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$_{2\text{max}}$) in sedentary adults. We hypothesized that increases in VO$_{2\text{max}}$ after both interventions would not significantly differ between groups. 24 participants (27.1 ± 8.4 yr, VO$_{2\text{max}}$ = 27.7 ± 6.7 ml*kg$^{-1}$*min$^{-1}$) completed 4 wk (3 sessions/wk) of MICT (30 min, 70-75% HR$_{\text{max}}$). 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$_{\text{max}}$, 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$_{\text{max}}$). VO$_{2\text{max}}$ 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$_{2\text{max}}$ 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$_{2\text{max}}$ 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$_{2\text{max}}$ 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$_{2\text{max}}$ 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\textsubscript{2max}

Maximum oxygen uptake (VO\textsubscript{2max}) is defined as the maximal amount of oxygen that can be consumed by the body during maximal exercise. VO\textsubscript{2max} assesses the body’s ability via the pulmonary, cardiovascular, & skeletal muscle systems to uptake, transport, & utilize O\textsubscript{2} in contracting muscle mitochondria at maximal exercise (24). Hence, measuring VO\textsubscript{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 mL*kg\textsuperscript{-1}*min\textsuperscript{-1}) increase in VO\textsubscript{2max} has been associated with a 13% reduction in risk of all-cause mortality & a 15% reduction in risk of CVD (18). Furthermore, VO\textsubscript{2max} is used in the development of exercise prescriptions for athletes, patients, & clients. Exercise programs may utilize percentages of VO\textsubscript{2max} to determine intensity of exercise sessions. In research settings, changes in VO\textsubscript{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\textsubscript{2max}

VO\textsubscript{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\textsubscript{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 VCO₂/VO₂ 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 VO₂max. 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\textsubscript{max} (220 – 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\textsubscript{2} plateau; thus, another method is required.

Although secondary criteria are utilized commonly to determine if a VO\textsubscript{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\textsubscript{2}\textsubscript{max} 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\textsubscript{2}\textsubscript{max}: Fick Equation**

VO\textsubscript{2} is defined via the Fick Equation, which states that VO\textsubscript{2} (ml*kg\textsuperscript{-1}*min\textsuperscript{-1}) is equal to the product of cardiac output [CO (L*min\textsuperscript{-1})] & the arteriovenous difference in oxygen concentration [a-v\textsubscript{O\textsubscript{2}}\textsubscript{diff} (mL*L\textsuperscript{-1})] (6):

1. \[\text{VO}_2 = \text{CO} \times \text{a-vO}_2\text{diff}\]

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

2. \[\text{VO}_2 = [\text{HR} \times (\text{EDV} - \text{ESV})] \times (\text{CaO}_2 - \text{CvO}_2)\]
In order to alter VO$_{2\text{max}}$, 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$_{2\text{max}}$ 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$_{2\text{max}}$. VO$_{2\text{max}}$ 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 O$_2$ 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$_{2\text{max}}$ 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$_{2\text{max}}$ 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; $P < 0.05$) & in 8 younger men (pre: 3.82 ± 0.47; 3 weeks: 4.27 ± 0.52 L/min; $P < 0.05$) (22).

**Changing VO$_{2\text{max}}$: 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$_{2\text{max}}$, blood volume, stroke volume, heart rate, & cardiac output. Mujika et al. (21) report that decreases in VO$_{2\text{max}}$ 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$_{2\text{max}}$ after only 12 days of cessation ($P < 0.05$); ultimately, after 56 days, VO$_{2\text{max}}$ 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$_{\text{max}}$ of 4, 5, 6 & 5% at 12, 21, 56, & 84 days, respectively (8). As a consequence of the changes in HR$_{\text{max}}$ & SV$_{\text{max}}$, CO$_{\text{max}}$ has also been observed to decrease after training cessation (21). In the study by
Coyle et al. (8), CO\textsubscript{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 VO\textsubscript{2max}**

Several variables may act as the limiting factor for VO\textsubscript{2max}. Central factors consist of pulmonary diffusing capacity, CO\textsubscript{max}, & the oxygen carrying capacity of the blood (4). Pulmonary diffusing capacity is typically not the limiter of VO\textsubscript{2max} 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\textsubscript{2} desaturation & become a limiting factor of VO\textsubscript{2max} (4,9). To combat this limitation, an increase in O\textsubscript{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 VO\textsubscript{2max}. However, because HR\textsubscript{max} does not change significantly after exercise training, change in SV\textsubscript{max} is the main determinant of CO\textsubscript{max}. In a study by Saltin et al. (28), VO\textsubscript{2max} decreased after 20 days of bed rest & increased after 50 days of training, with the majority of the change in VO\textsubscript{2max} attributable to alterations in SV, emphasizing that SV is often the limiter of VO\textsubscript{2max}. The oxygen carrying capacity of blood may also act as a central limiter of VO\textsubscript{2max}. 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, VO$_{2\text{max}}$ may improve by 4-9% indicating that the oxygen-transport capacity of the blood may limit VO$_{2\text{max}}$ (4).

