Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow mediated dilation in healthy humans

Mark Rakobowchuk, Sophie Tanguay, Kirsten A. Burgomaster, Krista R. Howarth, Martin J. Gibala & Maureen J. MacDonald

Department of Kinesiology, McMaster University, Hamilton, Ontario, L8S 4K1, Canada

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Address for correspondence:

Maureen J. Macdonald
Department of Kinesiology
Ivor Wynne Centre
1280 Main Street West
Hamilton, Ontario, Canada
L8S 4K1
P: 905-525-9140 x23580
F: 905-523-6011
E: macdonmj@mcmaster.ca
ABSTRACT

Low-volume sprint interval training (SIT), or repeated sessions of brief, intense intermittent exercise, elicits metabolic adaptations that resemble traditional high-volume endurance training (ET). The effects of these different forms of exercise training on vascular structure and function remain largely unexplored. To test the hypothesis that SIT and ET would similarly improve peripheral artery distensibility and endothelial function and central artery distensibility, we recruited 20 healthy untrained subjects (age: 23.3 ± 2.8) and had them perform 6 wk of SIT or ET (n = 5 men and 5 women per group). The SIT group completed 4-6 x 30s “all-out” Wingate Tests separated by 4.5 min of recovery, 3 d/wk. The ET group completed 40-60 min of cycling at 65% of their VO2peak, 5 d/wk. Popliteal endothelial function, both relative and normalized to shear stimulus, was improved after training in both groups (main effect for time, P<0.05). Carotid artery distensibility was not statistically altered by training (p=0.29) in either group, however popliteal artery distensibility was improved in both groups to the same degree (main effect, P<0.05). We conclude that SIT is a time-efficient strategy to elicit improvements in peripheral vascular structure and function that are comparable to ET. However, alterations in central artery distensibility may require a longer training stimuli and/or greater initial vascular stiffness than observed in this group of healthy subjects.
INTRODUCTION

The distensibility of the arterial tree has an important regulatory impact on cardiac performance, perfusion and homeostasis (23). A stiff arterial tree is also associated with adverse cardiovascular events (4). As well, decreased peripheral artery distensibility impacts coronary circulation through quicker pulsewave reflection, which augments systolic pressure while concomitantly lowering diastolic pressure and thus coronary perfusion pressure (39).

Traditional moderate-intensity exercise training improves central artery distensibility in populations with impaired vasculature (19, 43, 46) and most training studies showing improvements of artery distensibility have noted changes in the central arterial tree (aorta or carotid arteries) (19, 21, 43, 46, 49) while peripheral muscular arteries commonly show no exercise training induced improvements (10, 19, 35, 45). However, these investigations of peripheral muscular artery distensibility were made in the relatively stiff common femoral artery (10, 19, 35, 45).

Brachial endothelial function is a surrogate indicator of coronary endothelial function and an independent measure of atherosclerotic disease risk (40, 44, 50). As well, coronary artery disease patients exhibit reduced popliteal artery flow-mediated dilation (2). Similar to artery distensibility, exercise training is a potent stimulus that improves brachial flow-mediated dilation and endothelial function in young healthy (9, 14) and diseased populations (28, 29). The popliteal artery, unlike the brachial artery, is a common site of peripheral vascular disease and displays unique elastic-like properties (13). To date, it has not been established whether exercise training can improve the structural and functional properties of this disease prone artery. In models of integrated
vascular physiology, structure and function are tightly linked with decreased peripheral artery distensibility and endothelial function often occurring in concert, thereby creating an environment where disease progression accelerates. A recent review highlights the importance of exercise training in modifying traditional cardiovascular risk factors such as hypercholesterolemia and hypertension (17). However, 40% of the reduction of cardiovascular disease risk attributed to exercise cannot be explained by modifications of the mentioned risk factors (17). Other vascular indices such as artery endothelial function and distensibility may provide useful information about the link between exercise stimuli and cardiovascular risk reduction (17).

Recently, there has been renewed interest in interval training models, particularly sprint interval (above 100% peak aerobic power) training because of evidence that the ensuing metabolic adaptations mirror those observed after traditional endurance training (5, 6, 8, 26). High-intensity interval training has been shown to accelerate the kinetic responses of leg blood flow and oxygen uptake at the onset of high intensity single leg kicking exercise, indicating a more efficient cardiovascular response system (24). As well, studies in rodents reveal many alterations in vascular structure and function and eNOS protein expression with this type of training (25). However, the mechanisms responsible for these adaptations have not been fully examined. We suspect that changes in vascular structure and function may impact the kinetic responses of skeletal muscle blood flow at the onset of exercise.

