

Research article

## The Effects of Standardised versus Individualised Aerobic Exercise Prescription on Fitness-Fatness Index in Sedentary Adults: A Randomised Controlled Trial

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### Abstract

A poor Fitness Fatness Index (FFI) is associated with type 2 diabetes incidence, other chronic conditions (Alzheimer's, cancer, and cardiovascular disease) and all-cause mortality. Recent investigations have proposed that an individualised exercise prescription based on ventilatory thresholds is more effective than a standardised prescription in improving cardiorespiratory fitness (CRF), a key mediator of FFI. Thus, the aim of the current study was to determine the effectiveness of individualised versus standardised exercise prescription on FFI in sedentary adults. Thirty-eight sedentary individuals were randomised to 12-weeks of: (1) individualised exercise training using ventilatory thresholds ( $n = 19$ ) or (2) standardised exercise training using a percentage of heart rate reserve ( $n = 19$ ). A convenience sample was also recruited as a control group ( $n=8$ ). Participants completed CRF exercise training three days per week, for 12-weeks on a motorised treadmill. FFI was calculated as CRF in metabolic equivalents (METs), divided by fatness determined by waist to height ratio (WtHR). A graded exercise test was used to measure CRF, and anthropometric measures (height and waist circumference) were assessed to ascertain WtHR. There was a difference in FFI change between study groups, whilst controlling for baseline FFI,  $F(2, 42) = 19.382$   $p < .001$ , partial  $\eta^2 = 0.480$ . The magnitude of FFI increase from baseline was significantly higher in the individualised (+15%) compared to the standardised (+10%) ( $p = 0.028$ ) and control group (+4%) ( $p = <.001$ ). The main finding of the present study is that individualised exercise prescription had the greatest effect on improving FFI in sedentary adults compared to a standardised prescription. Therefore, an individualised based exercise prescription should be considered a viable and practical method of improving FFI in sedentary adults.

**Key words:** Ventilatory threshold, HRR, epidemiology, central obesity, physical activity.

### Introduction

A sedentary lifestyle which includes prolonged sitting time during waking hours and low energy expenditure, increases the risk of adverse health events, cardiovascular disease, and type two diabetes (T2DM) (Dempsey et al., 2014). This type of lifestyle leads to lower levels of cardiorespiratory fitness (CRF) and increased fatness, increasing cardiometabolic risk and mortality (Laukkanen and Kujala, 2018). Improving CRF and lowering fatness with exercise promotes cardiovascular health and longevity (Garber et al., 2011; Strasser and Burtcher, 2018; Garber, 2019). The Fitness-Fatness Index (FFI),

developed in 2016, combines these two cardiometabolic risk factors and has been shown to be better at identifying those at risk of adverse cardiovascular events than either measure alone (Barry et al., 2014; Sloan et al., 2016; Frith and Loprinzi, 2017a). A poor FFI has been associated with the development of major non-communicable conditions (T2DM, Alzheimer's disease, cancer, and cardiovascular disease) and all-cause mortality (Frith and Loprinzi, 2017a, b, c). FFI is calculated by measuring maximal CRF in metabolic equivalents (METs) divided by the waist to height ratio (WtHR) (Sloan et al., 2016). A 1-unit increase in FFI is clinically significant and has been found to reduce all-cause mortality (9%) and cardiovascular disease (CVD) specific mortality by 13% (Edwards et al., 2017).

Evidence shows that a vigorous to high-intensity exercise prescription can elicit more favourable cardiometabolic health benefits? relative to the current exercise guideline of moderate-intensity continuous training (Hussain et al., 2016; Su et al., 2019). Furthermore, vigorous to high-intensity exercise has shown to require less total exercise volume and time commitment to elicit these favourable health outcomes (Hussain et al., 2016; Su et al., 2019). The current physical activity guidelines targeting fitness and fatness alone utilise a standardised exercise prescription, using either relative percentages of maximal oxygen uptake ( $VO_2$  max),  $VO_2$  reserve ( $VO_2R$ ), or heart rate reserve (HRR) to establish exercise at moderate- (40 - 60% HRR or  $VO_2R$ ) or vigorous to high-intensity (60 - 80% HRR or  $VO_2R$ ) (Weatherwax et al., 2016). This generic method of exercise prescription, however, often results in a wide variability in responses due to the large inter-individual variation in the metabolic responses to exercise training (Bouchard et al., 2012; Scharhag-Rosenberger et al., 2012; Mann et al., 2014). To better account for the individual metabolic responses when prescribing vigorous to high-intensity exercise, a more individualised approach has been proposed since a standardized method both over- and under-estimates the metabolic responses (Weatherwax et al., 2019).

Recent evidence has suggested that individualised exercise prescription using ventilatory thresholds enhances the potential benefits of regular exercise on health outcomes (Wolpern et al., 2015). It has been shown that an exercise intensity prescribed relative to an individual's ventilatory thresholds 1 and 2 (VT1 and VT2) accounts for the individual variability in training responsiveness as

individual metabolic differences are taken into consideration (Wolpern et al., 2015). Specifically, a previous study demonstrated that training near VT2 improved exercise tolerance, exercise adherence and overall health outcomes in untrained individuals (Londeree, 1997).

