Low-Frequency Severe-Intensity Interval Training Improves Cardiorespiratory Functions

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ABSTRACT

NAKAHARA, H., S.-Y. UEDA, and T. MIYAMOTO. Low-Frequency Severe-Intensity Interval Training Improves Cardiorespiratory Functions. Med. Sci. Sports Exerc., Vol. 47, No. 4, pp. 789–798, 2015. Purpose: The present study investigated the effects of severe-intensity interval training at a frequency of once a week on cardiorespiratory function at rest and during exercise. Methods: Fourteen young healthy males were randomly assigned to either an interval training group or control group. Cardiorespiratory function was investigated by incremental maximal exercise test and constant work rate submaximal exercise test before and after the intervention period in all subjects. Submaximal exercise test was conducted at two work rates (80% ventilatory threshold (VT) level and 100% VT level plus 50% of the difference between VT and peak oxygen consumption (VO2peak)) for 8 min; the same work rates and duration were used before and after training. Left ventricular adaptations were assessed by echocardiography under supine resting conditions before and after training. In the interval training group, seven subjects performed cycle ergometer training once per week for 3 months. The training consisted of three bouts of exercises to volitional fatigue at 80% maximum work rate. Results: Increased VO2peak (+13%, P = 0.015), VT (+21%, P = 0.001), and left ventricular posterior wall thickness (+18%, P = 0.002) and reduced minute ventilation (~12%, P = 0.032) and blood lactate concentration (~16%, P = 0.025) during high-intensity exercise were observed after the training program compared with baseline. Although not significant, VO2 and cycling economy (VO2 per work rate) during high-intensity exercise decreased slightly after training. Conclusion: The present results indicate that severe-intensity interval training, even when performed at a low frequency, markedly improves cardiorespiratory function as well as induces cardiac morphological adaptations involving left ventricular hypertrophy and cardiorespiratory metabolic response during submaximal exercise. The present findings may provide new insights for low-frequency, severe-intensity interval training in the field of sports science. Key Words: CARDIOVASCULAR ADAPTATIONS, INTERVAL TRAINING, EXERCISE INTENSITY, EXERCISE DURATION

High-intensity interval training has been reported to confer beneficial effects not only in athletes (22) but also in patients with metabolic syndrome or respiratory and cardiovascular diseases by improving the patients’ cardiorespiratory function and disease state, ultimately enhancing their quality of life (33,37). In recent studies, high-intensity interval training was found to be more effective in improving cardiorespiratory function and endurance performance than continuous training (17,33,37). Furthermore, Wisløff et al. (37) reported that high-intensity interval training in heart failure patients was superior to moderate-intensity training with regard to reversal of left ventricular remodeling, aerobic capacity, and quality of life. They indicated that the risk of a cardiovascular event was low after high-intensity exercise training in a cardiovascular rehabilitation setting.

According to the American College of Sports Medicine (ACSM) guidelines on endurance training, moderate- to high-intensity continuous training and interval training performed at a frequency of three to five times a week for a period of 2 to 3 months effectively improve cardiorespiratory functions (1). The guidelines also indicate that training at a frequency of less than twice a week does not generally result in a meaningful increase in maximum oxygen consumption (VO2peak). On the other hand, Pollock (28) showed that even at a low frequency of once per week for 20 wk, a high-intensity endurance training program executed for 30 min induced 8% increase in VO2peak. Huttunen et al. (19) also reported that patients with insulin-dependent diabetes mellitus who underwent 3-month endurance training once a week at low to moderate intensity for 45 min achieved 9.5% increase in VO2peak. Furthermore, in a recent cross-sectional study focusing on lifelong exercise frequency as an index of exercise dose, Carrick-Ranson et al. (6) reported that maximal
arteriovenous oxygen difference (oxygen extraction) was greater in trained seniors who performed six exercise sessions per week. They also observed that even a low frequency (two to three sessions per week) of lifelong exercise improved oxygen extraction during maximal exercise. Goodman et al. (15) reported that moderate-intensity endurance training of only six times elicited rapid adaptation of left ventricular function including stroke volume during exercise. These previous findings thus indicate that physiological adaptations of the cardiorespiratory system may occur even after a small number of training sessions by selecting the appropriate mode of training, frequency, length, and intensity of the training stimulus. On the other hand, increases in muscle soreness, pain score, and serum creatine kinase induced by maximal effort exercise do not fully recover by 5 d after exercise (8). Therefore, development of a low-frequency training program may be helpful to reduce the risk of overtraining syndrome, participant burden, and program cost and increase program adherence (2,13,14). According to the above reports, the low frequency of once a week used in previous studies appears to be efficacious for cardiopulmonary improvement.