In addition to central factors, peripheral factors may also act as the limiting variable of 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 VO$_{2\text{max}}$, indicating that it is not a major limiter of 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 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 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 VO$_{2\text{max}}$ when measured via cycle ergometry. Therefore, increasing mitochondrial enzyme activity & capillary density may decrease CvO$_2$, increase a-Vo$_2$diff, & increase VO$_{2\text{max}}$, but tend not to be limiting factors of VO$_{2\text{max}}$ (4).

As aforementioned, limiting factors of 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 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 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 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 VO₂max in the untrained participants; thus, mitochondrial capacity limits VO₂max in untrained individuals. The limiting factors of VO₂max 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 VO₂max (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 VO₂max 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 VO₂max vary based on the training level of the individual, altitude, & O₂ saturation of the environment. VO₂max in untrained individuals may be limited due to mitochondrial O₂ demand or SV; in highly trained athletes, VO₂max 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 VO₂max to protect the body from physiological failure.
**Changing VO\textsubscript{2max}: Exercise Interventions**

Exercise interventions are utilized to increase VO\textsubscript{2max} in individuals. The intensity of exercise is a significant factor at determining the effectiveness of the protocol at raising VO\textsubscript{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\textsubscript{2} reserve), a vigorous-intensity group (75% VO\textsubscript{2} reserve), or near-maximal-intensity group (95% VO\textsubscript{2} reserve). Exercise volume & energy expenditure were controlled by varying duration & frequency. VO\textsubscript{2max} increased significantly ($P < 0.05$) by 3.4, 4.8, & 7.2 mL*kg\textsuperscript{-1}*min\textsuperscript{-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\textsubscript{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\textsubscript{2max}. In an additional RCT, Ross et al. (27) examined the separate effects of intensity & amount of exercise on VO\textsubscript{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\textsubscript{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\textsubscript{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\textsubscript{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$_{2\text{max}}$, altering intensity & volume simultaneously may not lead to significantly different increases in VO$_{2\text{max}}$ 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$_{2\text{max}}$); 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$_{2\text{max}}$ between groups after training (1: $+0.29 \pm 0.15 \text{L*min}^{-1}$; 2: $+0.26 \pm 0.10 \text{L*min}^{-1}$; 3: $+0.35 \pm 0.17 \text{L*min}^{-1}$) (32). Scribbans et al. (32) concluded that similar increases in VO$_{2\text{max}}$ 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$_{2\text{max}}$ 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$_{\text{HV}}$), or periodized interval training (PER) in 39 men & women (22.9 ± 5.4 yr, 39.6 ± 5.6 mL*kg$^{-1}$*min$^{-1}$) in comparison to 32 non-exercising controls (25.7 ± 4.5 yr, 40.7 ± 5.2 mL*kg$^{-1}$*min$^{-1}$). All 3 HIIT protocols led to significant increases in VO$_{2\text{max}}$ ($P < 0.001$) 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$_{2\text{max}}$, as well. In the 334 participants, a significant increase in VO$_{2\text{max}}$ of 0.51 L*min$^{-1}$ (95% CI: 0.43 to 0.60 L*min$^{-1}$) was observed. Of those 37 studies, 9 used longer intervals and led to a greater increase of approximately 0.80-0.90 L*min$^{-1}$ in VO$_{2\text{max}}$ (3).

