ORIGINAL ARTICLE

CrossMark

Extremely short duration sprint interval training improves vascular health in older adults

Simon Adamson[1] · Mykolas Kavaliauskas[2] · Takaki Yamagishi[1] · Shaun Phillips[3] · Ross Lorimer[1] · John Babraj[1]

Abstract

Exercise improves health and physical function in older people, but very few older people participate although the trend is for increasing participation. This study sought to determine whether short duration sprint interval training (SIT) improves health and physical function in older people. Seventeen (9 M and 8 F) older adults (age 66 ± 3 years) were recruited. Participants had blood pressure, physical function and blood lipid profile measured and were then allocated to a control group (CON $n = 7$) or a SIT group ($n = 10$). The control group maintained daily activities; the SIT group performed 10 weeks of twice-weekly training sessions of 6-s sprints. By week 10, training sessions lasted 11.6 ± 0.6-min. Ten weeks of SIT resulted in significant changes in pulse pressure (CONpre 59 ± 18 mmHg; CONpost 60 ± 9 mmHg; SITpre 56 ± 14 mmHg; SITpost 49 ± 7 mmHg; $p = 0.007$), mean blood pressure (CONpre 100 ± 10 mmHg; CONpost 97 ± 11 mmHg; SITpre 102 ± 7 mmHg; SITpost 93 ± 8 mmHg; $p = 0.003$), timed get up and go (CONpre 6.9 ± 1.1 s; CONpost 6.9 ± 1.0 s; SITpre 7.4 ± 1.2 s; SITpost 6.6 ± 1.0 s; $p = 0.005$), loaded 50 m walk (CONpre 6.9 ± 1.1 s; CONpost 6.9 ± 1.0 s; SITpre 7.4 ± 1.2 s; SITpost 6.6 ± 1.0 s; $p = 0.005$), and total cholesterol: HDL cholesterol ratio (CONpre 4.2 ± 0.7; CONpost 4.0 ± 0.7; SITpre 4.4 ± 1.1; SITpost 3.2 ± 0.7; $p = 0.01$). SIT is an effective way to maintain blood pressure, lipid profile, and physical function during aging and is an effective tool for promoting optimal aging.

Keywords Aging · Sprint interval training · Blood pressure · Physical function

Introduction

Aging is associated with a reduction in cardiac and arterial function with a marked increase in arterial stiffness, resulting in isolated systolic hypertension [1]. Pulse pressure is a surrogate pulsatile component of blood pressure made up of ventricular ejection, arterial stiffness and wave reflection [2]. At rest, ventricular ejection does not change over the lifespan [3] and following 12 months of endurance exercise ventricular ejection does not change at rest [4]. Therefore, it is suggested that at rest pulse pressure represents either arterial stiffness or wave reflection in older adults [2]. Furthermore,

arterial stiffening is a predictor for a number of vascular diseases including heart failure, stroke and dementia [5]. Arterial stiffening is a predictor of vascular disease, but is also strongly associated with the loss of physical function and onset of functional limitations of old age [6].

The development of arterial stiffness has been linked to both abnormal lipid and glucose metabolism [7]. However, with age there is a decrease in glucose tolerance, resulting in increased fasting and post-prandial blood glucose levels across the lifespan [8]. In addition, circulating total cholesterol, low-density lipoprotein (LDL) cholesterol and triglycerides concentrations can be elevated with age [9] and are strongly associated with cardiovascular disease risk [10]. Higher total cholesterol: high density lipoprotein (HDL) cholesterol ratios have been reported in older adults with atherosclerotic alterations compared to those with no atherosclerotic alterations [11]. The mechanisms linking lipid profile to arterial stiffness are multifactorial, including atherosclerosis, changes in the elastic elements of the arterial wall, endothelial dysfunction and inflammation [12]. Following 12 weeks of an aerobic and resistance

✉ John Babraj
j.babraj@abertay.ac.uk

1 Division of Sport and Exercise Science, Abertay University, Dundee DD1 1HG, Scotland, UK

2 School of Applied Sciences, Sport, Exercise and Health Sciences, Edinburgh Napier University, Edinburgh, UK

3 Division of Sport and Exercise Science, Edinburgh University, Edinburgh, UK

Springer

exercise programme there is an improvement in systolic blood pressure, pulse wave velocity and a reduction in lipid profile in obese women [13]. Likewise, a 5-week aerobic exercise programme has been shown to decrease circulating triglycerides and glycosylated heamoglobin (HbA1c) in older men [14]. Despite the benefits of aerobic and resistance exercise, the majority of older adults do not take part in traditional exercise with time, dislike of these exercise modes and risk of injury being barriers to exercise participation [15].