The aim of the present study was to investigate changes in FFI in sedentary adults following a 12-week individualised ventilatory threshold-based exercise prescription versus a standardised exercise prescription based on %HRR. It was hypothesised that there would be a greater effect from individualised exercise prescription on FFI, compared to a standardised exercise prescription.

## Methods

Participants in this study were recruited from a community wellness program and the surrounding community via newspaper advertisement at the local university and word of mouth. To be included in the study, participants had to be between the ages of 30 and 75 years, participate in less than 30 minutes of moderate intensity physical activity on three days a week or less, and be considered low to moderate risk as determined by the American College of Sports Medicine 2014 standards (Pescatello 2013). Participants were excluded from the study if signs or symptoms of cardiovascular, metabolic, or pulmonary conditions were identified from a medical history questionnaire and interview. After recruitment, men and women were randomised into one of two experimental groups. A third control group was also recruited as a separate convenience sample from the other experimental participants (Etikan, 2016). Control participants were interested in the multiple health indices from the laboratory testing; however, they were not interested in participating in an exercise intervention. This group was recruited separately due to the moral and ethical considerations of withholding a known psychological and physiological benefit (i.e., an exercise intervention). Control group participants were required to meet all of the inclusion/exclusion criteria. The control group completed all laboratory testing at baseline and 12 weeks and were encouraged to maintain their current lifestyle (physical activity and diet) after baseline testing. Twelve hours prior to all testing sessions, participants were instructed to only consume water and avoid any strenuous exertion. Baseline and post-week 12 testing occurred on the same day of the week as close to the same time of day to mitigate any possible changes as a result of the timing of the testing and to ensure consistency. All post-week 12 testing occurred within one to four days of the last exercise intervention session. All participants provided written and verbal consent prior to commencing the study at the initial screening. To be able to interpret baseline testing results and to prescribe correct targeted exercise intensities explained below, one primary investigator knew the group allocation of participants. In order to reduce bias research assistants supervised and ran CRF (cardiorespiratory fitness) sessions in place of the primary investigator. A CONSORT diagram is shown in Figure 1. The study was approved by the Western State Colorado University

Institutional Review Board (HRC2016-01-90R6).

### Establishment of Fitness Fatness Index (FFI)

To establish FFI and changes in FFI (Sloan et al., 2016), anthropometric measurements of waist (cm) and height (cm) and maximal CRF testing was required at baseline and 12-weeks post intervention. The FFI was calculated as maximal CRF expressed as metabolic equivalents (METs) divided by the waist-to-height ratio (WtHR).

$$[FFI = CRF (METs) / WtHR]$$

CRF was determined by the maximal oxygen uptake ( $VO_{2max}$  mL·kg<sup>-1</sup>·min<sup>-1</sup>) via a graded maximal exercise test (GXT), in further detail below, converted to METs by dividing by 3.5 mL·kg<sup>-1</sup>·min<sup>-1</sup>. WtHR was calculated by dividing the anthropometric measurements of waist (cm) by height (cm). A higher number indicates a greater FFI.

### Anthropometric measurements

Waist circumference (WC) was measured at baseline and 12 weeks post-intervention to the nearest 0.5cm adhering to the ACSM standardised guidelines (Liguori 2020). Participant height was measured to the nearest 0.5 cm using a WB-3000 stadiometer (Tanita Corp., Tokyo, Japan). Participant body mass was measured at baseline and 12 weeks post-intervention to the nearest 0.1kg using a medical grade scale.

