Low-Volume, High-Intensity Interval Training in Patients with CAD

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ABSTRACT

CURRIE, K. D., J. B. DUBBERLEY, R. S. MCKELVIE, and M. J. MACDONALD. Low-Volume, High-Intensity Interval Training in Patients with CAD. Med. Sci. Sports Exerc., Vol. 45, No. 8, pp. 1436–1442, 2013. Purpose: Isocaloric interval exercise training programs have been shown to elicit improvements in numerous physiological indices in patients with CAD. Low-volume high-intensity interval exercise training (HIT) is effective in healthy populations; however, its effectiveness in cardiac rehabilitation has not been established. This study compared the effects of 12 wk of HIT and higher-volume moderate-intensity endurance exercise (END) on brachial artery flow-mediated dilation (FMD) and cardiorespiratory fitness (VO2peak) in patients with CAD. Methods: Twenty-two patients with documented CAD were randomized into HIT (n = 11) or END (n = 11) based on pretraining FMD. Both groups attended two supervised sessions per week for 12 wk. END performed 30–50 min of continuous cycling at 58% peak power output (PPO), whereas HIT performed ten 1-min intervals at 89% PPO separated by 1-min intervals at 10% PPO per session. Results: Relative FMD was increased posttraining (END, 4.4% ± 2.6% vs 5.9% ± 3.6%; HIT, 4.6% ± 3.6% vs 6.1% ± 3.4%, P < 0.001 pre- vs posttraining) with no differences between groups. A training effect was also observed for relative VO2peak (END, 18.7 ± 5.7 vs 22.3 ± 6.1 mL·kg⁻¹·min⁻¹; HIT, 19.8 ± 3.7 vs 24.5 ± 4.5 mL·kg⁻¹·min⁻¹, P < 0.001 for pre- vs posttraining), with no group differences. Conclusions: Low-volume HIT provides an alternative to the current, more time-intensive prescription for cardiac rehabilitation. HIT elicited similar improvements in fitness and FMD as END, despite differences in exercise duration and intensity. Key Words: FLOW-MEDIATED DILATION, CARDIORESPIRATORY FITNESS, HEART RATE, BLOOD PRESSURE, REHABILITATION

Interval training has gained considerable attention as a suitable exercise program for patients with cardiovascular diseases including CAD and heart failure (2,5,10). Investigations using various types of high-intensity interval protocols in CAD patients demonstrate training-induced improvements in numerous physiological indices (19–21,27,35,36). In addition, when compared with the current exercise program used in cardiac rehabilitation of high-volume moderate-intensity endurance exercise (END), interval exercise has been shown to elicit superior improvements in indices of cardiorespiratory fitness (27,35,36) and endothelial function (36). These previous investigations, however, used isocaloric protocols, where the energy expenditure of the interval bout was matched to that of the END bout. No studies have examined the effectiveness of an interval protocol not matched to END for energy expenditure in patients with CAD.

Low-volume interval protocols, which are not matched for energy expenditure, are time-efficient strategies that have been shown to be effective in healthy populations (13,26). Given that “lack of time” is one of the commonly cited barriers to exercise adherence in cardiac rehabilitation (3), a low-volume, high-intensity interval protocol may be a superior treatment strategy in terms of adherence if the resultant physiological benefits are comparable. The purpose of this study was to compare the effects of 12 wk of END and low-volume high-intensity interval exercise training (HIT) on endothelial function and cardiorespiratory fitness in patients with CAD. These indices were chosen for examination because they are related to future risk of mortality (17,22). On the basis of evidence from investigations in healthy populations, it was hypothesized that the effects of HIT would be comparable with those found with END for the indices examined.

