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Effects of Short-Term High versus Continuous Moderate Intensity Training on Insulin Resistance in Overweight and Obese Adults: A Randomized Controlled Trial

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ABSTRACT

International Journal of Exercise Science 12(3): 1057-1069, 2019. In long-term exercise training studies (> 6 weeks), improvements in insulin resistance (IR) are amplified by decreased body fat and/or increased cardio-respiratory fitness. This presents a challenge in studying the independent effects of exercise training. Our study purpose was to determine the effects of short-term continuous moderate intensity training (CMIT) and high intensity interval training (HIIT) on IR in overweight/obese adults. Participants were stratified into insulin sensitive (IS) and IR groups, and randomized into non-exercise control (CNT), CMIT and HIIT sub-groups that underwent baseline and post testing. Exercise sessions were 18-24 minutes for 10 consecutive days. The CMIT sub-group continuously cycled at 60-70% of peak oxygen consumption ($V\dot{O}_2$peak) while the HIIT sub-group performed 60s of cycling at 90-100% $V\dot{O}_2$peak interspersed with 30 seconds of rest. Ninety-five participants (mean age and BMI 23.9 $\pm$ 3.9 years and 32.1 $\pm$ 5.0 kg/m^2) were enrolled into the study. Of these, 63% were IS and 37% had IR. CMIT or HIIT did not result in statistically significant improvements in IR. However, the reduction (32.4%) in IR observed with HIIT may be of clinical relevance. Cohen's (*d*) effect size (ES) for HIIT on IR was large (ES: *d* = -0.9; 95% CI: -1.7, -0.1) while that of CMIT was unclear (ES: *d* = -0.2; 95% CI: -1.0, 0.6). In the current study, CMIT or HIIT did not result in statistically significant improvements in insulin resistance. Future large-scale studies to clarify and confirm our findings are warranted.

KEY WORDS: Obesity, chronic disease, metabolic disorder, physical inactivity

INTRODUCTION

Insulin resistance commonly arises from a chronic energy surplus associated with overweight and obesity [1]. Individuals who are overweight/obese are at increased risk of cardiometabolic disorders associated with insulin resistance including dyslipidaemia, type 2 diabetes and cardiovascular diseases (CVDs) [2]. Current strategies for the managements of insulin resistance and associated disorders include lifestyle modification (diet and exercise) and pharmacological agents such as insulin, metformin and statins [3-5]. Pharmacological agents however, are often associated with undesirable side effects such as diarrhea, abnormal dreams and myopathy [6].

Exercise and dietary interventions such as high-fat-low-carbohydrate (HFLC) diet, high protein diet and the Mediterranean diet have been reported to result in superior therapeutic effects compared with pharmacological agents [7, 8]. Challenges with dietary interventions, however, include high cost and weak sustainability [9-11]. Conversely, exercise offers an efficient and cost-effective alternative for the management of chronic diseases including insulin resistance [12]. A large body of evidence indicates that exercise training results in improved insulin sensitivity, and consequently, the concentration of insulin required to bring about 50% of its maximal effect on glucose transport is lower [13, 14]. As a result, a maximal insulin stimulus produces a larger increase in glucose transport. Exercise, therefore, improves impaired glucose homeostasis associated with insulin resistance by regulating insulin action.

Metabolic effects of exercise training are primarily dependent on the intensity and duration of the intervention [15, 16]. Chronic training studies have shown that high intensity interval training (HIIT) characterized by brief bouts of all-out effort interspersed with recovery, leads to similar improvements in metabolic outcomes when compared with traditional continuous moderate intensity training (CMIT) currently prescribed for individuals with metabolic disorders [17, 18]. Asymptomatic individuals with insulin resistance or type 2 diabetes are encouraged to achieve a minimum 30 minutes of moderate intensity (less than 60% of heart rate reserve) exercise per day, on at least 5 days per week [19]. Conversely, HIIT requires only 10-20% of the 150 minutes required for CMIT, with the same or possibly better therapeutic effects [20, 21]. However, with long-term training, effects of exercise on insulin action are influenced by decreases in body fat and/or improvements in cardiorespiratory fitness, factors which have been found to independently improve insulin resistance [22, 23]. As a result, short-term interventions (7-10 days) are increasingly being studied to better understand independent effects of exercise on insulin resistance. Despite being less understood, short-term HIIT has been reported to improve metabolic health in insulin resistant and diabetic patients [24, 25]. In these studies, however, the cohorts are often of a single gender, with no distinction made between individuals with and without insulin resistance, and no comparison to a non-exercise control and CMIT group [26]. The aim of the current randomized controlled trial, therefore, was to assess short-term effect of CMIT and HIIT on markers of beta cell function, insulin sensitivity and insulin resistance in physically inactive overweight/obese adults.

