Reduction in Obesity and Related Comorbid Conditions after Diet-Induced Weight Loss or Exercise-Induced Weight Loss in Men

A Randomized, Controlled Trial

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Background: **The independent effects of diet- or exercise-induced weight loss on the reduction of obesity and related comorbid conditions are not known. The effects of exercise without weight loss on fat distribution and other risk factors are also unclear.**

Objective: **To determine the effects of equivalent diet- or exercise-induced weight loss and exercise without weight loss on subcutaneous fat, visceral fat, skeletal muscle mass, and insulin sensitivity in obese men.**

Design: **Randomized, controlled trial.**

Setting: **University research center.**

Participants: **52 obese men (mean body mass index [±SD], 31.3 ± 2.0 kg/m^2) with a mean waist circumference of 110.1 ± 5.8 cm.**

Intervention: **Participants were randomly assigned to one of four study groups (diet-induced weight loss, exercise-induced weight loss, exercise without weight loss, and control) and were observed for 3 months.**

Measurements: **Change in total, subcutaneous, and visceral fat; skeletal muscle mass; cardiovascular fitness; glucose tolerance and insulin sensitivity.**

Results: **Body weight decreased by 7.5 kg (8%) in both weight loss groups and did not change in the exercise without weight loss and control groups. Compared with controls, cardiovascular fitness (peak oxygen uptake) in the exercise groups improved by approximately 16% ($P < 0.01$). Although total fat decreased in both weight loss groups ($P < 0.001$), the average reduction was 1.3 kg (95% CI, 0.3 to 2.3 kg) greater in the exercise-induced weight loss group than in the diet-induced weight loss group ($P = 0.03$). Similar reductions in abdominal subcutaneous, visceral, and visceral fat–to–subcutaneous fat ratios were observed in the weight loss groups ($P < 0.001$). Abdominal and visceral fat also decreased in the exercise without weight loss group ($P = 0.001$). Plasma glucose and insulin values (fasting and oral glucose challenge) did not change in the treatment groups compared with controls ($P = 0.10$ for all comparisons). Average improvement in glucose disposal was similar in the diet-induced weight loss group (5.6 mg/kg skeletal muscle per minute) and in the exercise-induced weight loss group (7.2 mg/kg skeletal muscle per minute) ($P > 0.2$). However, these values were significantly greater than those in the control and exercise without weight loss groups ($P < 0.001$).**

Conclusions: **Weight loss induced by increased daily physical activity without caloric restriction substantially reduces obesity (particularly abdominal obesity) and insulin resistance in men. Exercise without weight loss reduces abdominal fat and prevents further weight gain.**

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In 1997, the World Health Organization published a landmark document recognizing obesity as a worldwide disease that poses a serious threat to public health (1). Persons who are overweight or obese have substantially increased risk for morbidity from numerous chronic disorders, such as diabetes (2, 3), hypertension (4, 5), and cardiovascular disease (6, 7). Obesity-related health risk is greater when excess fat is deposited in the abdomen region because this phenotype is a stronger predictor of cardiovascular disease and type 2 diabetes mellitus than general obesity is (8–11). This may be partially explained by excess accumulation of visceral fat, an independent correlate of insulin resistance (9–11) and dyslipidemia (8, 9). These observations highlight the need to identify appropriate treatment strategies to prevent and reduce obesity and suggest that the effectiveness of these treatments would be enhanced if abdominal obesity, particularly visceral fat, was substantially reduced.

Diet restriction remains the most common method of obesity reduction (12). Despite the observation that low levels of physical activity are a major cause of obesity (13), increased physical activity alone is not thought to be a useful strategy for obesity reduction. Some reports have suggested that physical activity in obese adults results in only modest weight loss (approximately 1 to 2 kg) independent of the effects of diet restriction (14). However, these conclusions are drawn from studies in which individual energy intake and expenditure were not rigorously controlled or accurately measured (15–17). Moreover, in most studies, the negative energy balance induced by exercise

was modest enough that substantial weight loss was not expected (15–17). Currently, no compelling evidence supports the observation that exercise alone is not a useful method for reducing total or abdominal obesity.

It is well known that a single exercise session is associated with a significant improvement in insulin-stimulated glucose uptake (18, 19). It is also clear that weight loss is associated with an improvement in insulin action (20–24). It is unclear, however, whether regular exercise improves glucose metabolism after the short-term effects of exercise and changes in body fat distribution are considered. Segal and colleagues (25) controlled for the confounding effect of the last exercise session and body composition changes and found that exercise had no independent effect on insulin sensitivity (25). Given the established importance of insulin resistance as an antecedent to both cardiovascular disease and type 2 diabetes mellitus (26), it is important to clarify whether exercise improves insulin action independent of fat loss.

We performed a randomized, controlled trial to determine the independent effect of diet-induced or exercise-induced weight loss on obesity and insulin resistance in moderately obese men. We also evaluated whether exercise without weight loss was associated with reductions in abdominal obesity and insulin resistance.

