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Effect of aerobic exercise training followed by a low-calorie diet on metabolic syndrome risk factors in men

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Abstract *Background and aims:* Whether low-volume, high-intensity, interval training (HIIT) is an adequate exercise method for improving metabolic risk factors is controversial. Moreover, it is not known if performing a short-term, low-calorie diet intervention (LCDi) after a HIIT program affects risk factors. This study investigated how an 8-week, 3 times/week exercise intervention (EXi) incorporating either HIIT or moderate-intensity continuous training (MICT) followed by a 4-week LCDi affects risk factors.
Methods and results: Twenty-six male workers with metabolic risk factors (47.4 ± 7.1 years; cardiorespiratory capacity ($\dot{V}O_{2peak}$) of 28.5 ± 3.9 ml/kg/min) were randomly assigned to either the HIIT (3 sets of 3-min cycling with a 2-min active rest between sets, 180 kcal) or MICT (45 min, 360 kcal) group. After the EXi, all subjects participated in a 4-week LCDi (4 counseling sessions). During the EXi, $\dot{V}O_{2peak}$ improved more ($P < 0.05$) through HIIT (25.4 ± 14.6%) than through MICT (14.9 ± 12.8%), whereas improvements in body fat and HDL cholesterol were similar. During the LCDi, some risk factors improved further ($P < 0.05$) without any group differences, while $\dot{V}O_{2peak}$ in the HIIT group decreased ($P < 0.05$) to the same level as in the MICT group.
Conclusion: $\dot{V}O_{2peak}$ increased more with HIIT than with MICT during the EXi despite HIIT having a lower exercise volume than MICT, but this advantage of HIIT promptly disappeared through detraining. An intervention strategy consisting of 8 weeks of either HIIT or MICT followed by a 4-week LCDi has a positive effect on metabolic risk factors.
Clinical Trial Registration: UMIN11352.

Introduction

Low-volume, high-intensity interval training (HIIT) induces efficient metabolic adaptation and can be a time-efficient strategy to improve metabolic risk factors [1–4]. Some studies [5,6] showed that HIIT achieved an equal or greater improvement in metabolic risk factors compared with traditional, moderate-intensity continuous training (MICT). These studies emphasized the primacy of exercise intensity rather than its duration and volume; however, some researchers [7] dispute this, and believe that exercise volume is more important than intensity for improving metabolic risk factors [8,9]. Thus, it is still controversial as to whether low-volume HIIT is the best training method for improving metabolic risk factors.

Our previous studies [10–12] showed that an 8-week HIIT program improved cardiorespiratory capacity ($\dot{V}O_{2peak}$) more than MICT, despite HIIT (18 min, 180 kcal per session) having a substantially shorter duration and lower volume than MICT (45 min, 360 kcal). While these results were consistent with results of recent meta-

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analysis [13], the effects of this type of HIIT on metabolic risk factors remains unknown. In the present study, therefore, subjects with metabolic risk factors participated in an 8-week exercise intervention (EXi) in which they performed either HIIT or MICT to clarify whether low-volume HIIT is a time-efficient strategy for treating metabolic disorders.

On the other hand, many studies [14–16] have shown that calorie restriction, i.e., a low-calorie diet intervention (LCDi), has a considerable impact on improving metabolic risk factors, even if the duration is only 7–10 days [14,16]. These studies suggest that calorie restriction, per se, is the crucial factor for improving metabolic risk factors. It is reasonable to assume that adding a LCDi to the HIIT would improve metabolic risk factors even more, even if the duration of the LCDi was short. Generally, subjects in metabolic syndrome studies perform a concurrent program of both EXi and LCDi. While this strategy may generate synergistic effects, it can also be burdensome to many of these subjects due to time constraints and the changes to their lifestyle [17]. Separating the interventions, such as adding a short-term LCDi following the HIIT intervention, may be a possible solution to this problem. Although many studies show the effects that HIIT can have on human health, research is limited on HIIT interventions followed by a LCDi.

The purpose of this study, therefore, was 1) to test the hypothesis that MICT would have more impact than HIIT on some metabolic risk factors because the exercise volume of MICT is double that of HIIT, while $\dot{V}O_{2peak}$ would improve more with HIIT than MICT, and 2) to investigate how adding a 4-week LCDi following the 8-week EXi affects risk factors.