Hickson et al. (18) reported that improvement in VO2max is intensity dependent, and that training intensity was more critical to maintain cardiorespiratory function at given training levels than either exercise frequency or duration. Indeed, Matsuo et al. (23) reported that compared with a continuous training program, a high-intensity interval training program with far lower volume and shorter time achieved greater improvement in VO2max. Thus, accumulating evidence has indicated that interval training at a higher intensity level to ensure an adequate total work (exercise intensity–duration) is important to achieve training effects (28,34). Animal studies provide further evidence for the importance of exercise intensity, showing that high aerobic intensity of exercise training yields effects at a greater magnitude than low to moderate exercise intensity, including the effect on cardiac hypertrophy (20,21). Kemi et al. (20) demonstrated that changes in VO2max closely paralleled cardiomyocyte dimensions and contractile capacity, and that as with intensity, training volume was an important stimulus for improving exercise performance.

Based on the above findings in the literature, we hypothesized that once a week and low-volume severe-intensity interval training induces cardiac morphological adaptations involving left ventricular hypertrophy and improves cardiorespiratory function during exercise and exercise performance. In the present study, we adopted a training program of severe-intensity exercise consisting of three bouts of exercise, each continued until volitional fatigue. Studies of exercise training at low frequency may provide new insights in defining the optimal interval training methods not only in the field of sports science but also for exercise therapy in the clinical setting. However, few studies have investigated the effectiveness of once a week and low-volume severe-intensity interval training programs. In addition, the effects of severe-intensity interval training on exercise performance and/or cardiorespiratory and metabolic functions at rest and during exercise at different intensities are poorly studied, especially at a low frequency of once a week.

Previous studies have elucidated that improved cardiac function reflected by an increase in maximum cardiac output is the major mechanism involved in the improvement of VO2max through endurance training (15,35). Many cross-sectional studies in the past have verified that these cardiac morphological changes vary greatly depending on the type of training (3,27). However, there is no report of longitudinal study that investigates the effects of low frequency of once a week and low-volume severe-intensity interval training on cardiorespiratory function under maximal and submaximal exercise conditions and at the same time examined the cardiac morphological and functional changes in the same subjects. In the present study, in addition to examination of cardiorespiratory function, we also followed the training-induced cardiac morphological changes using an echocardiographic device in the same subjects.

**METHODS**

**Subjects**

Fourteen healthy male volunteers (age, 20.6 ± 2.5 yr; range, 18 to 29 yr; height, 174.2 ± 6.2 cm; weight, 70.8 ± 14.5 kg) without cardiovascular risk factors participated in the study. All subjects were informed about the experimental procedures, potential risks, and discomfort and signed an informed consent form. Participants were familiar with performing maximal cycle ergometer exercise and the laboratory procedures for obtaining cardiorespiratory data. The protocol was submitted to and approved by the Ethical Committee of the Morinomiya University.

**Experimental Procedures and Protocols**

Subjects were randomly assigned to one of the two following groups; interval training group (n = 7) and control (n = 7). For 24 h preceding the day of exercise tests, the subjects were instructed to avoid strenuous exercise and to continue their usual diet, but to avoid food with a high salt content. Food, alcohol, and caffeine were prohibited 4 h before each test. Each subject underwent both maximal and submaximal exercise tests before and after the exercise training program. Before the submaximal exercise test, an intravenous catheter (0.47 mm ID, 24-gauge) for blood collection was placed in the forearm of the subject who was seated in a comfortable chair.

**Maximal exercise test.** A computer-controlled bicycle ergometer (232CXL; Combi Co., Tokyo, Japan) with an incremental protocol was used to assess VO2max, maximal work rate, and ventilatory threshold (VT) by the V-slope method (4). The work rate was set at 20 W initially and increased by 1 W every 3 s (a total of 20 W·min⁻¹) until the subject could no longer maintain the pedaling frequency of 60 rpm despite strong verbal encouragement. The criteria for the achievement of VO2max were a plateau in O2 uptake.
(\dot{V}O_2) despite an increased work rate and an RER above 1.10. The above-mentioned criteria are widely adopted in the literature of exercise physiology as primary validation for the attainment of \dot{V}O_{2\text{max}}. On the other hand, previous reports advocated the use of secondary criteria to validate \dot{V}O_{2\text{max}} because an RER above 1.10 may lead to significant under-measurement of \dot{V}O_{2\text{max}} (10,30).

