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Kathryn Holloway, Denise Roche and Peter Angell.

Evaluating the progressive cardiovascular health benefits of short-term high intensity interval training.

Liverpool Hope University, School of Health Sciences, Hope Park, Taggart Avenue, Liverpool, L16 9JD.

Corresponding author:

Kathryn Holloway

hollowk@hope.ac.uk

01512913227

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Abstract

Purpose High-intensity training is recognised as a time efficient way of improving aerobic fitness. However, there is a lack of consensus regarding the temporal nature of adaptation response and which peripheral and cardiac changes occur using the same exercise stimulus and protocol. Therefore, this study aimed to evaluate the progression of vascular and cardiac changes over a 6-week training period.

Methods Twelve healthy males (age 21±2 years; 42.5±8.3 $ml.min^{-1}.kg^{-1}$) participated in a high-intensity training programme consisting of 1-minute sprints, interspersed with 2 minutes active recovery, 3 days/week for 6 weeks on a cycle ergometer. Cardiac, vascular, blood lipids and VO_{2max} measurements were taken at 0, 3 and 6 weeks and compared against a participant-matched control group (age 21±2 years; 37.7 ± 8.3 $ml.min^{-1}.kg^{-1}$).

Results There was a significant improvement in VO_{2max} (42.5±8.3 $ml.min^{-1}.kg^{-1}$ to 47.4 ± 8.5 $ml.min^{-1}.kg^{-1}$; p=0.009) in the training group and a significant decrease in systolic blood pressure (8%) from 0-6 weeks (p=0.025). There was a small yet significant decrease in ejection fraction and increased end-systolic volume in both groups over time (p=0.01) with no significant interaction effect (p>0.05). A between-group difference in peak velocity of early diastolic mitral annular motion was also observed (p=0.01). No improvements were seen in blood lipid profiles, central arterial stiffness and cardiometabolic risk score.

Conclusions Six weeks of high-intensity training increases aerobic fitness and is enough to stimulate initial reductions in peripheral pressure, but not sufficient to elicit structural and functional cardiac changes, reduce arterial stiffness or lower CV risk.

Key words

High-intensity, exercise training, cardiac function, vascular structure, cardiovascular risk

Abbreviations

A	Peak velocity of late transmitral flow
A’	Peak velocity of diastolic mitral annular motion
AIx	Augmentation index
AP	Central augmented pressure
a-VDO_2	Arterial-venous difference
BMI	Body mass index
CO_{max}	Maximal cardiac output

1	CRF	Cardiorespiratory fitness
2	CVD	Cardiovascular disease
3	DBP	Diastolic blood pressure
4	DP	Central aortic diastolic pressure
5	E	Peak velocity of early diastolic transmitral flow
6	E’	Peak velocity of early diastolic mitral annular motion
7	EF	Ejection fraction
8	FBG	Fasting blood glucose
9	HDL-C	High-density lipoprotein cholesterol
10	HIIT	High intensity interval training
11	HR	Heart rate
12	IVSd	Interventricular septum thickness at end -diastole
13	LDL-C	Low-density lipoprotein cholesterol
14	LV	Left ventricle
15	LVEDV	Left ventricular end-diastolic volume
16	LVESV	Left ventricular end-systolic volume
17	LVIDd	Left ventricular internal diameter end diastole
18	LVIDs	Left ventricular internal diameter end systole
19	LVPWd	Left ventricular posterior wall thickness at end –diastole
20	MAP	Mean arterial pressure
21	NO	Nitric oxide
22	PP	Central aortic pulse pressure
23	PWV	Pulse wave velocity
24	S’	Peak velocity of systolic mitral annular motion
25	SBP	Systolic blood pressure
26	SIT	Sprint interval training
27	SP	Central aortic systolic pressure

SV	Stroke volume
SV_{max}	Maximal stroke volume
TC	Total cholesterol
TG	Triglycerides

Introduction

An emerging body of evidence is highlighting the positive impact that high intensity interval training (HIIT) has on an individual's physical health. HIIT can be defined as repeated short bouts of high intensity aerobic exercise (typically >90% VO_{2max}; Kessler et al. 2012) interspersed by periods of lower intensity passive or active recovery (Astorino et al. 2012). Numerous training studies have demonstrated the effectiveness of endurance training on cardiorespiratory fitness (CRF; Kemi et al. 2005; Leon, 1997). However, more current research has identified HIIT as a more time efficient method compared to endurance training (Gormley et al. 2008; Helgerud et al. 2007) with HIIT eliciting greater increases in CRF within a variety of population groups including healthy sedentary (Weston et al. 2014) and heart failure patients (Angadi et al. 2015). HIIT has also shown increased adherence rates compared to continuous training in healthy (Heisz et al., 2016) and diseased groups (Jung et al., 2015), highlighting its potential for wider use amongst the general population. With a large percentage of the population not meeting the current physical activity guidelines, and lack of time as the biggest barrier (Godin et al. 1994), understanding how HIIT can improve overall cardiovascular health is fundamental. In addition, with 64% of participants dropping out of a long-term exercise programmes within the first 8 weeks (Heisz et al., 2016), it is important to understand the immediate benefits of short-term HIIT.

