European Journal of Applied Physiology (2019) 119:981–989
https://doi.org/10.1007/s00421-019-04087-9

ORIGINAL ARTICLE

Cardiovascular response of postmenopausal women to 8 weeks of sprint interval training

Daniel Zhang[1] · Tornike Janjgava[1] · Stephen H. Boutcher[1] · Yati N. Boutcher[1]

Received: 30 August 2018 / Accepted: 29 January 2019 / Published online: 9 February 2019

Abstract

Introduction Menopause is accompanied by decreased aerobic fitness and increased risk of cardiovascular disease. Sprint interval training (SIT) is a time-efficient intervention for improving cardiovascular function and aerobic fitness of young adults.

Aim To determine the effect of an 8-week SIT program on the cardiovascular function and aerobic fitness of overweight postmenopausal women.

Method Thirty overweight postmenopausal women were randomized into exercise ($n=15$) or control ($n=15$) groups. The intervention group completed three SIT sessions a week for 8 weeks. Each session consisted of 20 min of 8-s sprints and 12 s of light pedalling. Participants also completed 8 min of light aerobic cycle exercise, before and after the SIT intervention. Cardiovascular function including heart rate, stroke volume (SV), and diastolic filling time (DFT) was assessed before and after the intervention and during 8 min of light aerobic exercise. Estimated maximal oxygen uptake ($\dot{V}O_{2max}$) was also assessed.

Results Resting SV was increased ($p=0.001$) from pre- (77.5 ± 17.0 mL) to post-SIT (81.3 ± 17.0 mL), whereas SV during 8 min of light aerobic exercise was increased ($p=0.000$), from pre- (97.8 ± 1.6 mL) to post-test (103.5 ± 17.8 mL). Resting DFT was increased, ($p=0.010$), at pre- (333.4 ± 94.4 mL) to post-SIT (357.4 ± 88.2 mL), whereas DFT during 8 min of aerobic exercise was increased, ($p=0.000$), from pre- (480.1 ± 99.5 mL) to posttest (527.2 ± 123.0 mL). Predicted $\dot{V}O_{2max}$ was increased, ($p=0.016$), from pre- (19.5 ± 5.87 mL kg^{-1} min^{-1}) to post-SIT (21.4 ± 7.02 mL kg^{-1} min^{-1}).

Conclusion SIT improved cardiovascular function and aerobic fitness of overweight postmenopausal women after 8 weeks of exercise.

Keywords Sprint interval training · Postmenopausal women · Stroke volume · Diastolic filling time · Aerobic fitness

Abbreviations

ANCOVA	Analysis of co-variance
BMI	Body mass index
BPM	Beats per minute
BSA	Body surface area
BP	Blood pressure
CI	Cardiac index
CO	Cardiac output
COP	Cardiac output program
CRF	Cardiorespiratory fitness
DFT	Diastolic filling time
ECG	Electrocardiography
HR	Heart rate
SIT	Sprint interval training
LV	Left ventricular
LVET	Left ventricular ejection time
PEP	Pre-ejection period
RPE	Rating of perceived exertion
RPM	Revolutions per minute
SI	Stroke index
SSE	Steady-state exercise
SV	Stroke volume
TPR	Total peripheral resistance
VER	Ventricular emptying rate
$\dot{V}O_2$	Oxygen uptake
$\dot{V}CO_2$	Carbon dioxide production
$\dot{V}O_{2max}$	Maximal oxygen uptake
VFT	Ventricular filling time
W	Watts

Communicated by Keith Phillip George.

✉ Yati N. Boutcher
y.boutcher@unsw.edu.au

[1] School of Medical Sciences, Faculty of Medicine, University of New South Wales, Sydney 2052, Australia

Introduction

Maximal aerobic power ($\dot{V}O_{2max}$) and cardiac function deteriorate with age and in postmenopausal women these changes occur more rapidly compared to similar aged males (Zhao et al. 2014). Postmenopausal women's more pronounced cardiac deterioration likely explains why they are at greater risk of developing cardiovascular disease when compared to males (Perez-Lopez et al. 2009). Therefore, preventing the deterioration of $\dot{V}O_{2max}$ and cardiac function in postmenopausal women is important so that increased risk of cardiovascular and other chronic diseases can be reduced (Lavie et al. 2015).

