

Effects of Aerobic Exercise on Metabolic Syndrome Improvement in Response to Weight Reduction

Tomohiro Okura,*† Yoshio Nakata,*† Kazunori Ohkawara,‡ Shigeharu Numao,‡ Yasutomi Katayama,‡ Tomoaki Matsuo,‡ and Kiyoji Tanaka*†

Abstract

OKURA, TOMOHIRO, YOSHIO NAKATA, KAZUNORI OHKAWARA, SHIGEHARU NUMAO, YASUTOMI KATAYAMA, TOMOAKI MATSUO, AND KIYOJI TANAKA. Effects of aerobic exercise on metabolic syndrome improvement in response to weight reduction. *Obesity*. 2007;15:2478–2484.

Objective: The objective was to test effects of aerobic exercise training on metabolic syndrome (MetSyn) improvement in response to weight reduction.

Research Methods and Procedures: A total of 459 overweight and obese women (age, 49 ± 9 years; BMI, 28 ± 3 kg/m²) were recruited for a baseline examination to test the relationship between cardiorespiratory fitness and metabolic syndrome prevalence; among these, 67 subjects with MetSyn were treated with 14-week weight-loss programs, which included low-calorie diet and aerobic exercise. The MetSyn was defined according to the Examination Committee of Criteria for “Metabolic Syndrome” in Japan. Maximal oxygen uptake ($\dot{V}O_{2max}$) during a maximal cycling test was measured as an index of cardiorespiratory fitness at baseline and after the intervention.

Results: In the baseline examination, age- and BMI-adjusted odds ratios for MetSyn prevalence in the low, middle, and upper thirds of $\dot{V}O_{2max}$ were 1.0 (referent), 0.50 (95% confidence interval, 0.26 to 0.95), and 0.39 (95% confidence interval, 0.14 to 0.96), respectively (linear trend, $p =$

0.02). The adjusted odds ratios for MetSyn improvement in the two interventions with diet alone and diet plus exercise were 1.0 and 3.68 (95% confidence interval, 1.02 to 17.6; $p = 0.04$), respectively.

Discussion: These results suggest that adding aerobic exercise training to a dietary weight-reduction program further improves MetSyn (adjusted odds ratio, 3.68) in obese women, compared with diet alone. Further studies on an association between $\dot{V}O_{2max}$ change and MetSyn improvement are needed.

Key words: exercise intervention, diet, aerobic exercise, metabolic syndrome

Introduction

Metabolic syndrome is a cluster of interrelated risk factors (visceral obesity, dyslipidemia, hyperglycemia, and hypertension) (1) that increase susceptibility to cardiovascular disease (2,3) and type 2 diabetes (4,5). The National Cholesterol Education Program’s Adult Treatment Panel III report (6) stated that the increasing prevalence of obesity has been accompanied by a parallel increase in the prevalence of metabolic syndrome, which together constitutes the “obesity epidemic.”

Cross-sectional data indicate that high levels of cardiorespiratory fitness are associated with low prevalence of metabolic syndrome (7–9). Several prospective studies have found that cardiorespiratory fitness is a significant predictor for metabolic syndrome incidence (10,11). Another study found that 20 weeks of aerobic exercise training reduced metabolic syndrome prevalence (12). Clinical intervention studies in obese people have also revealed that regular aerobic exercise training clearly improves risk factors for metabolic syndrome (13,14).

Detecting metabolic syndrome in asymptomatic obese individuals is useful in identifying high-risk individuals for intensive primary preventive therapy (15), and lifestyle

Received for review June 5, 2006.

Accepted in final form February 26, 2007.

The costs of publication of this article were defrayed, in part, by the payment of page charges. This article must, therefore, be hereby marked “advertisement” in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

*Graduate School of Comprehensive Human Sciences, †Center for Tsukuba Advanced Research Alliance, and ‡Doctoral Program in Graduate School of Comprehensive Human Sciences, University of Tsukuba, Tsukuba, Ibaraki, Japan.

Address correspondence to Tomohiro Okura, 1–1–1 Ten-nodai, Tsukuba, Ibaraki 305-8574, Japan.

E-mail: okura@taiiku.tsukuba.ac.jp

Copyright © 2007 NAASO

therapy is recognized as an important approach in the various clinical and educational settings of obesity treatment. However, little is known of the effects of diet and/or aerobic exercise training on metabolic syndrome improvement in obese individuals.

We have investigated these issues in overweight and obese Japanese women. On the basis of the studies cited above, we hypothesized that change in cardiorespiratory fitness, defined as maximal oxygen uptake ($\dot{V}O_{2max}$)¹, would be a predictor for improvement in metabolic syndrome in obese subjects during weight reduction. We first determined whether cardiorespiratory fitness was associated with metabolic syndrome prevalence at baseline. Next, we assigned subjects with metabolic syndrome to two treatment groups, which received diet therapy alone or with aerobic exercise training, and we investigated the effects of cardiorespiratory fitness change and these two treatments on metabolic syndrome improvement during weight reduction.

Research Methods and Procedures

Subjects

Participants were sedentary overweight and obese Japanese women who were recruited through advertisements in local newspapers in Ibaraki prefecture, Japan, and participated in a 14-week weight-reduction program between 2000 and 2004. Before the program, we excluded subjects who smoked, had concomitant renal, hepatic, or cardiac disease, or were being treated with hormone replacement or drugs, which could affect the variables of the study. Consequently, 459 women, 34 to 66 years of age, were chosen as subjects (Table 1) after they met the following criteria: 1) sedentary-ness, defined as exercise-induced energy expenditure of <60 minutes/wk, and 2) overweight or obesity, defined as a BMI of, respectively, >25 kg/m² and >30 kg/m² (16). Of these women, 185 were postmenopausal and 274 were premenopausal. Menopause was defined as the absence of menses for at least 12 months, as reported by questionnaire. This study conformed to the principles outlined in the Helsinki Declaration and was approved by the Review Board of the University of Tsukuba. The aim and design of the study were explained to each subject before she gave her written, informed consent.

Research Procedures

First, we cross-sectionally examined the relationship between cardiorespiratory fitness and metabolic syndrome prevalence in all subjects. Next, 67 subjects were diagnosed as having the metabolic syndrome according to the criteria for the Japanese population, which are described below

¹ Nonstandard abbreviations: $\dot{V}O_{2max}$, maximal oxygen uptake; CT, computed tomography; CI, confidence interval.

Table 1. Baseline characteristics of subjects ($n = 459$)

Characteristic	Value
Age (yrs)	49 ± 9
BMI (kg/m ²)	27.5 ± 3.4
Waist (cm)	99.4 ± 9.5
Visceral fat area (cm ²)	96 ± 47
Systolic BP (mm Hg)	132 ± 18
Diastolic BP (mm Hg)	83 ± 11
Triglycerides (mM)	1.21 ± 0.86
HDL-C (mM)	1.64 ± 0.39
Glucose (mM)	5.49 ± 1.16
$\dot{V}O_{2max}$ (mL/kg per min)	25.2 ± 4.0
$\dot{V}O_{2max}$ (mL/min)	1714 ± 280
Visceral fat obesity (%)	42
High BP (%)	54
High triglycerides (%)	16
Low HDL-C (%)	2
High glucose (%)	13
No. of subjects with metabolic syndrome (%)	67 (15)

BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; $\dot{V}O_{2max}$, maximal oxygen uptake. Values are mean ± SD unless specified otherwise.

(17). To increase subjects' adherence to the weight loss programs, the subjects' personal lifestyles (occupations, daily schedules, etc.) and preferences were taken into account, and the 67 subjects were assigned to two 14-week weight-reduction programs consisting of a low-calorie diet ($n = 24$; target energy intake, 1200 kcal/d) or the diet-plus-aerobic exercise ($n = 43$). Three subjects in the diet alone group and 5 in the diet plus exercise group were unable to complete the study successfully for personal reasons. As a consequence, 21 subjects in the diet alone group and 38 subjects in the diet plus exercise group completed the study requirements. Assays and measurements were carried out before and after the 14-week intervention period. We prospectively examined the relationship between cardiorespiratory fitness change and metabolic syndrome improvement in response to weight reduction.

Anthropometric Variables

Body mass was measured to the nearest 0.1 kg using a digital scale, height was measured to the nearest 0.1 cm using a wall-mounted stadiometer, and BMI was calculated as mass (kg) divided by height squared (m²). Waist girth was measured to the nearest 0.1 cm at the level of the umbilicus with subjects in the standing position.

Visceral Fat Area by CT Scans

Visceral fat area (cm^2) was measured at the level of the umbilicus (L4–L5) using computed tomography (CT) scans (SCT-6800TX, Shimadzu, Tokyo, Japan) performed on subjects in the supine position and was calculated using a computer software program (FatScan, N2system, Osaka, Japan) (18). The intra-class correlation for repeated determinations of visceral fat area in our laboratory was 0.99.

Definition of Metabolic Syndrome

For the Japanese population, the Examination Committee of Criteria for “Metabolic Syndrome” in Japan (17) defined metabolic syndrome as the presence of visceral fat obesity (visceral fat area $\geq 100 \text{ cm}^2$) and two or more of the following criteria: 1) triglycerides $\geq 1.70 \text{ mM}$ (150 mg/dL) and/or high-density lipoprotein cholesterol $< 1.04 \text{ mM}$ (40 mg/dL), 2) systolic blood pressure $\geq 140 \text{ mm Hg}$ and/or diastolic blood pressure $\geq 90 \text{ mm Hg}$, and 3) fasting plasma glucose $\geq 6.1 \text{ mM}$ (110 mg/dL). Systolic and diastolic blood pressures were taken from the left arm using a sphygmomanometer after the subjects rested at least 20 minutes in a sitting position. Cuff sizes were selected based on upper arm girth and length. A blood sample of $\sim 10 \text{ mL}$ was drawn from each subject after an overnight fast. Triglycerides were determined enzymatically, and fasting plasma glucose was assayed by a glucose oxidase method. Serum high-density lipoprotein-cholesterol was measured by the heparin-manganese precipitation method.

Maximal Oxygen Uptake

Maximal oxygen uptake ($\dot{V}O_{2\text{max}}$, mL/kg per min and mL/min) was determined during a graded exercise test using a cycle ergometer (818E, Monark, Stockholm, Sweden). After a 2-minute warm-up, the subject started with a workload of 15 W, which was increased by 15 W each minute until volitional exhaustion occurred. Pulmonary ventilation and gas exchange were measured breath-by-breath with an online data acquisition system (Oxycon α System, Mijnhardt, Breda, Netherlands).

Diet and Exercise Regimens

Dietary Protocol. Subjects were instructed to take a well-balanced supplemental food product (MicroDiet, Sunny-Health, Nagano, Japan) every day. It was developed for very low-energy diets (170 kcal per pack) and is comprised of protein, carbohydrates, fat, various amino acids, vitamins, and minerals. Two other meals per day were allowed, consisting, on average, of 240 kcal of protein, 480 kcal of carbohydrate, and 240 kcal of fat. Subjects also kept daily food diaries during the 14-week intervention period and learned about proper daily nutrition through weekly lectures and counseling by skilled dietitians.