Fifteen minutes of daily high intensity exercise can reduce all-cause mortality risk by 25%, which is equivalent to completing 60 minutes of daily moderate intensity physical activity (Wen et al. 2011). It has therefore been suggested that there is an inverse dose-response relationship between mortality risk and the proportion of high intensity exercise performed, which would suggest a need to endorse high intensity exercise (Gebel et al. 2015). More specifically, HIIT has been shown to improve a variety of direct and indirect cardiovascular indices, with as little as six sessions of HIIT improving VO_{2max} by 10% (Esfandiari et al. 2014). Notwithstanding the improvements seen in CRF, the key indices contributing to this enhancement remain elusive and so have been associated with a number of different physiological adaptations. MacPherson et al. (2011) ascertained that 6 weeks of sprint-interval training mediated no change in cardiac function, but the augmentation in CRF was increased due to peripheral adaptations. This has primarily been associated with enhanced vascular structure and function, provoking a greater blood flow through the vessels, consequently increasing oxygen availability and promoting a greater shear stress-induced nitric oxide (NO) bioavailability (Wisloff et al. 2007). In support of this, Thijssen et al. (2009) showed a parallel relationship between an increase in blood flow and increase in shear stress with increasing exercise intensity. Consequently, an increase in NO release has been associated with an improvement in CV risk profile with recurring intermittent increases in shear stress instigating vascular adaptations and remodelling of the artery (Wilson et al. 2015). The augmented shear associated with high-intensity exercise would predict an increased likelihood of notable vascular adaptations to HIIT, although there is limited data on the response in healthy individuals with studies mainly having been undertaken in diseased populations (Thijssen et al. 2009). In fact, Tjonna et al. (2011) showed that HIIT is sufficient in maintaining normal vascular function in healthy adults but very limited potential for further vascular improvements due to the healthy nature of the subjects. Despite this, Tinken et al. (2008) showed that exercise training initiates improvement in vasodilator function in healthy young men in as little as 2 weeks. This therefore raises the question of the effectiveness of HIIT in the general population who are currently free from cardiovascular disease but are of a sedentary nature, since peripheral adaptations are associated with CRF and consequently CV risk.

Conversely, increases in cardio-respiratory fitness have also been associated with cardiac adaptations that have predominantly occurred during the active recovery phase that occurs in-between the high-intensity intervals. Astorino et al. (2017) found that 6 weeks of HIIT significantly increased CRF through an increase in maximal stroke volume (SV_{max}) and maximal cardiac output (CO_{max}), but found no increase in arterial-venous difference (a-VDO_2). Cardiac output is enhanced through the increase of LV volume and a decrease in peripheral resistance (Tschakert et al. 2013) which enhances end-diastolic volume and stroke volume (SV). Buchheit et al. (2013) describes that working at an intensity close to maximal SV triggers these cardiac adaptations in HIIT. However, it is important to note that HIIT may not induce structural changes quickly (<6 weeks) as increases in SV have also been attributed to an increase in plasma volume thus eliciting increases in blood volume and subsequently SV. Therefore, it is important to ascertain at which point cardiac structural changes appear with HIIT since the intermittent nature of exercise and recovery has the potential to improve CRF and thus reduce CVD risk.

High intensity interval training protocols vary amongst research groups in regards to exercise intensity, work: rest ratios, duration and frequency of exercise. It is therefore difficult to fully elucidate the extent of potential simultaneous peripheral and cardiac changes that are induced by HIIT. Only one study to date has incorporated both cardiac and vascular measurements using HIIT in adult males (Heydari et al. 2013) but this study adopted a sprint interval training (SIT) based protocol (8 second sprint, 12 second recovery) rather than an HIIT protocol. HIIT has been shown to produce a lower perceived exertion and a higher positive affect in comparison to SIT (Wood et al. 2015) and therefore could be deemed more appropriate for the general population. In addition, HIIT has also been deemed as a safe exercise regime to undertake even with high risk groups (Weston et al. 2014), thus furthermore allowing the protocol to be used beyond the general population if required.