Regular aerobic exercise significantly increases $\dot{V}O_{2max}$ and cardiac function of young males and females (Weiner and Baggish 2012). Both central and peripheral cardiovascular adaptations are thought to contribute to increases in $\dot{V}O_{2max}$ brought about by aerobic exercise training. Central adaptations include exercise-induced blood volume expansion, an increase in stroke volume (SV) and diastolic filling time (DFT), as well as a decrease in resting and exercise heart rate (HR) (Weiner and Baggish 2012). An exercise-induced increase in blood volume has been shown to enhance left ventricular filling resulting in increased resting and exercise SV (Convertino 2007). In the periphery, vascular and metabolic adaptations also occur that include reduced total peripheral resistance and an increase in the ability of muscles to utilize more oxygen ($a-\bar{v}O_2$ difference) caused by enhanced capillarization and metabolic activity of muscle tissue (Warburton et al. 2004).

Fast bursts of exercise interspersed with recovery periods, typically referred to as sprint interval training (SIT), have resulted in decreased fat loss and increased muscle mass in young overweight women and men (Dunn et al. 2014; Gahreman et al. 2016; Heydari et al. 2012; Trapp et al. 2008) and postmenopausal women (Maillard et al. 2016). In addition, SIT has increased $\dot{V}O_{2max}$ of premenopausal females (Dunn et al. 2014; Trilk et al. 2011) and young males (Gahreman et al. 2016; Heydari et al. 2012) in significantly less time and volume compared to aerobic exercise (Arena et al. 2013; Boutcher 2011). Compared to aerobic exercise, the mechanisms underlying SIT-induced increases in $\dot{V}O_{2max}$ have not been well identified. Trilk et al. (2011), however, showed that 4 weeks of SIT consisting of 4–7, 30-s flat out sprints by premenopausal women resulted in an 11% increase in SV which was accompanied by a 12% increase in $\dot{V}O_{2max}$. In addition, Heydari et al. (2013) found that 12 weeks of SIT, which involved continuous 8-s sprints and 12-s recovery periods for 20 min, three times per week, significantly increased SV at rest and during light aerobic exercise. Therefore, in young females and males, increases in SV brought about by both aerobic and SIT training appear to make a major contribution to increases in $\dot{V}O_{2max}$.

Information, however, about SIT effects on $\dot{V}O_{2max}$ increase and cardiac adaptations in overweight postmenopausal women is scarce. Regular aerobic exercise training by older men and women in their sixties and seventies resulted in increased $\dot{V}O_{2max}$ (Haykowsky et al. 2005). Spina et al. (1993), however, found gender-related differences in physiological adaptations to moderate aerobic exercise training. Unlike older men, postmenopausal women increased their $\dot{V}O_{2max}$ via peripheral adaptations only, showing no significant increase in plasma volume or SV after participating in a 9–12-month light aerobic training program exercising four times per week (Spina et al. 1993). Although, moderately light steady-state aerobic exercise does not appear to enhance the SV of postmenopausal women, the effect of a more intense form of exercise such as SIT on SV and plasma volume of menopausal women is yet to be determined.

Therefore, the aim of this study was to investigate the effect of SIT on predicted $\dot{V}O_{2max}$ and to examine the contribution of central cardiovascular adaptations underlying an increase in aerobic power. It was hypothesised that 8 weeks of SIT would significantly increase $\dot{V}O_{2max}$ of overweight postmenopausal women with the major contributor being an enhancement of SV caused by plasma volume expansion.

Methodology

Participants

Thirty sedentary, overweight postmenopausal women (determined by self-reported cessation of menstruation) aged between 47 and 59 years with a body mass index (BMI) of between 25 and 35 kg m^2 ($\geq$ 23 kg m^2 for women of Asian descent) were recruited, consented, and randomized into exercise ($n=15$) or control ($n=15$) groups. Average time since onset of menopause was 4.2 ($\pm$0.3) years. Exclusion criteria included: taking medication that could interact negatively with exercise, non-natural menopause (surgically induced), taking hormone replacement therapy, and smoking. Prior to acceptance into the study, a health history questionnaire was used to assess participants' medical history and a clearance letter from their general practitioner was required to confirm study eligibility. A 7-day physical activity recall was used to confirm a sedentary lifestyle and to assess level of current physical activity of all participants (Sallis et al. 1993). The pre-admission interview included information about all procedures and requirements for the participant and informed consent was given and documented. This study was approved by a University Human Experimentations Ethics committee. All procedures conformed to the standards of the Declaration of Helsinki.

