Efficiency of a Free-Living Physical Activity Promotion Program Following Diet Modification for Fat Loss in Japanese Obese Men

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Summary The aim of the current study was to examine the efficiency of a free-living physical activity promotion (PAP) program following a diet modification program for fat loss. Fifty obese men, aged 51.4±7.0 y, received a 6-mo regimen consisting of 2 phases. Weekly 90-min free-living PAP sessions were provided for 3 mo immediately after a 3-mo diet modification phase. Fat mass (FM) was measured at baseline and at months 3 and 6 using dual energy X-ray absorptiometry. The time spent in moderate-vigorous physical activity (MVP A) was monitored by a validated single-axis accelerometer. The total energy intake of all participants was assessed at baseline and during weeks 9 and 10 of both the diet modification and PAP programs. The change (Δ) in FM was −1.4±1.9 kg (p<0.05) during the PAP program after the diet modification program (−4.3±2.9 kg, p<0.01). Although there was no significant relationship between ΔFM and ΔMVP A during the PAP phase (r=0.11), MVP A was significantly increased during the PAP phase (+76.0±146.5 min/wk, p<0.01). However, a significant correlation was observed between energy intake/weight and MVP A during the PAP program (r=0.39). Our results suggest that the magnitude of expected FM loss induced by an increase in PA may be suppressed as a result of increased energy intake, even during a PAP program after a diet modification program.

Key Words physical activity, fat loss, obesity, energy compensation

Weight loss can be induced by diet and exercise. Although weight loss resulting from the combination of diet and exercise can be significant (−11.4%), compared to diet (−11.1%) or exercise (−3.5%) alone (1), the combination of diet and exercise does not always achieve the same level of weight loss as diet and exercise alone (2, 3). A study by Martin et al. (4) reported that diet restriction resulted in reduced energy expenditure by physical activity (PA). This report suggested that a combination of simultaneous diet and exercise might compensate the efficiency of exercise-induced weight loss. However studies by Fogelholm et al. (5) and Kukkonen-Harjula et al. (6) recommended the use of a weight-loss program that incorporates exercise after diet-induced weight loss. This approach resulted in successful weight loss (men: 13.2 kg in 8 mo, women: 13.8 kg in 12 mo). As many obese individuals are sedentary and are at risk of knee or back injuries during exercise secondary to excess weight, the initiation of exercise after weight loss may be a better approach from both the weight-loss and safety standpoints (7–9).

Although PA is well known to prevent obesity, all individuals do not lose the same amounts of weight in response to exercise. Bouchard et al. (10) reported the results from a highly controlled study in which participants were sequestered to an isolated research station. Energy intake was prescribed at a specific level to maintain baseline weight, and exercise was prescribed to create a 1,000 kcal daily energy deficit. Nevertheless, weight loss ranged from −3 to −12 kg. Donnelly et al. (11) conducted a randomised controlled exercise trial in which all exercises were performed under direct supervision, and total energy expenditure was measured according to the doubly labeled water (DLW) method. In this report, the range of weight change was +4 to −14 kg (12). Individual differences in response
to increased PA can be greatly affected by increases in energy intake (13) or decreases in non-training PA (14, 15) during an exercise regimen.

Compensation for altered energy balance in individuals adopting a diet and exercise program may be caused by several factors, including a decrease in energy expenditure as a result of diet restriction, an increase in energy intake, or a decrease in non-training PA. Hence, a program divided between diet and exercise could be more effective at promoting weight loss because the separation of diet and exercise may be able to prevent reductions in energy expenditure and non-training PA. However, little information is available as to whether a PA promotion program after a period of diet modification is effective at promoting additional weight loss. The purpose of this study was to examine the efficiency of a free-living PA promotion after the diet modification program for fat mass (FM) loss.

