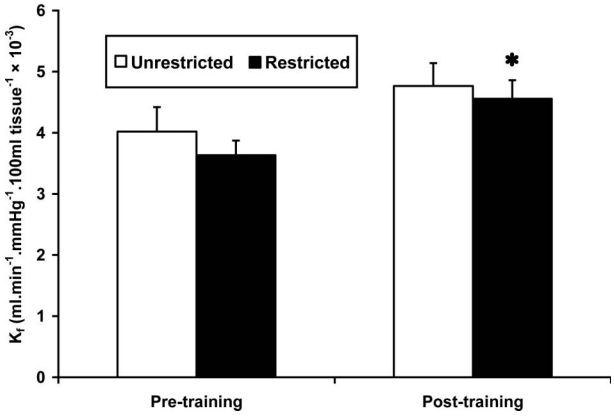

Figure 4. Calf filtration capacity (K_f) of the triceps surae pre- and post-training under conditions of restricted (solid bars) or unrestricted (open bars) blood flow. Values shown are means ± standard errors ($n = 9$). Calf filtration capacity increased after training in the leg with restricted blood flow only ($^{*}P < 0.05$).

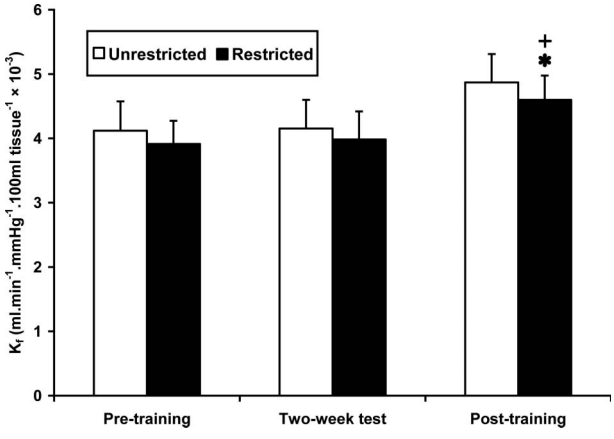

Figure 5. Calf filtration capacity (K_f) of the triceps surae pre-training, after 2 weeks of training, and post-training under conditions of restricted (solid bars) or unrestricted (open bars) blood flow. Values shown are means ± standard errors ($n = 3$). Calf filtration capacity increased after 4 weeks of training compared with pre-training and 2 weeks of training in the leg with restricted blood flow only (* and $^{+}P < 0.05$).

comparison by Brown et al. (2001). Charles et al. (2006) correlated their training-induced increases in calf filtration capacity with the morphometric gain in surface contact between capillaries and muscle fibres, indicating expansion of the microvascular supply. The capacity for this to occur rapidly in humans clearly exists, since increases in capillary-fibre ratio of 25–30% have been noted after as little as 4 weeks very high-intensity aerobic training (Jensen, Bangsbo, & Hellsten, 2004). The current results imply that 2–4 weeks of low-intensity resistance training combined with vascular occlusion is required to increase microvascular filtration capacity. Exercise *per se* appears to increase microvascular filtration capacity, which was slightly augmented by blood flow restriction.

Exercise-induced capillary growth can be stimulated by hypoxia, which up-regulates a variety of angiogenic growth factors and cytokines (Adair, Gay, & Montani, 1990). Hypoxic conditions up-regulate the expression of HIF1a, which leads to increases in VEGF expression and capillary growth (Hoppeler & Vogt, 2001). Endurance training increases the expression and activity of HIF1 (Gustafsson et al., 1999) and the expression of VEGF, changes that are augmented by conditions of restricted blood flow (Gustafsson et al., 1999; Hoppeler, 1999). The increases in HIF1 and VEGF expression in response to resistance training with partial occlusion are strongly correlated (Hoppeler, 1999; Wagner, 2001). Other angiogenic HIF target genes up-regulated by conditions of hypoxia include placental growth factor (PLGF), stromal cell-derived factor (SDF) 1, and adenosine (Adair, 2005). Vascular occlusion may therefore promote microvascular filtration capacity in response to resistance training via HIF1-mediated release of cytokines and growth factors such as VEGF.

The increase in calf filtration capacity in the leg trained under conditions of restricted blood flow may also be stimulated by reactive hyperaemia following thigh cuff release. Angiogenesis is facilitated by capillary shear stress, thus reactive hyperaemia may contribute to the increase in calf filtration capacity in response to resistance training with partial occlusion (Hudlicka & Brown, 2009; Suzuki et al., 2000).