Exercise Protocol. In addition to restricting energy intake, the subjects from the diet plus exercise training group

performed a bench-stepping exercise 3 days/wk for 45 minutes per session, supervised in the hospital by two or three physical trainers. The bench-stepping exercise is a combination exercise of low impact aerobic dance and stepping with a step bench (10 to 20 cm high) (19). The exercise started with basic steps for the first 4 weeks and then progressed to combination of basic steps and lunge steps for the next 6 weeks, and finally progressed to more advanced lunge steps for the last 4 weeks. Subjects were instructed to perform the aerobic dance at a level that raised their heart rate to 70% to 85% of the corresponding heart rate at their $\dot{V}O_{2\text{max}}$. The target Borg's scale (ratings of perceived exertion) (20) ranged from 13 (fairly hard) to 17 (very hard).

Statistical Analysis

Values are mean \pm standard deviation. Paired *t* tests were used to assess differences between variables before and after the weight-reduction intervention period. Unpaired *t* tests were used to test difference in variables between the two treatment groups. Qualitative data were analyzed by a χ^2 test. We used logistic regression to estimate odds ratios and 95% confidence intervals (CIs) as an index of the strength of associations between cardiorespiratory fitness and metabolic syndrome prevalence or improvement, and between treatment (diet alone vs. diet plus exercise) and metabolic syndrome improvement. Multivariate analyses were adjusted for age (years), menopause (yes/no), BMI (kg/m^2), and body weight change (kg). General linear model analyses [repeated-measure two-by-two way (baseline vs. after treatment) ANOVA with post hoc tests] were used to test for difference in measurement variables between groups with diet alone and diet plus exercise, and between baseline and after treatment. In each statistical analysis, probability values below 0.05 were regarded as significant. The data were analyzed with the Statistical Analysis System, version 9.01 for Microsoft Windows (SAS Institute, Inc., Cary, NC).

Results

At baseline, we observed an inverse gradient (linear trend, $p < 0.05$) of age- and BMI-adjusted odds ratios for metabolic syndrome prevalence in the low (average $\dot{V}O_{2\text{max}}$, 20.8 mL/kg per min), middle (average $\dot{V}O_{2\text{max}}$, 25.2 mL/kg per min), and upper (average $\dot{V}O_{2\text{max}}$, 29.5 mL/kg per min) thirds of $\dot{V}O_{2\text{max}}$. They were 1.0 (referent), 0.50 (95% CI, 0.26 to 0.95), and 0.39 (95% CI, 0.14 to 0.96), respectively (linear trend, $p = 0.02$) (Table 2). The significant trend (linear trend, $p = 0.03$) remained after adjustment for age, BMI, and menopausal status. The adjusted risks of metabolic syndrome were 48% (–6% to 75%) and 63% (–4% to 87%) lower in the middle and upper thirds of fitness, respectively, compared with the lower third. On average, each 1 mL/kg per min increment in $\dot{V}O_{2\text{max}}$ was associated with 7% lower risk of metabolic syndrome.

Table 2. Odds ratios and 95% CIs for metabolic syndrome according to $\dot{V}O_{2max}$ (mL/kg per min)

Covariates	$\dot{V}O_{2max}$ tertile			Linear trend (<i>p</i>)
	Low	Middle	High	
All (<i>n</i> = 459)				
No adjustment	1.0 (referent)	0.38 (0.21 to 0.68)	0.13 (0.05 to 0.28)	<0.001
Age, baseline BMI	1.0 (referent)	0.50 (0.26 to 0.95)	0.39 (0.14 to 0.96)	0.02
Age, menopause, baseline BMI	1.0 (referent)	0.52 (0.25 to 1.06)	0.37 (0.13 to 1.04)	0.03
Postmenopausal (<i>n</i> = 143)				
No adjustment	1.0 (referent)	0.39 (0.15 to 0.96)	0.24 (0.08 to 0.66)	<0.01
Age, baseline BMI	1.0 (referent)	0.45 (0.16 to 1.21)	0.45 (0.13 to 1.47)	0.14
Premenopausal (<i>n</i> = 212)				
No adjustment	1.0 (referent)	0.35 (0.09 to 1.12)	0.18 (0.03 to 0.70)	0.02
Age, baseline BMI	1.0 (referent)	0.53 (0.13 to 1.83)	0.34 (0.06 to 2.14)	0.26

CI, confidence interval; $\dot{V}O_{2max}$, maximal oxygen uptake.

Sixty-seven women (15% of all subjects) were diagnosed as having metabolic syndrome. The subjects were assigned to two groups, treated with a low-calorie diet (*n* = 24) or the diet-plus-aerobic exercise training (*n* = 43) (Table 3). Three subjects in the diet alone group and five in the diet plus exercise group were unable to complete the weight-reduction program successfully, for personal reasons. Consequently, 21 subjects in the diet alone group and 38 subjects in the diet plus exercise group were included in the final analysis. The average weight reductions in the diet group and diet plus exercise group were 6.0 kg and 8.8 kg, respectively. The prevalence of metabolic syndrome and metabolic syndrome risk factors was significantly decreased and improved in both groups. For the group treated with diet alone, of the 21 subjects with the metabolic syndrome at baseline, 15 (71%) were no longer diagnosed with the metabolic syndrome after the weight-loss treatment. For the group treated with diet plus exercise, of the 38 subjects with the metabolic syndrome at baseline, 36 (95%) were no longer diagnosed as having the metabolic syndrome after the weight-loss treatment.

We next examined whether treatment (diet alone vs. diet plus exercise) affected metabolic syndrome improvement in response to weight reduction (Table 4). The adjusted odds ratios in the groups with diet alone and diet plus exercise for metabolic syndrome improvement were 1.0 (referent) and 3.68 (95% CI, 1.02 to 17.6; linear trend, *p* = 0.04).

Discussion

Several organizations have recommended clinical criteria for the diagnosis of the metabolic syndrome (1,21). There are some slight differences in the criteria for diagnosis of

the metabolic syndrome used by these organizations. According to the definition of the World Health Organization (22), insulin resistance is a required component and two other risk factors are sufficient for a diagnosis of metabolic syndrome. The National Cholesterol Education Program's Adult Treatment Panel III has stated that when three of five listed characteristics are present, a diagnosis of metabolic syndrome can be made (6). The criteria of the International Diabetes Federation include "central obesity" as an essential component and ethnic-specific values for waist girth (23). In the present study, we used Japanese-specific criteria recommended by the Examination Committee of Criteria for Metabolic Syndrome in Japan (17). This is in accordance with the International Diabetes Federation definition, whereas a slight difference was found in the criteria of low high-density lipoprotein cholesterol and high fasting plasma glucose between the two organizations. It is well known that Japanese individuals are likely to develop obesity-related disorders with even mild obesity (24). Inter-relations among anthropometric variables, body composition, fat distribution, and lipid/glucose metabolism, which may be affected by genetic factors, are quite different in the Japanese, U.S., and European populations. Therefore, we decided to use the Japanese-specific definition of metabolic syndrome.

A few prospective studies have revealed that physical activity and cardiorespiratory fitness are predictors of metabolic syndrome incidence (10,11). One study reported that a 20-week supervised aerobic exercise training reduced metabolic syndrome prevalence by 31% (12). The subjects in the above studies, however, were not all obese. Clinical intervention studies have shown that regular aerobic exercise training clearly improved risk factors for metabolic syndrome in obese people (13,14), but no study, to our

Table 3. Descriptive characteristics of subjects with metabolic syndrome at baseline ($n = 67$) and after treatment ($n = 59$)

	Diet alone			Diet plus exercise		
	Baseline ($n = 24$)	After treatment ($n = 21$)	<i>p</i>	Baseline ($n = 43$)	After treatment ($n = 38$)	<i>p</i>
No. of subjects						
With metabolic syndrome	24	6		43	2	
Without metabolic syndrome	0	15	<0.001	0	36	<0.001
Age (yrs)	52 ± 8			55 ± 6		
BMI (kg/m ²)	30.4 ± 4.9	27.1 ± 4.3	<0.001	29.2 ± 2.3	25.8 ± 2.2	<0.001
Visceral fat area (cm ²)	161 ± 52	119 ± 45	<0.001	136 ± 24	92 ± 26	<0.001
Systolic BP (mm Hg)	150 ± 15	133 ± 11	<0.001	147 ± 19	133 ± 16	<0.001
Diastolic BP (mm Hg)	91 ± 10	87 ± 9	<0.05	89 ± 10	81 ± 10	<0.01
Triglycerides (mM)	1.77 ± 0.75	1.23 ± 0.55	<0.001	2.19 ± 0.94	1.09 ± 0.29	<0.001
HDL-C (mM)	1.43 ± 0.34	1.46 ± 0.29	NS	1.43 ± 0.34	1.51 ± 0.29	<0.05
Glucose (mM)	6.82 ± 2.27	5.77 ± 2.27	<0.01	6.99 ± 2.11	5.60 ± 1.11	<0.001
$\dot{V}O_{2max}$ (mL/kg per min)	22.6 ± 3.4	24.0 ± 3.5	<0.05	22.9 ± 3.2	27.0 ± 3.8	<0.001
$\dot{V}O_{2max}$ (mL/min)	1617 ± 320	1584 ± 291	NS	1596 ± 258	1657 ± 284	NS
Visceral fat obesity (%)	100	76	<0.001	100	47	<0.001
High BP (%)	96	76	<0.001	84	74	<0.01
High triglycerides (%)	58	48	<0.05	67	13	<0.001
Low HDL-C (%)	8	5	NS	10	5	NS
High glucose (%)	67	48	<0.01	58	34	<0.01

BP, blood pressure; HDL-C, high-density lipoprotein cholesterol; $\dot{V}O_{2max}$, maximal oxygen uptake; NS, not significant. Values are mean ± standard deviation unless otherwise specified. Qualitative data were analyzed by a χ^2 test.

knowledge, has confirmed the improvement of metabolic syndrome with increased cardiorespiratory fitness. To our knowledge, the present study is, therefore, the first to investigate the association between cardiorespiratory fitness and metabolic syndrome in overweight and obese populations.

Table 2 suggests that, even with overweight and obesity, high $\dot{V}O_{2max}$ is associated with low prevalence of metabolic syndrome. This finding is in accordance with previous

cross-sectional studies on the association of physical activity and cardiorespiratory fitness with the prevalence of metabolic syndrome (7–9). In obese patients, high cardiorespiratory fitness may prevent metabolic syndrome. Hence, clinicians should counsel their sedentary patients with obesity to become more physically active.

It is well known that an excess visceral fat accumulation is strongly associated with a high prevalence of risk factors

Table 4. Odds ratios and 95% confidence intervals for improvement of metabolic syndrome according to treatment

	Treatment		Linear trend (<i>p</i>)
	Diet alone	Diet plus exercise	
No adjustment			
1.0 (referent)		7.20 (1.47–53.1)	0.02
Adjusted for age and body weight change			
1.0 (referent)		3.68 (1.02–17.6)	0.04

for coronary heart disease, such as lipid metabolic disorders, hypertension, and type 2 diabetes. Moreover, several studies have shown that the visceral fat area above which metabolic disturbances increase is 100 or 110 cm² (25,26). Japan Society for the Study of Obesity has adopted visceral fat area of 100 cm² as the cut-off point for diagnosing high-risk obesity (16). Our data showed that average visceral fat area became <100 cm² in the group treated with diet plus exercise after weight loss, but that still remained >100 cm² in the group with diet alone. Table 3 shows that, although the prevalence of metabolic syndrome and metabolic syndrome risk factors was significantly decreased in both groups, the decreases tended to be larger in the group with diet plus exercise compared with the diet alone group. These data were accordance with previous studies on the association of visceral fat accumulation with the prevalence of risk factors for coronary heart disease.

