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Effects of Lifestyle Modifications on Improvement in the Blood Lipid Profiles in Patients with Dyslipidemia

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Abstract

Aim: This study was designed to clarify the difference in the effects of aerobic exercise training and diet on the improvement in the blood lipid profiles in patients with dyslipidemia.

Subjects and Methods: The study enrolled 86 patients with dyslipidemia [34 males and 52 females; age, 55 ± 10 years (33 to 71 years); low-density lipoprotein cholesterol (LDL-C), 150 ± 33 mg/dl (74 to 206 mg/dl); high-density lipoprotein cholesterol (HDL-C), 54 ± 12 mg/dl (35 to 87 mg/dl) and triglycerides, 165 ± 65 mg/dl (68 to 318 mg/dl)]. The subjects were randomly allocated to exercise training (n=42) or diet (n=44) group. These patients in the exercise training group were instructed to exercise for more than 300 min per week at the lactate threshold intensity. In the diet group, the target caloric intake was 25 kcal/kg of ideal body weight [height $(m)^2 \times 22$] according to the guideline of the Japan Society for the Study of Obesity.

Results: After the 12-week intervention, the LDL-C, triglyceride level and body weight decreased in both the exercise training and diet groups (p<0.05). There was no significant interaction effect for group × time on the LDL-C, fasting triglyceride level or body weight between the groups. The HDL-C increased only in the exercise training group, and a significant interaction effect for group × time was seen between the exercise training and diet groups for the HDL-C levels (p<0.05).

Conclusions: Based on our results, an improvement in the HDL-C level was observed in the exercise training group, but not in the diet group, despite the fact that the reductions in the LDL-C, triglycerides and body weight were not significantly different between the two groups. Therefore, these results suggest that lifestyle modification, especially exercise training, is considered to be important to reduce the risk of cardiovascular disease through by increasing the HDL-C.

Keywords: High-density lipoprotein cholesterol; Aerobic exercise training; Diet; Dyslipidemia

Introduction

A low high-density lipoprotein cholesterol (HDL-C) level is an important independent risk factor for coronary artery disease (CAD) [1]. The Japan Lipid Intervention Trial (J-LIT) observed that the risk of coronary events increased by 18% in males and 21% in females with each 10 mg/dl elevation in the low-density lipoprotein cholesterol (LDL-C), and decreased by 39% in males and 33% in females with each 10 mg/dl elevation of the HDL-C [2].

Lifestyle modifications, such as increases in the daily physical activity and changes in diet are an initial step for the prevention of CAD [3]. It has been well known that physical inactivity and decreased aerobic capacity are associated with significantly lower levels of HDL-C [4-6]. Conversely, habitual exercise training may be able to increase the HDL-C level [7]. On the other hand, it is well known that a low-fat diet has been considered to be an effective method for weight loss, and improvements in the blood lipid profiles by weight loss have been demonstrated [8,9].

Moreover, the weight loss is also effective for improving the HDL-C level. Dattilo et al. observed that a 1.0 kg reduction in body weight was associated with a significant increase in the HDL-C by 0.009 mmol/l (about 0.35 mg/dl) [10]. In prior reports, the National Cholesterol Education Program (NCEP) adult treatment panel published data the emphasizing the impact of weight control by implementing lifestyle modifications combined with exercise training and diet on the level of blood lipids [11]. However, at present, it is still unknown whether exercise training or diet is more important to improve the HDL-C,

despite the observation that weight loss influences the increase in the HDL-C level. Furthermore, the differences in the effects and the mechanisms underlying the improvements in the HDL-C by both exercise training and diet have yet to been elucidated.

This study was designed to clarify the difference in the effects of exercise training and diet on improving the HDL-C level in patients with dyslipidemia. We hypothesized that the effects of exercise training and diet on the improvement in the HDL-C level may be different, because the increase in HDL-C occurs via different mechanisms for the two treatments. If the differences between the effects of exercise training and diet on HDL-C can be clarified, it may contribute to demonstrating which lifestyle modifications can good help to prevent dyslipidemia and CAD. Therefore, we focused on patients with dyslipidemia to clarify the differences in the effects of exercise training and diet on the improvement in the HDL-C level, because dyslipidemia is a major risk factor for future CAD.