Therefore, the purpose of the current study was to evaluate whether 6 weeks of high intensity, low volume, sprint interval training (SIT) improves central (carotid) artery distensibility and, peripheral (popliteal) artery distensibility and endothelial function in
the trained legs to the same extent as high-volume, moderate intensity endurance training (ET). We hypothesized that central artery distensibility would increase to a similar degree with both training methods (ET and SIT). Further we hypothesized that popliteal artery distensibility would improve in concert with enhanced endothelial function indicating improved peripheral vascular structure and function. Note that metabolic and performance adaptations to the training protocol have been previously described in a separate publication (7).

**METHODS**

**Subjects**

Twenty young healthy men and women (n = 5 men and 5 women per group) volunteered for the study (Table 1). A preliminary screening process was employed to establish that subjects: (a) were free of risk factors associated with cardiovascular, pulmonary or metabolic disease; (b) were deemed safe to begin a physical activity program; and (c) other than activities of daily living, were not engaged in a regular training program (i.e. 2 sessions per week and 30 min per session, for at least 1 year prior to the study including recreational activity such as sport or leisure activities). Other exclusion criteria included cardiovascular disease, diabetes, obesity, hypertension (resting blood pressure > 140/90 mmHg), medication use, and smoking as assessed through pre-testing screening. The experimental procedures and potential risks were fully explained to the subjects prior to the study, and all subjects provided written, informed consent. Hamilton Health Sciences Research Ethics Board approved the experimental protocol.

**Pre-experimental procedures**
Subjects initially performed a progressive exercise test (increasing 1 W every 2 s) on an electronically braked cycle ergometer (Lode BV, Excalibur Sport V2.0, the Netherlands) in order to determine their peak oxygen uptake using an on-line gas collection system (Moxus Modular VO2 System, AEI Technologies Inc., Pittsburgh, PA, USA). The value used for \( VO_{2\text{peak}} \) corresponded to the highest value achieved over a 30 s collection period. All subjects also performed a 30 s test of all out effort (Wingate Test) on the same cycle ergometer against a resistance equivalent to 0.075 kg • (kg body mass)\(^{-1}\). After the familiarization procedures, subjects were randomly assigned to either a SIT) group or an ET group in a matched fashion based on sex and \( VO_{2\text{peak}} \).

**Training protocol**

ET consisted of continuous cycling on an ergometer, 5 days per week (Monday–Friday) for 6 weeks, at a power output corresponding to 65% \( VO_{2\text{peak}} \). Subjects performed 40 min of exercise per training session for the first 2 weeks. Exercise time was increased to 50 min per session during weeks 3 and 4, and subjects performed 60 min of exercise per session during the final 2 weeks. \( VO_{2\text{peak}} \) tests were re-administered after 3 weeks of training and training loads were adjusted in order to maintain a training intensity equivalent to 65% \( VO_{2\text{peak}} \). SIT consisted of repeated Wingate Tests on an ergometer 3 days per week (Monday, Wednesday and Friday) for 6 weeks. The number of Wingate Tests performed during each training session increased from four during week 1 and 2, to five during week 3 and 4, and finally to six during week 5 and 6. For all training sessions, the recovery interval between Wingate Tests was fixed at 4.5 min, during which time subjects cycled at a low cadence (< 50 r.p.m.) against a light resistance (30 W) to reduce
venous pooling in the lower extremities and minimize feelings of light-headedness or nausea. The ET program was based on general guidelines recommended by a leading public health agency (1) whereas the SIT program was modeled on recent studies conducted in our laboratory that have examined metabolic and performance adaptations to low-volume, high-intensity interval training (5-8, 15). By design, the protocols differed substantially in terms of total training volume and time commitment in order to evaluate vascular adaptations to two diverse training programs.