METHODS AND PROCEDURES

Participants. Participants were recruited from the community through the use of advertisements in local newspapers and flyers. We received phone calls from 74 male volunteers. We adopted the following eligibility criteria: male gender, aged between 40 to 64 y, without a history of cardiovascular disease (CVD) (such as myocardial infarction or stroke) and with a body mass index (BMI) greater than 25 kg/m², according to the guideline set by the Japan Society for the Study of Obesity (16). In Japan, although only 2–3% of the population has been characterized as having a BMI ≥30 kg/m², the prevalence of metabolic disorders is relatively high (16, 17). Thus, the cutoff value for the definition of obesity among Asian populations was used (18). In total, 62 obese men who met these criteria participated in this study. Twelve participants were excluded because they had incomplete data or they did not successfully complete the study program. Therefore, data on the remaining 50 men were used for the subsequent analyses. We fully explained the purpose and design of the study to each participant before they provided written informed consent. The research protocol was approved by the institutional review board at the University of Tsukuba (No. 21-210) and thus met the standards of the Declaration of Helsinki.

Weight loss program. This 6-mo weight loss program consisted of 2 phases: a 3-mo free-living PA promotion program followed a 3-mo diet modification program.

1) Dietary modification program: The dietary program consisted of weekly group-based, 90-min instructional sessions for 3 mo, as well as individual counseling by trained staff. In each session, participants received lectures on low-calorie diets and eating behaviors. Our dieticians instructed them to consume a well-balanced, 1,680-kcal diet per day. The energy intake per meal (three times per day) was 80 kcal from eggs and/or dairy products, 80 kcal from vegetables and fruits, 80 kcal from meat, fish and/or soybean products, and 320 kcal from carbohydrates and oils. Participants kept a daily food diary in which they recorded all the foods that they ate. The dieticians carefully reviewed the participants’ diaries and provided individualized feedback to the participants regarding energy intake (1,680 kcal) and nutritional balance at every session. The participants were instructed to maintain their current exercise habits during the diet program as much as possible. These methods have been previously described in detail elsewhere (19).

2) Free-living physical activity promotion program: Weekly 90-min PA sessions were provided for 3 mo. Each session began with 30 min of lectures followed by 60 min of brisk walking performed outdoors. The lectures consisted of a sequential presentation of various self-control techniques that were typically used in behavioral programs for weight reduction (20). Techniques included self-monitoring, stimulus control strategies, self-efficacy, decisional balance and processes of change. Well-trained physical instructors supervised all PA promotion sessions. The targeted time spent in moderate-vigorous PA (MVPA) was 250 min/wk (21), which included PA performed at each session. Participants were encouraged to increase PA during daily living at a moderate-to-vigorous intensity. For the first 4 wk, the targeted MVPA duration was 200 min/wk. Subsequently, the target duration increased to 250 min/wk for the next 4 wk and 300 min/wk for the last 4 wk. During the PA promotion program, there was no instruction related to dietary habits.

Anthropometry and body composition. Body heights were measured at baseline to the nearest 0.1 cm (YG-200; Yagami, Nagoya, Japan). Participants were weighed at baseline and at months 3 and 6. Body weights were measured to the nearest 0.05 kg while participants were wearing their underwear and a cloth hospital gown (WB-150; TANITA, Tokyo, Japan). BMI values were calculated as weight (in kilograms) divided by height (in meters) squared. Fat mass, fat-free mass and the percentage of fat mass were measured at baseline and at months 3 and 6 by dual energy X-ray absorptiometry (DXA) using a Hologic QDR 4500A densitometer (Hologic, MA, USA). During each measurement, the participant remained motionless in the supine position while the fat-scanning arm passed over his body in parallel 1-cm strips. To minimize technical error, the same examiner operated the densitometer and positioned the participant for all DXA examinations.

The intra-abdominal fat area (IFA) and the subcutaneous fat area (SFA) were measured at the level of the umbilicus using computed tomography scans performed in the supine position. The IFA and SFA were calculated using a computer software program (FatScan; N2system, Ibaraki, Japan).

