Effects of strength or aerobic training on body composition, resting metabolic rate, and peak oxygen consumption in obese dieting subjects[1–4]

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ABSTRACT Given that resting metabolic rate (RMR) is related largely to the amount of fat-free mass (FFM), the hypothesis was that strength training, which stimulates muscle hypertrophy, would help preserve both FFM and RMR during dieting. In a randomized controlled intervention trial, moderately obese subjects (aged 19–48 y) were assigned to one of three groups: diet plus strength training, diet plus aerobic training, or diet only. Sixty-five subjects (25 men and 40 women) completed the study. They received a formula diet with an energy content of 70% of RMR or 5150 ± 1070 kJ/d ($\bar{x}$ ± SD) during the 8-wk intervention. They were seen weekly for individual nutritional counseling. Subjects in the two exercise groups, designed to be isoenergetic, trained three times per week under supervision. Those in the strength-training group performed progressive weight-resistance exercises for the upper and lower body. Those in the aerobic group performed alternate leg and arm cycling. After 8 wk, the mean amount of weight lost, 9.0 kg, did not differ significantly among groups. The strength-training group, however, lost significantly less FFM ($P < 0.05$) than the aerobic and diet-only groups. The strength-training group also showed significant increases ($P < 0.05$) in anthropometrically measured flexed arm muscle mass and grip strength. Mean RMR declined significantly, without differing among groups. Peak oxygen consumption increased the most for the aerobic group ($P = 0.03$). In conclusion, strength training significantly reduced the loss of FFM during dieting but did not prevent the decline in RMR. *Am J Clin Nutr* 1997;66:557–63.

KEY WORDS Exercise, weight reduction, lean tissue, fat-free mass, obesity, strength training, aerobic training, humans

INTRODUCTION

Exercise is considered an important component of a weight-reduction program in conjunction with dieting (1–3). Dieting alone without exercise results in loss of not only fat but lean tissue or fat-free mass (FFM) as well (4). Resting metabolic rate (RMR) is also lowered during dieting (5), a useful adaptation to periods of famine, but an impediment for dieters trying to lose weight. The decline in RMR is due in part to the loss of FFM (6), given that RMR correlates highly with FFM (7–10). Incorporating exercise into a weight-loss program may help reduce the decline in RMR and FFM, but it is unclear whether to emphasize strength training or aerobic training. Theoretically, strength training should lessen the decline in RMR if it preserves FFM by inducing hypertrophy of skeletal muscle (11, 12), which may comprise > 50% of FFM (13). For example, muscular men who weigh the same as obese men have a significantly higher RMR (14). Two studies incorporating strength training during dieting in obese women found contradictory results: one study (15) found an increase in FFM (RMR was not measured); a second study found no effect of strength training on FFM or RMR (16). The lack of an effect on FFM in the second study may have been due to the relatively low energy intake, 2184 kJ/d (522 kcal/d), overriding the potential effect of strength training.

On the other hand, aerobic exercise, although it often does not significantly increase muscle mass, may have other advantages over strength training. Aerobic training may be more effective in increasing peak oxygen consumption ($\dot{V}O_2$peak), an index of cardiorespiratory capacity because aerobic training by definition stimulates more oxygen consumption than the relatively anaerobic strength training (11). There are conflicting results on whether aerobic exercise can prevent the decline in RMR during dieting, with both positive (3, 17, 18) and negative effects (19–22) being noted. Aerobic training is generally recommended (2) because it results in greater utilization of fat stores and greater energy expenditure in a typical training session than does anaerobic training (11). Studies equating the energy expenditure for the two modes of training are lacking. Energy expenditure, however, can be equalized by increasing the time period for strength training relative to aerobic training. Our study addressed the controversy over which mode of exercise is most advantageous during weight reduction. We

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hypothesized that combined with moderate energy restriction, strength training would best conserve FFM and RMR.

