

Effects of a Low-Volume Aerobic-Type Interval Exercise on $\dot{V}O_{2\max}$ and Cardiac Mass

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ABSTRACT

MATSUO, T., K. SAOTOME, S. SEINO, N. SHIMOJO, A. MATSUSHITA, M. IEMITSU, H. OHSHIMA, K. TANAKA, and C. MUKAI. Effects of a Low-Volume Aerobic-Type Interval Exercise on $\dot{V}O_{2\max}$ and Cardiac Mass. *Med. Sci. Sports Exerc.*, Vol. 46, No. 1, pp. 42–50, 2014. **Purpose:** The aim of this study was to compare the effects of time-efficient, low-volume interval exercises on cardiorespiratory capacity and left ventricular (LV) mass with traditional continuous exercise in sedentary adults. **Methods:** Forty-two healthy but sedentary male subjects (age 26.5 ± 6.2 yr) participated in an 8-wk, five times per week, supervised exercise intervention. They were randomly assigned to one of three exercise protocols: sprint interval training (SIT, 5 min, 100 kcal), high-intensity interval aerobic training (HIAT, 13 min, 180 kcal), and continuous aerobic training (CAT, 40 min, 360 kcal). Cardiorespiratory capacity ($\dot{V}O_{2\max}$) and LV mass (3T-MRI) were measured preintervention and postintervention. **Results:** We observed significant ($P < 0.01$) increases in $\dot{V}O_{2\max}$ in all three groups, and the effect of the HIAT was the greatest of the three (SIT, $16.7\% \pm 11.6\%$; HIAT, $22.5\% \pm 12.2\%$; CAT, $10.0\% \pm 8.9\%$; $P = 0.01$). There were significant changes in LV mass, stroke volume (SV), and resting HR in both the SIT (LV mass, $6.5\% \pm 8.3\%$; SV, $5.3\% \pm 8.3\%$; HR, $-7.3\% \pm 11.1\%$; all $P < 0.05$) and HIAT (LV mass, $8.0\% \pm 8.3\%$; SV, $12.1\% \pm 9.8\%$; HR, $-12.7\% \pm 12.2\%$; all $P < 0.01$) but not in the CAT (LV mass, $2.5\% \pm 10.1\%$; SV, $3.6\% \pm 6.6\%$; HR, $-2.2\% \pm 13.3\%$; all $P > 0.05$). **Conclusions:** Our study revealed that $\dot{V}O_{2\max}$ improvement with the HIAT was greater than with the CAT despite the HIAT being performed with a far lower volume and in far less time than the CAT. This suggests that the HIAT has potential as a time-efficient training mode to improve $\dot{V}O_{2\max}$ in sedentary adults. **Key Words:** CARDIAC FUNCTION, CARDIAC MAGNETIC RESONANCE IMAGING, EXERCISE TRAINING, MAXIMAL OXYGEN CONSUMPTION, MYOCARDIUM

Research has shown that in our time-pressed society, a major barrier to exercise is lack of time (28). High-intensity interval exercise has gained attention recently, not only as an efficient training method for athletes but also as an appropriate exercise prescription for public health (18). Training programs using interval exercises are expected to be a highly effective, time-efficient strategy for public health. High-intensity interval exercises are divided into two main categories: sprint and aerobic types. Previous studies indicate that sprint-type interval training improves maximal oxygen consumption ($\dot{V}O_{2\max}$) mainly through increased oxidative capacity in peripheral muscles (11), whereas aerobic-type interval training improves $\dot{V}O_{2\max}$ mainly through

improved cardiac function (37). Representative examples of these two types of interval training include the modified Wingate protocol (2,10,22) and the 4×4 protocol (32,34,39), respectively. The sprint-type, modified Wingate protocol comprises four to seven repetitions of 30-s all-out maximal cycling with a 4-min rest between each bout. The aerobic-type 4×4 protocol comprises 4-min intervals repeated four times with 90%–95% $HR_{\max}$ and a 3-min active recovery period at approximately 70% $HR_{\max}$ between intervals.

From a public health perspective, however, these protocols might be excessive for daily routine exercise in sedentary adults; hence, we tried to find easier interval protocols in our preliminary experiment (24) and devised two candidate cycling protocols. In our previous study, we compared the exercise energy expenditure of our interval exercises with that of traditional, moderate-intensity continuous exercise (23). Table 1 shows detailed protocols of the three forms of cycling training, that is, sprint interval training (SIT), high-intensity interval aerobic training (HIAT), and continuous aerobic training (CAT) with each protocol's total exercise duration and exercise energy expenditure.

Although many studies (12,25,34,39) revealed superior outcomes from high-intensity interval exercise compared with moderate-intensity continuous exercise, the exercise volumes

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TABLE 1. The three exercise protocols in this study.

	Program	Total Time (min)	Volume ^a (kcal)	HR During Exercise ^a (bpm)	
				Average	Peak
SIT	2 min (30 W, 60 rpm) 30 s (120% $\dot{V}O_{2max}$, >85 rpm) and 15 s (rest) $\times$ 7 times (no rest at 7th)	10	110	161	179
HIAT	3 min (30 W, 40–60 rpm) 2 min (30 W, 60 rpm) 3 min (85%–90% $\dot{V}O_{2max}$, 70–80 rpm) 2 min (50% $\dot{V}O_{2max}$, 60 rpm) 3 min (85%–90% $\dot{V}O_{2max}$, 70–80 rpm) 2 min (50% $\dot{V}O_{2max}$, 60 rpm) 3 min (80%–85% $\dot{V}O_{2max}$, 70–80 rpm)	18	180	170	184
CAT	3 min (30 W, 40–60 rpm) 2 min (30 W, 60 rpm) 40 min (60%–65% $\dot{V}O_{2max}$, 60 rpm) 3 min (30 W, 40–60 rpm)	45	360	152	160

