High intensity interval training (HIIT) improves resting blood pressure, metabolic (MET) capacity and heart rate reserve without compromising cardiac function in sedentary aging men

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ABSTRACT

Background: This study examined a programme of pre-conditioning exercise with subsequent high intensity interval training (HIIT) on blood pressure, echocardiography, cardiac strain mechanics and maximal metabolic (MET) capacity in sedentary (SED) aging men compared with age matched masters athletes (LEX).
Methods: Using a STROBE compliant observational design, 39 aging male participants (SED; n = 22, aged 62.7 ± 5.2 yrs) (LEX; n = 17, aged = 61.1 ± 5.4 yrs) were recruited to a study that necessitated three distinct assessment phases; enrolment (Phase A), following pre-conditioning exercise in SED (Phase B), then following 6 weeks of HIIT performed once every five days by both groups before reassessment (Phase C). Hemodynamic, echocardiographic and cardiac strain mechanics were obtained at rest and maximal cardiorespiratory and chronotropic responses were obtained at each measurement phase.
Results: The training intervention improved systolic, mean arterial blood pressure, rate pressure product and heart rate reserve (each $P < 0.05$) in SED and increased MET capacity in both SED and LEX ($P < 0.01$) which was amplified by HIIT. Echocardiography and cardiac strain measures were unremarkable apart from trivial increase to intra-ventricular septum diastole (IVSd) ($P < 0.05$) and decrease to left ventricular internal dimension diastole (LVId) ($P < 0.05$) in LEX following HIIT.
Conclusions: A programme of preconditioning exercise with HIIT induces clinically relevant improvements in blood pressure, rate pressure product and encourages recovery of heart rate reserve in SED, while improving maximal MET capacity in both SED and LEX without inducing any pathological cardiovascular remodeling. These data add to the emerging repute of HIIT as a safe and promising exercise prescription to improve cardiovascular function and metabolic capacity in sedentary aging.

1. Introduction

Normal aging is accompanied by diffuse alterations to cardiovascular structure and function that contrive to increase cardiovascular morbidity and mortality during advancing years. Since the landmark Dallas bed-rest study (Saltin et al., 1968), the pleiotropic effects of preserving cardiorespiratory fitness (CRF) during advancing age have become more widely appreciated such that achievable improvements in CRF (~1 MET) can profoundly impact health and survival (Kaminsky et al., 2013; Kodama et al., 2009) by improving CRF through effective physical activity regimens. However, because epidemiological studies consistently identify older adults as the least physically active demographic (Knowles et al., 2015) and because CRF is a greater prognostic indicator of mortality than 'physical activity' (Lee et al., 2010), then alternative strategies that improve CRF in older persons have become increasingly important.

High-intensity interval training (HIIT), is characterized by brief, intermittent bursts of vigorous exercise, interspersed by periods of low intensity recovery (Saltin et al., 1968). More recently, HIIT has meta-analytical support as a viable method to improve cardiovascular health by increasing cardiorespiratory fitness (CRF) in young healthy (Weston et al., 2014) and cohorts with lifestyle-induced cardiometabolic disease (Elliott et al., 2015; Weston et al., 2013). Of the few available data in older cohorts, HIIT appears to offer promising results in patients with coronary artery disease (Munk et al., 2009), following myocardial infarction (Moholdt et al., 2009) and hypertension (Molmen-Hansen et

al., 2012) but there is an absence of data in sedentary aging (Weston et al., 2014). Of contrasting note, a recent experiment described the potential for to HIIT to promote pathological adaptations in the left ventricle of hypertensive rats (Holloway et al., 2015), underlining the need for further study of HIIT on cardiac structure, hemodynamic strain and strain mechanics in sedentary aging.

Encouragingly, both the feasibility of HIIT and its prescription following an initial period of conditioning exercise have recently been established in aging cohorts (Knowles et al., 2015; Sculthorpe et al., 2017). However, because aging men can take longer to recover from single strenuous HIIT session than their younger counterparts (Herbert et al., 2015a), caution should be exercised when prescribing the volume of HIIT training in aging cohorts. With these aspects in mind, the present study set out to examine the effects of low frequency HIIT (once every 5 days) on: i) resting cardiac structure and function ii) blood pressure and rate pressure product, and iii) metabolic capacity (MET's) in a cohort of sedentary but otherwise healthy aging men (SED). These were compared with a positive control group of age-matched lifelong exercising masters athletes (LEX). We hypothesized that 6 weeks (9 sessions) of low frequency HIIT would (i) not alter cardiac structure or strain mechanics (ii) would favourably affect indicators of resting blood pressure and rate pressure product in SED compared with LEX. We further hypothesized that HIIT would (iii) positively impact maximal metabolic capacity (MET's) in SED compared with LEX.