Blood pressure and blood biochemistries. Blood pressure measurements and blood biochemistries were collected at baseline and at months 3 and 6. Systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured from the left arm using a mercury manometer after the participants rested for at least 15 min in a sitting position. The lower value of two readings was...
considered to represent the outcome measure. A blood sample of approximately 10 mL was drawn from the antecubital vein of each participant after an overnight fast (of at least 8 h). Serum total cholesterol (TC), serum high-density lipoprotein cholesterol (HDLC) and triglyceride (TG) levels were determined enzymatically. Fasting plasma glucose (FPG) was assayed using the glucose oxidase method, and serum low-density lipoprotein cholesterol (LDLC) was estimated according to Friedewald’s formula (22). Hemoglobin A1c (HbA1c) was measured using a particle-enhanced immunoturbidimetric assay (Kyowa Medex, Tokyo, Japan). The inter-assay coefficients of variation for each measurement were 0.4–2.2% for TG, 0.5–0.8% for FPG, and 0.8–1.0% for TC, 0.5–0.9% for HDLC, 0.9–1.6% for LDLC, and 0.9–1.6% for HbA1c.

### Aerobic fitness

Peak oxygen uptake (VO2peak) for each participant was determined according to a graded exercise test using a cycling ergometer (828E, Monark, Stockholm, Sweden). Following a 2-min warm-up period at 0 W, the workload increased every minute by 15 W. A validated equation (n=83, r=0.83, standard error of the estimate=3.66 mL/kg/min) using a workload at a ratings of perceived exertion (RPE) of 15 during the test was applied to estimate the VO2peak (23).

### Energy intake

Total energy intake, in kilocalories, was assessed according to 3-d weighed dietary records (WDR) (24), which included 2 weekdays and 1 weekend day. Total energy intake was measured prior to the onset of the program and during weeks 9 and 10 of both the diet modification and increased PA regimens. Dietary recall interviews for each participant were performed by skilled dieticians who explained to the participants how to complete the 3-d WDR in detail before taking the measurements. The dietary data for each participant were analyzed with commercially available computer software (Excel Eiyo-kun, Kenpakusha, Tokyo, Japan).

### Physical activity

PA levels were measured using single-axis accelerometers (Lifecorder-EX; Suzuken Co. Ltd., Nagoya, Japan). The accelerometer was firmly attached to the participant’s clothing (belt or waistband) during all waking hours (except while bathing). PA levels were measured 2 wk before starting the intervention (for the baseline examination) and throughout the dietary modification and PA promotion programs. The accelerometer was designed to determine the level of movement intensity every 4 s. The intensity of PA was determined according to the frequency of steps and the magnitude of vertical acceleration, which was categorized into 4 components with 4 thresholds (threshold 1: 0.06 g, threshold 2: manufacturer’s fixed values, threshold 3: manufacturer’s fixed values, and threshold 4: 1.96 g) (25, 26).

### Table 1. Descriptive characteristics and their changes throughout the study period.

|                          | Baseline | Month 3 | Month 6 |
|--------------------------|----------|---------|---------|
| Age, y                   | 51.4±7.0 |         |         |
| Height, cm               | 170.4±6.0|         |         |
| Weight, kg               | 88.4±16.6| 80.5±13.8a| 79.4±14.5ab|
| BMI, kg/m²               | 30.4±4.8 | 27.7±4.0a| 27.3±4.2ab|
| Energy intake, kcal/d    | 2.368±0.469| 1.601±26.7a| 1.685±36.2a|
| Percentage of FM,%       | 25.5±4.3 | 22.7±4.3a| 21.2±4.4ab|
| FFM, kg                  | 63.1±8.5 | 59.8±7.7a| 60.0±8.3a |
| FM, kg                   | 23.0±8.5 | 18.8±6.8a| 17.4±6.7ab|
| IFA, cm²                 | 150±52   | 119±44a  | 103±46ab |
| SEA, cm²                 | 232±84   | 187±99a  | 169±98ab |
| SBP, mmHg                | 130.5±16.7| 121.2±15.3a| 121.8±15.1a|
| DBP, mmHg                | 84.8±15.0 | 76.9±12.2a| 78.7±10.7a|
| TC, mg/dl                | 203.2±29.9| 184.6±27.0a| 202.3±26.4a|
| HDLC, mg/dl              | 127.3±28.8| 116.6±24.6a| 126.9±25.2a|
| TG, mg/dl                | 46.0±10.4 | 49.9±10.2a| 55.6±11.7ab|
| FPG, mg/dl               | 154.4±122.4| 90.9±37.8a| 98.4±57.6a |
| HbA1c, %                 | 5.3±0.9   | 5.3±0.7a  | 5.2±0.7a  |
| VO2peak, mL/kg/min       | 32.8±3.7 | 34.5±4.1a | 35.4±4.0ab|