^aOur previous study (23) shows the exercise volume including 3-h excess postexercise oxygen consumption and HR during the exercise of the three protocols.

of the interval exercises and continuous exercise were the same in those studies. However, although the exercise volumes of the two protocols were identical, the subjects performing interval protocols had to repeat many bouts of high-intensity exercise (12). An exercise protocol of such great intensity and duration may not be an appropriate prescription for public health. Consequently, it must still be established whether high-intensity interval exercise can have a greater impact on human health even when the interval exercise is performed for a substantially shorter duration and at a lower volume compared with moderate-intensity continuous exercise. The present study focused on the effect of cycling exercise on cardiac mass using cardiac MRI because cardiac MRI is considered to be the gold standard for assessing cardiac mass (5,9). There have been no previous studies comparing the effects of SIT, HIAT, and CAT on the heart using cardiac MRI.

A previous study using isoenergetic work comparing interval and continuous exercises demonstrated that the percentage increase in $\dot{V}O_{2max}$ derived from interval exercise substantially exceeded that of continuous exercise (46% vs 14%) (39). It is logical to assume that the outcome for interval exercise would be superior to that of continuous exercise, even with a substantially shorter duration and lower volume of interval exercise than continuous exercise. Therefore, in the present study, we tested the hypothesis that despite the exercise volumes of SIT and HIAT being 30% and 50% of CAT, respectively, SIT and HIAT would have more impact than CAT on $\dot{V}O_{2max}$. In addition, we tested the second hypothesis that left ventricular (LV) mass increased only through the HIAT. The purpose of this study was, therefore, to compare the effects of time-efficient, low-volume, sprint- and aerobic-interval exercises on $\dot{V}O_{2max}$ and LV mass with traditional continuous exercise in sedentary adults.

METHODS

Participant Recruitment and Randomization

Participants were recruited through advertisements posted on the campus of the University of Tsukuba and local newspaper

advertisements. This study was conducted in accordance with the guidelines proposed in the Declaration of Helsinki. The Ethical Committees of both JAXA and the University of Tsukuba reviewed and approved the study protocol. The aim and design of this study were explained to every subject before each gave their written, informed consent.

The inclusion criteria in this study were as follows: 1) male adults in their 20s or 30s and 2) sedentary lifestyles (no participation in regular exercise activities for the past year). The exclusion criteria were as follows: 1) body mass index $\geq 25 \text{ kg}\cdot\text{m}^{-2}$, 2) smokers, and 3) adverse medical issues (all candidates had a medical interview and a 12-lead resting ECG examination to confirm eligibility by a doctor). The flow of participants in the study is shown in Figure 1. Consequently, 42 subjects were selected and underwent a maximal graded exercise test to determine their baseline (week 0) $\dot{V}O_{2max}$.

A 4-wk nonexercise period was set after the baseline (week 0) $\dot{V}O_{2max}$ measurement as a control period, during which time subjects engaged in their normal lifestyles. Subsequently, preintervention measurements (week 4) were performed, whereupon the subjects were randomly assigned to one ($n = 14$) of three exercise intervention groups, stratified according to age and $\dot{V}O_{2max}$. The study was single blind; assessors for all outcomes were blind to the subject group assignment, and data on all outcomes were kept blind until final data entry for postintervention assessments was completed.

Exercise Intervention

All subjects participated in an 8-wk, five times per week, supervised cycling exercise intervention. In our previous study (23), we measured exercise energy expenditure, including a 3-h excess postexercise oxygen consumption and HR during exercise of our three cycling protocols. Table 1 shows the detailed exercise protocols, the total exercise duration, the exercise energy expenditure, and the HR during the three exercises. Both SIT and HIAT protocols were originally developed in our laboratory for sedentary individuals (24). In