**Submaximal exercise test.** Four to 6 d after the maximal exercise test, each subject underwent constant work rate exercise tests at two different work rates. The work rates were chosen on the basis of the VT and \dot{V}O_{2\text{max}} measured in the maximal exercise test under baseline condition for each subject. The two work rates represented 80% work intensity at the subject’s VT (low intensity) and 100% of the VT level plus 50% of the difference between VT and peak \dot{V}O_2 (high intensity). Subjects were evaluated sequentially during 2 min of rest followed by 8 min of cycling exercise on a bicycle ergometer at low intensity and then 8 min of exercise at high intensity. Exercise intensity was increased in a stepwise fashion to obtain work rates that corresponded to low and high intensities. Before and after the training program, the submaximal exercise test was conducted at the same work rates and for identical duration. In all experimental sessions, the pedaling speed was maintained between 55 and 60 rpm.

**Exercise training program.** Interval training was conducted at one session per week for 3 months. The exercise training program involved bicycle ergometer exercise. Each training session consisted of three bouts of cycling at 80% of maximum work rate. Each subject completed a bout until volitional fatigue. Interval training sessions are generally prescribed on the basis of a combination of independent variables, namely, intensity of training, duration of training (exercise time), number of training bouts, and recovery period. Previous studies indicate that the potential benefits of interval training are derived through varying training bouts of interval training, ranging from 3 bouts to 12 bouts (17,22,24,37). Optimal training bouts are yet to be defined fully. In the present study, the training bout was prescribed at a minimum (i.e., 3 bouts) to set the highest exercise intensity level (intensity and duration) for each training bout performed until volitional fatigue. The recovery period between training bouts was fixed at 2 min of active recovery at 0 W, which provided an appropriate balance between intracellular restitution and maintenance of high oxygen uptake kinetics (32), and 1 min of rest. In the case that the exercise duration during the first bout (trial 1) was extended by 30% or more in response to the training program, the training work rate for the next session (second stage) was increased by 10% from the initial setting. Polar Accurex HR monitors (S810i; Polar, Tokyo, Japan) were attached to the chest during the interval training in all subjects to assess HR response and exercise duration. In each training session, all subjects were interviewed to ensure that they had not changed their routine activities excluding training. The control group did not perform any exercise other than routine activities during the training period.

**Experimental Apparatus and Measurements**

Respiratory and metabolic data during the experiments were recorded using an automatic breath-by-breath gas analyzing system (ARCO2000-MET; Arcosystem, Chiba, Japan) consisting of a differential pressure transducer, sampling tube, filter, suction pump, and mass spectrometer. We recorded expired flow, CO2 and O2 concentrations at 200 Hz, derived tidal volume (VT), respiratory frequency, minute ventilation (VE), end-tidal CO2 tension (PETCO2), VO2, and CO2 output (VCO2) from the digitized data. The gas analyzers were calibrated before each test. HR was monitored via a three-lead ECG (BSM-7201; Nihon Kohden, Tokyo, Japan), and the beat-to-beat HR was recorded continuously using a personal computer in on-line mode at a sampling rate of 200 Hz during each test. Echocardiographic examinations were conducted using M-mode echocardiography (SSD 6500; Aloka, Tokyo, Japan) and a sector probe, with the subject supine. M-mode measurements of left ventricle were derived from the parasternal long-axis view, and the septal and posterior wall thicknesses of the left ventricle and the dimensions of the ventricle cavities were measured in triplicate according to the recommendations of the American Society of Echocardiography (31). Left ventricular mass was calculated using Devereux’s formula (11). Left ventricular volumes were also computed using the Teichholz rule. All analyses were performed by one investigator. In the study, the intraobserver coefficients of variation calculated from the mean of three repeated echocardiographic measurements performed on the same day in the 14 subjects before interval training were 7.1% for septal thickness, 1.8% for left ventricular end-diastolic dimension, 5.9% for posterior wall thickness, and 6.0% for left ventricular mass. During the constant work rate submaximal exercise test, venous blood samples were measured using an automatic blood lactate analyzer (YSI-2300; Yellow Springs Instruments Inc., Yellow Springs, OH) within 1 min after sampling. The analyzer was calibrated against a standard lactate solution.