Vascular assessment sessions

All participants arrived at the laboratory at the same time of the day (at all testing sessions) for all vascular assessments. Time of testing was specific to each subject with some participants arriving in the morning and others in the afternoon. Female participants were tested in the same phase of their individual menstrual cycle to control for this potential confounding factor. Due to the pragmatic constraints of scheduling and the need to perform metabolic measurements (7) within a reasonable time, 2 participants were tested during the luteal phase while the remaining 8 were tested during the follicular phase. Further, the 2 participants tested during the luteal phase were subsequently removed from the endothelial function data set due to poor image quality. Therefore, all female participants included in the endothelial function portion of the experiment were in the follicular phase at both pre and post testing time points. Prior to arriving in the lab participants were instructed to abstain from caffeine and no participant was taking medication or using nicotine products for at least 12 hours. Testing sessions were performed 4 hours postprandial following the consumption of a commercially available standardized meal replacement drink (237ml BOOST®, Mead Johnson Nutritionals,
Ottawa, ON, Canada) to control for the acute effects of diet. Measurements were taken while subjects were in the supine position in a temperature controlled (22-24°C) room. Vascular measurements were conducted twice prior to the initiation of training and at 48 and 72 hours following their final exercise session. All measurements were taken following a 20-min supine rest period. Because no differences were noted between the 2 PRE and 2 POST testing sessions, the average of these tests was used for subsequent analysis.

Resting heart rate, central and peripheral arterial blood pressure

Electrocardiography was used to record ventricular depolarization via a three-lead set-up, while simultaneous measurements of continuous brachial blood pressure were acquired by automated applanation tonometry (model CBM-7000, Colin Medical Instruments, San Antonio, USA). Both signals were acquired and recorded using commercially available hardware (Powerlab model ML795, ADInstruments, Colorado Springs, USA) and software (Chart 5, ADInstruments, Colorado Springs, CO, USA).

Direct arterial distensibility

Measurements of vascular structure and artery distensibility were determined using two methods by the same investigator (MR) who has 6 years of experience imaging the peripheral vasculature in similar research applications. Arterial distensibility in the vessel of interest (carotid or popliteal artery) was assessed directly using a combination of Ultrasound imaging (System FiVe, GE Medical Systems, Horten, The Netherlands) for the measurement of lumen diameter and vessel specific blood pressure via applanation tonometry (model SPT-301, Millar Instruments Inc., Texas, USA) or automated oscillatory cuff. These methods have been previously described (37), but have been
modified slightly. Briefly, the same investigator throughout the protocol imaged the carotid artery and used baseline images as visual feedback to ensure similar ultrasound probe placement and imaging of the common carotid artery 2-3cm proximal to the bifurcation. At the popliteal artery, the designated peripheral artery, measurements were made either proximal or distal to the branching of the middle genicular artery, yet consistent within each subject as verified by visualization of landmarks. This variation between subjects was needed to ensure the highest possible image quality.

Two ultrasound video clips of ten heart cycles each were acquired at a frame rate of 15 frames per second, simultaneous to measurements of carotid or ankle pulse pressure. Simultaneous to imaging at the carotid artery, a hand-held pressure transducer (model SPT-301, Millar Instruments Inc., Texas, USA) sensitive to hold-down pressure, was held against the carotid artery to acquire arterial blood pressure waveforms while simultaneous measurements of continuous absolute brachial blood pressure was obtained for the purpose of calibrating the carotid waveforms to diastolic and mean pressures (model CBM-7000, Colin Medical Instruments, San Antonio, USA). The Colin model CBM-7000 device combines blood pressure from a brachial cuff and a wrist sensor to determine beat-to-beat brachial blood pressure. The device calibrates the radial blood pressure waveform to the brachial cuff derived blood pressure thus giving the equivalent of beat-to-beat brachial blood pressure. This modification of previous methods (22) simply provides brachial blood pressure for each beat so that carotid blood pressure calibration is beat specific. Briefly, it was assumed both DBP and MAP are similar in all conduit arteries when an individual is in the supine position while SBP is amplified through the arterial tree (33). The mean and minimum BP values obtained from the
carotid waveform were equated to the MAP and DBP of the radial artery. The maximum BP waveform value recorded in the carotid artery was then used as an extrapolation point from the calibrated MAP and DBP. For popliteal measurements, ankle pulse pressure from 2 ankle cuff (model CBM-7000, Colin Medical Instruments, San Antonio, USA) derived measurements was used because brachial cuff values do not correlate with posterior tibial pressures due to pulsewave amplification.

All video clips used to determine artery distensibility were analyzed by the same investigator using a semi-automated edge detection software program (AMS II, Chalmers University of Technology, Göteborg, Sweden).