SUBJECTS AND METHODS

Subjects

Overweight men and women aged 19–48 y were recruited by local advertising. The women had to be premenopausal. Their body weight was ≥ 20% above the desired amount (23) and stable within 5% for the past 3 mo. They were also sedentary—not engaged in regular systematic exercise such as aerobic or strength training (not including routine walking) for ≥ 3 mo. Subjects were screened medically with a history, physical exam including electrocardiogram, and blood analysis (general chemistry, thyroid profile, cholesterol, triacylglycerols, and complete blood cell count). Except for obesity, subjects had to be in good health, without hypertension, diabetes, or gastrointestinal, heart, kidney, or liver disease. The subjects could not be abusing drugs or taking medications that affect RMR or body weight. Smokers were excluded because nicotine can raise RMR (24). Pregnant women (determined by urine test) were also excluded. For women, the day of the menstrual cycle when they began and ended the study was noted because fluctuations of 2.7% in RMR can occur during the cycle (25). Because the study period was 8 wk, most women were at about the same point of their cycle as they were originally when RMR and body composition were remeasured.

The subjects were assigned to one of the following groups: *1*) strength training and diet, *2*) aerobic training and diet, or *3*) diet only. The sequence for randomization was first to stratify by sex and then to assign three subjects at a time to a group. There were 13 dropouts (6 in strength training, 3 in aerobics, and 4 in diet only) due to employment relocation, change in vacation dates, illness, and noncompliance. The baseline characteristics of the 65 participants who completed the study are shown in **Table 1**.

Restrictive diet

Subjects received a liquid-formula diet based on Pro-Cal (R-Kane, Pennsauken, NJ) that provided 70% of RMR, 5375 ± 1175 kJ (1286 ± 281 kcal), or ≈50% of daily energy intake for sedentary subjects who require ≈140% of RMR to maintain stable body weight (19). A formula diet was provided to improve control of energy intake. Five packets of powder were taken daily that contained 70 g protein as calcium caseinate, 32.5 g carbohydrate as fructose and corn syrup, 10 g fat as soybean oil, and 2 g fiber. An additional 10 g fiber was provided in three packets of Metamucil (sugar-free effervescent; Proctor & Gamble, Cincinnati) consumed daily. The packets were combined with variable amounts of 1%-fat milk to provide 70% of RMR. Daily potassium exceeded 80 mmol. Other minerals and vitamins provided slightly more than the recommended dietary allowance (RDA; 26) . Subjects obtained ≥ 1.5 g protein/kg ideal body weight and 1 g/kg actual weight, an intake recommended to maximize increases in $\dot{V}O_2$peak and muscle growth (11).

TABLE 1
Baseline characteristics of subjects in the three intervention groups[1]

Group and sex	Age	Weight	Percentage fat[2]
	y	*kg*	*%*
Strength training + diet			
Males (n = 8)	35 ± 3	114.2 ± 16.1	38.1 ± 6.0
Females (n = 12)	36 ± 8	92.1 ± 21.3	43.1 ± 6.8
Both (n = 20)	35 ± 6	101.0 ± 21.9	41.1 ± 6.7
Aerobic training + diet			
Males (n = 9)	37 ± 6	115.9 ± 20.6	37.9 ± 5.1
Females (n = 14)	35 ± 8	83.1 ± 13.4	40.9 ± 6.2
Both (n = 23)	36 ± 7	96.0 ± 23.0	39.8 ± 5.9
Diet only			
Males (n = 8)	33 ± 9	107.5 ± 20.9	36.0 ± 6.2
Females (n = 14)	37 ± 8	92.0 ± 17.7	44.4 ± 4.3
Both (n = 22)	36 ± 8	97.6 ± 19.9	41.3 ± 6.4

[1] $\bar{x}$ ± SD. There were no significant differences among the three groups.
[2] Based on densitometry.

Subjects were seen individually each week for 30 min of nutritional counseling, with an emphasis on behavior modification that included recording daily food intake. Behavior modification has been shown to improve the long-term weight loss associated with a formula diet (27). Subjects were asked to record and maintain their usual sedentary activity pattern during the study except for the exercise prescribed. Body weights were measured weekly. After the 8-wk study period, subjects were asked to return once a week for 4 more weeks for a supervised transition to solid food. Subjects who needed to lose more weight after this transition were encouraged to follow a 5016-kJ/d (1200-kcal/d) solid diet. The study protocol was approved by St Luke's–Roosevelt Hospital's Institutional Review Board. All subjects gave their informed consent.