Exercise intensity, as opposed to duration, has been deemed as the most important variable in determining CRF (Karlsen et al. 2017). With a clear association between cardio-respiratory fitness and CVD risk (Karlsen et al. 2017), it is important to understand which physiological indices are primarily adapting with HIIT and the time-course of responses. Therefore, this study investigated the simultaneous changes in peripheral and cardiac indices from a temporal perspective using a HIIT based protocol to elucidate potential cardiovascular improvements. As a consequence, we hypothesise that HIIT will significantly improve indices of vascular function, which in turn may elicit cardiac changes in the form of an improvement in systolic function.

Methods

Study design

Subjects were individually assigned to one of two parallel groups, an exercise intervention and control group. The study was an open-label study, with an allocation ratio of 1:1. The study took place in the exercise physiology laboratories, Liverpool Hope University, Liverpool, UK from January 2016 to April 2016.

Subjects

Subjects were recruited through University advertisements, email bulletins and notice boards. Inclusion criteria included healthy males, non-smokers, free from cardiac or respiratory pathologies and not taking regular medication. Twenty-one subjects were recruited, with 14 subjects assigned to the intervention group to complete a 6-week high-intensity exercise programme (age 21±2 years, body mass 73.5±9.5 kg, BMI 24±3 kg/m^2) and 7 subjects were assigned to a control group (age 21 ± 2 years, body mass 71.4±7.6 kg, BMI 23±3 kg/m^2). Two subjects from the intervention group withdrew from the study due to injury that was gained outside of the exercise programme.

This study was approved by the Ethics Committee of Liverpool Hope University and all subjects gave their written informed consent.

Experimental design

All physiological measurements were taken at baseline, 3 weeks and 6 weeks. All subjects refrained from consuming caffeine (6 hours), food (12 hours), alcohol (12 hours) and strenuous exercise (24 hours) before measurements were conducted. Subjects successively completed 18 HIIT sessions, with an average 89% completion rate. Subjects were removed from the study if their completion rate was below 80%. Subjects were tested and trained at the same time of day to avoid diurnal variation. Post training tests at 3 and 6 weeks were taken within 48-120 hours after the last HIIT session. Subjects were asked to maintain the same dietary intake and physical activity level as undertaken prior to the study.

All data was collected over 2 days of testing in a standard laboratory environment (room temperature 22°C, relative humidity 10%).

Anthropometrics

Total body and segmental body fat analysis was conducted using bioelectric impedance (Tanita T5896, Tokyo). Height was measured to the nearest 0.1cm using a stadiometer (SECA, Germany) and weight was measured to the nearest 0.1kg using scales (SECA, Germany). Body mass index was then calculated using the formula body mass (kg) divided by height squared (m).

VO_{2MAX}

Subjects performed the VO_{2max} test on a cycle ergometer (Lode Excalibur Sport, Lode B.V Technology, Groningen, Netherlands). Breath by Breath analysis was collected through a Respiratory Ergostik (Geratherm, Bremen, Germany), evaluating the expired O_2 and carbon dioxide (CO_2) using the Blue Cherry software (CV% = 3%). Calibration of the Ergostik occurred prior to every test with O_2, CO_2 and nitrogen gas percentages accounting for 16%, 4% and 80% respectively. Subjects were fitted with a H2 Polar HR monitor (Polar Team 2 Heart Rate Monitor System) and then sat resting for 2 minutes to gain baseline respiratory values. Following this, a 5-minute warm up was instructed at a power output corresponding to that of the initial stage of the ramp test (50 watts). Once the warm up was complete, the ramp test instantaneously began with intensity increasing 25 watts per minute. Subjects cycled at an rpm between 60 and 70 for as long as possible until exhaustion. The test was terminated when rpm fell below 60 for longer than 10 seconds, while HR within 10 beats of the age related max and RER $\geq$1.15 displayed if a true VO_{2max} was achieved. Following this, a 2-minute cool down was advised, at 50 watts. The VO_2 for each 30 second epoch was averaged. The maximum 30 second average value was reported.

Assessment of vascular structure and function.

For all vascular measurements, subjects remained in a supine position for 10 minutes before testing commenced in a temperature-controlled room.