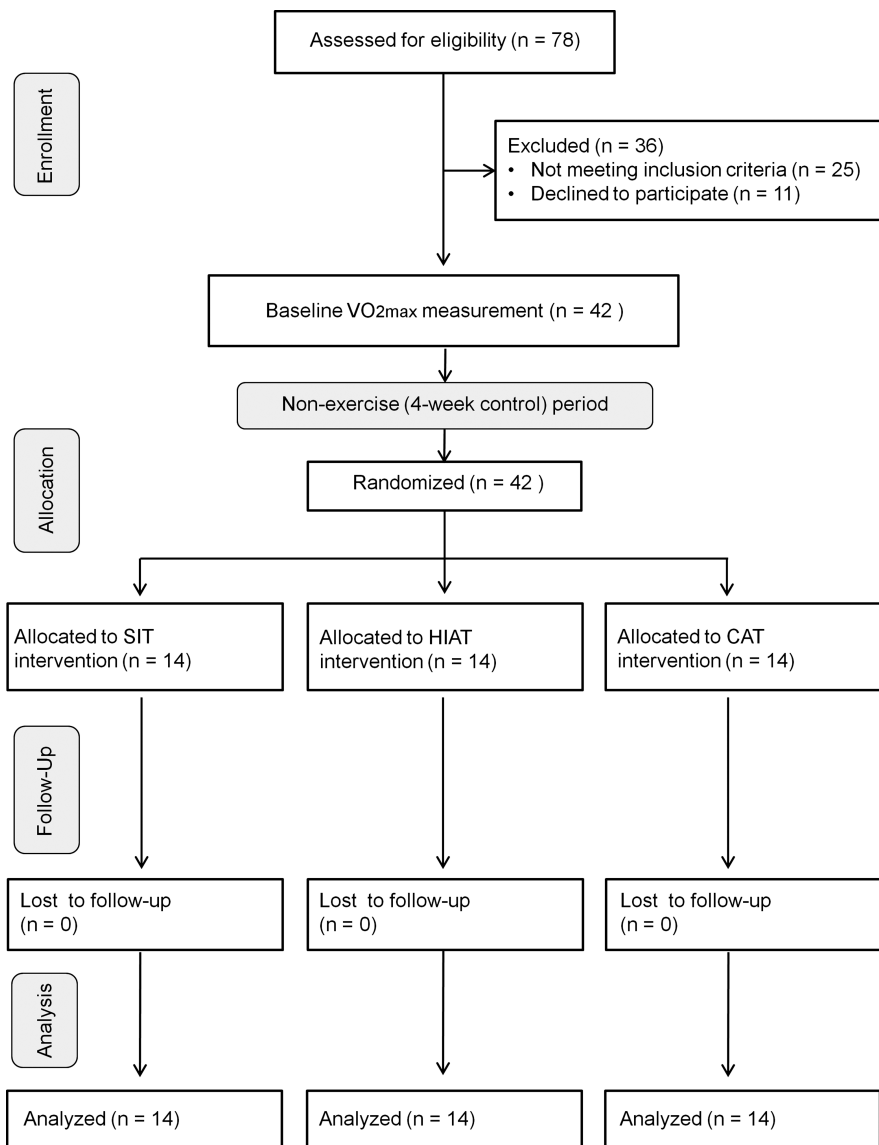


FIGURE 1—CONSORT flow diagram of participants through each stage of the trial. SIT, sprint interval training; HIAT, high-intensity interval aerobic training; CAT, continuous aerobic training.

particular, during the third stage of our HIAT, intensity (80%–85% $\dot{V}O_{2max}$) was lower than the first and second stages (85%–90% $\dot{V}O_{2max}$) because we consider a lower intensity third stage to be crucial in daily exercise for sedentary individuals. To determine each subject's exercise intensity during the exercise session, we used the $\dot{V}O_{2max}$ measurement data (i.e., values per minute for workload and $\dot{V}O_2$) and calculated a simple linear regression equation for each subject: $Y = \beta x + c$, with $Y = \text{workload (W)}$, $x = \dot{V}O_2 \text{ (mL)}$, and β and c as constants. Subsequently, the given $\dot{V}O_{2max}$ data (mL) (e.g., 120% $\dot{V}O_{2max}$ for SIT) were applied to the equation, whereupon each subject's exercise intensity (workload, W) was determined. We recalculated and changed exercise intensity for each subject in all three groups recording mid-intervention (week 8) $\dot{V}O_{2max}$ measurements. As we treated the first week (week 5) of exercise intervention as a break-in

period, we allowed subjects to take 1 d off (i.e., subjects came to the laboratory four rather than five times). Furthermore, during the entire intervention period, if subjects in the SIT and HIAT groups could not complete their protocols with the predetermined (calculated) workloads, the workloads were reset 10–15 W lower than the predetermined. In particular, during the first 2 wk (weeks 5 and 6) or the middle 2 wk (weeks 9 and 10, i.e., after intensity recalculating), the workloads for subjects assigned to SIT or HIAT groups were gradually and progressively increased until they reached the calculated workloads. As for the CAT group, to adjust their exercise intensity (60%–65% $\dot{V}O_{2max}$), we measured exercise oxygen uptake ($\dot{V}O_2$) using an open-circuit computerized indirect calorimeter (AE-310S, Minato Medical Science, Osaka, Japan) once every 2 wk and monitored the exercise HR at every exercise session. The postintervention (week 13)

measurements were performed within one week of completing the exercise intervention.

Measurements

Anthropometric measurements. Body weight was measured once to the nearest 0.1 kg using a digital scale (InBody-3.2, Biospace, Seoul, Korea), and height was measured once to the nearest 0.1 cm using a wall-mounted stadiometer (YG-200, Yagami, Nagoya, Japan) with the subjects in underwear and barefoot. Abdominal circumference was measured at the level of the umbilicus, and hip and left-side thigh circumferences were measured at the greater curvature. These circumference measurements were taken in duplicate to the nearest 0.1 cm in the standing position. Fat mass and fat-free mass were measured by dual-energy x-ray absorptiometry using a Hologic QDR 4500A densitometer (Hologic, MA, USA). During the dual-energy x-ray absorptiometry measurement, the subjects remained motionless in the supine position while the fan-scanning arm passed over their bodies in parallel 1-cm strips.

Cardiorespiratory capacity. The subjects underwent a maximal graded exercise test on a cycling ergometer (75XL III, Konami, Tokyo, Japan) to determine $\dot{V}O_{2\max}$ at weeks 0, 4, 8, and 13. After a 2-min warm-up at 15 W, the subjects began the actual exercise protocol at a 30-W level. The workload was increased every 1 min by 15 W until volitional exhaustion (33). Data on maximal $\dot{V}O_2$ values were accepted only if the subjects met the following two criteria: 1) the subjects were too fatigued to continue pedaling on the bicycle and 2) the highest respiratory exchange ratio was >1.10 . During the test, ventilation and expired gases ($\dot{V}O_2$ and carbon dioxide production [$\dot{V}CO_2$]) were measured using the mixing chamber method with an open-circuit computerized indirect calorimeter (AE-310S, Minato Medical Science, Osaka, Japan). The gas analyzer was calibrated before each trial. The coefficient of variation (CV) from the mean figure for the three resting $\dot{V}O_2$ measurements in the 10 subjects was 4.3%. HR at rest and during the exercise test was monitored using an electrocardiogram monitor (Dyna Scope, Fukuda Denshi, Tokyo, Japan).