2. Materials and methods

2.1. Participants

Following health screening, 44 aging male participants were enrolled to the study and allocated to one of two groups; (i) sedentary men (SED, n = 25; aged 62.3 ± 4.6 yrs) who did not participate in any formal exercise training and (ii) a positive control group of masters athletes (LEX, n = 19; aged 61.3 ± 5.1 yrs). LEX included active masters national competitors in sports including triathlon, athletics, sprint cycling and racquet sports. Participant characteristics are outlined in Table 1. Participants provided written informed consent in addition to a physical activity readiness questionnaire (PAR-Q) prior to enrolment to the study, which conformed with ethical guidelines of the 1975 Declaration of Helsinki and approved by institutional research board. The flow of participants through the study is outlined in Fig. 1.

2.2. Protocol and experimental procedures

The study employed a STROBE compliant observational design where SED undertook six weeks of supervised conditioning exercise (training block 1) as recently advised (Riebe et al., 2015), while LEX maintained their normal exercise regimens. SED and LEX participants kept a weekly log detailing exercise training, which were confirmed using telemetric heart rate data (Polar, Kempele, Finland). To account for the influence of preconditioning exercise, the study required 3 distinct measurement phases outlined in Fig. 2.

On each assessment phase, participants arrived in the laboratory following an overnight fast having abstained from caffeine and alcohol consumption for 36 h, and avoided strenuous exercise for a minimum of 5 days. Resting blood pressure was obtained using the auscultatory method according to guidelines of American Heart Association (Pickering et al., 2005), Resting heart rate was obtained following 10. mins supine rest (Polar T31, Kempele, Finland). Rate-pressure product (RPP) was calculated as the product of resting heart rate (bpm) and systolic arterial pressure (mm Hg) divided by 100. Echocardiographic measures were obtained according to current recommendations (Supplementary information 1) and left ventricular mass established according to the Penn convention (Devereux and Reichek, 1977) Maximal aerobic capacity was determined using open circuit spirometry using a Cortex II Metalyser 3B-R2 (Cortex, Biophysik, Leipzig, Germany) conforming to procedures outlined in more detail elsewhere (Grace et al., 2015; Knowles et al., 2015) and Metabolic Equivalents (MET's) established by dividing relative $\dot{V}O_{2\ max}$ by 3.5.

Table 1

Participant characteristics, confirmation of maximal effort and maximal cardiorespiratory mechanics in lifelong sedentary (SED) and lifelong exercisers (LEX) on enrolment to the study (Phase A); following conditioning exercise (Phase B) and following high intensity interval training exercise (HIIT; Phase C). Data are presented as mean ± S.D.

	SED group			LEX group		
	Phase A	Phase B	Phase C	Phase A	Phase B	Phase C
Participant characteristics						
Participant number (n)	25	22	22	19	17	17
Age (yrs)	62.7 ± 5.2			61.1 ± 5.4		
Height (cm)	175 ± 6.1			173 ± 5.5		
Body mass (kg)	89.9 ± 17.2[*]	88.9 ± 16.6[*,a]	89.0 ± 17.5[*,a]	79.5 ± 12.3	79.3 ± 12.3	80.1 ± 12.6
Confirmation of maximal effort (max)						
BLa⁻ max (mmol·L⁻¹)	9.8 ± 2.6	10.2 ± 2.3	9.1 ± 2.0	10.3 ± 2.3	10.7 ± 2.5	9.2 ± 2.4[b]
RERmax	1.10 ± 0.07	1.09 ± 0.05	1.14 ± 0.08	1.13 ± 0.07	1.11 ± 0.06	1.15 ± 0.07
RPEmax	17.4 ± 1.4	17.4 ± 1.2	18.2 ± 1.1	18.2 ± 1.3	18.2 ± 1.2	18.6 ± 1.0
HRmax	156 ± 9	159 ± 9	161 ± 8.8	161 ± 14	165 ± 12	163 ± 12
Maximal cardiorespiratory function						
$\dot{V}O_{2\ max}$ (L·min⁻¹)	2.46 ± 0.43[**]	2.52 ± 0.36[**]	2.82 ± 0.58[**,a,b]	3.08 ± 0.45	3.2 ± 0.57	3.45 ± 0.47[a,b]
$\dot{V}E_{max}$ (L·min⁻¹)	87.1 ± 20.6[**]	92.8 ± 17.7[**]	108.1 ± 21.3[**,a,b]	117.4 ± 19.3	116.2 ± 21.1	129.4 ± 18.3[a,b]
MET capacity	7.9 ± 1.6[**]	8.5 ± 1.2[**]	9.4 ± 1.4[**,a,b]	11.2 ± 1.7	11.6 ± 1.9	12.6 ± 1.8[a,b]
VT% $\dot{V}O_{2\ max}$	72.9 ± 10.3[*]	79.4 ± 8.8	81.3 ± 9.8	79.6 ± 8.5	79.1 ± 9.6	77.6 ± 7.5
AT% Abs $\dot{V}O_{2\ max}$	82.6 ± 10.0	83.4 ± 7.7	79.5 ± 8.9	84.6 ± 5.5	84.3 ± 8.6	82.6 ± 9.7
AT Abs $\dot{V}O_{2\ max}$ (L·min⁻¹)	1.97 ± 0.39[**]	2.1 ± 0.38[**]	2.24 ± 0.49[**,a]	2.61 ± 0.46	2.70 ± 0.55	2.84 ± 0.50[a]
O_2 pulse $\dot{V}O_{2\ max}$ (mL·beat⁻¹)	15.4 ± 2.5[**]	15.9 ± 2.3[**]	18.0 ± 4.0[**,a]	19.2 ± 2.7	21.0 ± 3.2	21.1 ± 2.3[a]
PO-$\dot{V}O_{2\ max}$ (W)	201 ± 42[**]	201 ± 33[**]	222 ± 30[**,a,b]	253 ± 37	254 ± 34	264 ± 41[a,b]