Methods

Participants

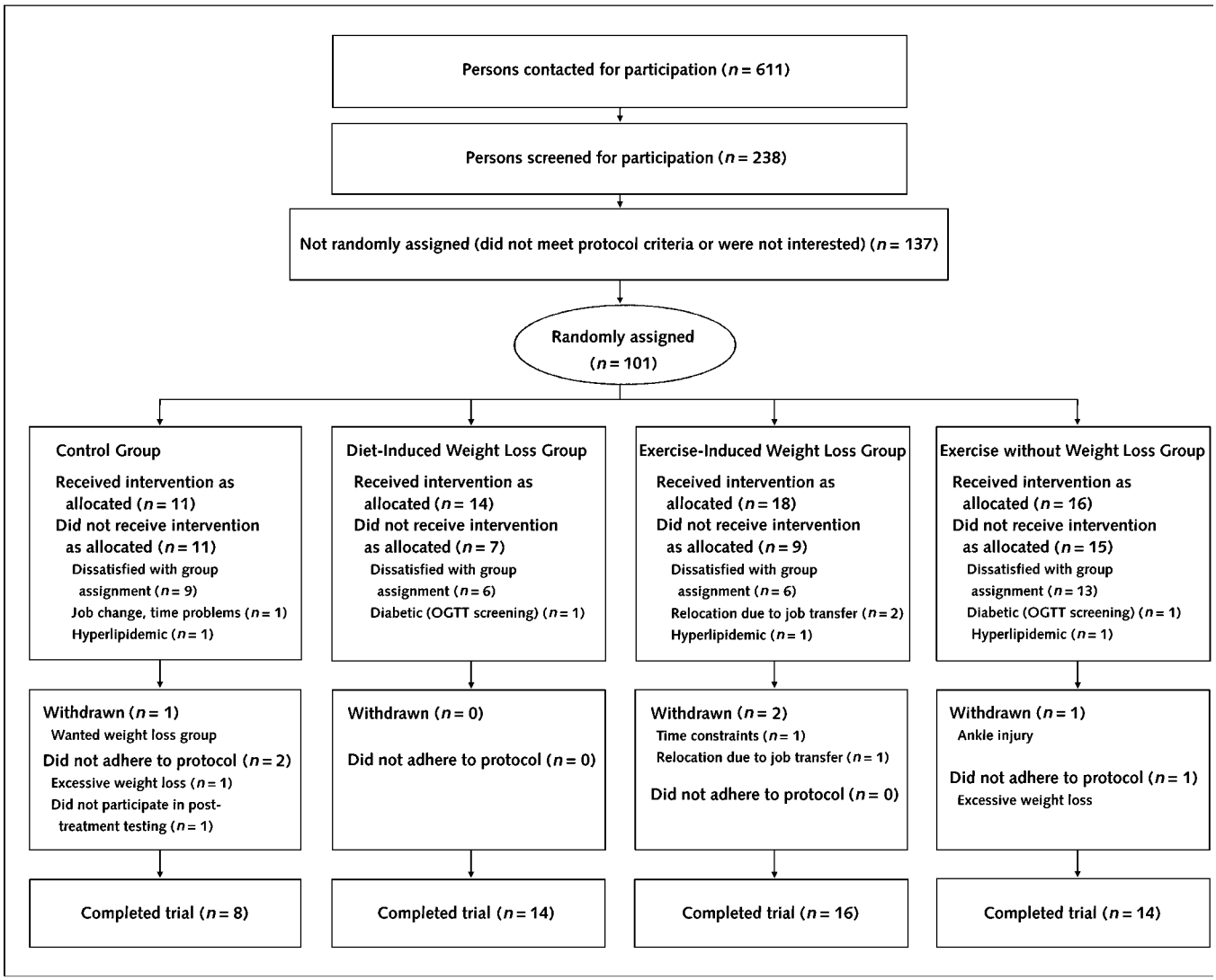
Participants were recruited from Kingston, Ontario, Canada, a typical suburban region, through the general media. We selected men with a body mass index greater than 27 kg/m^2 and a waist circumference greater than 100 cm whose weight had been stable ($\pm$2 kg) for 6 months before study entry. Participants were nonsmokers who consumed an average of fewer than two alcoholic beverages per day, had a sedentary lifestyle, and took no medications known to affect the principal outcome measures. All participants had a preparticipation medical examination that included screening for normal glucose tolerance and plasma lipid profile. A computer program was used to randomly assign eligible men to one of the following four groups: control, diet-induced weight loss, exercise-induced weight loss, and exercise without weight loss (**Figure 1**). Of the 101 participants who were randomly assigned to groups, 34 chose not to participate because they were dissatisfied with their assigned group, 5 were diabetic or dyslipidemic, and 3 were relocated because of job transfers. Those who chose not to participate and those who completed the trial were similar with regard to anthropometric variables. In addition, those who completed the trial were similar to those who did not in each group ($P > 0.10$). Baseline characteristics among groups were similar for all participants (**Table 1**). All participants gave fully informed written consent. The study was conducted in accordance with the ethical guidelines of Queen's University. The participants did not receive monetary compensation.

Diet and Exercise Regimen

During the baseline period, daily energy requirements for all participants were determined by estimating resting energy expenditure and multiplying the obtained value by a factor of 1.5 (27). All participants followed a weight maintenance diet (55% to 60% carbohydrate, 15% to 20% protein, and 20% to 25% fat) for a 4- to 5-week baseline period. During this period, body weight was monitored to determine the accuracy of the prescribed energy requirement, which was adjusted accordingly to maintain body weight. Controls were asked to maintain body weight throughout the 12-week treatment period. Participants in the diet-induced weight loss group were asked to reduce the isocaloric diet by 700 kcal/d during the treatment period to achieve a weekly weight loss of approximately 0.6 kg. To lose the same amount of weight, participants in the exercise-induced weight loss group were asked to maintain the isocaloric diet for the duration of the treatment period and to perform exercise that expended 700 kcal/d. Participants assigned to exercise without weight loss were asked to maintain body weight. Therefore, they consumed enough calories to compensate for the energy expended during the daily exercise sessions (approximately 700 kcal). At the end of the treatment period, isocaloric requirements were determined and prescribed for a 2-week weight stabilization period.