METHODS

Participants

Study participants were male and female adults aged between 18 and 35 years who were physically inactive (less than 30 minutes of physical activity five days a week), overweight/obese (BMI ≥ 25 kg/m2), non-smokers and did not use medications that are known to affect body composition and metabolism.

Staff and Students of the University of KwaZulu-Natal were invited to volunteer to take part in the study through posters and social media outlets. Consenting individuals provided written consent for participation and publication of the results. The study was conducted between August 2016 and October 2017 at the Human Performance Laboratory (HPL) of the University of KwaZulu-Natal, Durban, South Africa. A target sample size of 150 was calculated using an online sample size calculator (Raosoft®) with the confidence level and confidence interval set at 95% and 5% respectively. Recruitment of study participants was stopped at 95 (63% of target sample size) due to financial constraints. The study protocol of this was approved by the University of KwaZulu-Natal Biomedical Research Ethics Committee (reference: BFC098/16).

Protocol

This was a 10-day, multiple-arm randomized controlled trial designed to determine the effects of short-term CMIT and HIIT on insulin sensitivity (HOMA-IS), beta cell function (HOMA-β) and insulin resistance (HOMA-IR) in physically inactive, overweight/obese adults. Enrolled participants were screened for insulin resistance and categorized as insulin sensitive (IS; HOMA-IR > 1.1 - < 2) or insulin resistant (IR; HOMA-IR > 2) [34]. Using a pseudo-random number generator software (Research Randomizer version 4.0), participants in the IS and IR groups were assigned to a control group (CNT), continuous moderate intensity training (CMIT) or high intensity interval training (HIIT) sub-group. Participants in the CNT sub-group underwent baseline and follow-up evaluation without taking part in the intervention. The CMIT and HIIT sub-groups underwent baseline assessments, ten consecutive days of either CMIT or HIIT, and had follow-up evaluation after the exercise intervention period. Primary outcome measures included body mass index [BMI (kg/m^2)], percentage body fat (%BF), HOMA-IS, HOMA- β, HOMA-IR and peak oxygen consumption ($\dot{V}O_2$peak).

Baseline measurements included exercise pre-participation questionnaire, clinical examination, laboratory and physiological tests. A repeat of baseline measures was conducted at follow-up which was undertaken at least 24 hours after the last exercise bout, within a 48-hour period. Post-training blood draws were conducted at least 24 hours before $\dot{V}O_2$peak test.

The exercise pre-participation health screening questionnaire focused on the participant's current level of physical activity as well as screening for signs or symptoms of cardio-metabolic disease; physical activity screening questionnaire (PASQ) and pre-exercise intervention questionnaire (PEIQ). The PASQ (themes; activity at work/school, travel to and from places, recreational activities and sedentary behaviour) was adapted from the World Health Organization (WHO) global physical activity questionnaire (GPAQ). The PEIQ (themes; history

of cardiovascular events, symptoms, other health issues and cardiovascular risk factors i.e. age, blood pressure and cholesterol) was adapted the American Council for Sport Medicine (ACSM) pre-participating questionnaire.

Height (cm) was measured on a stadiometer without shoes. Waist circumference [WC (cm)] was measured at the approximate midpoint between the lower margin of the last palpable rib and the top of the iliac crest [27]. Hip circumference [HC (cm)] was measured around the widest part of the buttocks [27]. Height, WC and HC were all measured to the nearest centimeter. Waist and hip circumference were used to calculate waist-hip ratio (WHR). An electronic body composition monitor (Omron® BF511 Body Composition Scale) was used to measure body weight [BW (kg)], BMI, %BF, muscle mass percentage [MM (%)] and visceral fat (VF).