Furthermore, another 2016 meta-analysis by Batacan et al. (5) examined 65 intervention studies to determine the effects of HIIT on VO$_{2\text{max}}$ & cardiometabolic health. Batacan et al. observed
that short-term HIIT protocols (< 12 weeks) increased VO\(_{2}\text{max}\) 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 (≥ 12 weeks) increased VO\(_{2}\text{max}\) 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\(_{2}\text{max}\) (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\(_{2}\text{max}\). They observed an increase of 4.9 mL*kg\(^{-1}\)*min\(^{-1}\) (95% CI: 3.5 to 6.3 mL*kg\(^{-1}\)*min\(^{-1}\)) following a continuous endurance training program. However, they also observed an increase of 5.5 mL*kg\(^{-1}\)*min\(^{-1}\) (95% CI: 4.3 to 6.7 mL*kg\(^{-1}\)*min\(^{-1}\)) following a HIIT program. Thus, both types of protocols induced increases in VO\(_{2}\text{max}\), yet HIIT led to greater gains (19). Another meta-analysis by Weston et al. (36) examined the effects of HIIT & MICT protocols on VO\(_{2}\text{max}\) 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\(_{2}\text{max}\) following a HIIT protocol than a MICT protocol with a mean difference of 3.03 mL*kg\(^{-1}\)*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\(_{2}\text{max}\) significantly increased by 4.4 ± 1.2
mL*kg\(^{-1}\)*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 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 VO\(_{2\text{max}}\). 46 participants followed a HIIT protocol with repeated sprints (15-60s, > 90% HR\(_{\text{max}}\)) & active recovery (≤ 25 min/session, 3 sessions/week). 44 participants followed a MICT protocol with continuous cycling (~70% HR\(_{\text{max}}\), 30-45 min/session, 5 sessions/week) (34). VO\(_{2\text{max}}\) increased by a mean difference of 2.8 mL*kg\(^{-1}\)*min\(^{-1}\) (95% CI: 2.0 to 3.6) after the HIIT protocol; VO\(_{2\text{max}}\) increased by a mean difference of 2.4 mL*kg\(^{-1}\)*min\(^{-1}\) (95% CI: 1.7 to 3.3) after the MICT protocol. Thus, the protocols did not lead to significantly different increases in 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 VO\(_{2\text{max}}\) in 17 obese participants (BMI ≥ 30 kg/m\(^2\)) following HIIT & MICT protocols. 8 participants completed 8 weeks of HIIT (10 1-min intervals at 90-95% HR\(_{\text{max}}\) 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\(_{\text{max}}\)). In the HIIT group, VO\(_{2\text{max}}\) increased significantly \((P = 0.01)\) from 2.19 ± 0.65 L/min to 2.64 ± 0.88 L/min; in the MICT group, VO\(_{2\text{max}}\) increased significantly \((P < 0.01)\) from 2.24 ± 0.48 L/min to 2.55 ± 0.61 L/min (31). Therefore, HIIT & MICT interventions may induce similar increases in VO\(_{2\text{max}}\) in individuals.

When examining the general effects of aerobic exercise interventions on VO\(_{2\text{max}}\), the aforementioned studies show that greater intensity & greater volume of aerobic exercise may lead to more significant increases in VO\(_{2\text{max}}\) (14,26); however, no significant difference in increases in 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$_{2\text{max}}$ (2,3,5) & that HIIT is more effective than MICT (17,19,36, yet several studies observed no significant difference between increases in VO$_{2\text{max}}$ following HIIT & MICT protocols (31,34).
Introduction

Maximum oxygen uptake (VO\textsubscript{2max}) is defined as the maximal amount of oxygen that can be consumed by the body during maximal exercise. VO\textsubscript{2max} assesses the body’s ability via the pulmonary, cardiovascular, & skeletal muscle systems to uptake, transport, & utilize O\textsubscript{2} in contracting muscle mitochondria at maximal exercise (24). Hence, measuring VO\textsubscript{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 mL*kg\textsuperscript{-1}*min\textsuperscript{-1}) increase in VO\textsubscript{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\textsubscript{2max} is critical to improve the health of individuals at risk of CVD & other chronic diseases.

Aerobic exercise interventions can be used to increased CRF. Specifically, high-intensity interval training (HIIT) & moderate-intensity continuous training (MICT) are two common interventions utilized to improve VO\textsubscript{2max}. Generally, previous literature has shown that HIIT induces a more significant increase in VO\textsubscript{2max} than MICT (17,19,36). However, other studies have observed similar increases in VO\textsubscript{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\textsubscript{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\textsubscript{2max} in sedentary adults. We
hypothesized that 8 weeks of low-volume HIIT would not improve VO2max 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.