Sprint interval training (SIT) can be a time-efficient exercise paradigm but duration of SIT protocols can vary between 10-min [16] and 30-min [17] depending on sprint duration and recovery. Longer SIT protocols have typically utilised $4-6 \times 30$-s sprints in a 1:8 work to rest interval on 3 days per week and produce similar adaptations to traditional endurance training [18]. However, these protocols last close to 30 min. To overcome this number of recent studies have looked at utilising 10-min protocols using fewer sprints but still requiring a minimum of 3 training days per week. Metcalfe et al. [19] demonstrated improved VO_2 max (13%) and insulin sensitivity (28%) in young males when carrying out 2×20-s sprints. Gillen et al. [20] demonstrate similar improvements in VO_2 max (12%) in young adults when using 3×20-s sprints at a lower resistance and an 8% reduction in mean arterial pressure after 6 weeks of training. Despite this reduction in total time, the use of 20-s sprints may not be appropriate for older adults due to the extended cardiovascular load, which is similar to 30-s sprints in young adults [21]. Further, these studies still require training to be carried out on 3 days of the week, which may limit people's ability to follow these protocols. In a middle-aged population, twice weekly SIT sessions consisting of $6-10 \times 6$-s sprints, with a maximum training duration of 10-min, has been shown to increase VO_2 peak (8%), decrease blood glucose area under the curve (6%) and improve physical function [16]. Recently, it has been demonstrated that a progressive SIT protocol performed twice weekly over 6 weeks will also improve physical function and blood glucose control in older adults [22]. However, the effectiveness of short sprints on blood pressure components has not been investigated.

Longer duration sprints have been shown to improve cardiovascular function and lower blood pressure in young adults [18]. However, shorter duration sprints have been shown to improve physical function and glucose metabolism in older adults [22]. Therefore, the aim of this study was to determine the effectiveness of a 10-week extremely short duration SIT protocol (6-s) on blood pressure and health in an older adult population. It was hypothesized that SIT group would have improvements in physical function and resting blood pressure.

Materials and methods

Participants

17 older adults (age range 60–71 years) were recruited for the study via local newspaper advertisement and were allocated using a stratified approach to ensure that baseline age was similar in the control group (CON: three males and four females; Table 1) or a twice per week SIT group (SIT: four females, six males; Table 1). More participants were recruited to the SIT group to allow for potential dropout, although dropout rate was zero. All participants were inactive and participants were excluded if they had any metabolic disease or cardiovascular disease. All participants had well-controlled hypertension (blood pressure ≤ 160/90 mmHg) and were taking oral hypertensive medication which remained unchanged for 6 months prior to and during the study. There was no significant difference in baseline characteristics of the two groups (Table 1), therefore, differences in the group sizes do not bias the result [23]. Participants allocated to the control group were asked to maintain their normal lifestyle throughout the study period but took part in no structured training. Participants allocated to the SIT group took part in a twice weekly, 10-week SIT intervention, but no other exercise training. All participants were asked to report any changes in lifestyle or medication during the study. All participants provided verbal and written informed consent. The study had ethical approval from Abertay University Ethics Committee (SHS0701615) and conducted in accordance with the declaration of Helsinki.

Baseline testing

Participant reported to the laboratory having fasted overnight and height was recorded using a SECA 217 Stadiometer (SECA United Kingdom, Birmingham, UK) and weight determined using a SECA Medical 780 weighing scales (SECA United Kingdom, Birmingham, UK).

Blood pressure

Prior to obtaining blood pressure measurements, all participants sat quietly for 5 min with both arms in a forward position, on a flat table surface. Blood pressure, pulse pressure and mean arterial pressure was measured using a WatchBP Office ABI Automatic Office Blood Pressure Measurement Device (Microlife WatchBP AG, Widnau, Switzerland). Triplicate blood pressure measurements were made, with a 1-min interval in-between, and the average value for blood pressure, pulse pressure (pulse pressure = systolic − diastolic pressure) and mean arterial pressure was recorded for both

Table 1 Participant characteristics, physical function and circulating lipids

	CONTROL		SIT	
	Pre	Post	Pre	Post
Participant characteristics				
Age (years)	66 ± 2	66 ± 2	66 ± 4	66 ± 4
Height (cm)	164 ± 10	164 ± 10	169 ± 9	169 ± 9
Weight (kg)	70 ± 13	71 ± 13	77 ± 13	75 ± 12^a
BMI (kg m^{-2})	25.9 ± 3.3	26.2 ± 3.5	26.9 ± 3.5	26.3 ± 3.5
Blood measures				
Total cholesterol (mmol l^{-1})	4.8 ± 1.4	4.8 ± 1.2	5.8 ± 1.5	4.6 ± 1.3
HDL cholesterol (mmol l^{-1})	1.1 ± 0.4	1.2 ± 0.3	1.4 ± 0.4	1.5 ± 0.4
LDL cholesterol (mmol l^{-1})	3.0 ± 0.9	2.9 ± 1.0	3.6 ± 1.1	2.6 ± 1.1
Total triglyceride (mmol l^{-1})	1.2 ± 0.6	1.3 ± 0.8	1.7 ± 1.0	1.2 ± 0.7
TC:HDL-C	4.2 ± 0.7	4.0 ± 0.7	4.4 ± 1.1	3.2 ± 0.7^b
LDL:HDL-C	2.7 ± 0.6	2.4 ± 0.7	2.7 ± 0.9	1.7 ± 0.6^a
TG:HDL-C	1.4 ± 1.0	0.9 ± 0.6	1.0 ± 0.3	1.1 ± 0.6
Physical function				
Get up and go (s)	6.9 ± 1.1	6.9 ± 1.0	7.4 ± 1.2	6.6 ± 1.0^b
Loaded 50 m walk (s)	37.3 ± 4.3	36.8 ± 4.0	38.3 ± 4.8	34.9 ± 5.1^b
Stair climb power (W)	208 ± 86	200 ± 98	253 ± 46	285 ± 59^a

[a] $p < 0.05$ SIT post versus control post

[b] $p < 0.01$ SIT post versus control post

left and right arms. It has been suggested that differences in right and left arm blood pressure provides evidence of peripheral vascular disease and is of clinical importance [24].