In the present study, relative values of $\dot{V}O_{2max}$ (unit, mL/kg per min) increased significantly between baseline and after weight reduction, whereas a significant increase was not found in absolute values of $\dot{V}O_{2max}$ (unit, mL/min), even in the group treated with diet plus exercise. A study design that includes exercise of higher intensity and greater frequency and a longer intervention period, which could increase cardiorespiratory fitness, might be needed.

Aerobic exercise training may, however, be essential in treating obese patients with metabolic syndrome, even if the exercise does not increase their $\dot{V}O_{2max}$. The adjusted odds ratio for metabolic syndrome improvement in our study was 3.68 in the group with diet plus exercise training compared with diet alone, when adjusted for age and body weight change. That is to say, adding aerobic exercise training to dietary weight reduction may have a synergistic effect on the improvement of risk factors for metabolic syndrome. This is partly supported by our previous studies (27,28). In one (27), we found that the addition of exercise training contributes to the maintenance of fat-free mass and might be more effective for improving physical fitness and risk factors for coronary heart disease during weight reduction in obese women, compared with diet alone. Another study revealed that a 14-week weight-loss program with diet plus aerobic exercise training reduced visceral adipose tissue by a factor of 1.3 (diet plus exercise, 49.3 cm²; diet alone, 37.8 cm² by CT scans) compared with diet alone, after adjustment for age, menopausal status, and body weight reduction (28). These studies suggest that adding aerobic exercise training to a dietary weight-reduction program further reduces visceral adipose tissue and further improves coronary heart disease risk factors compared with diet alone, even if weight reduction is the same with either treatment.

This study has some limitations. Our findings apply primarily to overweight and obese Japanese women. Although the external validity of our data is limited, the homogeneity of our subjects reduces confounding by sociodemographic

factors, thus enhancing its internal validity. Second, subjects were not randomized to the treatments. Because our goal, in particular, was to increase subjects' adherence to the weight loss programs, the subjects' personal lifestyles and preferences were preferentially taken into account. Consequently, the numbers of subjects were imbalanced in two treatment groups. The study design without randomization may be concomitant with a type II error because of some confounding variables. This factor might partly preclude our definitive conclusion. However, at baseline, no differences were found in any variables between the groups treated with diet alone and with diet plus exercise. This suggests that assigning rather than randomizing subjects had little, if any, influence on the measurement variables.

In summary, our cross-sectional data suggest that, for overweight and obese women, a physically active lifestyle and maintenance of high cardiorespiratory fitness can be useful in primary prevention of metabolic syndrome. Our interventional study revealed that, for overweight and obese women with metabolic syndrome, adding aerobic exercise training to dietary weight reduction is a more effective (adjusted odds ratio = 3.68) treatment for improving metabolic syndrome than diet alone. However, weight-loss intervention trials of longer duration, with more frequent, higher-intensity exercise training and larger samples of obese patients with metabolic syndrome are needed to confirm the association between cardiorespiratory fitness change and metabolic syndrome improvement.

Acknowledgments

This work was supported in part by Grants-in-Aid from the Japanese Society of Physical Fitness and Sports Medicine (1998–2000), by Grants-in-Aid from the Uehara Memorial Foundation (2005), by the Tanaka Project (2004–2006) of TARA (Tsukuba Advanced Research Alliance) at University of Tsukuba, and by the twenty-first century COE (Center of Excellence) program, Ministry of Education, Culture, Sports, Science and Technology (2002–2006 Nishihira Project: Promotion of Health and Sport Scientific Research).

References

1. Grundy SM, Brewer HB Jr, Cleeman JI, Smith SC Jr, Lenfant C. Definition of metabolic syndrome: report of the National Heart, Lung, and Blood Institute/American Heart Association Conference on scientific issues related to definition. *Circulation*. 2004;109:433–8.
2. Lakka HM, Laaksonen DE, Lakka TA, et al. The metabolic syndrome and total and cardiovascular disease mortality in middle-aged men. *JAMA*. 2002;288:2709–16.
3. Hu G, Qiao Q, Tuomilehto J, Balkau B, Borch-Johnsen K, Pyorala K. Prevalence of the metabolic syndrome and its relation to all-cause and cardiovascular mortality in nondiabetic European men and women. *Arch Intern Med*. 2004;164:1066–76.

4. Lorenzo C, Okoloise M, Williams K, Stern MP, Haffner SM. The metabolic syndrome as predictor of type 2 diabetes: the San Antonio Heart Study. *Diabetes Care*. 2003;26:3153-9.
5. Laaksonen DE, Lakka HM, Niskanen LK, Kaplan GA, Salonen JT, Lakka TA. Metabolic syndrome and development of diabetes mellitus: application and validation of recently suggested definitions of the metabolic syndrome in a prospective cohort study. *Am J Epidemiol*. 2002;156:1070-7.
6. National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III) final report. *Circulation*. 2002;106:3143-21.
7. Lakka TA, Laaksonen DE, Lakka HM, et al. Sedentary lifestyle, poor cardiorespiratory fitness, and the metabolic syndrome. *Med Sci Sports Exerc*. 2003;35:1279-86.
8. Jurca R, Lamonte MJ, Church TS, et al. Associations of muscle strength and fitness with metabolic syndrome in men. *Med Sci Sports Exerc*. 2004;36:1301-7.
9. Farrell SW, Cheng YJ, Blair SN. Prevalence of the metabolic syndrome across cardiorespiratory fitness levels in women. *Obes Res*. 2004;12:824-30.
10. Carnethon MR, Gidding SS, Nehgme R, Sidney S, Jacobs DR Jr, Liu K. Cardiorespiratory fitness in young adulthood and the development of cardiovascular disease risk factors. *JAMA*. 2003;290:3092-100.
11. LaMonte MJ, Barlow CE, Jurca R, Kampert JB, Church TS, Blair SN. Cardiorespiratory fitness is inversely associated with the incidence of metabolic syndrome: a prospective study of men and women. *Circulation*. 2005;112:505-12.
12. Katzmarzyk PT, Leon AS, Wilmore JH, et al. Targeting the metabolic syndrome with exercise: evidence from the HERITAGE family study. *Med Sci Sports Exerc*. 2003;35:1703-9.
13. Watkins LL, Sherwood A, Feinglos M, et al. Effects of exercise and weight loss on cardiac risk factors associated with syndrome X. *Arch Intern Med*. 2003;163:1889-95.
14. Frank LL, Sorensen BE, Yasui Y, et al. Effects of exercise on metabolic risk variables in overweight postmenopausal women: a randomized clinical trial. *Obes Res*. 2005;13:615-25.
15. Grundy SM, Hansen B, Smith SC Jr, Cleeman JI, Kahn RA. Clinical management of metabolic syndrome: report of the American Heart Association/National Heart, Lung, and Blood Institute/American Diabetes Association Conference on scientific issues related to management. *Circulation*. 2004;109:551-6.
16. Japan Society for the Study of Obesity. Examination Committee of Criteria for 'Obesity Disease' in Japan: new criteria for 'obesity disease' in Japan. *Circ J*. 2002;66:987-92.
17. Examination Committee of Criteria for 'Metabolic Syndrome' in Japan. Criteria for 'metabolic syndrome' in Japan [in Japanese]. *J Jpn Soc Intern Med*. 2005;94:188-203.
18. Yoshizumi T, Nakamura T, Yamane M, et al. Abdominal fat: standardized technique for measurement at CT. *Radiology*. 1999;211:283-6.
19. Olson MS, Williford HN, Blessing DL, Greathouse R. The cardiovascular and metabolic effects of bench stepping exercise in females. *Med Sci Sports Exerc*. 1991;23:1311-7.
20. Borg G. Perceived exertion: a note on 'history' and methods. *Med Sci Sports*. 1973;5:90-3.
21. Alberti KGM, Zimmet P, Shaw J. The metabolic syndrome: a new worldwide definition. *Lancet*. 2005;366:1059-62.
22. World Health Organization. *Definition, diagnosis and classification of diabetes mellitus and its complications: report of a WHO consultation. Part 1: diagnosis and classification of diabetes mellitus*. http://whqlibdoc.who.int/hq/1999/WHO_NCD_NCS_99.2.pdf (Accessed March 30, 2006).
23. International Diabetes Federation. *The IDF consensus worldwide definition of the metabolic syndrome*. http://www.idf.org/webdata/docs/Metac_syndrome_def.pdf (Accessed March 30, 2006).
24. Nakamura T, Tsubono Y, Kameda-Takemura K, et al. Magnitude of sustained multiple risk factors for ischemic heart disease in Japanese employees: a case-control study. *Jpn Circ J*. 2001;65:11-7.
25. Despres JP, Lamarche B. Effects of diet and physical activity on adiposity and body fat distribution: implications for the prevention of cardiovascular disease. *Nutr Res Rev*. 1993;6:137-59.
26. Williams MJ, Hunter GR, Kekes-Szabo T, et al. Intra-abdominal adipose tissue cut-points related to elevated cardiovascular risk in women. *Int J Obes Relat Metab Disord*. 1996;20:613-7.
27. Okura T, Nakata Y, Tanaka K. Effects of exercise intensity on physical fitness and risk factors for coronary heart disease. *Obes Res*. 2003;11:1131-9.
28. Okura T, Nakata Y, Lee DJ, Ohkawara K, Tanaka K. Effects of aerobic exercise and obesity phenotype on abdominal fat reduction in response to weight loss. *Int J Obes*. 2005;29:1259-66.

原一著

減量介入前の体格, 食事摂取量, 身体活動量が 体重減少量に及ぼす影響

松尾 知明^{*1}, 室武由香子^{*2}, 齋藤 義浩^{*3}, 大藏 倫博^{*4}, 中田 由夫^{*4}, 田中喜代次^{**}

^{*1}筑波大学大学院博士課程人間総合科学研究科 ^{*2}袖ヶ浦市保健福祉部 ^{*3}財団法人体力づくり指導協会

^{**}筑波大学大学院人間総合科学研究科

索引用語: 減量介入, BMI, 食事摂取量, 身体活動量

減量介入によりもたらされる体重変化は一概ではなく, 個体差が生じるのが常である。同一の減量プログラムを提供した場合, 対象者の体重減少量を予測する因子が事前に把握できれば, 肥満改善の現場における利用価値は高い。そこで本研究では, 減量介入による体重変化に影響を及ぼすことが予測される減量介入前の情報(体格, 食事摂取量, 身体活動量)が, 体重変化にどのように関わっているかを検討した。対象者は, 12週間の食事改善指導に運動指導を加えた減量プログラムに参加した女性103名(平均年齢 51.9 ± 7.3 歳)であった。総摂取エネルギー量および三大栄養素摂取量は, 3日間の食事記録による秤量法とその後の管理栄養士による個別聞き取り調査により推定した。また, 総消費エネルギー量および身体活動量は, 加速度計(Lifecorder)と運動記録を併用することで算出した。

減量介入によって対象者の体重($66.6 \pm 8.8\text{kg} \rightarrow 58.4 \pm 8.0\text{kg}$)とBMI($27.3 \pm 2.8\text{kg}/\text{m}^2 \rightarrow 24.0 \pm 2.5\text{kg}/\text{m}^2$)は有意に減少した。体重減少量を目的変数, 減量介入前のBMI, 食事摂取量, 身体活動量を説明変数とした重回帰分析の結果, 標準偏回帰係数はそれぞれ 0.47 ($p < 0.0001$), 0.09 ($p = 0.30$), 0.32 ($p = 0.0003$), 重相関係数は 0.56 であった。減量介入前のBMIと身体活動量が減量介入による体重減少量に関与することが示された。また, 減量を効果的に行うためには, 運動指導による身体活動の増加だけでなく, 日常生活活動量の増加が重要であることが示唆された。