METHODS

Patients. Patients were recruited on entry to a phase II outpatient cardiac rehabilitation program and therefore were not undergoing previous exercise training. Inclusion criteria included a recent CAD event that was defined as the patient having at least one of the following: angiographically documented stenosis ≥50% in at least one major coronary artery; myocardial infarction, percutaneous coronary intervention, or...
coronary artery bypass graft surgery; and positive exercise stress test determined by a positive nuclear scan or symptoms of chest discomfort accompanied by ECG changes of >1 mm horizontal or down sloping ST-segment depression. Twenty-seven men and three women with documented CAD were recruited from the Cardiac Health and Rehabilitation Centre at the Hamilton Health Sciences General Site (Ontario, Canada). Three men and one woman dropped out due to reasons unrelated to the exercise interventions. Three patients had changes to their beta-blockers, and one patient was put on a calcium channel blocker during the study period. Therefore, 22 patients were included in the study. Exclusion criteria included smoking within 3 months, noncardiac surgical procedure within 2 months, myocardial infarction or coronary artery bypass graft within 2 months, percutaneous coronary intervention within 1 month, New York Heart Association class II-IV symptoms of heart failure, documented valve stenosis, documented severe chronic obstructive pulmonary disease, symptomatic peripheral arterial disease, unstable angina, uncontrolled hypertension, uncontrolled atrial arrhythmia or ventricular dysrhythmia, and any musculoskeletal abnormality that would limit exercise participation. The study protocol was reviewed and approved by the Hamilton Health Sciences/Faculty of Health Sciences Research Ethics Board, conforming to the Declaration of Helsinki on the use of human subjects, and written informed consent was obtained from patients before participation. Participant characteristics, medical history, and medications are presented in Table 1.

**Study protocol.** This study used a factorial repeated-measure design. Brachial artery endothelial-dependent function, which was assessed using flow-mediated dilation (FMD), was the primary outcome. Therefore, to ensure equivalent FMD values between exercise groups, patients were randomized into END or HIT after pretraining assessments, based on their relative FMD. Assessments were performed at baseline (pretraining) and after 12 wk of exercise training (posttraining). Timing of sessions was different between participants, but within-subject sessions were scheduled at the same time of day. Before testing sessions, participants were instructed to fast for at least 8 h; to abstain from exercise for 24 h, caffeine and alcohol consumption for 12 h; and to take all medications and vitamins as usual, except for nitroglycerin (NTG), which was withheld on testing days. All testing was performed in a temperature-controlled room (23.1°C ± 1.2°C).

Height (cm) and weight (kg) were measured, and body mass index was calculated. Seated blood pressure was measured in triplicate using an automated oscillometric device (Dinamap Pro 100; Critikon LCC, Tampa, FL) after 10 min of quiet rest. The first was considered a calibration measure, so brachial blood pressure was determined from the average of the second and third measures (25). Heart rate was measured throughout the testing session using a single-lead (CC5) ECG (model ML 123; ADInstruments Inc., Colorado Springs, CO) and is reported as the average value from a 5-min sample collected after 10-min of supine rest.

**Cardiorespiratory fitness assessment.** Fitness was assessed using a medically supervised graded exercise test to exhaustion on a cycle ergometer (Ergoline, Bitz, Germany). Heart rate was monitored throughout the test using a 12-lead ECG (MAC 5500; General Electric, Freiburg, Germany). After a brief warm up, participants cycled at approximately 70 rpm for 1 min at a workload of 100 kpm. After the first minute, the workload was increased 100 kpm every minute until exhaustion. Expired gas was analyzed using a semi-automated metabolic cart (Vmax 229; SensorMedics Corporation, Yorba Linda, CA), and oxygen consumption was determined at peak (\(\text{VO}_{2\text{peak}}\)) and anaerobic threshold (respiratory quotient = 1.0) from breath-by-breath samples averaged for 20 s. One participant did not complete the posttraining test due to a musculoskeletal injury (unrelated to the exercise training); therefore, analysis was performed on 21 patients (END = 10, HIT = 11).

**Brachial artery assessments.** All brachial artery endothelial-dependent function assessments were conducted in the supine position after 20 min of quiet rest. Brachial artery endothelial-dependent function was assessed using the FMD test, based on previously established guidelines (6,32). Duplex ultrasound (Vivid Q; GE Healthcare, Horten, Norway) was used to capture simultaneous images of the right brachial artery (13 MHz) at a frame rate of 7.7 frames per second and blood velocity measurements (4 MHz) throughout the FMD protocol. Baseline preclosure images and velocities were collected 3–5 cm proximal to the antecubial fossa for 30 s. A pneumatic cuff positioned on the forearm distal to the antecubial fossa was inflated using a rapid cuff inflator (model E20 and AG101; Hokanson, Bellevue, WA) to an occlusion pressure of 200 mm Hg. After 5 min of occlusion, the cuff was released and duplex postocclusion images, and velocities were collected for 3 min.

<table>
<thead>
<tr>
<th>TABLE 1. Participant characteristics and exercise training data.</th>
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<td><strong>Variable</strong></td>
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<td>Height (m)</td>
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<td>Weight (kg)</td>
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<td>BMI (kg/m(^2))</td>
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<td>CAD criteria (n)</td>
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<td>MI</td>
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<td>PCI</td>
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<td>CABG</td>
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<td>Time since CAD event (d)</td>
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<td>Calcium channel blockers</td>
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<td>Mean work/session (kJ)</td>
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<td>Mean heart rate (bpm)</td>
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<td>Attendance per 24 sessions</td>
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Data are expressed as mean ± SD.