Subjects and Methods

Subjects

The subjects were recruited by advertisements in newspaper, on the website and on public transportation. Among the 319 subjects who participated in our Metabolic Syndrome Intervention Program (Fukuoka University Randomized Controlled Trial; FURCT), 98 patients with dyslipidemia (41 males and 57 females, age; 55 ± 9 years) were enrolled in this study. The protocol for this program was described in a previous study [12]. Dyslipidemia was defined according to the criteria of the Japan Atherosclerosis Society (LDL-C ≥ 140 mg/dl and/or HDL-C < 40mg/dl and/or triglycerides ≥ 150 mg/dl) [13]. Patients taking anti-hyperlipidemic agents, such as statins or fibrates, were included in this study (statins use, five patients; fibrates use, two patients). Patients with a history of CAD, cerebrovascular diseases or dialysis treatment were excluded.

The patients were randomly allocated to an exercise training (n=50) or diet (n=48) groups using a random number table. Of the 12 patients who did not complete this program (exercise training group: n=8, diet group: n=4), three left for employment-related reasons, three were lost to follow-up, four were in poor physical condition during the 12-week intervention and two left for family reasons. Thus, 86 patients [34 males and 52 females; age, 55 ± 10 years (33 to 71 years); LDL-C, 150 ± 33 mg/dl (74 to 206 mg/dl); HDL-C, 54 ± 12 mg/dl (35 to 87 mg/dl) and triglycerides, 164.9 ± 65.2 mg/dl (68 to 318 mg/dl)] completed the 12-week intervention, with 42 in the exercise training group (17 males and 25 females; age, 55 ± 11 years; LDL-C, 149 ± 29 mg/dl; HDL-C, 55 ± 12 mg/dl and triglycerides, 154 ± 59 mg/dl) and 44 in the diet group (17 males and 27 females; age, 55 ± 9 years; LDL-C, 151 ± 36 mg/dl; HDL-C, 52 ± 11 mg/dl and triglycerides, 176 ± 70 mg/dl).

The flow of participants through the study is shown in Figure 1. All patients gave their informed consent after agreeing with the purpose, methods and significance of the study. This study was approved by the Ethics Committee of Fukuoka University (No. 09-05-01).
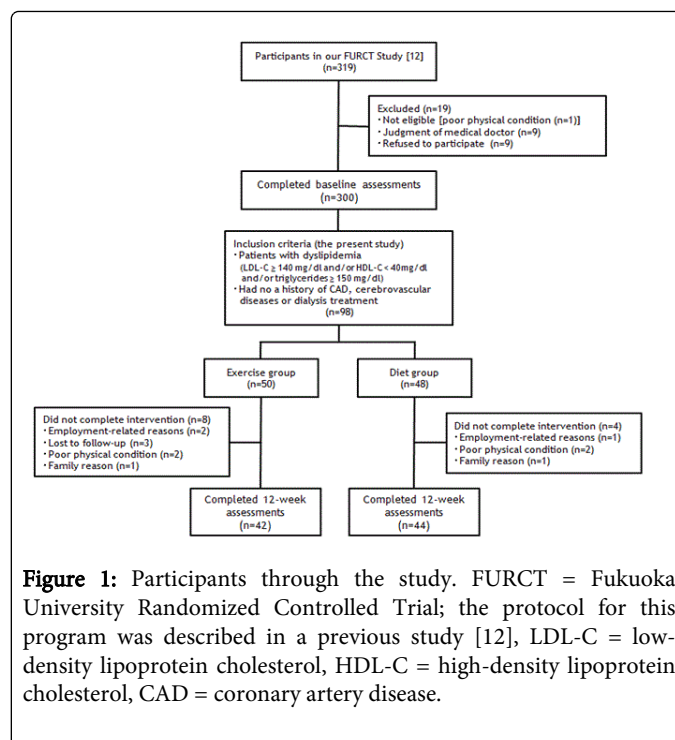

Figure 1: Participants through the study. FURCT = Fukuoka University Randomized Controlled Trial; the protocol for this program was described in a previous study [12], LDL-C = low-density lipoprotein cholesterol, HDL-C = high-density lipoprotein cholesterol, CAD = coronary artery disease.