**Data Analysis**

Resting values of cardiorespiratory and metabolic variables averaged over 3 min before starting exercise were calculated for each subject. Average values of cardiorespiratory and metabolic variables during submaximal exercise were computed over a 2-min period from 6 to 8 min after the start of exercise. Venous blood samples (2.5 mL) were collected at 6.5 and 7.5 min for each work intensity, and the averaged value for each work intensity was analyzed.

**Statistical Analysis**

All data are expressed as mean ± SD. Differences between groups for all baseline physiological variables were analyzed using an unpaired Student’s t-test. Intra-individual comparisons of variables before and after the training program were performed by a two-way ANOVA (group × training). If an interaction existed, tests for simple effects were performed.
RESULTS

Subjects and training program. Height and body weight before intervention were not significantly different between the interval training group (172.1 ± 4.1 cm and 73.1 ± 16.3 kg) and control group (176.3 ± 7.6 cm and 66.9 ± 13.8 kg). Relative value of VO2max measured before the interval training period was also not significantly different between the interval training and control groups (46.1 ± 5.2 vs 48.3 ± 8.8 mL·kg⁻¹·min⁻¹).

Figure 1 shows the changes in HR over time of a representative subject in the interval training group recorded during interval training at the first and final (fourth) sessions of the first stage and at the first and final (seventh) session of the second stage of the training program. The maximum HR during three bouts of exercises interspersed with 3-min rest intervals reached approximately 200 beats·min⁻¹ in all the trials. The maximum exercise durations in all three trials were extended as training progressed.

In the interval training group (n = 7), the mean maximum exercise duration in trial 1 was extended from 406.1 ± 91.2 to 560 ± 122.4 s (40% increase, P = 0.006) in the first stage and from 263.4 ± 92.7 to 382.6 ± 94.8 s (45% increase, P = 0.04) in the second stage. The training work rate increased from 220.7 ± 12.1 to 259.8 ± 14.9 W after an average 5.0 ± 1.7 sessions of training. The mean total maximum exercise duration of all trials increased from 738.1 ± 190.8 to 914.6 ± 239.0 s (24%, P = 0.02) in the first stage and from 480.9 ± 84.1 to 652.6 ± 148.2 s (36%, P = 0.02) in the second stage. The subjects’ mean maximum HR recorded during training was 190.6 ± 5.0 beats·min⁻¹ in the first stage and 192.6 ± 4.0 beats·min⁻¹ in the second stage.

Maximal exercise stress test. Table 1 shows the mean maximum ventilatory variables, maximum HR, and maximum work rate during maximal exercise test before and after the intervention program in the interval training (n = 7) and control (n = 7) groups. ANOVA revealed a significant group–training interaction. The values of pre- and posttraining physiological variables are presented in Table 1.

![Figure 1](http://www.acsm-msse.org)

**TABLE 1.** Comparison of pre- and posttraining physiological variables during maximal exercise test.

<table>
<thead>
<tr>
<th></th>
<th>Interval Training Group (n = 7)</th>
<th>Control Group (n = 7)</th>
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<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>VO2max (mL·min⁻¹·kg⁻¹)</td>
<td>3226 ± 566</td>
<td>3717 ± 486</td>
</tr>
<tr>
<td>(mL·min⁻¹)</td>
<td>46.1 ± 5.2</td>
<td>51.0 ± 7.2</td>
</tr>
<tr>
<td>VT (mL·min⁻¹)</td>
<td>2096 ± 251</td>
<td>2524 ± 296</td>
</tr>
<tr>
<td>VE (L·min⁻¹)</td>
<td>159.7 ± 26.0</td>
<td>163.3 ± 29.5</td>
</tr>
<tr>
<td>VT, tidal volume</td>
<td>2529 ± 329</td>
<td>2761 ± 528</td>
</tr>
<tr>
<td>RR, respiratory frequency</td>
<td>63.2 ± 4.9</td>
<td>60.7 ± 12.9</td>
</tr>
<tr>
<td>HRmax (beats·min⁻¹)</td>
<td>192.8 ± 5.9</td>
<td>195.7 ± 4.8</td>
</tr>
<tr>
<td>Work rate max (W)</td>
<td>282.4 ± 16.6</td>
<td>307.9 ± 32.5</td>
</tr>
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</table>

Values are presented as mean ± SD.