The twenty measurements of diameter change were subsequently used to calculate distensibility. The following equation was used to calculate distensibility (34):

\[
\text{Dist} = \frac{\pi (d_{\text{max}}/2)^2 - \pi (d_{\text{min}}/2)^2}{\pi (d_{\text{min}}/2)^2 \cdot \text{PP}}
\]

Where, Dist is distensibility, \(d_{\text{max}}\) is maximum diameter, \(d_{\text{min}}\) is minimum diameter and PP is pulse pressure.

**Vascular structure measurements**

Arterial diameters acquired for arterial distensibility measurements were also used to determine resting vascular structure. Measurements of minimum, maximum and mean arterial diameter were determined from carotid and popliteal arteries as described above. Mean arterial diameter was determined using a weighted average calculation (1/3 x systolic diameter + 2/3 x diastolic diameter). Intima-media thickness (IMT) was also determined from carotid images. The average of twenty frames was used to determine IMT from images taken at end-diastole and each frame consisted of between 150-200
measures of IMT within a designated region of interest (AMS II, Chalmers University of Technology, Göteborg, Sweden).

**Vascular function of the popliteal artery**

Flow mediated dilation (FMD) was used to assess vascular function in the legs at the popliteal artery using a combination of Doppler Ultrasound and B-mode imaging. Participants were positioned prone throughout the FMD protocol. Briefly, a pneumatic cuff connected to a rapid inflation system (model E20 and AG101, Hokanson, Bellevue, WA) was placed around the leg 2-3 cm distal to the popliteal fossa. The cuff was inflated to a pressure of at least 250 mmHg to ensure complete occlusion of the popliteal artery. Occlusion was maintained for a period of 5-min. Longitudinal popliteal artery images and blood velocity measurements were made using a 10 MHz (18 participants) or 5MHz (2 participants) linear array pulse Doppler ultrasound probe (System FiVe, GE Medical Systems, Horten, Norway) which was positioned ~3-5 cm proximal to the popliteal fossa either 2 cm proximal or distal to the branching of the middle genicular artery. This was consistent between testing days within each subject to ensure maximal image quality. All pre images were available to the ultrasonographer and displayed on an additional monitor throughout subsequent testing to ensure identical probe placement. Continuous video recording of the image of the popliteal artery was obtained from 15s prior to cuff deflation until 4 min following cuff deflation. In addition, a single heart cycle digital video clip was stored at 15s intervals from 30s to 4 min following cuff deflation. This digital video clip contained images acquired at a rate of 15Hz. Simultaneous to the imaging of the popliteal artery, mean blood velocity (MBV) was obtained using the duplex function of the previously described linear array probe 15s prior to cuff deflation until 25s after cuff
release to determine peak and mean post-occlusion blood flow and shear rates. The raw audio signal corresponding to blood velocity was output from the Doppler ultrasound system to an external spectral analysis system (model Neurovision 500M TCD, Multigon Industries, Yonkers, NY) which applies a fast Fourier transform (FFT) to the raw audio signal to determine MBV continuously. Blood velocity was corrected for insonation angle during post acquisition analysis. MBV, like all other physiological signals, was acquired and recorded using the previously described Powerlab system.

*Image analysis of relative flow mediated dilation*

Using semi-automated analysis software (AMS II, Chalmers University of Technology, Göteborg, Sweden) diameters at end-diastole were acquired from leading edge to leading edge from all images acquired for 4 min post occlusion. The maximal post-occlusion end-diastolic value was compared to resting end-diastolic diameters and expressed as a relative change. The following equation was used to calculate relative FMD (12):

\[
FMD = \frac{FMD_{\text{peak end diastolic diameter}} - \text{Resting end diastolic diameter}}{\text{Resting end diastolic diameter}} \times 100
\]

*Post-occlusion reactive hyperemia*

As previously described, blood velocity measurements were acquired 15s prior to until 25s following cuff release. Mean blood flow was calculated (vessel cross-sectional area x MBV) and used to quantify the hyperemic response. Also, mean wall shear rate (MWSR) was determined as:

\[
\text{MWSR} = \frac{4 \times \text{MBV}}{\text{mean diameter}}.
\]

Where MBV is mean blood velocity.
Normalized flow mediated diameter

Resultant measurements of flow-mediated dilation were normalized to the average MWSR during the first 25 s after cuff release since the amount of dilation is dependent on the resultant hyperemic flow stimulus as represented by mean wall shear rate (36). The following equation was used:

\[
\text{normalized FMD} = \frac{\text{relative FMD}}{\text{MWSR}_{25s}}
\]