Laboratory analyses were conducted at the Lancet Pathology Laboratory (Durban, South Africa). Following a 12-hour overnight fast by participants, venous blood samples were drawn from the ante cubital region of the arm, collected into plain tubes and NaF tubes for the analysis of insulin and glucose, respectively. The samples were stored at -80 °C until further biochemical analysis. Fasting serum insulin was measured by radioimmunoassay [28]. Fasting plasma glucose was measured using a glucose hexokinase method on a Roche automated analyzer [29, 30]. The fasting insulin and glucose were used to calculate HOMA-%β, HOMA%S and HOMA-IR (HOMA2 Calculator© The University of Oxford 2013) [31].

Physiological parameters, $\dot{V}O_2$peak and power output (PO) were assessed by a 2-minute continuous incremental cycling test performed on an electronically braked ergometer (Lode Excalibur Sport, Groningen, The Netherlands) [32, 33]. After participants pedalled at 50 watts for 3 minutes (warm-up), the workload was increased by 25 watts every 2 minutes until volitional fatigue. Blood pressure (mmHg) (Omron® MIT Elite Plus Blood Pressure Monitor) and heart rate (bpm) (Polar S810TM HRM) were measured at rest (before the exercise test), every 2 minutes during the $\dot{V}O_2$peak test and post exercise testing until the values returned toward resting measures. Oxygen consumption ($\dot{V}O_2$) was analyzed by an online breath by breath gas collection system (Cortex MetaMax 3b gas analyser). The highest $\dot{V}O_2$ achieved during the last stage was recorded as the $\dot{V}O_2$peak [34]. Studies have reported that acute exercise can influence insulin up to 72 hours post-exercise [35]. Thus, to minimize the effects of the $\dot{V}O_2$peak test on insulin, cardiorespiratory fitness testing was conducted no less than 4 days before blood samples were collected [35]. Study participants were asked to refrain from exercise before blood samples were collected for insulin assay.

The HIIT exercise protocol was designed based on findings from previous studies which indicated that during HIIT, a 2:1 work to rest ratio yields appropriate training stimulus and is perceived as less difficult by participants [36]. The CMIT group was matched for time. Exercise sessions were performed on an electronically braked cycle ergometer (Lode Excalibur Sport, Groningen, The Netherlands) for 10 consecutive days, with at least 24 hours in-between sessions. Study participants were afforded 3 minutes of warm-up and 3 minutes of cool-down on the ergometer at 50W and 60rpm. During active exercise, participants were required to maintain a workload that corresponded with a wattage that solicited 60-70% $\dot{V}O_2$peak for the CMIT sub-

group and 90-100% of $V\dot{O}_2$peak for the HIIT. The workloads were determined from pre-intervention $V\dot{O}_2$peak assessments.

The total time for exercise sessions started at 18 minutes and progressed to 24 minutes over the intervention period. In the current study, we were exploring one of the numerous work-to-rest ratios of HIIT. In studies on trained athletes, HIIT sessions are generally 15-30 minutes. However, little is known about the optimal tolerable combination of intensity and volume necessary for adaptations in clinical populations. The gradual progression in time and intensity was in accordance with *American College of Sports Medicine Guidelines for Exercise testing and Prescription* [37]. Over the 10 days of exercise intervention, the HIIT group spent 33.3% less time in active exercise than the CMIT group, 96 minutes versus 144 minutes of CMIT. Exercise sessions were performed under standard laboratory conditions. The primary investigator documented attendance and ensured that sessions were performed as prescribed. Post-training assessments (repeat of baseline measures) were conducted at least 24 hours after the last exercise bout, within a 48-hour period. A summary of procedures conducted at baseline, during the intervention and post intervention is shown in Table 1, below.

