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RAPID VASCULAR MODIFICATIONS TO LOCALIZED RHYTHMIC HANDGRIP TRAINING AND DETRAINING

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Abstract

Introduction—Despite the evidence describing the rapid vascular function modifications to commencement and cessation of large muscle exercises (i.e. cycling), no studies examined the time-course vascular modifications to localized training and detraining.

Purpose—To examine the effects of 4-week rhythmic handgrip exercise training and 2-week detraining on reactive hyperemic forearm blood flow and vascular resistance in 11 young men.

Methods—Rhythmic handgrip exercise was performed in the non-dominant forearm for 20 minutes/day, 5 days/week, at 60% of maximum voluntary contraction for 4 weeks, followed by 2 weeks of no training. Forearm blood flow and vascular resistance were evaluated, in both arms, at rest and following arterial occlusion. These vascular function indices were obtained in 5 visits; before, after 1 and 4 week(s) of training as well as after 1 and 2 week(s) of training cessation.

Results—Resting cardiovascular measures were not altered during the study period. A 2 (arms) * 5 (visits) ANOVA revealed significant arms-by-visits interactions for reactive hyperemic forearm blood flow ($p=0.02$) and vascular resistance ($p=0.02$). Subsequent comparison demonstrated increased trained forearm reactive hyperemic blood flow 1 week after training, then returned to pre-training values 1 week following training cessation. In contrast, vascular resistance decreased 1 week after training commencement, only to return to pretraining level 1 week after training cessation.

Conclusion—These results indicate a rapid, unilateral improvement in regional reactive hyperemic blood flow and vascular resistance following localized exercise-training. However, the improvements are transient and return to pretraining levels 1 week after detraining.

Keywords

regional blood flow; rhythmic handgrip; exercise training/detraining; time-course

Introduction

Recent data report rapid improvements in regional (Alomari and Welsch 2007) and single vessel (Allen et al. 2003) vascular function after one week of rhythmic handgrip exercise training confirming previous data from animal models (Laughlin 1995). Although the

precise mechanisms for these rapid modifications are not entirely known, exercise-mediated muscle contractions trigger a sequence of vascular adaptations, secondary to fluctuating shear stress. Apparently, fluctuating shear stress on the arterial wall induces vascular changes secondary to endothelium-dependent and independent structural and functional modifications (Barakat 1999; Clarkson et al. 1999; Delp 1998; Prior et al. 2003). In fact, recent evidence from in vivo studies indicates that exercise-induced shear stress contributes to an anti-atherogenic vascular phenotype (Laughlin et al. 2008).

Arguably, one might hypothesize that if muscular contractions induced fluctuations in shear stress evoke rapid vascular adaptations, removal of this stimulus could result in rapid loss of vascular benefits. Indeed, there are several studies that indicate that vascular adaptations to exercise training are transient and return to pre-training upon removal of the training stimulus (Hornig et al. 1996; Murray et al. 2006; Thijssen et al. 2006; Wang 2005). However, those studies used whole-body exercise training protocols (e.g. cycling) (Murray et al. 2006; Wang 2005) or involved patients with chronic (e.g. heart failure) or disabling (e.g. spinal cord injury) diseases (Hornig et al. 1996; Thijssen et al. 2006). Although, those studies provide important information about vascular adaptations to exercise training, it is important to understand that any reported changes in vascular responses were more than likely influenced by changes in other physiological systems, or the presence of pathology. Thus it is difficult to interpret whether the vascular changes to exercise training are a direct (locally mediated) or indirect (mediated through other physiological systems). This is an important point to consider, if we are to appreciate the role of exercise training on vascular biology. Regional-specific exercises typically do not evoke significant heart rate or blood pressure changes. Thus vascular modifications to regional-specific training and after cessation of such training are more than likely a direct consequence of alterations in the local milieu (e.g. muscle contraction-induced shear stress). Interestingly, the literature is not clear how regional vascular function responds across a period of localized training and detraining, in young healthy individuals.