$\dot{V}O_{2\ max}$: maximal aerobic capacity; BLa⁻$_{max}$: maximum blood lactate; RER = respiratory exchange ratio; RPE = rating of perceived exertion; HR$_{max}$: maximum heart rate; $\dot{V}E_{max}$: maximum minute ventilation; MET: metabolic equivalent; VT% $\dot{V}O_{2\ max}$: ventilatory threshold as percentage of maximal capacity; AT% Abs $\dot{V}O_{2\ max}$: anaerobic threshold as percentage absolute maximal capacity; O_2 pulse: oxygen pulse at maximal capacity; PO-$\dot{V}O_{2\ max}$: peak power output at maximal capacity.

[*] P < 0.05 versus LEX at same time-point.
[**] P < 0.01 versus LEX and same time-point.
[a] P < 0.01 versus Phase A in the same group.
[b] P < 0.01 versus Phase B in the same group.

Assessed for eligibility
(n = 47)

Excluded (n = 3)

Not meeting inclusion criteria
(n = 3*)
Refused to participate
(n = 0)

(*3 x Known cardiovascular disease -
confirmed by General Practitioner)

Enrolled to Study (n = 44)

Allocated to SED (n = 25)

Received Training Block 1 (n =25)

Completed Training Block 1 (n =22)

Non Completions (n = 3*)

(*Reasons: 1 x Lower back Pain; 2 x
Dysrhythmia)

Allocated to LEX (n = 19)

Received Training Block 1 (n = 19)

Completed Training Block 1 (n=17)

Non Completions (n=2*)

(*Reasons: 1 x Dysrhythmia; 1 x
Personal issues unrelated to project)

Received Training Block 2 (n=22)

Completed Training Block 2 (n=22)

Non-Completions (n=0)

Received Training Block (n=17)

Completed Training Block 2 (n=17)

Non-Completions (n=0)

Analysed (n = 22)

Excluded from analysis
(n = 0)

Analysed (n = 17)

Excluded from analysis
(n = 0)

