Effects of low-intensity bench press training with restricted arm muscle blood flow on chest muscle hypertrophy: a pilot study

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Summary

Single-joint resistance training with blood flow restriction (BFR) results in significant increases in arm or leg muscle size and single-joint strength. However, the effect of multijoint BFR training on both blood flow restricted limb and non-restricted trunk muscles remain poorly understood. To examine the impact of BFR bench press training on hypertrophic response to non-restricted (chest) and restricted (upper-arm) muscles and multi-joint strength, 10 young men were randomly divided into either BFR training (BFR-T) or non-BFR training (CON-T) groups. They performed 30% of one repetition maximal (1-RM) bench press exercise (four sets, total 75 reps) twice daily, 6 days week^{-1} for 2 weeks. During the exercise session, subjects in the BFR-T group placed elastic cuffs proximally on both arms, with incremental increases in external compression starting at 100 mmHg and ending at 160 mmHg. Before and after the training, triceps brachii and pectoralis major muscle thickness (MTH), bench press 1-RM and serum anabolic hormones were measured. Two weeks of training led to a significant increase ($P<0.05$) in 1-RM bench press strength in BFR-T (6%) but not in CON-T (-2%). Triceps and pectoralis major MTH increased 8% and 16% ($P<0.01$), respectively, in BFR-T, but not in CON-T (-1% and 2%, respectively). There were no changes in baseline concentrations of anabolic hormones in either group. These results suggest that BFR bench press training leads to significant increases in muscle size for upper arm and chest muscles and 1-RM strength.

Introduction

Age-related skeletal muscle loss (sarcopenia) inhibits mobility and increases the risk of developing several diseases such as diabetes, osteoporosis and heart disease (Visser *et al.*, 2002; Guillet & Boirie, 2005). High-intensity resistance training can induce appendicular and trunk muscle hypertrophy and improve insulin resistance and type-2 diabetes in the elderly (Frontera *et al.*, 1988; Fiatarone *et al.*, 1990; Dunstan *et al.*, 2002), suggesting that high-intensity resistance training leads to preventing and/or improving the sarcopenia in the elderly. However, the high intensity required for muscle adaptation with traditional resistance exercise may not be practical and may even be dangerous when carried out without proper supervision in the elderly.

In the past decade, several studies have reported that low-intensity resistance training combined with muscular blood flow restriction (BFR) elicits similar muscle hypertrophy as traditional high-intensity resistance training regardless of age (Takarada *et al.*, 2000b, 2002; Abe *et al.*, 2005; Fujita *et al.*, 2008). Because BFR requires the use of an elastic cuff that is placed at the proximal end of the limbs, the restricted blood flow is only applicable to appendicular muscles. Consequently, previous BFR training studies have focused on the physiological adaptations of appendicular muscles. However, the effect of low-intensity BFR training on non-flow-restricted trunk musculature has not been explored. Our previous study indicated that neuromuscular activity during low-intensity BFR bench press exercise increases not only in the blood flow restricted arm muscle (triceps brachii) but also in non-restricted chest muscle (pectoralis major) compared with same exercise without BFR (Yasuda *et al.*, 2006). We hypothesized that appendicular as well as trunk muscle hypertrophy may be observed following low-intensity multijoint BFR exercise training. Thus, the purpose of this pilot study was to determine the impact of low-intensity bench press exercise training with BFR on muscular strength and hypertrophic responses in chest and upper arm muscles.

Methods

Subjects

Ten young men (ages 23–38 years) volunteered to participate in this study. Their standing height, body weight and one repetition maximal (1-RM) bench press strength (mean ± SE) were 172 ± 5 cm, 66 ± 7 kg and 58 ± 8 kg, respectively, before training. The subjects in this study were physically active, with four of 10 participated in regular aerobic-type exercise (walking, jogging or cycling; 2–3 times per week for approximately 30 min in duration). Four of all subjects had light-to-moderate resistance training experience in performance of the bench press, but none of the subjects had participated in regular resistance training for a minimum of 1 year prior to the start of the study. The subjects were randomly divided into either a BFR training group (n = 5, BFR-T) or a non-BFR training group (n = 5, CON-T). Each subject was informed of the risks associated with the training and measurements and gave written consent to participate in this study, which was approved by the Ethics Committee of the University.

Training protocol

One week prior to training programme, all subjects completed an orientation session to practice and familiarize with 1-RM bench press testing and training equipment. During training programme, each subject performed a supervised free weight flat bench press exercise twice daily (morning and afternoon sessions, with at least 4 h between sessions), 6 days week^{-1} for 2 weeks (total 24 sessions). Training intensity and volume were set at 30% of predetermined 1-RM and 75 repetitions (30 reps followed by three sets of 15 reps, with 30 s rest between sets), respectively and remained constant throughout the training period.

