

The Effects of High-Intensity Interval Training versus Moderate-Intensity Continuous Training on Maximal Oxygen Uptake in Sedentary Adults

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Abstract

The purpose of the investigation was to compare the effects of 8 weeks of low-volume high-intensity interval training (HIIT) versus the effects of high-volume moderate-intensity continuous training (MICT) on maximum oxygen uptake (VO_{2max}) in sedentary adults. We hypothesized that increases in VO_{2max} after both interventions would not significantly differ between groups. 24 participants (27.1 ± 8.4 yr, $VO_{2max} = 27.7 \pm 6.7$ ml*kg⁻¹*min⁻¹) completed 4 wk (3 sessions/wk) of MICT (30 min, 70-75% HR_{max}). After the 4 wk conditioning period, 13 participants (28 ± 9.7 yr) were randomized into 8 wk (3 session/wk) of HIIT (10 X 1 min, 90-95% HR_{max}, 1-min active recovery) & 11 participants (26 ± 6.9 yr) were randomized into 8 wk (5 sessions/wk) of MICT (30 min, 70-75% HR_{max}). VO_{2max} increased significantly in all participants after the interventions (Baseline = 2.01 ± 0.47 L*min⁻¹, Week 12 = 2.30 ± 0.41 L*min⁻¹, $P < 0.001$). VO_{2max} increased significantly in the HIIT group from week 4 to week 12 (Week 4 = 2.08 ± 0.21 L*min⁻¹, Week 12 = 2.29 ± 0.19 L*min⁻¹, $P < 0.001$). VO_{2max} also increased significantly in the MICT group from week 4 to week 12 (Week 4 = 2.18 ± 0.63 L*min⁻¹, Week 12 = 2.30 ± 0.58 L*min⁻¹, $P = 0.010$). There was no group X time interaction for changes in VO_{2max} from week 4 to week 12 ($P = 0.755$). Therefore, 8 weeks of low-volume HIIT (480 total min) & 8 weeks of high-volume MICT (1200 total min) led to increases in VO_{2max} that did not differ significantly in 24 sedentary adults.

Key Words: Maximal oxygen uptake, high-intensity interval training, moderate-intensity continuous training, cardiorespiratory fitness

Definitions & Uses of VO_{2max}

Maximum oxygen uptake (VO_{2max}) is defined as the maximal amount of oxygen that can be consumed by the body during maximal exercise. VO_{2max} assesses the body's ability via the pulmonary, cardiovascular, & skeletal muscle systems to uptake, transport, & utilize O_2 in contracting muscle mitochondria at maximal exercise (24). Hence, measuring VO_{2max} is the gold standard for assessing cardiorespiratory fitness (CRF) in individuals. Understanding CRF is essential in clinical practice; low CRF levels are associated with an increased risk of cardiovascular disease (CVD) and all-cause mortality (26). This higher risk has been observed in healthy men & women, as well as those with CVD & comorbid conditions such as hypertension, type II diabetes mellitus (T2DM), obesity, & lipid abnormalities (26). Additionally, improvements in CRF are associated with an increased rate of survival; a 1-MET ($3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) increase in VO_{2max} has been associated with a 13% reduction in risk of all-cause mortality & a 15% reduction in risk of CVD (18). Furthermore, VO_{2max} is used in the development of exercise prescriptions for athletes, patients, & clients. Exercise programs may utilize percentages of VO_{2max} to determine intensity of exercise sessions. In research settings, changes in VO_{2max} can be used to identify a training effect—a change in CRF levels—after an exercise intervention or detraining period (4, 24).

Measuring VO_{2max}

VO_{2max} can be measured directly via a maximal graded exercise test (GXT) or indirectly estimated via a submaximal GXT, aerobic field tests, or non-exercise algorithms. A GXT allows for the observation of the relationship between exercise workload & integrated physiological systems of the human body (6). To measure VO_{2max} using a maximal GXT, typical modes of

exercise include walking, jogging, or running with incline, as well as cycling & rowing. Thus, a cycle ergometer, treadmill, or rowing machine is required to complete the GXT. O₂ & CO₂ gas analyzers & a metabolic cart are needed for instantaneous respiratory gas flow measurements. A heart rate (HR) monitor is also required. Using these technologies, ventilation, VO₂, VCO₂, HR, and the respiratory exchange ratio (RER) are measured during the GXT to monitor the test participant. The RER is an estimate of the respiratory quotient (RQ), which is the ratio of V_{CO2}/V_{O2} or the ratio of CO₂ produced to O₂ consumed.

A maximal GXT protocol consists of a ramp or incremental test. A ramp test is a form of GXT that utilizes a linear increase in work rate via increased resistance over time until volitional exhaustion. On the other hand, an incremental test is a form of GXT that utilizes stages of equal duration that involve discontinuous increases in work rate until volitional exhaustion. Resistance can be altered by changing the wattage on a cycle ergometer or the speed & incline on a treadmill. As a result, cycle ergometry allows for a more quantifiable work rate in Watts & is typically used for a ramp protocol, while the treadmill is generally reserved for incremental GXTs (6). Total test duration varies based on protocol and may last between 8 & 12 minutes, as determined by Buchfuhrer et al. (7). The researchers suggest the ideal time to bring a participant to his or her tolerance limit is 10 minutes.

Various criteria are utilized to determine if a participant has achieved VO_{2max}. The primary criterion is the achievement of a VO₂ plateau; the plateau is represented by an increase in work rate without a further increase in oxygen consumption during the GXT. However, only about 50% of participants reach a VO₂ plateau (4). The low incidence of plateau attainment may be attributed to age, testing modality, gas sampling rate, & data analyses methodology (6). Consequently, secondary criteria are necessary. The criteria include achieving a HR that is ± 10

bpm of the age-predicted HR_{max} ($220 - \text{age}$), a rate of perceived exertion (RPE) ≥ 18 on Borg's Scale, an RER ≥ 1.10 , & blood lactate accumulation ≥ 8 mM (6, 24). A major limitation of secondary criteria is that meeting the criteria does not guarantee the attainment of a VO_2 plateau; thus, another method is required.

Although secondary criteria are utilized commonly to determine if a VO_2 plateau is achieved, the verification phase (VP) is a superior method to ensure plateau attainment. The VP, a subsequent validation test after the initial GXT, can be used to verify if the VO_{2max} is a true maximal value. The VP generally consists of exercising at or near the maximal achieved work rate from the GXT without progression following a period of active rest of 10-20 min (24). During a VP, the participant works at 90-115% of the maximal achieved work rate until volitional exhaustion (6). The total duration of the VP generally lasts between 3-6 min (24).

Changing VO_{2max} : Fick Equation

VO_2 is defined via the Fick Equation, which states that VO_2 ($\text{ml} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) is equal to the product of cardiac output [CO ($\text{L} \cdot \text{min}^{-1}$)] & the arteriovenous difference in oxygen concentration [$a-vO_{2diff}$ ($\text{mL} \cdot \text{L}^{-1}$)] (6):

$$1. \quad VO_2 = CO \times a-vO_{2diff}$$

The equation can be expanded by representing CO as the product of HR ($\text{beats} \cdot \text{min}^{-1}$) and stroke volume [SV ($\text{mL} \cdot \text{b}^{-1}$)], which is calculated as the difference between left ventricular (LV) end-diastolic volume [EDV ($\text{mL} \cdot \text{b}^{-1}$)] and end-systolic volume [ESV ($\text{mL} \cdot \text{b}^{-1}$)]. Moreover, $a-vO_{2diff}$ can be expanded as the difference between arterial oxygen concentration [CaO_2 ($\text{mL} \cdot \text{L}^{-1}$)] & venous oxygen concentration [CvO_2 ($\text{mL} \cdot \text{L}^{-1}$)] (6):

$$2. \quad VO_2 = [HR \times (EDV - ESV)] \times (CaO_2 - CvO_2)$$

In order to alter VO_{2max} , SV & CvO_2 are the typical variables that change with training or detraining. Following an exercise intervention, SV may increase via increases in blood volume, LV chamber diameter, &/or LV wall thickness. For instance, Spence et al. (35) reported a significant increase in LV mass, wall thickness, and EDV following a 24-week endurance training intervention. The cardiac hypertrophy evident in the 10 young healthy male participants led to a significant increase in VO_{2max} from 45.8 ± 1.6 to 49.3 ± 2.2 mL*kg⁻¹*min⁻¹ ($P < 0.05$) (35). In a classic comparative study by Morganroth et al. (20), collegiate swimmers & runners presented with greater mean LV internal dimensions at end-diastole, LV EDV, & LV mass when compared with untrained control participants of the same age & gender ($P < 0.001$). Consequently, aerobic endurance exercise via swimming & running alters LV architecture in ways that increase SV, & thus VO_{2max} . VO_{2max} may increase via a decrease in CvO_2 , as well (20). CvO_2 may decrease after an exercise intervention through increases in capillary density or mitochondrial capacity, which will improve the rate of O₂ extraction at the exercising muscles.

The time course of changes for variables such as blood volume, LV chamber diameter, LV chamber thickness, capillary density, mitochondrial capacity, & VO_{2max} varies. A 10% increase in blood volume following aerobic training has been observed to occur after 1-4 days, as noted by a meta-analysis by Sawka et al. (30); the initial increase in blood volume is accounted for via plasma expansion, yet erythrocyte volume expansion is observed after 2-3 weeks of training. With regards to LV dimensions, Ehsani et al. (11) observed a significant increase ($P < 0.005$) in LV end-diastolic dimension from 48.7 ± 1.7 to 53 ± 0.2 mm in 8 swimmers after only 1 week of training; the researchers also observed a significant increase ($P < 0.005$) in LV posterior wall thickness from 9.4 ± 0.4 to 10.1 ± 0.4 mm after 9 weeks of training. Significant increases in capillary density generally take longer to occur than the aforementioned central factors.