### Resting measures - heart rate and blood pressure

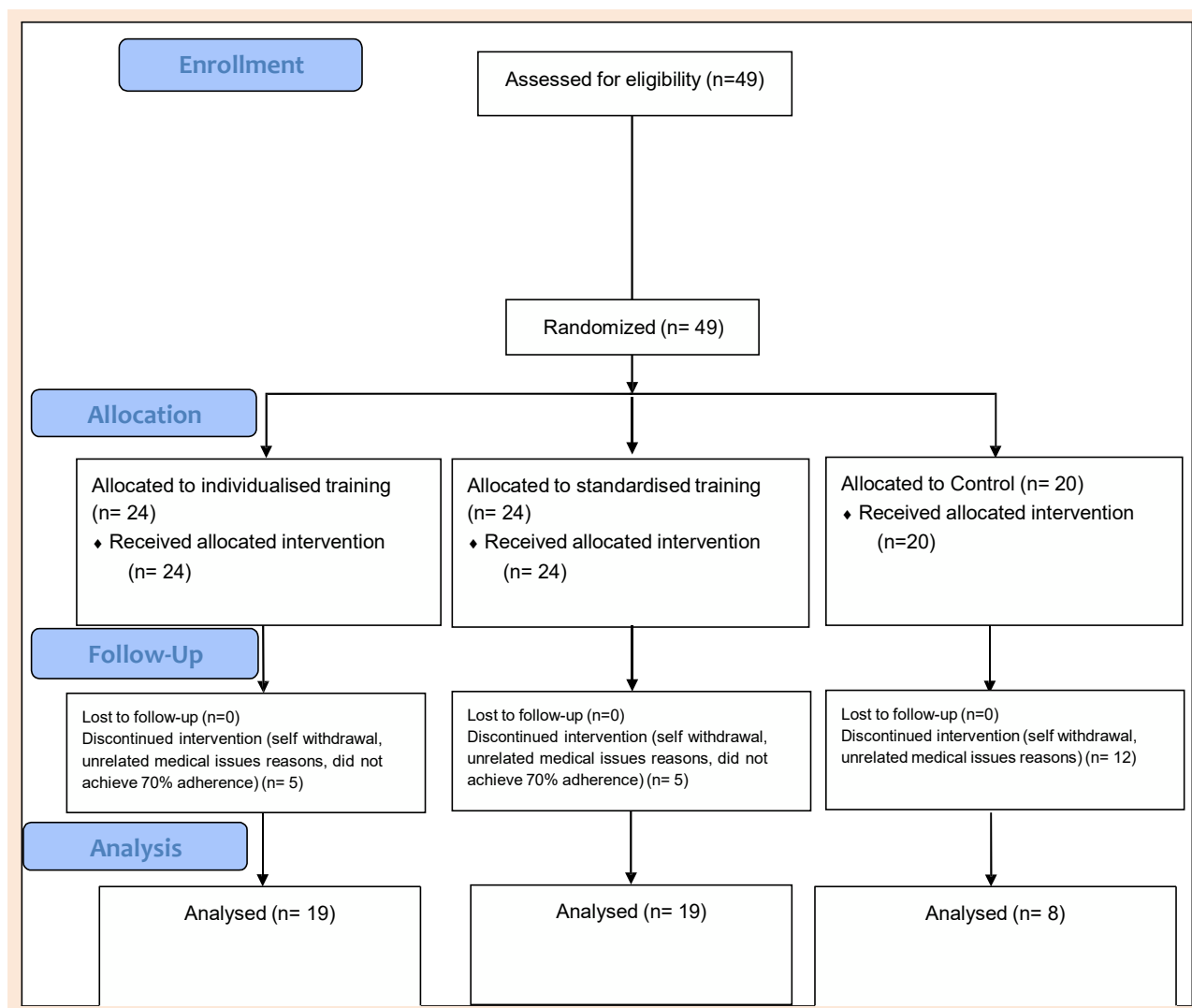
Prior to a graded maximal exercise test (GXT) a pre-exercise heart rate (HR) was measured using a medical-grade pulse oximeter and blood pressure (BP) using a sphygmomanometer (American Diagnostic Corporation Diagnostic 700 Series, USA) and standard stethoscope using ACSM Standardised Guidelines (Liguori 2020).

### Analysis of physical activity and diet

Participants for both experimental groups and control group completed the IPAQ - International Physical Activity Questionnaire (Lee et al., 2011) at baseline and 12-weeks post-intervention, to establish participants' daily activity and weekly PA levels (METs min·wk<sup>-1</sup>). While completing the baseline testing, researchers ensured participants understood the associated time and intensity levels of their PA, while also assisting in identifying participants' sedentary behaviour levels met the study inclusion criteria. All participants were asked to verbally agree to maintain their current diets for the duration of the 12-week research intervention and asked to complete a dietary log for three days at baseline and 12-weeks post intervention, including one weekend day, and two weekdays.

### Maximal exercise testing and verification protocol

The GXT was completed on a motorised treadmill (Powerjog GX200, USA) using a Balke pseudo-ramp protocol (Taylor et al., 2015) beginning at a participant self-selected starting pace to establish each participant's VT1 and VT2 and  $VO_{2max}$ .



**Figure 1.** Consort flow diagram for current study. Please increase the fonts to make it readable.

The GXT started with a 4-minute warm up at 0% incline, with an increase of work rate until the participant's chosen selected starting pace was reached. Once the participant's self-selected pace was reached, the incline percentage was then increased each minute by 1% until the participant expressed their inability to continue. During each GXT the expired air and gas exchange data was monitored and recorded using a Parvo metabolic analyser (Parvo Medics True One 2.0, USA), while HR during the GXT was monitored utilising a HR chest strap monitor (Polar Electro, USA). Prior to the GXT, calibration of the Parvo metabolic analyser as per the Parvo Medic user manual guidelines was completed, with a calibrated gas mixture of 16% O<sub>2</sub>, 4% CO<sub>2</sub>. Room air was measured at 20.93% O<sub>2</sub> and 0.03% CO<sub>2</sub>. At the termination of each GXT, the final data point was determined by averaging the last fifteen seconds of gas analysis data. The fifteen seconds prior to this data was averaged, and the two averages were used as final data points to represent the VO<sub>2</sub> max reached for each participant's GXT. To calculate %HRR, resting HR was subtracted from the maximal HR reached during the GXT. Guidelines from previous research were used to determine VT1 and VT2 during the GXT by using time points and the ventilatory equivalents

of O<sub>2</sub> (VE/VO<sub>2</sub>) and CO<sub>2</sub> (VE/VCO<sub>2</sub>) while also visually analysing the gas exchange data (Wolpern et al., 2015; Dalleck et al., 2016; Weatherwax et al., 2016). Previous research suggests VT1 occurs when VE/VO<sub>2</sub> increases without a linear increase in VE/VCO<sub>2</sub>, while VT2 occurs where both VE/VO<sub>2</sub> and VE/VCO<sub>2</sub> increase concurrently.