* \(P < 0.001\) versus END.

** \(P < 0.01\) versus END.
Duplex images were stored in Digital Imaging and Communications in Medicine (DICOM) format for offline analysis. End-diastolic frames, determined by the R-spike of the ECG trace, were extracted and stacked in a new DICOM file using commercially available software (Sante DICOM Editor, Version 3.0.12; Santesso, Athens, Greece). Brachial artery diameters were determined using edge-detection software (Artery Measurement System; Image and Data Analysis, Gothenburg, Sweden). Preocclusion diameters were determined from the average of the 30-s sample. Postocclusion diameters were averaged in rolling five-cycle bins (32). Peak postocclusion diameter was defined as the maximum five-cycle average. Absolute FMD was calculated as the difference between the peak postocclusion diameter and the preocclusion diameter. Relative FMD is the ratio of the absolute FMD to the preocclusion diameter. We previously reported day-to-day intraclass correlations coefficients in a similar population of 0.89 for absolute and relative FMD (7).

Blood velocity raw audio signals were continuously analyzed by an external spectral analysis system (model Neurovision 500M TCD; Multigon Industries, Yonkers, NY) to determine continuous intensity weighted mean blood velocity (MBV). The MBV was sampled at 100 kHz during the FMD tests using commercially available hardware (Powerlab model ML795; ADInstruments) and analyzed offline using LabChart 7 Pro for Windows (Powerlab ML 795; ADInstruments). MBV signals were corrected for the angle of insonation (all ≤70°). Blood flow was calculated by multiplying brachial artery cross-sectional area by MBV. Postocclusion reactive hyperemic blood flows and MBV were averaged into five-cycle bins to align with the five-cycle diameter bins. Peak reactive hyperemic blood flow is reported as the maximum value during the 3-min postocclusion period. Shear rate for each bin was calculated by multiplying the MBV bin by 8 and by dividing it by the corresponding reactive hyperemic five-cycle bin. The reactive hyperemic stimulus until peak dilation was quantified as shear rate area under the curve (AUC).

Endothelial-independent function was assessed 10 min after cuff release using a 0.4-mg sublingual spray of NTG (6). Longitudinal B-mode images (8 MHz) of the right brachial artery were collected before the administration of NTG (pre-NTG; 10 cardiac cycles) and for 10 cardiac cycles every minute post-NTG up to 10 min at a frame rate of 22.9 frames per second. End-diastolic frames were extracted and stacked, and peak-NTG diameter was determined from the average of 10 cardiac cycles at each minute. NTG results are expressed in absolute and relative units. One participant declined the NTG assessment due to a history of severe headaches; therefore, NTG results are reported for 21 patients.

Exercise training protocols. Participants attended two supervised sessions per week for 12 wk at the Cardiac Health and Rehabilitation Centre at the Hamilton Health Sciences General Site (Hamilton, Ontario). Each session involved a 10- to 15-min standardized warm-up and cooldown involving light aerobic exercise and dynamic stretching. Exercise intensities for each protocol were based on the peak power output (PPO) achieved during the pretraining exercise stress test. The prescription for END was based on the Canadian Association of Cardiac Rehabilitation exercise guidelines (30) and involved continuous cycling at 58% of PPO (range 51%–65%). Participants progressed from 30 min (weeks 1–4) of cycling to 40 min (weeks 5–8) to 50 min (weeks 9–12). The protocol for HIT was based on previous research in adults with type 2 diabetes (18) and involved ten 1-min cycling intervals at 89% PPO (range, 80%–104%) separated by 1-min intervals at 10% PPO. Rather than increasing duration, workload was increased every 4 wk to elicit the heart rate responses achieved during 89% pretraining PPO. Consequently, patients were training at 102% pretraining PPO during weeks 5–8 and 110% pretraining PPO for weeks 9–12. In addition to the supervised training sessions, patients were instructed to perform lower limb exercise at least one additional day per week, using similar exercise durations and intensities as their exercise protocol. Unsupervised sessions were tracked using Polar heart rate monitors (RS300X; Polar Electro Inc., Lake Success, NY).