Andersen et al. (1) found that capillary density increased significantly ($P < 0.01$) from 329 to 395 cap*mm⁻² in 5 participants after 8 weeks of training on a cycle ergometer; they also observed a significant increase ($P < 0.01$) in capillaries per muscle fiber after only 5 weeks of training. Lastly, significant increases in VO_{2max} have been observed after only 3 weeks of training on a cycle ergometer in 8 older men (pre: 2.29 ± 0.49 ; 3 weeks: 2.48 ± 0.42 L*min⁻¹; $P < 0.05$) & in 8 younger men (pre: 3.82 ± 0.47 ; 3 weeks: 4.27 ± 0.52 L*min⁻¹; $P < 0.05$) (22).

Changing VO_{2max} : Detraining

Detraining is defined as the partial or complete loss of training-induced anatomical, physiological, & performance adaptations due to training reduction or cessation (21). Detraining impacts several measures, including VO_{2max} , blood volume, stroke volume, heart rate, & cardiac output. Mujika et al. (21) report that decreases in VO_{2max} have been observed after 14 & 15 days of training cessation. Further, Coyle et al. (8) completed a detraining study with 7 endurance-trained participants & observed a 6% decrease in VO_{2max} after only 12 days of cessation ($P < 0.05$); ultimately, after 56 days, VO_{2max} decreased & stabilized at a level 14% below trained levels ($P < 0.05$). After a period of inactivity, blood volume also decreases as soon as 2 days after cessation. This decrease in blood volume induces a decrease in stroke volume, as well (21). Coyle et al. (8) observed a 10% decrease in stroke volume after 12 days & up to a 14% decrease after 84 days ($P < 0.05$). As a result of decreased stroke volume, heart rate during submaximal & maximal exercise has been observed to increase by 5-10% (21). Correspondingly, the 7 endurance-trained participants experienced significant increases ($P < 0.05$) in HR_{max} of 4, 5, 6 & 5% at 12, 21, 56, & 84 days, respectively (8). As a consequence of the changes in HR_{max} & SV_{max} , CO_{max} has also been observed to decrease after training cessation (21). In the study by

Coyle et al. (8), CO_{max} was significantly reduced ($P < 0.05$) by 8% after 21 days & up to 9% after 84 days of detraining. Factors that did not significantly change following detraining in the 7 endurance-trained individuals included myoglobin concentration & muscle capillarization (8).

Limiters of $\text{VO}_{2\text{max}}$

Several variables may act as the limiting factor for $\text{VO}_{2\text{max}}$. Central factors consist of pulmonary diffusing capacity, CO_{max} , & the oxygen carrying capacity of the blood (4). Pulmonary diffusing capacity is typically not the limiter of $\text{VO}_{2\text{max}}$ in average individuals exercising at sea level. However, elite individuals may have an exceptionally high CO, resulting in a decreased transit time of red blood cells in the pulmonary capillaries. The reduced transit time may result in arterial O_2 desaturation & become a limiting factor of $\text{VO}_{2\text{max}}$ (4,9). To combat this limitation, an increase in O_2 saturation (from 90.6% to 95.9%) during maximal work is evident in elite individuals with the use of hyperoxic air (4,25). Pulmonary diffusing capacity may act as the limiter in individuals exercising at moderately high altitudes (9), as well as those with asthma or chronic obstructive pulmonary disease (4). CO, defined as the product of HR & SV, is another possible central limiter of $\text{VO}_{2\text{max}}$. However, because HR_{max} does not change significantly after exercise training, change in SV_{max} is the main determinant of CO_{max} . In a study by Saltin et al. (28), $\text{VO}_{2\text{max}}$ decreased after 20 days of bed rest & increased after 50 days of training, with the majority of the change in $\text{VO}_{2\text{max}}$ attributable to alterations in SV, emphasizing that SV is often the limiter of $\text{VO}_{2\text{max}}$. The oxygen carrying capacity of blood may also act as a central limiter of $\text{VO}_{2\text{max}}$. Blood doping, which is the practice of reinfusing red blood cells to increase hemoglobin content, has been observed to increase the oxygen carrying capacity of blood in a systematic review by Gledhill (13). When the hemoglobin content of blood is

increased via blood doping, $\text{VO}_{2\text{max}}$ may improve by 4-9% indicating that the oxygen-transport capacity of the blood may limit $\text{VO}_{2\text{max}}$ (4).

In addition to central factors, peripheral factors may also act as the limiting variable of $\text{VO}_{2\text{max}}$. Peripheral factors involve skeletal muscle characteristics, including mitochondrial enzyme levels & capillary density (4). Mitochondrial enzyme levels are representative of mitochondria sites for O_2 uptake, yet an increase in mitochondrial enzyme levels does not significantly increase $\text{VO}_{2\text{max}}$, indicating that it is not a major limiter of $\text{VO}_{2\text{max}}$. For instance, Saltin et al. (29) noted 2.2-fold increase in mitochondrial enzyme activity induced only a 20-40% increase in $\text{VO}_{2\text{max}}$. Capillary density is important for oxygen delivery & extraction at the muscle; consequently, increasing capillary density will elongate mean O_2 transit time and may improve $\text{VO}_{2\text{max}}$. In the aforementioned study by Saltin et al. (29), the number of capillaries per fiber in the vastus lateralis was strongly related to relative $\text{VO}_{2\text{max}}$ when measured via cycle ergometry. Therefore, increasing mitochondrial enzyme activity & capillary density may decrease CvO_2 , increase $a\text{-vO}_{2\text{diff}}$, & increase $\text{VO}_{2\text{max}}$, but tend not to be limiting factors of $\text{VO}_{2\text{max}}$ (4).

As aforementioned, limiting factors of $\text{VO}_{2\text{max}}$ differ between highly fit & average individuals. In elite & highly-trained individuals, a pulmonary limitation may exist as a result of high CO decreasing the diffusion time in pulmonary capillaries (9). Consequently, O_2 saturation decreases & impacts $\text{VO}_{2\text{max}}$ by decreasing the amount of oxygen delivered to exercising muscles. Moreover, Gifford et al. (12) used *in vivo* and *in vitro* methods of measuring O_2 supply & demand in 10 young untrained males & 10 young endurance-trained males. The researchers observed that $\text{VO}_{2\text{max}}$ is limited by oxygen supply in trained participants, indicating that central factors are indeed limiting in trained individuals (12). On the contrary, the $\text{VO}_{2\text{max}}$ of average

individuals is generally limited by SV & mitochondrial capacity. As noted by Gifford et al. (12) in the same study, mitochondrial O₂ demand was the main limiter of VO_{2max} in the untrained participants; thus, mitochondrial capacity limits VO_{2max} in untrained individuals. The limiting factors of VO_{2max} differ between normal conditions & hypoxic conditions, as well. At hypoxic conditions, O₂ saturation is decreased, effectively lowering the ability of the cardiopulmonary system to delivery oxygen to the muscles (4). At normal conditions at sea-level, SV is the primary limiter of VO_{2max} (4).

In addition to physiological limitations, Noakes (23) argues for the central governor model (CGM), in which the brain & central nervous system (CNS) maintain ultimate regulation of exercise performance. Thus, the CGM predicts that the VO_{2max} achieved from a GXT may be limited by the CNS ending exercise before physiological failure of the body's systems to deliver oxygen to exercising muscles. Continuous feedback from the body's organs on fuel reserves, heat accumulation, hydration, & other variables signal the CNS to modify pace or end exercise. Noakes also notes research that displays the effects of music, placebos, prior experience, self-belief, knowledge of endpoint, presence of competitors, & other psychological factors on exercise performance (23).

Overall, the limiters of VO_{2max} vary based on the training level of the individual, altitude, & O₂ saturation of the environment. VO_{2max} in untrained individuals may be limited due to mitochondrial O₂ demand or SV; in highly trained athletes, VO_{2max} may be limited due to the pulmonary diffusing capacity or the O₂ saturation of the environment. Additionally, the CNS in Noakes' CGM may prevent all individuals from achieving true VO_{2max} to protect the body from physiological failure.

Changing VO_{2max} : Exercise Interventions

Exercise interventions are utilized to increase VO_{2max} in individuals. The intensity of exercise is a significant factor at determining the effectiveness of the protocol at raising VO_{2max} . For example, Gormley et al. (14) completed a randomized controlled trial (RCT) with 61 young healthy adult participants; all were randomly assigned to a non-exercising control group, a moderate-intensity group (50% VO_2 reserve), a vigorous-intensity group (75% VO_2 reserve), or near-maximal-intensity group (95% VO_2 reserve). Exercise volume & energy expenditure were controlled by varying duration & frequency. VO_{2max} increased significantly ($P < 0.05$) by 3.4, 4.8, & 7.2 $mL \cdot kg^{-1} \cdot min^{-1}$ in the moderate-intensity, vigorous-intensity, & near-maximal-intensity groups, respectively. Therefore, the researchers observed that increasing intensities will induce greater increases in VO_{2max} in young healthy adults when volume is controlled (14).

The amount of exercise prescribed is also an important factor at determining the effectiveness of a protocol at raising VO_{2max} . In an additional RCT, Ross et al. (27) examined the separate effects of intensity & amount of exercise on VO_{2max} in 121 sedentary, middle-aged, obese adults. 39 were randomly assigned to low-amount, low-intensity (LALI) exercise, 51 to high-amount, low-intensity (HALI) exercise, & 31 to high-amount, high-intensity (HAHI) exercise for 24 weeks. After the 24 weeks, all 3 groups experienced a significant increase in VO_{2max} ($P < 0.001$). 38.5%, 17.6%, & 0% of the participants in the LALI, HALI, & HAHI groups, respectively were nonresponsive to changes in VO_{2max} at 24 weeks. Consequently, when exercise amount was fixed, increased exercise intensity eliminated nonresponse ($P < 0.001$); when exercise intensity was fixed, increased exercise amount reduced the rate of nonresponse ($P < 0.02$). Thus, Ross et al. (26) observed that increasing exercise intensity & amount separately both led to greater increases in VO_{2max} in obese, middle-aged participants.