MRI Measurements

Parameters protocol. A cardiac MRI was performed on all subjects before (week 4) and after (week 13) the intervention at the Center for Cybernetics Research in the University of Tsukuba. All data were acquired using a 3.0-T MR scanner (Achieva, Release 3.2.1.1, Philips, The Netherlands) with a 32-channel phased array sensitivity encoding (SENSE) cardiac coil and an ECG gating. The subject was positioned supine on the MRI table, and ECG monitoring leads were placed on the chest. The data set was acquired with the following imaging protocol. Balanced turbo field echo sequence (45° flip angle, reaction time/echo time = 2.4 ms/1.22 ms, matrix size = 128 × 128 interpolated 256 × 256,

field of view = 380 mm) was used to acquire 14–19 short-axis slices, with no slice gap and 6-mm thickness. All images were acquired during breath hold at end expiration of 15–25 s, depending on the subject's condition.

Image analysis. Short-axis slices were used for LV mass calculation using a standard system software analysis tool (Extended MR WorkSpace, PHILIPS, The Netherlands). LV mass was determined from myocardial volumes (epicardial minus endocardial volume) after multiplication by the specific myocardium density (1.05 g·cm⁻³). Previous studies demonstrated that MRI with Simpson's rule technique resulted in highly accurate and reproducible measurements of LV mass (9,15). LV mass was divided by body surface area (BSA) to obtain the LV mass index according to the Devereaux and Reichek (6) formula, and BSA was calculated using the following formula: $BSA = \text{height}^{0.725} \times \text{weight}^{0.425} \times 0.007184$. LV volume was determined by manually identifying the endocardial border at both end diastole and end systole. The end diastole was the first frame in every sequence, and the end systole was defined as the frame with the smallest endocardial chamber area. LV volumes were then calculated by summation using Simpson's rule (14,27). Stroke volume (SV) was calculated as the average of the end diastole minus end-systole volumes. Papillary muscles were included in the LV mass calculations but excluded from SV calculations. The interventricular septum was treated as LV mass. We excluded one subject in the SIT group from MRI analyses because we could not analyze his data due to poor image quality. All analyses were performed by one observer. The CV from the mean for the two LV mass measurements in 10 subjects was 3.1%.

Blood pressure and biochemical assays of blood.

Blood pressure and biochemical assays of blood were measured at weeks 4 and 13. One trained nurse measured the systolic and diastolic blood pressures (SBP and DBP) of subjects via the right arm using a mercury manometer and a standard protocol after the subjects had rested for at least 20 min in a seated position. Using the first and fifth Korotkoff sounds as indicative of SBP and DBP, respectively, these values were estimated as the mean of two readings.

Blood samples were collected from the antecubital vein of each subject. The sample was drawn into two polypropylene tubes, one containing ethylenediamine tetra-acetic acid for whole blood cell count and one for serum collection. The whole blood cell count, including red blood cell count, hemoglobin concentration, and hematocrit, was measured using an automated hematology analyzer (XE-210, Sysmex, Hyogo, Japan). With serum samples, the creatinine level was quantified by the enzymatic method using a JCA-BM8060 (JEOL, Tokyo, Japan), and N-terminal pro brain natriuretic peptide (NT-proBNP) was measured via an electrochemiluminescence immunoassay (ECLIA) method using Modular Analytics E170 (Roche Diagnostics Japan, Tokyo, Japan). Serum samples were stored at -80°C until analyzed. The inter- and intra-assay CV were $<5\%$ for all blood parameters. Percentage changes in the volumes of blood and plasma were calculated according to Dill and Costill's (8) formula.

Statistical Analyses

A priori power analysis was performed to determine the sample size. The primary outcome variable of this study was the increase of $\dot{V}O_{2\max}$ achieved through three types of exercise intervention. On the basis of data from both a previous study (39) and our preliminary study (24) on changes in $\dot{V}O_{2\max}$, we assumed a 15% difference in the training effect between the three groups with an SD estimate of 10%. With an alpha error rate of 0.017 (with Bonferroni adjustment for *post hoc* tests) and statistical power of 80%, the minimal sample size in each group was estimated to be 11 subjects (33 subjects in total). Assuming subject attrition such as dropout, we recruited 14 subjects for each group (42 subjects in total) in this study.

Values are expressed as the mean $\pm$ SD. Paired Student's *t*-tests were performed to test the significance of changes in values measured preintervention and postintervention, and Cohen's *d* values were used as an effect size (ES) index. To analyze differences among multiple groups at baseline and during the intervention, we used one-way ANOVA. Tukey–Kramer's *post hoc* tests were applied when the difference was significant ($P < 0.05$) according to the results of the ANOVA. To compare any change in $\dot{V}O_{2\max}$ among the three groups, a two-way repeated-measures ANOVA (time $\times$ group) was applied. Chi-square tests were used to analyze categorical values. We used SAS, version 9.2 (SAS Institute Japan, Tokyo, Japan) to analyze the data.

RESULTS

The characteristics of the study groups at preintervention (week 4) are shown in Table 2. No differences were observed at week 4 across the three groups in any of the measures. All subjects completed the 8-wk exercise training (no dropouts). Adherence to the interventions (i.e., attendance rate at the exercise training sessions) was similar among the three groups (SIT, 96.1% $\pm$ 5.7%; HIAT, 97.6% $\pm$ 4.3%; CAT, 95.9% $\pm$ 7.3%; $P = 0.71$).

Figure 2 presents changes in $\dot{V}O_{2\max}$ during the entire study period by group. No significant $\dot{V}O_{2\max}$ changes were observed in all three groups from weeks 0 to 4 (4-wk control period), nor any significant interaction (time–group) during the same period. Meanwhile, significant increases in $\dot{V}O_{2\max}$ were observed in all three groups from weeks 4 to 13 (exercise intervention period). During this period, significant ($P = 0.03$) interaction (time–group) was observed among the three exercise groups. As for the period from weeks 8 to 13, we observed significant changes in $\dot{V}O_{2\max}$ in both the SIT and HIAT groups, but not in the CAT group.