*Significantly different (P < 0.05) from the pretraining value.
interaction for absolute $\dot{V}O_2^{max}$, relative $\dot{V}O_2^{max}$, VT, and maximum work rate, indicating greater changes in these variables in the interval training than that in the control group. Analysis of simple interaction effect also confirmed that the changes were due to the significant training effects in improving absolute $\dot{V}O_2^{max}$, relative $\dot{V}O_2^{max}$, VT, and maximum work rate in the interval training group. In the training group, absolute $\dot{V}O_2^{max}$, relative $\dot{V}O_2^{max}$, VT, and maximum work rate were improved by 13%, 11%, 21%, and 9%, respectively, after training compared with before training. On the other hand, ANOVA detected no group–training interaction for HR and other ventilatory variables.

Submaximal constant work rate exercise stress test. Mean work rates at low intensity and high intensity during submaximal constant work rate exercise test were not significantly different between the interval training and control groups (low intensity, 102 ± 15 vs 90 ± 15 W; high intensity, 159 ± 17 vs 150 ± 20 W). Figure 2 shows the changes in mean $\dot{V}O_2$, VE, and HR over time in the interval training and control groups recorded at rest and during low-intensity and heavy-intensity exercises. In both groups, $\dot{V}O_2$, VE, and HR increased with exercise and depended on the exercise intensity. In the interval training group, although VE at rest and during low-intensity exercise did not change after training compared with before training, a ventilatory depression effect was observed after training only under a high-intensity exercise condition. On the other hand, $\dot{V}O_2$ during high-intensity exercise decreased slightly after training compared with before training. Such difference was not observed in the control group.

ANOVA revealed a significant group–training interaction for VE during high-intensity exercise, indicating greater
change in this variable during high-intensity exercise in the interval training group than that in the control group. Analysis of simple interaction effect also confirmed that the change was due to the significant training effect in ameliorating V̇E in the interval training group. In the interval training group, V̇E during high-intensity exercise decreased by 12% after training compared with before training. On the other hand, ANOVA confirmed no group–training interaction for V̇O2 and HR during low- and high-intensity exercise.

Figure 3 shows the changes in mean blood lactate concentration in the interval training and control groups recorded at rest and at low- and high-intensity exercise. In both groups, blood lactate concentration increased with exercise and was dependent on the exercise intensity. In the interval training group, however, although blood lactate concentrations at rest and during low-intensity exercise did not change after training compared with before training, the posttraining concentration was lower than pretraining concentration only during high-intensity exercise. No difference was observed in the control group.

ANOVA revealed a significant group–training interaction for blood lactate concentration during high-intensity exercise, indicating greater change in this variable during high-intensity exercise in the interval training group than that in the control group. Analysis of simple interaction effect also confirmed that the change was due to the significant training effect in ameliorating blood lactate in the interval training group. In the interval training group, blood lactate concentration during high-intensity exercise decreased by 16% after training compared with before training (Table 2).

**Left ventricular morphology.** Table 3 shows mean left ventricular morphology as well as HR measured under supine resting condition before and after the intervention program in both the interval training and control groups. ANOVA revealed a significant group–training interaction for posterior wall thickness and left ventricular mass, indicating greater changes in these variables in the interval training than that in the control group. Analysis of simple interaction effect also confirmed that the changes were due to the significant training effects on posterior wall thickness and left ventricular mass in the interval training group. In the training group, posterior wall thickness and left ventricular mass at rest increased by 18% and 28%, respectively, after training compared with before training. On the other hand, ANOVA detected no group–training interaction