Therefore, the purpose of this study was to examine the effects of a localized training stimulus followed by a detraining period on forearm vascular function. Specifically, changes in forearm reactive hyperemic blood flow responses and vascular resistance were monitored during 4 -weeks of rhythmic handgrip exercise training, followed by 2 weeks of detraining. On the basis of previous findings, it was hypothesized that forearm reactive hyperemic blood flow responses and vascular resistance would improve within the first 2 weeks of training, followed by a rapid decline during the detraining period in the trained arm only.

Methodology

Inclusion/Exclusion Criteria

Apparently healthy and sedentary men between 18 and 35 years old were recruited to partake in the study. Smokers, as well as candidates taking any medications which could affect cardiovascular function were excluded. All participants signed an informed consent, approved by the Institutional Review Board of the sponsoring institution, following detailed description of the study and advised of the inherited risks, potential benefits and expected commitments.

Experimental Design and Training Protocol

The study was a randomized, prospective design aimed at examining forearm vascular changes during 4 weeks of unilateral rhythmic handgrip exercise training followed by 2 weeks of detraining. Vascular function measurements for each subject were obtained 5 times including prior to (pretrain), after the 1st (train1) and 4th (train2) week of training, as well as

1 (detrain1) and 2 (detrain2) week(s) after cessation of training. The training protocol consisted of five, 20-minute sessions per week. The intensity of training was 60% of maximum handgrip strength. During rhythmic handgrip exercise training session, the length of the contraction and relaxation was about 4 seconds with each phase approximately 2 seconds. Workloads were adjusted at the beginning of each week of the 4-week training period to account for changes in handgrip strength. Training was performed in the non-dominant arm while the dominant arm served as control under the supervision of study personnel.

Measurements

Handgrip strength and forearm skinfold and circumference—The participants' maximal handgrip strength was measured using a hand dynamometer (Lafayette instruments, OH), from a standing position. Maximal handgrip strength was determined from the average of 3 consecutive all-out gripping trials. Additionally, forearm skinfold (SF) was estimated with standard skinfold caliper and circumference was determined using tape measure 10 cm distal to the midpoint between the lateral epicondyle and olecranon process. Each measurement was obtained before and at the end of each week of the training program, and detraining period.

Cardiac hemodynamics and vascular function—Prior to the assessment of cardiovascular indices, all participants were allowed to rest in a supine position for 15 minutes. Subsequently, forearm blood flow and vascular resistance were measured non-invasively in both arms using mercury strain gauge plethysmography (model EC5R, D. E. Hokanson Inc, Bellevue, WA). Measurements were obtained at rest and following 5 minutes of upper arm arterial occlusion, in both arms. Prior to obtaining the measurements, 2 pressure cuffs were positioned above the elbow and on the wrist with the forearm elevated, extended, and supinated on a styrofoam block slightly above the heart. Additionally, the strain gauge was placed around the forearm approximately 10 cm distal to the olecranon process (Alomari et al. 2004; Alomari and Welsch 2007).

Immediately before measuring resting blood flow, hand circulation was occluded for 1 minute by inflating the cuff at the wrist to 240 mmHg. Forearm blood flow was obtained after occluding the upper arm venous circulation at 7 mmHg below diastolic blood pressure (Alomari et al. 2004). Subsequently, reactive hyperemic forearm blood flow was measured following arterial occlusion achieved by inflating the upper arm cuff to 240 mmHg for 5 minutes. Forearm blood flow was then determined as described at rest. The same measures were then performed in the other arm. Additionally, automated noninvasive standard auscultatory method was used to measure blood pressure and heart rate simultaneously at rest and immediately after 5 minutes of arterial occlusion.

Calculations of blood flows were computed as previously described (Alomari et al. 2004; Alomari and Welsch 2007), however they will be briefly explained. Resting and reactive hyperemic blood flows were recorded at a paper speed of 5 and 25 mm/seconds, respectively. Immediately after occluding the upper arm venous circulation, the flows were estimated from the slope drawn on the plethysmography paper graph using the first 2 beats and expressed in $\text{ml} \cdot 100 \text{ ml tissue}^{-1} \cdot \text{min}^{-1}$. Forearm vascular resistance was then determined by dividing mean arterial pressure by the blood flow.