Methods

Research design

This study was a randomized, controlled trial conducted on Japanese male workers with metabolic risk factors. After baseline measurements, the subjects were randomly assigned to either the HIIT or MICT group stratified according to age and $\dot{V}O_{2peak}$. A computerized random number generator was used to select permutation blocks. Both exercise programs consisted of an 8-week, 3 times/week, supervised cycling exercise training. After the EXi, all subjects participated in a 4-week, non-exercise LCDi. The LCDi was the same for both HIIT and MICT groups. The protocol of this study was registered with an approved clinical trial registry (UMIN11352).

Participants

Participants were recruited through local newspaper advertisements. This study was conducted in accordance with the guidelines proposed in the Declaration of Helsinki. The Ethical Committee of the National Institute of Occupational Safety and Health, Japan reviewed and approved the study protocol. The aim and design of this study were explained to every participant before each gave their written, informed consent.

The inclusion criteria in this study were 1) male workers aged 30–59 years, 2) no participation in regular exercise activities ($\leq$1 session per week and $\leq$30 min per session) over the past year, and 3) patients who fit the Japanese definition for metabolic syndrome or pre-metabolic syndrome [18]. The Japanese definition for pre-metabolic/metabolic syndrome includes central obesity (abdominal circumference $\geq$85 cm) and one (pre-metabolic syndrome) or more (metabolic syndrome) of the following three components: 1) dyslipidemia (triglyceride (TG) $\geq$150 mg/dl and/or HDL cholesterol (HDLC) $<$ 40 mg/dl, or specific treatment for these lipid abnormalities); 2) hypertension (systolic blood pressure (SBP) $\geq$130 mmHg and/or diastolic blood pressure (DBP) $\geq$85 mmHg, or treatment of diagnosed hypertension); or 3) hyperglycemia (fasting plasma glucose (FPG) $\geq$110 mg/dl). The exclusion criteria were 1) participation in a weight reduction program within the past year, and 2) adverse medical issues (all candidates underwent a medical interview and a 12-lead resting ECG examination by a physician to confirm eligibility). Seventy four applicants were assessed for eligibility and 48 were excluded (25 did not meet inclusion criteria; 23 declined to participate). Consequently, 26 subjects participated in this study and completed the 8-week EXi and 4-week LCDi. There were no dropouts.

Exercise intervention

The EXi and detailed descriptions of the determination of each subject's exercise intensity have been published elsewhere [11]. All subjects participated in an 8-week, 3 days/week, supervised cycling program. Table 1 shows the detailed exercise protocols, total exercise duration, exercise energy expenditure [10] and average training workload. The exercise intensity for each subject was recalculated and adjusted following the mid-intervention (Week 4) $\dot{V}O_{2peak}$ measurements recording. Subjects were instructed to maintain their usual diet during the EXi. No major complications occurred during the EXi.

Low-calorie diet intervention

The 4-week LCDi included a once weekly dietary modification program with a trained dietician. The program was comprised of lectures, practical training sessions, and individual counseling (4 sessions of 90 min each). Detailed descriptions of the dietary program have been described in our previous study [19]. Briefly, the program is based on four food groups (FGs) method: FG 1 (dairy products and eggs), FG 2 (meat, fish, and beans), FG 3 (vegetables and fruits) and FG 4 (grains, oil, and sugar). To simplify calculations for energy intakes and nutrient balances, a cluster of foods equal to 80 kcal is equivalent to one point in this method. To consume a well-balanced daily diet, each person could choose 3 points worth of food from each of FGs 1, 2, and 3 and 12 points worth of food from FG 4 (total of 21 points, 1680 kcal/day as a

Table 1 The two cycling exercises in this study.