Enrollment
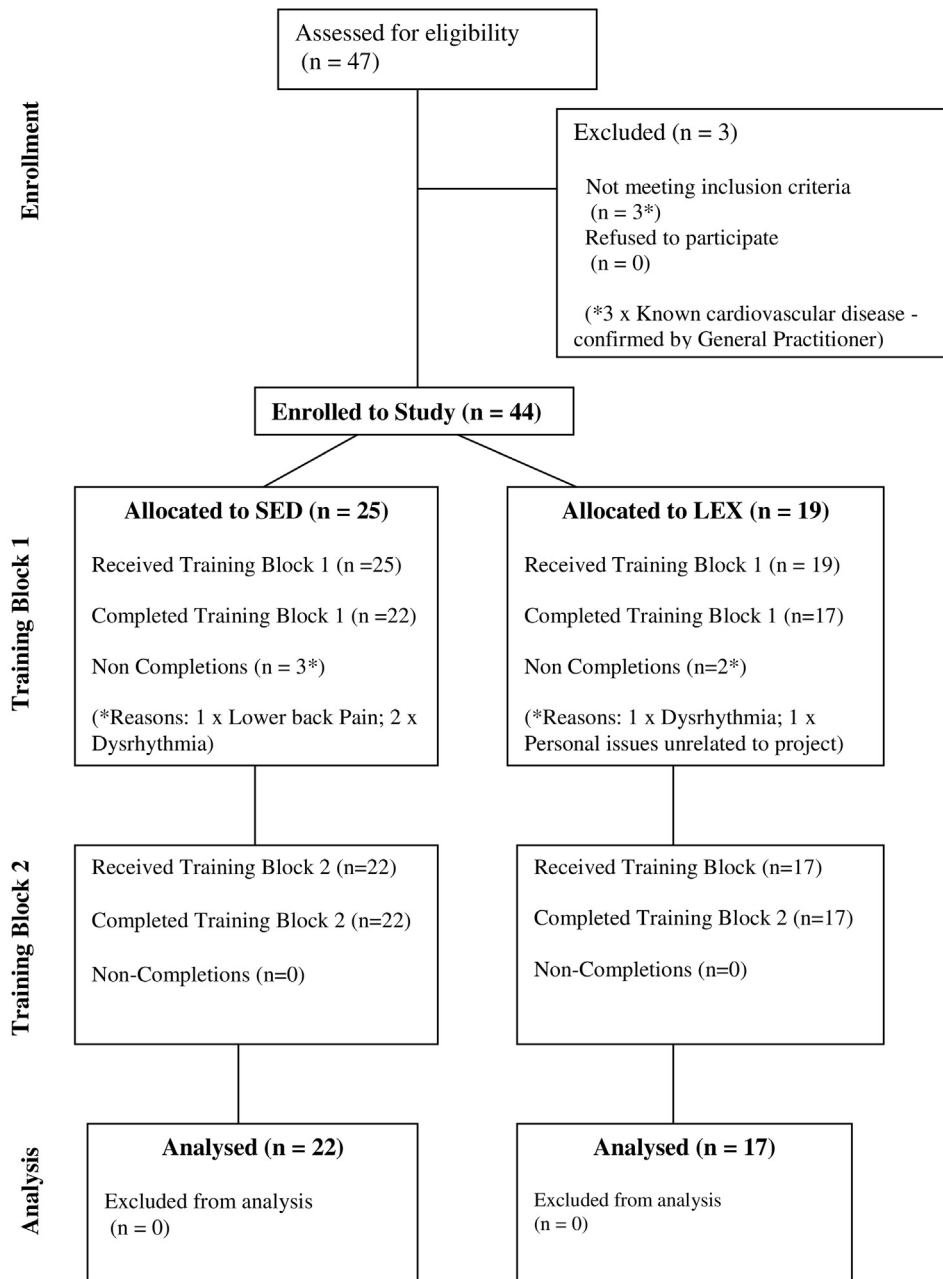
Training Block 1

Training Block 2

Analysis

Fig. 1. Flow of sedentary aging (SED) and lifelong exercising masters' athletes (LEX) through the study.

2.3. Exercise training protocol

During training block 1, SED participants underwent 6 weeks of supervised aerobic pre-conditioning exercise endorsed by ACSM guidelines for older adults (Chodzko-Zajko et al., 2009) where telemetrically derived heart rate reserve (HRR) was used to inform weekly progression in exercise load. During this time, LEX participants continued and recorded their habitual exercise training regimens. High intensity interval training (HIIT) during training block 2 consisted of HIIT exercise performed once every 5 days (HIIT) for 6 weeks (9 sessions in total) by both SED and LEX. Each session consisted of 6×30 s sprints at 50% of peak power output (determined using the Herbert 6 s peak power test (Herbert et al., 2015b)), interspersed with intervals of 3 min active recovery against a low (0–50 W) resistance and self-selected cadence. HIIT sessions were conducted in groups of 4–6 participants using Wattbike Pro cycle ergometers (Wattbike Ltd.,

Nottingham, UK). The HIIT sessions were the only exercise performed by either cohort during this training block period which preceded Phase C measurement.

2.4. Statistical analyses

Sample power was calculated using a single-tailed within-group comparisons with $\alpha = 0.05$ and $\beta = 0.95$, using $\dot{V}O_{2 \text{ max}}$ as described elsewhere (Grace et al., 2015) resulting in a required sample of n = 17. Data were analysed using SPSS version 20.0. Q-Q plots were employed to confirm normal distribution of data. Training effects were compared using a 2×3 (group $\times$ time) mixed design ANOVA with pairwise comparisons of within and between group simple effects including a Bonferroni correction. An alpha value of $P \leq 0.05$ was used to indicate statistical significance. Data are presented as mean $\pm$ standard deviation (S.D).
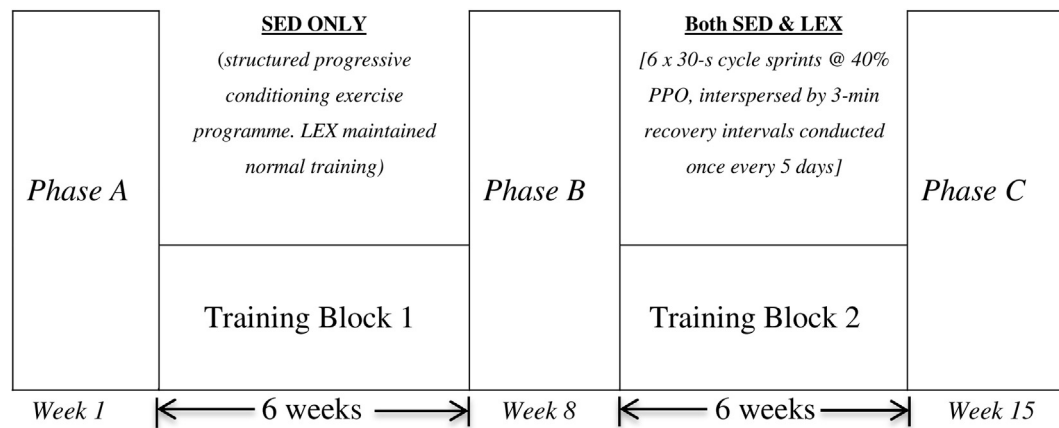
	SED ONLY			**Both SED & LEX**	
	(structured progressive conditioning exercise programme. LEX maintained normal training)			*[6 x 30-s cycle sprints @ 40% PPO, interspersed by 3-min recovery intervals conducted once every 5 days]*	
Phase A			*Phase B*		*Phase C*
	Training Block 1			Training Block 2	
Week 1	←——— 6 weeks ———→		*Week 8*	←——— 6 weeks ———→	*Week 15*

Fig. 2. Study schematic showing 3 measurement phases (Phases A; B; C) interspersed by 2 training blocks of 6 weeks duration; Training block 1: conditioning exercise for sedentary aging (SED) participants and Training block 2: high intensity interval training (HIIT) undertaken by lifelong sedentary (SED) and lifelong exercisers (LEX).