Intervention

The exercise group ($n = 15$) took part in a sprint interval training (SIT) program that consisted of three sessions a week for a total of 8 weeks. Each session was conducted on a Monark Ergomedic 828E exercise bike and consisted of a 5-min warm-up of light pedalling at ~50 revolutions per minute (RPM), followed by 20 min of alternating 8-s sprints at near-maximal exertion (100–120 RPM) and 12-s rest periods of light pedalling, and ending with a 5-min cool-down of light pedalling. The load was initially set at 80–85% of each participant's peak HR with a pedal cadence of between 100 and 125 RPM. Recovery was set at the same amount of resistance but at a pedal cadence of 50 RPM. Participants were asked to keep their exercise intensity at a level which ensured their average exercise HR fell below their individual peak HR. SIT was supervised to ensure compliance and HR was measured using a Polar Electro HR monitor, whereas participant's rating of perceived exertion (RPE) was assessed using Borg's scale (Borg 1982) recorded at 2 min intervals.

Measures

Anthropometric measurements and metabolic profiles

Height, weight, and BMI were initially collected at pre-admission interview to confirm eligibility within the recruitment criteria and were re-measured prior to intervention. Participants were also instructed to fast for 10 h prior to the first pre-intervention testing after which venous blood was taken from the antecubital vein after 5 min of rest in an upright sitting position and was then analysed for haemoglobin and haematocrit. Hematocrit was determined by filling and centrifuging a capillary tube and then reading hematocrit level using a Micro-Haematocrit Reader (MSI, Great Britain). Haemoglobin was assessed by placing a drop of blood on a Microcuvette and analyzing it using a Hemocue analyzer (Hemocue, Medipac, Australia). Haemoglobin and haematocrit were used to calculate blood and plasma volume changes (Dill and Costill 1974).

Cardiovascular function

Cardiovascular function was assessed using impedance cardiography (Minnesota Model 304B; Surcom, Minneapolis) and a 3-lead electrocardiogram (ECG). Impedance cardiography is a non-invasive method used to measure SV, cardiac output (CO), pre-ejection period (PEP), and left ventricular ejection time (LVET). Resting impedance-derived CO corresponds closely with CO measured by the thermodilution technique (Goldstein et al. 1986), electromagnetic flow probe (Ehlert and Schmidt 1982), dye dilution (Milsom et al. 1983), and isotope dilution and radionuclide angiocardiography (Williams and Caird 1985). The correlation between impedance cardiography and other methods has generally been greater than 0.80, indicating that impedance cardiography is a valid measure of CO. Impedance measures are highly reliable with the coefficient of variation being ±5% in this and other laboratories (Godshall et al. 1996). Two impedance electrode tapes were attached to the participants' neck and the other two were attached at the level of the xiphoid process of the sternum. A small AC current was applied to the outside loops, while the central loops recorded the impedance to this current. As the heart went through systole and diastole, there were changes in blood volume and velocity in the large vessels in the thorax which alters the impedance. These changes were recorded and analysed through the Cardiac Output Program (COP, Microtronics Inc, Chapel Hill, NC, USA) (Sherwood et al. 1990). SV and CO were determined by the Kubicek equation (Kubicek et al. 1966). Body surface area (BSA) was calculated using the Du Bois formula (Dubois and Dubois 1916): $\mathrm{BSA} = 0.007184 \times \mathrm{height}^{0.725}\ (\mathrm{cm}) \times \mathrm{weight}^{0.425}\ (\mathrm{kg})$. BSA was used to derive stroke index (SI) and cardiac index (CI) by dividing SV and CO by BSA. PEP was calculated as the time between the *Q* and *B* points on the d*Z*/d*t* waveform, while LVET was the time between the *B* and *X* points. DFT was then calculated by subtracting PEP and LVET from the inter-beat interval (Sherwood et al. 1990). Ventricular filling rate (VFR) was calculated by dividing SV with DFT and ventricular emptying rate (VER) was determined by dividing SV by LVET (Boutcher et al. 2003).

Measurements during rest and during 8 min of light aerobic exercise

Measurements during rest and light aerobic exercise for 8 min were performed following blood collection after a 30-min rest interval to avoid heightened cardiovascular response due to venepuncture procedure (Dimsdale and Ziegler 1991). Measurements included SV, CO, PEP, LVET, DFT, VFR, VER, total peripheral resistance, blood pressure (BP), and HR. All participants were required to rest in a seated upright position for at least 10 min, after which, the resting measures were collected for 5 min. Following the resting measurements, while sitting upright, participants were required to perform 8 min of light aerobic exercise on a stationary bike, with a pedal cadence of 50 RPM and a 0.5 kg load, which was equal to 25 watts (W). Participants avoided movement of the torso during exercise by resting their arms on foam blocks positioned next to the stationary cycle. This protocol was repeated at the postintervention. Cardiovascular measures (averaged across exercise) were collected during the 8 min of light aerobic exercise. During the 8 min of light aerobic exercise, blood pressure (BP)

was also recorded every minute using an automatic arm-cuff blood pressure monitor (OMRON, Bannockburn, IL, USA).