Statistical Analyses

A 2 (arms) * 5 (visits) split-plot ANOVA was used to examine the influence of the 4-week rhythmic handgrip exercise training and the 2-week detraining on vascular function indices. A repeated measure ANOVA was used to compare changes in blood pressure, heart rate,

arm circumference, and handgrip strength throughout the study. Alpha was set a-priori at $p < 0.05$. All statistical analyses were completed using SAS (version 9.00, Cary, NC) statistical package.

Results

Participants

Participant characteristics are presented in table 1. Eleven men completed the 5 study visits with average age, height, and weight of 24 ± 4.8 years old, 174.6 ± 6.6 cm, and 72.2 ± 6.1 kg, respectively.

Forearm circumference, handgrip strength, and hemodynamics

No significant changes in handgrip strength and forearm circumference were noted in table 1 between baseline and detrain2.

Additionally, table 2 shows that heart rate and blood pressure did not significantly change between all visits. Furthermore, as presented in table 2, no changes were observed in resting forearm blood flow and vascular resistance throughout the study period. However, the ANOVA tests revealed significant interactions between arms and visits for reactive hyperemic blood flow ($p = 0.02$) and post occlusion vascular resistance ($p = 0.02$) in the trained arm only. In fact, as depicted in figure 1, the post-hoc comparison showed that reactive hyperemic blood flow increased 14% at train1 ($p = 0.03$) and remained elevated at train2 ($p = 0.005$) as compared to baseline, then returned to pretraining level at detrain1 ($p = 0.94$) and detrain2 ($p = 0.86$). Figure 2 reveals similar changes in post occlusion vascular resistance, with a 15% decrease at train1 ($p = 0.05$) and train2 ($p = 0.03$) as compared to pretrain, followed by an increase to pretraining level at detrain1 ($p = 0.9$) and detrain2 ($p = 0.4$).

Discussion

Consistent with our hypothesis, forearm reactive hyperemic blood flow responses and vascular resistance improved after 1 week of rhythmic handgrip training, but rapidly declined within 1 week after cessation of training.

Training Adaptations

Post-occlusion blood flow was greater and vascular resistance was less, in the dominant than in the non-dominant forearm during pretrain; obviously due to habitual usage (Sinoway et al. 1986). However, they were similar after 1 week of non-dominant rhythmic handgrip training and remained the same for the rest of the training period. This is quite significant and indicates that matching vascular function after lifelong usage can be achieved rapidly, even in young apparently healthy individuals. The 18% improvement in the reactive hyperemic blood flow response and 15% decline in vascular resistance after 4 weeks of training are consistent with previous reports using similar training protocol (Alomari and Welsch 2007; Alomari et al. 2001; Sinoway et al. 1987). Also as previously demonstrated in our laboratory, forearm reactive hyperemic blood flow increased 14% and vascular resistance decreased 15% after 1 week of rhythmic handgrip training in the trained arm only (Allen et al. 2003; Alomari and Welsch 2007). Given there were no significant changes in the contralateral arm and/or overall cardiovascular hemodynamics, including blood pressure, and heart rate, the training adaptations reported in the current study appear to be locally driven. Although the present study was not designed to address specific mechanisms for the vascular changes, data indicate that the fluctuating shear stress on the arterial wall during muscle contractions signal formation of beneficial endothelial cell phenotype. These benefits

include increased nitric oxide production, eNOS, PGI₂, anti-oxidant defenses, and a reduction in reactive oxygen species, adhesion molecules, and vasoconstriction factors such as endothelin⁻¹ (Laughlin et al. 2008). However, it is important to recognize that the regional adaptations observed in the current study, certainly extend beyond the endothelium, as reactive hyperemia is not thought to be largely influenced by nitric oxide (Engelke et al. 1996). These other factors are believed to include metabolic (Delp 1998), neural (Shoemaker et al. 1998a; Shoemaker et al. 1998b), and structural (Lehoux et al. 2002; Prior et al. 2003) modifications. For example, the greater reactive hyperemic response in this study may be a consequence of an increase in muscle capillary cross-sectional area (Suzuki et al. 1997; Suzuki et al. 2001). Evidence indicates, that vasodilating substances (such as prostaglandins, adenosine and nitric oxide) released following each contraction, stimulate vascular endothelial growth factors, which are coupled to muscle capillary supply. In fact, an increase in “pre-existing” and “de-novo” capillaries has been reported after only 1 week of exercise training (Suzuki et al. 2001). Additional evidence suggests that smooth muscle sensitivity is enhanced with training, thus allowing for greater relaxation, and reactive hyperemic blood flow (Delp and Laughlin 1997). However, future studies are needed to further reveal the contribution of these mechanisms in vascular adaptations to exercise training.