Table 1. Procedures conducted at baseline, during the intervention and post intervention

Baseline	Physical activity questionnaire	Conducted only at baseline		
	Clinical examination	Conducted between 07:00-09:00 AM		
	Venipuncture			
	Aerobic fitness test	Conducted 24 hrs after venipuncture		
Intervention	Days	1-4	5-8	9-10
	Warm-up at 50W and 60rpm for 3 minutes			
	CMIT sessions	12 min cycling with no rest 1 set	15 min cycling with no rest 1 set	18 min cycling with no rest 1 set
	HIIT sessions	60 sec cycling/30 sec rest 8 sets	60 sec cycling/30 sec rest 10 sets	60 sec cycling/30 sec of rest 12 sets
	Cool-down at 50W and 60rpm for 3 minutes			
Post measures	Clinical examination	Conducted between 07:00-09:00 AM, 24 hrs after the last exercise bout		
	Venipuncture			
	Aerobic fitness test	Conducted 24 hrs after venipuncture within 48 hrs of the last exercise bout		

Note: CMIT; continuous moderate intensity training, HIIT; high intensity interval training, RPM; revolutions per minute, Hrs; hours

Statistical Analysis

Data was analyzed using SPSS version 24 (IBM, USA). Values are expressed as means and standard deviations (SD). Descriptive statistics were conducted to examine the general characteristics of study participants such as age, BMI, HOMA-IR and $V\dot{O}_2$peak. The independent t-test was applied to compare the means at baseline between the sub-groups (CNT, CMIT and HIIT) to ensure no significant differences at the baseline. The comparison was also made between insulin sensitive and insulin resistant participants in the sub-groups. A repeated measure analysis of variable (ANOVA) was used to examine the interaction effect (sub-group × time) for all variables. Where appropriate, pairwise multiple comparison were performed using the Bonferroni post-hoc test. The significance level was set at $p < 0.05$. Percentage changes from

baseline to follow-up were calculated for all outcome variables. To determine the magnitude of the changes from baseline to follow-up, Cohen's (*d*) effect sizes 95% confidence intervals (CI) were calculated. Magnitudes of the standardized effects were interpreted using thresholds of $d \leq 0.2$ (small), $d \geq 0.5$ (moderate) and $d \geq 0.8$ (large) [38]. An effect size was only accepted if the confidence interval did not cross both the positive and negative 0.2 ES thresholds otherwise were considered to be unclear [38].

RESULTS

A total of 95 participants were enrolled into the study. Of these, 38 (40%) were male and 57 (60%) were female. The mean age and BMI were 23 ± 3.9 years and 32.1 ± 5.0 kg/m^2, respectively. Figure 1 outlines the consort diagram. Of the total study group (n = 95), 60 participants (63%) were categorized as insulin sensitive (IS) and 35 (37%) as insulin resistant (IR). When the IS and IR groups were randomised into sub-groups, the CNT and HIIT sub-groups each included 31 participants: IS (n = 20) and IR (n = 11); the CMIT sub-group had 33 participants: IS (n = 20) and IR (n = 13).

Sixty participants were included in the pre-versus post-intervention analysis; of these, 31 were from the insulin sensitive group [CNT (n = 9); HIIT (n = 11); CMIT (n = 11)] and 29 from the insulin resistant group [CNT (n = 5); HIIT (n = 11); CMIT (n = 13)].

Thirty-five participants were lost to follow-up, of whom 29 were from the insulin sensitive group [CNT (n = 10); HIIT (n = 8); CMIT (n = 11)], and 6 were from the insulin resistant CNT group (n = 6). Reasons for discontinuing the study were, relocation (to study at other institutions or for employment) and busy schedule. There was no statistically significant difference in baseline characteristics between participants who completed the study and those who did not.

Table 2 shows baseline characteristics of the sub-sample that was followed-up (n = 60). When compared with IS participants, those with IR had significantly higher HOMA-IR in all sub-groups; CNT (p = 0.00), CMIT (p = 0.01) and HIIT (p = 0.02). IR participants had significantly lower aerobic fitness (p = 0.01) compared with IS participants in the CNT sub-group.