Although it is accepted that separate increases in intensity & volume of training generally lead to greater gains in VO_{2max} , altering intensity & volume simultaneously may not lead to significantly different increases in VO_{2max} after training between protocols. For example, in a meta-analysis of 28 studies by Scribbans et al. (32), the authors formed 3 groups based on intensity (1: 60-70%; 2: 80-92.5%; 3: 100-250% VO_{2max}); however, as each group increased in intensity, session dose & total training volume decreased significantly ($P < 0.05$). Nonetheless, there was no significant difference in mean change in VO_{2max} between groups after training (1: $+0.29 \pm 0.15 \text{ L}\cdot\text{min}^{-1}$; 2: $+0.26 \pm 0.10 \text{ L}\cdot\text{min}^{-1}$; 3: $+0.35 \pm 0.17 \text{ L}\cdot\text{min}^{-1}$) (32). Scribbans et al. (32) concluded that similar increases in VO_{2max} can be achieved with low training doses at higher intensities & high training doses at lower intensities.

High-intensity interval training (HIIT) interventions have led to significant increases in VO_{2max} after 2-12 weeks of training (2). Astorino et al. (2) completed an RCT & examined the effects of 10 sessions of HIIT, followed by 10 sessions of sprint interval training (SIT), high-volume interval training ($HIIT_{HI}$), or periodized interval training (PER) in 39 men & women ($22.9 \pm 5.4 \text{ yr}$, $39.6 \pm 5.6 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) in comparison to 32 non-exercising controls ($25.7 \pm 4.5 \text{ yr}$, $40.7 \pm 5.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$). All 3 HIIT protocols led to significant increases in VO_{2max} ($P < 0.001$) due to significant increases in CO ($P < 0.04$) & SV ($P < 0.04$) (2). Additionally, Bacon et al. (3) completed a meta-analysis of 37 studies with 334 total participants to examine the effects of HIIT on VO_{2max} , as well. In the 334 participants, a significant increase in VO_{2max} of $0.51 \text{ L}\cdot\text{min}^{-1}$ (95% CI: 0.43 to $0.60 \text{ L}\cdot\text{min}^{-1}$) was observed. Of those 37 studies, 9 used longer intervals and led to a greater increase of approximately 0.80 - $0.90 \text{ L}\cdot\text{min}^{-1}$ in VO_{2max} (3). Furthermore, another 2016 meta-analysis by Batacan et al. (5) examined 65 intervention studies to determine the effects of HIIT on VO_{2max} & cardiometabolic health. Batacan et al. observed

that short-term HIIT protocols (< 12 weeks) increased VO_{2max} by a large effect in normal weight populations by a standardized mean difference (SMD) of 0.83 (95% CI: 0.56 to 1.10) and by a medium effect in overweight/obese populations by an SMD of 0.74 (95% CI: 0.36 to 1.12). The researchers also observed that long-term HIIT protocols (\geq 12 weeks) increased VO_{2max} by a large effect in overweight/obese populations by an SMD of 1.20 (95% CI: 0.57 to 1.83) (5).

In comparison to moderate-intensity continuous training (MICT), HIIT generally induces a more significant increase in VO_{2max} (15,17,34). For example, Milanovic et al. (19) completed a meta-analysis of 28 studies with 723 total participants in 2015 comparing the effects of HIIT & continuous endurance training on VO_{2max} . They observed an increase of $4.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: 3.5 to $6.3 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) following a continuous endurance training program. However, they also observed an increase of $5.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: 4.3 to $6.7 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) following a HIIT program. Thus, both types of protocols induced increases in VO_{2max} , yet HIIT led to greater gains (19). Another meta-analysis by Weston et al. (36) examined the effects of HIIT & MICT protocols on VO_{2max} in participants with coronary artery disease, heart failure, hypertension, metabolic syndrome, & obesity. The researchers analyzed 10 studies with 273 total participants & observed a greater increase in VO_{2max} following a HIIT protocol than a MICT protocol with a mean difference of $3.03 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: 2.00 to 4.07), showing the superiority of HIIT at improving CRF (36). Karstoft et al. (17) conducted an RCT to examine the effects of MICT-walking & HIIT-walking in participants with T2DM. 12 participants in the MICT-walking group exercised at 55% of the peak energy-expenditure rate, while 12 participants in the HIIT-walking group exercised above & below 70% of the peak energy-expenditure rate in 3-min cycles. All participants exercised 5 times/week, 60 min/session for 4 months. Following the HIIT-walking protocol, VO_{2max} significantly increased by 4.4 ± 1.2

$\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ ($P < 0.001$), while no change occurred following the MICT-walking protocol (17).

Although most studies indicate HIIT is superior to MICT at improving $\text{VO}_{2\text{max}}$, some researchers observe no difference. Sheperd et al. (34) compared the effects of HIIT & MICT on spinning bikes in a gym setting on $\text{VO}_{2\text{max}}$. 46 participants followed a HIIT protocol with repeated sprints (15-60s, $> 90\%$ HR_{max}) & active recovery (≤ 25 min/session, 3 sessions/week). 44 participants followed a MICT protocol with continuous cycling ($\sim 70\%$ HR_{max} , 30-45 min/session, 5 sessions/week) (34). $\text{VO}_{2\text{max}}$ increased by a mean difference of $2.8 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: 2.0 to 3.6) after the HIIT protocol; $\text{VO}_{2\text{max}}$ increased by a mean difference of $2.4 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: 1.7 to 3.3) after the MICT protocol. Thus, the protocols did not lead to significantly different increases in $\text{VO}_{2\text{max}}$ ($P = 0.90$), indicating that MICT protocols are not always inferior to HIIT (34). Additionally, Sawyer et al. (31) observed similar increases in $\text{VO}_{2\text{max}}$ in 17 obese participants ($\text{BMI} \geq 30 \text{ kg/m}^2$) following HIIT & MICT protocols. 8 participants completed 8 weeks of HIIT (10 1-min intervals at 90-95% HR_{max} with 1-min of cycling at a low intensity between intervals) & 9 participants completed 8 weeks of MICT (30 min of cycling at 70-75% HR_{max}). In the HIIT group, $\text{VO}_{2\text{max}}$ increased significantly ($P = 0.01$) from $2.19 \pm 0.65 \text{ L/min}$ to $2.64 \pm 0.88 \text{ L/min}$; in the MICT group, $\text{VO}_{2\text{max}}$ increased significantly ($P < 0.01$) from $2.24 \pm 0.48 \text{ L/min}$ to $2.55 \pm 0.61 \text{ L/min}$ (31). Therefore, HIIT & MICT interventions may induce similar increases in $\text{VO}_{2\text{max}}$ in individuals.

When examining the general effects of aerobic exercise interventions on $\text{VO}_{2\text{max}}$, the aforementioned studies show that greater intensity & greater volume of aerobic exercise may lead to more significant increases in $\text{VO}_{2\text{max}}$ (14,26); however, no significant difference in increases in $\text{VO}_{2\text{max}}$ has been observed between groups of high-amount, low-intensity exercise &

low-amount, high-intensity exercise (32). Additionally, previous studies agree that HIIT is an effective protocol for increasing VO_{2max} (2,3,5) & that HIIT is more effective than MICT (17,19,36, yet several studies observed no significant difference between increases in VO_{2max} following HIIT & MICT protocols (31,34).

Introduction

Maximum oxygen uptake (VO_{2max}) is defined as the maximal amount of oxygen that can be consumed by the body during maximal exercise. VO_{2max} assesses the body's ability via the pulmonary, cardiovascular, & skeletal muscle systems to uptake, transport, & utilize O_2 in contracting muscle mitochondria at maximal exercise (24). Hence, measuring VO_{2max} is the gold standard for assessing cardiorespiratory fitness (CRF). CRF is an important marker in clinical practice. Low CRF is associated with an increased risk of cardiovascular disease (CVD) and all-cause mortality (26). Furthermore, improvements in CRF are associated with an increased rate of survival; a 1-MET ($3.5 \text{ mL} \cdot \text{kg}^{-1} \cdot \text{min}^{-1}$) increase in VO_{2max} has been associated with a 13% risk reduction of all-cause mortality & a 15% risk reduction of CVD (18). Consequently, creating effective exercise interventions that increase VO_{2max} is critical to improve the health of individuals at risk of CVD & other chronic diseases.

Aerobic exercise interventions can be used to increase CRF. Specifically, high-intensity interval training (HIIT) & moderate-intensity continuous training (MICT) are two common interventions utilized to improve VO_{2max} . Generally, previous literature has shown that HIIT induces a more significant increase in VO_{2max} than MICT (17,19,36). However, other studies have observed similar increases in VO_{2max} when comparing the two protocols (31,34). Additionally, comparisons of HIIT & MICT in previous literature have typically used differences in intensity or volume to determine their effects on VO_{2max} . On the contrary, the current study utilizes a 4-week MICT conditioning period, followed by 8-week interventions of HIIT or MICT that differ in both intensity & volume in comparison to one another.

The purpose of the investigation was to compare the effects of 8 weeks of low-volume HIIT versus the effects of 8 weeks of high-volume MICT on VO_{2max} in sedentary adults. We

hypothesized that 8 weeks of low-volume HIIT would not improve VO_{2max} more than 8 weeks of high-volume MICT; similar increases would be evident due to the different intensities, different volumes of exercise, & equal use of both the cycle ergometer & treadmill in the intervention.

Methods

Participants & Testing Overview:

The study was approved by Point Loma Nazarene University's Institutional Review Board. All participants provided written informed consent before participation. 83 participants volunteered for the study; 24 final participants completed the study (21 female, 3 male). The process of participant enrollment, allocation, & analysis is displayed in Figure 1. All participants met the following inclusion criteria: between 18-55 years of age, completion of the Physical Activity Readiness Questionnaire (PAR-Q) without "yes" answers, free from known chronic disease, capable of performing physical activity, & are currently non-active (engage in < 30 minutes of daily aerobic activity). Baseline anthropometric measurements of all participants are displayed in Table 1.