In order to determine if participants reached maximal CRF capacity during the GXT, a supramaximal verification test was completed. This verification is effective in confirming if a VO<sub>2</sub> max has been reached in middle-aged and older adults (Dalleck et al., 2012). Verification testing was completed 20 minutes after the GXT, based on current evidence by previous studies (Nolan et al., 2014; Weatherwax et al., 2016). The verification test utilised a work rate of 105%, which was adequate at obtaining verification testing durations of approximately 2-3 minutes (Dalleck et al., 2012; Astorino et al., 2013). The same protocols for analysis of the GXT was followed in the verification test. Following guidelines from previous research and the measurement of error of the metabolic analyser, if the GXT and verification test were equal or <3%, a "true" VO<sub>2</sub>max was confirmed (Dalleck et al., 2012). If participants had a VO<sub>2</sub>max >3% during their verification test they were asked to repeat the verification

test 24-72 hours after the initial verification test until a difference of <3% was achieved.

### Exercise intervention - prescription and training

For the exercise intervention, participants attended the exercise laboratory three days per week (Monday, Wednesday, and Friday). Participants were asked to be seated for five minutes on arrival for an initial resting period. Following the rest period pre-exercise HR and BP was measured for relative and absolute contraindications to exercise (Liguori 2020). Following resting measures participants were asked to begin a warm-up using a motorised treadmill (Powerjog GX200, USA) beginning at the participant's self-selected pace for 5 minutes, gradually increasing the speed and/or inclination until the determined individualised or standardised prescribed exercise HR had been reached. Participants were then asked to exercise within their prescribed individualised or standardised HR 'intensity domain' for their prescribed time, based on a participant calculated energy expenditure which was determined from values acquired during the GXT and explained in further detail in determination of exercise intensity, duration, and progression below. To ensure the relative prescribed exercise intensity was being adhered to an Exercise Physiologist (EP) or research assistant continually monitored HR using a HR chest strap monitor (Polar Electro, USA). HR was monitored continually with the treadmill work rate adjusted accordingly throughout the session to ensure the prescribed intensity or target HR range (as shown below) was maintained. Participants completed a cool-down for five minutes following the exercise session. The exercise intensity during the cool-down was progressively reduced until a HR within 15 beats per minute ( $b \text{ min}^{-1}$ ) of the participants' pre-exercise HR value was reached.

### Determination of exercise intensity, duration and progression

Target exercise intensity prescription for the individualised group was achieved by the relevant HR value associated with established ventilatory threshold VT1 and VT2 (Table 1) which were determined during the GXT. The calculation of HR values correlated with VT1 and VT2 values was established prior to the initiation of exercise sessions, with the following three target HR exercise intensity ranges:

Individualised Group Exercise Intensity:

- HR range of HR at VT1 and 10bpm below =  $HR < VT1$
- HR range of 15bpm directly between VT1 and VT2 = Target  $HR \geq VT1$  to  $< VT2$
- HR range of 10bpm above VT2 = Target  $HR \geq VT2$

Week to week progression of exercise prescriptions is shown in Table 1.

For the standardised group, exercise intensity prescription was determined based on the %HRR achieved during the maximal GXT and determined prior to exercise sessions (Table 1) using the following calculation:

Standardised Group Exercise Intensity:

$$\%HRR = [(Maximal HR - Resting HR) \times Desired \%] + Resting HR$$

Evidence suggests that an energy expenditure in the ranges of 4 (Church et al., 2007) to 23kcal  $\text{kg}^{-1} \text{week}^{-1}$  (Kraus et al., 2001; Slentz et al., 2004; Lee et al., 2012; Dalleck et al., 2015) showed improvements in CRF and has positive effects on metabolic and cardiovascular responses to exercise. Consequently, this RCT utilised a twelve-week exercise intervention protocol similar to previous research to determine exercise duration (Wolpern et al., 2015). Exercise duration for the experimental groups was calculated based on weekly energy expenditure per kg body mass ( $\text{kcal} \text{kg}^{-1} \text{week}^{-1}$ ) - instead of a determined duration for each exercise session - to confirm an isocaloric volume was achieved within each experimental group for the duration of the intervention (Table 1). Exercise progression followed ACSM standardised guidelines (Liguori 2020) for the twelve-week intervention for the experimental groups (Table 1).