3. Results

From 44 enrolled participants, 39 completed the study and were included in the final analysis. Withdrawals (n = 5) occurred during training block 1 and included n = 3 from SED (1 × lower back pain; 2 × dysrhythmia) and n = 2 from LEX (1 × dysrhythmia, 1 × personal reasons). There was complete adherence to training block 2 (Fig. 1).

Maximal cardiorespiratory function during Phase A, B, C is documented in Table 1. Resting echocardiographic, hemodynamic and cardiac strain mechanics are documented in Table 2; Fig. 3. The effects of the intervention on cardiovascular, echocardiographic and cardiorespiratory measures are further dialogued in Supplementary information 2.

4. Discussion

The main findings of this study were that a programme of pre-conditioning with HIIT promotes clinically relevant improvements in resting haemodynamic stress (SBP, MAP, RPP) in SED and improves cardiovascular reserve (MET capacity) in both SED and LEX. without prompting cardiac strain or pathological cardiovascular remodeling. These data provide preliminary evidence for the prescription of the combination of pre-conditioning exercise and HIIT as a method to improve cardiovascular health in sedentary aging.

Significant improvements in BP (~− 7.7/− 4.6 mm Hg) and mean arterial blood pressure (~− 5.5 mm Hg) in the SED group are clinically relevant as arterial blood pressure is one of the most readily modifiable risk factors for cardiovascular disease (Lopez et al., 2006). It is notable

Table 2
Cardiac morphology (echocardiography), haemodynamic and cardiac strain mechanics obtained at rest in lifelong sedentary (SED) and lifelong exercisers (LEX) on enrolment to the study (Phase A); following conditioning exercise (Phase B) and following high intensity interval training exercise (HIIT; Phase C). Data are presented as mean ± S.D.

	SED group			LEX group		
	Phase A	Phase B	Phase C	Phase A	Phase B	Phase C
Echocardiography						
LVM (g)	223 ± 48	232 ± 44	241 ± 39	249 ± 41	254 ± 59	265 ± 70
LVMI (g·m^{-2})	89 ± 16.9	91.2 ± 11.8	97.2 ± 13.0	96 ± 16.2	98 ± 19.6	101 ± 22.3
IVSd (cm)	1.0 ± 0.15	1.0 ± 0.12	1.1 ± 0.08	1.0 ± 0.10	1.1 ± 0.16	1.2 ± 0.14[a]
LVIDd (cm)	5.14 ± 0.52	5.10 ± 0.30	5.16 ± 0.53	5.27 ± 0.48	5.13 ± 0.64	5.07 ± 0.51[a]
PWd (cm)	0.97 ± 0.13	1.01 ± 0.15	1.00 ± 0.09	1.05 ± 0.13	1.06 ± 0.12	1.10 ± 0.15
Hemodynamics						
SV (mL·beat^{-1})	67 ± 23	69 ± 19	70 ± 18	77 ± 19	73 ± 21	75 ± 23
EF (%)	55.6 ± 8.6	53.6 ± 4.6	53.5 ± 7.1	60.9 ± 5.1	55.3 ± 4.4	56.2 ± 4.9
CO (L·min^{-1})	4.3 ± 1.5	4.7 ± 1.7	4.4 ± 1.4	4.7 ± 1.2	4.8 ± 1.3	4.5 ± 1.4
E wave (m/s)	0.68 ± 0.14	0.66 ± 0.13	0.68 ± 0.14	0.70 ± 0.11	0.68 ± 0.10	0.70 ± 0.97
A wave (m/s)	0.63 ± 0.15	0.54 ± 0.13	0.55 ± 0.15[*]	0.54 ± 0.07	0.52 ± 0.06	0.49 ± 0.09
E:A	1.12 ± 0.26	1.26 ± 0.27	1.28 ± 0.30[*],[a]	1.31 ± 0.25	1.32 ± 0.26	1.46 ± 0.20
E′ (cm/s)	− 6.2 ± 1.3	− 6.2 ± 1.3	− 6.1 ± 1.3	− 7.4 ± 1.4	− 6.5 ± 1.5	− 6.4 ± 1.4
DBP (mm Hg)	87 ± 4.7[*]	83 ± 7.8	82 ± 7.8	82 ± 6.6	80 ± 6.6	79 ± 10.0
MAP (mm Hg)	104 ± 5.8[**]	101 ± 9.8	99 ± 7.8[a]	98 ± 7.7	96 ± 8.1	95 ± 10.4
PP (mm Hg)	53 ± 7.4	52 ± 11.7	49 ± 9.4	48 ± 7.9	47 ± 8.6	49 ± 8.8
RHR (bpm)	65 ± 11.8	64 ± 10.6	60 ± 7.6[*]	59 ± 9.7	57 ± 10.3	56 ± 4.4
Cardiac strain/strain rate						
PLS (%)	− 12.1 ± 2.78	− 14.8 ± 2.1	− 13.3 ± 3.9	− 16.37 ± 5.2	− 15.75 ± 4.6	− 15.77 ± 2.9
SRS (%/s)	− 1.2 ± 0.32	− 1.04 ± 0.28	− 1.18 ± 0.28	− 1.0 ± 0.27	− 1.0 ± 0.19	− 0.94 ± 0.16
SRE (%/s)	1.4 ± 0.48	1.3 ± 0.31	1.22 ± 0.33	1.46 ± 0.31	1.47 ± 0.29	1.34 ± 0.29
SRA (%/s)	1.26 ± 0.36	1.27 ± 0.21	1.31 ± 0.26	1.23 ± 0.26	1.19 ± 0.31	1.22 ± 0.34