Blood sampling

Blood samples were collected early in the morning by venipuncture from an antecubital vein after at least 12 hours of fasting. A blood biochemistry analysis was conducted by Special Reference Laboratories (SRL Inc., Tokyo, Japan). The fasting blood samples were used to measure the following parameters: the HDL-C and LDL-C levels by the direct method, the triglyceride levels by the enzyme method, the high-sensitivity C-reactive protein (hs-CRP) level by a nephelometry method, the plasma glucose by an ultraviolet/ hexokinase method and the hemoglobin A1c (HbA1c; National Glycohemoglobin Standardization Program value) by high performance liquid chromatography. The insulin resistance was assessed using Matthews's homeostasis model assessment (HOMA-IR) [14] based on the following formula: fasting glucose (mg/dl) × fasting insulin (μIU/ml)/405.

Anthropometry measurement and body composition

The anthropometric measurement and body composition studies were conducted after 12 hours of fasting. The height and body weight were measured, and the body mass index (BMI) was calculated as the ratio of the body weight (kg) to the height squared (m²). The waist circumference was measured at the level of the umbilicus. The body composition was measured using the underwater weighing method, and the body density was estimated after correction for residual air by the O2 re-breathing method during underwater weighing [15]. The percentage of body fat was calculated using the formula proposed by Brozek et al. [16]. The body fat mass and lean body mass (LBM) were respectively calculated using the following formula: "body weight × percentage of body fat/100" and "body weight – fat mass".

Computed tomography (CT) measurements

The visceral fat area (VFA), subcutaneous fat area (SFA) and middle-thigh muscle area were measured using computed tomography (CT; Toshiba Multi-CT Aquilion TSX-101A Scanner, Toshiba Medical Systems, Tokyo, Japan). For the CT measurements, the examination was conducted after a three-hour fast with only water intake. Images were captured with a maximum voltage of 135 kVp, at 400 mm or 500 mm, to adapt to the anthropometry of each patient. The VFA and SFA were measured at the level of the umbilicus, which were calculated using an image analysis software program (M900PRIMAL, Ziosoft Inc., Tokyo, Japan). The thigh muscle area was measured at the level of the middle thigh circumference, which was divided into low and normal-density muscle. The low- and normal-density muscle areas, markers of lipid-rich skeletal muscle and the contractile component of skeletal muscle were quantified. The data from six images, each 2 mm thick, from the intermediate position between the ends of the anterior iliac crest and patella, were superimposed to create volume data of 10 mm. The low- and normal-density muscle areas were determined based on the calculation of the Hounsfield units (HU) values, using a CT image analysis software program on a Macintosh computer (OsiriX ver 3.3; OsiriX Foundation, Geneva, Switzerland). The low- and normal-density muscle areas were quantified within a 0-29 HU and 30-100 HU attenuation value [17].