Age (yr)	Weight (kg)	Height (m)	Body Fat (%)	Fat Mass (kg)	Fat-Free Mass (kg)
27.1 ± 8.4	75.3 ± 23.4	1.71 ± 0.10	31.6 ± 9.2	25.1 ± 15.5	50.3 ± 11.4

Table 1. Mean ± SD of baseline anthropometric measurements of all participants (n = 24).

Prior to baseline testing, the participants visited the lab to sign the consent form, fill out the PAR-Q, & become accustomed to exercise on the cycle ergometry at 70% of their age-predicted HR_{max} ($220 - \text{age} = \text{predicted } HR_{max}$) for 20 minutes if necessary. Exercising at 70% of HR_{max} is considered moderate intensity by the American College of Sports Medicine; it prepared participants for future exercise sessions. All participants completed testing at baseline, after 4 weeks, after 8 weeks, & after 12 weeks of exercise. On testing days, the participants arrived in a fasted state (no food in the previous 8 hours & no caffeine, alcohol, or dietary supplements in the previous 24 hours) & did not exercise in the previous 24 hours.

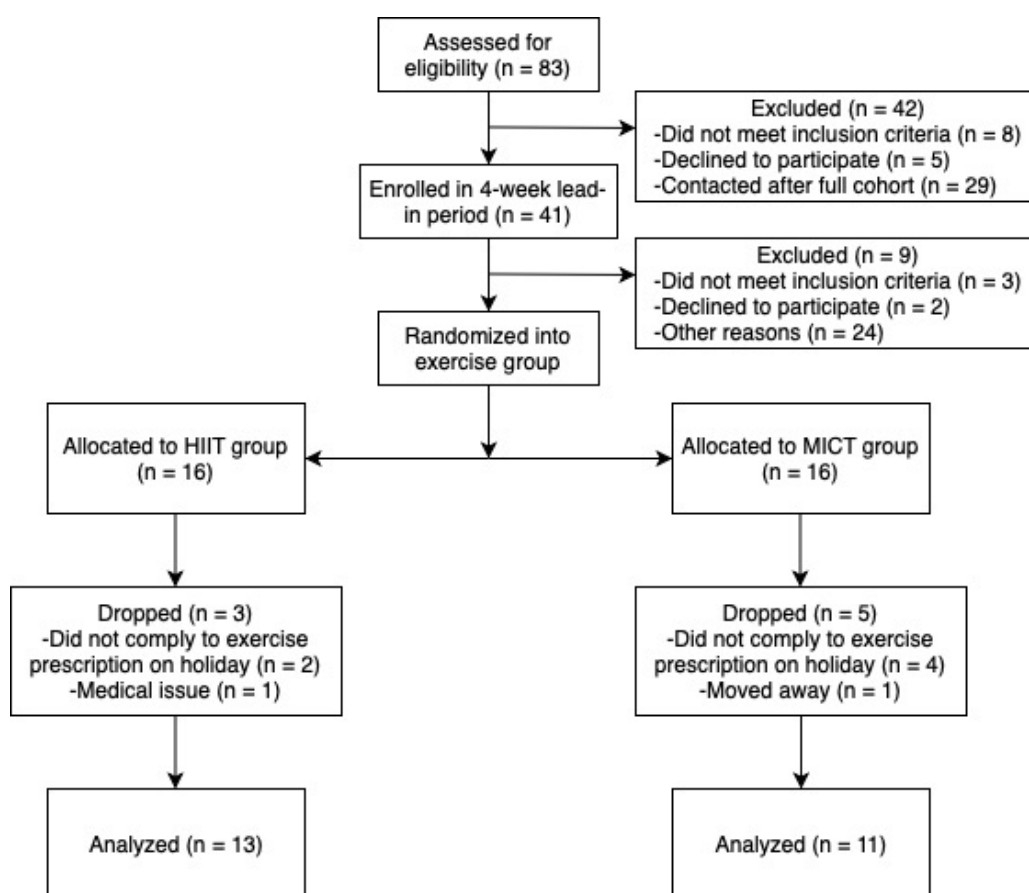


Figure 1. Flowchart of participant allocation & drop-out throughout the intervention from initial recruitment.

Body Composition Assessment:

On the testing days at baseline, after 4 weeks, & 12 weeks of exercise, all participants completed a body composition assessment utilizing air displacement plethysmography via the BodPod (Cosmed, Rome, Italy). Air displacement plethysmography involves measuring the amount of air the participant's body displaces, enabling the measurement of two-compartment (fat & fat-free) body density. The non-invasive test is completed in approximately 5 minutes. To avoid air-trapping in the BodPod, participants wore spandex shorts & a speedo cap with minimal cotton; women also wore sports bras with no padding. Air within the lungs is accounted for via a thoracic gas volume (TGV) measurement, as well. Body mass, body fat percentage, fat mass, & fat-free mass were measured. Height was measured via a wall-mounted stadiometer (Seca, Hamburg, Germany).

Cardiorespiratory Fitness Assessment:

On the testing days at baseline, after 4 weeks, 8 weeks, & 12 weeks of exercise, all participants completed a CRF assessment via a ramp graded exercise test (GXT) on an electronically braked cycle ergometer (Lode Corival, Groningen, Netherlands). Pulmonary ventilation and gas exchange were measured continuously via a Parvo Medics TrueOne 2400 metabolic cart (Parvo Medics, Sandy, UT). Flowmeter calibration & gas calibration were completed to manufacturer standards. Heart rate was measured via a Polar HR monitor (Polar Electro, Lake Success, NY). During the GXT, the participants were equipped with the HR monitor, headgear, & a mouthpiece connected to a hose. After collecting resting oxygen data for 2 minutes, the participants completed a warm-up at 0 Watts, 10 Watts, & 25 Watts for 3 minutes

at each stage. Prior to the GXT, VO_{2peak} was estimated utilizing a non-exercise VO_{2peak} prediction equation determined by Jackson et al. (16). The equation states:

$$VO_{2peak} \text{ (mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}\text{)} = 56.363 + 1.921\cdot(\text{PA-R}) - 0.381\cdot(\text{Age}) - 0.754\cdot(\text{BMI}) + 10.987\cdot(\text{F} = 0, \text{M} = 1)$$

Thus, the estimated VO_{2peak} allows for the calculation of the estimated increase in resistance (~10-30 watts/min). Consequently, during the GXT, the resistance increases continuously at the pre-determined rate until volitional exhaustion. Following a 10-minute active recovery at 25 watts, the participants completed a verification phase at 95% of the max wattage achieved during the GXT, although both submaximal & supramaximal protocols are equally effective at verifying VO_{2max} (33). Final results utilized averages of the two highest consecutive 15-second recordings. The VO_{2max} value recorded at each time point was the highest average VO_{2max} value attained after the GXT & the verification phase.

Exercise Intervention:

During the 12-week exercise intervention, all participants warmed up & cooled down for 5 minutes before & after each session at 50 watts on the cycle ergometer or 3.0 miles/hour on the treadmill. Polar HR monitors were utilized to maintain HR within the desired range. The exercise sessions were split evenly on the cycle ergometer & the treadmill (Trackmaster TMX428CP, Newton, KS) to prevent the participants' familiarization with the exercise modalities. HR, cycle ergometer RPM, wattage, treadmill speed, & incline were recorded every 5 minutes to ensure the participants stayed within the desired ranged.

During the initial 4 weeks of conditioning, all participants completed MICT sessions 3 days/week, 30 minutes/session at 70-76% HR_{max} as calculated utilizing the HR_{max} achieved during the initial GXT. After the initial 4 weeks & reassessment of body composition & CRF, all participants were randomly assigned via a randomizer into low-volume HIIT or high-volume

MICT. For the remaining 8 weeks of exercise, participants in the low-volume HIIT group exercised 3 days/week, 20 minutes/session with 10 1-min intervals at 90-95% HR_{max} & 1-min recovery periods between; additionally, participants in the high-volume MICT group exercised 5 days/week, 30 minutes/session at 70-76% HR_{max} . HR ranges were calculated according to the participant's most recent GXT.

Statistical Analysis:

Descriptive statistics (means \pm SD) for the study participants were calculated across intervention groups (HIIT & MICT), as displayed in Table 2 & Table 3. 2-way ANOVA tests were used to detect differences in several variables over time, between exercise groups, & with a group x time interaction. A Bonferroni post hoc test was used to adjust for multiple comparisons. The best-fitting linear mixed-effect models were determined using forward stepwise model selections using likelihood ratio tests for several variables, as displayed in Table 4. The exercise group, gender, & exercise group x gender interaction were set as fixed effects; time was set as a random effect. Correlation coefficients were calculated between GXT time-to-exhaustion (TTE) & the difference between VP VO_{2max} & GXT VO_{2max} (VP-GXT VO_{2max}), as well as VP TTE & VP-GXT VO_{2max} . All *P* values were two-tailed & values less than 0.05 were considered to indicate statistical significance. All statistical procedures were performed via SPSS 20 (IBM, Armonk, NY) & RStudio (RStudio, Boston, MA).

Results

Cardiorespiratory Fitness:

After the 4-week conditioning period of MICT, all 24 participants (27.1 ± 8.4 yr, $VO_{2max} = 27.7 \pm 6.7$ ml*kg⁻¹*min⁻¹) were randomized into the low-volume HIIT group or the high-volume MICT group. 13 participants (28 ± 9.7 yr, $VO_{2max} = 29.0 \pm 6.0$ ml*kg⁻¹*min⁻¹) were randomized into the HIIT group. 11 participants (26 ± 6.9 yr, $VO_{2max} = 26.2 \pm 7.3$ ml*kg⁻¹*min⁻¹) were randomized into the MICT group.