Lipid profile

A finger prick blood sample was taken to allow measurement of lipid profile. The initial blood droplet was discarded and the second blood droplet taken for analysis of fasting lipid levels (CardioChek PA, Polymer Technology Systems Inc, Indianapolis, USA, within run CV for total cholesterol 4.7%, HDL cholesterol 5.9% and triglyceride 4.3%). LDL cholesterol was calculated using the modified Friedewald calculation (LDL-C (mg dl^{-1}) = non-HDL-C $\times$ 90% $-$ TG $\times$ 10%; where TG = triglyceride, non-HDL-C = total-C $-$ HDL-C) [25].

Physical function [26]

Get up and go test: Participants began seated with their arms folded across their chest. On the command go they rose from the chair, without using their arms, and walked 6 m, as fast as possible without running, before sitting down on a chair. This was repeated two times with the average time taken reported.

Loaded 50 m walk test: Participants were instructed to walk 50 m, as fast as possible without running, whilst carrying 20% of their bodyweight for males and 15% of their bodyweight for females. This was repeated two times with the average time taken reported.

Step test: Participants were instructed to ascend the nine stairs (each stair had a height of 16 cm) as quickly as they could. Power was calculated as the product of (total vertical height of the stairs/time) $\times$ (body weight $\times$ 9.81).

SIT intervention

All SIT sessions were fully supervised and took place at Abertay University. A bioharness 2 (Zephyr Technology, Annapolis, USA) was attached to participants prior to the training session to allow continuous recording of heart rate (bpm) and breathing frequency. Participants then completed 6×6-s all-out effort cycle sprints, against 7% bodyweight for males and 6% bodyweight for females, with a minimum of 60-s passive recovery or until heart rate was below 120 bpm. Passive recovery was chosen to allow the heart rate to recover. The cycle sprint began once the participant reached 100 rpm. Each week one extra sprint was added until the participants were completing ten sprints. No warm-up or cool down was performed before or after the session, with no adverse effects reported by any participant. All participants completed 100% of the training sessions. In week 1 training sessions averaged 8 ± 1.5 min and in week 10 training sessions averaged 11.6 ± 0.6 min.

Post-testing

All tests were repeated at the same time of day, with the same fast period and in the same order as baseline after 10 weeks. There was 5 ± 2 days between last training session and retesting of participants. This was to ensure that a training effect has being measured rather than a response to the last training session.

Data and statistical analysis

Heart rate area under the curve for the first 6 sprints in training session 1 and training session 20 was calculated using the trapezoidal rule. Statistical analysis was carried out using SPSS version 23. All data were checked for normal distribution using a Shapiro–Wilk test and was within normal values for skewness and kurtosis. Independent samples t test was used to determine the differences in baseline characteristics between the groups. Effects of training on each variable were analysed using an ANCOVA to determine difference between groups. A one way repeated measures ANOVA was used to analyse the heart rate area under the curve and average power data from training session 1 and training session

20. Significance was accepted as $p < 0.05$. Partial eta squared (η^2) was defined as 0.02 small, 0.13 medium and 0.26 large as proposed by Bakeman [27]. A Pearson's correlation was carried out between dependent variables.