はじめに

Body mass index (BMI)が高いと糖尿病や動脈硬化性疾患などの罹患率や, それらを原因とする死亡率が高くなることが多くの疫学研究により報告されている^{1,2)}。我が国では日本肥満学会により, BMIによる肥満の判定規準が規定されており(普通体重: $18.5 \leq \text{BMI} < 25.0$, 肥満1度: $25.0 \leq \text{BMI} < 30.0$, 肥満2度: $30.0 \leq \text{BMI} < 35.0$, 肥満3度: $35.0 \leq \text{BMI} < 40.0$, 肥満4度: $40.0 \leq \text{BMI}$), BMIが高い者に対しては減量

の必要性を呼びかけている³⁾。また, 2005年4月に新しいメタボリックシンドロームの診断基準が設定され, 内臓脂肪量の多い者に対する減量指導が強く求められるようになった⁴⁾。

肥満をエネルギー代謝の観点からみると, エネルギー摂取量がエネルギー消費量を上回り, 体内に脂肪が過剰に蓄積した状態であるといえる。このエネルギーのインバランスに起因する肥満者は増加の一途であり, 肥満対策は我が国において重要な課題の一つである。一方, 冠危険因子の異常所見や肥

満に関連(起因)する健康障害は, 減量によってその多くが改善することが知られており, この分野のさらなる発展が期待されている。

このような背景から, 我々は, これまでに減量介入を数多く行い, 肥満症患者の冠危険因子の改善⁵⁾, BMI $25\text{kg}/\text{m}^2$ 以上の肥満者の内臓脂肪量の減少⁶⁾などの成果を上げてきた。しかし, 減量介入による参加者の冠危険因子数や内臓脂肪量の変化は一概ではなく, 体重減少量だけをみても個体差が生じるのが常である。減量プログラム

表1 減量介入前後の各測定項目の変化

	減量介入前				減量介入後				変化量			
	全体 n=103	普通体重 n=20	肥満1度 n=66	肥満2度 n=17	全体 n=103	普通体重 n=20	肥満1度 n=66	肥満2度 n=17	全体 n=103	普通体重 n=20	肥満1度 n=66	肥満2度 n=17
体重(kg)	66.6±8.8	57.1±4.2	66.1±5.8	80.0±5.6	58.4±8.0	50.6±4.0	57.8±5.5	70.3±6.2	-8.2±2.3*	-6.6±1.6*	-8.3±2.0*	-9.7±2.8*
BMI(kg/m ²)	27.3±2.8	24.0±0.8	27.1±1.4	32.0±1.4	24.0±2.5	21.3±0.9	23.8±1.4	28.2±1.7	-3.3±0.9*	-2.7±0.6*	-3.4±0.8*	-3.8±1.1*
腹囲(cm)	94.2±8.4	86.7±5.6	93.9±6.7	103.8±8.2	86.1±8.2	80.2±6.9	85.5±6.2	95.4±8.6	-8.1±3.6*	-6.5±3.0*	-8.5±3.5*	-8.4±4.3*
SBP(mmHg)	130±18	117±13	130±18	144±14	116±16	109±15	115±15	128±13	-14±13*	-8±8*	-15±13*	-16±15*
DBP(mmHg)	81±10	74±8	81±8	89±10	72±9	70±9	71±9	78±11	-9±8*	-5±5*	-10±8*	-11±9*
TC(mg/dl)	223±40	211±39	227±41	218±36	198±33	194±36	197±32	204±35	-25±26*	-17±20*	-30±23*	-13±35
TG(mg/dl)	115±88	73±28	123±97	135±88	72±29	68±28	71±29	80±29	-43±76*	-5±28	-51±82*	-55±77*
HDL(mg/dl)	60±13	63±16	59±12	61±16	61±12	63±15	61±11	61±14	1±8	1±6	2±9	0±6
LDL(mg/dl)	140±34	134±31	144±34	131±36	122±30	117±28	122±28	127±36	-18±24*	-17±17	-22±20*	-3±36
FPG(mg/dl)	99±20	97±20	97±14	108±34	90±9	88±8	90±9	94±10	-9±16*	-9±20*	-7±10*	-13±27
VFA(cm ²)	107±43	81±38	105±37	145±49	80±36	59±29	78±31	112±41	-27±24*	-23±23*	-27±25*	-33±19*

BMI: body mass index, SBP: systolic blood pressure, DBP: diastolic blood pressure, TC: total cholesterol, TG: triglycerides, HDL: high-density lipoprotein cholesterol, LDL: low-density lipoprotein cholesterol, FPG: fasting plasma glucose, VFA: visceral fat area

* $p < 0.05$

に参加しても必要と考えられる減量(ベースライン体重の5~10%)^{7,8)}が達成できず、目的であった内臓脂肪量の減少などの効果が得られない者も一部いる。体重減少量に個体差が生じる理由としては主に環境要因、そして遺伝要因や性格要因が考えられるが、そのいずれに関しても、これまでに十分な検討はなされていない。特に環境要因については、減量介入前の調査で容易に把握できるものもあり、体重減少量との関係が解明されれば、減量処方との関係が現場での利用価値は高い。減量処方の現場においては、画一化、マニュアル化された従来の手法に従うのではなく、個々人の身体的特徴などの情報を考慮した、より柔軟な対応が求められている。減量介入前の情報で体重減少量を推定できれば、より効果的な処方が可能となり、肥満が蔓延する現代社会において大きな貢献ができるであろう。そこで本研究では、体重減少量に影響を及ぼす可能性がある減量介入前の参加者の体格、食事摂取量、身体活動量が体重減少量にどのような影響を及ぼすかを検討することとした。

対象と方法

1. 対象者

本研究の対象者は、平成16年度から平成18年度に袖ヶ浦健康づくり支援センター(千葉県袖ヶ浦市)で行われた「減量教室」に参加し、食事改善指導に運動指導を加えた12週間の減量プログラムを受けた平均年齢51.9±7.3歳(30~65歳)の女性103名(平成16年度教室28名、平成17年度教室①24名、教室②23名、平成18年度教室28名)であった。対象者は、普通体重20名、肥満1度66名、肥満2度17名で構成された。普通体重であっても、内臓脂肪面積100cm²以上もしくは腹囲90cm以上の者は本研究の対象者とした。さらに、非肥満者(普通体重、内臓脂肪面積<100cm²、腹囲<90cm)であっても本人の強い希望があれば教室への参加を受け入れ、本研究の対象者とした。非肥満者は11名であり、減量介入前の非肥満者の平均BMIは24.0±1.0kg/m²(22.3~24.8kg/m²)、介入後は21.2±1.1kg/m²(19.4~22.6kg/m²)であった。また、介入前に、医師より高血圧と診断された

者20名(服薬15名)、高脂血症と診断された者13名(服薬10名)、糖尿病と診断された者5名(服薬5名)がいたが、本研究ではこれらの対象者も含めて分析を行った。介入前の全対象者の平均BMIは27.3±2.8kg/m²(22.3~34.6kg/m²)であった(表1)。

対象者には、研究の目的や減量プログラムの詳細な内容、測定内容についての説明会を開催し、測定内容を十分に理解したとみなしたうえで文書にて研究参加への同意を得た。これらの研究遂行手続きについては、筑波大学の倫理委員会の承認を得た。

2. 測定項目

1) 身長、体重、BMI、内臓脂肪面積

身長および体重はTANITA TBF-215を使用し、身長は0.1cm単位、体重は0.1kg単位で計測した。その際、着衣分の目安重量(下着のみの場合は0.5kg、下着以外の着衣がある場合は1.0kg)を計測値から差し引いて測定値とした。身長および体重の測定はすべて熟練した同一の検者が行った。BMIは体重/身長²(kg/m²)として算出した。

内臓脂肪面積(visceral fat area;VFA)は、仰臥位でCTスキャン(TSX-002A, Toshiba, Tokyo)を用いて臍高位(およそL4-L5)を撮影し、内臓脂肪面積計測ソフトFat Scan(ver. 2.0, N2システム)を用いて算出した。

2) 血圧と血液生化学

安静時の収縮期血圧(systolic blood pressure;SBP)と拡張期血圧(diastolic blood pressure;DBP)は20分以上安静座位後、ヤガミ製水銀血圧計を用いて計測した。また、12時間以上の絶食状態で採血し、総コレステロール(total cholesterol;TC), 中性脂肪(triglycerides;TG), 高比重リポ蛋白コレステロール(high-density lipoprotein cholesterol;HDLc), 空腹時血糖(fasting plasma glucose;FPG)を測定した。TCはCholesterol oxidase HDAOS法, TGはGPO-HDAOS法, HDLcはModified Enzymatic法, FPGはGOD Immobilized Membrane-Oxygen Electrode法で測定した。また、TC, HDLc, TGの値からFriedewaldの式 $(TC - HDLc - TG/5)^9$ により低比重リポ蛋白コレステロール(low-density lipoprotein cholesterol;LDLc)を算出した。血圧測定は熟練した看護師が行い、採血は医師により行われた。

3) 総摂取エネルギー量および三大栄養素摂取量の調査

本研究における総摂取エネルギー量および三大栄養素摂取量は、3日間(平日2日+休日1日)の食事記録(自己申告)による秤量法により調査した。調査開始前の集団説明会にて管理栄養士による調査方法の詳細な説明がなされた。対象者にはデジタルクッキングスケールを用いるよう指示し、秤量が困難な菓子類等については製造会社名と分量を記入するよう求めた。3日間の自記式調査後、対象者一人ひとりに対して、管理栄養士による個別聞き取

り調査を行った。また、調味料については、対象者の味覚を基準食(ほうれん草の胡麻和え)により確認し、記入用紙に記録された摂取量と照合した。総摂取エネルギー量および三大栄養素摂取量の計算には、五訂食品成分表を用いて3日間の平均摂取量を算出した。なお、減量介入中の総摂取エネルギー量および三大栄養素摂取量の調査は、減量介入開始10週目に行った。

4) 総消費エネルギー量および身体活動量の推定

本研究における総消費エネルギー量および身体活動量は、メモリ機能を持つ1軸加速度計(Lifecorder, 株式会社スズケン)の情報と運動記録の情報を併用することで算出した。Lifecorderにより性・年齢・身長・体重から基礎代謝量を算出し、それに縦方向への加速度計測から算出される運動量を加算することで、総摂取エネルギー量および身体活動量を求めた¹⁰⁾。このようにして推定された身体活動量は「運動量」と「微小運動量」の2種に分けられる。運動量は、加速度信号から運動強度(1~9)を算定し、それに対応する運動係数(Ka)に体重を乗じて算出される。

$$C = K_a \times W$$

C:運動量, K_a :運動係数(運動強度別固定値, 製造会社非公表), W:体重
また、微小運動量は、加速度信号による運動強度0.5に該当する運動量であり、立位保持など弱い強度のエネルギー量の算定を意図したものである。以下の式により算出される。

$$X = K_x \times B$$

X:微小運動量, K_x :微小運動係数(非公表), B:基礎代謝量

基礎代謝量, 総消費エネルギー量の算出式は以下の通りである。

$$B = K_b \times A \times T$$

K_b :体表面積あたり基礎代謝基準値, A:体表面積, T:時間

$A = 0.444W \times 0.663H \times 88.83$ (W:体重, H:身長)

$$E = B + C + X + 1/10E$$

E:総エネルギー消費量, 1/10E:食事摂取に伴うエネルギー消費量

測定終了後、Lifecorderに蓄えられた情報は、パーソナルコンピュータにデータ送信することにより、表計算ソフト(Excel, マイクロソフト社)のファイル上で総消費エネルギー量(E), 運動量(C)および2分ごとの運動強度(0~9)を確認することができる。なお、本研究では運動量(C)に微小運動量(X)を加えた値を身体活動量(CX)とみなした。