	Protocol	Total time	Volume[a]	Average exercise workload at Week 8
HIIT	2 min (30 W, 60 rpm, warm-up)	18 min	180 kcal	30 W
	3 min (85%VO_{2peak}, 70–80 rpm)			180 ± 20 W
	2 min (50%VO_{2peak}, 60 rpm)			96 ± 13 W
	3 min (85%VO_{2peak}, 70–80 rpm)			180 ± 20 W
	2 min (50%VO_{2peak}, 60 rpm)			96 ± 13 W
	3 min (80%VO_{2peak}, 70–80 rpm)			168 ± 18 W
	3 min (30 W, 40–60 rpm, cool-down)			30 W
MICT	2 min (30 W, 60 rpm, warm-up)	45 min	360 kcal	30 W
	40 min (60–65%VO_{2peak}, 60 rpm)			88 ± 20 W
	3 min (30 W, 40–60 rpm, cool-down)			30 W

HIIT, high-intensity interval training; MICT, moderate-intensity continuous training.
[a] Our previous study[10] measured the exercise energy expenditure of the two cycling exercises.

rough indication). The subjects kept a daily log in which they recorded in detail every food they ate during the 4-week intervention period. Subjects were instructed to refrain from exercise activities during the LCDi.

Measurements

Anthropometric measurements

Anthropometric measurements were performed at baseline (Week 0), post-EXi (Week 9), and post-LCDi (Week 14). With the subjects in underwear and barefoot, 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). 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 made 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).

Cardiorespiratory capacity

The subjects underwent a maximal graded exercise test on a cycling ergometer (75XL III, Konami, Tokyo, Japan) to determine $\dot{V}O_{2peak}$ at baseline (Week 0), mid-EXi (Week 4), post-EXi (Week 9), and post-LCDi (Week 14). After a 2-min warm up at 15 watt (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 [20]. 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, i.e., $\dot{V}O_2$ and $\dot{V}CO_2$, 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. HRs at rest and during the exercise test were monitored using an electrocardiogram monitor (Dyna Scope, Fukuda Denshi, Tokyo, Japan).

Blood pressure and biochemical assays of blood

Blood pressure and biochemical assays of blood were measured at baseline (Week 0), post-EXi (Week 9) and post-LCDi (Week 14). One trained nurse measured the 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.

Blood samples were collected from the antecubital vein of each subject after a 12-h fast. Automated laboratory methods were used to measure serum glucose and lipids. HbA1c was determined with a latex agglutination method (Kyowa Medex, Tokyo, Japan), uric acid (UA) with a uricase method (Kyowa Medex, Tokyo, Japan) and fasting insulin (FINS) via an electrochemiluminescence immunoassay using Modular Analytics E170 (Roche Diagnostics Japan, Tokyo, Japan). LDL cholesterol was calculated according to Friedewald's formula [21] and HOMA-IR according to a previous study [22]; HOMA-IR = [(FINS, μU/ml) × (FPG, mg/dl)]/405. Serum samples were stored at −80 °C until analyzed. The inter- and intra-assay CV were <5% for all blood parameters.

Dietary assessments

Total energy intake in kilocalories and the amounts of each nutrient (carbohydrates, fat, and protein in grams) were assessed at baseline (Week 0), mid-EXi (Week 4) and mid-LCDi (Week 12) using a three-day weighed dietary record method. During each three day period, subjects photographed and recorded the names and amounts of every food item they ate. One skilled dietician collected the recorded sheets and photographs and codified the food items and food weights. The dietary data were converted to energy and nutrient data using a standard system software analysis tool (Eiyoukun version 6.0, Kenpakusya, Tokyo, Japan).

Statistical analysis

A priori power analysis was performed to determine the sample size. The primary outcome variable of this study

was the change in $\dot{V}O_{2peak}$ throughout the intervention. Based on data from our previous study [12], a 13% difference was assumed in the training effect between the HIIT and MICT with a standard deviation estimate of 10%. With an alpha error rate of 0.05 and statistical power of 80%, the minimal sample size was estimated in each group to be 11 subjects. Assuming subject attrition such as dropout, 13 subjects were recruited for each group in this study.

Values are expressed as the mean ± standard deviation. Paired Student's *t* tests were performed to test the significance of changes in values within groups. Cohen's *d* values were used as an effect size (ES) index. ANCOVA with adjustments for respective baseline values was applied to compare changed values between groups. When ANCOVA assumptions were violated, unpaired Student's *t*-tests were performed [23]. Chi-square tests were used to analyze categorical values. For the analyses, a two-tailed *P*-value $\leq$0.05 was considered statistically significant. SAS, version 9.3 (SAS Institute Japan, Tokyo, Japan) was used to analyze the data.