Results

Blood pressure changes

There were no significant differences in blood pressure variables between the control and SIT group at baseline or between right and left arm blood pressure measures (Fig. 1). Following 10 weeks of SIT, there was a significant reduction compared to control for both right and left arm systolic blood pressure (right arm: $p < 0.01$, $\eta^2 = 0.54$; left arm: $p < 0.01$, $\eta^2 = 0.61$; Fig. 1); right and left arm diastolic blood pressure (right arm: $p = 0.05$, $\eta^2 = 0.24$; left arm: $p = 0.02$, $\eta^2 = 0.33$; Fig. 1); right and left arm pulse pressure (right arm: $p < 0.01$, $\eta^2 = 0.55$; left arm: $p < 0.01$, $\eta^2 = 0.53$; Fig. 1); right and left arm mean arterial pressure (right arm: $p = 0.01$, $\eta^2 = 0.37$; left arm: $p < 0.01$, $\eta^2 = 0.49$; Fig. 1). Sex responses are shown in Table 2.
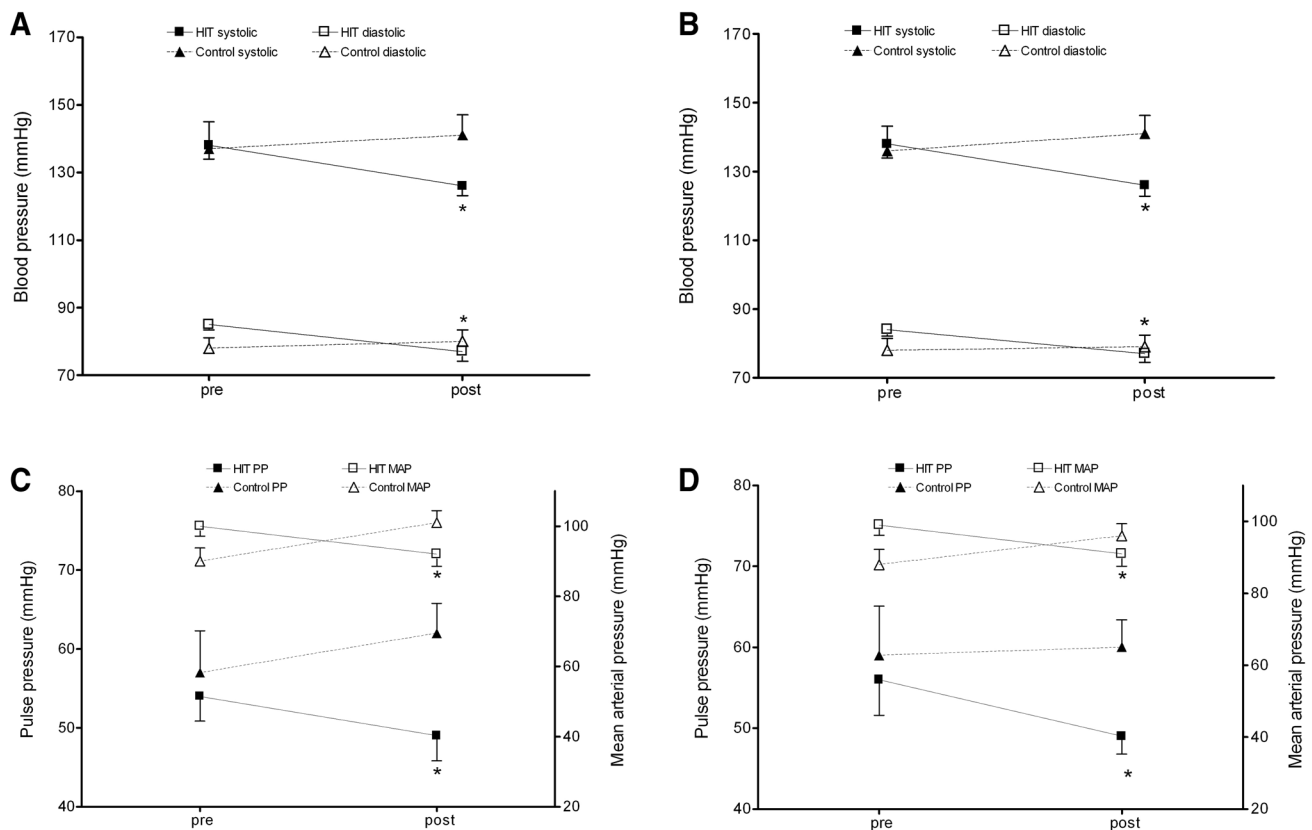

Fig. 1 Changes in vascular measures; **a** right arm systolic and diastolic blood pressure; **b** left arm systolic and diastolic blood pressure; **c** right arm pulse pressure and mean arterial pressure; **d** left arm pulse pressure and mean arterial pressure. *$p < 0.05$ SIT post versus control post

Table 2 Male and Female data for blood pressure components and physical function

	SIT pre		SIT post		CONTROL pre		CONTROL post	
	M ($n=6$)	F ($n=4$)	M ($n=6$)	F ($n=4$)	M ($n=3$)	F ($n=4$)	M ($n=3$)	F ($n=4$)
BP (mmHg)								
R Sys	136 ± 13	141 ± 13	122 ± 9	131 ± 6	137 ± 4	138 ± 13	149 ± 9	135 ± 6
R Dia	85 ± 5	85 ± 5	77 ± 9	84 ± 4	82 ± 9	76 ± 8	87 ± 5	74 ± 6
L Sys	138 ± 14	137 ± 15	122 ± 11	131 ± 7	137 ± 6	135 ± 15	146 ± 4	137 ± 7
R Sys	84 ± 5	82 ± 7	77 ± 7	78 ± 11	81 ± 9	75 ± 9	87 ± 7	74 ± 7
PP (mmHg)								
Right	53 ± 8	56 ± 13	45 ± 6	54 ± 14	54 ± 4	60 ± 15	62 ± 3	62 ± 13
Left	57 ± 14	55 ± 14	48 ± 5	52 ± 10	55 ± 5	61 ± 15	59 ± 6	61 ± 11
MAP (mmHg)								
Right	104 ± 9	94 ± 8	94 ± 10	88 ± 11	96 ± 4	86 ± 12	106 ± 5	96 ± 9
Left	104 ± 8	93 ± 7	94 ± 8	87 ± 12	94 ± 7	83 ± 11	104 ± 7	90 ± 8
GUAG (s)	8.0 ± 1.2	6.5 ± 0.6	7.0 ± 1.1	5.9 ± 0.4	6.4 ± 1.2	7.2 ± 1.2	6.7 ± 1.6	7.0 ± 0.6
L50m (s)	39 ± 5	36 ± 4	36 ± 6	33 ± 3	35 ± 4	39 ± 4	34 ± 4	39 ± 3
Power (W)	269 ± 54	229 ± 13	317 ± 56	236 ± 10	272 ± 99	159 ± 19	264 ± 99	152 ± 21