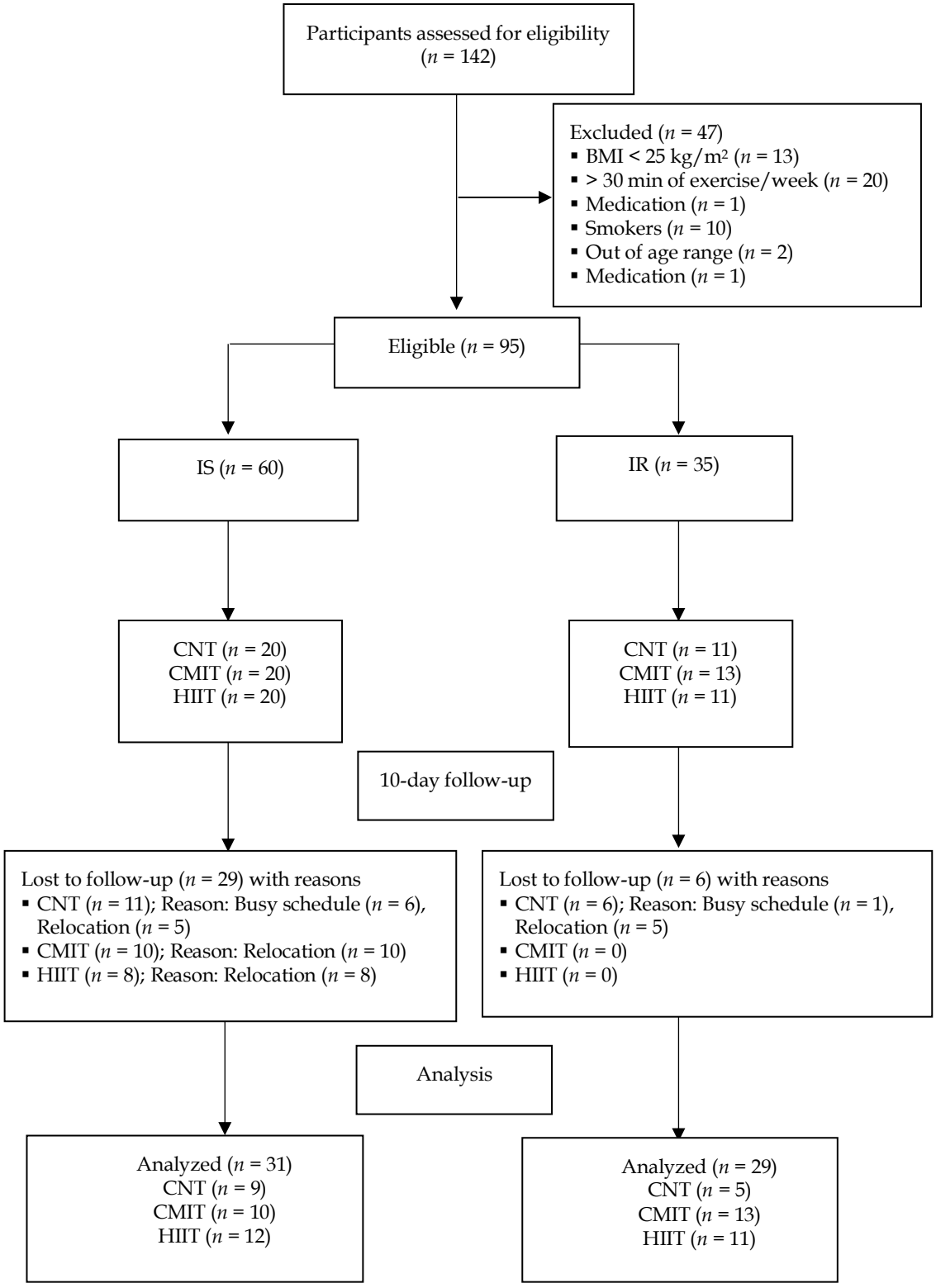

Figure 1. Consort diagram. IS-insulin sensitive; IR-insulin resistant; CNT-control; CMIT-continuous moderate intensity training; HIIT-high intensity interval training; BMI-body mass index.

Table 2. Baseline characteristics of the sub-sample that was followed-up (*n*:60) by training intensity.