Results

Subject's characteristics and use of medications at baseline are shown in Table 2. There were no significant group differences at baseline between the two groups. Adherence to the interventions (i.e., attendance rates) was also similar between the two groups: EXi (HIIT 97.1 ± 6.2% vs. MICT 97.4 ± 2.7%) and LCDi (HIIT 100% vs. MICT 100%).

Table 3 shows mean measurement variables at baseline, post-EXi and post-LCDi along with effect sizes by group.

Table 2 Subject characteristics at baseline.

	HIIT (n = 13)	MICT (n = 13)	Group differences
Age, year	47.5 ± 7.0	47.4 ± 7.5	0.98
Height, cm	174.3 ± 7.0	171.0 ± 4.3	0.16
Body weight, kg	83.7 ± 7.7	80.1 ± 5.9	0.19
Body mass index	27.6 ± 2.5	27.4 ± 2.1	0.85
Abdominal circumference, cm	95.6 ± 5.9	94.0 ± 5.5	0.50
Medications, n			
Acetylsalicylic acid	0	1	0.31
Angiotensin receptor blockers	0	2	0.14
Calcium antagonists	0	3	0.07
DPP-4 inhibitors	1	0	0.31
Eicosapentaenoic acid	0	1	0.31
Fibrates	1	1	1.00
Glitazones	0	1	0.31
Metformin	2	1	0.54
Statins	0	2	0.14
Sulfonylureas	0	1	0.31
Xanthine oxidase inhibitors	1	1	1.00

Values are presented as the mean ± standard deviation.
HIIT, high-intensity interval training; MICT, moderate-intensity continuous training.
VO_{2peak}, peak oxygen consumption.

There were significant group differences in changed values of HR at rest (during the LCDi and during the EXi + LCDi) and $\dot{V}O_{2peak}$ (during the EXi and during the LCDi). During the EXi, $\dot{V}O_{2peak}$ improved more ($P < 0.05$) with HIIT (25.4 ± 14.6%) than with MICT (14.9 ± 12.8%). Meanwhile, during the LCDi, $\dot{V}O_{2peak}$ in the HIIT group decreased (−4.2 ± 8.0%) but remained the same in the MICT group (3.1 ± 9.8%). The number of subjects who increased their $\dot{V}O_{2peak}$ at least 10% during the EXi was significantly greater ($P < 0.01$) in the HIIT group (n = 13, 100%) than the MICT group (n = 7, 53.9%). On the other hand, the number of subjects who decreased their $\dot{V}O_{2peak}$ more than 5% during the LCDi was significantly greater ($P < 0.05$) in the HIIT group (n = 7, 53.9%) than the MICT group (n = 2, 15.4%). Body weight, abdominal circumference and body fat decreased ($P < 0.05$) during the EXi and the values decreased further ($P < 0.05$) during the LCDi in both groups. Meanwhile, body muscle, SBP and DBP did not change during the EXi, but the values decreased ($P < 0.05$) during the LCDi in both groups. Conversely, HR at rest decreased ($P < 0.05$) and HDLC increased ($P < 0.05$) in both groups during the EXi, but did not change during the LCDi. During the entire study period (EXi + LCDi), abdominal circumference, body fat, blood pressure, HbA1c, UA and $\dot{V}O_{2peak}$ improved ($P < 0.05$) in both groups. Fig. 1 shows the changes in $\dot{V}O_{2peak}$, body fat and HDLC.

Discussion

The major finding in this study is that, compared to MICT, HIIT had a greater effect on $\dot{V}O_{2peak}$ and a similar effect on HDLC even though the exercise volume of HIIT (54 min/week) was substantially lower than the MICT exercise volume (135 min/week). The other key finding of this study is that the advantage on $\dot{V}O_{2peak}$ with HIIT was promptly lost during the 4-week detraining period. Furthermore, the study suggests that adding a LCDi of only 4 weeks after an EXi that incorporates either HIIT or MICT induces significant effects on metabolic syndrome.