Aerobic fitness

A submaximal exercise test on an electronic cycle ergometer (Monark 319E, Stockholm, Sweden) was used to predict $\dot{V}O_{2max}$ as an indicator of aerobic fitness. After a 5-min warm-up at 15 W, the load was slowly increased by 5 W every minute, while the participant was instructed to maintain 60 RPM. The test was continued until the participant reached 70% of their age-estimated maximum HR, calculated through the following equation (Robergs and Landwehr 2002): maximal heart rate = 205.8–0.685 × age. Throughout the test, participant's respiration gases were collected via a metabolic cart (TrueOne Model 2400, ParvoMedics Inc., Utah, UT, USA) and were analysed to determine oxygen consumption ($\dot{V}O_2$) and carbon dioxide production ($\dot{V}CO_2$). $\dot{V}O_{2max}$ was then calculated (Fairbarn et al. 1994).

Statistical analyses

Data were analysed using IBM's Statistical Package for the Social Sciences software (SPSS v25, IBM, USA). Analysis of co-variance (ANCOVA) was conducted to determine if there were statistically significant differences in the recorded variables between the exercise and control groups. The covariate used was the pre-test measure of each variable. If one ANCOVA assumption was violated, an independent *t* test was conducted on the difference between pre- and post-intervention results between groups. The results were considered statistically significant when the *p* value was < 0.05. Eta-squared (η^2) was used to determine effect size, with values of 0.02, 0.13, and 0.26 and above being considered to be small, medium, and large effect sizes. Data are reported as mean and standard deviation (SD).

Results

Participant characteristics

There were no significant differences (independent *t* tests) in age and height, $p > 0.05$, between the two groups (Table 1). In addition, there were no significant differences (ANCOVA) for weight, BMI, BSA, haematocrit, haemoglobin, and blood and plasma volume change, $p > 0.05$, at postintervention between the two groups (Table 1). The exercise program of 24 SIT cycling sessions over 8 weeks was completed with 100% compliance.

Resting cardiovascular response

The 8-week intervention program resulted in no significant difference (ANCOVA between groups) for the following resting variables: CO, PEP, PEP/LVET, BP, total peripheral resistance, CI, VFR, and VER (Table 2). However, there was a significant difference between groups in resting SV, $F(1, 27) = 13.6$, $p = 0.001$, $\eta^2 = 0.33$ (Fig. 1a), after 8 weeks of SIT. The exercise group increased their resting SV by 4.9% at post-SIT compared to the control group who reduced their resting SV at post-SIT by 3%. In addition, a significant difference between groups existed in SI at post-SIT, $F(1, 27) = 14.5$, $p = 0.001$, $\eta^2 = 0.35$. At post-SIT, the exercise group improved their SI by 4.4%. A significant difference between groups was also found in LVET at post SIT, $F(1, 27) = 5.97$, $p = 0.021$, $\eta^2 = 0.18$. The exercise group's LVET increased by 3.5% compared to a 2.8% drop for the control group at post-SIT. DFT was also significantly different between groups, $F(1,27) = 7.72$, $p = 0.010$, $\eta^2 = 0.22$ (Fig. 1b). The exercise group's DFT increased by 9.8% at post SIT. Resting HR was also found to be significantly different between groups, $F(1, 27) = 13.9$, $p = 0.001$, $\eta^2 = 0.34$ (Fig. 2). The exercise group's resting HR was decreased by

Table 1 Participant characteristics of exercise and control groups pre and post 8 weeks of intervention

Variable	Exercise ($n = 15$)		Control ($n = 15$)	
	Pre	Post	Pre	Post
Age (years)	53.2 (3.5)		53.0 (3.1)	
Height (cm)	164.1 (7.4)		161.4 (3.1)	
Weight (kg)	76.1 (13.9)	76.8 (13.9)	70.1 (2.8)	70.8 (2.9)
Body mass index (kg m^2)	28.2 (3.9)	28.4 (3.9)	26.9 (0.9)	27.2 (0.9)
Body surface area (m^2)	1.82 (0.2)	1.83 (0.2)	1.74 (0.03)	1.75 (0.03)
Haematocrit (%)	41.8 (2.7)	42.1 (3.1)	42.7 (2.7)	42.5 (2.3)
Haemoglobin (g L^{-1})	134.7 (8.7)	133.9 (10.1)	135.2 (7.7)	130.8 (7.7)
Blood volume change (%)	2.03 (4.7)		2.99 (5.7)	
Plasma volume change (%)	2.51 (8.9)		2.59 (6.8)	