Pulse wave analysis

Pulse wave analysis measurements were attained using a SphygmoCor System (AtCor Medical, Sydney, Australia). An automated cuff was placed around the upper left arm and was inflated to measure brachial systolic and diastolic pressures and central pressures. Measurements were taken twice. A third test was completed if the previous two measurements were >5mmHg different from each other. Following this, the cuff was re-inflated to capture the brachial waveforms and used to determine pulse wave reflection characteristics. Central

Aortic Diastolic Pressure (DP) was calculated by the lowest pressure value recorded in a pulse and Central Aortic Systolic Pressure (SP) was calculated as the maximum pressure during aortic injection. Central Aortic Pulse Pressure (PP) was then calculated as Central Aortic Systolic Pressure (SP) minus the Central Aortic Diastolic pressure (DP). The Central Augmented Pressure (AP) was calculated as the difference between the two pressure peaks during systole and the Augmentation Index (Alx) is the ratio of AP to PP. Mean Arterial Pressure (MAP) and Heart rate (HR) were also measured.

Pulse wave velocity

Pulse wave velocity measurements were attained using a SphygmoCor System (AtCor Medical, Sydney, Australia). Measurements were standardised according to recommendations by Van Bortel et al. (2012). PWV was taken from the left carotid artery to the left femoral artery with subjects laying in a supine position. The distance between the carotid site and the manubrium sterni and the carotid to femoral artery distance was determined to the nearest millimetre (mm). PWV was measured by aligning the high-fidelity tonometer with the carotid artery pulse, holding in position until full assessment was completed. A pressure cuff was positioned around the upper left thigh. Time delays between proximal and distal waveforms were then used to calculate PWV (m/s) by the following equation: $PWV = D/\Delta t$ in which D is the distance (m) and Δt is the time interval (s). The SphygomoCor software will only accept values that are within a standard deviation of $\leq$10%, which has been shown to be highly reproducible (Wilkinson et al. 1998).

Vascular structure

Brachial artery imaging was conducted using a VividQ ultrasound scanner (GE Healthcare, UK). Scans were performed on the right arm with the subject in a supine position. The ultrasound probe (6-13MHz) was placed above the antecubital fossa in the longitudinal plane. Femoral artery measurements were taken on the right leg, in a supine position, at the midpoint of the inguinal ligament. Three wall-to-wall measures were taken of each artery using the Vividq calliper option and an average was calculated to give a true diameter (cm) value.

Assessment of cardiac structure and function.

Global and segmental cardiac structure and function were assessed using ultrasound echocardiography (Vivid Q, GE Healthcare, Norway). Images were taken of the left ventricle (LV) in multiple planes from parasternal and apical acoustic windows. All measurements were performed by a single experienced echocardiographer with the participant in the left lateral decubitus position.

Parasternal long axis views allowed the collection of M-mode images at the tips of the mitral valve leaflets perpendicular to septal and posterior walls taking care to ensure clear endocardial definition. From M-mode traces septal (IVSd) and LV posterior wall thickness (LVPWd) in diastole as well as LV chamber dimensions at end-diastole and systole (LVIDd, LVIDs) were assessed following American Society of Echocardiography guidelines (Lang et al. 2015).

Apical 4-chamber views were digitised to assess LV end-diastolic (LVEDV) and end-systolic (LVESV) volumes, which allowed the estimation of stroke volume (SV) and ejection fraction (EF) using Simpsons' bi-plane method. From the 4-chamber view, colour Doppler and then pulsed wave Doppler were used to assess peak flow velocities across the mitral valve. Using a 4 mm sample volume in the area of peak flow LV early (E) and late (A) diastolic in-flow velocities were recorded. From the same view, tissue-Doppler measures of myocardial wall velocities were recorded. Taking care to adjust filters and scale and with the septal wall parallel to the ultrasound beam, we interrogated the mitral annulus at the septal wall, recording peak systolic (S') as well as early (E') and atrial (A') diastolic tissue velocities. The ratio of early to late diastolic filling and tissue velocities were formulated.

Two-dimensional image optimisation including maintaining frame rate between 40 and 90 fps was performed. For all measurements, images were analysed off-line by a single experienced technician. Data reflect the average of 3-5 continuous cardiac cycles.

Blood analysis

A fasting 35 μm finger-capillary blood sample was collected in sterile conditions for the subsequent determination a number of metabolic markers including: fasting blood glucose (FBG, $mmol.L^{-1}$), Total Cholesterol (TC, $mmol.L^{-1}$), low-density lipoprotein cholesterol (LDL-C, $mmol.L^{-1}$), high-density lipoprotein cholesterol (HDL-C, $mmol.L^{-1}$), Triglycerides (TG, $mmol.L^{-1}$) and TC:HDL-C ratio. The free-flow whole blood was analysed using a LDX Cholestech analyser (Cholestech Corporation, USA).