Despite the lower volume and shorter duration of the HIIT compared to the MICT, we saw a greater $\dot{V}O_{2peak}$ improvement with HIIT than with MICT during the 8-week EXi. This was consistent with previous studies [5,11–13]. All 13 subjects in the HIIT group completed the EXi (no dropouts) and increased their $\dot{V}O_{2peak}$ more than 10%, suggesting that the 3 × 3 HIIT in our study may be an adequate method to improve $\dot{V}O_{2peak}$ in low-fitness individuals. During the 4-week LCDi (detraining) period, however, $\dot{V}O_{2peak}$ in the HIIT group apparently decreased, whereas $\dot{V}O_{2peak}$ in the MICT group remained unchanged. This rapid decrease of $\dot{V}O_{2peak}$ after exercise cessation in the HIIT group may have occurred for several reasons. First, the $\dot{V}O_{2peak}$ level at the start of detraining (Week 9) was related to the magnitude of $\dot{V}O_{2peak}$ reduction during the detraining period, i.e., the higher the level of $\dot{V}O_{2peak}$ at Week 9, the greater it declined during detraining [24,25]. Second, the exercise volume of the HIIT may have been insufficient and thus led to a rapid $\dot{V}O_{2peak}$ reduction. Nevertheless, at the final measurement session (at Week

Table 3 Measurement values at Weeks 0, 9 and 14 by exercise group.