Reproducibility of measurements

The reproducibility of the measurements in our laboratory was determined in the 20 participants of this study through evaluation of all measures at 2 time-points prior to training separated by 5-7 days. All participants underwent identical procedures to those outlined above on both of these testing days. Carotid diameter, pulse pressure, and distensibility showed very good reproducibility with coefficients of variation of 2%, 8% and 8% respectively. Popliteal diameter, pulse pressure and distensibility also showed good reproducibility with coefficients of variation of 2%, 8%, and 18%, respectively. Measurements of IMT also showed very good reproducibility with a CV of 5%. Relative popliteal flow mediated dilatation also showed reproducibility with a CV of 28% in 16 participants.

Data Analysis and statistics

Data are expressed as mean ± SD. Measures acquired twice prior to and twice following training were averaged. All variables were analyzed using a two-way mixed analysis of variance, with the repeated factor “Time” (PRE vs POST) and the between factor “Group” (SIT vs ET) using commercially available software (SPSS 11.0 for Mac OS X, SPSS Inc. Chicago, IL). Significance for all analysis was set at P≤0.05. Analyses
of popliteal parameters were performed on 18 rather than 20 participants due to image quality issues with one participant from each group, which were removed from the dataset. Analysis of FMD was performed on 16 participants rather than 20 because of image quality issues with two participants from each of the groups, which were removed from the dataset.

RESULTS

Evidence of a training effect and average weekly work

Training increased $VO_{2\text{peak}}$, with no difference between groups (SIT: PRE 41±2, POST 44±2, ET: PRE 41±2, POST 45±2) (7). As previously described, training reduced steady-state exercising HR and improved Wingate peak power with no differences between groups (7). The SIT group performed on average 225 kJ of work per week while the ET group performed on average 2250 kJ of work per week. The average workload for the sprint intervals was ~500 W while the ET workload was ~150 W.

Heart rate and resting arterial blood pressure

Resting heart rate (p=0.16) and brachial blood pressure were not significantly altered (SBP p=0.69, DBP p=0.38) with training in either group (Table 1).

Arterial distensibility and structure

Popliteal artery distensibility was increased after training in both groups (p<0.01, main effect for time) while differences of carotid artery distensibility were not statistically significant (Figure 1, p=0.29). Ankle pulse pressure was not statistically altered with training in either group (Table 2, p=0.41). However, the change in popliteal cross-sectional area within each heart cycle (delta CSA) increased after training in both
groups (Table 2, p<0.01). Resting arterial structure, as estimated by mean diameter, was not statistically different with training in either the carotid (p=0.10) or popliteal arteries (p=0.10) (Table 2). IMT was not statistically different after training (Table 2, p=0.69)).

**Vascular function assessed by flow mediated dilation**

Absolute popliteal artery flow mediated dilation was improved with training (PRE: 0.28 ± 0.03 mm vs. 0.36 ± 0.03mm); however, it did not reach statistically significant levels (p=0.06). When normalized to resting end diastolic diameter popliteal relative flow mediated dilation increased after training in both groups (p=0.05) (Figure 2a). The enhanced endothelial-dependent dilation was also apparent in both groups after normalization to post-occlusion MWSR (Figure 2b, p=0.047).

Resting blood flow in the popliteal artery was not statistically altered with training (Table 3). Post occlusion blood flow and MWSR following cuff release evaluated during the flow mediated dilation test were also not statistically different (Table 3, p=0.23).

**DISCUSSION**

To our knowledge, this is the first study to show that both low-volume SIT and traditional high volume ET improve popliteal artery distensibility and endothelial function to the same extent in young healthy men and women. As previously reported, the time commitment and total training volumes were much lower with SIT (7) compared to ET. As well, contrary to recent studies of high-intensity training (3, 16), endothelial function improved with SIT. Finally, the increases in $VO_{2peak}$ of ~10% show the effectiveness of both ET and SIT training programs.

**Alterations of peripheral arterial distensibility**
The current observation of improved popliteal artery distensibility is unique because peripheral distensibility is not often measured and in cases when it has been examined there is usually no indication of change with exercise training (10, 19, 35). Only one study that evaluated stiffness in the superficial femoral artery showed improvements with training in older participants (48). Possible reasons for the contrasting findings may include differences in exercise modes (rowers) (10, 35), and populations (elderly) (19). Recent evidence suggests the popliteal artery has some structural and functional characteristics of an elastic artery rather than a muscular artery since it exhibits age-related stiffening (13). The popliteal artery may therefore be a site of significant vascular disease and may be a prime target for exercise intervention.