Calculation of clustered metabolic risk score

Standardised z scores were calculated for percentage body fat, TC-HDL ratio, Glucose, Triglycerides and MAP (based on the clustered risk score by Lamb et al. 2016). These were then summed to create a composite clustered risk score. This composite score of cardiometabolic risk may compensate for day to day fluctuations in individual risk factors.

High intensity training intervention

A five minute warm up of unloaded cycling at 60rpm preceded five 1 minute cycling bouts at 90% of the subject's VO_{2max}, interspersed with 2 minutes, resistance free active recovery at 60 rpm. This was repeated 3 times a week for 6 weeks, reaching 15 minutes of exercise per session, using a cycle ergometer (Monark, Ergomedic 874E, Vansbro, Sweden) throughout. All training sessions were supervised to ensure completion of training. Subject's training load was adjusted at 3 weeks to align with any changes in VO_{2max} found at the 3-week assessment point.

Statistics

All statistical analysis was performed using SPSS v. 23 (IBM Statistics, UK). To determine differences at 0, 3 and 6 weeks, an Analysis of Covariance (ANCOVA) was performed with the pre-variable as the covariate and the group as the between-subjects factor. Bonferroni pairwise comparisons were used to determine at which time points these differences occurred. Partial eta squared (ŋ2) values provide estimates of effect sizes for the main analyses where partial ŋ2 ≥ 0.01, 0.09 and 0.25 classified as small, medium and large effect sizes respectively (MRC, 2009).

Results

Aerobic capacity

Pairwise comparisons revealed that there was an improvement in maximal oxygen consumption (VO_{2max}) in the exercise group from 0-6 weeks of 10% (p=0.009; partial $ŋ^2$= 0.191). The control group exhibited no change in VO_{2max} across the 6 weeks (p=.686).

Vascular structure and function

There was a significant decrease in peripheral SBP (p=0.005; partial $ŋ^2$= 0.232) in the training group from 0-6 weeks. There was also a significant main effect for PP over time (p=0.041; partial ŋ2= 0.204). There were no other significant group differences in vascular structure and function across the training programme.

PWV and vascular structure measurements did not significantly change after the exercise intervention (table 1).

Cardiac structure and function

There was a small yet significant decrease in ejection fraction and increase in end-systolic volume in both groups over time (p=0.01; partial $ŋ^2$= 0.295 and partial $ŋ^2$= 0.429 respectively) with no significant group effect or interaction (p>0.05). A between group difference in E was also observed (p=0.01; partial $ŋ^2$= 0.361) however this was due to greater early diastolic filling velocities in the training group pre-intervention that persisted throughout the study. All other cardiac measurements were unchanged by the training intervention and not significantly different from the control group.

Blood analysis and CV risk factors

There were no significant group differences or training effects on clustered risk score (table 1; F (2, 22) = 0.483, p=0.623). Anthropometric measurements were also not changed with the training programme (p>0.05).

Discussion

This study, to current knowledge, is the first to investigate the simultaneous temporal changes in peripheral and cardiac indices to elucidate the cardiovascular response to HIIT in a 6 week period. The main finding of this study is that HIIT elicited improvements in aerobic capacity, which are likely to be attributed to improved oxygen delivery and/or mitochondrial adaptations since no improvements in cardiac function or vascular function were seen. In addition, there were no beneficial reductions in CV risk factors or central arterial stiffness, but an improvement in peripheral blood pressure was evident.

Enhancements in cardiorespiratory fitness are strongly associated with cardiovascular risk reduction (Wilson et al. 2015). Kaminsky et al. (2013) reported that a rise of one metabolic equivalent (3.5 ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) in VO_{2max} improved CVD survival by 10-25%. The present study found that aerobic fitness was significantly increased by 11% (5ml $O_2 \cdot kg^{-1} \cdot min^{-1}$) with the 6 week training programme, suggesting a reduction in overall CV risk. However, the clustered metabolic risk score was not significantly changed with training, suggesting that a longer training period or greater volume of training is required to improve cardiometabolic profiles. This is supported by Nybo et al (2010) who did not find any changes in total cholesterol, HDL and LDL after 12 weeks of HIIT. However, they did show a significant reduction in fasting glucose levels from 5.7±0.2mM to 5.2±0.1mM. Our subjects already had baseline fasting glucose values of 5.2±0.4mM and this therefore could explain why no reductions were observed with the training program, suggesting the lowest threshold had already been reached.