	Week 0 (baseline)		Week 9 (after EXi)		Week 14 (after LCDi)		Effect size From weeks 0–9 (EXi)		Group differences[c]	Effect size From weeks 9–14 (LCDi)		Group differences[c]	Effect size From weeks 0–14 (Exi + LCDi)		Group differences[c]
	HIIT	MICT	HIIT	MICT	HIIT	MICT	HIIT	MICT		HIIT	MICT		HIIT	MICT	
Body weight, kg	83.7 ± 7.7	80.1 ± 5.9	82.8 ± 7.1[a]	79.2 ± 5.8[a]	79.4 ± 7.8[b,a]	76.1 ± 5.9[b,a]	−0.11	−0.16	0.53	−0.47	−0.53	0.59	−0.56	−0.68	0.70
Abdominal circumference, cm	95.6 ± 5.9	94.0 ± 5.5	93.9 ± 5.5[a]	91.7 ± 5.1[a]	90.3 ± 6.4[b,a]	88.4 ± 5.2[b,a]	−0.29	−0.44	0.50	−0.60	−0.64	0.81	−0.85	−1.06	0.68
Hip circumference, cm	100.8 ± 4.6	98.8 ± 3.8	98.8 ± 4.9[a]	96.4 ± 3.2[a]	97.1 ± 4.9[b,a]	94.9 ± 3.5[a]	−0.43	−0.68	0.42	−0.35	−0.43	0.72	−0.79	−1.06	0.91
Thigh circumference, cm	53.9 ± 3.2	53.6 ± 2.8	53.8 ± 3.3	53.4 ± 2.6	51.4 ± 2.8[b,a]	50.9 ± 2.0[b,a]	−0.01	−0.09	0.70	−0.78	−1.06	0.77	−0.80	−1.10	0.59
Body muscle, kg	60.5 ± 4.7	59.0 ± 4.4	60.5 ± 4.5	58.7 ± 4.4	58.6 ± 4.6[b,a]	56.9 ± 4.3[b,a]	0.00	−0.08	0.37	−0.41	−0.42	0.90	−0.40	−0.50	0.64
Body fat, kg	21.5 ± 4.7	19.6 ± 2.7	20.8 ± 4.2[a]	19.1 ± 3.1[a]	19.2 ± 4.6[b,a]	17.9 ± 3.1[b,a]	−0.17	−0.19	0.78	−0.37	−0.37	0.10	−0.51	−0.58	0.16
HR at rest, bpm	72.6 ± 10.8	75.9 ± 10.6	66.8 ± 8.3[a]	72.2 ± 10.7[a]	65.2 ± 6.4[a]	74.2 ± 10.2	−0.61	−0.35	0.20	−0.21	0.19	0.04	−0.83	−0.16	0.03
SBP, mmHg	128 ± 10	132 ± 12	129 ± 10	134 ± 16	120 ± 10[b,a]	128 ± 16[b]	0.10	0.13	0.79	−0.90	−0.42	0.51	−0.81	−0.34	0.34
DBP, mmHg	88 ± 8	89 ± 9	86 ± 8	90 ± 10	80 ± 5[b,a]	83 ± 11[b,a]	−0.18	0.09	0.33	−0.89	−0.71	0.58	−1.05	−0.64	0.85
TC, mg/dl	207 ± 29	199 ± 22	209 ± 27	203 ± 26	191 ± 23[b,a]	197 ± 33	0.08	0.13	0.90	−0.71	−0.20	0.23	−0.60	−0.10	0.33
HDLC, mg/dl	47 ± 6	45 ± 6	50 ± 7[a]	48 ± 9[a]	49 ± 9	47 ± 6	0.53	0.42	0.93	−0.12	−0.20	0.83	0.30	0.28	0.81
LDLC, mg/dl	129 ± 27	122 ± 22	134 ± 26	122 ± 26	122 ± 22[b]	122 ± 35	0.17	0.02	0.52	−0.48	0.00	0.30	−0.28	0.02	0.54