R right arm, *L* left arm, *BP* blood pressure, *Sys* systolic, *Dia* diastolic, *PP* pulse pressure, *MAP* mean arterial pressure, *GUAG* get up and go, *L50m* loaded 50 m walk

Heart rate area under the curve

There was no significant sprint × time interaction for average power ($p = 0.358$) between session 1 and session 20 (Fig. 2). There was a significant sprint × time interaction ($p = 0.048$, $\eta^2 = 0.17$) for heart rate area under the curve (Fig. 2). Following 10 weeks of SIT, heart rate AUC averaged across the first six sprints of a SIT session was significantly reduced (session 1: 6994 ± 372 beats min^{-1} s^{-1}; session 20: 6426 ± 153 beats min^{-1} s^{-1}; $p < 0.01$). There were no significant differences between the sessions for sprints 1 and 2 ($p > 0.05$), however, there was a significant difference between sessions for sprints 3, 4, 5 and 6 ($p < 0.05$, Fig. 2).

Physical function

There were no significant differences in physical function between the control and SIT group at baseline (Table 1). Following 10 weeks of SIT, there was a significant improvement in physical function compared to the control group (get up and go: $p = 0.01$, $\eta^2 = 0.44$; loaded 50 m walk: $p < 0.01$, $\eta^2 = 0.50$; stair climb power: $p = 0.04$, $\eta^2 = 0.26$; Table 1). There is a significant correlation between the change in pulse pressure and change in get up and go ($R = 0.55$; $p = 0.023$) and loaded 50 m walk ($R = 0.50$; $p = 0.041$) but not with change in mean arterial pressure ($p > 0.05$) or change in systolic blood pressure ($p > 0.05$). Sex responses are shown in Table 2.

Blood measures

There were no significant differences in blood measures between the control and SIT group at baseline (Table 1). Following 10 weeks of SIT, Total Cholesterol/HDL cholesterol ($p = 0.01$, $\eta^2 = 0.42$) and LDL cholesterol/HDL cholesterol ($p = 0.02$, $\eta^2 = 0.34$) were significantly reduced compared to the control group (Table 1). There was a trend for reduction in total cholesterol ($p = 0.07$, $\eta^2 = 0.23$), triglyceride ($p = 0.06$, $\eta^2 = 0.24$) and triglyceride/HDL cholesterol ($p = 0.06$, $\eta^2 = 0.24$; Table 1).

Discussion

The major finding from this study is that training twice weekly, with each session lasting no longer than 12-min, is sufficient to improve resting blood pressure and physical function in older adults. This demonstrates that reducing the training frequency does not impede adaptation to sprint interval training in older adults. It could be possible that a further reduction in training frequency could still promote health benefits in an older population. This could have major implications as to how we encourage older adults to exercise. It would appear that fewer training sessions are required than is currently recommended for health benefits [20]. In the current study, there was a significant reduction in left and right arm systolic and diastolic blood pressure, pulse pressure and mean arterial
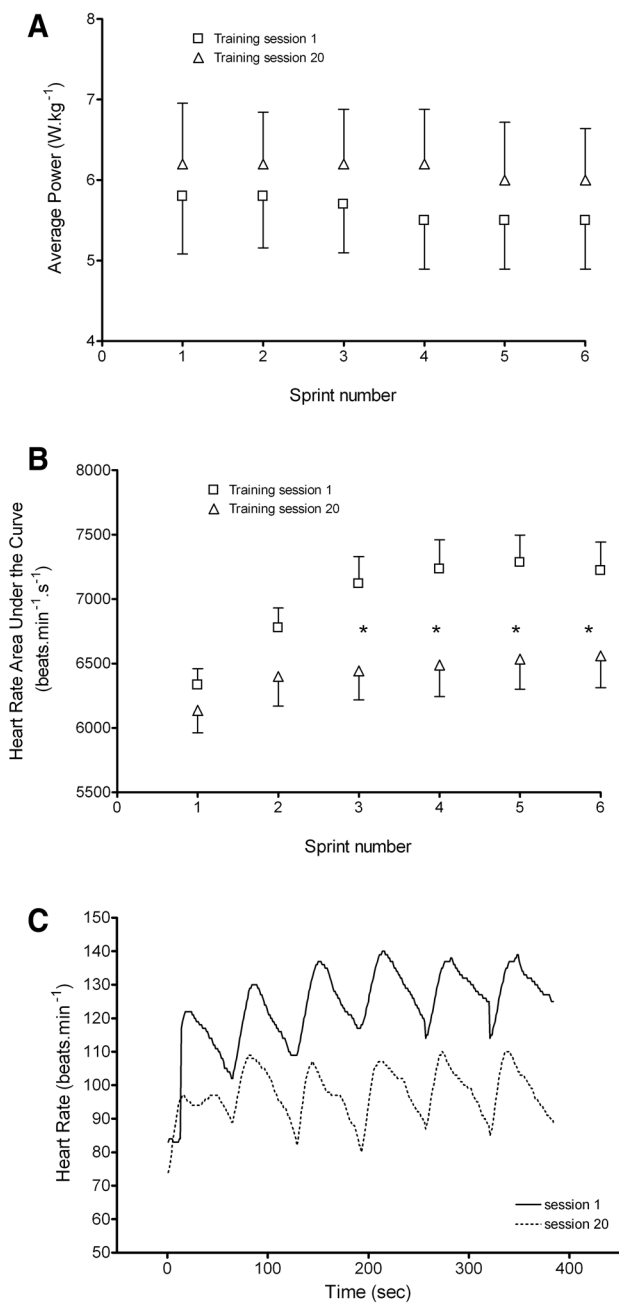
Fig. 2 Changes in heart rate during training; **a** average power across the first six sprints; **b** heart rate area under the curve for each sprint and recovery period; **c** example trace of heart rate across 6 sprints. *$p < 0.05$ session 1 compared to session 20