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Weight (kg)</th>
<th>Height (m)</th>
<th>Body Fat (%)</th>
<th>Fat Mass (kg)</th>
<th>Fat-Free Mass (kg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>27.1 ± 8.4</td>
<td>75.3 ± 23.4</td>
<td>1.71 ± 0.10</td>
<td>31.6 ± 9.2</td>
<td>25.1 ± 15.5</td>
<td>50.3 ± 11.4</td>
</tr>
</tbody>
</table>

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\textsubscript{max} (220 – age = predicted HR\textsubscript{max}) for 20 minutes if necessary. Exercising at 70% of HR\textsubscript{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$_{2\text{peak}}$ was estimated utilizing a non-exercise VO$_{2\text{peak}}$ prediction equation determined by Jackson et al. (16). The equation states:

$$\text{VO}_2\text{peak} (\text{mL}\cdot\text{kg}^{-1}\cdot\text{min}^{-1}) = 56.363 + 1.921*(\text{PA-R}) - 0.381*(\text{Age}) - 0.754*(\text{BMI}) + 10.987*(F = 0, M = 1)$$

Thus, the estimated VO$_{2\text{peak}}$ 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$_{2\text{max}}$ (33). Final results utilized averages of the two highest consecutive 15-second recordings. The VO$_{2\text{max}}$ value recorded at each time point was the highest average VO$_{2\text{max}}$ 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$_{\text{max}}$ as calculated utilizing the HR$_{\text{max}}$ 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\textsubscript{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\textsubscript{max}. HR ranges were calculated according to the participant’s most recent GXT.

Statistical Analysis:

Descriptive statistics (means ± 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\textsubscript{2max} & GXT VO\textsubscript{2max} (VP-GXT VO\textsubscript{2max}), as well as VP TTE & VP-GXT VO\textsubscript{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$_{2\text{max}}$ = 27.7 ± 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$_{2\text{max}}$ = 29.0 ± 6.0 ml*kg$^{-1}$*min$^{-1}$) were randomized into the HIIT group. 11 participants (26 ± 6.9 yr, VO$_{2\text{max}}$ = 26.2 ± 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$_{2\text{max}}$ values were determined as the highest VO$_{2\text{max}}$ attained in the GXT or VP. In all participants, VO$_{2\text{max}}$ 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$_{2\text{max}}$ 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$_{2\text{max}}$ did increase significantly from week 4 to week 12 ($P < 0.001$), as well. In the MICT group, VO$_{2\text{max}}$ 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$_{2\text{max}}$ did increase significantly from week 4 to week 12 ($P = 0.010$), as well. Changes in VO$_{2\text{max}}$ in HIIT & MICT from baseline to week 12 are displayed in Figure 2. There
was no group X time interaction for changes in VO$_{2\text{max}}$ from baseline to week 12 ($P = 0.878$) or from week 4 to week 12 ($P = 0.755$)

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

The VP-GXT VO$_{2\text{max}}$ values from baseline to week 12 are displayed in Figure 3. The VP-GXT VO$_{2\text{max}}$ values did not change significantly in the HIIT or MICT groups (HIIT: Baseline = 0.0 ± 0.11 L*min$^{-1}$, Week 12 = -0.07 ± 0.09 L*min$^{-1}$, $P = 0.401$; MICT: Baseline = -0.1 ± 0.10 L*min$^{-1}$, Week 12 = -0.03 ± 0.07 L*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$_{2\text{max}}$ & GXT TTE ($R = -0.2167$), as well as VP-GXT VO$_{2\text{max}}$ & VP TTE ($R = 0.3636$).
HIIT (n = 13) | MICT (n = 11) | Time Effect | Group X Time Interaction P value
---|---|---|---
Baseline | Wk 4 | Wk 8 | Wk 12 | Baseline | Wk 4 | Wk 8 | Wk 12 | P value

### Highest VO_{2max} (L·min⁻¹)

| Baseline | 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)

| Baseline | 186 ± 10 | 182 ± 12 | 181 ± 11 | 182 ± 14 | 0.385 | 188 ± 12 | 187 ± 11 | 187 ± 10 | 0.698 | 0.739 |
| 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 ± 0.6 | 2.5 ± 0.5 | 2.2 ± 0.5 | 0.455 | 0.228 |

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 ± SD VO$_{2\text{max}}$ in HIIT, MICT, & both over 12 weeks of training. *Significant time effect from baseline to week 12 ($P = 0.0182$); no group x time interaction ($P < 0.878$).

Figure 3. Mean ± SD Verification phase VO$_{2\text{max}}$ – GXT VO$_{2\text{max}}$ in HIIT & MICT over 12 weeks of training. No time effect from baseline to week 12 ($P = 0.592$); no group x 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 ± 6.6%, Week 12 = 30.8 ± 7.5%, *P* = 0.952; MICT: Baseline = 32.6 ± 11.8%, Week 12 = 34.5 ± 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).