TG, mg/dl	156 ± 58	163 ± 79	128 ± 29	161 ± 82	102 ± 39[b,a]	144 ± 90	−0.61	−0.03	0.21	−0.76	−0.20	0.66	−1.09	−0.23	0.05
TC/HDLC ratio, mg/dl	4.48 ± 0.63	4.51 ± 0.89	4.24 ± 0.67[a]	4.34 ± 1.13	4.02 ± 0.79[a]	4.21 ± 0.63	−0.37	−0.17	0.69	−0.30	−0.14	0.49	−0.64	−0.39	0.40
FPG, mg/dl	102 ± 24	109 ± 32	99 ± 20	101 ± 15	95 ± 18[a]	102 ± 19	−0.17	−0.32	0.50	−0.19	0.07	0.08	−0.35	−0.27	0.29
HbA1c, %	5.8 ± 1.0	5.8 ± 0.9	5.7 ± 0.9	5.6 ± 0.7[a]	5.5 ± 0.6[a]	5.6 ± 0.7[a]	−0.09	−0.22	0.29	−0.25	−0.04	0.17	−0.34	−0.26	0.27
HbA1c, mmol	39.5 ± 10.5	40.1 ± 10.0	38.5 ± 9.7	38.1 ± 7.5[a]	36.3 ± 6.7[a]	37.8 ± 7.3[a]	−0.10	−0.23	0.32	−0.26	−0.04	0.14	−0.36	−0.26	0.21
FINS, μU/ml	13.3 ± 10.7	14.1 ± 9.0	10.7 ± 3.0	11.6 ± 7.8	8.0 ± 3.1[b]	9.5 ± 3.5	−0.33	−0.30	0.76	−0.89	−0.34	0.30	−0.67	−0.67	0.27
HOMA-IR	3.6 ± 3.8	4.2 ± 4.3	2.6 ± 0.7	3.0 ± 2.2	1.9 ± 0.8[b]	2.5 ± 1.2	−0.36	−0.37	0.63	−0.97	−0.27	0.19	−0.62	−0.55	0.16
UA	6.3 ± 1.0	7.0 ± 1.0	6.0 ± 1.5	6.4 ± 1.0[a]	5.7 ± 1.2[a]	6.1 ± 0.8[a]	−0.21	−0.61	0.18	−0.22	−0.32	0.62	−0.50	−0.99	0.39
VO_{2peak}, ml/kg/min	28.4 ± 3.2	28.5 ± 4.5	35.4 ± 4.0[a]	32.9 ± 6.6[a]	33.9 ± 5.0[a]	33.8 ± 6.9[a]	1.93	0.77	0.08	−0.33	0.14	0.05	1.32	0.90	0.89
VO_{2peak}, L/min	2.37 ± 0.33	2.28 ± 0.40	2.93 ± 0.39[a]	2.59 ± 0.50[a]	2.68 ± 0.37[b,a]	2.57 ± 0.53[a]	1.55	0.68	0.03	−0.66	−0.04	0.02	0.88	0.62	0.87
HR_{max} during exercise testing, bpm	177.2 ± 11.2	177.5 ± 19.2	177.2 ± 9.1	175.9 ± 19.7	178.2 ± 10.0	176.9 ± 19.3	0.00	−0.08	0.55	0.10	0.05	1.00	0.09	−0.03	0.64
Total energy intake, kcal/d	2197 ± 456	2124 ± 248	2120 ± 372	2013 ± 303	1741 ± 225[b,a]	1632 ± 410[b,a]	−0.18	−0.40	0.54	−1.23	−1.06	0.52	−1.27	−1.45	0.47
Carbohydrates intake, g/d	273 ± 68	287 ± 37	267 ± 43	270 ± 46	229 ± 41[b,a]	217 ± 50[b,a]	−0.10	−0.41	0.71	−0.92	−1.08	0.53	−0.78	−1.58	0.47
Fat intake, g/d	77 ± 23	69 ± 12	76 ± 22	60 ± 14	55 ± 9[b,a]	51 ± 17[b,a]	−0.06	−0.66	0.30	−1.28	−0.62	0.96	−1.29	−1.23	0.63
Protein intake, g/d	75 ± 16	72 ± 10	77 ± 14	69 ± 15	69 ± 12	63 ± 20	0.16	−0.21	0.21	−0.66	−0.34	0.88	−0.44	−0.55	0.44