Data are means with the standard deviation of the means in parenthesis

Table 2 Resting cardiovascular variables of exercise and control groups at pre and post 8 weeks of intervention

Variable	Exercise ($n=15$)		Control ($n=15$)	
	Pre	Post	Pre	Post
Heart rate (bpm)	66.6 (8.1)	62.8 (8.9)*	67.1 (8.5)	67.7 (8.9)
Stroke volume (mL)	77.5 (17.0)	81.3 (17.0)*	67.3 (13.5)	63.9 (14.3)
Cardiac output (L min^{-1})	5.2 (1.1)	5.0 (1.0)	4.5 (0.7)	4.3 (0.9)
PEP (ms)	131.0 (13.9)	133.1 (11.6)	131.5 (18.6)	129.4 (10.1)
LVET (ms)	303.3 (21.7)	314.0 (23.9)*	306.2 (26.7)	297.7 (34.0)
PEP/LVET	0.43 (0.04)	0.43 (0.08)	0.44 (0.08)	0.44 (0.08)
SBP (mmHg)	118.7 (13.5)	117.3 (14.3)	115.6 (18.6)	113.2 (16.3)
DBP (mmHg)	78.5 (12.0)	78.8 (9.7)	74.7 (9.7)	78.1 (11.6)
TPR (dyne s cm^{-5})	1486 (352)	1511 (313)	1627 (313)	1749 (383)
Stroke index (mL min m^2)	42.8 (10.0)	44.7 (10.0)*	39.0 (8.9)	36.8 (8.9)
Cardiac index (L min m^2)	2.9 (0.7)	2.7 (0.5)	2.6 (0.4)	2.5 (0.5)
Diastolic filling time (ms)	480.1 (99.5)	527.2 (123)*	471.2 (106)	475.2 (110)
Ventricular filling rate (mL s^{-1})	154.6 (37.5)	152.5 (38.3)	146.0 (27.9)	138.7 (37.2)
Ventricular emptying rate (mL s^{-1})	243.6 (50.7)	249.2 (44.1)	219.4 (36.8)	215.6 (47.2)

Data are means with the standard deviation of the means in parenthesis. Significant difference ($p<0.05$) between groups at post-SIT is represented by asterisks (*)

PEP pre-ejection period, *LVET* left ventricular ejection time, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *TPR* total peripheral resistance

5.7%, as opposed to a 1.0% increase for the control group at post-SIT.

Cardiovascular response during 8 min of light aerobic exercise

There were no significant differences (ANCOVA between exercise and control groups) for the following variables during the 8 min of light aerobic exercise at pre-to-postintervention: CO, PEP, PEP/LVET, CI, VER, and VFR (Table 3). However, there were significant differences between groups in SV, $F(1, 27)=20.7$, $p=0.000$, $\eta^2=0.43$ (Fig. 1a), SI, $F(1, 27)=19.1$, $p=0.000$, $\eta^2=0.43$, LVET, $t(28)=2.51$, $p=0.018$ (independent t test on change scores) (Table 3), and HR, $F(1,27)=4.38$, $p=0.046$, $\eta^2=0.14$. Exercise SV was increased by 5.8%, SI by 5.4%, LVET by 6%, and HR decreased by 4.6%. A significant difference between groups was also found for DFT, $F(1, 20)=6.32$, $p=0.021$, $\eta^2=0.24$, (Fig. 1b) with an increase of 7%. Due to technical difficulty during exercise DFT data collection, only 23 (13 exercise and 10 control participants) out of 30 participants were included in the data analysis.

Aerobic fitness

Predicted $\dot{V}O_{2max}$ was significantly (ANCOVA between exercise and control groups; $p=0.000$) different between groups, $F(1, 27)=2.57$, $p=0.016$ (Fig. 3). The post-SIT predicted $\dot{V}O_{2max}$ of the exercise group was significantly greater (21.4 ± 7.02 mL kg^{-1} min^{-1}) than that of the control group (18.7 ± 4.92 mL kg^{-1} min^{-1}).

Overall exercise heart rate during sprint interval training

Over 8 weeks of SIT, the average HR rate was 151 beats per minute (bpm) (week 1: 152 bpm; week 8: 150 bpm) with an average 122 RPM pedal cadence (week 1: 119 rpm; week 8: 125 rpm), 0.68 kg of load (week 1: 0.52 kg; week 8: 0.77 kg), 83.6 W of power output (week 1: 62 W; week 8: 97.6 W), and 14.5 on rating of perceived exertion (week 1: 14.3; week 8: 14.7). The exercise group consistently worked at over 70% of their estimated maximum HR of between 150 and 160 bpm. Increases in anaerobic fitness were also suggested by increases in RPM, load, and power, whereas HR and RPE decreased or stayed relatively constant, demonstrating that participants were able to generate more power at a lower HR.