Factors other than capillarization can also contribute to microvascular filtration capacity. Increases in calf filtration capacity may be due to increased vessel permeability, which occurs during capillary growth to enable the branching of new blood vessels from existing ones (Conway, Collen, & Carmeliet, 2001). Vessel permeability is increased by VEGF, for example, which is likely to be up-regulated during

ischaemic training. Lymph flow, inflammatory cell infiltration, and number of open capillaries could also affect microvascular filtration. Changes in capillarity could be confirmed morphologically by immunostaining of muscle biopsies and magnetic resonance imaging (MRI).

Our data also demonstrate that the increase in strength in response to short-term resistance training is augmented by vascular occlusion. Maximal voluntary contraction increased by 18% in the triceps surae trained under restricted but not unrestricted blood flow conditions. Similar changes in strength after 4 weeks of resistance training with or without vascular occlusion have been reported previously (Shinohara et al., 1998). Other authors found vascular occlusion did not enhance the gain in strength following 8 weeks of resistance training (Burgomaster et al., 2003). The discrepancy between these and the current findings could be due to the muscle group chosen for training. Muscles of the upper arm are generally in a less trained state than muscles of the lower leg and may therefore show a greater response to training, which could make it difficult to detect differences in the efficacy of different training programmes, especially in the short term. Blood flow to the biceps brachii reaches zero at $\sim$50% maximal voluntary contraction (Sadamoto, Bonde-Petersen, & Suzuki, 1983), which is the exercise intensity used by Burgomaster et al. (2003). Training at this exercise intensity may therefore prevent blood flow to both the non-occluded and occluded arms, which would silence the effect of cuff occlusion *per se*.

In the early stages of resistance training, neural changes occur that include increased action potential firing frequency, synchronization of action potentials, and number of motor units activated (Enoka, 1997). These learning effects are increased in movements controlled by a small number of muscle groups (Enoka, 1997), such as plantar flexion of the ankle during heel raises. The vascular occlusion-induced increase in strength cannot be easily explained by an improved exercise technique, however, because identical movements are exerted by both restricted and unrestricted triceps surae throughout training. Vascular occlusion limits oxygen availability to the muscle, which has been shown to increase neuromuscular activity (Moritani, Sherman, Shibata, Matsumoto, & Shinohara, 1992), and this could contribute to the gain in strength found in the triceps surae trained under conditions of restricted blood flow.

The increase in strength in response to resistance training with vascular occlusion could also be due to muscle hypertrophy. Resistance training with vascular occlusion decreases intramuscular oxygen concentration (Yamada et al., 2004) and increases the accumulation of hydrogen ions and inorganic phosphate (Suga et al., 2009). It has been hypothesized that the metabolic stress during resistance training with occluded blood flow may increase the release of IGF-1, which in turn could stimulate muscle hypertrophy (Schott, McCully, & Rutherford, 1995; Shinohara et al., 1998). Low intramuscular oxygen concentration increases the activity of several signalling pathways (e.g. the PI3K pathway) that up-regulate the transcription of genes responsible for muscle growth (Rennie, Wackerhage, Spangenburg, & Booth, 2004). Although muscle hypertrophy was not measured in the current study, it has been shown to increase after 4 weeks of exercise training with vascular occlusion, as demonstrated by an increase in muscle cross-sectional area (Nygren et al., 2000). The increase in strength in the triceps surae trained under vascular occlusion is probably stimulated by conditions of hypoxia, which induce increased neuromuscular activity and expression of genes that control protein synthesis and muscle hypertrophy.

Local muscle endurance under complete blood flow restriction did not improve in response to resistance training with or without vascular occlusion in the current study. Other studies have shown that vascular occlusion augments the gain in local endurance in response to exercise training (Kaijser et al., 1990; Terrados et al., 1990). The intensity and duration of training used in these studies were greater than in the present study, which could in part explain why local endurance did not increase in our study. The discrepancy between current and previous findings could also be explained by differences in the method used to induce conditions of restricted blood flow during training. Disparate findings of the current and previous studies (Kaijser et al., 1990; Terrados, Jansson, Sylven, & Kaijser, 1990) could also be due to differences in the nature of the tests themselves (electrically invoked vs. voluntary contractions) and severity of blood flow restriction during tests (complete vs. partial occlusion). There is unlikely to be any training-induced alteration in muscle metabolism in the current study, since this must be the main determinant of force under conditions of vascular occlusion as in the fatigue test.

In summary, strength – and to a lesser extent microvascular filtration capacity (an index of capillarity) – is enhanced by vascular occlusion during short-term resistance training of human triceps surae. Future work should investigate the mechanisms responsible for skeletal muscle adaptations to resistance training under conditions of restricted blood flow.

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