pressure in older adults after 10 weeks of training. We also demonstrate an improved heart rate recovery in older adults when performing repeated sprint exercise. Further, we also demonstrate that 10-week sprint interval training lowers circulating lipid profile in older adults. This suggests that SIT could be a useful exercise paradigm to reduce cardiovascular disease risk in older adults.

Blood pressure changes

Systolic blood pressure (7%), diastolic blood pressure (9%), pulse pressure (9%) and mean arterial pressure (8%) were all significantly reduced with a medium to large effect size ($\eta^2 \geq 0.24$ for all measures; Fig. 1) following twice weekly SIT. In normotensive young men and women longer duration intervals performed on 3 days per week have been shown to reduce systolic and diastolic blood pressure by 2–7% and mean arterial pressure by 6% [20, 28]. Given our findings are of a similar magnitude, even though total weekly duration is much lower, then this suggests that the duration of the sprint is not a driving factor in blood pressure adaptation to SIT. Ejection fraction does not appear to change with either endurance training [4] or with 30-s repeated high intensity bursts [29] and resting heart rate was unchanged in this study (data not reported). It could be speculated that improvements in pulse pressure at rest, reported in this study, reflect changes in arterial stiffness or wave reflection. The reduction in MAP (8%) is similar to that seen in obese adults (6%) after 2-week of 4–6 30-s sprints [30], but greater than that reported in young people following 1–6 30-s sprints [31]. Obesity like ageing is associated with an increase in total peripheral resistance that may reflect endothelial dysfunction [32]. Therefore, the reduction in mean arterial pressure may reflect improved endothelial function seen in other SIT protocols [31]. Given that hypertension is the most common risk factor for cardiovascular morbidity and mortality across the lifespan [33], then twice-weekly 6-s SIT is an effective intervention to lower hypertension risk in older adults.

Heart rate area under the curve

Following 10 weeks of SIT, heart rate AUC during sprints and recovery was significantly reduced by 8% (Fig. 2). This reduction in heart rate AUC occurs despite the average power produced across the six sprints being similar (Fig. 2). This reduction in heart rate AUC following SIT could be related to altered autonomic function. Short duration sprints (8 s sprint with 12 s recovery over 20 min) have been shown to alter autonomic control of the heart via greater vagal influence on heart rate at rest in young overweight women [34]. When looking at the individual heart rate trace there is a reduction in peak heart rate following each sprint, with a more rapid recovery after exercise (Fig. 2). This suggests that there is either lower sympathetic activity post training or withdrawal of vagal tone [35] during each sprint resulting in a lower heart rate response. Given that HR recovery is associated with several pathophysiological abnormalities and is considered as an independent risk factor for the development of cardiovascular disease [33] these findings may have important clinical implications for older adults.

Lipid profile

The ratio of total cholesterol/HDL cholesterol (26%) and LDL cholesterol/HDL cholesterol (33%) were significantly reduced compared to control (Table 2) following SIT, with a strong trend for reduction of circulating total cholesterol (19%) and triglyceride levels (23%; Table 2). The magnitude of change is similar to that reported in older adults following a 3 times per week endurance programme lasting 50 min per session [36] and a treadmill-based sprint protocol in inactive females, consisting of 10×15 s sprints with 30 s rest on 3 days per week [37]. However, SIT was only performed on 2 days per week compared to the 3 days per week in endurance and run-based high intensity with a much lower total sprint time. This may reflect increased clearance of lipids from the blood or reduced secretion of lipids from the liver. Fat oxidation has been shown to be increased following longer duration SIT protocols [38] and VLDL secretion has been shown to be reduced following long duration (4 min intervals) high intensity training [39]. However, this remains to be determined with this short sprint protocol.

Physical function

Following 10 weeks of twice per week SIT, there was a significant improvement in get up and go (11%), loaded 50 m walk (9%) and stair climb power ($d_{pcc2} \geq -0.8$ for all measures Table 2). In older adults, a 10-week endurance training programme, carried out 3 days per week for 50 min, resulted in a 15% reduction in 1 mile walk [36]. Likewise, a 14-week progressive resistance training programme improved timed get up and go by 15% [40]. The improvements in physical function are similar to those reported after 6 weeks of 6 s SIT, 7% improvement in loaded 50 m walk, 7% improvement in get up and go [22], suggesting that improvements in physical function occur early during SIT but continue to improve as training progresses. There is a significant correlation between the change in resting pulse pressure and the change in loaded 50 m walk ($R = 0.5$; $p = 0.04$) and get up and go ($R = 0.55$; $p = 0.02$). This is similar to the correlation reported for improvements in arterial stiffness and physical function with endurance exercise [6]. Given at rest in the elderly, pulse pressure reflects either arterial stiffness or wave amplification then this suggests that changes within the blood vessel structure are important for physical function adaptations following SIT. Further these adaptations are seen within only twice weekly training sessions lasting approximately 10 min.