All cardiorespiratory fitness measurements are displayed in Table 2. The highest VO_{2max} values were determined as the highest VO_{2max} attained in the GXT or VP. In all participants, VO_{2max} increased significantly from baseline to week 4 (Baseline = 2.01 ± 0.47 L*min⁻¹, Week 4 = 2.13 ± 0.44 L*min⁻¹, $P = 0.003$), from baseline to week 8 (Week 8 = 2.22 ± 0.40 L*min⁻¹, $P < 0.001$), & from baseline to week 12 (Week 12 = 2.30 ± 0.41 L*min⁻¹, $P < 0.001$). In the HIIT group, VO_{2max} did not increase significantly from baseline to week 4 (Baseline = 1.99 ± 0.25 L*min⁻¹, Week 4 = 2.08 ± 0.21 L*min⁻¹, $P = 0.117$), yet it did increase significantly from baseline to week 8 (Week 8 = 2.18 ± 0.24 L*min⁻¹, $P = 0.016$) & from baseline to week 12 (Week 12 = 2.29 ± 0.19 L*min⁻¹, $P = 0.001$). However, VO_{2max} did increase significantly from week 4 to week 12 ($P < 0.001$), as well. In the MICT group, VO_{2max} did not increase significantly from baseline to week 4 (Baseline = 2.04 ± 0.65 L*min⁻¹, Week 4 = 2.18 ± 0.63 L*min⁻¹, $P = 0.067$), yet it did increase significantly from baseline to week 8 (Week 8 = 2.27 ± 0.54 L*min⁻¹, $P = 0.012$) & from baseline to week 12 (Week 12 = 2.30 ± 0.58 L*min⁻¹, $P = 0.002$). However, VO_{2max} did increase significantly from week 4 to week 12 ($P = 0.010$), as well. Changes in VO_{2max} in HIIT & MICT from baseline to week 12 are displayed in Figure 2. There

was no group X time interaction for changes in VO_{2max} from baseline to week 12 ($P = 0.878$) or from week 4 to week 12 ($P = 0.755$)

GXT VO_{2max} values increased significantly in the HIIT group (Baseline = 29.0 ± 6.0 $ml \cdot kg^{-1} \cdot min^{-1}$, Week 12 = 34.1 ± 7.5 $ml \cdot kg^{-1} \cdot min^{-1}$, $P = 0.0496$), but GXT VO_{2max} values did not increase significantly in the MICT group (Baseline = 26.2 ± 7.3 $ml \cdot kg^{-1} \cdot min^{-1}$, Week 12 = 29.6 ± 7.3 $ml \cdot kg^{-1} \cdot min^{-1}$, $P = 0.199$). Additionally, VP VO_{2max} values did not increase significantly in the HIIT or MICT groups (HIIT: Baseline = 28.4 ± 6.8 $ml \cdot kg^{-1} \cdot min^{-1}$, Week 12 = 33.0 ± 6.8 $ml \cdot kg^{-1} \cdot min^{-1}$, $P = 0.0779$; MICT: Baseline = 25.4 ± 6.7 $ml \cdot kg^{-1} \cdot min^{-1}$, Week 12 = 29.2 ± 7.3 $ml \cdot kg^{-1} \cdot min^{-1}$, $P = 0.179$).

The VP-GXT VO_{2max} values from baseline to week 12 are displayed in Figure 3. The VP-GXT VO_{2max} values did not change significantly in the HIIT or MICT groups (HIIT: Baseline = 0.0 ± 0.11 $L \cdot min^{-1}$, Week 12 = -0.07 ± 0.09 $L \cdot min^{-1}$, $P = 0.401$; MICT: Baseline = -0.1 ± 0.10 $L \cdot min^{-1}$, Week 12 = -0.03 ± 0.07 $L \cdot min^{-1}$, $P = 0.911$). GXT TTE did not change significantly in the HIIT or MICT groups (HIIT: Baseline = 10.1 ± 1.7 min, Week 12 = 9.2 ± 1.3 min, $P = 0.056$; MICT: Baseline = 9.5 ± 2.8 min, Week 12 = 9.4 ± 0.7 min, $P = 0.971$). Furthermore, VP TTE also did not change significantly in the HIIT or MICT groups (HIIT: Baseline = 2.5 ± 0.6 min, Week 12 = 2.3 ± 0.6 min, $P = 0.33$; MICT: Baseline = 2.1 ± 0.6 min, Week 12 = 2.2 ± 0.5 min, $P = 0.455$). GXT wattage increased significantly in all participants (Baseline = 169 ± 50 W, Week 12 = 204 ± 39 W, $P < 0.002$), but there was no group x time interaction ($P = 0.785$). GXT wattage increased significantly in the HIIT group (Baseline = 165 ± 19 W, Week 12 = 202 ± 24 W, $P < 0.001$), but not the MICT group (Baseline = 175 ± 58 W, Week 12 = 206 ± 53 W, $P = 0.144$). Correlation coefficients were calculated to examine the relationship between VP-GXT VO_{2max} & GXT TTE ($R = -0.2167$), as well as VP-GXT VO_{2max} & VP TTE ($R = 0.3636$).

	HIIT (n = 13)					MICT (n = 11)					Group X Time Interaction <i>P</i> value
	Baseline	Wk 4	Wk 8	Wk 12	Time Effect <i>P</i> value	Baseline	Wk 4	Wk 8	Wk 12	Time Effect <i>P</i> value	
Highest VO _{2max} (L*min ⁻¹)	1.99 ± 0.25	2.09 ± 0.21	2.18 ± 0.24	2.29 ± 0.19	0.0006	2.04 ± 0.65	2.18 ± 0.63	2.27 ± 0.54	2.30 ± 0.58	0.002	0.878
GXT VO _{2max} (ml*kg ⁻¹ *min ⁻¹)	29.0 ± 6.0	30.2 ± 6.5	31.9 ± 7.7	34.1 ± 7.5	0.0496	26.2 ± 7.3	27.1 ± 6.8	28.8 ± 6.4	29.6 ± 7.3	0.199	0.684
VP VO _{2max} (ml*kg ⁻¹ *min ⁻¹)	28.4 ± 6.8	30.5 ± 6.7	31.3 ± 6.4	33.0 ± 6.8	0.0779	25.4 ± 6.7	27.2 ± 6.9	28.2 ± 6.7	29.2 ± 7.3	0.179	0.858
VP-GXT VO _{2max} (L*min ⁻¹)	0.0 ± 0.11	0.03 ± 0.11	-0.03 ± 0.10	-0.07 ± 0.09	0.401	-0.1 ± 0.10	0.01 ± 0.13	-0.05 ± 0.12	-0.03 ± 0.07	0.911	0.517
Highest HR _{max} (bpm)	186 ± 10	182 ± 12	181 ± 11	182 ± 14	0.385	188 ± 12	187 ± 11	187 ± 11	187 ± 10	0.698	0.739
Highest RER _{max}	1.29 ± 0.10	1.24 ± 0.06	1.28 ± 0.08	1.27 ± 0.08	0.958	1.26 ± 0.07	1.28 ± 0.08	1.28 ± 0.07	1.26 ± 0.07	0.831	0.917
GXT TTE (min)	10.1 ± 1.7	9.6 ± 0.5	9.5 ± 0.5	9.2 ± 1.3	0.056	9.5 ± 2.8	9.1 ± 1.4	9.5 ± 0.4	9.4 ± 0.7	0.971	0.269
VP TTE (min)	2.5 ± 0.6	2.7 ± 0.7	2.5 ± 0.9	2.3 ± 0.6	0.33	2.1 ± 0.6	2.4 ± 0.7	2.5 ± 0.5	2.2 ± 0.5	0.455	0.228
GXT Peak Wattage (W)	165 ± 19	174 ± 18	187 ± 21	202 ± 24	< 0.001	175 ± 58	183 ± 55	196 ± 50	206 ± 53	0.144	0.785

Table 2. Mean ± SD of cardiorespiratory fitness measurements at baseline, 4 weeks, 8 weeks, & 12 weeks of training. Group X time interaction from baseline to 12 weeks. Time effect from baseline to 12 weeks.

The highest HR_{max} values (the highest HR_{max} attained in the GXT or VP) did not change significantly in the HIIT or MICT groups (HIIT: Baseline = 186 ± 10 bpm, Week 12 = 182 ± 14 bpm, *P* = 0.385; MICT: Baseline = 188 ± 12 bpm, Week 12 = 187 ± 10 bpm, *P* = 0.698). The highest RER_{max} values (the highest RER_{max} attained in the GXT or VP) also did not change significantly in the HIIT or MICT groups (HIIT: Baseline = 1.29 ± 0.10, Week 12 = 1.27 ± 0.08, *P* = 0.958; MICT: Baseline = 1.26 ± 0.07, Week 12 = 1.26 ± 0.07, *P* = 0.831). No significant group X time interactions were observed in any of the cardiorespiratory fitness measures.