LVM: left ventricular mass; LVMI: left ventricular mass index; IVSd: intra-ventricular septum diastole; LVIDd: left ventricular internal dimension diastole; PWd: posterior wall diastole; SV: stroke volume; EF: ejection fraction; CO: cardiac output; DBP: diastolic blood pressure; MAP: mean arterial blood pressure; PP: pulse pressure; RHR: resting heart rate; PLS: peak longitudinal strain; SRS: systolic strain rate, SRE: early diastolic strain rate, SRA: atrial systolic strain rate.

[*] P < 0.05 versus LEX at same time-point.
[**] P < 0.01 versus LEX and same time-point.
[a] P < 0.05 versus Phase A in the same group.
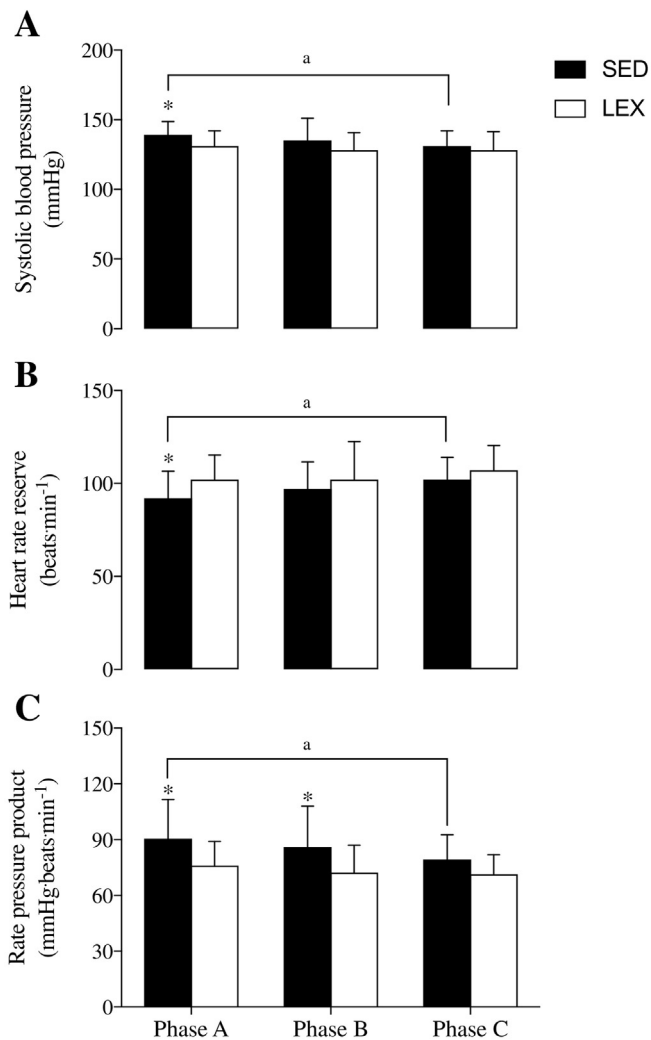
Fig. 3. A–C. A: Systolic blood pressure (mm Hg) B: heart rate reserve (beats·min^{-1}) C: rate pressure product (RPP; mm Hg·beats·min^{-1}) in lifelong sedentary (SED) and lifelong exercisers (LEX) on enrolment to the study (Phase A); following conditioning exercise (Phase B) and following high intensity interval training exercise (HIIT; Phase C). * = P < 0.05 versus LEX at same time-point; [a] = P < 0.01 Phase A versus Phase C in SED. Data are presented as mean ± S.D.

balance of sympathetic/parasympathetic drive resulting from the HIIT epochs during the intervention. Coupled with an improved O_2 pulse at maximal exercise in both SED and LEX (~+9%; +11% respectively) these data indicate an improved cardiometabolic efficiency at both rest and peak exercise.