Characteristics	CNT (*n* = 14)		CMIT (*n* = 23)		HIIT (*n* = 23)	
	IS mean (SD)	IR mean (SD)	IS mean (SD)	IR mean (SD)	IS mean (SD)	IR mean (SD)
n (M; F)	9 (4; 5)	5 (2; 3)	12 (6; 6)	11 (5; 6)	10 (4; 6)	13 (7; 6)
BMI (kg/m²)	31.6 (4.0)	36.4 (4.4)	30.1 (6.7)	35.3 (4.6)	30.3 (4.2)	32.7 (3.4)
BF (%)	40.2 (7.6)	48.7 (8.0)	39.3 (11.1)	45.1 (8.5)	37.9 (6.9)	43.7 (8.7)
FBG (mmol/L)	4.6 (0.3)	4.8 (0.4)	4.8 (0.5)	4.9 (0.3)	4.7 (0.2)	5.0 (0.4)
HOMA-%ß	118.6 (29.2)	161.7 (27.6)	90.4 (20.0)	151.5 (30.2)	104.0 (12.2)	151.8 (35.4)
HOMA-%S	105.6 (41.8)	54.0 (12.7)	138.3 (50.6)	57.6 (12.4)	106.2 (14.2)	57.2 (18.4)
HOMA-IR	1.4 (0.4)	2.6 (0.4)*	1.2 (0.3)	3.1 (0.7)*	1.5 (0.3)	3.4 (1.3)*
$\dot{V}O_2$peak (ml/min/kg)	26.4 (7.2)	18.8 (1.8)*	26.2 (5.6)	23.0 (4.0)	24.1 (4.5)	22.1 (2.9)

Note: Data are expressed as mean (SD). CNT: control; CMIT: continuous moderate intensity training; HIIT: high intensity interval training; IS: insulin sensitive; IR: insulin resistant; BMI: body mass index; BF: body fat; FBG: fasting blood glucose; HOMA-%ß: homeostasis model of assessment for beta-cell function; HOMA: %S: Homeostasis Model of Assessment for insulin sensitivity; HOMA-IR: homeostatic model of assessment for insulin resistance; $\dot{V}O_2$peak: peak oxygen consumption. *$p < 0.05$: IS vs IR. Fasting glucose reference range: 3.3-6.0 mmol/L.

Table 3 shows percentage changes in the insulin resistance (IR) group at follow-up (*n* = 24). High intensity interval training increased HOMA-%S by 33.3% vs. 9.33% in the continuous moderate intensity training (CMIT) sub-group. Insulin resistance (HOMA-IR) was reduced by 32.4% in the HIIT sub-group vs 6.45% in the CMIT sub-group.

Cohen's effect sizes (ES) showed that the effects of CMIT on HOMA-%S and HOMA-IR were unclear in the insulin resistant group while HIIT produced a large (ES: *d* = 0.9; 95% CI: 0.04.7, 1.8) increase in HOMA-%S and large (ES: *d* = -0.9; 95% CI: -1.7, -0.1) decrease in HOMA-IR.

Table 3. Changes in characteristics of the insulin resistant (IR) group from baseline to follow-up

Characteristics	Sub-group	Baseline	Follow-up	% change
BMI (kg/m^2)	CNT	36.4 (4.4)	36.2 (4.4)	-0.55
	CMIT	35.3 (4.6)	35.8 (4.5)	1.42
	HIIT	32.7 (3.4)	32.6 (3.4)	-1.21
BF (%)	CNT	48.7 (8.0)	49.2 (7.7)	1.03
	CMIT	45.1 (8.5)	45.7 (8.5)	1.33
	HIIT	43.7 (8.7)	44.0 (8.6)	0.69
FBG (mmol/L)	CNT	4.8 (0.4)	4.9 (0.4)	2.08
	CMIT	4.9 (0.3)	4.9 (0.3)	0.00
	HIIT	5.0 (0.4)	4.8 (0.3)	-4.00
HOMA-%ß	CNT	161.7 (27.6)	166.5 (56.4)	2.97
	CMIT	151.5 (30.2)	143.9 (28.7)	-5.02
	HIIT	151.8 (35.4)	130.5 (24.7)	-14.0
HOMA-%S	CNT	54.0 (12.7)	51.8 (14.9)	-4.07
	CMIT	57.6 (12.4)	63.1 (18.5)	9.93
	HIIT	57.2 (18.4)	76.4 (23.1)	33.3
HOMA-IR	CNT	2.6 (0.4)	3.2 (1.2)	23.1
	CMIT	3.1 (0.7)	2.9 (0.9)	-6.45
	HIIT	3.4 (1.3)	2.3 (1.2)	-32.4
$\dot{V}O_2$peak (ml/min/kg)	CNT	18.8 (1.8)	19.3 (2.5)	2.66
	CMIT	23.0 (4.0)	23.2 (3.9)	0.87
	HIIT	22.1 (2.9)	21.5 (4.3)	-2.71