BMI: body mass index, FM: fat mass, FFM: fat-free mass, IFA: intra-abdominal fat area, SEA: subcutaneous fat area, SBP: systolic blood pressure, DBP: diastolic blood pressure, TC: total cholesterol, HDLC: low-density lipoprotein cholesterol, LDLC: high-density lipoprotein cholesterol, TG: triglyceride, FPG: fasting plasma glucose, HbA1c: hemoglobin A1c, VO2peak: maximal oxygen uptake.

Values are presented as the mean±SD.

Significance was determined by one-way ANOVA.

*The value are significantly different from baseline.

abThe value are significantly different from month 3.

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during a 4-s period, a value of 1–9, according to the intensity of the PA (1 = minimal, 9 = maximal intensity), was recorded. The PA was classified into three activity categories: light PA (LPA: PA levels 1–3), representing 1.8 to 3.5 metabolic equivalents (METs); moderate PA (MPA: PA levels 4–6), representing 3.5 to 6.1 METs; and vigorous PA (VPA: PA levels 7–9), representing greater than 6.1 METs (25). Data from participants who had worn the accelerometer for at least 10 h per day for at least 4 weekdays and 1 weekend day were considered valid (26). The PA variables used in this study consisted of the time spent in LPA, MPA, VPA and MVP A (MPA plus VPA). Total PA represented the sum from LPA to VPA. PA variables were calculated on a daily basis and then used to estimate weekly activity by taking a weighted average of daily weekday and weekend activity. Weekly MVP A was calculated using the following formula: (average daily weekday MVP A×5)+(average daily weekend MVP A×2).

Statistical analyses. Values are expressed as the mean±SD. One-way analysis of variance (ANOVA) was employed to compare serial changes (at baseline and at months 3 and 6) in the studied variables. If a significant difference was detected, a post hoc comparison with the Bonferroni correction was applied. The relationships between energy intake/weight and PA, changes (Δ) in weight and ΔPA, Δweight and ΔMVP A, and Δmeasurement items and ΔPA during the increased PA program were assessed using Pearson’s product-moment correlation. Multiple-regression analysis was used to explore the influence of ΔMVP A, Δenergy intake/weight and age on ΔFM. A p value less than 0.05 was considered statistically significant. The data were analyzed with SPSS, version 18.0 (SPSS Japan Inc., Tokyo, Japan).

RESULTS

The attendance rate was 86.4% (range 45.5 to 100%) for the diet modification phase and 74.3% (range 16.7 to 100%) for the PA promotion phase. The average recorded heart rate during brisk walking in the PA promotion phase was 113.7 ± 10.1 bpm, which corresponded to 66.2 ± 10.9% of the age-predicted maximal heart rate (220 minus chronological age). Of the 50 total participants, 27 met the target MVP A duration at month 4, 22 attained this value at month 5, and 18 attained this value at month 6.

The change in weight was −7.9 ± 2.1 kg (−8.7 ± 4.6%, p<0.01) from baseline to month 3 and −1.1 ± 2.8 kg (−1.3 ± 2.9%, p<0.05) from month 3 to 6. The change in FM was −4.3 ± 2.9 kg (−18.3 ± 8.9%, p<0.01) from baseline to month 3 and −1.4 ± 1.9 kg (−5.9 ± 7.5%, p<0.01) from month 3 to 6 (Table 1). The BMI value and percentages of FM, FFA, SFA, HDLC and VO2peak significantly improved during the diet modification phase and further improved during the PA promotion phase. The energy intake, FFM, SBP, DBP, TG, FPG and HbA1c measurements significantly decreased during the diet modification phase and did not change.
during the PA promotion phase. In addition, TC and LDL-C decreased significantly during the diet modification phase but returned to baseline during the PA promotion phase.