De-Training Responses

Uniquely, the present data indicate a rapid return of forearm reactive hyperemic blood flow (–14%) and vascular resistance (17%), to pretraining levels. After only 1 week of rhythmic handgrip training, vascular function indices in the non-dominant were less than in the dominant forearm. The reversals of training adaptations were not accompanied by changes in autonomic nervous balance, heart rate or blood pressure suggesting involvement of local factors.

The present findings are difficult to compare since the majority of previous studies have examined changes in vascular function subsequent to 3 deconditioning models distinctly different from the one used in the current study. These models used either exercise cessation after large muscle group training protocols (e.g. cycling) (Klausen et al. 1981; Maeda et al. 2001; Murray et al. 2006; Vona et al. 2004; Wang 2005), simulated inactivity (i.e. bed rest or leg suspension) (Bleeker et al. 2005a; Bleeker et al. 2005b; Bonnin et al. 2001), or detraining due to chronic (Hornig et al. 1996) and/or disabling diseases (i.e., spinal cord injury) (de Groot et al. 2005; de Groot et al. 2006b; de Groot et al. 2004; De Groot et al. 2003; Thijssen et al. 2006; Wecht et al. 2000). Thus, our understanding of vascular modifications to training/detraining might be limited due to interference of other physiologic changes (e.g. autonomic balance) or diseases.

Arguably, vice versa to training, the removal of the fluctuating shear stress on the arterial wall during muscle contractions is the most likely reason for the reversal in vascular adaptations. In fact, a number of studies have suggested that training cessation results in reduced nitric oxide synthase expression, thereby, decreased nitric oxide synthesis, (Hornig et al. 1996; Vona et al. 2004; Wang 2005), and increased endothelin⁻¹ (Maeda et al. 2001).

Once again, we must be cautious attributing reduction in reactive hyperemic responsiveness following training cessation to just endothelial factors as the phenomena seems to be multifactorial thus complex. One must also consider the influence of the training cessation on metabolic (Delp 1998), neural (Shoemaker et al. 1998a; Shoemaker et al. 1998b), and structural (Lehoux et al. 2002; Prior et al. 2003) factors. For example, changes in vascular function have been examined after inactivity using either leg suspension or bed rest. The decay in blood flow after 1 leg suspension was attributed to reduction in venous capacitance and pressure without changes in nitric oxide pathway (Bleeker et al. 2005a). Hypotension

and orthostatic intolerance associated with bed rest were related to increased arterial diameter and dilation (Bonnin et al. 2001) subsequent to either enhanced nitric oxide sensitivity or smooth muscle reactivities to vasodilators (Bleeker et al. 2005b). However, given reactive hyperemic blood flow reflects mainly metabolic vasodilation (Engelke et al. 1996), the decay in vascular function might be due to local metabolic modifications. Opposite to exercise training, the cessation of training stimulus might reduce metabolically-induced vasodilation, particularly prostaglandins (Carlsson et al. 1987; Carlsson and Wennmalm 1983), adenosine and bradykinin (Clifford and Hellsten 2004; Sadowski and Badzyska 2008). Nevertheless, future studies should certainly further examine the metabolic, structural and other theories.

The rapid decay in vascular function was observed after 3 weeks of spinal cord injury (de Groot et al. 2006b) and 1 week after cessation of “function electric stimulation cycling” used to train the paralyzed leg in patients with spinal cord injury (Thijssen et al. 2006). However the authors suggested the mechanisms involved in vascular function decay found in spinal cord patients are not necessarily a mere reversal of vascular adaptations to exercise training (de Groot et al. 2006a). As much as there is no sufficient understanding of the rapid blood flow improvements to exercise training, data describing vascular function decay is sparse since only few studies examined vascular changes after exercise training cessation. Therefore, future studies should examine the mechanism(s) attributing to the transient vascular modifications associated with exercise training and detraining.