Discussion

The major findings of this study were that following the 8-week SIT intervention, resting, and light aerobic exercise SV and DFT, and predicted $\dot{V}O_{2max}$ all significantly increased. These changes were accompanied by a decrease in resting and light aerobic exercise HR. No increase in plasma volume occurred. These results indicate that SIT is

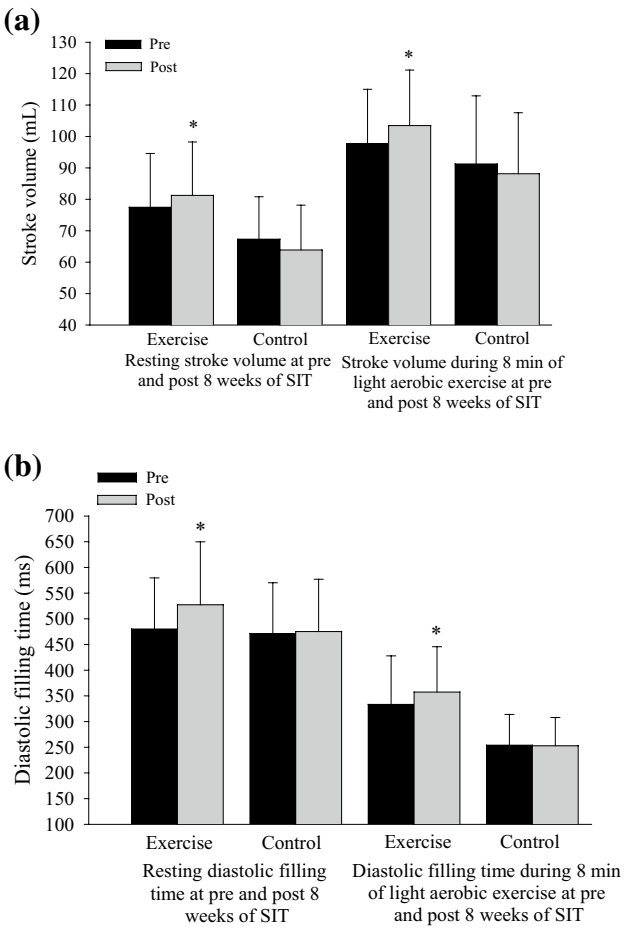

Fig. 1 **a** Resting (before and after the 8-week SIT intervention) and exercise stroke volume (during 8 min of light aerobic exercise, before and after the 8-week SIT intervention) and **b** resting (before and after the 8-week SIT intervention) and exercise diastolic filling time (during 8 min of light aerobic exercise, before and after the 8-week SIT intervention) in exercise and control groups. Significant differences ($p < 0.05$) between groups at post-SIT and during 8 min of light aerobic exercise are represented by asterisks (*)

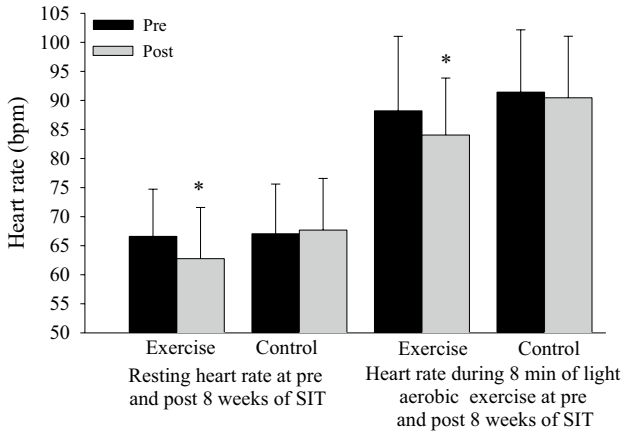

Fig. 2 Resting (before and after the 8-week SIT intervention) and exercise heart rate (during 8 min of light aerobic exercise, before and after the 8-week SIT intervention) in exercise and control groups. Significant differences ($p < 0.05$) between groups at post-SIT and during 8 min of light aerobic exercise are represented by asterisks (*)

an effective and time-efficient form of exercise for improving cardiac function and $\dot{V}O_{2max}$ of overweight postmenopausal women.