Blood flow restriction

Subjects in the BFR-T group wore elastic cuffs around the most proximal region of both arms during training. On the first day of training, the cuffs were set at 30 mmHg and gradually inflated to 100 mmHg (Day 1). The training air pressure was increased by 10 mmHg each day until 160 mmHg (Day 7) was reached. The restriction pressure was selected by a previous report (Yasuda *et al.*, 2009).

Measurements

Prior to starting the training programme and 3 days after the final training session, several measurements were performed. Maximal dynamic strength (1-RM) was assessed using a free weight flat bench press test. The 1-RM was determined by progressively increasing the weight lifted until the subject failed to lift the weight (Abe *et al.*, 2000). Muscle size was measured using B-mode ultrasound (Aloka SSD-500, Tokyo, Japan) at two anatomical sites [chest (at the site between third and fourth of costa under the clavicle midpoint) and posterior upper arm (at 60% distal between the lateral epicondyle of the humerus and the acromial process of the scapula)] of the left side as has been described previously (Abe *et al.*, 1994, 2000). Briefly, the measurements were carried out while the subjects stood with their elbows extended and relaxed. A 5-MHz scanning head was placed on the measurement site without depressing the dermal surface. The subcutaneous adipose tissue–muscle interface and the muscle–bone interface were identified from the ultrasonic image, and the distance between two interfaces was taken as muscle thickness (MTH; Fig. 1). Ink markers on the triceps brachii and pectoralis major muscles were used to ensure similar positioning over repeated MTH measurement. The estimated coefficient of variation of MTH measurement from test–retest was 1.6% for triceps brachii and 1.7% for pectoralis major muscle. Test–retest reliability correlation coefficients (r) across sessions on different days were 0.99 for triceps brachii and 0.98 for pectoralis major MTH. This measurement was carried out each morning prior to the training session and prior to the post-testing. Previous studies have reported that MTH is strongly correlated with muscle cross-sectional area (CSA) in limb muscle (Abe *et al.*,
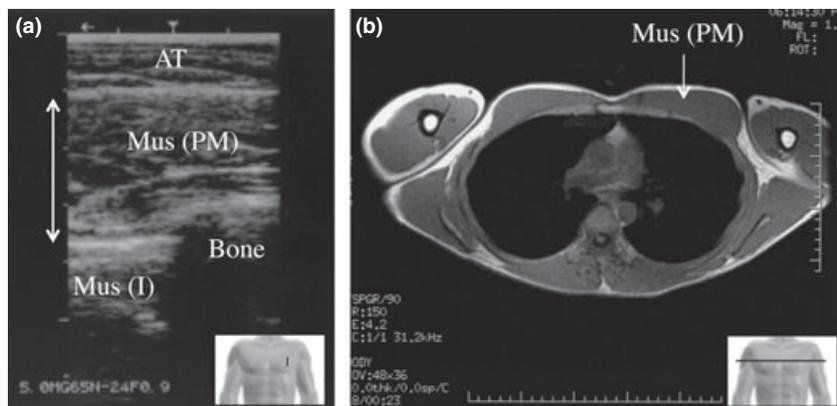

Figure 1 Typical ultrasonographic image (a) and magnetic image showing transverse section (b). Image a is vertical scan on the left of the chest. Image b is transverse scan of the chest. AT, subcutaneous adipose tissue; Mus, skeletal muscle tissue; PM, pectoralis major muscle; I, intercostalis internus muscle; Bone, costa.

1997; Miyatani et al., 2004) although there is no published study for trunk muscle. To examine a relationship between MTH and CSA (at the same site as MTH measurements) in the pectoralis major muscle, another 20 young men were measured using 1.5-T magnetic resonance imaging (GE Signa, Milwaukee, WI, USA). A T1-weighted, spin-echo, axial plane sequence was performed with 1500-ms repetition time and a 17-ms echo time with 1.0-cm slice thickness at the site between third and fourth of costa (Fig. 1). Subjects rested quietly in the magnet bore in a spine position with their arms extended. MTH of the chest was measured by ultrasound, at the same sites as CSA measurements. Results indicate that MTH was strongly correlated (r = 0.92, P<0.001) with pectoralis major muscle CSA (Fig. 2), which suggested applicability of MTH for evaluation of muscle size. Resting venous blood samples were drawn from each subject on the same day prior to the first training (Pre) and 2 days after the final training (post). All blood samples were obtained at the same time of day (9:00–10:00 AM) following an overnight fast (12–13 h). Serum hormones [growth hormone (GH), insulin-like growth factor-1 (IGF-1), and IGF-binding protein-3 (IGF-BP3)] and markers of muscle damage (creatine phosphokinase and myoglobin) were determined using a commercially available kit (SRL Co. Ltd., Tokyo, Japan).