Measurement procedures

Subjects underwent several measurements before starting the diet and after 8 wk while still dieting. Measurements at the end were conducted ≥ 48 h after a previous exercise session. Subjects were requested to fast for 13 h beforehand and void bowels and bladder in the morning. The technicians performing the procedures were blind to the subjects' group assignments. The procedures required took ≈3 h and were performed in the following order: RMR, blood tests, psychologic ratings, body composition, and $\dot{V}O_2$peak.

Resting metabolic rate

RMR was determined after the subject rested comfortably for 45 min, in a supine position, while trying not to move or fall asleep. A face mask was used for 15 min; the last 10 min of the measurements were used for analysis. The amount of oxygen consumed and carbon dioxide produced were recorded by using open-circuit spirometry with a metabolic cart (Sensormedics-Horizon, Yorba Linda, CA) after calibration with 100% nitrogen, room air, and a mixture of 4% CO_2 and 16% O_2. The energy expended was calculated by indirect calorimetry (Weir formula). Reproducibility for this measurement in our laboratory has a CV of 3.8% (28).

Serum measurement

Because thyroid hormones may influence RMR (29, 30), blood samples for hormone measurements were drawn after RMR was measured, and serum was separated to measure total and free triiodothyronine (T_3) and thyroxine (T_4) by radioimmunoassay kits (Diagnostic Products, Los Angeles).

Body composition

After measurement of RMR, body density was determined by underwater weighing (Precision Biomedical Systems, University Park, PA). The subject was first weighed in air on an electronic scale (Weightronix; Scale Electronics Development, New York) to the nearest 0.05 kg. Then, wearing a weight belt, the subject was weighed underwater 10 times while exhaling maximally. Residual lung volume was measured by oxygen dilution (31). Body density was calculated (32), and the percentage fat and FFM were derived (33). The CV for hydrodensitometry in our laboratory is < 1% (34). To provide additional assessments of FFM, total-body electrical conductivity (TOBEC) was measured with the subject inside a large magnetic coil (EM Scan, Auburn, IL) (35); the CV in our laboratory is < 2% (36). Bioelectrical impedance analysis (BIA; Valhalla, San Diego) was also performed with electrodes attached to the arm and leg (36), with a CV of < 1% (36).

Triceps and biceps skinfold thicknesses were measured at the midupper arm with Lange calipers (Cambridge Scientific, Cambridge, MD), and the circumference of the midupper arm was obtained with a tape measure. From the change in triceps skinfold thickness and upper arm circumference, the change in the circumference of the upper arm muscle was estimated (37). The biceps as well as the triceps skinfold thickness was included in the calculation. Additionally, these arm measurements were repeated with the arm extended to flex the triceps. Grip strength (mean of two trials) was assessed with a dynamometer (Country Tech, Gay Mills, WI).

Peak oxygen consumption

$\dot{V}O_2$peak was determined with a modified Balke protocol by using a treadmill (Quinton, Seattle) with speed maintained at 3.3 miles/h (5.3 km/h). Treadmill walking was chosen to differentiate from the training received by either exercise group. Subjects first walked for 2 min at 0 grade, then the treadmill was elevated by 1°/min until the subject could not continue. At each workload, the subject rated perceived exertion (RPE) on a Borg scale from 6 to 20. Expired gas was continuously analyzed for oxygen and carbon dioxide with a metabolic cart (Sensormedics-Horizon). $\dot{V}O_2$peak was considered the largest value for oxygen consumption per minute. The subject wore electrocardiogram leads for continuous monitoring of heart rate.

Ratings of mood

To assess mood state, subjects filled out the Beck Depression Inventory (38) (two questions were excluded on changes in eating and body weight that would be influenced by the dieting component of the study). A high score indicates more depressed mood.

Exercise training

Subjects assigned to either strength- or aerobic-training groups exercised under supervision three times per week on Monday, Wednesday, and Friday. Missed exercise sessions, ≈5% of the total, were made up the same week. The strength and aerobic exercise sessions were designed, according to published guidelines, to be isoenergetic with a mean net energy expenditure of 627 kJ (150 kcal) (39, 40). The aerobic sessions lasted ≈30 min and the strength sessions ≈60 min. Self reports were collected to confirm that subjects remained sedentary outside of supervised sessions.