Conclusion

In this study, we show for the first time the adaptation in blood pressure and physical function in older adults to a twice-weekly extremely short duration SIT. These results should be considered in context of the limitations of the study and future studies should look to record all medication being taken by participants and seek to record daily activity and nutritional intake across the intervention period. However, there are significant improvements in vascular and physical function and these improvements in physical function are strongly correlated to the improvements in pulse pressure ($R = 0.55$), which may reflect greater arterial compliance following SIT. Given that one of time and dislike of traditional exercise are barriers to participation [20] then there is a need to reappraise current exercise advice for older adults to do 150 min of moderate or 75 min of vigorous intensity exercise a week [41]. The current study has a much lower time commitment but more research is required to determine minimum effective training load of SIT to promote optimal aging in older adults.

Compliance with ethical standards

Conflict of interest The authors declare no conflict of interest.

Ethical approval The study was granted ethical approval by the institutional ethics committee (SHS0701615) and was carried out in line with the Declaration of Helsinki (World Medical Association 2013).

Informed consent All participants were given verbal and written information about the study prior to providing informed consent.

References

1. Scuteri A, Najjar SS, Muller DC, Andres R, Hougaku H, Mette EJ et al (2004) Metabolic syndrome amplifies the age-associated increases in vascular thickness and stiffness. J Am Coll Cardiol 43:1388–1395
2. Asmar R, Rudnichi C, Blacher J, London GM, Safar ME (2001) Pulse pressureand aortic wave are markers of cardiovascular risk in hypertensive populations. AJH 14:91–97
3. Port S, Cobb FR, Coleman RE, Jones RH (1980) Effect of age on the response of the left ventricular ejection fraction to exercise. N Engl J Med 303:1133–1137
4. Stratton JR, Levy WC, Schwartz RS, Abrass IB, Cerqueira MD (1994) Beta-adrenergic effects on left ventricular

filling: influence of aging and exercise training. J Appl Physiol 77:2522–2529

5. Vaccarino V, Berger A, Abramson J, Black H, Setaro J, Davey J et al (2001) Pulse pressure and risk of cardiovascular events in the systolic hypertension in the elderly program. Am J Cardiol 88:980–986

6. Brunner EJ, Shipley MJ, Witte DR, Singh-Manoux A, Britton AR, Tabak AG et al (2011) Arterial stiffness, physical function, and functional limitation: the Whitehall II study. Hypertension 57:1003–1009

7. Ferrier KE, Muhlmann MH, Baguet JP, Cameron JD, Jennings GL, Dart AM et al (2002) Intensive cholesterol reduction lowers blood pressure and large artery stiffness in isolated systolic hypertension. J Am Coll Cardiol 39:1020–1025

8. DeFronzo RA (1981) Glucose intolerance and aging. Diabetes Care 4:493–501

9. Chubert CM, Rogers NL, Remsberg KE, Sun SS, Chumlea WC, Demerath EW et al (2006) Lipids, lipoproteins, lifestyle, adiposity and fat-free mass during middle age: the Fels longitudinal study. Int J Obes 30:251–260

10. Nomikos T, Panagiotakos D, Georgousopoulou E, Metaxa V, Chrysohoou C, Skoumas I et al (2015) Hierarchical modelling of blood lipids' profile and 10-year (2002–2012) all cause mortality and incidence of cardiovascular disease: the ATTICA study. Lipids Health Dis 14:108

11. Blacher J, Asmar R, Djane S, London GM, Safar ME (1999) Aortic pulse wave velocity as a marker of cardiovascular risk in hypertensive patients. Hypertension 33:1111–1117

12. Wilkinson I, Cockcroft JR (2007) Cholesterol, Lipids and arterial stiffness. Safar ME, Frohlich ED (eds) Atherosclerosis, large arteries and cardiovascular risk. Advances in cardiology, vol 44. Karger, Basel, pp 261–277

13. Yang SJ, Hong HC, Choi HY, Yoo HJ, Hwang TG, Baik SH, Choi DS, Kim SM, Choi KM (2011) Effects of a three-month combined exercise programme on fibroblast growth factor 21 and fetuin-A levels and arterial stiffness in obese women. Clin Endocrinol 75:464–469

14. Taniguchi H, Tanisawa K, Sun X, Kubo K, Higuchi M (2016) Endurance exercise reduces hepatic fat content and serum fibroblast growth factor 21 levels in elderly men. J Clin Endocrinol Metab 101:191–198

15. McPhee JS, French DP, Jackson D, Nazroo J, Pendleton N, Degens H (2016) Physical activity in older age: perspectives for healthy ageing and frailty. Biogerontology 17:567–580

16. Adamson S, Lorimer R, Cobley JN, Lloyd R, Babraj J (2014) High intensity training improves health and physical function in middle aged adults. Biology (Basel) 3:333–344

17. Burgomaster KA, Cermak NM, Phillips SM, Benton CR, Bonen A, Gibala MJ (2007) Divergent response of metabolite transport proteins in human skeletal muscle after sprint interval training and detraining. Am J Physiol Regul Integr Comp Physiol 292:1970–1976