 Statistical analysis. Statistical analyses were performed using the Statistical Package for the Social Sciences (Version 11.5; SPSS, Chicago, IL). Factorial (END versus HIT group) repeated-measures (pre- versus posttraining) analyses of variance were used to compare indices of cardiorespiratory fitness, brachial artery endothelial function, and resting hemodynamics. Group differences in pretraining characteristics and exercise training data were compared using independent t-tests. Data are presented as mean ± SD, with P ≤ 0.05 considered statistically significant.

RESULTS

There were no differences in pretraining age, height, weight, body mass index, CAD criteria, time since CAD event, or medications between HIT and END (P ≤ 0.05). Posttraining weight decreased (END, −1.2 ± 1.8 kg; HIT, −0.6 ± 2.2 kg, P ≤ 0.05). Exercise training data for the supervised sessions are reported in Table 1. The END group performed twice as much total work and average work per supervised exercise session compared with the HIT group (P ≤ 0.01). The HIT group had higher mean heart rates during the supervised sessions than the END group. HIT trained at 73% ± 10% of their age-predicted maximal heart rate, whereas END trained at 65% ± 4%. There was no difference in exercise attendance between exercise groups. There were also no group differences in the frequency or duration of unsupervised exercise sessions (P ≥ 0.05). During the 12-wk training period, END performed 14 ± 14 unsupervised exercise sessions for 45 ± 3 min per session, whereas HIT performed 11 ± 10 unsupervised sessions for 40 ± 17 min per session. HIT trained at 68% ± 5% of their age-predicted heart rate maximum during the unsupervised sessions, which was higher than the END group, which trained at 60% ± 7% (P < 0.05).

Cardiorespiratory fitness indices are presented in Table 2. After 12 wk of training, fitness increased 19% and 24% in the
END and HIT groups, respectively. Relative VO₂peak, relative VO₂ at anaerobic threshold, and PPO were significantly larger posttraining, with no differences between exercise groups. There was no change in heart rate at peak or anaerobic threshold after training or between exercise groups.

Brachial artery FMD results are reported in Figure 1. Absolute and relative FMD were increased posttraining, with no differences between exercise groups. Baseline brachial artery diameters for the FMD and NTG tests, time to peak dilation, and FMD peak reactive hyperemic blood flows and AUC are reported in Table 3. There was no difference in preocclusion FMD diameters and pre-NTG diameters between exercise groups, and diameters were unchanged posttraining. Preocclusion FMD diameters were also not significantly different from the pre-NTG diameters at pre- and posttraining, suggesting the artery had returned to a baseline state before the administration of NTG. Time to peak dilation during the FMD and NTG assessments were unchanged with training, with no differences between exercise groups. Peak dilation during the FMD test occurred around 1 min after cuff release, whereas maximal NTG-mediated diameters were observed between 7 and 8 min after administration. Peak reactive hyperemic blood flows and shear rate AUC were unchanged

### Table 2. Cardiorespiratory fitness data pre- and posttraining.

<table>
<thead>
<tr>
<th>Variable</th>
<th>END Pre</th>
<th>END Post</th>
<th>HIT Pre</th>
<th>HIT Post</th>
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<tbody>
<tr>
<td>Peak exercise capacity (W)</td>
<td>18.7 ± 5.7</td>
<td>22.3 ± 6.1*</td>
<td>19.8 ± 3.7</td>
<td>24.5 ± 4.5*</td>
</tr>
<tr>
<td>Relative VO₂ peak (mL·kg⁻¹·min⁻¹)</td>
<td>120 ± 24</td>
<td>123 ± 16</td>
<td>133 ± 16</td>
<td>139 ± 15</td>
</tr>
<tr>
<td>Peak HR (bpm)</td>
<td>1.21 ± 0.06</td>
<td>1.25 ± 0.08</td>
<td>1.25 ± 0.12</td>
<td>1.23 ± 0.09</td>
</tr>
<tr>
<td>Peak power output (W)</td>
<td>108 ± 30</td>
<td>133 ± 42*</td>
<td>133 ± 51</td>
<td>159 ± 52*</td>
</tr>
<tr>
<td>Anaerobic threshold (W)</td>
<td>13.2 ± 2.2</td>
<td>15.3 ± 3.3*</td>
<td>14.4 ± 2.0</td>
<td>17.7 ± 3.0*</td>
</tr>
<tr>
<td>HR (bpm)</td>
<td>96 ± 12</td>
<td>99 ± 10</td>
<td>103 ± 13</td>
<td>110 ± 18</td>
</tr>
</tbody>
</table>

Data are presented as mean ± SD. Anaerobic data and RER data, n = 18 (END = 9, HIT = 9). Peak HR, n = 20 (END=10; HIT=10).