All participants were free-living and consumed self-selected foods. No vitamins or other nutritional supplements were prescribed. Each person participated in a series of weekly 1-hour seminars in which a dietitian taught proper food selection and preparation. Participants were told that the composition of the maintenance and energy-reduced diets should be approximately 55% to 60% carbohydrate, 15% to 20% protein, and 20% to 25% fat. Participants kept and analyzed daily, detailed food records

Figure 1. **Flow of participants through the study.**



OGTT = oral glucose tolerance test.

for the duration of the study period (approximately 20 weeks); the study dietitian also reviewed these records. For the 2-week period during which doubly labeled water measurements were acquired (weeks 6 and 7), the diet records were analyzed by using a computerized program (Food Processor, Esha Research, Salem, Oregon).

Participants in both exercise groups performed daily exercise (brisk walking or light jogging) on a motorized treadmill for the duration of the 12-week trial. The length of each exercise session was determined by the time required to expend 700 kcal. Participants were asked to exercise at an intensity not greater than 70% of their peak oxygen uptake ($\dot{V}O_2$) (approximately 80% of maximal heart rate). Energy expenditure was determined by using the heart rate and oxygen consumption data that were obtained from the pretreatment graded exercise test and were adjusted according to the results of two subsequent tests performed at weeks 4 and 8. During each session, heart rate was monitored every 5 minutes by using an automated heart rate monitor (Polar Oy, Kempele, Finland). All exercise sessions were by appointment and were supervised. Peak $\dot{V}O_2$ was determined by using a graded treadmill test and standard open-circuit spirometry techniques (SensorMedics, Yorba Linda, California).

Energy Expenditure

Total energy expenditure for 14 days was measured by using a two-point doubly labeled water method (28). Deuterium enrichment was analyzed by using a 903 deuterium dual-inlet isotope ratio mass spectrometer (VG Isogas, Cheshire, United Kingdom). Oxygen-18 was determined by using a SIRA 12 isotope ratio mass spectrometer (VG Isogas). Total energy expenditure was calculated by using the DeWeir formula (29). After an overnight stay in the hospital, resting metabolic rate was measured at 7:00 a.m. by using indirect calorimetry with a modified mask system (Teem 100, Aerosport, Inc., Ann Arbor, Michigan). Values were obtained for 30 minutes, and the last 25 minutes were used to determine resting metabolic rate. Resting systolic and diastolic blood pressure were measured when the participant was supine.

Magnetic Resonance Imaging and Anthropometric Measurements

Whole-body data from magnetic resonance imaging (approximately 45 equidistant images) were obtained with a General Electric 1.5-Tesla magnet (Milwaukee, Wisconsin) by using an established protocol described in detail elsewhere (30). Once acquired, the data were transferred to a stand-alone work station (Silicon Graphics, Mountain View, California) for analysis with specially designed computer software (Tomovision, Inc., Montreal, Canada), the procedures for which are described elsewhere (31). For adipose tissue (fat) and skeletal muscle, volume units (L) were converted to mass units (kg) by multiplying the volumes by the assumed constant density for fat (0.92 kg/L) and fat-free skeletal muscle (1.04 kg/L) (32). All anthropometric circumference measurements were obtained by using standard procedures described elsewhere (30).

Insulin Sensitivity and Glucose Tolerance

Participants consumed a weight-maintenance diet consisting of at least 200 g of carbohydrate for a minimum of 4 days and were asked to avoid strenuous physical activity for 3 days before insulin sensitivity was measured. Posttreatment data for the exercise group were obtained 4 days after the last exercise session. Participants stayed in the hospital the night before insulin sensitivity was measured. All studies were performed at 8:00 a.m. after a 12- to 14-hour overnight fast. An antecubital vein was catheterized for infusion of insulin and 20% glucose. An intravenous catheter was inserted in a retrograde fashion in a hand vein, and the hand was placed in a heating pad for sampling of arterialized blood. Insulin was infused at a rate of 40 mU/m^2 per minute for 3 hours. Plasma glucose was measured by using an automated glucose analyzer (YSI 2300 Glucose Analyzer, YSI, Yellow Springs, Ohio) every 5 minutes in arterialized blood. Indirect calorimetry was performed during the last 30 minutes of insulin infusion by using an open-circuit spirometry metabolic monitoring system (Teem 100, Ann Arbor, Michigan) to estimate glucose oxidation. Glucose disposal rate was calculated by using the average exogenous glucose infusion rate during the final 30 minutes of euglycemia. Nonoxidative glucose metabolism was determined by subtracting glucose oxidation, measured by calorimetry, from glucose disposal.

Before treatment and approximately 6 days after treatment, a 2-hour, 75-g oral glucose tolerance test was administered the morning after an overnight fast. Blood samples were collected from the antecubital vein at −15, 0, 30, 60, 90, and 120 minutes. Glucose was measured by using an automated glucose analyzer (YSI), and insulin was measured by using a radioimmunoassay kit (Intermedico, Toronto, Ontario, Canada). Areas under the glucose and insulin curves were determined by using a trapezoid model (33).