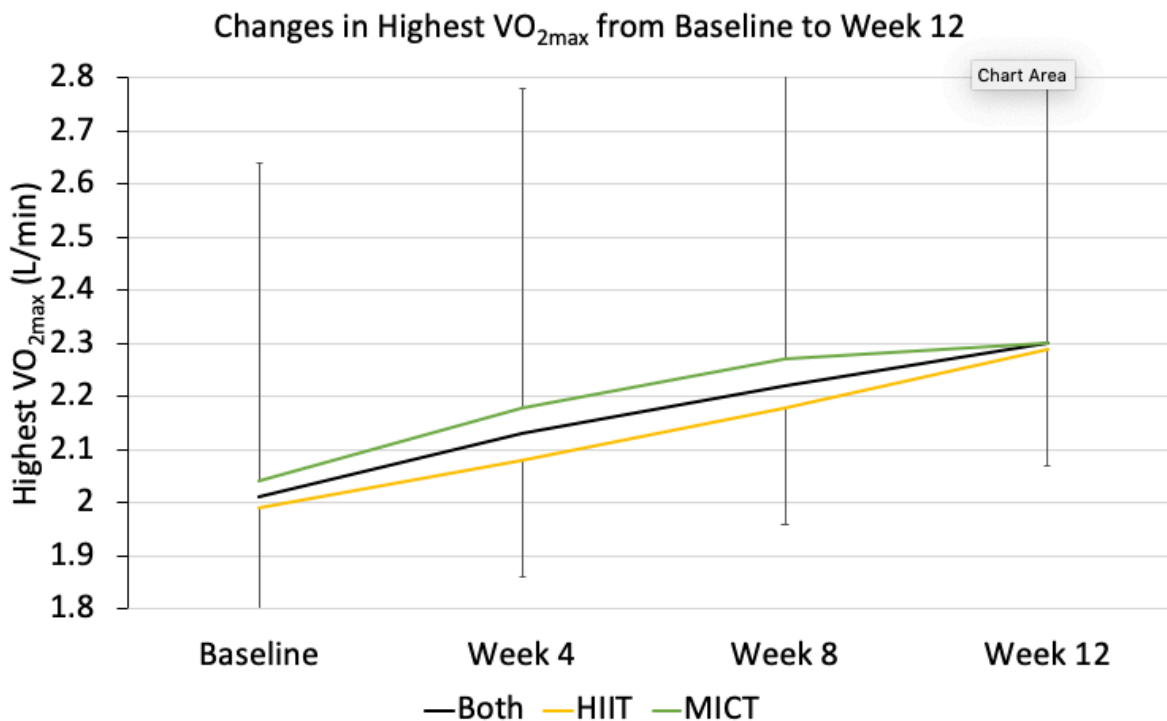


Figure 2. Mean \pm SD VO_{2max} in HIIT, MICT, & both over 12 weeks of training. *Significant time effect from baseline to week 12 ($P = 0.0182$); no group \times time interaction ($P < 0.878$).

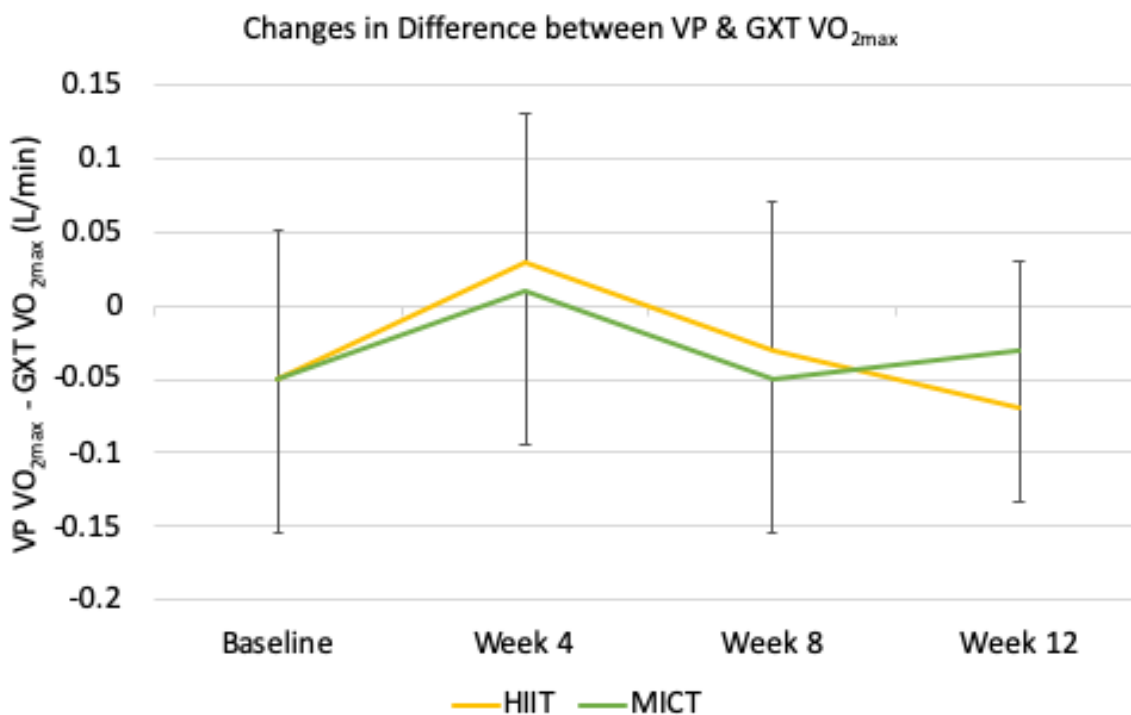


Figure 3. Mean \pm SD Verification phase $VO_{2max} - GXT VO_{2max}$ in HIIT & MICT over 12 weeks of training. No time effect from baseline to week 12 ($P = 0.592$); no group \times time interaction ($P = 0.517$).

Body Composition:

All changes in the anthropometric measurements are displayed in Table 3. Weight did not change significantly after the exercise interventions (HIIT: Baseline = 70.2 ± 15.2 kg, Week 12 = 69.6 ± 14.7 kg, $P = 0.939$; MICT: Baseline = 81.4 ± 30.0 kg, Week 12 = 81.6 ± 29.0 kg, $P = 0.978$). Changes in weight from baseline to week 12 are displayed in Figure 4. Body fat percentage also did not change after the exercise interventions (HIIT: Baseline = $30.7 \pm 6.6\%$, Week 12 = $30.8 \pm 7.5\%$, $P = 0.952$; MICT: Baseline = $32.6 \pm 11.8\%$, Week 12 = $34.5 \pm 8.9\%$, $P = 0.636$). Changes in body fat percentage from baseline to week 12 are displayed in Figure 5. Consequently, fat mass did not change significantly (HIIT: Baseline = 22.2 ± 8.6 kg, Week 12 = 22.2 ± 9.0 kg, $P = 0.993$; MICT: Baseline = 28.4 ± 21.0 kg, Week 12 = 30.2 ± 20.6 kg, $P = 0.836$) & fat-free mass did not change significantly (HIIT: Baseline = 48.0 ± 7.8 kg, Week 12 = 47.4 ± 7.1 kg, $P = 0.838$; MICT: Baseline = 52.9 ± 14.6 kg, Week 12 = 50.7 ± 10.9 kg, $P = 0.653$).

	HIIT (n = 13)					MICT (n = 11)					Group X Time Interaction P value
	Baseline	Wk 4	Wk 8	Wk 12	Time Effect P value	Baseline	Wk 4	Wk 8	Wk 12	Time Effect P value	
Weight (kg)	70.2 ± 15.2	70.1 ± 14.7	70.3 ± 15.4	69.6 ± 14.7	0.939	81.4 ± 30.0	81.9 ± 30.1	82.2 ± 29.3	81.6 ± 29.0	0.978	0.951
Body Fat (%)	30.7 ± 6.6	30.5 ± 6.7	N/A	30.8 ± 7.5	0.952	32.6 ± 11.8	32.0 ± 11.5	N/A	34.5 ± 8.9	0.636	0.688
Fat Mass (kg)	22.2 ± 8.6	22.0 ± 8.4	N/A	22.2 ± 9.0	0.993	28.4 ± 21.0	28.3 ± 21.5	N/A	30.2 ± 20.6	0.836	0.836
Fat-Free Mass (kg)	48.0 ± 7.8	48.1 ± 7.5	N/A	47.4 ± 7.1	0.838	52.9 ± 14.6	53.6 ± 13.6	N/A	50.7 ± 10.9	0.653	0.743

Table 3. Mean \pm SD of anthropometric measurements at baseline, 4 weeks, 8 weeks, & 12 weeks of training. Group X time interaction from baseline to 12 weeks. Time effect from baseline to 12 weeks.

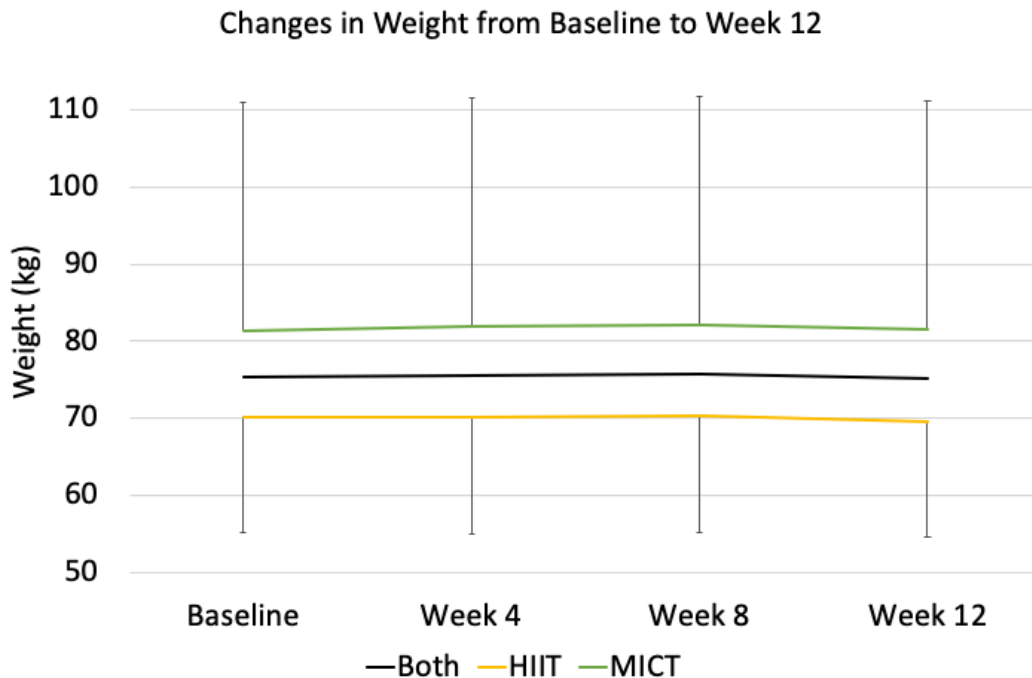


Figure 4. Mean \pm SD Weight in HIIT, MICT, & both over 12 weeks of training. No time effect from baseline to week 12 ($P = 0.990$); no group \times time interaction ($P = 0.951$).