Table 3 presents changes in measurement values during the exercise intervention period by group with the results of the ANOVA analysis and the ES (Cohen's *d*). We observed increases in thigh circumference with moderate ES in all three groups. Body fat decreased and body muscle increased in all three groups. The ES for body muscle and fat were

small, whereas those of the HIAT were the largest among the three groups. There was a significant group difference in increased $\dot{V}O_{2\max}$. We observed large ES for this value in both the SIT and HIAT and moderate ES in the CAT, whereas the ES of the HIAT was the largest of the three. As for the cardiac measurements, significant decreases in resting HR and increases in LV mass (also its index) were observed in both the SIT and HIAT with moderate to large ES, whereas no significant changes were observed in the CAT. SV increased in all three groups, although there was a group difference, that is, a large ES was observed only in the HIAT. Cardiac output remained unchanged in all three groups. The ejection fraction tended to increase in both HIAT and CAT with moderate ES. No significant changes were observed in blood variables in all three groups except for change in hematocrit of the SIT. The percentage changes in blood volume by SIT, HIAT, and CAT averaged $-1.0\% \pm 3.7\%$ ($P = 0.32$), $-0.1\% \pm 3.4\%$ ($P = 0.94$), and $-0.2\% \pm 2.8\%$ ($P = 0.76$), and those in plasma volume (PV) averaged $-2.5\% \pm 6.1\%$ ($P = 0.15$), $0.2\% \pm 6.4\%$ ($P = 0.93$), and $-0.9\% \pm 4.9\%$ ($P = 0.52$), respectively. There were no significant changes in all three groups and no significant group differences.

Figure 3 shows comparisons of percentage changes in $\dot{V}O_{2\max}$, LV mass, SV and resting HR, respectively, during the exercise intervention among the three groups. Significant increases in $\dot{V}O_{2\max}$ were observed in all three groups (SIT, 16.7% $\pm$ 11.6%; HIAT, 22.5% $\pm$ 12.2%; CAT, 10.0% $\pm$ 8.9%). We also observed significant changes in LV mass, SV, and resting HR in both the SIT (LV mass, 6.5% $\pm$ 8.3%; SV, 5.3% $\pm$ 8.3%; HR, $-7.3\% \pm 11.1\%$) and HIAT groups (LV mass, 8.0% $\pm$ 8.3%; SV, 12.1% $\pm$ 9.8%; HR, $-12.7\% \pm 12.2\%$), but not in the CAT group (LV mass, 2.5% $\pm$ 10.1%; SV, 3.6% $\pm$ 6.6%; HR, $-2.2\% \pm 13.3\%$). There were significant group differences in increased $\dot{V}O_{2\max}$ and SV.

DISCUSSION

We observed significant increases in $\dot{V}O_{2\max}$ in all three groups during the exercise intervention period, and there was a significant time–group interaction (Fig. 2). The ES values for the changes in $\dot{V}O_{2\max}$ in both the SIT (1.10) and HIAT (1.43) were large, whereas the ES value in the CAT (0.63) was moderate (Table 3). Also, the percentage change in $\dot{V}O_{2\max}$ of the HIAT (22.5% $\pm$ 12.2%) significantly exceeded that of the CAT (10.0% $\pm$ 8.9%) (Fig. 3). The significant group difference in $\dot{V}O_{2\max}$ improvement between HIAT and CAT in our study is noteworthy because several studies (12,25,34,39) comparing the 4 $\times$ 4 HIAT protocol with a moderate-intensity continuous exercise (the CAT) revealed that the increase in $\dot{V}O_{2\max}$ of their HIAT exceeded that of their CAT when the exercise volumes were matched. In the present study, however, the exercise duration and volume of our HIAT (13 min, 180 kcal) were substantially lower than (approximately half) those of the CAT (40 min, 360 kcal). These results emphasize the primacy of exercise intensity

TABLE 2. Characteristics of subjects before intervention.

	SIT (n = 14)	HIAT (n = 14)	CAT (n = 14)	Group Differences ANOVA, P
Age, yr	26.4 ± 6.5	27.2 ± 6.4	25.9 ± 6.0	0.86
Height, cm	170.9 ± 5.0	171.6 ± 4.9	172.2 ± 3.4	0.74
Body weight, kg	62.4 ± 5.4	63.1 ± 6.7	63.0 ± 7.2	0.95
Body mass index	21.3 ± 1.2	21.4 ± 1.9	21.2 ± 2.4	0.97
Abdominal circumference, cm	76.6 ± 4.7	76.9 ± 5.8	75.6 ± 6.8	0.84
Hip circumference, cm	90.8 ± 3.2	91.5 ± 4.0	91.1 ± 5.2	0.92
Thigh circumference, cm	47.4 ± 2.3	46.6 ± 2.9	45.9 ± 3.6	0.44
Body fat, kg	10.2 ± 2.9	12.1 ± 3.2	10.8 ± 3.7	0.32
Body muscle, kg	50.6 ± 4.8	49.5 ± 4.4	50.4 ± 4.2	0.77
Maximal oxygen consumption, mL·kg ⁻¹ ·min ⁻¹	43.9 ± 6.7	41.9 ± 5.6	42.0 ± 6.8	0.66
Maximal oxygen consumption, L·min ⁻¹	2.74 ± 0.52	2.63 ± 0.33	2.64 ± 0.50	0.77
Systolic blood pressure, mm Hg	111 ± 12	110 ± 8	112 ± 9	0.86
Diastolic blood pressure, mm Hg	62 ± 12	61 ± 6	60 ± 9	0.89
HR at rest, bpm	68 ± 12	66 ± 9	67 ± 10	0.92
Left ventricular mass, g	103 ± 14	102 ± 15	110 ± 17	0.30
Left ventricular mass index	59.3 ± 7.6	58.4 ± 8.3	63.1 ± 7.8	0.26
Left ventricular end-diastolic volume, mL	128 ± 20	126 ± 12	137 ± 14	0.17
Left ventricular end-systolic volume, mL	52 ± 10	50 ± 8	57 ± 9	0.09
SV, mL	76.0 ± 13.0	76.4 ± 8.1	79.5 ± 9.7	0.64
Cardiac output, L·min ⁻¹	5.2 ± 0.9	5.0 ± 0.7	5.3 ± 0.8	0.62
Ejection fraction, %	59.6 ± 4.4	60.7 ± 4.6	58.2 ± 4.2	0.36
Blood variables				
Red blood cell, ×10 ⁴ ·μL ⁻¹	515 ± 20	513 ± 26	509 ± 44	0.88
Hemoglobin, g·dL ⁻¹	15.5 ± 0.6	15.3 ± 0.6	15.3 ± 1.1	0.68
Hematocrit, %	45.6 ± 2.2	45.2 ± 2.4	45.2 ± 3.1	0.89
NT-proBNP, pg·mL ⁻¹	19 ± 10	16 ± 10	18 ± 8	0.72
Creatinine, mg·dL ⁻¹	0.83 ± 0.11	0.82 ± 0.14	0.81 ± 0.06	0.90