Statistical Analyses

We analyzed data from participants who completed the study; therefore, this was not an intention-to-treat analysis. A one-way analysis of variance was performed to examine differences among treatments before intervention. A 4 × 2 (group × time)–way analysis of variance with repeated measures was used to evaluate main treatment effects and interactions. A Bonferroni post hoc comparison test was used to locate significant treatment differences (interactions). The influence of treatment on fat distribution was determined by using analysis of variance (group × time) with the visceral–to–subcutaneous fat ratio. Relations between fat distribution (for example, visceral fat) and metabolic variables were determined by using Pearson correlation coefficients. Statistical procedures were performed by using SYSTAT (SYSTAT, Inc., Evanston, Illinois).

Role of the Funding Source

The funding source had no role in the analysis or interpretation of the data or in the decision to submit the report for publication.

Table 1. **Selected Anthropometric, Magnetic Resonance Imaging, and Metabolic Variables before Treatment and 3 Months after Treatment***

Characteristic	Control Group			Diet-Induced Weight Loss Group		
	Did Not Complete (n = 12)	Completed (n = 8)		Did Not Complete (n = 6)	Completed (n = 14)	
		Pretreatment	Post-Treatment		Pretreatment	Post-Treatment
Anthropometric						
Age, *y*	44.6 ± 11.2	46.0 ± 10.9		41.8 ± 7.6	42.6 ± 9.7	
Weight, *kg*	97.5 ± 10.9	96.7 ± 9.0	96.8	100.9 ± 11.6	96.1 ± 8.7	88.7†‡
Body mass index, kg/m^2	31.4 ± 2.3	30.7 ± 1.6	30.7	32.1 ± 3.3	30.7 ± 1.9	28.3†‡
Waist circumference, *cm*	108.8 ± 6.4	108.7 ± 4.7	108.6	113.8 ± 4.8	109.1 ± 5.6	102.0†‡
Waist-to-hip ratio	1.01 ± 0.02	1.00 ± 0.03	1.00	1.03 ± 0.05	1.01 ± 0.03	0.97†‡
Magnetic resonance imaging findings						
Total fat, *kg*		30.5 ± 4.5	29.9		28.4 ± 4.7	23.6†‡
Subcutaneous fat, *kg*		22.3 ± 3.5	22.1		21.8 ± 4.9	18.4†‡
Visceral fat, *kg*		4.1 ± 1.7	4.1		3.2 ± 1.0	2.3†‡
Visceral fat at L4–L5, cm^2		198 ± 71	198		170 ± 47	126†‡
Skeletal muscle, *kg*		34.1 ± 3.8	33.6		35.2 ± 5.0	33.5†
Metabolic						
Fasting glucose level, *mmol/L (mg/dL)*		5.0 ± 0.7 (91 ± 13)	4.9 (90)		5.3 ± 0.5 (97 ± 10)	5.1 (92)
Fasting insulin level, *pmol/L*		52 ± 29	54		56 ± 21	46
OGTT glucose level, *mmol/L · 2 h (mg/dL · 2 h)*		29.0 ± 6.8 (527 ± 124)	28.8 (523)		29.7 ± 5.9 (539 ± 107)	26.8 (489)
OGTT insulin level, *pmol/L · 2 h*		1457 ± 1200	1465		1508 ± 1122	1114
Glucose disposal, *mg/kg muscle per minute*		15.4 ± 6.0	14.4		13.0 ± 6.1	18.6†
Oxidative		3.4 ± 1.2	2.9		2.7 ± 1.5	2.8
Nonoxidative		12.0 ± 5.9	11.7		11.5 ± 5.4	17.1
Systolic blood pressure, *mm Hg*		119 ± 15	122		118 ± 11	118
Diastolic blood pressure, *mm Hg*		80 ± 10	80		83 ± 11	77
Maximum oxygen uptake, *L/min*		3.7 ± 0.8	3.7		3.6 ± 0.6	3.4

* Data are group means ± SD. OGTT = oral glucose tolerance test.
† Change (pre–post) is significantly greater compared with change in control group ($P < 0.05$).
‡ Change (pre–post) is significantly greater compared with change in exercise without weight loss group ($P < 0.05$).
§ Change (pre–post) is significantly greater compared with change in diet-induced weight loss group ($P < 0.05$).

Results

Adherence to the Exercise Program

For both exercise groups, average attendance at exercise sessions was 98% (range, 94% to 100%). Participants assigned to exercise without weight loss and participants assigned to exercise-induced weight loss did not differ in average duration (63.3 minutes compared with 60.4 minutes), intensity (77.0% compared with 77.0% of maximum heart rate), and energy expenditure per exercise session (692 kcal compared with 698 kcal) ($P > 0.2$ for all comparisons).

Daily Energy Intake and Expenditure

The values for average daily energy intake (diet records) and expenditure (doubly labeled water method) during a 2-week period (weeks 6 and 7) are given in **Table 2**. Although weight loss groups did not differ significantly in negative energy balance achieved ($P > 0.2$), daily energy expenditure in the exercise-induced weight loss group was greater than in the diet-induced weight loss group ($P = 0.01$). This finding, combined with the observation that the diet-induced weight loss group had a lower energy intake than the exercise-induced weight loss group, confirms that the negative energy balance in the diet-induced group was achieved through caloric restriction, whereas the negative energy balance in the exercise-induced group was caused by increased energy expenditure. Controls and participants assigned to exercise without weight loss were in energy balance.