Strength training

Subjects performed progressive-resistance weight training with Nautilus equipment (Independence, VA). Eight stations were used to exercise upper- and lower-body large muscle groups: leg extension (quadriceps), leg curl (hamstring), chest press (pectoralis major), super pullover (latissimus dorsi), lateral raise (medial shoulder), arm flexion (biceps), arm extension (triceps), and leg press (buttocks, hip, and quadriceps). At each station, subjects performed three consecutive sets of repetitions, 30 s apart. The first two sets consisted of six repetitions each, followed by a third set of as many repetitions as possible. If the subjects performed eight or more repetitions on the third set, the resistance was increased at the next session. Subjects raised and lowered the weights slowly in a continuous motion to a count of 5 s in each direction. A warm-up of 5 min on a cycle ergometer, set at 0 resistance, preceded the strength training.

Aerobic training

Subjects exercised first on a stationary leg cycle ergometer (Monark; Varberg, Sweden) at a starting speed of 60 rpm, at low resistance, for 8 min. This was followed by 8 min on an upper-body ergometer (Monark), with the arm cycling direction reversed each minute. Subjects concluded with leg cycling for 8 more min. To maintain heart rate in the aerobic range as subjects progressed through the study, the rpms were increased without raising the resistance. A warm-up and cool down of 2.5 min on the leg cycle at 0 resistance preceded and followed the session. The aerobic training was designed to exercise the upper and lower body, like the strength training, with the person's body weight supported. Both upper and lower body work were first set to be 55% of the subject's initial $\dot{V}O_2$peak. Heart rate was monitored continuously with a heart rate monitor (Polar, Port Washington, NY) and kept just above 70% of maximal rate.

Statistical analysis

The data were subjected to two-way analysis of variance (ANOVA) with repeated measures on one of the factors, pre- and postintervention, to assess changes between groups and determine interactions (PC ANOVA; Human Systems Dynamics, Northridge, CA). Post hoc tests were performed with the Duncan multiple-range test. The data are given for the sexes combined because there were no significant interactions between the sexes for the changes between groups. For the mood scores, the initial mood score was entered as a covariate for the change in scores. Differences with $P < 0.05$ (two-tailed) were considered significant.

RESULTS

Mean weight loss in the three intervention groups did not differ significantly: 9.0 kg or 9.2% of initial weight (**Table 2**). The combined groups lost a significant amount of FFM, based on densitometry, but the strength-training group lost the least ($P < 0.05$, ANOVA) (**Figure 1** and Table 2). As a component of the weight loss, the FFM lost represented 8% for the

TABLE 2
Before and after measurements for the three intervention groups[1]