**Table 1. A summary of the exercise prescription for the intervention groups.**

Week	Energy Expenditure ( $\text{kcal} \cdot \text{kg}^{-1} \cdot \text{wk}^{-1}$ )	Individualised (Target HR)	Standardised (Target HR, %HRR)
1	5.6	$HR < VT1$	40-45
2	8.4	$HR < VT1$	40-45
3	11.2	$HR < VT1$	40-45
4	11.2	$HR \geq VT1$ to $< VT2$	50-55
5	11.2	$HR \geq VT1$ to $< VT2$	55-60
6	11.2	$HR \geq VT1$ to $< VT2$	55-60
7	12.6	$HR \geq VT1$ to $< VT2$	55-60
8	14.0	$HR \geq VT1$ to $< VT2$	55-60
9	14.0	$HR \geq VT2$	60-65
10	14.0	$HR \geq VT2$	60-65
11	15.4	$HR \geq VT2$	60-65
12	15.4	$HR \geq VT2$	60-65

### Sample Size Calculation

A previous study's means and standard deviations were examined and the effect size for the current research study was calculated (Wolpern et al., 2015). Projected sample size was determined with difference in FFI as the main outcome variable. The calculated effect size was 0.30 for difference in FFI, with an assumed power of 0.80. From this it was determined that approximately 16 participants would be required for each group. An approximate 20% drop out rate was assumed, therefore 20 participants for each group were recruited.

### Data and Confidentiality

All data was de-identified and stored in a secured password protected computer at Flinders University, Sturt Campus, Bedford Park, South Australia, Australia. Only the authors, research assistants and supervising exercise physiologists had access to the data.

### Statistical Analysis

IBM SPSS version 27 package (IBM, New York, NY, USA) was used for statistical analysis. A previously

published power calculation, with an assumption of a 20% dropout rate was used to calculate the desired number of 20 for each group (Weatherwax et al., 2016). The assumption of normality of data was confirmed by Shapiro–Wilk tests (Cohen, 2013). If the assumption of normality was violated the data was log transformed, and an equivalent non-parametric test was completed. Analysis of covariance (ANCOVA) or a non-parametric equivalent (Kruskal Wallis) was used to analyse between-group differences in the change values in outcome variables, using the baseline values as a covariate and the 12-week post intervention change values as the dependent variables. A Bonferroni post hoc test was completed when appropriate to determine significant differences between intervention groups. FFI unit values are expressed as FFI values at 12 weeks minus the baseline FFI value. Continuous variables are reported as mean  $\pm$  standard deviation or median (range). A participant with a FFI value change  $\geq 1$  unit was considered a likely responder to a clinically meaningful change in FFI (Edwards et al., 2017). A *p* value  $< 0.05$  was considered significant.

## Results

A total of 48 experimental and 20 control participants were recruited for the present study. Both individualised and standardised intervention groups had 19 participants each. Participants successfully completed all pre- and post-intervention data to determine the primary outcome of the study. Only eight control participants completed all pre- and post-intervention testing (Table 2). A large proportion of the control group ( $n = 12$ ) withdrew from the study. The primary factor ( $n = 10$ ) participants stated for self-withdrawal was an initial desire to obtain free health-related measurements at baseline but no motivation to complete the intervention by increasing their physical activity levels. Table 2 provides a summary of the number of participants in each group and the rationale for exclusion. Table 3 provides the baseline data of the 46 participants who were included in the data analysis. The individualised and standardised groups had adherence rates of  $86.1 \pm 4.7\%$  and  $82.9 \pm 5.7\%$ , respectively, of the prescribed exercise training sessions.

## Fitness Fatness Index

There was a significant difference in the change in FFI between study groups, whilst controlling for baseline FFI, ( $F(2, 42) = 19.382$   $p < 0.001$ , partial  $\eta^2 = 0.48$ ) (Table 4). The magnitude of FFI increase from baseline was significantly higher in the individualised exercise group from  $16.93 \pm 5.41$  to  $19.54 \pm 6.01$  (+ 15%) compared to the standardised exercise group  $13.04 \pm 4.15$  to  $14.45 \pm 4.11$  (+ 10%;  $p = 0.028$ ) and the control group  $15.23 \pm 2.48$  to  $15.87 \pm 2.91$  (+ 4%;  $p < 0.001$ ) (Table 4).

When analysing the individual variables of FFI, METs was significantly different for the individualised group  $8.4 \pm 2.1$  to  $9.4 \pm 2.4$  (+ 12%;  $p = 0.034$ ) compared to the standardised group  $7.0 \pm 1.3$  to  $7.5 \pm 1.2$  (+ 7%) and compared to the control group ( $p < 0.001$ ) (Table 4). The standardised group was different compared to the control group ( $p = 0.014$ ) which had a decrease in METs from  $8.1 \pm 1.3$  to  $7.9 \pm 1.3$  (- 2%). Relative  $\text{VO}_2\text{max}$  ( $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) for the individualised group was significantly different, from  $29.5 \pm 7.5$  to  $32.9 \pm 8.5$   $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  (+ 11%) compared to the standardised ( $p = 0.084$ ) and control groups ( $p < 0.001$ ). Relative  $\text{VO}_2\text{max}$  ( $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ) also improved from  $24.4 \pm 4.7$  to  $26.2 \pm 4.2$   $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$  (+ 7%) in the standardised group compared to control ( $p = 0.014$ ) (Table 4). Absolute  $\text{VO}_2\text{max}$  ( $\text{L}\cdot\text{min}^{-1}$ ) also improved in the individualised group,  $2.4 \pm 0.8$  to  $2.6 \pm 0.9$  (+ 8%) compared to the control ( $p = 0.001$ ) and standardised group,  $2.0 \pm 0.6$  to  $2.2 \pm 0.6$  (+ 10%) versus control ( $p = 0.014$ ) (Table 4).