Resting Energy Expenditure and Blood Pressure

The treatment groups had similar resting energy expenditure compared with controls ($P > 0.1$). However, the average increase in resting metabolism in the exercise without weight loss group (371 kcal/d) differed significantly from the decrease observed in both the diet-induced weight

Table 1—**Continued**

Exercise-Induced Weight Loss Group			Exercise without Weight Loss Group		
Did Not Complete (n = 10)	Completed (n = 16)		Did Not Complete (n = 15)	Completed (n = 14)	
	Pretreatment	Post-Treatment		Pretreatment	Post-Treatment
43.0 ± 6.6	45.0 ± 7.5		43.9 ± 9.0	44.7 ± 7.6	
96.7 ± 4.7	101.5 ± 7.7	94.0†‡	101.4 ± 8.6	97.9 ± 9.0	97.4
31.5 ± 1.4	32.3 ± 1.9	29.9†‡	32.5 ± 2.6	31.3 ± 2.3	31.1
108.4 ± 5.5	112.0 ± 5.0	105.5†‡	111.5 ± 8.2	110.0 ± 6.7	108.2
1.00 ± 0.04	1.01 ± 0.04	0.98†‡	1.01 ± 0.05	1.00 ± 0.05	0.99
	33.1 ± 5.5	27.0†‡§		30.6 ± 6.7	29.1
	24.9 ± 5.3	20.7†‡		23.4 ± 5.3	22.6
	3.9 ± 1.0	2.8†‡		3.4 ± 1.0	3.0†
	186 ± 59	134†‡		191 ± 88	159†
	35.2 ± 2.7	33.9		34.5 ± 3.1	34.9
	5.4 ± 0.4 (98 ± 7)	5.1 (93)		5.4 ± 0.6 (97 ± 11)	5.3 (97)
	70 ± 30	41		65 ± 38	55
	32.6 ± 5.5 (592 ± 100)	29.2 (530)‡		30.2 ± 7.5 (548 ± 136)	32.7 (594)
	2118 ± 1255	1219‡		1260 ± 625	1396
	11.2 ± 4.9	18.4†‡		11.8 ± 5.1	15.4
	2.8 ± 1.8	3.4		2.8 ± 1.6	2.8
	8.9 ± 4.0	14.5†		9.0 ± 4.9	12.6
	123 ± 14	118		125 ± 15	122
	83 ± 11	77		84 ± 9	83
	3.8 ± 0.8	4.3†§		3.7 ± 0.5	4.4†§

loss group (−211 kcal/d) and the exercise-induced weight loss group (−126 kcal/d). The weight loss groups did not differ significantly in average reduction in resting energy expenditure ($P > 0.2$) (**Table 3**). Compared with controls, changes in blood pressure in the other groups were similar ($P > 0.2$).

Weight Loss and Anthropometric Variables

The average weekly weight loss was similar for both the diet-induced weight loss group (0.6 kg) and the exercise-induced weight loss group (0.6 kg). The average total weight loss for the diet-induced weight loss group (7.4 kg) and the exercise-induced weight loss group (7.6 kg) repre-

Table 2. **Values for Energy Intake and Expenditure during the Doubly Labeled Water Period (14 Days during Weeks 6 to 7)***

Variable	Control Group	Diet-Induced Weight Loss Group	Exercise-Induced Weight Loss Group	Exercise without Weight Loss Group
	← kcal/d →			
Energy intake†	2706 ± 577‡	2019 ± 333	2612 ± 464‡	3335 ± 703§
Energy expenditure‖	3172 ± 436	2682 ± 464	3652 ± 724‡	3280 ± 520
Energy balance	−466 ± 566	−663 ± 447	−1039 ± 800¶	55 ± 804

* Data are expressed as group means ± SD.
† Determined by using daily diet records.
‡ Significantly greater than the diet-induced weight loss group ($P < 0.05$).
§ Significantly greater than the control, diet-induced weight loss, and exercise-induced weight loss groups ($P < 0.05$).
‖ Determined by using the doubly labeled water method (data were not obtained for two participants in the diet-induced weight loss group).
¶ Significantly greater than the exercise without weight loss group ($P < 0.05$).

sented approximately 8% of initial body weight. The average reduction in waist circumference was − 7.4 cm in the diet-induced weight loss group and −6.8 cm in the exercise-induced weight loss group. These values were similar to each other ($P > 0.2$) but significantly greater than values for controls ($P < 0.001$). The average change in waist circumference in participants assigned to exercise without weight loss was similar to that in controls ($P = 0.15$).

Cardiovascular Fitness

Compared with controls, the exercise groups had similar improvements in cardiovascular fitness (approximately 16%) ($P < 0.001$). However, cardiovascular fitness in the diet-induced weight loss group did not change ($P > 0.2$) (**Table 1**).

Total Fat and Skeletal Muscle

Compared with controls, both weight loss groups showed a significant reduction in total fat ($P < 0.001$). However, the average reduction in total fat was 1.3 kg (95% CI, 0.3 to 2.3 kg) greater in the exercise-induced weight loss group than in the diet-induced weight loss group ($P = 0.03$) (**Table 3**). The reduction in total fat in participants assigned to exercise without weight loss was similar to that in controls ($P > 0.2$). Compared with controls, skeletal muscle mass decreased in the diet-induced weight loss group but was unchanged in both exercise groups ($P > 0.10$) (**Table 1**).