対象者にはLifecorderを、減量介入前と介入中(介入開始10週目)の各7日間以上装着するよう求めた。Lifecorder装着期間中、自記式の記録用紙に、1日のLifecorderの装着状況と実践した運動内容の詳細(運動種類, 時間, ratings of perceived exertion (RPE))を記入するよう指示した。Lifecorderに蓄えられた電子情報と記録用紙からLifecorderの装着状況を確認し、睡眠時と入浴時を除く1日の装着時間が12時間を下回ると推定される日のデータは削除した¹¹⁾。対象者の減量介入前の平均装着日数は 7.3 ± 1.0 日, 介入中は 7.2 ± 1.0 日であった。記録用紙に記載された内容と2分ごとの運動強度を照合し、水泳や自転車運動などLifecorderが捉えられない消費エネルギー量についてはMETsを用いて換算した。測定期間終了後、Lifecorderによる値に、記録用紙から推定した身体活動量を加えることで、総消費エネルギー量および身体活動量を算出した。1METは体重1kgあたり1kcalのエネルギー消費とし、単位時間・単位体重あたりの消費エネルギー量に体重を乗じ、該当身体活動の消費エネルギー量を求めた¹²⁾。

5) 運動習慣の有無

本研究では、ウォーキングやスポーツジムでのエクササイズなど、意識的に行う運動を1回30分以上、週2回以上、3ヵ月以上継続していることを運動習慣ありと定義し、減量教室参加前の運動習慣の有無を介入前の測定の際、個別問診により調査した。

3. 減量プログラム

本研究における減量プログラムは食事改善指導と運動指導を組み合わせた内容である。すべてのプログラムは袖ヶ浦健康づくり支援センターで行われた。食事改善指導はすべての対象者に対して週1回提供した。一方、運動は対象者の意志に基づき、(1)1回90分、週2回の群と、(2)運動に関する講話を行い、それ以外の運動実践は個人に任せる群の2群に分けて行った。すべての対象者は介入期間中、袖ヶ浦健康づくり支援センター内の運動施設(ジム、プールなど)を自由に使用することができ、体力づくり支援士や健康運動指導士ら専門員による個別運動指導を随時受けられる体制を整えた。減量介入中の2群間の身体活動量に差はなかった(運動指導群 19.2 ± 4.7 kcal/(kg/m²)/d vs. 自主運動群 17.7 ± 3.8 kcal/(kg/m²)/d)。

食事改善指導の内容は、原則としてすべての対象者に共通したものである。食事改善のための講話(11回)と調理実習(1回)を週に1回(90分間)、合計12回提供した。講話や調理実習は1教室25名ほどを対象とした集団指導であり、管理栄養士1名、補助スタッフ4名(栄養士、保健師)により行われた。本研究で指導した食事内容は1食あたり400kcalを目安に、4群点数法¹³⁾を用いて、栄養バランスのよい食事を摂取するように指導した。具体的には、毎食、第1群(乳・乳製品/卵)から1点(80kcal)、第2群(魚介・肉類/豆・豆製

品)から1点、第3群(野菜/芋類/果物/きのこ/海藻)から1点、第4群(穀類/砂糖/油脂/その他の嗜好品)から2点(160kcal)の範囲内で食品を選んで摂取するように指導した。教室期間中、対象者にはダイエット日誌を毎日記載するよう求めた。ダイエット日誌は、毎日の体重、体調、4群点数法に基づいた食事内容の詳細を記載するように構成されており、週に1回の教室開催時、講話が行われる90分間で、補助スタッフにより全対象者の体調、摂取エネルギー量(4群点数法に基づく計算により簡易算出)、栄養摂取バランスのチェックを行った。毎回の教室後、ダイエット日誌を返却する際、4群点数法の理解が遅れている者、栄養摂取バランスのよくない者に対し、補助スタッフが1名5~10分の個別指導を行った。個別指導の対象者は1回の教室あたり2~3名であった。

週2回の専門員による運動プログラムは、デンマーク体操、エアロビックダンスなど有酸素性運動を中心に構成した。運動強度は対象者の健康状態や体力に合わせて徐々に上げていくこととし、RPEは11~15(平均13)となるように設定した。運動に関する講話は、運動時の注意点、運動方法などを中心にプログラム初期に行った。なお、運動指導以外の場面での身体活動量の制限は、全対象者に対して指示しなかった。

4. 統計解析

各項目の測定結果は平均値±標準偏差で表した。減量介入前、介入後における各測定項目の平均値の比較には、対応のあるt検定を適用した。3群間の平均値の比較には分散分析を適用した。有意差が認められた場合、post hoc testとしてTukey-Kramer's法を用いた。また、3群間の比率の比較にはカイ2乗検定を適用した。体重減少量

および体重減少率と各変数間の相関関係を検討するために、Pearsonの積率相関係数を算出し、2変数間に交絡因子が存在する場合には偏相関係数を算出した。また、体重減少量を目的変数、各測定項目の減量介入前の値を説明変数とした重回帰分析を施した。重回帰分析は強制投入法を用い、標準偏回帰係数(β)と寄与率(R^2)を検討した。これらの統計解析には、SAS 9.01 Windows版を用いた。なお、すべての検定において、統計学的有意水準を5%に設定した。

結果

1. 減量に伴う各項目の変化

減量に伴う各測定項目の変化を表1に示した。対象者の体重、BMI、腹囲、SBP、DBP、TC、TG、LDLC、FPG、VFAは有意に減少した。

対象者の減量介入前の総摂取エネルギー量は $1,863 \pm 339$ kcal/d、炭水化物摂取量は 260 ± 53 g/d(総摂取エネルギー量に対する比率 $55.8 \pm 5.5\%$)、脂質摂取量は 57 ± 16 g/d($27.2 \pm 5.5\%$)、たんぱく質摂取量は 70 ± 12 g/d($15.2 \pm 2.2\%$)であった。また、減量介入中の総摂取エネルギー量は $1,197 \pm 129$ kcal/d、炭水化物摂取量は 169 ± 24 g/d($56.6 \pm 5.5\%$)、脂質摂取量は 32 ± 8 g/d($23.7 \pm 4.6\%$)、たんぱく質摂取量は 60 ± 10 g/d($20.2 \pm 2.8\%$)であった。減量介入中の総摂取エネルギー量、炭水化物摂取量、脂質摂取量、たんぱく質摂取量は介入前に比べて有意に少なかった。また、総摂取エネルギー量に対する脂質摂取量の比率は有意に減少し、たんぱく質摂取量の比率は有意に増加した(表2)。

対象者の減量介入前の総消費エネルギー量は $1,893 \pm 193$ kcal/d、身体活動量は 447 ± 111 kcal/dであった。また、減量介入中の総消費エネルギー量は

表2 総摂取エネルギー量・三大栄養素摂取量と総消費エネルギー量・身体活動量の変化

n=103	減量介入前	減量介入中	変化量
総摂取エネルギー量(kcal/d)	1,863±339	1,197±129	-666±336*
炭水化物摂取量(g/d) (総摂取エネルギー量に対する比率%)	260±53 (55.8±5.5)	169±24 (56.6±5.5)	-90±52* (+1.0±6.8)
脂質摂取量(g/d) (総摂取エネルギー量に対する比率%)	57±16 (27.2±5.5)	32±8 (23.7±4.6)	-25±17* (-3.6±6.4*)
たんぱく質摂取量(g/d) (総摂取エネルギー量に対する比率%)	70±12 (15.2±2.2)	60±10 (20.2±2.8)	-10±14* (+5.0±3.1*)
総消費エネルギー量(kcal/d)	1,893±193	1,836±173	-57±105*
総消費エネルギー量/BMI(kcal/d)	69.6±6.2	73.0±7.0	+3.5±4.2*
身体活動量(kcal/d)	447±111	468±106	+20.9±93.2*
身体活動量/BMI(kcal/d)	16.4±3.9	18.7±4.4	+2.2±3.6*

* p < 0.05

表3 体重減少量と減量介入前, 介入中の各項目との相関関係

n=103	相関係数 vs. 体重減少量	偏相関係数 (BMI調整) vs. 体重減少量	相関係数 vs. 体重減少率
【減量介入前】			
体重	0.46*	0.15	0.01
BMI	0.45*		0.06
総摂取エネルギー量	0.15	0.13	0.07
炭水化物摂取量	0.15	0.12	0.09
脂質摂取量	0.10	0.06	0.002
たんぱく質摂取量	-0.002	0.01	-0.05
総消費エネルギー量	0.53*	0.37*	0.21*
身体活動量	0.45*	0.37*	0.33*
身体活動量/0 wk BMI	0.28*	0.36*	0.32*
【減量介入中】			
総摂取エネルギー量	-0.06	0.05	-0.03
炭水化物摂取量	-0.08	0.004	-0.04
脂質摂取量	-0.01	0.03	-0.02
たんぱく質摂取量	-0.06	0.04	-0.03
総消費エネルギー量	0.48*	0.29*	0.15
身体活動量	0.48*	0.43*	0.40*
身体活動量/10 wk BMI	0.40*	0.48*	0.44*

* p < 0.05

1,836±173kcal/d, 身体活動量は468±106kcal/dであった。総消費エネルギー量は減量介入前と比べて介入中の値が有意に少なかったが, 体格を考慮した(BMIで除した)総消費エネルギー量は減量介入中の値が介入前の値より有意に多かった。身体活動量は絶対値もBMIで除した値も減量介入中の値が介入前の値より有意に多かった(表2)。

2. 体重減少量と各項目の相関関係

体重減少量と各項目の相関分析の結果を表3に示した。体重減少量は, 減

量介入前の体重(r=0.46), BMI(r=0.45), 総消費エネルギー量(r=0.53), 身体活動量(r=0.45), BMIで除した身体活動量(r=0.28), 減量介入中の総消費エネルギー量(r=0.48), 身体活動量(r=0.48), BMIで除した身体活動量(r=0.40)と有意な相関関係にあった。また, 減量介入前のBMIで調整した偏相関分析, さらに, 体重減少率との相関分析を施したところ, いずれにおいても減量介入前, 介入中の身体活動量は体重減少量, 体重減少率と有意な相

関関係にあった。

3. 減量介入前の身体活動量High群,

Middle群, Low群の体重減少量

体重減少量と身体活動量の関係を検討するため, 減量介入前の身体活動レベルをHigh群(25名, 身体活動量上位25%), Middle群(54名, 中位50%), Low群(24名, 下位25%)に分けた。身体活動量(減量介入前のBMIで除した値)の平均値はHigh群21.3±2.3kcal/d, Middle群16.3±1.9kcal/d, Low群11.5±1.2kcal/dであった。図1は, 減量介入前のBMIを共変量としたうえで, 3群間の体重減少量を一元配置の分散分析により比較したものである。体重減少量の平均値はHigh群9.17±0.40kg, Middle群8.11±0.27kg, Low群7.38±0.40kgであり, High群の体重減少量はMiddle群, Low群よりも有意に多かった。