Statistical analyses

Results are expressed as means ± standard deviations (SD). The data were tested for normality using Shapiro-Wilk test. Because all variables were normally distributed, parametric statistical analyses were performed. A two-way analysis of variance (ANOVA) with repeated measures was used to compare BFR-T and CON-T with the effects being group (BFR-T and CON-T) and time (pre and post). Mean value of per cent changes were calculated based on individual changes. Per cent changes from baseline were also compared between groups with student's t-test. Statistical significance was set at P<0.05.
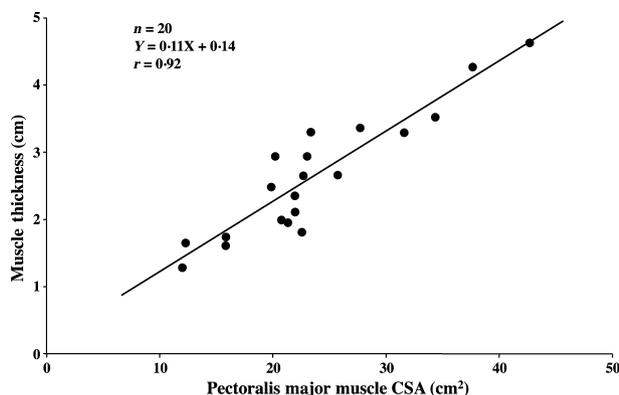

Figure 2 The relationship between muscle cross-sectional area measured by magnetic resonance imaging and muscle thickness by B-mode ultrasound.

Results

At baseline, before the training, there were no differences between BFR-T and CON-T groups for age (25.8 ± 6.3 and 25.6 ± 3.2 years, respectively), standing height (1.72 ± 0.05 and 1.72 ± 0.05 m), body weight (65.4 ± 5.4 and 67.6 ± 7.9 kg), triceps brachii MTH (3.62 ± 0.39 and 3.67 ± 0.78 cm), pectoralis major MTH (2.34 ± 1.9 and 2.24 ± 5.0 cm) and bench press 1-RM (58.5 ± 5.5 and 59.0 ± 17.0 kg). There was no change in body weight for either group following the training.

After the training, MTH for triceps brachii and pectoralis major were increased 8% (pre, 3.62 ± 4.2 cm; post, 3.89 ± 3.9 cm, P<0.05) and 16% (pre, 2.34 ± 1.9 cm; post, 2.76 ± 2.0 cm, P<0.05), respectively, in BFR-T group. No significant changes in MTH were observed in CON-T group (−1% and 2% for triceps brachii and pectoralis major, respectively). Increases in MTH for both triceps brachii and pectoralis major were significantly larger in BFR-T group when compared to CON-T group (Fig. 3). Per cent change in muscular strength as assessed by bench press 1-RM was greater in the BFR-T (6%) than that of the CON-T (−2%) (Fig. 4). There were no significant changes in resting serum hormones (GH, IGF-1 and IGF-BP3) or markers of muscle damage (CK and myoglobin) for either group (Table 1).