The mechanisms responsible for training induced improvements in popliteal artery distensibility may be local alterations in vessel wall structural and/or alterations in vascular tone. Substances such as endothelin-1 (ET-1), nitric oxide (NO), prostaglandins, or reactive oxygen species (ROS) regulate resting vascular tone along side sympathetic output and are influenced by training (16, 27, 49). Specifically, ET has been shown to decrease ET-1 (27) increase basal NO levels (49) and reduce basal ROS levels (16), which combine to improve vascular tone and potentially artery stiffness. Structural alterations such as collagen/elastin ratios, reductions of uncoiled collagen fibres, and fewer frayed elastic fibres may contribute to an altered extracellular matrix and a less stiff artery (53). Finally, basal sympathetic tone, which has been shown to acutely regulate distensibility (42), may be altered. Resting diameters of both the popliteal and carotid artery and resting brachial blood pressure were not statistically different with either
exercise training program, limiting the likelihood that basal sympathetic nervous tone accounts for the observed improvements in popliteal distensibility.

*Unaltered central artery distensibility*

In central arteries like the carotid artery, age-associated stiffening is often reversed or attenuated with ET (19, 43, 46, 49) and with combined endurance-strength training (10). However, similar to the current study, young sedentary participants do not exhibit differences compared to endurance-trained athletes (46). Our participants had relatively high levels of baseline carotid distensibility compared to older cohorts leaving little room for improvement with training (46). This is evident from our carotid distensibility measures, which were quite high and similar to those of healthy young subjects previously reported by Miyachi et al. (32) (~0.007 mm/mmHg).

*Structural adaptations- IMT, artery diameter size*

Contrary to our hypothesis, resting central and peripheral artery structure estimated using end-diastolic diameters and carotid IMT were not statistically altered with either training program in the current study. Similar to previous research, IMT in our group was low at baseline, which likely explains why there was no reduction (47). ET lasting 6 weeks has been shown previously to result in increased cross-sectional area of the common femoral artery (31). Contrary to these results, our study did not show an increased popliteal artery diameter which may be related to our observation of improved distensibility or a different time-course of adaptation specific to this artery (20).

*Endothelial function in the exercised limb*

Popliteal artery endothelial function, another measure associated with cardiovascular disease, was improved equally with both training methods. Relative and
normalized FMD increased while the mean hyperaemic blood flow response to occlusion was not statistically different with training. Enhanced FMD accompanied by no change in post-occlusion blood flow points specifically to a training induced improvement. Had there been an increase of the shear stimulus following training, the stimulus may have been the cause of increased FMD rather than exercise training. Mechanistically, improved endothelial function likely relates to reduced oxidative stress (16), an improved antioxidant defense system (38) (circulating and reactive oxygen species scavenging enzyme capacity) or an upregulation of endothelial nitric oxide synthase (eNOS) gene expression (18). All of these factors would improve NO bioavailability upon shear-induced endothelial stimulation and have been noted previously with training.

The current observation of improved popliteal FMD contrasts with the results of two previous studies in healthy young populations (3, 16) that have shown no improvement (16) or reduced endothelial function (3). In the current study, vascular assessment sessions were conducted at 48 and 72 hours following the last training session. This delay before measurement was designed to limit the effects of oxidative stress induced by the final training session on FMD, which has been noted by other researchers following ischemia inducing exercise (30, 41). The stimulus used to evaluate endothelial function was different. Previous studies used invasive measures (intra-arterial Ach infusion) specific to endothelial derived NO release (3, 16), while the current study used flow mediated shear, which may cause the release of other vasoactive substances in the popliteal artery other than NO. Finally, we evaluated endothelial function in the vasculature of the trained limb rather than a non-trained limb and specifically a conduit artery rather than the resistance vessels, as was the case with both previous studies (3, }
16). It is likely that SIT training in the current study resulted in eNOS protein upregulation specific to this vessel that also contributed to greater local NO bioavailability beyond improved or maintained antioxidant defenses.