Note: Data are expressed as mean (SD). CNT: control; CMIT: continuous moderate intensity training; HIIT: high intensity interval training; IS: insulin sensitive; IR: insulin resistant; BMI: body mass index; BF: body fat; FBG: fasting blood glucose; HOMA-%ß: homeostasis model of assessment for beta-cell function; HOMA: %S: Homeostasis Model of Assessment for insulin sensitivity; HOMA-IR: homeostatic model of assessment for insulin resistance; $\dot{V}O_2$peak: peak oxygen consumption.

DISCUSSION

This study on physically inactive overweight and obese adults has shown that CMIT or HIIT for 10 consecutive days does not result in statistically significant improvements in beta cell function, insulin sensitivity and insulin resistance. Cohen's (*d*) effects sizes showed that in insulin resistant individuals, HIIT leads to a large (33.3%) increase and a large (32.4%) decrease in insulin sensitivity and insulin resistance, respectively. Thus, although not statistically significant, the effects of HIIT on insulin sensitivity and insulin resistance may be of clinical relevance.

The absence of statistically significant changes (based on *p*-value statistics) in cardiometabolic parameters may be due to small sample size and may not directly indicate that an effect does not exist [39, 40]. Cohen's effect sizes indicated that HIIT produced a large (d = 0.9) increase in HOMA%S and a large (d = -0.9) decrease in HOMA-IR while those of CMIT were unclear. Previous studies have indeed reported that when compared with CMIT, HIIT results in similar

improvements in insulin sensitivity and insulin resistance even with the significantly reduced time spent exercising for the HIIT groups [41-43]. In those studies, however, there was concomitant improvement in cardiovascular fitness and decreases in body fat following HIIT. Improved cardiovascular fitness and decreased body fat have been reported to independently improve insulin sensitivity. Consequently, this presents a challenge in drawing conclusions on whether the improvements were from the exercise *per se*. In the present study, the positive effects of HIIT on insulin sensitivity and insulin resistance occurred in the absence of changes in body composition and cardiorespiratory fitness, suggesting that the observed improvements may be related to HIIT training. Possible mechanism for HIIT-induced metabolic adaptations include enhanced insulin signaling in skeletal muscle via increased insulin-stimulated phosphorylation of AS160 which regulates GLUT4 translocation [44]. Our findings however, are to be interpreted cautiously as other studies have found no clear advantage in the improvement of insulin sensitivity in short-term studies [45].

The strengths of this study include the randomized controlled design, categorization of participants into insulin sensitive and insulin resistant groups and use of Cohens (*d*) effect sizes to quantify the effects of the exercise intervention. Our study findings, however, need to be interpreted with caution. Limitations included high attrition rate, small sample and uncontrolled diet. Relocation to areas outside the study site and frequent visits to the laboratory may have contributed to the high attrition rate. The small sample size (63% of the estimated sample) which decreased the statistical power of the study. Participants were asked to maintain their regular diet, some individuals however, may have been encouraged to make healthier dietary choices upon enrollment into the study, this may have influenced the results.

Future large-scale studies with controlled diet and a broader representation of the South African population are necessary to enable a much wider interpretation and application of the findings. Furthermore, the current study explored one of the numerous possible combinations of work to rest ratio during HIIT. The ideal HIIT exercise prescription for individuals with insulin resistance/diabetes remains to be elucidated. Studies to establish the ideal combination of work to rest ratio, frequency and duration during HIIT training are needed.

In conclusion, the present study revealed that CMIT or HIIT for 10 consecutive days does not result in statistically significant improvements in beta cell function, insulin sensitivity and insulin resistance. However, future large-scale studies to clarify and confirm the effects of CMIT and HIIT in physically inactive overweight/obese adults.

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