表4は減量介入前と介入中の各項目の値とその変化量を, 身体活動レベル3群間で比較したものである。減量介入前では, 年齢, 体重, BMI, 総摂取エネルギー量, 三大栄養素摂取量, 運動習慣有る者の割合, いずれの項目においても3群間に差は認められなかった。一方, 総消費エネルギー量, 身体活動量は, 3群間に有意な差が認められた。また, 微小運動量(加速度信号による運動強度0.5に該当する運動量であり, 弱い強度の身体活動量)は, High群, Middle群がLow群よりも有意に大きかった。減量介入中では, 炭水化物摂取量において, High群がLow群よりも有意に大きかった。また, 総消費エネルギー量, 身体活動量は3群間に有意な差が認められた。さらに, 各項目の変化量では, 総摂取エネルギー量, 三大栄養素摂取量の減少量では3群間に有意な差はなく, 身体活動量において, Low群がMiddle群, High群よりも増加量が有意に大きかった。

4. 減量介入前と介入中の身体活動量の相関関係

減量介入前と介入中の身体活動量の関係を散布図(図2)で示した。2群間に有意な相関関係($r=0.64$)が認められた。なお、各身体活動量は減量介入前、介入中のそれぞれのBMIで除した値を用いた。

5. 体重減少量を目的変数とした重回帰分析

体重減少量を目的変数、減量介入前の情報を説明変数とした重回帰分析の結果を表5に示した。説明変数に減量介入前のBMIのみを投入したModel 1においては、標準偏回帰係数(β)は $0.45(p<0.0001)$ 、重相関係数(R) 0.47 、調整済み寄与率($\text{Adj } R^2$)は 21% であった。説明変数に減量介入前のBMIと身体活動量を投入したModel 2においては、 β はそれぞれ $0.48(p<0.0001)$ 、 $0.32(p=0.0002)$ 、 R は 0.55 、 $\text{Adj } R^2$ は 29% であった。説明変数に減量介入前のBMI、身体活動量、総摂取エネルギー量、年齢を投入したModel 3においては、 β はそれぞれ $0.47(p<0.0001)$ 、 $0.32(p=0.0003)$ 、 $0.09(p=0.30)$ 、 $-0.01(p=0.95)$ 、また R は 0.56 、 $\text{Adj } R^2$ は 29% であった。

考察

減量プログラムにおける全対象者の体重($66.6 \pm 8.8\text{kg} \rightarrow 58.4 \pm 8.0\text{kg}$)、BMI($27.3 \pm 2.8\text{kg}/\text{m}^2 \rightarrow 24.0 \pm 2.5\text{kg}/\text{m}^2$)、腹囲($94.2 \pm 8.4\text{cm} \rightarrow 86.1 \pm 3.2\text{cm}$)、VFA($107 \pm 43\text{cm}^2 \rightarrow 80 \pm 36\text{cm}^2$)は有意に減少した(表1)。それぞれの減少量の平均値は体重 8.2kg 、BMI $3.3\text{kg}/\text{m}^2$ 、腹囲 8.1cm 、VFA 23cm^2 であった。日本肥満学会の基準³⁾からみると、減量介入前に肥満1度であった対象者の平均BMI($27.1 \pm 1.4\text{kg}/\text{m}^2$)は、介入後には正常域($23.8 \pm 1.4\text{kg}/\text{m}^2$)になり、メタボリックシ

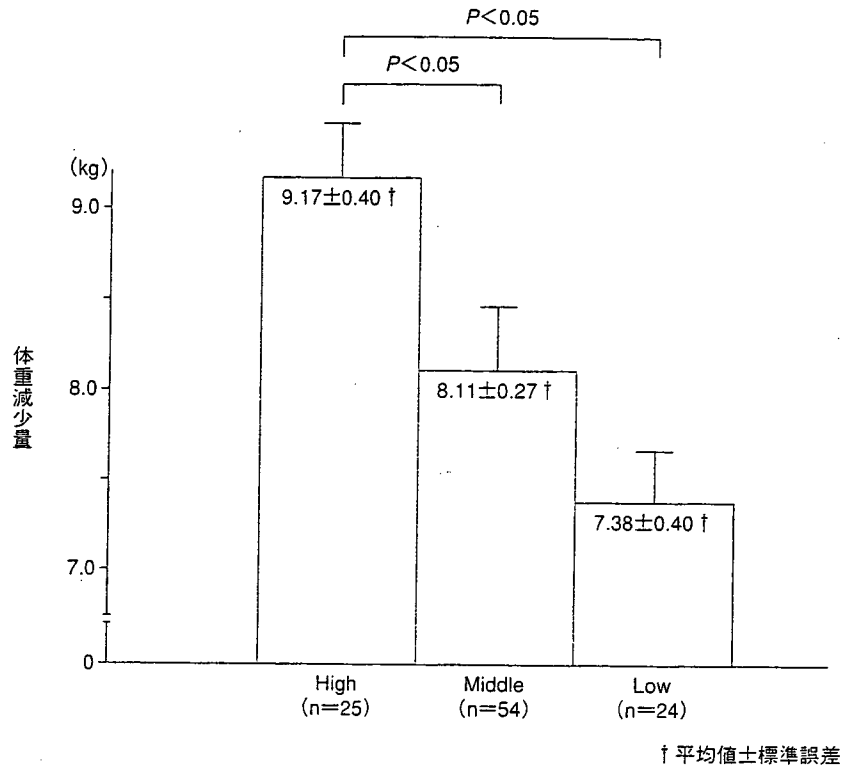


図1 減量介入前の身体活動量High群, Middle群, Low群の体重減少量

ドロームの観点からみても、減量介入前の肥満1度の対象者の腹囲、VFAの平均値は基準値(90cm, 100cm^2)を上回っていたが、介入後は基準値を下回った。肥満2度の対象者では、腹囲やVFAの値は介入後も基準値を上回っているものの、顕著に減少した。その結果、血圧や血液生化学の項目も改善した(表1)。

体重減少量とエネルギー出納との関連を検討するには、摂取エネルギー量および消費エネルギー量の評価が不可欠である。摂取エネルギー量の推定においては、日常生活における対象者の食事内容の詳細を調査しなければならないが、それには対象者自身による申告が必要となる。特にBMIが高い女性は摂食量を過小申告する可能性があり¹⁴⁾、十分な注意が必要である。本研究では、総摂取エネルギー量および三大栄養素の摂取量の推定に3日間の食

事記録による秤量法を採用した。秤量法は食物摂取頻度調査(food frequency questionnaire; FFQ)の妥当基準とされており¹⁵⁾、実態との誤差が懸念される食事調査の中で最も信頼性の高い評価方法の一つである。本研究では秤量法の導入にあたり、総摂取エネルギー量および三大栄養素の摂取量の推定精度を高めるため、3日間の自記式による調査後、対象者一人ひとりに対して管理栄養士による聞き取り調査を行った。調味料については、基準食(ほうれん草の胡麻和え)を用いて対象者の味覚を確認したうえで、記録された調味料の使用量と照合し、聞き取り調査時に確認するなど、可能な範囲で対象者個人の詳細な情報を得ることに努めた。減量プログラム開始10週目(減量プログラム中)における総摂取エネルギー量の平均値は $1,197 \pm 129\text{ kcal}$ であり、減量介入中の対象者の1日あたり

表4 減量介入前の身体活動量 High群, Middle群, Low群の減量介入前, 減量介入中の各項目の値と変化量

	High (n= 25)	Middle (n= 54)	Low (n= 24)	3群比較
【減量介入前】				
年齢 (y.o.)	50.9±7.4	52.0±7.8	53.0±6.2	ns
体重 (kg)	66.9±9.4	67.2±8.2	65.2±9.6	ns
BMI (kg/m ²)	27.0±3.1	27.4±2.7	27.3±2.6	ns
総摂取エネルギー量 (kcal/d)	1,973±354	1,819±345	1,848±295	ns
炭水化物摂取量 (g/d)	281±50	253±54	252±49	ns
脂質摂取量 (g/d)	56±19	56±16	57±13	ns
たんぱく質摂取量 (g/d)	72±14	69±10	69±14	ns
総消費エネルギー量/ 0 wk BMI (kcal/d)	76.1±5.0	69.5±4.5	62.9±2.6	H > M > L
身体活動量/ 0 wk BMI (kcal/d)	21.3±2.3	16.3±1.9	11.5±1.2	H > M > L
微小運動量/ 0 wk BMI (kcal/d)	7.6±1.2	7.6±1.1	6.5±1.4	H > L, M > L
運動習慣有り (n / %)	10/40	18/33	4/17	ns
【減量介入中】				
総摂取エネルギー量 (kcal/d)	1,241±149	1,198±121	1,152±111	ns
炭水化物摂取量 (g/d)	178±27	170±21	160±22	H > L
脂質摂取量 (g/d)	32±8	32±8	31±9	ns
たんぱく質摂取量 (g/d)	62±11	60±10	59±8	ns
総消費エネルギー量/ 10 wk BMI (kcal/d)	79.0±7.3	72.4±5.7	68.2±5.0	H > M > L
身体活動量/ 10 wk BMI (kcal/d)	22.6±4.8	18.1±3.2	15.8±3.6	H > M > L
微小運動量/ 10 wk BMI (kcal/d)	9.0±1.5	9.0±1.5	8.6±1.5	ns
【変化量】				
総摂取エネルギー量 (kcal/d)	-733±317	-621±359	-696±298	ns
炭水化物摂取量 (g/d)	-103±45	-84±57	-92±48	ns
脂質摂取量 (g/d)	-24±19	-25±17	-26±14	ns
たんぱく質摂取量 (g/d)	-10±15	-9±14	-10±12	ns
総消費エネルギー量/BMI (kcal/d)	+2.9±5.0	+2.9±3.6	+5.3±4.1	ns †
身体活動量/BMI (kcal/d)	+1.3±3.8	+1.8±3.1	+4.3±3.6	L > H, L > M †
微小運動量/BMI (kcal/d)	+1.3±1.4	+1.4±1.4	+2.1±1.8	ns †

† ベースライン値で補正

の摂取エネルギー量は1,200 kcalになるよう指導したことを考えると, 本研究における摂取エネルギー量の評価が一定の水準に達していたと考えられる。

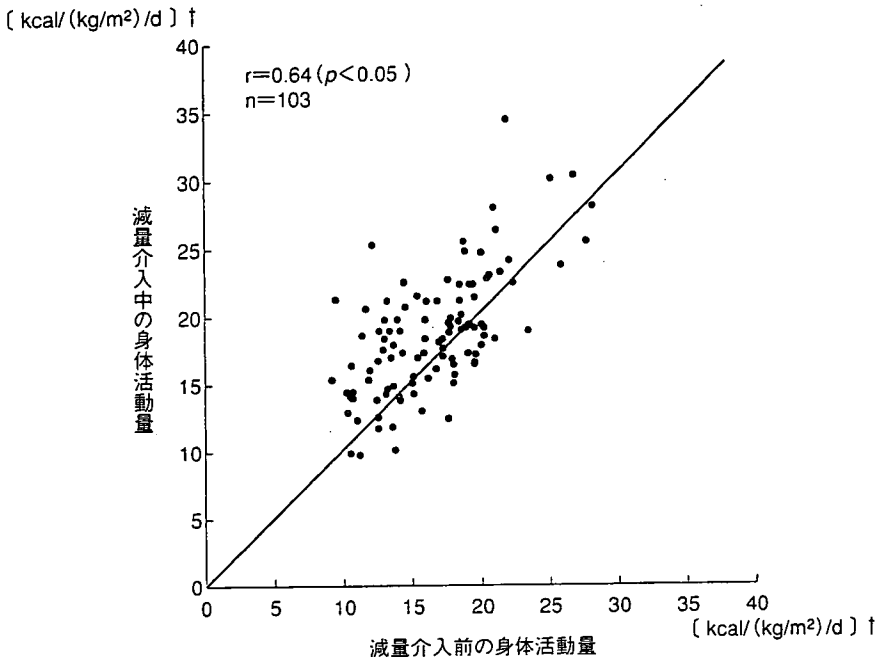
しかしながら, 本研究では体重減少量を予測する因子として, 摂取エネルギー量に関わる因子は採択されなかった。一般に体重減少量に摂食エネルギー量が影響を及ぼさないとは考えにくい。本研究では減量介入中の総摂取エネルギー量を全員一律に1,200kcal/dに規定したが, 減量介入前の総摂取エネルギー量には個体差(1,863±339kcal/d)があるため, 減量介入前の総摂取エネルギー量が多い者ほど総摂取エネルギー量の減少幅が大きく, 体重減少量も大きくなると予想された。しかし, 減