18. Burgomaster KA, Hughes SC, Heigenhauser GJ, Bradwell SN, Gibala MJ (2005) Six sessions of sprint interval training increases muscle oxidative potential and cycle endurance capacity in humans. J Appl Physiol 98:1985–1990

19. Metcalfe RS, Babraj JA, Fawkner SG et al (2012) Towards the minimal amount of exercise for improving metabolic health: beneficial effects of reduced-exertion high-intensity interval training. Eur J Appl Physiol 112:2767–2775

20. Gillen JB, Percival ME, Skelly LE et al (2014) Three minutes of all-out intermittent exercise per week increases skeletal muscle oxidative capacity and improves cardiometabolic health. PLoS One 9(11):e111489

21. Yamagishi T, Babraj J (2017) Effects of reduced-volume of sprint interval training and the time course of physiological and performance adaptations. Scand J Med Sci Sports 7:1662–1672

22. Adamson SB, Lorimer R, Cobley JN, Babraj J (2014) Extremely short-duration high-intensity training substantially improves the physical function and self-reported health status of elderly adults. J Am Geriatr Soc 62:1380–1381

23. Dumville JC, Hahn S, Miles JN, Torgerson DJ (2006) The use of unequal randomisation ratios in clinical trials: a review. Contemp Clin Trials 27:1–12

24. Clark CE, Taylor RS, Shore AC, Ukoumunne OC, Campbell JL (2012) Association of a difference in systolic blood pressure between arms with vascular disease and mortality: a systematic review and meta-analysis. Lancet 379:905–914

25. Chen Y, Zhang X, Pan B, Jin X, Yao H, Chen B et al (2010) A modified formula for calculating low-density lipoprotein cholesterol values. Lipids Health Dis 9:52

26. Bennell K, Dobson F, Hinman R (2011) Measures of physical performance assessments. Arthritis Care Res 63:S350–S370

27. Bakeman R (2005) Recommended effect size statistics for repeated measures designs. Behav Res Methods 37:379–384

28. Ciolac EG, Bocchi EA, Bortolotto LA, Carvalho VO, Greve JM, Guimarães GV (2010) Effects of high-intensity aerobic interval training vs. moderate exercise on hemodynamic, metabolic and neuro-humoral abnormalities of young normotensive women at high familial risk for hypertension. Hypertens Res 33:836–843

29. Eskelinen JJ, Heinonen I, Löyttyniemi E, Hakala J, Heiskanen MA, Motiani KK, Virtanen K, Pärkkä JP, Knuuti J, Hannukainen JC, Kalliokoski KK (2016) Left ventricular vascular and metabolic adaptations to high-intensity interval and moderate intensity continuous training: a randomized trial in healthy middle-aged men. J Physiol 59:7127–7140

30. Whyte LJ, Gill JM, Cathcart AJ (2010) Effect of 2 weeks of sprint interval training on health-related outcomes in sedentary overweight/obese men. Metabolism 59:1421–1428

31. Rakobowchuk M, Tanguay S, Burgomaster KA, Howarth KR, Gibala MJ, MacDonald MJ (2008) Sprint interval and traditional endurance training induce similar improvements in peripheral arterial stiffness and flow-mediated dilation in healthy humans. Am J Physiol Regul Integr Comp Physiol 295:236–242

32. Wildman RP, Mackey RH, Bostom A, Thompson T, Sutton-Tyrrell K (2003) Measures of obesity are associated with vascular stiffness in young and older adults. Hypertension 42:468–473

33. Ciolac EG (2012) High-intensity interval training and hypertension: maximizing the benefits of exercise? Am J Cardiovasc Dis 2:102–110

34. Boutcher SH, Park Y, Dunn SL, Boutcher YN (2013) The relationship between cardiac autonomic function and maximal oxygen uptake response to high-intensity intermittent-exercise training. J Sports Sci 31:1024–1029

35. White DW, Raven PB (2014) Autonomic neural control of heart rate during dynamic exercise: revisited. J Physiol 592:2491–2500

36. Fahlman MM, Boardley D, Lambert CP, Flynn MG (2002) Effects of endurance training and resistance training on plasma lipoprotein profiles in elderly women. J Gerontol A Biol Sci Med Sci 57:54–60

37. Zaer Ghodsi N, Zolfaghari MR, Fattah A (2016) The impact of high intensity interval training on lipid profile, inflammatory markers and anthropometric parameters in inactive women. Med Lab J 10:56–60

38. Boutcher SH (2011) High-intensity intermittent exercise and fat loss. J Obes 2011:868305

39. Tsekouras YE, Magkos F, Kellas Y, Basioukas KN, Kavouras SA, Sidossis LS (2008) High-intensity interval aerobic training reduces hepatic very low-density lipoprotein-triglyceride secretion rate in men. Am J Physiol Endocrinol Metab 295:851–858

40. Sousa N, Sampaio J (2005) Effects of progressive strength training on the performance of the Functional Reach Test and the Timed Get-Up-and-Go Test in an elderly population from the rural north of Portugal. Am J Hum Biol 17:746–751

41. Elsawy B, Higgins KE (2010) Physical activity guidelines for older adults. Am Fam Physician 81:55–59