Discussion

It has been demonstrated that muscle CSA/volume in arm or leg muscles increases after a low-intensity single joint resistance training in which the blood flow to the working muscles are restricted during exercise (Takarada et al., 2000b, 2002; Fujita et al., 2008). For example, Fujita et al. (2008) have examined the effect of 20% 1-RM-intensity knee extension training combined with BFR on quadriceps muscle CSA/volume and knee extension strength in young men. They found that significant increases in muscle CSA/volume and maximal strength had occurred after 6 days of twice daily training. In this study, we examined the impact of low-intensity multijoint bench press exercise training with BFR on hypertrophic responses to blood flow restricted upper arm muscles as well as non-restricted chest muscle. The results support our hypothesis that muscle hypertrophy in triceps brachii as well as pectoralis major were observed following low-intensity multijoint BFR bench press training. The muscle hypertrophy results from increased protein accretion and from the accumulation of contractile protein, which occurs when the balance between protein synthesis and degradation shifts towards synthesis. A previous study (Fujita et al., 2007) demonstrated that a single bout of 20% 1-RM intensity BFR knee extension exercise increased both vastus lateralis muscle protein synthesis and the Akt/mTOR signalling pathway in young men. These anabolic responses may contribute significantly to BFR training induced muscle hypertrophy in both blood flow restricted upper arm and non-restricted chest muscles.
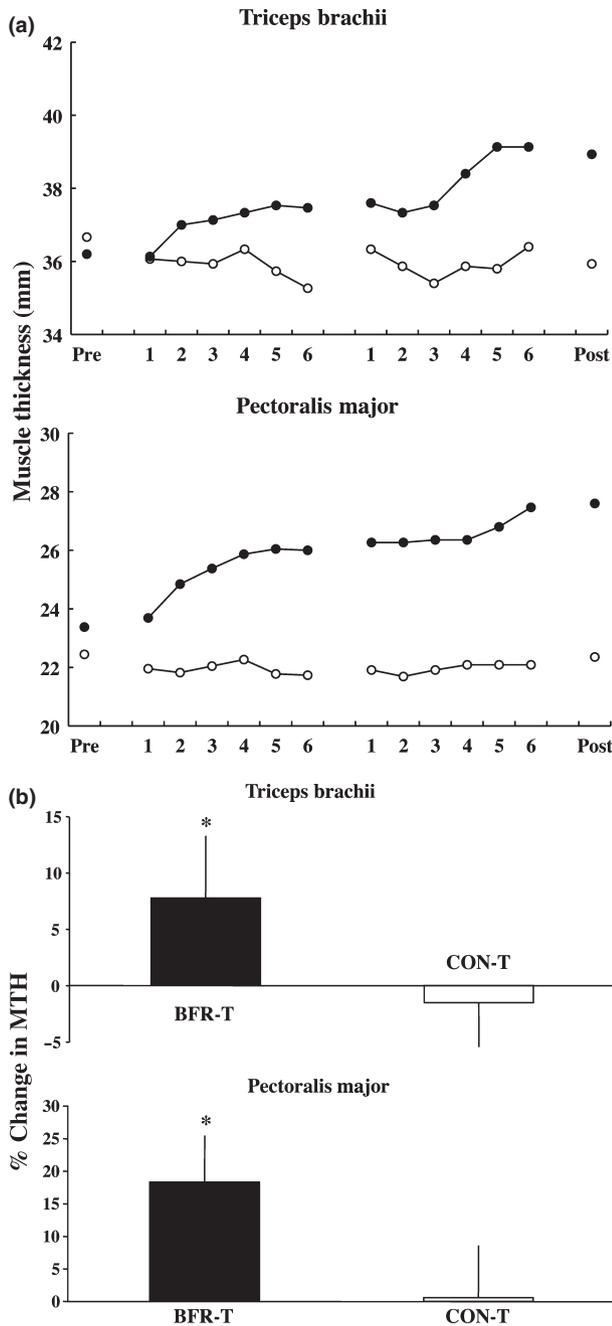
(a)

Triceps brachii

Pectoralis major

(b)

Triceps brachii

Pectoralis major

Figure 3 Changes in muscle thickness of the triceps brachii and pectoralis major muscles following the training period. Data are means ± SD. BFR-T is blood flow restriction group (filled symbols), and CON-T is non-blood flow restriction group (unfilled symbols). *Different from CON-T, P<0.05.

Bench press 1-RM

Figure 4 Per cent changes in one repetition maximal bench press strength following the training period. Data are means ± SD. BFR-T is blood flow restriction group (filled symbols), and CON-T is non-blood flow restriction group (unfilled symbols). *Different from CON-T, P<0.05.

Table 1 Changes in resting serum hormones and markers for muscle damage following the training.

	BFR-T		CON-T	
	Pre	**Post**	**Pre**	**Post**
GH (ng ml^{-1})	0.37 (0.59)	1.86 (2.21)	0.22 (0.32)	0.77 (1.55)
IGF-I (ng ml^{-1})	241 (44)	229 (51)	231 (18)	235 (28)
IGF-BP3 (μg ml^{-1})	2.4 (0.1)	2.3 (0.2)	2.3 (0.4)	2.4 (0.3)
T (ng ml^{-1})	8.0 (2.4)	6.8 (1.4)	4.8 (1.8)	5.4 (1.7)
CPK (IU l^{-1})	340 (457)	280 (237)	290 (294)	312 (267)
MYO (ng ml^{-1})	113 (152)	40 (20)	40 (16)	42 (13)

Values are means (SD).
BFR-T, blood flow restriction training group; CON-T, non-blood flow restriction training group; GH, growth hormone; IGF-I, insulin-like growth factor-1; IGF-BP3, insulin-like growth factor–binding protein-3; T, total testosterone; CPK, creatine phosphokinase; MYO, myoglobin.

were measured integrated electromyography (iEMG) activity in both upper arm and chest muscle during bench press exercise with and without BFR (Data are not shown). The results are similar to our previous investigation (Yasuda et al., 2006) that iEMG activity is synergistically increased in blood flow restricted arm muscle as well as non-restricted chest/deltoid muscles during BFR bench press exercise. The greater muscle activation in chest/deltoid muscles may have taken place to compensate for the deficit in force development with triceps brachii muscle during BFR bench press. Increased muscle activation during low external load (30% 1-RM) with BFR appears to result in greater internal activation intensity (50–90% 1-RM at fourth set) such that activation is comparable to that observed when training at high external load.