4.1. Cardiac structure and function

Echocardiographic measures in the present study were largely unremarkable. There were some trivial physiological changes to LEX that were not evident in SED. For instance, a small increase in diastolic septal thickness along with a concomitant reduction in chamber diameter were evident in LEX only. This suggests an initiation of the cardiac remodeling process following HIIT, possibly related to the sudden reduction in training volume, where LEX group quite dramatically reduced their normal volume of exercise induced cardiac preload. The unchanged cardiac morphology within the SED group agrees with recent work which also failed to detect LV structural changes following 8 weeks of HIIT in sedentary seniors (Hwang et al., 2016). However, in contrast to our study, Hwang et al. (2016) reported increased LV ejection fraction at rest, the discrepancies between their results and those of the present study are likely due to the different methods of HIIT employed. Hwang et al. (2016) utilised 4 × 4 min HIIT and 3 × 3 min recovery intervals at 90 and 70% HRmax respectively, using an all-extremity ergometer, representing 16 min of HIIT work per session. The present study used 6 × 30 s representing 3 min of HIIT work per session. Hwang et al. (2016) further present a significant, positive relationship between changes in ejection fraction and $\dot{V}O_{2peak}$ suggesting that enhanced aerobic fitness during whole body exercise could be the result of central, in addition to peripheral adaptations (Hwang et al., 2016). In contrast, the improved maximal MET capacity presented herein are attributable to peripheral adaptations as ultrasonically derived cardiac structure was unaltered by a combination of conditioning exercise and HIIT. Clearly, volume dependant adaptations following different modes of HIIT exercise in older individuals requires further study.

With regards to newer methods of assessing LV systolic function, reductions in longitudinal strain have been suggested as an early marker of LV dysfunction (D'Ascenzi et al., 2016), especially in recreationally active non-elite populations. However, in the present study there were no changes in longitudinal strain, and no differences between groups. Correspondingly, the present data disagree with a recent hypothesis that HIIT may induce maladaptive responses in humans, as demonstrated in rats with hypertensive heart failure (Holloway et al., 2015). Findings of unchanged PLS, SRS, SRE and SRA observed in this study agree with previous work employing either shorter (12 days) (Wright et al., 2014) or longer (16-weeks,) (Scharf et al., 2015) of HIIT training. Similarly, investigations using a young healthy cohort have also failed to demonstrate changes in longitudinal strain or strain rates at rest following HIIT (12 days - 6 sessions; (Esfandiari et al., 2014)). Thus, the present study aligns ageing data with current consensus in young healthy groups. Recently, a recent meta-analytical review of resistance and endurance trained sportsmen (Beaumont et al., 2016a). Observed that exercise training has a negligible effect on resting measurements of global longitudinal strain. This could be due to the minimal change in longitudinal strain even during submaximal exercise (Doucende et al., 2010) and thus, a baseline adaptation may not be necessary. Conversely, LV twist has shown progressive augmentation with increasing exercise intensity, (Beaumont et al., 2016b; Doucende et al., 2010; Stohr et al., 2011) which may indicate a more fundamental role of cardiac twist mechanics in supporting systolic function. Indeed, a baseline adaptation in twist has recently been observed in elite endurance athletes (Beaumont et al., 2016a). Since LV twist increases with advancing age (Takeuchi et al., 2006) and since chronic endurance training is associated with reduced twist at rest (Maufrais et al., 2014) and augmented twist during exercise (Lee et al., 2012) in older athletes compared to their untrained

that the magnitude of improvement is comparable to the 8.2 mm Hg observed in a recent meta-analysis of 26 randomized controlled trial groups of hypertensives undergoing endurance training, and greater than pre-hypertensive (−2.1/−1.7 mm Hg) and normal BP (−0.75/−1.1 mm Hg) subject groups reported in the same meta-analysis (Cornelissen and Smart, 2013). Similarly, when considered alongside the previous work of Molmen-Hansen and colleagues in their clinical hypertensive model (2012), the present data provide encouraging support for HIIT to induce clinically meaningful improvements blood pressure. This, alongside a concomitant improvement in RPP (−12%) and MAP (~5%) provides further indirect evidence of improved cardiac efficiency in the SED group.

One of the inexorable facts of advancing age is a gradual reduction in cardiac output, due in large part, to a concomitant decline in maximum heart rate in all aging demographics (Astrand et al., 1973; Heath et al., 1981). Although there were no statistically significant changes in either resting or max heart rate in SED, small but favourable margins of improvement in both, coalesced to induce a significant improvement in HRR (~10%) following the HIIT exercise (Fig. 3). This novel finding indicates some form of chronotropic plasticity that is augmented by HIIT exercise and is previously unreported. We propose that improved HRR is unlikely to be an effect of intrinsic firing rate, but rather an improved

counterparts, the potential for low frequency HIIT to produce similar functional adaptations in the aging population is an interesting prospect. Nevertheless, when taken in context with the inexorable fact that advancing age is the most powerful predictor of cardiovascular disease, the present findings provide encouraging support for the prescription of HIIT (following preconditioning) in sedentary aging men, despite recent caution raised by Holloway et al. (2015) in their hypertensive rat model.