The SIT group increased exercise SV by 5.8%, whereas in a previous study, using SIT training young overweight males recorded an 18% increase in exercise SV (Heydari et al. 2013). This greater increase may have been brought about by the different ages (18–35 years versus 47–59 years) and gender of the participants and the longer duration of the intervention (12 weeks versus 8 weeks). SV is normally augmented by increased end-diastolic volume or preload and enhanced myocardial contractility (Lavie et al. 2015). Typically, aerobic exercise-induced blood volume expansion leads to increased venous return resulting in a higher preload, and due to the Frank–Starling law, an increase in myocardial contractility, thereby resulting in enhanced SV (Baggish et al. 2008). Plasma volume expansion after aerobic exercise training has been shown to play a major role in enhancement of aerobic power (Convertino 2007). In the present study, however, the SIT group showed no significant increase in blood or plasma volume. It is possible that the short duration of the work intervals (8 s) and low volume of SIT training (8 h) were unable to stimulate plasma volume expansion. These findings, however, are consistent with the previous studies that have demonstrated the inability of postmenopausal women to significantly expand their plasma volume with exercise (Stachenfeld et al. 1998).

However, the exercise used in these studies was aerobic in nature (Stachenfeld et al. 1998), thus more research examining the effect of sprint interval training on postmenopausal plasma volume expansion needs to be performed. This inability to expand plasma volume may be caused by the dramatic decrease in estrogen levels that occur after menopause (Wenner and Stachenfeld 2012). Research in this area, however, is limited; therefore, more studies need to be carried out before it can be concluded that estrogen reduction prevents plasma volume expansion. Lack of plasma volume expansion suggests that the enhanced SV of the SIT group was most likely a result of the 7% increase in DFT. Possible contributors to the increased DFT of these postmenopausal women are likely to be enhanced myocardial compliance (Levy et al. 1993; Moore 2006).

Interval compared to aerobic exercise has been shown to induce greater improvements in rats' myocardial compliance (Wisloff et al. 2009). Higher intensity exercise has also been shown to lead to greater expression of mRNA-encoding sarcoendoplasmic Ca^{2+} ATP-ase channels. Importantly, these channels augment myocardial compliance by increasing calcium resequestration during

Table 3 Cardiovascular variables averaged over 8 min of light aerobic exercise for exercise and control groups pre- and postintervention

Variable	Exercise ($n=15$)		Control ($n=15$)	
	Pre	Post	Pre	Post
Heart rate (bpm)	88.2 (12.8)	84.1 (9.7)*	91.4 (10.8)	90.5 (10.4)
Stroke volume (mL)	97.8 (17.4)	103.5 (17.8)*	91.3 (21.7)	88.2 (21.7)
Cardiac output (L min^{-1})	8.54 (1.5)	8.60 (1.2)	8.25 (1.7)	7.92 (1.7)
PEP (ms)	111.0 (13.2)	111.3 (11.6)	101.2 (21.3)	105.6 (15.1)
LVET (ms)	271.4 (27.9)	287.7 (22.5)*	295.3 (27.9)	274.4 (31.7)
PEP/LVET	0.40 (0.05)	0.37 (0.04)	0.35 (0.10)	0.39 (0.08)
Stroke index (mL min m^2)	54.1 (10.8)	57.0 (10.8)*	52.0 (15.1)	50.0 (13.2)
Cardiac index (L min m^2)	4.73 (0.9)	4.74 (0.8)	4.60 (1.1)	4.43 (1.2)
Diastolic filling time (ms)	333.4 (94.4)	357.4 (88.2)*	254.1 (73.1)	252.8 (67.3)
Ventricular filling rate (mL s^{-1})	357.6 (110.3)	347.8 (78.6)	345.8 (107.9)	313.9 (126.5)
Ventricular emptying rate (mL s^{-1})	355.2 (59.2)	352.3 (44.1)	304.7 (75)	314.4 (109.1)

Data are means with the standard deviation of the means in parenthesis. Significant difference ($p<0.05$) between groups at 8 min of light aerobic exercise is represented by asterisks (*)

PEP pre-ejection period, *LVET* left ventricular ejection time

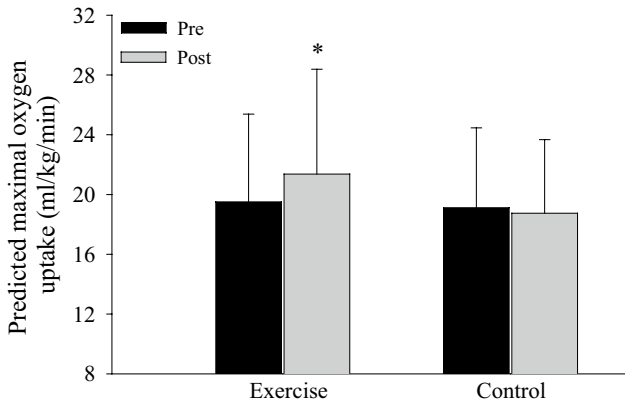

Fig. 3 Predicted maximal oxygen uptake of exercise and control groups at pre and post 8 weeks of sprint interval training. Significant difference ($p<0.05$) between groups at post-SIT is represented by asterisks (*)