Another possible factor for the muscle hypertrophy observed in the blood flow non-restricted chest muscle might be the acute increases in endogenous anabolic hormones, such as GH and IGF-1, during and after exercise training session. Several low-intensity BFR exercise studies (Takarada et al., 2000a; Abe et al., 2005, 2006; Reeves et al., 2006; Fujita et al., 2007) have observed that serum GH as well as IGF-1 increases during and

The reasons for low-intensity BFR training-induced increase in muscle protein metabolism and muscle hypertrophy, especially in blood flow non-restricted muscle, are poorly understood, but several possibilities are presented. A major factor for the blood flow non-restricted muscle hypertrophy may be increased in muscle activity and apparent elevation in contraction intensity during training session. In this study, our subjects
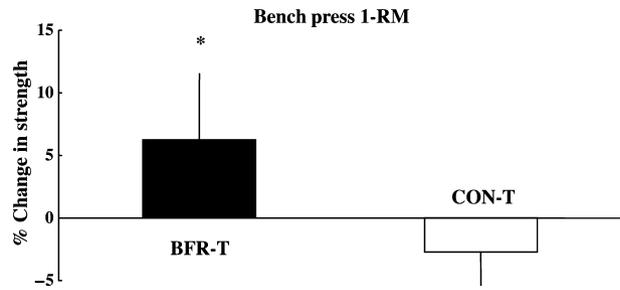
after the exercise session although the present study did not measure acute hormonal response. The exercise-induced increase in blood GH stimulates hepatic production of IGF-1 resulting in elevated circulating blood IGF-1 and stimulates muscle protein synthesis (Borst et al., 2001; Marx et al., 2001). Furthermore, circulating GH directly stimulates endogenous muscle production of IGF-1 (Florini et al., 1996). Recently, Madarame et al. (2008) demonstrated that 10 weeks of low-intensity arm curl resistance training without BFR increased biceps muscle size when it was combined with low-intensity BFR knee extension resistance exercise, indicating a 'cross-transfer' effect for the growth of other skeletal muscles.

It was anticipated that resting serum IGF-I concentration would increase following BFR bench press training, because our previous study had reported increases in resting serum IGF-I following BFR resistance training at same training frequency (Abe et al., 2005). However, our current results showed that serum IGF-I did not change following BFR training. The reasons are not clear, but it might be related to the training volume or type of the exercise. To date, there has been no systematic study on the interactions of altering frequency, intensity, duration or type of BFR training. More work is needed to understand how these variables would affect muscle adaptation by the BFR training.

In this study, resting blood markers for muscle damage (CPK and myoglobin) were not elevated on average. Both pre- and post-training values showed large variability, possibly associated with physical activity (only one subject in each group) performed outside of the BFR training. Previous studies reported that there are no changes in markers of muscle damage and oxidative stress between before and after acute bout of low-intensity BFR exercise (Abe et al., 2005, 2006; Fujita et al., 2008; Goldfarb et al., 2008). Therefore, the results of this study along with the previous studies suggest that the rapid response of muscle hypertrophy following low-intensity BFR training is not associated with cell swelling induced by muscle damage or inflammation of the muscle tissues.

In this study, a per cent increase in 1-RM strength is not larger than that of increase in muscle size. Previous studies have reported that relative strength (i.e. the maximal strength per unit of muscle size) of the knee extensor and elbow flexor muscle did not change significantly between pre- and post-training following low-intensity BFR training (Takarada et al., 2000b, 2002; Abe et al., 2006; Fujita et al., 2008). This suggests that changes in muscle strength are more closely tied to changes in muscle hypertrophy as opposed to change in neural adaptations. Taken together, these data suggest that a main contributor of increased muscle strength after BFR training is the increase in muscle size (physiological muscle CSA), which surpass the neural adaptation such as fibre recruitment patterns.

In conclusion, low-intensity bench press training combined with BFR of the arms leads to significant increases in 1-RM bench press strength and muscle size of both the blood flow restricted upper arm muscles as well as non-restricted pectoralis major muscle.

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