	Interventions			Comparison of changes: *P*	
	Strength + diet	Aerobic + diet	Diet only	Combined groups	Between groups
Weight (kg)					
Before	100.9 ± 21.9	96.0 ± 23.0	97.6 ± 19.9		
After	93.2 ± 19.9	86.4 ± 19.8	88.1 ± 19.2		
Change	−7.8 ± 3.8	−9.6 ± 4.5	−9.5 ± 3.1	< 0.00005	NS
Fat-free mass (kg)[2]					
Before	58.9 ± 11.5	57.5 ± 12.8	57.0 ± 12.2		
After	57.7 ± 11.1	55.2 ± 11.7	54.3 ± 11.6		
Change	−1.1 ± 2.3	−2.3 ± 2.4	−2.7 ± 2.1	< 0.00005	0.04 (S > A,D)[3]
Fat (kg)[2]					
Before	42.1 ± 14.0	38.4 ± 12.5	40.6 ± 11.2		
After	35.4 ± 12.4	31.2 ± 11.0	33.8 ± 11.5		
Change	−6.7 ± 2.8	−7.2 ± 3.0	−6.8 ± 2.6	< 0.00005	NS
Arm muscle, relaxed (cm)[4]					
Before	28.5 ± 5.2	27.6 ± 4.3	27.5 ± 4.1		
After	29.1 ± 4.3	27.2 ± 4.2	26.9 ± 3.9		
Change	0.6 ± 2.6	−0.4 ± 2.4	−0.6 ± 2.7	NS	NS
Arm muscle, flexed (cm)[5]					
Before	30.6 ± 5.7	29.8 ± 4.6	30.2 ± 5.4		
After	31.7 ± 6.8	29.5 ± 4.9	28.7 ± 4.9		
Change	1.1 ± 4.1	−0.3 ± 2.3	−1.6 ± 3.1	NS	0.04 (S > A,D)
Grip strength (u)					
Before	36.6 ± 11.9	34.7 ± 11.6	34.5 ± 12.0		
After	39.9 ± 12.6	35.3 ± 11.3	34.2 ± 9.9		
Change	3.3 ± 4.5	0.6 ± 5.3	−0.3 ± 3.5	NS	0.03 (S > A,D)
RMR (kJ/d)					
Before	7679 ± 1679	7290 ± 1447	7214 ± 1496		
After	7147 ± 1618	6667 ± 1561	6845 ± 1420		
Change	−532 ± 1054	−623 ± 857	−369 ± 1055	0.0003	NS
$\dot{V}O_2$peak (mL/min)					
Before	2170 ± 646	2074 ± 702	2231 ± 616		
After	2152 ± 730	2280 ± 706	1963 ± 502		
Change	−18 ± 313	206 ± 574	−268 ± 337	NS	0.01 (A > D)
Total thyroxine (μg/L)					
Before	79.7 ± 14	82.4 ± 15	77.7 ± 16		
After	79.1 ± 16	78.6 ± 18	70.6 ± 15		
Change	−0.6 ± 7.0	−3.9 ± 9.0	−7.1 ± 9.0	0.0007	0.04 (S > D)
Depression score[6]					
Before	8.3 ± 6.2	8.1 ± 4.5	9.7 ± 6.8		
After	4.7 ± 4.7	3.6 ± 3.6	7.2 ± 5.4		
Change	−3.6 ± 4.9	−4.5 ± 5.1	−2.5 ± 6.3	< 0.00005	0.02 (S,A < D)

[1] $\bar{x}$ ± SD.
[2] By densitometry.
[3] Post hoc comparison, for example, the change for the strength-training group (S) was significantly more positive than the changes for both the aerobic (A) and diet-only (D) groups.
[4] Circumference of midarm muscle in relaxed state.
[5] Circumference of midarm muscle with triceps flexed.
[6] Assessed by Beck Depression Inventory (38).

strength-training group compared with 20% for the aerobic-training group and 28% for the diet-only group. Body fat was significantly reduced in all groups, without differing between them (Table 2). Significantly smaller losses of lean tissue in the strength-training group were confirmed with both TOBEC (P = 0.007, ANOVA) and BIA (P = 0.04, ANOVA).

The circumference of arm muscle in the relaxed state showed a nonsignificant trend toward an increase only in the strength group, and in the flexed state increased significantly in the strength group (Table 2). The correlation between the changes in flexed arm muscle circumference and FFM was significant (r = 0.34, P < 0.02). Grip strength (P = 0.03, ANOVA) increased the most in the strength-training group.

Mean RMR declined significantly without differing between groups (Table 2). Adjusting RMR for either body weight or FFM eliminated the significant decline over the intervention period, but the difference between changes among groups was still not significant. $\dot{V}O_2$peak increased the most (P = 0.01,

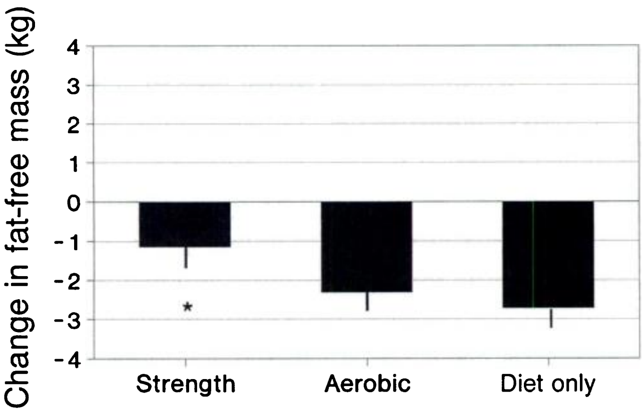

FIGURE 1. Mean (± SEM) change in fat-free mass in the three groups. The strength-training group lost significantly less fat-free mass, based on densitometry, than the other groups (*P = 0.04, ANOVA).