*P ≤ 0.001 versus pretraining.

HR, heart rate; RER, respiratory exchange ratio; VO₂, oxygen consumption.

**FIGURE 1**—Absolute (A) and relative (B) brachial artery flow-mediated dilation (FMD) responses pretraining (white) and posttraining (gray) for END (moderate-intensity endurance exercise) and HIT (low-volume high-intensity interval exercise) groups. *P ≤ 0.001 versus pretraining.

**FIGURE 2**—Absolute (A) and relative (B) brachial artery nitroglycerin (NTG)-mediated dilation responses pretraining (white) and posttraining (gray) for END (moderate-intensity endurance exercise) and HIT (low-volume high-intensity interval exercise) groups.
with training, with no differences between groups. Resting hemodynamics are also presented in Table 3. There was a training effect for seated brachial diastolic blood pressure and resting heart rate, with no differences between groups. Seated brachial systolic blood pressure was unchanged with training.

**DISCUSSION**

This is the first study to examine the effectiveness of low-volume HIT in patients with CAD. We demonstrated comparable increases in brachial artery FMD and cardiorespiratory fitness in both END and HIT training programs, although the END group performed approximately twice as much total work as the HIT group. Schnohr et al. (29) recently demonstrated that cycling intensity rather than duration is more closely associated with determining the risk of all-cause and cardiovascular mortality in men and women. Our findings lend further support to the notion that exercise intensity may be more important than exercise duration in terms of cardiovascular health. Recent work has also demonstrated the safety of high-intensity interval exercise in patients with cardiovascular disease (28). Similar to previous investigations using isocaloric high-intensity interval exercise prescriptions (19, 20, 27, 35, 36), we reported no adverse events in either our low-volume HIT or END groups. Taken together, the findings from this study provide preliminary evidence that low-volume HIT provides the same benefit as END and therefore may be a suitable exercise prescription for patients with CAD.

**Cardiorespiratory fitness.** Previous investigations in patients with CAD demonstrate comparable (35) and superior (27, 36) improvements in cardiorespiratory fitness with interval exercise training when compared with traditional endurance exercise. Although we did not observe a difference in the magnitude of change in fitness between END and HIT, the 24% increase in relative VO₂peak observed after HIT is larger than the changes observed in these previous interval exercise investigations (19, 21, 27, 35). Low-volume HIT also increased relative VO₂peak at anaerobic threshold, similar to the findings reported by Warburton et al. (35). Previous work has demonstrated a 12% improvement in survival for each 3.5-mL·kg⁻¹·min⁻¹ increase in VO₂peak (22). On average, VO₂peak increased 3.6 and 4.7 mL·kg⁻¹·min⁻¹ in the END and HIT groups, respectively, which may indicate the potential for improvements in survival rates after both training programs.

**Brachial artery endothelial function.** Brachial artery endothelial function is an accepted noninvasive surrogate for coronary artery endothelial function (31). Endothelial dysfunction is associated with the presence and severity of CAD (14, 24), making it an important therapeutic target. The observation of improved brachial artery FMD after both END and low-volume HIT was not surprising, given previous evidence of training-induced improvements in brachial artery FMD after lower-limb interval (19, 21, 36) and endurance (9, 36) exercise training in CAD patients. Currently, there is no consensus regarding a clinical cutoff value for brachial artery FMD (32); however, increased cardiovascular morbidity and mortality has been shown to be associated with a persistently impaired relative FMD value <5.5% after optimized anti-atherosclerotic therapy in patients with existing CAD (17). The pretraining FMD averages for both END and HIT were less than 5.5%, suggesting our sample may have been at an increased risk of future cardiovascular events. We observed a 34% and 33% increase in endothelial-dependent function in the END and HIT groups, respectively, resulting in posttraining group averages higher than 5.5%.