Abdominal Fat

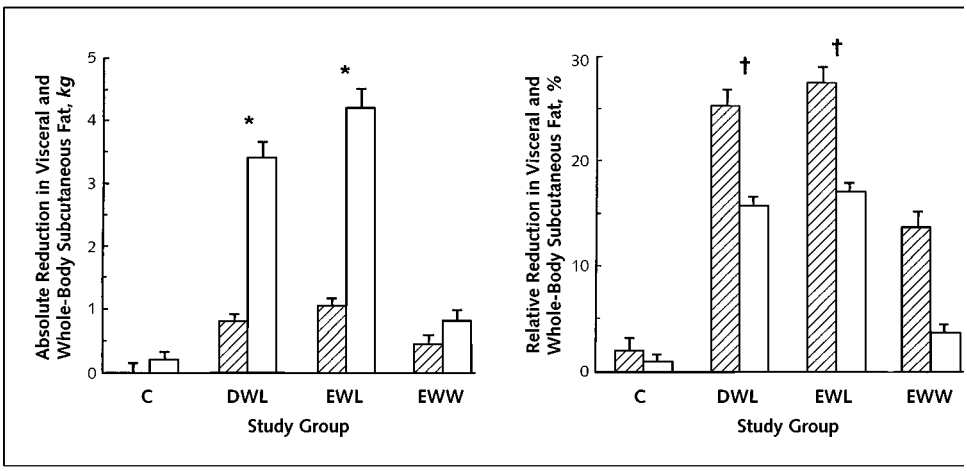
The average reduction in total abdominal fat (abdominal subcutaneous and visceral fat) in the diet-induced weight loss group (change, −1.5 kg) and exercise-induced weight loss group (change, −1.9 kg) differed significantly from that in controls ($P < 0.001$). However, the weight loss groups were similar to each other (difference, 0.4 kg [CI, 0.0 to 0.7 kg]; $P > 0.10$) (**Table 3**). The reduction in abdominal fat in the exercise without weight loss group was greater than in the control group ($P < 0.001$) but significantly less than in the weight loss groups ($P < 0.001$). The reduction in abdominal subcutaneous fat was similar in the weight loss groups ($P = 0.2$); however, both groups differed significantly from controls ($P < 0.001$) (**Table 3**). Reductions in visceral fat were significant in all treatment groups compared with controls ($P < 0.001$) (**Table 1**). The weight loss groups had similar reductions in visceral fat; however, this reduction was significantly greater than that seen in the exercise without weight loss group ($P < 0.001$) (**Table 3**). Collapsed across all groups ($n = 52$), changes in abdominal fat mass ($r = 0.69$) and visceral fat mass ($r = 0.59$) were signifi-

Table 3. **Comparison of Change Scores between the Diet and Exercise Weight Loss Groups**

Variable	Diet-Induced Weight Loss Group* ($n = 14$)	Exercise-Induced Weight Loss Group* ($n = 16$)	Difference [95% CI]
Anthropometric			
Weight, *kg*	−7.4 ± 0.9	−7.6 ± 0.6	0.2 [−0.4 to 0.8]
Waist circumference, *cm*	−7.4 ± 2.3	−6.9 ± 2.5	0.5 [−1.2 to 2.2]
Waist-to-hip ratio	−0.04 ± 0.02	−0.03 ± 0.02	0.01 [−0.04 to 0.06]
Fat distribution, *kg*			
Total fat	−4.8 ± 1.2	−6.1 ± 1.5	1.3 [0.3 to 2.3]†
Total subcutaneous fat	−3.4 ± 1.1	−4.2 ± 1.3	0.8 [−0.1 to 1.6]
Abdominal subcutaneous fat	−0.7 ± 0.4	0.8 ± 0.3	0.1 [−0.7 to 0.9]
Visceral fat	−0.8 ± 0.4	−1.0 ± 0.3	0.2 [0.0 to 0.5]
Abdominal fat	−1.5 ± 0.5	−1.9 ± 0.4	0.4 [0.0 to 0.7]
Skeletal muscle	−1.7 ± 1.0	−1.3 ± 0.8	0.4 [−0.3 to 1.0]
Metabolic			
Fasting glucose level, *mmol/L (mg/dL)*	−0.2 ± 0.5 (−4.6 ± 9.8)	−0.3 ± 0.6 (−5.1 ± 11.8)	0.0 [−0.4 to 0.4] (0.5 [−3.0 to 4.0])
Fasting insulin level, *pmol/L*	−10.5 ± 27.6	−28.9 ± 31.1	18.4 [−2.6 to 39.4]
Glucose area, *mmol/L · 2 h (mg/dL · 2 h)*	−2.9 ± 5.2 (−52 ± 94)	−3.4 ± 5.0 (−61 ± 91)	0.5 [−3.1 to 4.2] (9 [−57 to 75])
Insulin area, *pmol/L · 2 h*	−393 ± 780	−898 ± 997	505 [−133 to 1141]
Glucose disposal, *mg/kg muscle per minute*	5.6 ± 3.4	7.2 ± 5.3	1.5 [−1.7 to 4.8]
Maximum oxygen uptake, *L/min*	−0.2 ± 0.3	0.5 ± 0.3	0.7 [0.5 to 1.0]†
Resting energy expenditure, *kcal/24 h*	−212 ± 413	−126 ± 297	86 [−174 to 346]

* Data are presented as the group means ± SD.
† Significant treatment differences (diet compared with exercise) for change scores ($P < 0.05$).