<table>
<thead>
<tr>
<th></th>
<th>HIIT (n = 13)</th>
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<td>Baseline</td>
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<td>Wk 8</td>
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<td>Fat-Free Mass (kg)</td>
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<td>48.1±7.5</td>
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Table 3. Mean ± 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 ± SD Weight in HIIT, MICT, & both over 12 weeks of training. No time effect from baseline to week 12 ($P = 0.990$); no group x time interaction ($P = 0.951$).

Figure 5. Mean ± 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 x 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$_{2\text{max}}$:** 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$_{2\text{max}}$.

- **GXT VO$_{2\text{max}}$:** 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$_{2\text{max}}$.

- **VP VO$_{2\text{max}}$:** 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$_{2\text{max}}$. 
• **VP-GXT VO\textsubscript{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\textsubscript{2max}.

• **Highest HR\textsubscript{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\textsubscript{max} values.

• **Highest RER\textsubscript{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\textsubscript{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.
Table 4. Forward stepwise model section using likelihood ratio tests of 12 variables. Best-fitting linear mixed effects are highlighted.
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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$_{2\text{max}}$ 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$_{2\text{max}}$ 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$_{2\text{max}}$ 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; BMI = 27.7 ± 4.8 kg*m²) 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 ~70% HR\(_{\text{max}}\) on the bikes. The researchers observed significant increases in VO\(_{\text{2max}}\) after 10 weeks of training for both HIIT (Baseline = 2.50 ± 0.77 L*min\(^{-1}\), Week 10 = 2.71 ± 0.78 L*min\(^{-1}\), \(P < 0.001\)) & MICT (Baseline = 2.43 ± 0.70 L*min\(^{-1}\), Week 10 = 2.59 ± 0.75 L*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 VO\(_{\text{2max}}\) = 40.8 ± 7.9 mL*kg\(^{-1}\)*min\(^{-1}\)) to determine the effectiveness of HIIT interventions on CRF. The researchers observed an increase of 5.5 mL*kg\(^{-1}\)*min\(^{-1}\) (95% CI: ± 1.2 mL*kg\(^{-1}\)*min\(^{-1}\)) after a HIIT intervention & an increase of 4.9 mL*min\(^{-1}\)*kg\(^{-1}\) (95% CI: ± 1.4 mL*min\(^{-1}\)*kg\(^{-1}\)) after a traditional endurance training intervention. Thus, Milanovic et al. found a small beneficial effect of HIIT on relative VO\(_{2\text{max}}\) of 1.2 mL*kg\(^{-1}\)*min\(^{-1}\) (95% CI: ± 0.9 mL*kg\(^{-1}\)*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 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 VO\textsubscript{2max} from 22.5 to 27.9 mL*kg\textsuperscript{-1}*min\textsuperscript{-1} after HIIT & a 10.3% increase from 22.6 to 25.2 mL*kg\textsuperscript{-1}*min\textsuperscript{-1} after MICT. The mean difference in change in VO\textsubscript{2max} between HIIT & MICT was 3.03 mL*kg\textsuperscript{-1}*min\textsuperscript{-1} (36).

Helgerud et al. (15) led a randomized controlled trial in which the researchers also concluded that HIIT significantly increased VO\textsubscript{2max} 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\textsubscript{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\textsubscript{max} with 3-min of active recovery at 70% HR\textsubscript{max} between each interval for 3 days/week for 8 weeks. After the HIIT protocol, VO\textsubscript{2max} increased by 7.2% (baseline = 4.56 ± 0.62 L*min\textsuperscript{-1}; week 8 = 4.89 ± 0.52 L*min\textsuperscript{-1}; P < 0.05); on the contrary, after the MICT protocol, VO\textsubscript{2max} did not increase significantly (baseline = 4.77 ± 0.49 L*min\textsuperscript{-1}; week 8 = 4.74 ± 0.46 L*min\textsuperscript{-1}; P > 0.05) (15). Thus, as opposed to the current investigation, Helgerud & co-investigators observed a significant increase in VO\textsubscript{2max} 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 VO\textsubscript{2max} 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 VO$_{2\text{max}}$ (L*min$^{-1}$), GXT VO$_{2\text{max}}$ (mL*kg$^{-1}$*min$^{-1}$) VP VO$_{2\text{max}}$ (mL*kg$^{-1}$*min$^{-1}$), highest HR$_{\text{max}}$ (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’ VO$_{2\text{max}}$ & HR$_{\text{max}}$ 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 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$_{2\text{max}}$, 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$_{2\text{max}}$ 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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