量介入前の総摂取エネルギー量と体重減少量の間に関連はみられなかった。本研究では, 摂取エネルギー量を可能なかぎり正確に評価するよう努めたが, 先行研究¹⁶⁾が示すように, 摂取エネルギー量の評価には誤差が大きく, 体重減少量を予測するための評価方法としては限界があったのかもしれない。

一方, 体重減少量と減量介入前の体重およびBMIとの相関は高く(表3), 体格の大きな者ほど体重減少量は大きいことが認められた。本研究における減量プログラムでは, 減量介入中の摂取エネルギー量は対象者の体型に関わらず, 指導効率を高める立場から, 全員1,200kcal/dになるよう導いた。そのため, 体重, 体格がもともと大きかつ

た者が, 食事制限に対する反応が大きくなり, 体重減少量も大きくなったと考えられる。

総消費エネルギー量および身体活動量は, メモリ機能を有する一軸の加速度計(Lifecorder)の情報と運動記録情報を併用することで推定した。消費エネルギー量の基準法としては, ヒューマンカロリーメータ^{10,17)}や二重標識水法^{18,19)}があるが, 多人数を対象とする場合は, 質問紙法が多くの研究^{20,21)}で用いられている。しかし, 対象者に1日の活動内容の詳細な記録を要求する必要があるので負担が大きく, また活動時間の過大推定や過小推定が起こりやすいという問題がある。そのような中, 加速度計は対象者の負担も少なく, 妥当基準となるヒューマンカロリーメ



† 減量介入前, 減量介入中の身体活動量は各測定時のBMIで除した値

図2 減量介入前と減量介入中の身体活動量の相関関係

表5 減量介入前の情報を説明変数, 体重減少量を目的変数とした回帰分析

	n=103	β	p	R(Adj R ²)
【Model 1】				
BMI		0.45	<0.0001	0.47 (21%)
【Model 2】				
BMI		0.48	<0.0001	0.55 (29%)
身体活動量/BMI		0.32	0.0002	
【Model 3】				
BMI		0.47	<0.0001	0.56 (29%)
身体活動量/BMI		0.32	0.0003	
総摂取エネルギー量		0.09	0.30	
年齢		-0.01	0.95	

β : 標準偏回帰係数, R: 重相関係数, Adj R²: 調整済み寄与率

一タや二重標識水法との相関も高い^{10,29)}ことから, 多人数を対象とする測定には適しているとされる¹⁷⁾.

体重減少量と高い相関関係が認められた減量介入前の身体活動量を下位25%の身体活動レベルLow群, 上位25%のHigh群, それ以外のMiddle群の3群に分けて, それぞれの体重減少量の平均値を, 体重減少量に影響を及ぼすと考えられる減量前のBMIを補正值とした共分散分析により比較したと

ころ, High群は, Middle群およびLow群よりも体重減少量が有意に大きかった(図1)。また, 減量介入前の総摂取エネルギー量, 三大栄養素摂取量およびそれらの変化量にはHigh群, Middle群, Low群間に有意差は認められず, 3群間の体重減少量の差に食事の影響は少ないことが認められた。これらの結果は, 相関分析の結果と同様, 減量介入前の身体活動量が体重減少量に影響を及ぼすことを示すものであ

る。

一方, 減量介入中の身体活動量と体重減少量との間にも有意な相関関係がみられた(表3)。この結果は, 減量介入期間中に身体活動量が大きかった者ほど体重減少量が大きいのことを示すものである。本研究のすべての対象者は日常生活における身体活動の上限, 下限は制限されていない。図2からうかがえるように, 減量介入中に身体活動レベルが高かった者は, もともと(プログラム参加前の日常)の身体活動レベルも高い。低い強度の身体活動として位置づけられる微小運動量が減量介入前でHigh群, Middle群がLow群よりも大きかったこと, また, 減量介入前に運動習慣がある者の割合が3群間に差がなかったことを考えると, 本研究における減量介入前の身体活動量High群は, 運動実践による身体活動ではなく, 日常生活における身体活動が多く, その積み重ねで身体活動量全体が高まった集団であった可能性がある。

一方, 減量介入による身体活動量の変化量をみると, 減量介入前の身体活動レベルの低いLow群がMiddle群, High群よりも増加量が大きいことが示された。減量介入中のLow群の身体活動レベルは, 運動プログラムにより介入前よりも高まったが, 体重減少量は, 日常の身体活動レベルの高かったHigh群に及ばなかった。これらの結果は, 身体活動量の大小は, 運動プログラムの有無よりも日常生活全般における身体活動量で検討されるべきであることを示すものである。減量介入中の身体活動レベルは体重減少量に関与するが, これは介入中に運動量を変化させたことによるものだけではなく, 運動プログラム以外の場面の影響も強かったものと考えられる。

減量介入前の体格, 食事摂取量, 身体活動量が, 体重減少量に与える影響

の程度を検討した結果, 減量介入前のBMIと身体活動量で, 体重減少量の29%を説明することが示された。Donnellyら²⁹⁾は, 体重減少に及ぼす身体活動の効果は食事制限の補佐的なものであるとする一方で, その特徴を理解し, 減量プログラムに生かすことが減量の成功につながると述べている。本研究では, 中年肥満女性の日常の生活活動レベルが, 食事制限を主体とした減量介入による体重減少量の個体差に影響を及ぼす可能性が示された。対象者の多くが食事による摂取エネルギー量を顕著に減少させた本研究では, 日常の身体活動レベルの差が消費エネルギー量の差となり, 体重減少量の差につながったのではないかと。減量介入における運動プログラムは, 週2~3回, 1回60~90分で構成される運動指導に頼るだけでなく, 参加者の日常の身体活動レベルを高める導きも重要である。

まとめ

減量介入前の体格, 食事摂取量, 身体活動量が, 体重減少量に与える影響の程度を検討したところ, 減量介入前のBMI, 身体活動量が体重減少量と関連した。減量介入前の身体活動量をHigh群, Middle群, Low群の3群に分けて分析したところ, High群は他の2群よりも有意に体重減少量が大きいたことが示された。以上のことから, 肥満者に対して減量支援を行う際, 対象者の日常の身体活動レベルを事前に把握することは有意義であることが示唆された。

謝辞

本研究の減量プログラムは袖ヶ浦市保健福祉部健康づくり支援センター課および財団法人体力づくり指導協会の協力を得て行ったものである。また,

本研究は筑波大学先端学際領域研究センター(TARA田中プロジェクト), 研究拠点形成費補助金(21世紀COEプログラム西平プロジェクト)の支援を受けて行ったものである。

文献

- 1) Matsuzawa Y, Tokunaga K, Kotani K, et al.: Simple estimation of ideal body weight from body mass index with the lowest morbidity. *Diabetes Res Clin Pract* 1990, **10**: S159-S164.
- 2) Hayashi R, Iwasaki M, Otani T, et al.: Body mass index and mortality in a middle-aged Japanese cohort. *J Epidemiol* 2005, **15**: 70-77.
- 3) 片岡邦三: 肥満の判定と肥満症の診断基準について. *肥満研究* 2003, **9**: 3-4.
- 4) メタボリックシンドローム診断基準検討委員会: メタボリックシンドロームの定義と診断基準. *日誌* 2005, **94**: 794-809.
- 5) Okura T, Tanaka K, Nakanishi T, et al.: Effects of obesity phenotype on coronary heart disease risk factors in response to weight loss. *Obes Res* 2002, **10**: 757-766.
- 6) Tanaka K, Okura T, Shigematsu R, et al.: Target value of intraabdominal fat area for improving coronary heart disease risk factors. *Obes Res* 2004, **12**: 695-703.
- 7) Goldstein DJ: Beneficial health effects of modest weight loss. *Int J Obes* 1992, **16**: 397-415.
- 8) Klein S: Outcome success in obesity. *Obes Res* 2001, **9**(Suppl 4): 354S-358S.
- 9) Friedewald WT, Levy RI, Fredrickson DS: Estimation of the concentration of low-density lipoprotein cholesterol in plasma, without use of the preparative ultracentrifuge. *Clin Chem* 1972, **18**: 499-502.
- 10) Kumahara H, Schutz Y, Ayabe M, et al.: The use of uniaxial accelerometry for the assessment of physical-activity-related energy expenditure: a validation study against whole-body indirect calorimetry. *Br J Nutr* 2004, **91**: 235-243.
- 11) Masse LC, Fuemmeler BF, Anderson CB, et al.: Accelerometer data reduction: a comparison of four reduction algorithms on select outcome variables. *Med Sci Sports Exerc* 2005, **37**: S544-54.
- 12) Ainsworth BE, Haskell WL, Leon AS, et al.: Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc* 1993, **25**: 71-80.
- 13) 香川芳子: 五訂版食品80キロカロリーガイドブック. 東京: 女子栄養大学出版部, 2002.
- 14) Okubo H, Sasaki S: Underreporting of energy intake among Japanese women aged 18-20 years and its association with reported nutrient and its association with reported nutrient and food group intakes. *Public Health Nutr* 2004, **7**: 911-917.
- 15) Bonifacj C, Gerber M, Scali J, et al.: Comparison of dietary assessment methods in a Southern French population: use of weighed records, estimated-diet records and a food-frequency questionnaire. 1997, *Eur J Clin Nutr* **51**: 217-231.
- 16) Black AE: Physical activity levels from a meta-analysis of doubly labeled water studies for validating energy intake as measured by dietary assessment. *Nutr Rev* 1996, **54**: 170-4.
- 17) Westerterp KR: Physical activity assessment with accelerometers. *Int J Obes* 1999, **23**: S45-S49.
- 18) Bouten CV, Verboeket-van de Venne WP, Westertirp KR, et al.: Daily physical activity assessment: comparison between movement registration and doubly labeled water. *J Appl Physiol* 1996, **81**: 1019-1026.
- 19) Westerterp KR: Assessment of physical activity level in relation to obesity: current evidence and research issues. *Med Sci Sports*

減量介入前の情報と体重減少量

- Exerc 1999, 31 : S522-S525.
- 0) Schuit AJ, Schouten EG, Westerterp KR, et al : Validity of the physical activity scale (PASE) for the elderly according to energy expenditure assessed by the doubly labeled water method. J Clin Epidemiol 1997, 50 : 541-546.
- 1) Philippaerts RM, Westerterp KR, Lefevre J : Doubly labeled water validation of tree physical activity questionnaires. Int J Sports Med 1999, 20 : 284-289.
- 22) Rafamantanantsoa HH, Ebine N, Yoshioka M, et al : Validation of three alternative methods to measure total energy expenditure against the doubly labeled water method for older Japanese men. J Nutr Sci Vitaminol 2002, 48 : 517-523.
- 23) Donnelly JE, Smith BK : Is exercise effective for weight loss with Ad libitum diet? Energy balance, compensation, and gender differences. Exerc Sport Sci Rev 2005, 33 : 169-174.