Exercise stress test

A ramp submaximal exercise stress test was performed on each subject using an electric bicycle ergometer (REhcor, Lode, Groningen, and The Netherlands) to determine the aerobic capacity and optimal exercise intensity. The work rate was initially set to 60 rpm and 10 watts for the first four minutes as a warm-up, after which it was increased by 15, 10 or 8 watts per minute during the exercise stress test. The increase in the work rate was determined based on the subjects' sex and age (<65-years-old males, 15 watts; ≥65-years-old males, 10 watts; <65-years-old females, 10 watts and ≥65-years-old females 8 watts) or based on the rate decided to be optimal for medical supervision. An exercise stress test was continued until subjective exhaustion was achieved and the oxygen uptake (VO_2) at the peak effort (peak VO_2) was calculated. A respiratory gas analysis was conducted using the mixing chamber method to evaluate the volume of expired air, and the O_2 and CO_2 fractions were analyzed by mass spectrometry (ARCO-2000, ARCO System, Chiba, Japan). Earlobe blood samples were obtained every 30 seconds to measure the lactate acid levels. These 20 μl blood samples were collected in blood collection tubes and evaluated using a lactate analyzer (Biosen 5040, EKF Diagnostik, Barleban, Germany). The workload at the first breaking point of the blood lactate level was used to determine the lactate threshold (LT), and five skilled staff members visually checked the results using log-log (work rate–lactate acid levels) graph paper, and the approximate mean value from three members out of the five members was taken. In this study, the peak VO_2 and metabolic equivalents (METs) at the LT intensity were used as the index for the aerobic capacity.

Exercise training

In the exercise training group, the patients were instructed to perform the bench stepping exercise, bicycle ergometry and walking or running for 60 minutes per session, three times per week under the supervision of exercise trainers, and for a further 120 minutes per week on their own at home to perform a total of at least 300 minutes of moderate exercise per week. The exercise intensity was set at the LT [18]. The difficulty of the training, including exercise at home, was controlled by heart rate at the LT intensity, which was determined by an exercise stress test. Polar FT1 monitors (Polar Electro, Kempele, Finland) were used to measure the heart rate.

Dietary record and diet intervention

Each patient's individual dietary intake was evaluated for three days using a self-record (two weekdays and either Saturday or Sunday) at baseline and during the post-intervention period. All of the meals were photographed to increase the accuracy of the measurement. In the diet group, the target calorie intake was 25 kcal/kg of ideal body weight [height (m)^2 × 22] according to the guideline of the Japan Society for the Study of Obesity [19]. These data analyses and the nutritional guidance were carried out by skilled dieticians.

Statistical Analysis

The data were expressed as the means ± SD. The statistical analyses were performed using the SPSS version 18.0 software package (SPSS Inc., Chicago, IL, USA). The differences in the improvements in the coronary risk factors between the exercise training and diet groups were determined using a two-way repeated-measures analysis of variance for the intervention and groups × time interactions. Comparisons of the data at the baseline and after the intervention were performed using a one way repeated-measure analysis of variance for continuous variables. The inter-group comparisons were performed using the unpaired t-test for continuous variables and chi-square test for categorical variables. Simple linear regression and a partial correlation analysis were performed to determine the associations between the continuous variables. A probability value <0.05 was considered to be statistically significant.

Results

Table 1 shows comparisons of the patients' characteristics at the baseline in both the exercise training and diet groups. There were no significant differences in the age, sex, drinking habits, smoking habits and prevalence of medications used between two groups.

	Exercise training group (n=42)	Diet group (n=44)	p value
Age (years)	55 ± 11	55 ± 9	0.795
Sex (males/females)	17 (40.5)/25 (59.5)	17 (38.6)/27 (61.4)	0.853
Hypercholesterolemia (n,%)	30 (71.4)	29 (65.9)	0.767
Low-HDL cholesterolemia (n, %)	4 (9.5)	5 (11.3)	0.637
Hypertriglyceridemia (n,%)	22 (52.4)	25 (56.8)	0.289
Drinking habit (n,%)	16 (38.1)	12 (27.3)	0.489
Smoking habit (n,%)	3 (7.1)	5 (11.4)	0.737
Taking medications			
Statins (n,%)	2 (4.8)	3 (6.8)	0.584
Fibrates (n,%)	1 (2.4)	1 (2.3)	0.958

Anti-hypertensive drugs (n,%)	8 (19.0)	10 (22.7)	0.476
Hypoglycemic drugs (n,%)	1 (2.4)	2 (4.5)	0.513

Table 1: Characteristics of patient's at the baseline in the exercise training and diet groups

Table 2 shows comparisons of the coronary risk factors before and after the 12-week intervention in both the exercise training and diet groups. There were no significant differences in any of the risk factors between the groups before the intervention. At the baseline, low-HDL cholesterolemia (HDL-C<40mg/dl) was observed in 4/42 patients (9.5%) in the exercise training group and 5/44 patients (11.3%) in the diet group. After the 12-week intervention, the number of patients with low-HDL cholesterolemia decreased from four to one (75% reduction) in the exercise training group and from five to four patients (20% reduction) in the diet group.