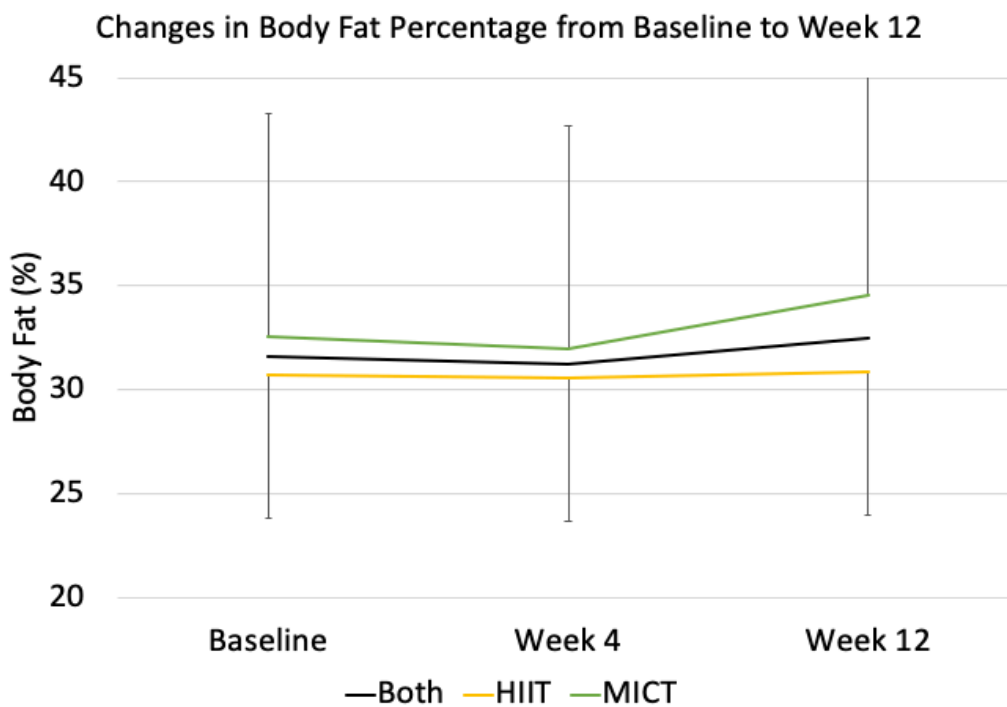


Figure 5. Mean \pm SD Body fat percentage in HIIT, MICT, & both over 12 weeks of training. No time effect from baseline to week 12 ($P = 0.673$); no group \times time interaction ($P = 0.688$).

Linear Mixed Effects Models:

Forward stepwise model selections using likelihood ratio tests were used to determine the best-fitting linear mixed effects model for 12 measured variables. The forward stepwise model selection is displayed in Table 4 & described in detail below. The estimates of the fixed effects of the best-fitting linear mixed effects models are displayed in Table 5.

- Highest VO_{2max} : ANOVA tests were conducted between the null model & the exercise group model ($P = 0.475$) & between the null model & the gender model ($P < 0.001$). Based on the significant p-value, an ANOVA test was conducted between the gender model & the exercise group x gender model ($P < 0.002$). Therefore, the exercise group x gender model best represents the effects of time & the group x gender interaction on the highest VO_{2max} .
- GXT VO_{2max} : ANOVA tests were conducted between the null model & the exercise group model ($P = 0.017$) & between the null model & the gender model ($P = 0.078$). Based on the significant p-value, an ANOVA test was conducted between exercise group model & the exercise group x gender model ($P < 0.004$). Therefore, the exercise group x gender model best represents the effects of time & the group x gender interaction on GXT VO_{2max} .
- VP VO_{2max} : ANOVA tests were conducted between the null model & the exercise group model ($P = 0.016$) & between the null model & the gender model ($P = 0.153$). Based on the significant p-value, an ANOVA test was conducted between exercise group model & the exercise group x gender model ($P < 0.004$). Therefore, the exercise group x gender model best represents the effects of time & the group x gender interaction on VP VO_{2max} .

- VP-GXT VO_{2max} : ANOVA tests were conducted between the null model & the exercise group model ($P = 0.998$) & between the null model & the gender model ($P = 0.069$). Without a significant p-value, an ANOVA test was conducted between the null model & the exercise group x gender model ($P = 0.321$). Therefore, no mixed effects model explains the results of VP-GXT VO_{2max} .
- Highest HR_{max} : ANOVA tests were conducted between the null model & the exercise group model ($P = 0.036$) & between the null model & the gender model ($P < 0.001$). Based on the significant p-value, an ANOVA test was conducted between the gender model & the exercise group x gender model ($P < 0.001$). Therefore, the exercise group x gender model best represents the effects of time & the group x gender interaction on HR_{max} values.
- Highest RER_{max} : ANOVA tests were conducted between the null model & the exercise group model ($P = 0.986$) & between the null model & the gender model ($P = 0.979$). Without a significant p-value, an ANOVA test was conducted between the null model & the exercise group x gender model ($P = 0.960$). Therefore, no mixed effects model explains the results of the RER_{max} values.
- GXT TTE: ANOVA tests were conducted between the null model & the exercise group model ($P = 0.450$) & between the null model & the gender group ($P < 0.001$). Based on the significant p-value, an ANOVA test was conducted between the gender model & the exercise group x gender model ($P = 0.864$). Therefore, the gender model best represents the effects of gender & time on GXT TTE values.
- VP TTE: ANOVA tests were conducted between the null model & the exercise group model ($P = 0.175$) & between the null model & the gender model ($P = 0.365$). Without a

significant p-value, an ANOVA test was conducted between the null model & the exercise group x gender model ($P = 0.454$). Therefore, no mixed effects model explains the results of the VP TTE values.

- Weight: ANOVA tests were conducted between the null model & the exercise group model ($P = 0.10$) & between the null model & the gender model ($P < 0.001$). Based on the significant p-value, an ANOVA test was conducted between the exercise group model & the exercise group x gender model ($P < 0.001$). Therefore, the exercise group x gender model best represents the effects of time & the group x gender interaction on weight.
- Body fat percentage: ANOVA tests were conducted between the null model & the exercise group model ($P = 0.267$) & between the null model & the gender model ($P = 0.732$). Without a significant p-value, an ANOVA test was conducted between the null model & the exercise group x gender model ($P = 0.332$). Therefore, no mixed effects model explains the results of the body fat percentage values.
- Fat mass: ANOVA tests were conducted between the null model & the exercise group model ($P = 0.058$) & between the null model & the gender model ($P = 0.061$). Without a significant p-value, an ANOVA test was conducted between the null model & the exercise group x gender model ($P = 0.040$). Therefore, the exercise group x gender model best represents the effects of time & the group x gender interaction on fat mass.
- Fat-free mass: ANOVA tests were conducted between the null model & the exercise group model ($P = 0.055$) & between the null model & the gender model ($P < 0.001$). Based on the significant p-value, an ANOVA test was conducted between the gender model & the exercise group x gender model ($P = 0.079$). Therefore, the gender model best represents the effects of gender & time on fat-free mass.

	Model	AIC	BIC	χ^2 (df)
Highest VO _{2max} (L*min ⁻¹)	Null	118.1	125.8	
	Exercise Group	119.6	129.9	
	Gender	95.7	105.9	24.47 (4)
	Exercise Group + Gender	97.7	110.5	
	Exercise Group * Gender	86.2	101.6	13.475 (6)
GXT VO _{2max} (ml*kg*min ⁻¹)	Null	654.2	661.9	
	Exercise Group	650.6	660.8	5.6602 (4)
	Gender	653.1	663.3	
	Exercise Group + Gender	650.4	663.2	
	Exercise Group * Gender	643.2	658.6	11.283 (6)
VP VO _{2max} (ml*kg*min ⁻¹)	Null	648.8	656.5	
	Exercise Group	645.0	655.2	5.8417 (4)
	Gender	648.8	659.0	
	Exercise Group + Gender	645.8	658.6	
	Exercise Group * Gender	637.7	653.1	11.218 (6)
VP-GXT VO _{2max} (L*min ⁻¹)	Null	-156.6	-148.9	
	Exercise Group	-154.6	-144.3	
	Gender	-157.9	-147.6	
	Exercise Group + Gender	-156.0	-143.1	
	Exercise Group * Gender	-154.0	-138.7	
Highest HR _{max} (bpm)	Null	742.4	750.1	
	Exercise Group	740.0	750.2	
	Gender	694.3	704.6	50.053 (4)
	Exercise Group + Gender	678.6	691.4	
	Exercise Group * Gender	675.9	691.3	22.41 (6)
Highest RER _{max}	Null	-219.8	-212.2	
	Exercise Group	-217.8	-207.6	
	Gender	-217.8	-207.6	
	Exercise Group + Gender	-215.8	-203.0	
	Exercise Group * Gender	-214.2	-198.8	
GXT TTE (min)	Null	335.9	343.6	
	Exercise Group	337.3	347.6	
	Gender	326.3	336.6	11.598 (4)
	Exercise Group + Gender	328.3	341.1	
	Exercise Group * Gender	330.0	345.4	
VP TTE (min)	Null	199.9	207.6	
	Exercise Group	200.1	210.4	
	Gender	201.1	211.4	
	Exercise Group + Gender	201.6	214.4	
	Exercise Group * Gender	203.3	218.7	
Weight (kg)	Null	877.4	885.1	
	Exercise Group	872.8	883.0	6.6204 (4)
	Gender	851.9	862.2	
	Exercise Group + Gender	849.6	862.4	
	Exercise Group * Gender	851.4	866.8	25.37 (6)
Body Fat (%)	Null	513.5	520.3	
	Exercise Group	514.3	523.4	
	Gender	515.4	524.5	
	Exercise Group + Gender	516.1	527.4	
	Exercise Group * Gender	516.1	529.7	
Fat Mass (kg)	Null	593.8	600.6	
	Exercise Group	592.2	601.2	
	Gender	592.3	601.3	
	Exercise Group + Gender	591.4	602.7	6.5254 (5)
	Exercise Group * Gender	592.9	606.4	
Fat-Free Mass (kg)	Null	538.29	545.1	
	Exercise Group	536.6	545.7	
	Gender	471.5	480.6	68.754 (4)
	Exercise Group + Gender	470.4	481.7	
	Exercise Group * Gender	470.5	484.0	

Table 4. Forward stepwise model selection using likelihood ratio tests of 12 variables. Best-fitting linear mixed effects are highlighted.