SIT, sprint interval training; HIAT, high-intensity interval aerobic training; CAT, continuous aerobic training; NT-proBNP, N-terminal pro brain natriuretic peptide.

rather than duration and volume in improving $\dot{V}O_{2\max}$ and suggest that the HIAT in our study would be a “time-efficient” exercise protocol for improving $\dot{V}O_{2\max}$ in public health.

Conversely, we observed no significant group difference in $\dot{V}O_{2\max}$ improvement between SIT and CAT ($P = 0.25$),

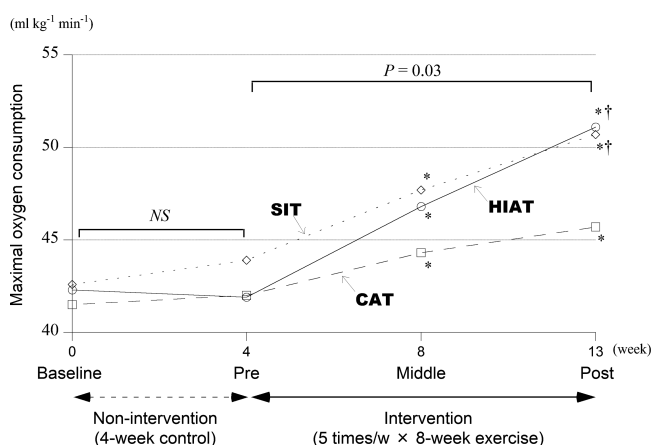


FIGURE 2—Changes in $\dot{V}O_{2\max}$ during the entire study period by group. No significant $\dot{V}O_{2\max}$ changes were observed in any of the three groups during the nonintervention period. There was no significant time-group interaction. Conversely, there was a significant ($P = 0.03$) time-group interaction among the three groups during the exercise intervention period. *Significant changes ($P < 0.01$) were observed from week 4. †Significant changes ($P < 0.01$) were observed from week 8. P value for interaction is displayed. SIT, sprint interval training; HIAT, high-intensity interval aerobic training; CAT, continuous aerobic training; NS, not significant.

nor between SIT and HIAT ($P = 0.35$), whereas the $\dot{V}O_{2\max}$ of the SIT significantly increased ($16.7\% \pm 11.6\%$; Fig. 3) and its ES was large (1.10) (Table 3). Improvements in SIT and CAT $\dot{V}O_{2\max}$ were equivalent and consistent with previous SIT studies (1,22) using the modified Wingate protocol. The exercise volume of the SIT might have been too small, or the sample size of the studies might not have been sufficient to detect the difference between SIT and other protocols.

Cardiac MRI revealed that an 8-wk (five times per week), 40-min, traditional, moderate-intensity continuous exercise (the CAT) had no significant impact on myocardial mass. Two previous studies (19,30) using the MRI method showed increases of 8.2% and 5.5% in LV mass after 3 months of endurance training (bicycle ergometer and jogging, respectively). Another MRI study by Spence et al. (31) also showed a significant 8.3% increase in LV mass after 6 months of exercise training, although their exercise protocol included walking, running, hill running, and short intervals. Compared with these studies (19,30,31), our CAT exercise period (8 wk) may have been relatively short for detecting a significant increase in LV mass. Conversely, although the exercise volumes of interval exercises in our study were small, LV mass increased with both sprint- and aerobic-type interval training. Moreover, percentage changes in SV and HR were significant in both SIT and HIAT (Fig. 3). These results are interesting because previous studies (11,37) indicated that sprint-type interval training improves $\dot{V}O_{2\max}$ by boosting the oxidative capacity of peripheral muscles, whereas aerobic-type interval training improves $\dot{V}O_{2\max}$ by improving cardiac function.

TABLE 3. Comparisons of changes in values during the exercise intervention period across the three groups.