Finally, the unilateral and transit modifications in vascular function confirms the specificity (Pogliaghi et al. 2006; Richardson et al. 2006; Tordi et al. 2001) and reversibility (Mujika and Padilla 2000a, b) principles of training, respectively. Therefore, its essential to design a well-rounded exercise program that target different body systems and organs including the cardiovascular system and major muscle groups.

Conclusion

Forearm reactive hyperemic blood flow and vascular resistance rapidly improved with rhythmic handgrip training, but returned to pretraining levels 1 week after training cessation. The transit vascular modifications imply that the vasculature is quite responsive to the training/detraining stimulus. These data suggest the importance of consistent exercise dosing and well-rounded exercise programs to maintain vascular health.

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Figure 1.

Average changes in reactive hyperemic forearm blood flow in the untrained (dominant) and the trained (non-dominant) arms of all participants across the 5 visits. Values are mean $\pm$ SE. * = $p < 0.05$ comparing the trained forearm to the untrained forearm of the same visit; † = $p < 0.05$ comparing the trained forearm of train1 and train2 to the trained forearm of pretrain, detrain1 & detrain2.

Figure 2.

Average changes in post-occlusion vascular the untrained (dominant) and the trained (non-dominant) arms of all participants across the 5 visits. Values are mean $\pm$ SE. * = $p < 0.05$ comparing the trained forearm to the untrained forearm of the same visit; † = $p < 0.05$ comparing the trained forearm of train1 and train2 to the trained forearm of pretrain, detrain1 & detrain2.

Table 1

Subject characteristics

	Pretrain	Detrain2
Age (yrs)	24±4.8	-----
Height (cm)	175 ± 6.6	-----
Weight (kg)	72.2 ± 6.1	72.9 ± 6.9
Sum of three SF (mm)	69.2 ± 10.4	60.3 ± 9.0
Control forearm SF (mm)	0.9 ± 0.1	1.2 ± 0.3
Trained forearm SF (mm)	0.75 ± 0.2	0.83 ± 0.3
Control arm circum (cm)	26.3 ± 0.5	26.5 ± 0.5
Trained arm circum (cm)	26.0 ± 0.5	26.1 ± 0.5
Control HG Strength (kg)	40.7 ± 2.4	40.1 ± 3.4
Trained HG Strength (kg)	37.2 ± 2.0	40.7 ± 3.2

Values are mean ± SE. SF: skinfold; Sum of three SF: chest, abdominal, and thigh; HG: handgrip.

Table 2

Cardiovascular hemodynamics

	Pretrain	Train1	Train2	Detrain1	Detrain2
HR (bpm)	57.4±1.6	58.9±1.3	57.5±1.8	58.1±1.8	59.4±2.3
SBP (mmHg)	113±2.2	112±2.2	111±2.2	109±2.5	111±2.3
DBP (mmHg)	59.8±1.8	61.1±2.2	59.7±1.8	60.6±1.9	62.4±2.2
MAP (mmHg)	77.3±1.8	78.1±1.9	76.8±1.8	76.7±2.0	78.4±1.9
Control arm RBF	2.9±0.2	2.6±0.3	2.6±0.2	2.8±0.2	2.5 ± 0.2
Trained arm RBF	3.1±0.3	2.7±0.2	2.8±0.3	2.3±0.1	2.8±0.3
Control arm RVR	31.5±3.5	34.0±4.3	33.5±2.3	30.1±3.6	33.3±4.1
Trained arm RVR	29.0±3.6	28.0±3.0	28.3±3.2	33.8±2.2	31.2±5.1

Values are mean ± SE. HR: heart rate; bpm: beats per minute; SBP: systolic blood pressure; DBP: diastolic blood pressure; MAP: mean arterial blood pressure; RBF: resting blood inflow (ml/100ml tissue·1·min⁻¹). RVR: resting vascular resistance.