	Highest VO _{2max} (L*min ⁻¹)	GXT VO _{2max} (ml*kg ⁻¹ *min ⁻¹)	VP VO _{2max} (ml*kg ⁻¹ *min ⁻¹)	VP-GXT VO _{2max} (L*min ⁻¹)
Group	0.10146	4.545	4.621	
Gender	0.93000	1.614	2.506	
Group * Gender	-0.86896	-12.984	-13.230	
	Highest HR _{max} (bpm)	Highest RER _{max}	GXT TTE (min)	VP TTE (min)
Group	-5.958			
Gender	-19.375		-1.376	
Group * Gender	-10.917			
	Weight (kg)	Body Fat (%)	Fat Mass (kg)	Fat-Free Mass (kg)
Group	-8.979		-6.031	
Gender	30.203		9.346	25.589
Group * Gender	5.448			

Table 5. Estimates of fixed effects of best-fitting linear mixed effects models.

Discussion

The primary finding of this study was that 8 weeks of low-volume HIIT & 8 weeks of high-volume MICT led to increases in VO_{2max} that did not differ significantly between groups; there was no significant group X time interaction from week 4 to week 12 ($P = 0.755$). The results support the hypothesis that increases in VO_{2max} would not differ significantly between the two groups. The results are also consistent with the findings of several other comparative studies of HIIT & MICT (31,34).

Sawyer et al. (31) observed similar results to the current investigation. 18 obese participants (35.1 ± 8.1 yr; body mass index (BMI) = 36.0 ± 5.0 kg*m⁻²) were randomized into HIIT or MICT groups with similar exercise protocols as the current study. The researchers observed significant increases in VO_{2max} after 8 weeks of training for both HIIT (Baseline = 2.19 ± 0.65 L*min⁻¹, Week 8 = 2.64 ± 0.88 L*min⁻¹, $P = 0.01$) & MICT (Baseline = 2.24 ± 0.48 L*min⁻¹, Week 8 = 2.55 ± 0.61 L*min⁻¹, $P < 0.01$), but there was no group X time interaction (P

= 0.53). However, as opposed to the current investigation, Sawyer et al. utilized 8-week interventions without a 4-week conditioning period. Additionally, the MICT protocol involved exercising only 3 sessions/week (31). Furthermore, Shepherd et al. (34) also observed similar findings. 90 inactive participants (42 ± 11 yr; $\text{BMI} = 27.7 \pm 4.8 \text{ kg}\cdot\text{m}^{-2}$) were randomized into HIIT or MICT groups. The HIIT protocol consisted of 3 sessions/week with 15-60 second sprints on mechanically-braked spinning bikes with 45-120 seconds of active recovery; sessions lasted 18-25 min. The MICT protocol consisted of 3 sessions/week for 30-45 min at $\sim 70\%$ HR_{max} on the bikes. The researchers observed significant increases in $\text{VO}_{2\text{max}}$ after 10 weeks of training for both HIIT (Baseline = $2.50 \pm 0.77 \text{ L}\cdot\text{min}^{-1}$, Week 10 = $2.71 \pm 0.78 \text{ L}\cdot\text{min}^{-1}$, $P < 0.001$) & MICT (Baseline = $2.43 \pm 0.70 \text{ L}\cdot\text{min}^{-1}$, Week 10 = $2.59 \pm 0.75 \text{ L}\cdot\text{min}^{-1}$, $P < 0.001$). No group X time interaction was observed ($P = 0.33$) (34).

Although the aforementioned studies support the results found in the current investigation, several meta-analyses do not. For example, a meta-analysis by Milanovic et al. (19) in 2015 examined 28 studies with 723 total participants (25.1 ± 5 yr; relative $\text{VO}_{2\text{max}} = 40.8 \pm 7.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) to determine the effectiveness of HIIT interventions on CRF. The researchers observed an increase of $5.5 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: $\pm 1.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) after a HIIT intervention & an increase of $4.9 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$ (95% CI: $\pm 1.4 \text{ mL}\cdot\text{min}^{-1}\cdot\text{kg}^{-1}$) after a traditional endurance training intervention. Thus, Milanovic et al. found a small beneficial effect of HIIT on relative $\text{VO}_{2\text{max}}$ of $1.2 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (95% CI: $\pm 0.9 \text{ mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) in comparison to traditional endurance training, indicating it is likely a superior method at improving CRF over MICT (19). Moreover, an additional meta-analysis by Weston et al. (36) also observed greater increases in $\text{VO}_{2\text{max}}$ following HIIT protocol over MICT protocols. The analysis utilized 10 studies with 273 patients with chronic diseases including coronary heart disease, heart failure,

obesity, metabolic syndrome, & hypertension. The researchers observed a 19.4% increase in relative $\text{VO}_{2\text{max}}$ from 22.5 to 27.9 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ after HIIT & a 10.3% increase from 22.6 to 25.2 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ after MICT. The mean difference in change in $\text{VO}_{2\text{max}}$ between HIIT & MICT was 3.03 $\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$ (36).

Helgerud et al. (15) led a randomized controlled trial in which the researchers also concluded that HIIT significantly increased $\text{VO}_{2\text{max}}$ more than MICT. 40 males (24.6 ± 3.8 yr; weight = 82.0 ± 12.0 kg) were equally randomized into 4 exercise protocols, including long slow distance running (MICT), lactate threshold running, 15/15 interval running, & 4 x 4-min interval running (HIIT). The long slow distance running protocol consisted of continuous running at 70% HR_{max} for 45 min & 3 days/week for 8 weeks. The 4 x 4-min interval running protocol consisted of running for 4 4-min intervals at 90-95% HR_{max} with 3-min of active recovery at 70% HR_{max} between each interval for 3 days/week for 8 weeks. After the HIIT protocol, $\text{VO}_{2\text{max}}$ increased by 7.2% (baseline = 4.56 ± 0.62 $\text{L}\cdot\text{min}^{-1}$; week 8 = 4.89 ± 0.52 $\text{L}\cdot\text{min}^{-1}$; $P < 0.05$); on the contrary, after the MICT protocol, $\text{VO}_{2\text{max}}$ did not increase significantly (baseline = 4.77 ± 0.49 $\text{L}\cdot\text{min}^{-1}$; week 8 = 4.74 ± 0.46 $\text{L}\cdot\text{min}^{-1}$; $P > 0.05$) (15). Thus, as opposed to the current investigation, Helgerud & co-investigators observed a significant increase in $\text{VO}_{2\text{max}}$ only after the HIIT intervention. However, only men who participated in leisure activity or endurance training at least 3 times per week enrolled in the study, indicating greater fitness than the sedentary adults in this investigation; thus, the MICT intervention may not induce significant increases in $\text{VO}_{2\text{max}}$ due to the increased experience & training level of the participants. Additionally, the HIIT protocol is greater in duration than the HIIT protocol utilized in the current study (25 total min/session vs. 20 total min/session).

In addition to 2-way ANOVA tests, linear mixed-effects models were utilized to determine best-fitting models for several measured variables, as displayed in Tables 4 & 5. The models examined the impact of gender, exercise group, exercise group X gender interaction, & time effect on the variables. Importantly, the linear mixed-effects model with an exercise group X gender interaction & time effect best explained the changes in highest $\text{VO}_{2\text{max}}$ ($\text{L}\cdot\text{min}^{-1}$), GXT $\text{VO}_{2\text{max}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$) VP $\text{VO}_{2\text{max}}$ ($\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}$), highest HR_{max} (bpm), & weight (kg). However, the models did not effectively explain changes in several other variables. Such results are likely due to the inclusion of 3 males; consequently, the effect of gender on variables is increasingly difficult to determine. Furthermore, many variables did not change over time.

The investigation had several strengths. For example, the participants completed GXTs with verification phases every 4 weeks, reassessing the participants' $\text{VO}_{2\text{max}}$ & HR_{max} values. Thus, HR-ranges for the exercise prescriptions were consistently adjusted. Additionally, adherence to exercise sessions was 100%. All 24 participants completed all exercise sessions. Research technicians supervised each exercise session & monitored the participants' HR, ensuring exercise was completed at the correct intensity & volume. The study also used a verification phase after the GXT to enhance the determination of $\text{VO}_{2\text{max}}$ (24). Use of the Cosmed BodPod to assess body composition via air displacement plethysmography is another strength due to the reliability & accuracy of the equipment in comparison to other common tools for body composition assessments.

A limitation of the study is the disparity in gender. Only 3 males participated in the study, reducing the applicability of the study's results to the general population. Inclusion of more males or removal of the 3 males from statistical analysis would improve the strength of the investigation's results & conclusions. The study did not have a sedentary control group, as well;

however, a lack of a sedentary control group is common in comparative studies of HIIT & MICT (13,31,34).

In conclusion, low-volume HIIT & high-volume MICT produced similar increases in VO_{2max} , as evident with no group X time interaction ($P = 0.755$). However, a significant time effect ($P = 0.019$) indicates both HIIT & MICT are capable of improving CRF in sedentary adults after 12 weeks of exercise. The time commitment for 8 weeks of low-volume HIIT (480 total min) was 60% less than the time commitment for 8 weeks of high-volume MICT (1200 total min); therefore, low-volume HIIT is a more time-efficient strategy for improving VO_{2max} than high-volume MICT. Previous literature observes a lack of time as the largest barrier to exercise in non-exercising college students & adults aged 18-64 (10). Thus, a time-efficient exercise intervention, such as low-volume HIIT in the current investigation, provides a reasonable solution to exercising with a lack of time.

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