	SIT (n = 14)	P ^a	HIAT (n = 14)	P ^a	CAT (n = 14)	P ^a	Group Differences (ANOVA, P)	ES (Cohen's d)		
								SIT	HIAT	CAT
Body weight, kg	0.1 ± 0.9	0.77	-0.1 ± 1.7	0.88	-0.4 ± 1.2	0.29	0.68	0.01	-0.01	-0.05
Abdominal circumference, cm	-0.6 ± 1.9	0.27	-1.0 ± 2.8	0.21	-0.8 ± 2.0	0.14	0.89	-0.13	-0.16	-0.12
Hip circumference, cm	-0.3 ± 0.9	0.31	0.1 ± 1.5	0.89	-0.8 ± 1.2	0.03	0.21	-0.09	0.01	-0.15
Thigh circumference, cm	1.3 ± 1.3	<0.01	1.0 ± 1.9	0.07	1.7 ± 1.3	<0.01	0.50	0.52	0.36	0.44
Body fat, kg	-0.4 ± 0.9	0.08	-0.7 ± 0.8	<0.01	-0.4 ± 0.7	0.06	0.46	-0.16	-0.23	-0.11
Body muscle, kg	0.7 ± 0.9	0.02	0.9 ± 1.2	0.01	0.5 ± 0.9	0.05	0.63	0.14	0.19	0.12
Maximal oxygen consumption, mL·kg ⁻¹ ·min ⁻¹	6.8 ± 4.4	<0.01	9.2 ± 5.0	<0.01	3.8 ± 2.9	<0.01	<0.01 (HIAT > CAT)	1.10	1.43	0.63
Maximal oxygen consumption, L·min ⁻¹	0.42 ± 0.28	<0.01	0.57 ± 0.33	<0.01	0.22 ± 0.16	<0.01	<0.01 (HIAT > CAT)	0.89	1.48	0.47
Number of subjects who achieved an increase in maximal oxygen consumption at least 10%, n (%)	9 (64.3)		12 (85.7)		5 (35.7)		0.02 ^b			
Systolic blood pressure, mm Hg	-3 ± 6	0.11	0 ± 6	0.79	5 ± 15	0.20	0.10	-0.26	0.05	0.40
Diastolic blood pressure, mm Hg	-1 ± 6	0.39	-1 ± 6	0.69	3 ± 10	0.24	0.23	-0.12	-0.09	0.33
HR at rest, bpm	-5.4 ± 7.9	0.02	-8.9 ± 8.6	<0.01	-2.3 ± 10.2	0.42	0.16	-0.49	-1.07	-0.26
Left ventricular mass, g	6.5 ± 8.8	0.02	7.3 ± 7.1	<0.01	2.7 ± 10.9	0.37	0.36	0.44	0.51	0.14
Left ventricular mass index	3.6 ± 5.2	0.03	4.3 ± 4.2	<0.01	1.6 ± 6.3	0.35	0.39	0.48	0.55	0.18
Left ventricular end-diastolic volume, mL	10 ± 12	0.01	8 ± 10	0.01	0 ± 9	0.99	0.04 (SIT > CAT)	0.54	0.68	0.00
Left ventricular end-systolic volume, mL	7 ± 8	0.02	-1 ± 10	0.81	-3 ± 9	0.23	0.03 (SIT > CAT)	0.68	-0.10	-0.37
SV, mL	3.5 ± 5.5	0.05	8.9 ± 7.0	<0.01	3.0 ± 5.4	0.06	0.03 (HIAT > SIT, CAT)	0.28	1.05	0.27
Cardiac output, L·min ⁻¹	-0.1 ± 0.7	0.61	-0.1 ± 0.6	0.62	0.0 ± 0.8	0.99	0.92	-0.10	-0.14	0.00
Ejection fraction, %	-1.9 ± 2.9	0.05	2.9 ± 5.9	0.10	2.1 ± 4.6	0.12	0.03 (SIT < HIAT)	-0.42	0.63	0.52
Blood variables										
Red blood cell, ×10 ⁴ ·μL ⁻¹	6 ± 19	0.23	-1 ± 19	0.92	4 ± 14	0.35	0.59	0.27	-0.02	0.08
Hemoglobin, g·dL ⁻¹	0.2 ± 0.6	0.27	0.0 ± 0.5	0.92	0.0 ± 0.4	0.77	0.66	0.26	0.03	0.03
Hematocrit, %	0.9 ± 1.5	0.05	0.0 ± 1.9	0.98	0.4 ± 1.4	0.31	0.36	0.41	-0.01	0.14
NT-proBNP, pg·mL ⁻¹	0 ± 10	0.94	4 ± 15	0.33	-2 ± 10	0.48	0.40	-0.01	0.41	-0.26
Creatinine, mg·dL ⁻¹	0 ± 0.08	0.81	-0.01 ± 0.08	0.68	0 ± 0.05	1.00	0.95	-0.12	-0.08	0.00

^aPaired Student's *t*-tests were performed to test the significance of changes in values measured preintervention and postintervention.

^bχ² test was performed.

SIT, sprint interval training; HIAT, high-intensity interval aerobic training; CAT, continuous aerobic training; NT-proBNP, N-terminal pro brain natriuretic peptide; ES, effect size.

Our study results indicate that myocardial mass is influenced not only by aerobic-type interval training but also by sprint-type interval training. Trilk et al. (35) using a CO₂-rebreathing method, showed that SIT with the modified Wingate protocol improved cardiac function by reducing HR and increasing SV. This was consistent with our study, although the exercise intensity of our SIT (120% $\dot{V}O_{2max}$) was substantially lower than that of their SIT (243%–284% $\dot{V}O_{2max}$). Ricci et al. (29) and Legaz et al. (20) showed the associations between sprint training and LV mass using an echocardiographic method. Ricci et al. (29) revealed that an 8-wk sprint running training did not increase LV mass among young male students; however, the mean baseline $\dot{V}O_{2max}$ of the subjects was relatively high (59.5 ± 4.1 mL·kg⁻¹·min⁻¹). The other study by Legaz et al. (20) showed that 3 yr of training for sprint-trained elite runners increased SV but not LV mass.