Figure 2. **Reduction in whole-body subcutaneous fat (*white bars*) and visceral fat (*striped bars*) after a 3-month treatment period.**



The asterisks indicate a greater within-treatment reduction in subcutaneous fat than in visceral fat compared with the control group ($P < 0.01$). The daggers indicate a greater within-group relative reduction in visceral fat than in subcutaneous compared with the control group ($P < 0.01$). Data are expressed as the mean + SE of measurement. C = control group; DWL = diet-induced weight loss group; EWL = exercise-induced weight loss group; EWW = exercise without weight loss group.

cantly related to corresponding change in waist circumference ($P < 0.001$).

The ratio of visceral to subcutaneous (whole-body) fat was calculated to determine whether treatment influenced fat distribution (**Figure 2**). Both weight loss groups had similar reductions in this ratio ($P > 0.10$); however, these reductions were significantly greater than those seen in the control group and in the exercise without weight loss group ($P < 0.001$). Therefore, independent of the treatment used to induce weight loss, the relative reduction in visceral fat was greater than that in subcutaneous fat. The change in the ratio of visceral to subcutaneous fat in participants assigned to exercise without weight loss did not differ significantly from that in controls ($P > 0.2$). **Table 3** shows a comparison of the change in fat distribution and metabolic measurements (with associated 95% CIs) between the diet- and exercise-induced weight loss groups.

Insulin Sensitivity and Glucose Tolerance

Plasma glucose and insulin values obtained during the last 30 minutes of the clamp studies (pre- and post-treatment) did not differ within groups ($P > 0.05$, data not shown). Average improvement in glucose disposal was similar in the diet-induced weight loss group (5.6 mg/kg skeletal muscle per minute) and exercise-induced weight loss group (7.2 mg/kg skeletal muscle per minute) ($P > 0.2$). However, these values were significantly greater than those in controls ($P < 0.01$). Participants in the exercise-induced weight loss group showed a greater improvement than those assigned to exercise without weight loss ($P = 0.01$). Improvement did not differ between participants assigned to exercise without weight loss and controls ($P = 0.09$). When partitioned into oxidative and nonoxidative fractions (**Table 1**), glucose oxidation was unchanged independent of treatment. However, compared with controls, the rate of nonoxidative glucose disposal increased in the exercise-induced weight loss group alone ($P < 0.001$).

Compared with controls, similar changes were observed for fasting, glucose tolerance, or insulin area values ($P > 0.20$) (**Table 1**). However, for both glucose tolerance and insulin area values, the reduction in the exercise-induced weight loss group was greater than in the exercise without weight loss group ($P = 0.01$).

Relations between Fat Distribution and Insulin Sensitivity

Total and abdominal subcutaneous fat were not related to fasting, oral glucose tolerance, or insulin sensitivity variables before or after treatment ($n = 52$) ($P > 0.2$). However, visceral fat was related to both glucose area ($r = 0.47$) and glucose disposal ($r = -0.39$) before treatment ($P = 0.01$). These relations persisted throughout the treatment period; significant correlation coefficients were obtained between corresponding changes in visceral fat and both glucose area ($r = 0.35$) and glucose disposal ($r = -0.49$) ($P = 0.01$).

Discussion

Our findings demonstrate that exercise-induced weight loss reduces total fat and improves cardiovascular fitness significantly more than equivalent diet-induced weight loss. They also show that when weight loss induced by diet restriction or an increase in exercise is carefully matched, reductions in abdominal obesity, visceral fat, and insulin resistance are similar. Because the previous view has been that exercise alone reduces total and abdominal obesity only modestly or not at all, our findings have considerable relevance to public health. They strongly support the recommendation that either modality, caloric restriction alone or daily exercise without caloric restriction, is an effective strategy for reducing obesity in moderately obese men. Our findings also suggest that exercise without weight loss is a useful method for reducing abdominal fat and preventing further increases in obesity.

Reviews (34, 35) and meta-analyses (15–17) have discussed the efficacy of exercise, with or without diet restriction, as a method of obesity reduction. These reports concluded that exercise alone in overweight adults results in only modest weight loss (approximately 1 to 2 kg). These observations are consistent with the findings from 13 randomized, controlled trials, which suggest that physical activity alone in overweight men and women reduces total and abdominal fat only modestly (approximately 2 kg or 2%) or not at all (14). Our findings are in stark contrast to previous observations. Although we reaffirm that diet restriction is effective for reducing total and abdominal obesity, our findings also demonstrate that 12 weeks of approximately 60 minutes of daily exercise without caloric restriction is associated with substantial reductions in body weight (7.6 kg), total fat (6.1 kg), abdominal fat (1.9 kg), and visceral fat (1.0 kg).

The discrepant findings can probably be explained by differences in study design. Our treatment groups were well matched; in addition, the negative energy balance induced by exercise was substantial (approximately 700 kcal/d) and was carefully matched with the negative energy balance induced by caloric restriction. That participants did not partake in substantial physical activity beyond that prescribed was confirmed by the 24-hour energy expenditure values derived from doubly labeled water. This finding is in contrast to those of previous studies, in which the exercise and diet groups were usually not well matched, 24-hour energy intake and expenditure values were not rigorously controlled or accurately measured, and the prescription of exercise programs did not induce a meaningful negative energy balance (15–17).