ANOVA) after aerobic training. Results were similar when $\dot{V}O_2$ was adjusted for body weight or FFM.

Among the thyroid hormones, free and total T_3 declined significantly overall but did not differ between groups. Free T_4 did not change significantly, but total T_4 declined significantly overall and declined significantly less for the strength-training group (Table 2).

Although none of the subjects were clinically depressed, depression scores declined significantly (showing enhanced mood) for all groups and declined significantly more in the exercise groups than in the diet only group (Table 2).

DISCUSSION

The results show that strength training combined with a moderate diet significantly diminished the loss of FFM compared with aerobic training or no exercise. The relative preservation of FFM in the strength-training group appeared to be due to muscular hypertrophy, given the increase in arm muscle mass and consistent with the increase in grip strength. Thus, lean tissue can be preserved during negative energy balance. In animals, muscle mass was shown to increase despite weight loss when rats performed progressive weight-resistance exercises (41). The reduced loss of lean tissue from ≈25% of the weight change (the aerobic and diet-only groups), typical for moderately restrictive diets (42), to < 10% in the strength-training group is impressive. The preservation of lean tissue and muscle mass is consistent with results from a recent study in dieting, obese subjects doing strength exercises as assessed with magnetic resonance imaging (43). In another study in obese humans, the failure to find preservation of FFM (16) after strength training may have been due both to the use of a very-low-energy diet with greater losses of FFM (44) and the relatively low protein intake of 50 g/d (11). In two studies that had combined sessions of aerobic and strength training in conjunction with a moderately restrictive diet [> 4181 kJ(1000 kcal)/d], the loss of FFM was also lessened relative to a no-exercise group in both premenopausal (45) and postmenopausal (46) obese women.

Our results also show that the preservation of FFM in the strength-training group did not translate into a conservation of RMR. This negative result is consistent with the finding (16) that strength training added to a very-low-energy diet also did not influence RMR, although in that study FFM was not preserved, as noted above. In the two other studies, with sessions combining both strength and aerobic exercises compared with no exercise during moderate dieting, no differential effect on RMR was observed (45, 46).

Thyroid hormones play a major role in regulating RMR (29, 30), and both T_3 and RMR generally decline more with a very-low-energy than with a moderate diet (30). In our study, the thyroid hormones, except for free T_4, declined postintervention in all groups. Although total T_4 declined significantly less for the strength-training group, this pattern apparently did not influence RMR, which decreased without differing between groups.

Weight loss after dieting has been shown to enhance mood (47). Exercise itself has also been noted to improve mood (48). Although clinical depression was absent in our study, mood improved for all three groups. Subjects in both exercise groups, although they did not lose more weight than those in the diet-only group, had a greater improvement in mood, illustrating a benefit for exercise regardless of mode when combined with dieting.

At the time of the postintervention measurements all the groups were still dieting, which acted as a constant. It is unlikely that the dieting obscured the potential effect of exercise on RMR because the moderate diet did not itself reduce RMR beyond that expected from a smaller body weight and FFM, as can sometimes result from a very-low-energy diet (49). It is also unlikely that a longer intervention period would have produced the effect because even 6 mo of combined aerobic and strength training failed to preserve RMR (45). Our results are consistent with the failure of a 12-wk resistance-training program to increase the RMR of young nondieting men (50). It is possible that the amount of lean tissue preserved (1.6 kg), when it is mostly muscle mass, is too small to induce a change in RMR. Resting muscle mass, although more metabolically active than fat mass, is still less active than organ tissue, which comprises another major part of the FFM (51). Thus, despite popular opinion and claims made by manufacturers of weight-resistance equipment, no evidence could be found for conservation of RMR when combining diet and strength training.

In our study aerobic training also failed to prevent the decline in RMR, which is consistent with other negative findings in the literature (19–22). However, the aerobic training significantly increased $\dot{V}O_2$peak, an index of cardiorespiratory capacity and exercise fitness, which is a known benefit conferred by aerobic exercise (11). The enhancement of $\dot{V}O_2$peak, however, may only occur with moderate energy restriction because aerobic exercise combined with a very-low-energy diet did not significantly raise $\dot{V}O_2$peak (16, 52).