myocyte relaxation (Strom et al. 2005; Wisloff et al. 2009). Myofilament responsiveness to calcium ions has also been demonstrated to increase in response to exercise training (Hammond et al. 1987). Therefore, enhanced myocardial compliance could be a major contributor to the increased SV of the SIT group. It has also been shown (Hammond et al. 1987) that down-regulation of β-adrenergic receptor number of the right atrium of pigs occurred after 10–19 weeks of treadmill running. Down-regulation of β-adrenergic receptor number of the right atrium would result in a decrease in exercise HR at the same absolute load, which would cause an increase in DFT. Therefore, it is feasible that due to the inability of postmenopausal women to expand their plasma volume, the most likely way for these women to increase their preload was by increasing DFT through cardiac adaptations such as increased myocardial compliance and/or down-regulation of β-adrenergic receptor number of the right atrium.

The SIT group significantly increased relative predicted $\dot{V}O_{2max}$ by 9.5%. Using the same protocol as that used in the present study, a 24% increase in $\dot{V}O_{2max}$ after a 15-week intervention was found in young women possessing normal weight (Trapp et al. 2008). The greater increase in $\dot{V}O_{2max}$ reported (Trapp et al. 2008) is likely to be due to the large age difference of the exercising participants (20 versus 53 years), as well as to the duration of intervention, which was almost twice as long (15 weeks). In addition, the inability of postmenopausal participants to expand blood volume may have contributed to their smaller increase in $\dot{V}O_{2max}$ (Stachenfeld et al. 1998). Nevertheless, the 9.5% increase in estimated $\dot{V}O_{2max}$ that followed after just 8 weeks of the current intervention may have significant clinical implications as low $\dot{V}O_{2max}$ is a major risk factor for cardiovascular disease mortality (Blair 2009).

At the end of the intervention, the SIT group increased their power output by 1.6-fold in 8 weeks. Impressively, despite exercising much harder closer to the end of the 8-week intervention program, participants still maintained a similar exercise HR to that in the beginning. Thus, these results confirm that SIT is a low-volume and time-efficient form of exercise that can be performed by sedentary and overweight postmenopausal women leading to significant cardiac changes in as little as 8 weeks or only 8 h of exercise.

One of the strengths of this study was that it was a supervised, randomized, control trial. Limitations include the characteristics of the participants who although overweight were relatively health conscious compared to the general population. Therefore, these findings should be only extrapolated to this kind of population. Another limitation is that the myocardial compliance of the participants was not directly

measured and also plasma volume was estimated and not directly assessed. Another limitation concerns the difficulty of getting impedance measurements when some participants were unable to keep their bodies still when exercising aerobically. Movement artefact may have contributed to the 3% reduction in stroke volume experienced by the control group after 8 weeks of no SIT exercise.

In summary, it was found that SIT improved predicted $\dot{V}O_{2max}$ of overweight, sedentary postmenopausal women. The increase in predicted $\dot{V}O_{2max}$ was largely brought about by increases in SV caused mainly by enhanced DFT. The SIT group also significantly decreased their exercise HR. In contrast, after SIT no significant changes in plasma volume occurred. Results also indicate that sedentary, overweight postmenopausal women were able to successfully complete this form of high-intensity exercise training. Increases in $\dot{V}O_{2max}$ have been shown to lead to decreased cardiovascular risk and mortality, suggesting that SIT can be used as a non-pharmacological way of enhancing the health of overweight postmenopausal women.

Acknowledgements We would like to thank Tze Yuen Ho, Vrischika Chabella, Alexandra Gleeson, Georgia Redmayne, Aengus Tran, Susan Li, and Helen Yoo for help with participant exercise training.

Author contributions DZ and TJ's contributions to the manuscript were equal. They shared first authorship. They organized data collection, data and statistical analyses, and contributed to the manuscript development. SB was responsible for the study design and manuscript development. YB organized participant recruitment, the testing timetable, statistical analyses, study design, and manuscript development.

Compliance with ethical standards

Conflict of interest None of the authors had a personal or financial conflict of interest. The study received no sources of funding. The results of the study are presented clearly, honestly, and without fabrication, falsification, or inappropriate data manipulation.

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