	Exercise training group (n=42)		Diet group (n=44)		Group × time Interaction (p value)
	Baseline	12 weeks	Baseline	12 weeks	
Body weight (kg)	70.5 ± 13.6	68.3 ± 12.2 *	72.0 ± 13.1	68.8 ± 13.4 **	0.104
BMI (kg/m2)	27.4 ± 4.6	26.4 ± 4.4 *	27.7 ± 3.5	26.4 ± 3.5 **	0.147
Waist circumference (cm)	95.9 ± 10.2	92.9 ± 9.9 **	96.5 ± 9.6	93.5 ± 7.7 **	0.631
Body fat mass (kg)	21.7 ± 8.0	19.6 ± 7.7 *	23.0 ± 6.6	20.2 ± 5.5 **	0.147
LBM (kg)	48.9 ± 8.4	48.6 ± 7.7	49.3 ± 10.2	49.1 ± 10.1 *	0.562
VFA (cm2)	173.3 ± 60.5	146.1 ± 551 **	191.7 ± 62.0	163.6 ± 49.4 **	0.729
SFA (cm2)	293.7 ± 114.4	268.6 ± 119.6 **	295.7 ± 102.9	266.3 ± 97.4 **	0.462
Low-density muscle area (cm2)	32.8 ± 11.6	32.9 ± 11.9	35.8 ± 12.3	34.7 ± 13.0	0.967
Normal-density muscle area (cm2)	209.6 ± 50.8	216.4 ± 49.0 **	199.6 ± 53.7	192.7 ± 48.2 *	<0.0001
LDL-C (mg/dl)	149 ± 29	143 ± 33 *	151 ± 36	141 ± 25 *	0.49
HDL-C (mg/dl)	55 ± 12	59 ± 15 *	52 ± 11	51 ± 10	0.002
Triglycerides (mg/dl)	154 ± 59	123 ± 45 *	176 ± 70	134 ± 60 *	0.39
HbA1c (%; NGSP value)	5.8 ± 0.6	5.7 ± 0.4	5.7 ± 0.4	5.6 ± 0.3	0.401
Glucose (mg/dl)	103 ± 17	102 ± 14	101 ± 14	100 ± 10	0.354
Insulin (μIU/ml)	8.6 ± 5.1	7.0 ± 4.1 *	8.9 ± 3.9	7.5 ± 3.4 *	0.97
HOMA-IR	2.3 ± 1.5	1.8 ± 1.1 *	2.3 ± 1.2	1.9 ± 0.9 *	0.891
hs-CRP (ng/ml)	1776.1 ± 2260.4	1292.2 ± 1965.2	1789.7 ± 4766.9	743.3 ± 929.8	0.865
Systolic blood pressure (mmHg)	138 ± 16	131 ± 17 *	142 ± 19	135 ± 18 *	0.552
Diastolic blood pressure (mmHg)	87.0 ± 10.3	82.5 ± 10.4 *	87.0 ± 9.7	83.2 ± 7.6 *	0.616
Peak VO2 (ml/min/kg)	23.4 ± 5.2	27.7 ± 7.0 *	21.7 ± 5.3	23.7 ± 8.4	0.001
METs at LT intensity	3.6 ± 0.7	3.9 ± 1.0	3.4 ± 0.7	3.7 ± 1.4	0.496
Energy intake (kcal/day)	2059 ± 326	1993 ± 391	2033 ± 452	1578 ± 321 **	<0.0001
Protein intake (% of energy)	14.9 ± 1.9	15.4 ± 2.5	14.6 ± 2.0	15.4 ± 1.9 *	0.516
Fat intake (% of energy)	28.7 ± 4.0	28.2 ± 5.8	27.6 ± 4.8	26.4 ± 4.8	0.61
Carbohydrate intake (% of energy)	52.9 ± 5.4	53.2 ± 6.7	54.9 ± 6.1	56.0 ± 5.3	0.519