There was a significant difference in WtHR ( $p = 0.006$ ) and WC ( $p = 0.003$ ) change values following the individualised and standardised interventions compared to the control group. Following the intervention period there was also a significant difference in WC change between the exercise groups and the control group (between group difference: individualised,  $p = 0.003$ ; standardised,  $p = 0.003$ ). However, a significant difference was not observed between the intervention groups ( $p = 0.981$ ). The individualised exercise group had a decrease from  $0.88 \pm 0.12$  at baseline to  $0.85 \pm 0.11\text{m}$  (- 3%). The standardised group also experienced a decrease from  $0.94 \pm 0.16$  at baseline to  $0.91 \pm 0.15$  m (- 3%). The control group had a small decrease from  $0.87 \pm 0.11$  at baseline to  $0.86 \pm 0.11$  m (- 1%) post intervention.

**Table 2.** Total number of participants recruited and rationale for exclusion of data.

	Standardised	Individualised	Control
<b>Participants Recruited</b>	24	24	20
<b>Participants that completed the study</b>	19	19	8
<b>Exclusion of participants (rationale):</b>			
Self-withdrawal	3	-	10
Unrelated medical issues	1	2	2
$\leq 70\%$ adherence	1	3	-

**Table 3.** Participant physical characteristics. Data are means  $\pm$  SD.

Variable	Standardised (n = 19)	Individualised (n = 19)	Control (n = 8)
Age (years)	$51 \pm 13$	$45 \pm 11$	$46 \pm 8$
Male sex (%)	21	26	25
Height (cm)	$169.2 \pm 8.7$	$172.1 \pm 7.1$	$171.7 \pm 6.4$
Weight (kg)	$84.5 \pm 21.1$	$80.6 \pm 16.2$	$75.3 \pm 15.1$

**Table 4.** All participants- changes in Fitness Fatness Index and other physical and physiological characteristics following the exercise interventions.

Parameters	(n = 19)			(n = 19)			(n = 8)			group difference p-value
	Pre	Post	Within Group Cohen d	Pre	Post	Within Group Cohen d	Pre	Post	Within Group Cohen d	
<b>FFI, (METs/WtHR)</b>	13.04 ± 4.15	14.45 ± 4.11	0.34	16.93 ± 5.41	19.54 ± 6.01	0.45	15.23 ± 2.48	15.87 ± 2.91	0.23	<0.001
<b>METs</b>	6.97 ± 1.34	7.49 ± 1.20	0.41	8.42 ± 2.14	9.39 ± 2.44	0.42	8.12 ± 1.29	7.91 ± 1.32	0.16	<0.001
<b>WtHR</b>	0.56 ± 0.08	0.54 ± 0.08	0.25	0.51 ± 0.06	0.49 ± 0.05	0.36	0.51 ± 0.06	0.50 ± 0.05	0.18	0.012
<b>WC (m)</b>	0.94 ± 0.16	0.91 ± 0.15	0.19	0.88 ± 0.12	0.85 ± 0.11	0.26	0.87 ± 0.11	0.86 ± 0.11	0.09	0.005
<b>Rel VO<sub>2</sub>max (mL·kg<sup>-1</sup>·min<sup>-1</sup>)</b>	24.38 ± 4.69	26.22 ± 4.19	0.41	29.48 ± 7.49	32.85 ± 8.55	0.42	28.41 ± 4.52	27.68 ± 4.63	0.15	<0.001
<b>Abs VO<sub>2</sub>max (Lmin<sup>-1</sup>)</b>	2.05 ± 0.62	2.21 ± 0.61	0.30	2.38 ± 0.79	2.61 ± 0.85	0.28	2.16 ± 0.73	2.09 ± 0.70	0.09	<0.001
<b>PA (MET·min·wk<sup>-1</sup>)</b>	838 ± 979	3680 ± 1671	2.07	937 ± 587	3855 ± 2261	1.77	1354 ± 1018	1176 ± 1109	0.17	0.007
<b>Time sitting (hour·day<sup>-1</sup>)</b>	5.6 ± 2.7	4.5 ± 2.3	0.43	6.3 ± 2.4	5.4 ± 2.4	0.38	6.5 ± 1.2	6.9 ± 2.5	0.20	0.049

FFI, fitness fatness index; METs, metabolic equivalents; WtHR, waist to height ratio; WC, waist circumference; Rel VO<sub>2</sub>max, relative VO<sub>2</sub> maximum; Abs VO<sub>2</sub>max, absolute VO<sub>2</sub>max; PA, physical activity