The changes in PA during the 6-mo study period are summarized in Fig. 1. PA levels were maintained during the diet modification phase. MVP A and MP A were increased gradually during the PA promotion phase. Total PA and LP A decreased at month 4, whereas VP A increased gradually until month 5 but slightly decreased by month 6.

The relationships between energy intake/weight and PA during the PA promotion phase are shown in Fig. 2. Significant positive correlations were observed between energy intake/weight and MVP A, total PA and MP A. No significant correlations existed between energy intake/weight and LP A or between energy intake/weight and VP A.

No significant correlations existed between ∆FM and ∆PA (at any time point or intensity) during the PA promotion phase.
motion phase (Fig. 3). In addition there was individual variability in MVPA and FM reduction (Fig. 4).

Multiple-linear regression analyses were performed to assess the independent effects of ΔMVPA, Δenergy intake/weight and age on ΔFM during the PA promotion phase. The overall model including ΔMVPA, Δenergy intake/weight and age was significant (p<0.05). However, a multiple coefficient of determination was 0.12, and none of the three independent variables were not significant.

Table 2 shows the relationship between changes in cardiovascular risks and ΔPA during the PA promotion phase. There were significant relationships between ΔMVPA and ΔIF A, ΔDBP and ΔHbA1c. Moreover, the change in total PA significantly correlated with ΔDBP and ΔHbA1c. The change in LPA and ΔMPA significantly correlated with ΔDBP.

**DISCUSSION**

This study was designed to examine whether a free-living PA promotion program after a diet modification facilitated additional fat loss without requiring increased energy intake to compensate for the increased physical activity. Previous studies have reported that long-term exercise programs do not achieve the expected levels of weight loss, because energy expenditure for other physical activities decreases after exercise and because total energy intake increases throughout the day (4, 13–15). We hypothesized that a diet modification program could help to maintain energy intake at an appropriate level during a physical activity program and that a free-living physical activity promotion program would be more beneficial for increasing total energy expenditure than supervised exercise. However, our results suggest that increasing energy intake, even with prior dietary modification, could compensate for the increased energy expenditure resulting from increased physical activity.

We further hypothesized that a program with separate dietary and exercise-related interventions could be more effective for FM loss. Estimates of activity energy expenditure from MVPA during the PA promotion phase were as follows: 3.5 METs×55.6 h×79.4 kg=15,451.2 kcal; 15,451.2 kcal/7,200 kcal (per FM, kg)=2.1 kg. However, our participants reduced their FM by only an average of 1.4 kg during the PA promotion phase. One

![Fig. 4. Individual variability of ΔFM and MVPA during the PA promotion phase. FM: fat mass, MVPA: moderate-vigorous physical activity.](image-url)
explanation for this result could be that individuals who increased their PA also increased their energy intake (Fig. 2), even after adhering to the previous dietary modifications. Previous studies have reported that long-term exercise (≥3 mo) can increase the orexigenic drive or energy intake, whereas acute and short-term exercise can improve appetite and reduce FM (27–29). However, the studies by Church et al. (30) and Ross and Janssen (31) were unable to demonstrate a dose-response relationship between PA and weight loss. Additionally, a recent report by King et al. (32) suggested that increased energy intake may represent a major source of energy compensation. In this previous study, after 12 wk of maintaining an energy expenditure of 2,500 kcal/wk through exercise training, there was an apparent increase in energy intake among individuals who did not lose the predicted amount of weight (compensators), whereas there was no change in energy intake among individuals who lost the predicted amount of weight (non-compensators). Among our participants, significant reductions in FM were observed, while significant increases in PA were also observed during the PA promotion phase (Table 1 and Fig. 1). However, we were unable to find a significant correlation between ΔFM and ΔPA during the PA promotion phase (Figs. 3 and 4), and a multiple-linear regression analysis that was performed to assess the independent contributions of ΔMVP A, Δenergy intake/weight and age to ΔFM during the PA promotion phase revealed that none of the three independent variables was significant. On the other hand, there were significant positive correlations between energy intake/weight and PA.