Table 2: Comparisons of the coronary risk factors at baseline and after the 12-week intervention between the exercise training and diet groups

Data are expressed as the means ± SD. *; p<0.05, **; p<0.01, compared to the values before intervention in each group. BMI: Body Mass Index; LBM: Lean Body Mass; VFA: Visceral Fat Area; SFA: Subcutaneous Fat Area: LDL-C: Low-Density Lipoprotein Cholesterol; HDL-C: High-Density Lipoprotein Cholesterol; Hba1c: Haemoglobin A1c; NGSP: National Glycohemoglobin Standardization Program;

HOMA-IR: Insulin Resistance Index By Homeostasis Model Assessment; Hs-CRP: High Sensitivity C-Reactive Protein; Peak VO2: Peak Oxygen Uptake; Mets At LT Intensity: Metabolic Equivalents At Lactate Threshold Intensity; NS: Not Significant.

After the 12-week of intervention, the LDL-C, triglyceride and insulin levels, as well as the HOMA-IR, body weight, BMI, waist circumference, body fat mass, VFA, SFA, systolic and diastolic blood pressure all decreased in both the exercise training and diet groups (p<0.05, respectively). The HDL-C, normal density muscle area (30-100 HU) and peak VO2 increased only in the exercise training group (p<0.05, respectively). The LBM and normal-density middle thigh muscle area (30-100 HU) decreased only in the diet intervention group (p<0.05, respectively).

A significant interaction effect for group × time was seen in the HDL-C, normal density muscle area (30-100 HU) and peak VO2 (p<0.05, respectively) between the exercise training and diet groups. There was no significant interaction effect for group × time in the other risk factors between the two groups.

Figure 2 shows the association between the changes in the HDL-C level and the amount of exercise duration per week, as determined by a simple regression analysis in the exercise training group. The mean exercise duration per week was 319 ± 76 minutes. The change in the HDL-C level was positively correlated with the amount of exercise duration per week (r=0.398, p=0.009).

Figure 2: The association between the changes in the HDL-C level and the amount of exercise duration per week in the exercise training group.
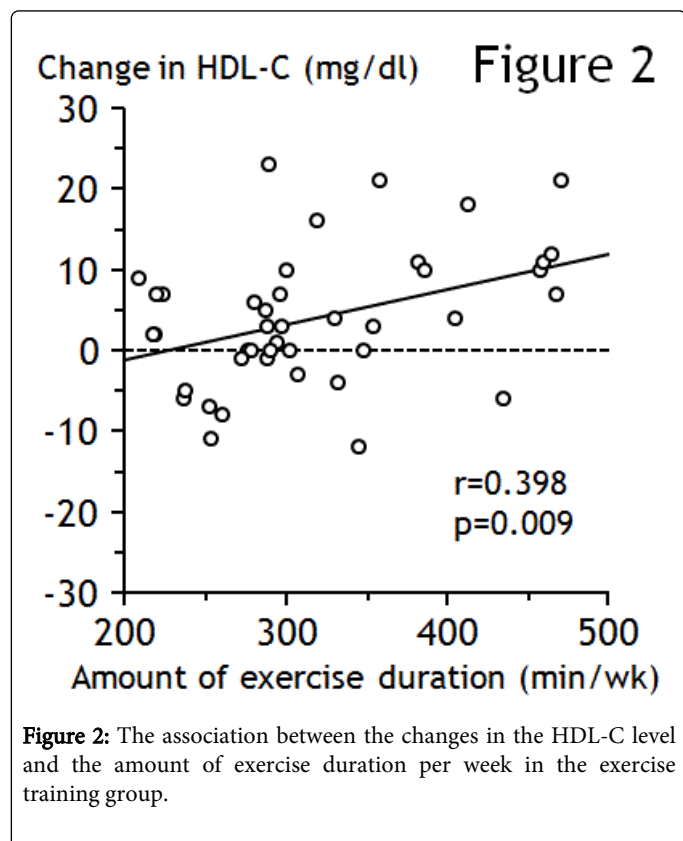
Figure 3 shows the association between the changes in the HDL-C level and the changes in the normal-density muscle area (30-100 HU), as determined by a simple regression analysis in both the exercise training and diet groups. The change in the HDL-C level was positively correlated with the change in normal-density muscle area