### Other variables (Physical activity and Time sitting)

There was a significant between group difference in PA ( $p = 0.007$ ) in both the individualised ( $937 \pm 587$  to  $3855 \pm 2261$ ;  $+ >100\%$ ;  $p = 0.008$ ) and standardised groups ( $838 \pm 979$  to  $3680 \pm 1671$ ;  $+ >100\%$ ;  $p = 0.016$ ) compared to control ( $1354 \pm 1018$  to  $1176 \pm 1109$  MET min·wk<sup>-1</sup>;  $- 13\%$ ) (Table 4). Time sitting was significantly different only in the standardised group ( $5.6 \pm 2.7$  to  $4.5 \pm 2.3$ ) compared to control ( $6.5 \pm 1.2$  to  $6.9 \pm 2.5$  hours·day<sup>-1</sup>) pre- versus post-intervention values respectively,  $p = 0.046$ ) (Table 4).

### Discussion

To our knowledge, this is the first study to investigate a change in FFI after an individualised and standardised exercise prescription in sedentary individuals. Previous studies have found FFI a better predictor of incidence risk of T2DM, Alzheimer's disease, cancer, cardiovascular disease, and all-cause mortality, rather than cardiorespiratory fitness or fatness alone (Sloan et al., 2016; Frith and Loprinzi, 2017a; 2017b; 2017c). As such, elucidating the best method of improving FFI could potentially have significant positive health outcomes and help guide future exercise prescription recommendations. The main finding of the present study was that individualised exercise training produced a significant difference in FFI change compared to standardised exercise training ( $p = 0.028$ ) and compared with a control group ( $p < 0.001$ ). Specifically, mean FFI change was greater in individualised exercise training ( $+ 2.61$ ,  $+ 15\%$ ) compared to standardised exercise training ( $+ 1.41$ ,  $+ 10\%$ ) and a control group ( $- 0.26$ ,  $+ 4\%$ ). Traditionally, standardised exercise prescription was used to target measures of fitness and fatness independently. Whereas, when these factors were combined using FFI, the present study showed that an individualised exercise prescription was more efficacious compared to a standardised exercise prescription in improving FFI. This is an important finding as Edwards et al. (2017) suggested that for every 1-FFI unit increase there was a reduction in all-cause mortality by 9% and CVD specific mortality by 13%. It is also suggested that for every 1-FFI unit increase

those already presenting with cardiovascular conditions had a 6% reduced all-cause mortality rate, a 14% reduction in Alzheimer-specific mortality, and an 8% reduction in cancer specific mortality (Edwards et al., 2017; Frith and Loprinzi, 2017a; 2017b; 2017c). Within the components of FFI, this is also significant, as for every 1-unit MET increase, individuals with an exercise capacity  $>7$  METs reduce their mortality risk by 50-70% lower than those with poor CRF  $<5$  METs (Kokkinos, 2008; Lee et al., 2011)

Our study found a significant difference in WC, the key mediator of WtHR, in the individualised exercise group compared to the control group ( $p = 0.003$ ). However, a significant between group difference was not observed between the intervention groups ( $p = .981$ ). This suggests that the benefit of an individualised exercise prescription on FFI may lie in changes in cardiorespiratory fitness (VO<sub>2</sub>max/METs) rather than changes in WtHR (fatness). Previous studies, and the current investigation, show an individualised exercise prescription is superior to standardised and a control group in improving CRF (Weatherwax et al., 2019). This is perhaps due to a higher exercise intensity or metabolic stress applied with an individualised relative to a standardised exercise prescription approach, with exercise intensity a key mediating factor of improvements in CRF. Additionally, Frith and Loprinzi (2017a) found that FFI change was largely driven by an increase in CRF, not WtHR. This is consistent with our study which showed no difference in WtHR (fatness), but a significant difference in relative VO<sub>2</sub>max/METs (fitness) in the individualised group ( $p = < .001$ ) compared to the other groups. Thus, CRF appears to be a key mediator of FFI change following a 12-week exercise intervention. Therefore, these results suggest that interventions targeting FFI should focus more on improving CRF rather than WtHR. However, further research is needed to elucidate a superior method of improving WtHR, to have the greatest impact on changes in FFI.