Thus our data suggest that the magnitude of the expected FM loss induced by an increase in PA may be suppressed by increased energy intake even when the PA promotion phase follows a diet modification phase. However, in this study, there may be some other interpretations for the Δweight observed during the free-living PA promotion program. For example, the magnitude of PA increase from the diet phase to the PA promotion phase may have not been large enough to substantially reduce FM during the free-living PA promotion program.

Our free-living PA promotion program after diet modification may have protected against compensation via the decrease in non-training PA as a result of an increase in free-living PA. In a review study, Westerterp (14) reported that aerobic exercise intervention was not shown to affect non-training PA in young participants but was shown to affect middle-aged and older patients due to fatigue. Moreover, from reports of middle-aged and older participants, the implementation of a resistance-training intervention was able to increase total energy expenditure and free-living PA. Additionally, Leon et al. (15) conducted an aerobic exercise intervention in young obese men but found that these participants failed to lose weight because non-training PA was reduced during the intervention. Therefore these previous studies suggest that it is difficult for obese middle-aged and older participants to increase free-living PA through an aerobic exercise intervention. However we speculate there was very little effect of fatigue on exercise training in our study because all levels of free-living PA increased gradually during the PA promotion phase. As a result of the free-living PA promotion following diet modification, our participants did not decrease their non-training PA levels, which indicates that obese individuals who lost weight could engage in increased free-living PA at this lower weight.

Although we were unable to identify a significant relationship between ΔFM and ΔPA during the PA promotion phase, there were significant correlations between ΔMVP A and ΔIF A. High-intensity exercise training has been shown to reduce IF A to a greater extent than low-intensity exercise, although it carries identical energy costs (33). The results of our study support this hypothesis, as changes in DBP correlated with ΔMVP A, ΔIF A and ΔMP A. Moreover, Ishikawa-Takata et al. (34) reported in a randomized dose-response study that the magnitude of the reduction in DBP was significantly related to exercise time. In support of these results, we found that there was a significant relationship between ΔDBP and ΔPA (except for ΔVP A). In addition changes in HbA1c significantly correlated with ΔMVP A and Δtotal PA, although ΔFPG was not associated with any particular ΔPA intensity. Hansen et al. (35) observed that HbA1c improved with vigorous-intensity exercise training (moderate- to high-intensity), although FPG was not improved. They also reported that HbA1c was a more reliable measure of glucose metabolism due to its stability.

There were some limitations of the current study. First, the study design did not include a control group. Second, we measured the participants’ energy intake by 3-d WDRs. There may have been an underestimation or overestimation of total energy intake along with carbohydrate, fat and protein intakes. Third, only men were included in the study. In Japan, the percentage of overweight/obese individuals continues to increase among the middle-aged population. However it remains unclear whether our results can be extrapolated to women and elderly adults. In addition, we acknowledge our relatively small sample size as a limitation.

One strength of the current study was our ability to detect changes in energy expenditure (accelerometers) in objective ways. Self-reporting appears to overestimate PA (36–38), especially with respect to assessing PA in overweight adults. Self-reporting can introduce recall bias and coding errors due to misclassification in the intensity, duration and frequency of PA. Another objective method for assessing PA is DLW; although DLW is one of the criteria used for measuring total energy expenditure (TEE) under free-living conditions, DLW cannot detect short-term TEE (time- or day-stamped) or the time and intensity of PA. Conversely, accelerometers are capable of measuring the intensity, frequency, and duration of PA, and accelerometers are more cost-effective than DLW. Moreover previous studies similar to ours have used accelerometers to quantify PA levels after diet-induced weight loss.

In conclusion, we examined the efficiency of a free-
living PA promotion program following diet modification on fat loss. Although we found that an increase in PA contributed to weight loss and improved CVD risks, we could not identify a significant relationship between ∆FM and ∆PA during the PA promotion phase. These results suggest that the magnitude of the expected FM loss induced by an increase in PA may be suppressed by an increase in energy intake, even when the PA phase follows a diet modification phase.

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