(30-100 HU) only in the exercise training group (r=0.382, p=0.016). However, there were no significant relationships between the change in the HDL-C level and the changes in the peak VO2, METs at LT intensity, body weight, BMI, waist circumference, body fat mass, LBM, VFA or SFA in either of the groups.

Figure 3: The association between the changes in the HDL-C level and the changes in the normal-density muscle area (30-100 HU) in the exercise training (left) and diet (right) groups.
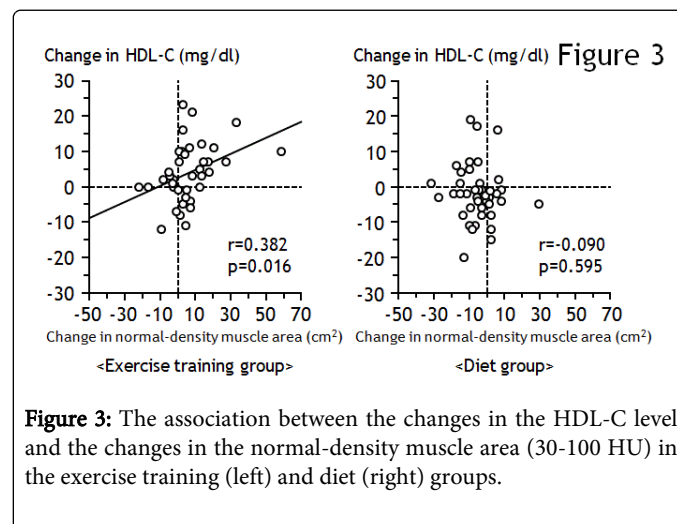
In the partial correlation analysis, which was after adjusted for the amount of exercise duration per week, there was no significant correlation found between the changes in the HDL-C level and the normal density muscle area (30-100 HU, partial correlation coefficient=0.251, p=0.128).

Discussion

The major findings of our study were that the HDL-C level increased only in exercise training group, and a significant interaction effect for group × time was seen between the exercise training and diet groups. Furthermore, the number of patients with low-HDL cholesterolemia decreased from four to one (75% reduction) in the exercise training group and from five to four (20% reduction) in the diet group. Interestingly, in the diet group, there was no significant change in the HDL-C level, despite the observation that there was no significant interaction effect for group × time on the LDL-C, triglyceride levels and body weight between the groups.

It has recently been demonstrated that the combination of exercise training and diet can effectively improve the blood lipid profiles [20-23]. However, the differences in the effects of exercise training and diet on the improvement in the blood lipid profiles have been unknown. There have been many studies [24,25] that showing diet or improvement in the LDL-C and triglyceride levels. However, it has not been elucidated whether the HDL-C level increases following changes in diet. Mensink et al. [26] observed that the HDL-C level was increased by replacing carbohydrates with lipids at the same energy intake. Conversely, an excessive intake of saturated fatty acids has been thought to raise the LDL-C level and to increase the risk of CAD [25]. On the other hand, weight loss is also effective for improving the HDL-C level. Moreover, it is well known that a low-fat diet has been considered to be an effective method to lose weight, and an improvement in the HDL-C level by weight loss has been demonstrated [9,10]. Dattilo et al. observed that a 1.0 kg reduction in body weight was associated with a significant increase in HDL-C by 0.009 mmol/l (about 0.35 mg/dl) [8]. Conversely, Yu-Poth et al. [24]