**Mechanistic insight into improved aerobic performance**

As described previously $VO_{2\text{peak}}$ was improved in the current study to the same degree with SIT and ET (7). Structural and functional vascular improvements may contribute to better performance noted previously with high intensity training (8, 24). Greater peripheral artery distensibility and enhanced endothelial-dependent vasodilation at the onset of exercise may facilitate greater oxygen availability through more efficient blood delivery; however, further study is needed to determine cause and effect.

**Study limitations and interpretations**

Although the magnitudes of the adaptations in vascular structure and function were similar between SIT and ET, the required dose of ET needed to improve popliteal artery endothelial function and distensibility is unknown and requires specific dose-response research. We also acknowledge that SIT is not practical for some populations as it requires high levels of motivation and possibly supervised training facilities. Therefore, further studies that determine ideal interval exercise intensities specific for each disease condition are warranted. In fact, initial studies in patients with coronary artery disease and chronic heart failure show that the effectiveness of a high-intensity training regime may be greater compared to traditional ET methods (51, 52) and high intensity aerobic interval training is now being recommended for several disease populations.

As well, we acknowledge that normalization of FMD to the area under the flow curve until the time of maximal dilation is the optimal method (36). We were unable to
capture the full post occlusion blood flow response due to technical limitations; however, given the lack of difference between our pre and post hyperaemic responses (MWSR$_{25s}$), we believe it is reasonable to assume we have accounted for most of the stimulus through normalization to the shear stimulus for the first 25 s after cuff release.

**PERSPECTIVES AND SIGNIFICANCE**

Both training protocols improved $VO_2$peak yet the actual total work performed over the period of 6 weeks was much different highlighting the time-efficiency of SIT. From a vascular health perspective this study shows that the beneficial effects of exercise on prognostic indicators of cardiovascular disease, such as peripheral artery distensibility and endothelial function, are modified effectively with either ET or SIT. Extension of interval training has already begun in populations with compromised health such as those with coronary artery disease (51), chronic obstructive pulmonary disease (11), and chronic heart failure (52). Whether the vascular benefits outlined in the present study are apparent in these populations awaits further attention.
Table 1 Subject characteristics over the course of 6 weeks of either sprint interval or endurance training.

<table>
<thead>
<tr>
<th></th>
<th>Sprint (n=10)</th>
<th></th>
<th>Endurance (n=10)</th>
<th></th>
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<tbody>
<tr>
<td></td>
<td>PRE</td>
<td>POST</td>
<td>PRE</td>
<td>POST</td>
</tr>
<tr>
<td>Age (years)</td>
<td>23.6 ± 3.2</td>
<td>___</td>
<td>23.0 ± 2.4</td>
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<tr>
<td>Height (cm)</td>
<td>171.2 ± 7.3</td>
<td>___</td>
<td>175.2 ± 12.1</td>
<td>___</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>69.1 ± 9.4</td>
<td>68.3 ± 8.9</td>
<td>75.4 ± 13.3</td>
<td>74.9 ± 12.7</td>
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<td>BMI (kg ·m⁻²)</td>
<td>23.6 ± 3.0</td>
<td>23.3 ± 3.0</td>
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<td>24.2 ± 2.0</td>
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<td>Heart Rate (bpm)</td>
<td>57 ± 8</td>
<td>56 ± 5</td>
<td>65 ± 10.0</td>
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<td>Brachial DBP (mmHg)</td>
<td>63 ± 5</td>
<td>63 ± 6</td>
<td>66 ± 5</td>
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<td>Brachial SBP (mmHg)</td>
<td>112 ± 9</td>
<td>114 ± 10</td>
<td>124 ± 14</td>
<td>121 ± 13</td>
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<td>Brachial MAP (mmHg)</td>
<td>80 ± 6</td>
<td>80 ± 7</td>
<td>85 ± 7</td>
<td>83 ± 7</td>
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</tbody>
</table>

Data are mean ± SD. Where, BMI is body mass index, BP is blood pressure, D is diastolic, S is systolic and MAP is mean arterial pressure.
Table 2 Vascular structure changes with sprint interval or endurance training.