4.2. Functional capacity

The observation that on enrolment LEX had ~44% higher $\dot{V}O_{2\,max}$ than SED is similar to recent comparisons in trained and untrained seniors (Iversen et al., 2011; Shibata and Levine, 2012), and corresponds with the commonly reported observation that age related decrements in aerobic capacity are more pronounced amongst untrained individuals (Seals et al., 2011). Furthermore, SED demonstrated improvements in relative $\dot{V}O_{2\,max}$ following both conditioning exercise and HIIT, which supports the tenet that older individuals can positively influence recovery of metabolic capacity with either aerobic or high intensity training (Murias et al., 2010; Poulin et al., 1992). Extended discussion in Supplementary information 3.

Comparison with previous work examining the effectiveness of HIIT in older populations is confounded by the lack of similar studies using apparently healthy sedentary aging participants. Indeed, it is more common that comparably age matched studies involve participants with existing pathologies. Such studies often demonstrate larger proportional improvements in $\dot{V}O_{2\,max}$ than is the case in the current study. Another recent meta-analysis examining HIIT in patients with existing lifestyle induced cardiometabolic disease reported a mean increase in aerobic capacity of 19.4% (Weston et al., 2013). The large effect is likely due to lower aerobic capacity at study enrolment (Wisløff et al., 2007). Direct comparison is further limited by the extensive vascular dysfunction in these populations, and compounded by confounding effects of concomitant pharmacological intervention. Nevertheless, the results of the present study contribute to the body of HIIT literature by demonstrating that healthy sedentary older participants can safely undertake high intensity training and enjoy clinically relevant improvements in metabolic capacity consistent with improved mortality (Kaminsky and others, 2013). Moreover, the magnitude of improvement in SED is comparable both with their younger counterparts and diseased older peers and does not seem to be adversely affected by a comparably lower training frequency. Extended discussion in Supplementary information 4.

Despite contravening of the recommended exercise volume for older participants endorsed by ACSM (Chodzko-Zajko et al., 2009) and AHA (Nelson et al., 2007), the HIIT portion of this study offers new information relating to the potential for HIIT to positively impact resting cardiovascular stress in SED and improve cardiovascular reserve in both SED and LEX. Although this observational study design precludes direct comparison between HIIT and aerobic exercise, the data are supported by the observation that LEX, despite routinely undertaking a high volume of exercise training, also enjoyed increases in MET capacity. Similarly, the contribution of aerobic conditioning should be noted. In contrast to the HIIT portion, aerobic preconditioning induced small but significant improvements in body composition and is likely to have contributed to overall improvements in resting cardiovascular hemodynamics, and provided a platform for SED to complete a HIIT programme. Consequently, we emphasise that HIIT is feasible in sedentary aging cohorts, when it is preceded by a programme of cardiovascular conditioning.

The present study has some important limitations that should be noted. One concerns the proximity of the conditioning programme (training block 1) to the HIIT intervention (training block 2), which makes it impossible to rule out the contribution of preconditioning exercise to the overall effect on SED following HIIT. However, this prudent

approach is endorsed by recent experimental work (Sculthorpe et al., 2017) and consensus statement (Riebe et al., 2015) and further justified in light of the complete adherence to the HIIT programme. Further, aerobic improvements in LEX in response to HIIT indicate that similar improvements in SED are as a consequence of the HIIT stimulus, rather than a residual effect of the cardiovascular conditioning exercise.

5. Conclusions

In conclusion, a programme of pre-conditioning and HIIT promotes clinically relevant improvements in resting haemodynamic stress (SBP, MAP, RPP) in SED and that HIIT similarly accelerates the improvement in MET capacity in both SED and LEX without compromising myocardial structure or strain mechanics. Consequently, these data support the prescription of HIIT with preconditioning as a method to improve cardiovascular health in sedentary aging men.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.exger.2017.05.010.

Conflict of interest

The authors have no conflicts of interest to declare.

Abbreviations

SED	sedentary aging men
LEX	lifelong exercising mas1ters athletes
HIIT	high intensity interval training
$\dot{V}O_{2\,max}$	maximal aerobic capacity
$\dot{V}E_{max}$	maximum minute ventilation
VT% $\dot{V}O_{2\,max}$	
ventilatory threshold as percentage of maximal capacity	
AT% Abs $\dot{V}O_{2\,max}$	
anaerobic threshold as percentage absolute maximal capacity	
O_2 pulse	oxygen pulse at maximal capacity.
PO-$\dot{V}O_{2\,max}$	
peak power output at maximal capacity	
LVM	left ventricular mass.
LVMI	left ventricular mass index
IVSd	intra-ventricular septum diastole
LVIDd	left ventricular internal dimension diastole
PWd	posterior wall diastole
SV	stroke volume
EF	ejection fraction
CO	cardiac output
SBP	systolic blood pressure
DBP	diastolic blood pressure
MAP	mean arterial blood pressure
PP	pulse pressure
RHR	resting heart rate
HRR	heart rate reserve
RPP	rate pressure product
PLS	peak longitudinal strain
SRS	systolic strain rate
SRE	early diastolic strain rate
SRA	atrial systolic strain rate

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