An increase in VO_{2max} of 11% is particularly large in comparison to similar studies, with Helgerud et al. (2007) showing an increase of only 7.2%. However, Gormley et al. (2008) have previously shown that 6 weeks of high intensity training improves VO_{2max} with moderate- (10.0%), vigorous- (14.3%), and the near-maximal-intensity (20.6%) exercise. They concluded that the highest intensity used for the significant portion of training was more important than the mean intensity across the exercise protocol. Across our 15 minute protocol, 33% of that time was spent at a high intensity work rate of 90% VO_{2max}. However, Helgerud et al. (2007) included 57% of high intensity work at 88% VO_{2max} suggesting that the proportion of high intensity training used is not necessarily indicative of improvements in VO_{2max} with high intensity training. Bacon et al. (2013) has recently observed that longer high intensity intervals of 3-5 minutes can generate a marked increase in VO_{2max}. However, our study deliberately included 1-minute work periods since Bacon et al. (2013) also observed that it would be unrealistic for significant proportions of the population to participate in such an exercise program that causes individuals to reach their upper limit for VO_{2max}. Our current study did not have any withdrawals from the training group, but did eliminate two subjects for not meeting the threshold attendance. Therefore, using this time-efficient (15 minutes total exercise time) design may be a useful tool in significantly improving aerobic fitness in young adults.

An association between higher aerobic fitness and lower central arterial stiffness has been previously identified (Eugene et al. 1986; Feske et al. 1988; Tarnawski et al. 1994), indicating the influence that aerobic capacity has on large artery stiffness. There was no significant change in PWV with training, suggesting no change in aortic stiffness, and no significant change in augmentation index (Alx) over the 6-week training period. Alx provides an indicator of systemic arterial stiffness by measuring the contribution that the wave reflection makes to the arterial pressure waveform. More recently, a dose-response relationship has been found between exercise intensity, rather than volume of exercise, in improvement in Alx (Ashor et al. 2014). However, 6 weeks of high intensity exercise training was not enough to elicit any improvement in Alx. Padilla et al. (2008) and Tanaka et al. (2006) have both shown that high intensity exercise is associated with the highest post-exercise shear stress. Therefore, augmented NO release would result in a greater reduction in peripheral vascular resistance and, consequently, Alx (Ashor et al. 2014). Conversely, Goto et al. (2003) showed that high intensity exercise augmented oxidative stress, which may negate the positive effect that shear stress has on endothelial function. In addition, high intensity exercise has been shown to stimulate increases in blood flow leading to a non-laminar shear stress and thus resulting in no enhancement in endothelial function (Harrison et al., 2006). Alx can be influenced by a number of factors including age, gender, height, heart rate and some blood lipids (Wilkinson et al. 2002). All subjects were matched in age and gender, and similar in height (176±5cm). However, no significant change in Alx was still observed when matched to 75% heart rate (Alx@75%).

In relation to this, there was a significant decrease in peripheral SBP of 7% following 6 weeks of training, suggesting an improvement in vascular resistance. Nybo et al. (2010) showed the same decrease (-8mmHg) following 12 weeks of HIIT, however these same changes also appeared in the prolonged running and strength training groups. Cornelissen et al. (2013) showed that a lower training intensity had a small effect (+0.073) on reducing SBP in comparison to moderate (-4.8) and high (-3.6) intensity exercise. However, these effects were dependent on hypertensive status. High intensity interval training may lower SBP due to an increase in endothelial NO availability resulting from an increase in blood velocity (Batacan et al., 2016). However, with no changes in Alx or central pressures, it would suggest that there may be other mechanisms which are enhancing vascular function with HIIT beyond increasing shear stress.

However, it is important to note that recent studies have shown that central pressure is more strongly related to future cardiovascular events than brachial pressures (Roman et al. 2007; Pini et al. 2008; Jankowski et al. 2008). Although central pulse pressure (PP) showed a significant main effect over time, both groups exhibited a decrease in PP which suggests a non-training effect. Smulyan et al. 2003 stated that PP measurements using the SphymoCor system can be variable (SD=13.6mmHg). This therefore may explain the significant decrease of 3 mmHg and 2mmHg found in the training and control groups respectively. In addition, central arterial pulse pressure has also been associated with carotid intima-media thickness (Boutouyrie et al. 1999) and therefore with no changes in brachial or femoral vascular diameter, it is suggestive that the HIIT programme had no effect on vascular structure.

There were no significant changes to any cardiac structural measures in response to training after 3 or 6 weeks. Previous findings have demonstrated an impact of HIT training on LV morphology, however the present study found no change in cardiac structural measures most likely due to the relatively short-duration of the study (Wislof et al, 2007). Whilst significant decreases in ejection fraction were observed in both groups, these changes were small and likely to be of little consequence as values were within the normal interstudy reliability range of 7% (Grothues et al. 2002). There was a significant difference in ESV between groups at each time-point but this is likely due to pre-intervention differences. With no meaningful change in systolic function, these differences could be due to subtle heart rate variations with changes within normal measurement variation.