(受付日 : 2006年11月22日)
(採択日 : 2007年 5月24日)



ELSEVIER



<http://www.ietf.org/oonet/aoaso.htm>

ORIGINAL ARTICLE

Effect of weight reduction on concentration of plasma total homocysteine in obese Japanese men

Tomohiro Okura^{a,b,*}, Yoshio Nakata^{a,b}, Kazunori Ohkawara^c, Shigeharu Numao^d, Yasutomi Katayama^a, Yohei Ono^e, Tomoaki Matsuo^e, Hirohito Sone^{a,f}, Kiyoji Tanaka^{a,b}

^a Graduate School of Comprehensive Human Sciences, University of Tsukuba, 1-1-1 Ten-nodai, Tsukuba, Ibaraki 305-8574, Japan

^b Center for TARA (Tsukuba Advanced Research Alliance), University of Tsukuba, Japan

^c Health Promotion and Exercise Program, The National Institute of Health and Nutrition, Japan

^d Faculty of Sport Sciences, Waseda University, Japan

^e Doctoral Program in Graduate School of Comprehensive Human Sciences, University of Tsukuba, Japan

^f Department of Nutrition, Ochanomizu University, Japan

Received 8 August 2006; received in revised form 29 June 2007; accepted 6 July 2007

KEYWORDS

Weight reduction;
Diet;
Exercise training;
Hyperhomocysteinemia;
Obesity

Summary

Objective: To test whether weight reduction would lower tHcys concentration, and whether adding exercise training to dietary weight reduction would further reduce tHcys concentration over diet alone.

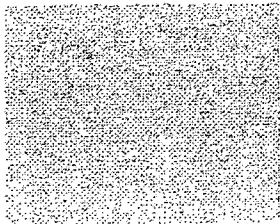
Design: A randomized, controlled, prospective, 14-week weight-loss intervention.

Setting and participants: Forty-eight obese Japanese men aged 27–66 years were assigned to 2 subgroups depending on the type of treatment: diet alone (DA) and diet plus exercise training (DE). Concentration of tHcys was measured in frozen plasma samples by an HPLC method.

Results: Body weight decreased significantly for both groups: DA -7.8 ± 3.2 kg and DE -9.1 ± 3.6 kg. A significant decrease was found in tHcys concentration of the DE group (-2.3 ± 5.0 $\mu\text{mol/L}$) but not of the DA (-0.3 ± 2.7 $\mu\text{mol/L}$). For subjects with hyperhomocysteinemia (≥ 15 $\mu\text{mol/L}$, $n=17$), tHcys concentration decreased from 20.1 ± 7.0 to 13.9 ± 3.0 $\mu\text{mol/L}$ ($p < 0.01$) for the DE group ($n=8$) but did not change for the DA group (16.6 ± 1.9 $\mu\text{mol/L}$ \rightarrow 15.4 ± 2.3 $\mu\text{mol/L}$, $n=9$). Furthermore, to consider a statistical phenomenon "regression to the mean", we excluded two

* Corresponding author at: Graduate School of Comprehensive Human Sciences, University of Tsukuba, 1-1-1 Ten-nodai, Tsukuba, Ibaraki 305-8574, Japan. Tel.: +81 29 853 2733; fax: +81 29 853 6507.

E-mail address: okura@taiiku.tsukuba.ac.jp (T. Okura).



subjects with the highest two tHcys values of the DE group. The DE group ($n=7$) still displayed a significant reduction after the exclusion.

Conclusions: A weight reduction with diet alone did not improve tHcy levels. Adding aerobic exercise training to a dietary weight-reduction program may be effective for a reduction in tHcy.

© 2007 Asian Oceanian Association for the Study of Obesity. Published by Elsevier Ltd. All rights reserved.

Introduction

Plasma/serum total homocysteine (tHcys) is thought to contribute to atherosclerosis and thrombosis [1]. Epidemiological studies have shown that elevated tHcys levels have been linked to increased risk of premature coronary artery disease, stroke, and thromboembolism (venous blood clots), even among people who have normal cholesterol levels [2,3]. Several studies have suggested that lowering homocysteine levels is associated with reduced incidence of heart attack and stroke [4–6].

Obesity is closely associated with some major health risk factors [7], and the prevalence of obesity continues to increase in developed countries [8]. It is well-known that individuals with android-type obesity are at greater risk for coronary heart disease and several metabolic disorders [9]. Some epidemiological studies have reported that body mass index (kg/m^2) [10] and waist-to-hip ratio [11] are positively associated with tHcys concentration. Hence, reduction in both tHcys concentration and fatness level is one of the most important goals to reduce the incidence of myocardial infarction and stroke. To our knowledge, however, very little information is available on the effect of weight reduction for android-type obese people on tHcys concentration. Moreover, to date, no prospective studies have examined the weight-reduction effect on tHcys in Asian populations.

Our first aim in this study was to investigate the effects of 14-weeks weight reduction intervention on tHcys concentration in obese Japanese men. A study by Randeve et al. [12] provided data that a 6-month regular brisk walking significantly lowered tHcys (from 10.1 to 7.4 $\mu\text{mol}/\text{L}$) in 21 young overweight and obese women with polycystic ovary syndrome. Therefore, we also tested an assumption that adding aerobic exercise training to a dietary weight-reduction program may further reduce tHcys concentration over diet alone.

Subjects and methods

Subjects

Ninety men were recruited through advertisements in local newspapers. Through medical history and physical examination, we excluded individuals who were non-obese or non-overweight (body mass index less than $25\text{ kg}/\text{m}^2$) [13], had concomitant renal, hepatic or cardiac disease or diabetes, or were being treated with drugs such as beta-blockers, which could affect the variables of the study. Forty-eight sedentary men, aged 27–66 years, were chosen as subjects with their baseline descriptive characteristics in Table 1.

They were randomly placed in either a diet alone (DA) group ($n=24$) or a diet plus exercise (DE) group ($n=24$). Three subjects in the DA group and two in the DE group were unable to complete the study successfully for personal reasons. Consequently, 21 subjects in the DA group and 22 subjects in the DE group completed the study requirements. Assays and measurements were carried out before and after the 14-week intervention period. This study conformed to the principles outlined in the Helsinki Declaration and was approved by the Review Board of the University of Tsukuba. The aim and design of the study were explained to each subject before they gave their written, informed consent.

Anthropometric variables

Body weight was measured to the nearest 0.1 kg using a digital scale, height was measured to the nearest 0.1 cm using a wall-mounted stadiometer,

Table 1 Descriptive characteristics of subjects ($n=48$)

	Mean \pm S.D.	Range
Age (year)	49.5 \pm 10.3	27–66
Body mass index (kg/m^2)	28.0 \pm 2.6	25.1–41.8
Percentage fat mass (%)	30.8 \pm 4.2	17.1–38.1
Homocysteine ($\mu\text{mol}/\text{L}$)	14.3 \pm 4.5	7.0–34.2

and body mass index was calculated as weight (kg) divided by height squared (m^2).

Body composition by DXA

A dual-energy X-ray absorptiometry (DXA) machine (DPX-L, Lunar, Madison, Wisconsin, USA) was used to evaluate segmental body composition, which consists of fat tissue and lean soft tissue. The reproducibility of body composition measurements was evaluated in 35 subjects. Two DXA procedures were performed on the same day separated by an interval of a few minutes. The within-participant coefficients of variation were 2.5% and 0.5% for fat tissue and lean soft tissue, respectively.

Abdominal adipose tissue area by CT

Visceral fat area and subcutaneous fat area (cm^2) were measured at the level of the umbilicus (L4-L5) using CT (computed tomography) scans (Somatom AR.C, Siemens, Erlangen, Germany) performed on subjects in the supine position. Visceral fat area and subcutaneous fat area were calculated using a computer software program (FatScan, N2system, Osaka, Japan). The intra-class correlation for repeated determinations in our laboratory are 0.99.

Blood pressure and biochemical blood assay

Systolic and diastolic blood pressures (SBP and DBP) were measured using a sphygmomanometer before 11 a.m. after the subjects rested at least 20 min in a sitting position. A blood sample was drawn from each subject after a 12-h fast.

Concentrations of tHcy (the sum of homocysteine, homocysteine, and homocysteine–cysteine mixed disulfides, free and protein bound) were measured in frozen plasma samples by an HPLC method [14] using a commercial kit (HPLC LC-9A, Shimadu, Tokyo, Japan). The intra- and inter-assay coefficients of variation for plasma total homocysteine are 3.0% ($n=12$) and 3.3% ($n=30$), respectively. Plasma folate and vitamin B₁₂ (cobalamin) concentrations were determined by means of Chemiluminescent Enzyme Immunoassay using a commercial kit (Access2, Beckman Coulter, Fullerton, CA, USA). Plasma vitamin B₆ (pyridoxal phosphate) concentrations were also determined by means of a HPLC method using a commercial kit (HPLC L-6000, Hitachi, Tokyo, Japan).

Serum total cholesterol (TC) and triglycerides (TG) were determined enzymatically, and fasting plasma glucose (FPG) was assayed by a glucose oxidase method. Serum high-density

lipoprotein–cholesterol (HDL) was measured by the heparin–manganese precipitation method. Low-density lipoprotein cholesterol (LDL) was estimated according to the Friedewald formula [15]. Insulin measurements were performed by an enzyme immunoassay method. To assess insulin sensitivity, HOMA-r index was calculated as insulin ($\mu U/mL$) \times FPG (mg/dL)/405.

Smoking status

Smoking status (smoking or not smoking) and number of current cigarettes per day were assessed by a questionnaire. Brinkman index [16] was calculated as number of current cigarettes per day \times years.

Maximal oxygen uptake

Maximal oxygen uptake ($\dot{V}O_{2\max}$) was determined during a graded exercise test using a cycle ergometer (818E, Monark, Stockholm, Sweden). Following a 2-min warm-up, the subject started with a workload of 15 watts, which was increased by 15 W each minute until volitional exhaustion occurred. Pulmonary ventilation and gas exchange were measured breath-by-breath with an on-line data acquisition system (Oxycon alpha System, Mijndhardt, Breda, Netherlands).

Diet and exercise regimens

Dietary protocol

We have reported the effect of a low-calorie diet intervention on body composition, body fat distribution and coronary heart disease risk factors [17]. Based on the diet protocol of the above study, all subjects were instructed to take meals per day consisting on average of 420 kcal of protein, 840 kcal of carbohydrate and 420 kcal of fat. Subjects kept daily food diaries during the 14-week intervention period and learned about proper daily nutrition (well-balanced protein, carbohydrates, fat, various amino acids, vitamins and minerals) through weekly lectures and counseling by skilled dieticians.

Exercise protocol

In addition to restricting energy intake, the subjects from the DE group performed exercise training with walking and jogging for 40 sessions (3 days per week) supervised by two or three physical trainers. In the first 2 months the exercise training was only walking with the target Borg's scale [18] ranged from 11 (light) to 13 (fairly hard). The distances were 3.5 km and 4.5 km in the first and second months, respectively. In the last month subjects performed a combination of a 3.0-km brisk walking and a 1.0-km middle-intensity jogging with the