<table>
<thead>
<tr>
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<th>Sprint (n=10)</th>
<th>Endurance (n=10)</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>PRE</td>
<td>POST</td>
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<tr>
<td>Carotid IMT (mm)</td>
<td>0.43 ± 0.04</td>
<td>0.42 ± 0.04</td>
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<td>Carotid Mean Dia (mm)</td>
<td>6.3 ± 0.3</td>
<td>6.3 ± 0.3</td>
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<td>Carotid Delta CSA within the heart cycle (mm²)</td>
<td>7.0 ± 0.4</td>
<td>6.6 ± 0.4</td>
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<td>Carotid PP (mmHg)</td>
<td>39 ± 8</td>
<td>40 ± 9</td>
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<td>Poiliteal Mean Dia (mm)</td>
<td>5.5 ± 0.5</td>
<td>5.3 ± 0.6</td>
</tr>
<tr>
<td>Poiliteal Delta CSA within the heart cycle (mm²)</td>
<td>2.0 ± 0.5</td>
<td>3.0 ± 1.2*</td>
</tr>
<tr>
<td>Ankle PP (mmHg)</td>
<td>60 ± 8</td>
<td>61 ± 6</td>
</tr>
</tbody>
</table>

Data are mean ± SD, * p<0.05 versus PRE, main effect for time. Where, Dia is diameter, CSA is cross-sectional area, PP is pulse pressure and PWV is pulsewave velocity.
Table 3 Flow and shear rate at rest and following cuff release (FMD protocol) before and after sprint interval or endurance training

<table>
<thead>
<tr>
<th></th>
<th>Sprint (n=10)</th>
<th>Endurance (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>PRE</td>
<td>POST</td>
</tr>
<tr>
<td>REST</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Popliteal MBV (cm ·sec⁻¹)</td>
<td>1.2 ± 0.4</td>
<td>1.4 ± 0.4</td>
</tr>
<tr>
<td>Popliteal MBF (ml ·min⁻¹)</td>
<td>17 ± 6</td>
<td>18 ± 7</td>
</tr>
<tr>
<td>Popliteal MWSR (sec⁻¹)</td>
<td>0.9 ± 0.3</td>
<td>1.0 ± 0.4</td>
</tr>
<tr>
<td>REACTIVE HYPERAEMIA</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Popliteal MBV₂₅s (cm ·sec⁻¹)</td>
<td>17.9 ± 1.8</td>
<td>16.7 ± 1.5</td>
</tr>
<tr>
<td>Popliteal MBF₂₅s (ml ·min⁻¹)</td>
<td>244 ± 48</td>
<td>226 ± 76</td>
</tr>
<tr>
<td>Popliteal MWSR₂₅s (sec⁻¹)</td>
<td>13.5 ± 3.9</td>
<td>12.5 ± 2.7</td>
</tr>
</tbody>
</table>

Data are mean ± SD, Where, MBV is mean blood velocity, MBF is mean blood flow, MWSR is mean wall shear rate. Reactive hyperaemic variables are the 25s average.
ACKNOWLEDGEMENTS

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REFERENCES


FIGURE LEGENDS

Figure 1 Arterial distensibility of the a) popliteal and b) carotid arteries before and after
6 weeks of either sprint interval (SIT) or endurance training (ET). Popliteal
artery distensibility was higher after training in both groups, whereas carotid
artery distensibility was unchanged. Values are mean ± SD, n = 10 per group. *
P<0.05 versus PRE, main effect for TIME.

Figure 2 Relative (a) and normalized (b) flow mediated dilation of the popliteal artery
before and after 6 weeks of either sprint interval (SIT) or endurance training
(ET). Relative and normalized popliteal FMD was higher after training in both
groups. Values are mean ± SD, n=8 per group. * P<0.05 versus PRE, main
effect for TIME.
Arterial distensibility of the a) popliteal and b) carotid arteries before and after 6 weeks of either sprint interval (SIT) or endurance training (ET). Popliteal artery distensibility was higher after training in both groups, whereas carotid artery distensibility was unchanged. Values are mean ± SD, n = 10 per group. * P<0.05 versus PRE, main effect for TIME.

Arterial distensibility of the a) popliteal and b) carotid arteries before and after 6 weeks of either sprint interval (SIT) or endurance training (ET). Popliteal artery distensibility was higher after training in both groups, whereas carotid artery distensibility was unchanged. Values are mean ± SD, n = 10 per group. * P<0.05 versus PRE, main effect for TIME.
Relative (a) and normalized (b) flow mediated dilation of the popliteal artery before and after 6 weeks of either sprint interval (SIT) or endurance training (ET). Relative and normalized popliteal FMD was higher after training in both groups. Values are mean ± SD, n=8 per group. * P<0.05 versus PRE, main effect for TIME.