The use of an individualised ventilatory threshold-based model to prescribe exercise was shown to be

significantly better compared to standardised prescription at improving CRF (Katzmarzyk et al., 2004; Wolpern et al., 2015; Dalleck et al., 2016). However, current guidelines do not prescribe exercise using a ventilatory threshold method (Liguori 2020). It is important to understand the mechanisms behind ventilatory threshold prescription, as inaccurate prescription above VT2 could lead to an adverse event (Hansen et al., 2019), while it is also suggested that exercise prescription at or above VT1 leads to optimal improvements in CRF (Guio de Prada et al., 2019). During light exercise intensity (<VT1), fats are a major fuel and only small amounts of lactate are produced and cleared (Deacon, 2020). Exercise at or above VT1 represents an exercise intensity where blood lactate begins to accumulate faster than it can be cleared, leading to an increased breathing rate as oxygen demands increase faster than oxygen delivery to the working muscles (Wolpern et al., 2015). The increased breathing rate also expires additional CO<sub>2</sub>, a by-product of buffering acid metabolites. The second increase in ventilation (VT2) occurs where lactate increases exponentially, hyperventilation is observed, together with a considerable reduction in ability to communicate. The expiration of CO<sub>2</sub> is no longer adequate at buffering the acid metabolites and exercise at this intensity can usually only last for up to two minutes in duration (Kunutsor et al., 2017). The ranges of VT1-VT2 are important to note for accurate cardiorespiratory exercise prescription and for determining individualised exercise intensities. In comparison, heart rate reserve is defined as the difference between basal HR and maximal HR (Mezzani et al., 2013) and is currently used as a standardised exercise prescription (Liguori 2020). Standardised ranges for cardiorespiratory exercise intensity prescribed at 40%-59% HRR have been suggested to improve and maintain CRF (Liguori 2020), however 60% HRR has been suggested to correspond to VT1 (Mezzani et al., 2013), with optimal improvements in CRF being prescribed at an exercise intensity at or above VT1 (Guio de Prada et al., 2019). The “range based” model of %HRR prescription has been found to be less accurate at both prescribing the correct exercise intensity and it fails to consider the individual blood lactate response to exercise intensity (Wolpern et al., 2015). It has been suggested that the variation in metabolic response using the %HRR method leads to ‘responders’ and ‘non-responders’ (Weatherwax et al., 2019). The mechanisms of individualised ventilatory threshold prescription compared to %HRR highlight the importance of why accurate prescription is important, not only for improving CRF but also for the individual’s safety (Anselmi et al., 2021).

### Limitations

The main limitation of this study is the convenience sample recruited for the control group. This group was recruited after the two intervention groups, and therefore was not a part of the randomisation process. However, all control group participants had to meet the same inclusion/exclusion criteria to minimise this limitation. Another limitation in the present study is the use of METs as the maximal CRF metric. The formula used to derive METs from VO<sub>2</sub>max (VO<sub>2</sub>max/3.5 mL·kg<sup>-1</sup>·min<sup>-1</sup>) is the

assumed metabolic rate, thus a true individualised resting metabolic rate was not calculated. Thus, to truly explore the importance of individualised exercise prescription an individualised approach to quantifying METs is required. Future research should investigate individual metabolic changes (METs) compared to changes in WtHR. Furthermore, as the principal investigator was aware of which treatment group participants were allocated to, a potential limitation is the possibility of assessor bias. However, this bias was minimised through the use of a verification protocol to confirm a true VO<sub>2</sub>max and high adherence to the set exercise prescriptions. Additionally, as the cohort of this study were relatively healthy sedentary individuals, although some had at least one cardiometabolic risk at baseline, these findings cannot be generalised to clinical populations. Further research on clinical populations is required to identify the efficacy of individualised versus standardised exercise prescription on FFI in this cohort. It is also noteworthy that males were under-represented in the study, only accounting for 23% of the study cohort, and due to the large age ranges, there may be heterogeneity in the results. Lastly, it is likely that ‘cardiovascular drift’ during the exercise sessions may have occurred in this study. Cardiovascular drift reflects a slow increase in HR over time during exercises lasting more than 10 min (Teso et al., 2022). Therefore, the target HR range used to provide a constant work rate or intensity during the training sessions could have led to a reduction in work rate/metabolic intensity, which may have, in turn influenced our overall results.

### Conclusion

The main finding of the present study was that individualised exercise training induced a significant difference in FFI change compared to standardised exercise training and a control group. This is an important finding as it has been reported that a favourable change in FFI is associated with a reduction in all-cause mortality, cancer, Alzheimer’s, and cardiovascular disease specific mortality. Whilst the exact mechanisms are yet to be fully understood, the use of ventilatory thresholds considers individual metabolic differences. Therefore, greater improvements in CRF, a key mediator of FFI change, can be achieved using this method. An individualised exercise prescription should be considered a viable and practical method of improving FFI in sedentary adults.

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The experiments complied with the current laws of the country in which they were performed. The authors have no conflicts of interest to declare. The datasets generated and analyzed during the current study are not publicly available, but are available from the corresponding author who was an organizer of the study.

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### Key points

- Cardiorespiratory exercise prescribed individually using ventilatory thresholds had a greater effect on improving Fitness-Fatness Index in sedentary adults compared to a comparable protocol using heart rate reserve.
- Individualized exercise prescription using threshold metrics should be considered a viable method for improving the Fitness-Fatness Index to help aid in mitigating the future progression of non-communicable disease.
- Greater improvements in the Fitness-Fatness Index were supported by positive improvements in cardiorespiratory fitness

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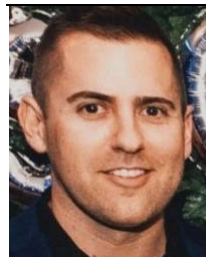
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