Although improved cardiac function was seen in the SIT group, we observed moderate to large ES for changes in HR, SV, and LV systolic performance (ejection fraction) in the HIAT group, in particular. This unique improvement in cardiac function with the HIAT was consistent with results from Wisloff et al. (37). Their studies (16,17,38) using a rodent model suggested that HIAT induced adaptive hypertrophy in cardiomyocytes with improved contractile function. At the

cellular level, Ca²⁺ has an important role in the contraction–relaxation rates of the cardiomyocyte. Changes in the rate of Ca²⁺ cycling are also associated with variation in the contraction–relaxation rates of the cardiomyocyte after exercise training. In other words, exercise training improves the contractile performance of the cardiomyocyte through improved Ca²⁺ cycling. A previous study (7) suggests that submaximal rather than maximal exercise intensity may be an important factor for increasing Ca²⁺ sensitivity in the cardiomyocyte. This means that the HIAT protocol using submaximal intensity, that is, 80%–90% $\dot{V}O_{2max}$ of our 3 × 3 HIAT (Table 1) or 90%–95% HR_{max} of the 4 × 4 HIAT by Wisloff et al. (37,39), may be well suited to positively impact the cardiomyocyte Ca²⁺ cycling and cardiac function. Our results showing improved cardiac function, particularly in the HIAT group, are consistent with these explanations. In our study, although subjects' workloads in the SIT group were set at a supramaximal level (120% $\dot{V}O_{2max}$), their HR during exercise (average 161 bpm; peak 179 bpm, Table 1) was submaximal. This suggests that the SIT exercise at submaximal HR may influence subjects' cardiomyocytes, although the volume is small. Conversely, ensuring sufficient volume of submaximal exercise in the HIAT may help boost participants' cardiac function considerably, although the exercise intensity may be insufficient to influence

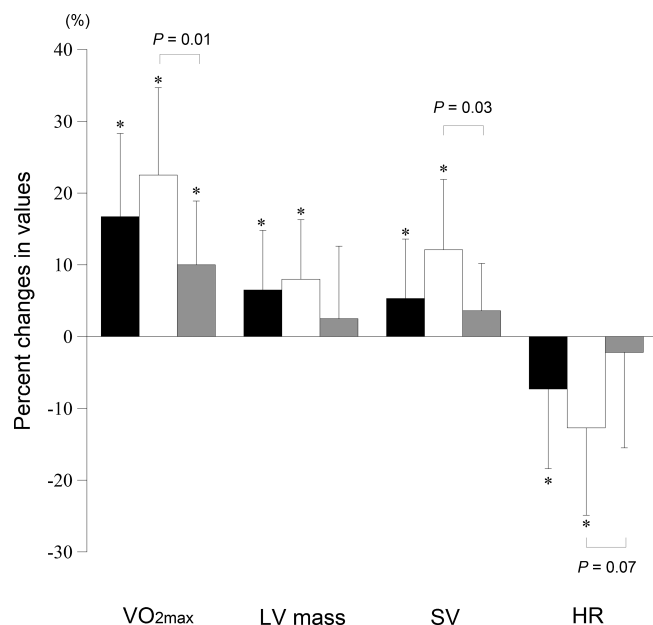


FIGURE 3—Comparisons of percentage changes in values during the exercise intervention period among the three groups. The figure shows *P* values for the *post hoc* test results. The figure also shows the results of paired Student's *t*-tests for each group. Black, white, and gray bars indicate sprint interval training (SIT), high-intensity interval aerobic training (HIAT), and continuous aerobic training (CAT) groups, respectively. *Significant changes ($P < 0.05$) were observed from preintervention to postintervention. $\dot{V}O_{2max}$, maximal oxygen consumption; LV, left ventricular; SV, stroke volume.

cardiomyocytes in the CAT group. Our MRI study revealed that the difference in impact on the heart among the three groups influenced changes in $\dot{V}O_{2max}$. On the other hand, in our study, we were unable to obtain more detailed cardiac function parameters with MRI. Further research using other techniques (19) is required to reveal more detailed effects of the HIAT on the heart.

Expansion of PV during exercise training has been well documented. Covertino (4) explained that a 1% increase in PV has been associated with a 1% reduction in exercise HR. In our study, $\dot{V}O_{2max}$ increased significantly in all three groups alongside a reduction in HR and elevated SV. Nevertheless, increased PV was not observed in any of the three

groups. Previous studies (3,36) showing increased PV with improved $\dot{V}O_{2max}$ have used Evan's blue dye technique to measure PV, whereas we used Dill's formula for PV estimation. The difference in PV measuring seemed to produce different results. Another SIT study (35) using Dill's formula was consistent with our results.

Previous studies showed the effects of the 4×4 HIAT protocol on coronary artery disease (13,39), cardiac rehabilitation (25), and metabolic syndrome (32,34). Further research is needed to investigate whether a lower-volume HIAT protocol (e.g., the 3×3 protocol in our study) would have similar effects on these diseases. On the other hand, we anticipate that our 3×3 HIAT protocol will become a time-efficient means of exercise for astronauts during space missions because astronauts in a microgravity environment experience a severe decrease in $\dot{V}O_{2max}$ (21) and myocardial atrophy (26).

In summary, our primary hypothesis is supported by the significantly increased $\dot{V}O_{2max}$ seen in the HIAT group compared with the CAT group. Our secondary hypothesis is partly supported by the significant increase in LV mass, that is, physiological cardiac hypertrophy, seen in the HIAT group, although this LV mass increase was also seen in the SIT group, which challenges our secondary hypothesis. Consequently, our study revealed that $\dot{V}O_{2max}$ improvement with the HIAT was greater than with the CAT despite the HIAT being performed with a far lower volume and in far less time than the CAT, suggesting the HIAT has potential as a time-efficient training mode to improve $\dot{V}O_{2max}$ in sedentary adults.

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