| TABLE 2. Comparison of pre- and posttraining physiological variables during submaximal exercise test. |
|---------------------------------------------------------|------------------|------------------|------------------|
|                                                       | Low Intensity    | High Intensity   |                  |
|                                                       | Interval Training Group (n = 7) | Control Group (n = 7) | Interval Training Group (n = 7) | Control Group (n = 7) |
|                                                       | Pre  | Post | Pre  | Post | Pre  | Post | Pre  | Post | Pre  | Post |
| V̇O2 (mL·min⁻¹)                                        | 1677 ± 345       | 1731 ± 348       | 1419 ± 310       | 1343 ± 267       | 2503 ± 270       | 2408 ± 218       | 2326 ± 257       | 2342 ± 263       |
| VE (L·min⁻¹)                                           | 47.7 ± 11.9      | 48.3 ± 13.0      | 38.1 ± 7.8       | 36.8 ± 7.3       | 74.5 ± 12.0      | 65.4 ± 10.7*     | 65.0 ± 23.3       | 69.0 ± 14.5       |
| HR (beats·min⁻¹)                                       | 121.7 ± 13.4     | 126.9 ± 14.0     | 120.2 ± 10.9     | 115.6 ± 9.2      | 161.2 ± 9.3      | 161.1 ± 9.9      | 156.4 ± 8.7       | 153.8 ± 10.7      |
| LA (mmol·L⁻¹)                                          | 1.6 ± 0.5        | 1.3 ± 0.3        | 1.8 ± 0.7        | 2.0 ± 0.5        | 3.1 ± 0.5        | 2.5 ± 0.7*       | 3.2 ± 0.8         | 3.5 ± 0.8         |

Values are presented as mean ± SD.

*Significantly different (P < 0.05) from pretraining value.

V̇O2, oxygen consumption; VE, minute ventilation; LA, blood lactate concentration.
TABLE 3. Left ventricular morphology measured at rest before and following intervention.

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Interval Training Group (n = 7)</th>
<th>Control Group (n = 7)</th>
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<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
</tr>
<tr>
<td>LVEDd (cm)</td>
<td>4.9 ± 0.7</td>
<td>4.9 ± 0.7</td>
</tr>
<tr>
<td>LVESd (cm)</td>
<td>3.2 ± 0.4</td>
<td>3.2 ± 0.4</td>
</tr>
<tr>
<td>IVS (cm)</td>
<td>0.8 ± 0.2</td>
<td>0.9 ± 0.1</td>
</tr>
<tr>
<td>PWT (cm)</td>
<td>1.0 ± 0.1</td>
<td>1.2 ± 0.2*</td>
</tr>
<tr>
<td>LV mass (g)</td>
<td>181.6 ± 95.2</td>
<td>218.0 ± 73.9*</td>
</tr>
<tr>
<td>HR (beats·min⁻¹)</td>
<td>59.1 ± 12.2</td>
<td>59.1 ± 11.0</td>
</tr>
<tr>
<td>Stroke volume (mL)</td>
<td>87.7 ± 44.5</td>
<td>90.3 ± 44.1</td>
</tr>
<tr>
<td>Cardiac output</td>
<td>5.0 ± 2.3</td>
<td>5.1 ± 2.2</td>
</tr>
<tr>
<td>(L·min⁻¹)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Ejection fraction (%)</td>
<td>69.6 ± 5.4</td>
<td>71.0 ± 8.3</td>
</tr>
</tbody>
</table>

Values are presented as mean ± SD.
*Significantly different (P < 0.05) from pretraining value.
LV, left ventricular; LVEDd, left ventricular end-diastolic dimension; LVESd, left ventricular end-systolic dimension; IVS, interventricular septal thickness; PWT, posterior wall thickness.

DISCUSSION

The present study demonstrated that severe-intensity interval training performed at a low frequency of once per week induced the following adaptive changes in resting cardiac morphology and cardiorespiratory responses during maximal and submaximal exercise. 1) As indicators of maximal aerobic capacity, VO₂max and VT increased significantly by 13% and 21%, respectively, after training compared with before training. Furthermore, exercise performance was drastically improved. 2) Lower VE response (12%) and a significant decrease in blood lactate concentration (16%) during submaximal exercise test at high intensity were observed after training compared with before training. Although not significant, VO₂ and cycling economy (VO₂ per work rate) during high-intensity exercise decreased slightly after training. 3) Cardiac morphological changes mainly as ventricular wall hypertrophy, observed as thickening of resting posterior wall thickness (18%) and enlargement of left ventricular mass (28%), were found after training compared with before training.