showed that a low-fat diet using the NCEP Step I and Step II dietary programs decreased the total cholesterol and LDL-C levels, although a reduction in the HDL-C level was also observed. In the present study, the HDL-C level increased only in the exercise training group, while demonstrating no relationship with the change in body weight. Therefore, the improvement in the HDL-C by exercise training may be considered to occur via a mechanism that does not require weight loss.

In our study, the changes in HDL-C level were positively correlated with amount of exercise duration per week in the exercise training group. However, after adjusting for the amount of exercise duration per week, there was no significant correlation between the changes in the HDL-C level and the normal-density muscle area (30-100 HU). At the present, the possible mechanisms underlying the improvement in the HDL-C level by aerobic exercise training have been regarded to include that there is a reduction of the portal free fatty acid level by the decreased amount of visceral fat, that there is an acceleration of lipoprotein lipase (LPL) activity in the muscle and adipose tissue and that the production of HDL-C increases after VLDL decomposition [7]. However, in our study, the change in the normal-density muscle area was not an independent predictive factor for the improvement of the HDL-C level.

According to our data, the change in the HDL-C level was positively correlated with amount of exercise duration per week. We previously evaluated the effects of aerobic exercise training on the HDL-C level in elderly healthy subjects. Our researches suggested that an aerobic exercise training program can result in increases in the HDL-C level, and we have found at the first time, that the increase in HDL-C level was dependent on the total exercise volume [27]. Thus, our present results support the previous study showing that increase in the HDL-C induced by exercise training is dependent on the exercise volume. Recently, Kodama et al. [28] also showed that the increase in the HDL-C dependent on the exercise volume, and the minimum weekly exercise volume necessary to increase the HDL-C level was estimated to be 900 kcal of energy expenditure per week, or 120 minutes of exercise per week. In our exercise training program, the patients were instructed to perform the bench stepping exercise, exercise on a bicycle ergometer and walking or running for 60 minutes per session, and to engage in a further 120 minutes per week on their own at home to perform a total of at least 300 minutes of moderate exercise per week. Therefore, the HDL-C level may have increased only in the exercise training group, because only these subjects engaged in a sufficient volume of exercise.

There are several limitations associated with in this study. First, the limited study population resulted in a small number of subjects, all of whom were free of complications, and in whom there was a predominance of hypercholesterolemia and hypertriglyceridemia. Therefore, it remains unclear whether our findings are consistent with CAD patients and those with other complications. Second, the present study could not measure the LPL activity and apolipoprotein A-I, so we were unable to clarify whether these were involved in the mechanisms underlying the improvement of the HDL-C by aerobic exercise training. Finally, our study period was, only 12-week, which did not provide a sufficient follow-up period to evaluate the influences of diet and exercise training on the longer-term blood lipid profiles. A longer-term additional study including control group should be required to more precisely clarify the effects and mechanisms of lifestyle modifications such as exercise training and diet on the improvement in HDL-C level.

However, in recent several studies, it has been clearly demonstrated that lifestyle modifications can control the development of dyslipidemia and CAD [3, 20-23]. Moreover, aerobic exercise training is an initial step for the prevention of dyslipidemia and CAD. Therefore, we believe our study provides a paradigm, habitual exercise, to prevent CAD in high-risk patients, and might be the good treatment to increase the HDL-C in patients with dyslipidemia.

Conclusions

We found that an improvement in the HDL-C level was observed in the exercise training group, but not in the diet group, despite the fact that the reductions in the LDL-C, triglycerides and body weight were not significantly different between the two groups. These results suggest that lifestyle modification, especially exercise training, are considered to be important to reduce the risk of cardiovascular disease through increasing the HDL-C level, and also suggest that exercise training may be more effective than dieting.

Acknowledgments

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