Our study has several important limitations. Many of the volunteers were dissatisfied with their group assignment and subsequently chose not to participate. Because of the high dropout rate and because we did not perform an intention-to-treat analysis, we cannot make unequivocal statements about causation. However, given that age, body mass index, and waist circumference were similar in participants who completed the trial and in those who did not and given that the mechanism by which diet or exercise induces a negative energy balance (weight loss) is understood, it seems reasonable to infer an etiologic relation. We also acknowledge that the small number of participants in each group may have underpowered the study and we may therefore have been unable to detect true differences between treatments. Finally, because the response for the principal outcome measures varied in the intervention groups, we note that our findings are preliminary and should be confirmed in studies with larger cohorts.

A novel finding in our study is that an 8% diet- or exercise-induced weight loss is associated with similar reductions in abdominal obesity, visceral fat, and waist circumference. A similar reduction in the ratio of visceral to subcutaneous fat was also observed; this result reinforces the observation that during periods of negative energy balance, change in fat distribution favors a reduction in visceral fat regardless of whether weight loss is induced by diet or exercise (36). The preferential reduction in visceral fat may be explained by previous observations, which suggest that visceral fat is more sensitive than subcutaneous fat to lipolytic stimulation (37) and less resistant to insulin suppression (38). A reduction in visceral fat of the magnitude observed has important clinical implications. Evidence suggests that accumulation of more than 135 cm^2 of visceral fat at the L4–L5 level in men is associated with distinct elevations in risk factors for cardiovascular disease and type 2 diabetes mellitus (39, 40). In both weight loss groups, visceral fat decreased below these values even though the men remained overweight or obese after treatment (body mass index, 28.3 to 29.9 kg/m^2) (**Table 1**). This suggests that the substantial reductions in health risk often associated with modest weight loss (<10%) (41) may be mediated in part by a preferential reduction in visceral fat. This concept is reinforced by our finding that reductions in visceral fat alone were related to improvements in glucose tolerance and insulin sensitivity. This is consistent with the

findings of Goodpaster and colleagues (20), who reported that diet-induced reductions in visceral fat (but not in subcutaneous fat) are related to improvements in insulin resistance in obese men and women. Although visceral fat cannot be routinely measured in clinical practice, the fact that changes in abdominal and visceral fat were related to corresponding reductions in waist circumference demonstrates the usefulness of this simple measure. Our findings support the recommendation that both body mass index and waist circumference be included in clinical examinations that seek to determine the efficacy of weight loss programs (1, 14).

In our study, exercise without weight loss was also associated with significant reductions in both abdominal and visceral fat. This confirms an earlier report in which reductions in visceral and abdominal subcutaneous fat were reported in men and women with type 2 diabetes mellitus after 8 weeks of exercise without weight loss (42). Of note, daily exercise in the exercise without weight loss group prevented the weight gain and negative metabolic consequences usually associated with increased caloric intake (43, 44). Given the consistent increase in the worldwide prevalence of obesity (1), prevention of further weight gain in overweight and obese persons by increasing physical activity is relevant.

It is well recognized that weight loss reverses the insulin resistance that is characteristic of obesity (20–24). Our results extend this observation and suggest that the reductions in insulin resistance after equivalent diet- or exercise-induced weight loss are similar. The increase in glucose metabolism in both groups was due almost entirely (90%) to enhancement of nonoxidative glucose disposal, a finding consistent with previous observations (20, 45). From a therapeutic perspective, it is noted that insulin sensitivity improved by approximately 60% in the weight loss groups, an increase greater than the 25% improvement in insulin sensitivity observed in response to metformin (46) and troglitazone (47). These observations confirm the importance of weight loss in the management of insulin resistance and highlight the benefits of nonpharmacologic strategies for its treatment.

Exercise without weight loss was also associated with a 30% improvement in glucose uptake. However, this improvement did not reach statistical significance when compared with controls. In addition, the improvement from baseline did not remain significant after we controlled for associated reductions in visceral fat, a finding consistent with those of Segal and colleagues (25). These findings do not argue against an important role for exercise in reducing insulin resistance. To the contrary, it is well established that short-term exercise is associated with substantial improvement in insulin sensitivity when measured within 48 hours of the last exercise session (19, 48). Our findings reinforce the notion that, in the absence of weight loss or reduction in visceral fat, the positive effect of exercise on insulin sensitivity attenuates quickly and that adherence to exercise is required to maintain related improvement in insulin sensitivity.

It has been shown that regular exercise reduces the morbidity and mortality associated with cardiovascular disease and diabetes (49–51) and that the intensity of physical activity required to gain the health benefits of exercise is less than originally believed (52). Accordingly, the U.S. Centers for Disease Control and Prevention recommend that regular, moderate-intensity physical activity, such as 30 to 60 minutes of brisk walking, be done on most days of the week (53). Our findings complement this recommendation and suggest that walking briskly for approximately 60 minutes per day without caloric restriction is an effective strategy for reducing obesity and insulin resistance. Combined with the observation that exercise enhances long-term maintenance of weight loss (54), our findings are directly relevant for the nearly one in three Americans who are currently trying to lose weight (8). We also suggest that diet restriction and exercise are effective methods of reducing abdominal obesity, and we confirm the importance of decreasing visceral fat to reduce insulin resistance. Finally, we suggest that exercise without weight loss should be recognized as a useful means of reducing abdominal fat and preventing further increases in obesity.

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