Effects of interval training on cardiorespiratory function at maximal exercise. Many studies using either continuous training or interval training have reported that endurance training in general increases VO₂max (7,17, 18,29). For example, Casaburi et al. (7) found that young normal male subjects who underwent continuous training at an intensity of approximately 180 beats·min⁻¹ for 45 min·d⁻¹, five times a week for 8 wk achieved 14% increase in VO₂max. Poole and Gaesser (29) assigned young normal male subjects to 35-min continuous training at an intensity of 70% VO₂max or interval training consisting of 10 sets of 2-min exercise at approximately 105% VO₂max interspersed with rest intervals. The two training procedures were designed to be equal to total work and were conducted at a frequency of three times a week for 8 wk. Comparison of the two groups showed similar increases in VO₂max by both continuous training (20%) and interval training (15%). On the basis of the above reports, ACSM currently recommends that as the lower threshold for endurance training for the purpose of improving cardiorespiratory function, the training should have an intensity at 40%–49% of the maximal oxygen uptake reserve or 55%–64% of maximum HR, conducted at a frequency of three to five times a week, for a duration of at least 20 min or longer (1). The interval training program designed in the present study consisted of three bouts of exercise at an intensity of 80% of the maximal work rate (obtained from a maximal exercise stress test) executed until volitional fatigue, conducted at a low-frequency of once a week for a total of 12 times. Despite a markedly lower exercise frequency than is recommended by ACSM, we obtained a similar rate of VO₂max increase as in the above-mentioned previous studies.

Laursen et al. (22) showed that even at a low frequency of twice per week for a short duration of 4 wk, high-intensity exercise executed until volitional fatigue induced 8% increase in VO₂max. A few studies showed that continuous training at a frequency of once a week was the effect on improved VO₂max (19,28) (see Introduction). Burgomaster et al. (5) found that intense sprint interval training at a frequency of three times per week over 2 wk increased resting muscle glycogen contents and endurance capacity during cycling exercise. Their work indicates that intense interval exercise is a time-efficient strategy to induce rapid adaptation in skeletal muscles and exercise performance compared with traditional endurance training. These findings support our results. Furthermore, among the three conditions of exercise intensity, duration, and frequency, exercise intensity has been reported to be most closely associated with the magnitude of VO₂max improvement (18). Indeed, Wisloff et al. (37) showed that in patients with postinfarction heart failure, the group that performed high-intensity training at over 90% of maximum HR achieved a greater VO₂max increase rate than the group that conducted moderate-intensity training. Although the exercise frequency of once per week adopted in our severe-intensity interval training program is incomparably lower than the frequencies used in previous study using interval training, we have shown that an adequate VO₂max improving effect almost equal to previous studies can be obtained. Therefore, the present findings provide new insight for the determination of low frequency for severe-intensity interval training even in the clinical setting.

Effects of interval training on cardiorespiratory metabolic response during submaximal exercise. Poole and Gaesser (29) reported that interval training consisting of 10 bouts of 2-min exercise at 105% VO₂max conducted three times a week for 8 wk significantly increased VT by 46%. Furthermore, Casaburi et al. (7) reported that continuous exercise training as described above resulted in 34% increase in anaerobic threshold; especially, high-intensity exercise yielded 40% decrease in blood lactate level and 30% reduction in VE response. The findings obtained from the above-mentioned interval and continuous training procedures are almost consistent with the results of the interval training program in the present study.
training used in the present study. The present study thus provides evidence that even using a different mode of training and a low exercise frequency, physiological effects at the same or even higher magnitude compared with previous findings can be obtained if adequate exercise intensity, duration, and work rate are ensured.

Although the present study does not allow elucidation of the mechanism of ventilatory depression resulting from endurance training, a relationship between VE response and blood lactate concentration during exercise has been reported. Casaburi et al. (7) observed a high correlation between ventilatory depression and decrease in blood lactate concentration after endurance training and concluded that the decrease in blood lactate concentration during high-intensity exercise above lactate threshold plays a role in causing ventilatory depression. In the present study also, lower VE response and decrease in blood lactate concentration were observed during submaximal exercise test at high intensity, suggesting that the decrease in blood lactate concentration may contribute to the mechanism of ventilatory depression observed during low-frequency interval training. On the other hand, the mechanism of ventilatory depression caused by training may involve not only peripheral factors such as potassium, hydrogen ion, and catecholamines (25) but also adaptations of respiratory controller and/or thermoregulation (26,36). Further studies are required to examine this possibility during interval training.