Previous research has found improvements in autonomic cardiovascular control following 12 weeks of training (Hedari et al. 2013), whilst improvements in cardiac function have been seen in cardiac patients including those with heart failure and coronary artery disease (Guiraud et al. 2012) and additionally, a reduction in endocardial damage (Cassidy et al. 2017). The lack of meaningful changes in cardiac function in the present study may be as a result of the good health status observed in both the training and control groups. In contrast to patients with cardiac disease, the subjects in the present study may have already been toward the upper end of the cardiac adaptations possible without substantial increases in training load. Contrastingly, the length of intervention may not have been sufficient to elicit significant structural and functional changes at the cardiac level as the majority of studies who have seen these improvements have been greater than 12 weeks (Wisloff et al. 2007).

Our study is strengthened by the supervision of all training sessions and the continual verification of participants working at the correct intensities and completing the training protocol. Participants were recreationally active and were therefore asked to continue their normal habitual exercise that was monitored by training logs during the 6 weeks. Control participants were recruited by convenience rather than random assignment and were matched for age, physical activity levels and health status. They did exhibit a lower mean VO_{2max} (4.75 $ml.min^{-1}.kg^{-1}$ mean difference) compared to the training group, but pairwise analysis showed no significant difference between the groups at baseline (p=.242). Although the control group were asked to maintain normal habitual exercise, this was not monitored. Whilst our participants were classed as active, none of them were attaining the 150 minutes per week (self-reported) that is nationally recommended (NHS, 2017). Therefore, even though it might be supposed that greater improvements may have been gained in more sedentary or diseased individuals, it is important to ascertain the degree of improvement that a healthy adult can obtain, since 33% of men are still not meeting the current recommended physical activity guidelines (British Heart Foundation, 2015). Although Gold standard methods were mainly employed, the measurement of resting arterial diameter was used solely as a marker of vascular structure; additional methods which assess functional vascular responsiveness (e.g. FMD) should be included in future studies. Furthermore, heart rate was not monitored during the exercise training which may have helped ensure participants were operating at the desired intensity.

Overall, six weeks of high-intensity training is enough to stimulate an increase in VO_{2max} and a reduction in peripheral systolic pressure, but did not elicit significant structural and functional changes at a cardiac level or improvements in central arterial stiffness and CV risk factors.

This study intentionally used a 15-minute protocol, since “lack of time” has been deemed the most cited reason for not exercising, and adopted a HIIT protocol, as opposed to a SIT protocol, as this has been deemed as more applicable to the general population (Ziemann, et al. 2011). However, these findings demonstrate that although 15 minutes of high-intensity exercise 3 times per week can improve cardiorespiratory fitness, it is not sufficient to improve cardiac indices, central arterial stiffness and blood lipid profiles and subsequently chronic disease risk reduction within a 6 week period. This could be a result of employing this particular time-efficient volume of exercise or the intensity of the work periods. Further investigation is required to elicit the time course at which improvement in these indices become apparent when adopting a time-efficient HIIT protocol.

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Table 1: Cardiometabolic and vascular indices before, mid-point and after the 6-week intervention period for the training group and control.

	Training group (n=12)			**Control group (n=9)**		
	0 weeks	**3 weeks**	**6 weeks**	**0 weeks**	**3 weeks**	**6 weeks**
VO_{2max} ($mL \cdot kg^{-1} \cdot min^{-1}$)	42.5 ± 8.3	44.3 ± 7.3	47.4 ± 8.5 *	37.7 ± 8.3	40.5 ± 8.7	40.0 ± 8.4
VO_{2max} ($L \cdot min^{-1}$)	3.1 ± 0.6	3.2 ± 0.6	3.4 ± 0.6	2.7 ± 0.7	3.0 ± 0.6	2.8 ± 0.7
Clustered risk score	-0.07 ± 0.57	-0.04 ± 0.89	-0.02 ± 0.88	-0.01 ± 0.32	0.05 ± 0.30	-0.03 ± 0.24
SBP (mmHg)	126 ± 8	120 ± 7	116 ± 8*	116 ± 7	125 ± 10	119 ± 8
DBP (mmHg)	70 ± 5	66 ± 8	65 ± 10	64 ± 8	65 ± 11	65 ± 7
DP (mmHg)	70 ± 6	67 ± 7	66 ± 9	64 ± 7	69 ± 8	68 ± 6
MAP (mmHg)	85 ± 6	80 ± 7	78 ± 8	77 ± 8	83 ± 9	80 ± 4
SP (mmHg)	109 ± 6	104 ± 6	102 ± 8	101 ± 8	106 ± 10	102 ± 6
PP (mmHg)	39 ± 6	37 ± 4	36 ± 6*	36 ± 5	37 ± 6	34 ± 7*
AP (mmHg)	4 ± 3	3 ± 4	4 ± 3	2 ± 3	1 ± 5	1 ± 5
Alx (%)	9 ± 8	8 ± 10	13 ± 9	7 ± 9	1 ± 13	4 ± 11
PWV (ms)	6 ± 1	5 ± 1	5 ± 1	5 ± 1	5 ± 1	5 ± 1
Brachial diameter (cm)	0.41 ± 0.05	0.44 ± 0.06	0.43 ± 0.04	0.42 ± 0.03	0.44 ± 0.06	0.43 ± 0.03
Femoral diameter (cm)	0.67 ± 0.06	0.70 ± 0.06	0.70 ± 0.05	0.65 ± 0.05	0.65 ± 0.05	0.63 ± 0.05