Values are presented as the mean ± standard deviation.

DBP, diastolic blood pressure; EXi, exercise intervention; FINS, fasting insulin; FPG, fasting plasma glucose; HbA1c, hemoglobin A1c; HDLC, high-density lipoprotein cholesterol; HIIT, high-intensity interval training; HOMA-IR, homeostasis model assessment insulin resistance; HR, heart rate; LCDi, low-calorie diet intervention; LDLC, low-density lipoprotein cholesterol; MICT, moderate-intensity continuous training; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; UA, uric acid; VO_{2peak}, peak oxygen consumption.

[a] Significant changes ($P < 0.05$) observed from Week 0.

[b] Significant changes ($P < 0.05$) observed from Week 9.

[c] ANCOVAs with adjustments for respective baseline values were applied to compare changed values between groups. When ANCOVA assumptions were violated, unpaired t-tests were applied and all results were consistent with the ANCOVA results.

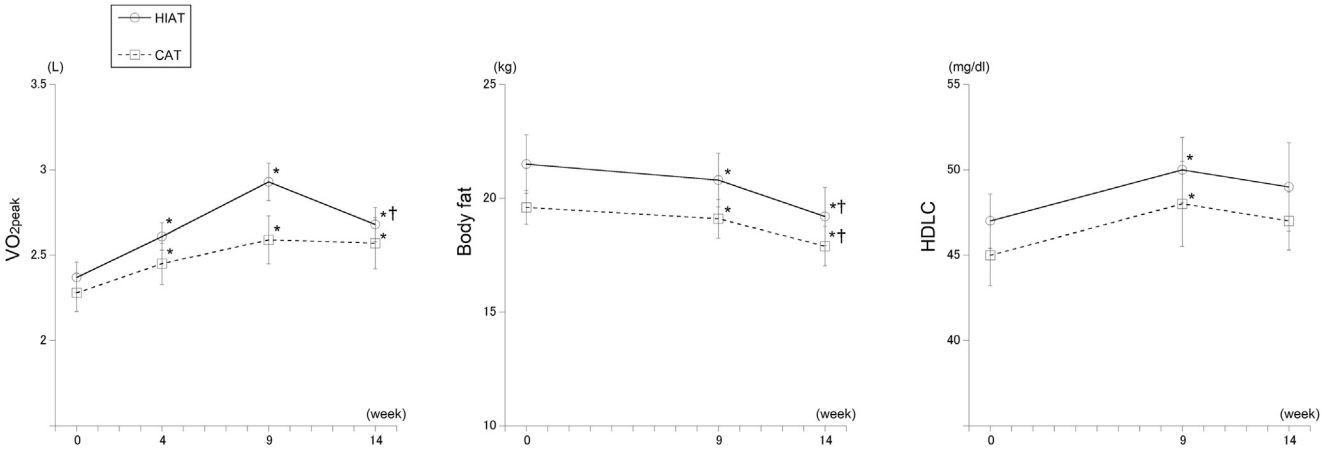

Figure 1 Changes in values during the entire study period by exercise group. The unbroken line indicates the high-intensity interval training (HIIT) group and the dotted line indicates the moderate-intensity continuous training (MICT) group. *Significant changes ($P \leq 0.05$) observed from Week 0. †Significant changes ($P \leq 0.05$) observed from Week 9. HDLC, high-density lipoprotein cholesterol; $\dot{V}O_{2peak}$, peak oxygen consumption.

14), $\dot{V}O_{2peak}$ improvement over baseline in the HIIT group (20.1 ± 17.1%) was similar and no lower than the MICT group (18.3 ± 15.3%). An improvement in cardiorespiratory capacity is meaningful for subjects with metabolic syndrome because cardiorespiratory capacity is a powerful predictor of mortality [26].

The present study further showed a similar and significant improvement in HDLC with both HIIT and MICT. Improved blood values with HIIT is consistent with other studies [6,27,28]. Tjonna et al. [27] showed that a 2 times/week 4 × 4 HIIT (4 sets of 4-min high-intensity running with a 3-min active rest between sets) for 3 months improved glucose metabolism among overweight adolescents. Their other study [6] also showed that the effects on patients with metabolic syndrome of the 4 × 4 HIIT performed 3 times/week for 16 weeks were superior to the effects of MICT. In addition, Nybo et al. [5] conducted a 2–3 times/week exercise intervention for 12 weeks and showed that glucose concentration decreased to a similar extent with both low-volume HIIT and high-volume MICT. In the present study, during the EXi, there were small to moderate effect sizes in many blood values in both groups, but these changes were not statistically significant except for HbA1c and UA in the MICT group. The length of time for the EXi (8 weeks) may not have been long enough to see significant changes [4,29], although a study using 24-h continuous glucose monitoring [2] showed that only 6 HIIT sessions in 2 weeks was enough to improve glucose control in patients with type 2 diabetes.

SBP and DBP did not change during the EXi, but they did improve during the LCDi in both groups. This suggests that calorie restriction has a strong influence on risk factors even if the duration is short [14,16]. However, in our study, it is possible that the EXi may have influenced the changes seen during the LCDi. Interestingly, there were relatively larger effect sizes in some blood values (e.g. total cholesterol, LDL cholesterol, TG, FPG, HbA1c, FINS and HOMA-IR) in the HIIT group compared to the MICT group during the LCDi, which presumes that the positive effects from HIIT may continue after exercise cessation. Until now, the strategy of adding a short-term LCDi after an EXi has not been well studied. Our results suggest that this type of intervention can be a highly effective and less burdensome way to improve metabolic syndrome in individuals, although further research is needed to confirm this phenomenon.

Our hypothesis is partly supported by the greater increase of $\dot{V}O_{2peak}$ seen with HIIT compared to MICT, but the study also revealed a rapid decrease in $\dot{V}O_{2peak}$ after stopping the HIIT. On the other hand, there was no large difference between HIIT and MICT in their effect on metabolic risk factors. The similar improvement in HDLC was especially meaningful because HIIT was performed with a far lower volume and in far less time than MICT, which challenges our hypothesis. The potential mechanisms for why such a low volume HIIT can positively influence $\dot{V}O_{2peak}$ and HDLC are not completely understood, but exercise intensity may play a key role in improving cardiac function [12,30] or skeletal muscle oxidative capacity [2].

In conclusion, our study revealed that, despite HIIT having lower exercise volume than MICT, the two training methods had similar effects on body fat and HDLC in subjects with metabolic risk factors. Furthermore, the increase in $\dot{V}O_{2peak}$ with HIIT was greater than with MICT, but this advantage on $\dot{V}O_{2peak}$ with HIIT promptly disappeared through detraining. Finally, the study suggests that an intervention strategy consisting of 8 weeks of either HIIT or MICT followed by 4 weeks of a low-calorie diet has a significant positive impact on metabolic syndrome.

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