Effects of interval training on left ventricular morphology. The present study demonstrated that severe-intensity interval training at once per week resulted in cardiac morphological changes including thickening of the posterior wall (18%) and increase in left ventricular mass (28%) measured at rest. Past studies investigating the effect of exercise training on heart morphology used either continuous training or a combination of interval and continuous training conducted at high frequencies. However, there is no report of longitudinal study examining changes in cardiac morphology and cardiorespiratory function together in subjects who undergo a training program consisting of low frequency of once a week interval training alone. Windecker et al. (35) reported increases in VO_{2max} (17%) and left ventricular mass (28%) after a training program of 60-min running or cycling at 80% peak HR conducted four times per week for 5 months. Cox et al. (9) reported that 40-min continuous training at 80%–90% VO_{2max} for 3 d combined with 5 × 5-min interval exercise for the remaining 3 d increased left ventricular end-diastolic diameter (LVEDd) (3%) and left ventricular mass (9%) in addition to augmenting VO_{2max} (32%). Hickson et al. (18) used a combined interval and continuous training program conducted six times per week for 10 wk and reported that the program resulted in increases in both LVEDd (6%) and left ventricular mass (19%). Fagard et al. (12) also reported that isometric exercise of the muscles of the upper arms by cycling primarily caused thickening of ventricular wall and myocardial contractility during exercise without increase in left ventricular internal diameter. Although supporting data are limited for the effect of once weekly training on left ventricular mass, the effects of only six training sessions on left ventricular performance (15) and endurance capacity (5) indicated beneficial adaptations after the exercise program. Goodman et al. (15) observed left ventricular adaptation response to exercise immediately after 6 d of endurance training at moderate intensity. Previous studies on the design of isometric training program reported that a low frequency of once weekly provided an effective training stimulus for development of muscle strength (13). Taken together, the exercise stimulus necessary to achieve left ventricular adaptation and endurance capacity in nonathletes with a high level of residual training plasticity may not require high frequency and high volume training if an adequate work intensity is ensured. On the other hand, earlier studies have demonstrated a decrease in HR both at rest and during exercise accompanied by an increase in LVEDd and stroke volume after interval and continuous exercise training (9,18). In the present study, a once weekly severe-intensity training program had no apparent benefits for HR, LVEDd, and stroke volume at rest compared with previous studies. The difference in results between studies may be explained by the fact that we used low-frequency and low-volume severe-intensity interval training program. Haykowsky et al. (16) observed that short-term intense altitude training of 3 wk elicited rapid adaptation of increased posterior wall thickness independent of left ventricular diastolic cavity dimension. Training-induced bradycardia at rest is also associated with cardiac vagal-mediated modulation, whereas neurally mediated change in HR is seen with longer period of training (15).

Implications. There is an urgent need to define the low-frequency exercise training conditions that improve cardiorespiratory function, not only for competing athletes but also for persons with low fitness level or patients with diseases. The present findings indicate that a good cardiorespiratory function improving effect is achievable even at an extremely low training frequency, if an adequate total work rate (exercise intensity and duration) is ensured in each training session. The training frequency of once per week that was proven to be effective in the present study is a frequency that can realistically be implemented without too much difficulty in our daily life. In the future, by exploring the low volume training conditions for achieving the training effects, including small total workload and short duration of one training session, progress will also be made in the once a week exercise prescription for patients with various diseases.

Limitations. Some limitations of the present study should be mentioned. First, the number of subjects used was relatively small. Using a larger number of subjects could increase the strength of the present study. Second, the training effects are strongly affected by the initial physical fitness level or training status of the subjects. The subjects in the study were normal range young males, and it remains unclear whether the severe-intensity interval training can induce similar results in athletes and patients. Especially, participants
in this study required substantial motivation during training period. Therefore, less motivated subjects, especially persons with low fitness level or patients with various diseases, may have difficulties in performing the severe-intensity interval training. Further study is thus needed to determine whether this severe-intensity training program can be used in persons with low fitness level or patients with various diseases. Finally, we only evaluated HR response at rest and during exercise as well as cardiac morphology at rest as variables of cardiovascular structure and function. Further studies are needed to examine whether the severe-intensity interval training affects other cardiac functions associated with general training, especially stroke volume and blood pressure during exercise.

CONCLUSION

The present study demonstrates that severe-intensity interval training implemented at a low frequency of once per week not only increases maximum oxygen consumption but also induces cardiac morphological adaptation involving left ventricular hypertrophy and cardiorespiratory metabolic response during submaximal exercise. These adaptive mechanisms may contribute to improve exercise performance during exercise. The present findings may provide new insights necessary for defining the low frequency of severe-intensity interval training not only in the field of sports science but also in prescribing exercise therapy in the clinical setting.

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