Data presented as mean ± SD. * $P<0.05$, significantly different from baseline. VO_{2max}, maximal oxygen consumption; SBP, systolic blood pressure; DBP, diastolic blood pressure; DP, central aortic diastolic pressure; MAP, mean arterial pressure; SP, central aortic systolic pressure; PP, central aortic pulse pressure; AP, central augmented pressure; Alx, augmentation index; PWV, pulse wave velocity.

Table 2: Cardiac indices before, mid-point and after the 6-week intervention period for the training group and control.

	Training group (n=12)			**Control group (n=9)**		
	0 weeks	**3 weeks**	**6 weeks**	**0 weeks**	**3 weeks**	**6 weeks**
IVSd (mm)	9.4 ± 1.4	8.9 ± 1.4	9.3 ± 1.3	8.0 ± 0.8	8.0 ± 1.1	8.3 ± 0.8
LVPWd (mm)	9.5 ± 0.9	9.3 ± 1.5	9.7 ± 1.3	9.5 ± 0.9	9.4 ± 1.1	9.9 ± 0.8
LVEDV (ml)	122.2 ± 17.2	118.4 ± 16.9	126.7 ± 12.7	120.2 ± 16.4	131.9 ± 16.9	122.1 ± 22.9
LVESV (ml)	47.8 ± 10.7	52.1 ± 11.1	51.5 ± 6.5*	48.6 ± 6.2	58.8 ± 8.6	55.8 ± 11.2*
SV (ml)	74.6 ± 12.8	66.3 ± 7.3	75.1 ± 9.9	71.6 ± 12.4	73.0 ± 9.8	66.2 ± 12.2
EF (%)	61.0 ± 6.5	56.3 ± 4.5	59.1 ± 4.0*	59.4 ± 3.8	55.4 ± 3.1	54.3 ± 2.1*
E (cm·s)	0.81 ± 0.07	0.84 ± 0.10	0.80 ± 0.10†	0.72 ± 0.04	0.74 ± 0.08	0.70 ± 0.10
A (cm·s)	0.46 ± 0.08	0.45 ± 0.08	0.4 ± 0.10	0.41 ± 0.06	0.40 ± 0.05	0.40 ± 0.10
E:A	1.82 ± 0.39	1.95 ± 0.49	2.07 ± 0.41	1.84 ± 0.33	1.91 ± 0.14	1.77 ± 0.15
E’ (cm·s)	0.14 ± 0.02	0.14 ± 0.02	0.14 ± 0.02	0.16 ± 0.03	0.15 ± 0.03	0.14 ± 0.02
A’ (cm·s)	0.08 ± 0.02	0.08 ± 0.02	0.07 ± 0.01	0.09 ± 0.04	0.08 ± 0.03	0.07 ± 0.01
S’ (cm·s)	0.09 ± 0.02	0.10 ± 0.02	0.09 ± 0.02	0.10 ± 0.03	0.13 ± 0.07	0.08 ± 0.01

Data presented as mean ± SD. * P<0.05, significantly different from baseline. † P<0.05, significantly different between groups. IVSd, interventricular septum thickness at end -diastole; LVPWd, left ventricular posterior wall thickness at end -diastole; LVEDV, left ventricular end-diastolic volume; LVESV, left ventricular end-systolic volume; SV, stroke volume; EF, ejection fraction; E, peak velocity of early diastolic transmitral flow; A, peak velocity of late transmitral flow; E:A, ratio of E to A; E’, peak velocity of early diastolic mitral annular motion; A’, peak velocity of diastolic mitral annular motion; S’, peak velocity of systolic mitral annular motion.