Posttraining improvements in brachial artery FMD were likely elicited by repetitive exercise-induced increases in localized shear stress in the brachial arteries, which has been shown to occur during lower-limb cycling (33). In addition, previous work has demonstrated the necessity of localized shear stress for FMD adaptations. In a previous study, the attenuation of brachial artery shear rate using forearm cuff inflation prevented the brachial artery FMD improvements, which were observed in the uncuffed arm, after 8 wk of lower limb cycle training (4). In the present study, patients were not using a recumbent cycle ergometer. Therefore, we cannot rule out the potential localized effects of gripping the handlebars during training and the contribution it may have had on the observed training responses in the brachial artery (34). Mechanisms mediating the observed improvement in brachial artery FMD may include, but are not limited...
to, increased nitric oxide bioavailability (8,11), decreased basal oxidative stress (1,36), or decreased sympathetic activity (12). We observed no change in NTG-mediated vasodilation after both training programs, which is consistent with other training studies (16,21,36). The absence of endothelial-independent improvements with exercise training lends further support to the ability of both HIT and END to selectively improve endothelial-dependent pathways. In addition, the absence of a change in preocclusion diameters and peak reactive hyperemic blood flow and shear rate AUC between pre- and posttraining assessments suggests a comparable FMD stimulus, lending further support to an increased capacity of the endothelium to respond to a given stimulus after 12 wk of exercise training.

Resting hemodynamic indices. Resting heart rate and diastolic blood pressure were decreased after training in both END and HIT, which is consistent with findings from previous interval exercise training studies (19,21). An elevated resting heart rate is associated with increased risk of mortality from CAD (23). For each increment of 10 bpm, there is approximately an 18% and 10% increased risk of death for women and men, respectively. The average reduction in heart rate observed in both END and HIT was <10 bpm, therefore not clinically significant. Diastolic blood pressure reductions equated to 10% and 3% for END and HIT, respectively. Although the reductions observed with HIT were smaller, they are comparable with the reductions reported by a meta-analysis of exercise training studies (15). Systolic blood pressure was not significantly lower after training, which contradicts the findings of the meta-analysis. However, previous interval exercise studies in CAD reported no training-induced reductions in systolic blood pressure (21,27,35,36). The absence of a change in systolic blood pressure and the minimal reductions in heart rate and diastolic blood pressure after training could be attributed to the optimal medical management and normotensive state of our patients or the sample size.

Limitations. We aimed to recruit an equal number of men and women; however, we had limited access to female patients with CAD. Therefore, only two women were included in the study. Although they were distributed between the two exercise groups, a larger female sample would be more representative of the cardiac rehabilitation population. Program wait times at our cardiac rehabilitation center delayed patient start times. On average, patients initiated exercise training 4–5 months after their CAD event, which is longer than desired based on cardiac rehabilitation guidelines (30). Although our patients were not undergoing previous exercise training during the waiting period, the use of a CAD sample closer to their date of event may have altered the training responses observed in our sample. The study did not include a control group with no exercise intervention. Rather, we used the current standard of care (END) as the control group. Previous interval training studies using a nonexercise control group found no change in physiological indices (21,36). Participants attended two supervised training sessions per week and exercised one additional day per week. Although our prescription satisfies the cardiac rehabilitation guidelines of three exercise sessions per week (30), public health agencies advocate for individuals to participate in activity during most days of the week. We observed significant training adaptations with only 3 d of exercise per week; however, larger adaptations may have been observed by increasing training frequency. Lastly, there are a variety of interval exercise training protocols currently being used. We chose the ratio of 1:1 for the high- and lower-intensity exercise intervals based on its success in middle-age populations (13,18). However, alternative time-efficient interval protocols should be tested. What is important is that our protocol was time efficient and involved approximately half of the work involved in a traditional endurance exercise session. We believe these features may be appealing to populations who are not motivated, or do not have the time to exercise.

CONCLUSIONS

Given that “lack of time” is one of the commonly cited barriers to exercise adherence in cardiac rehabilitation, time-efficient exercise programs pose an advantageous treatment strategy for patients with cardiovascular diseases. This study demonstrated training-induced improvements in fitness and brachial artery FMD after 12 wk of END and time-efficient low-volume HIT, with no difference in the magnitude of response between exercise programs. Our findings are consistent with the previous body of literature examining low-volume interval prescriptions in young healthy populations and suggest that shorter duration interval protocols may be a sufficient prescription to infer health benefits in more clinically relevant populations. Although it is difficult to draw conclusions regarding the clinical effect of the adaptations observed, the ability to elicit favorable improvements with low-volume HIT is noteworthy, especially given the HIT group performed approximately half the amount of work than the END group. This study also demonstrated the feasibility of low-volume HIT in cardiac rehabilitation. We reported no adverse events and no difference in exercise program adherence, despite differences in exercise intensities. In conclusion, the findings from this study provide preliminary evidence in support of low-volume high-intensity interval exercise prescriptions in patients with CAD and support the continued investigation of alternative exercise prescriptions for individuals with CAD, including low-volume HIT protocols.

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The results of the present study do not constitute any endorsement by the American College of Sports Medicine.
REFERENCES