In conclusion, strength training significantly preserved lean tissue relative to either aerobic exercise or no exercise in dieting obese subjects without conserving RMR. However, the protective effect for lean tissue was itself a major advantage of strength training.

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REFERENCES

1. Bjorntorp P. Physiological and clinical aspects of exercise in obese subjects. Exerc Sports Sci Rev 1983;11:159–80.
2. Geliebter A. Exercise and obesity. In: Wolman B, ed. Psychological aspects of obesity: handbook. New York: Van Nostrand Reinhold Company, 1982:291–310.
3. Pavlou KN, Krey S, Steffee WP. Exercise as an adjunct to weight loss and maintenance in moderately obese subjects. Am J Clin Nutr 1989;49:1115–23.
4. Garrow JS. New possibilities for the treatment of obesity. Bibl Nutr Dieta 1986;39:60–7.
5. Bray GA. Effect of caloric restriction on energy expenditure in obese patients. Lancet 1969;2:397–8.
6. Welle SL, Amatruda JM, Forbes GB, Lockwood DH. Resting metabolic rates of obese women after rapid weight loss. J Clin Endocrinol Metab 1984;59:41–4.
7. Bernstein RS, Thornton JC, Yang MU, et al. Prediction of the resting metabolic rate in obese patients. Am J Clin Nutr 1983;37:595–602.
8. Ravussin E, Burnand B, Shultz Y, et al. Twenty-four hour expenditure and resting metabolic rate in obese, moderately obese, and control subjects. Am J Clin Nutr 1982;35:366–73.
9. Ravussin E, Burnand B, Shultz Y, Jéquier E. Energy expenditure before and during energy restriction in obese patients. Am J Clin Nutr 1985;41:753–9.
10. Segal KR, Gutin B, Albu J, et al. Thermic effects of food in lean and obese men of similar lean body weight. Am J Physiol 1987;252:E110–7.
11. Walberg JL. Aerobic exercise and resistance weight training during weight reduction. Sports Med 1989;47:343–56.
12. Fleck SJ, Kraemer WJ. Resistance training: physiological responses and adaptations. Physician Sports Med 1988;16:108–24.
13. Martin AD, Spenst LF, Drinkwater DT, Clarys JP. Anthropometric estimation of muscle mass in men. Med Sci Sports Exerc 1990;22:729–33.
14. Segal KR, Gutin B, Nyman AM, et al. Thermic effect of food at rest, during exercise, and after exercise in lean and obese men of similar body weight. J Clin Invest 1985;76:1107–12.
15. Ballor DL, Katch VL, Becque MD, Marks CR. Resistance weight training during caloric restriction enhances lean body weight maintenance. Am J Clin Nutr 1988;47:19–25.
16. Donnelly JE, Pronk NP, Jacobsen DJ, Pronk SJ, Jakicic JM. Effects of a very-low-calorie diet and physical training regimens on body composition and resting metabolic rate. Am J Clin Nutr 1991;54:56–61.
17. Mole PA, Stern JS, Schultz CL, et al. Exercise reverses depressed metabolic rate produced by severe caloric restriction. Med Sci Sports Exerc 1989;21:29–33.
18. Tremblay A, Fontaine E, Poehlman ET, et al. The effect of exercise training on resting metabolic rate in lean and moderately obese individuals. Int J Obes 1986;10:511–7.
19. Hill JO, Sparling PB, Shields TW, Heller PA. Effects of exercise and food restriction on body composition and metabolic rate in obese women. Am J Clin Nutr 1987;46:622–30.
20. Woo R, Garrow JS, Pi-Sunyer FX. Voluntary food intake during prolonged exercise in obese women. Am J Clin Nutr 1982;36:478–84.
21. Heymsfield SB, Casper K, Hearn J, Guy D. Rate of weight loss during underfeeding: relation to level of physical activity. Metabolism 1989;38:215–23.
22. Phinney S, LaGrange B, O'Connell M, Danforth E. Effects of aerobic exercise on energy expenditure and nitrogen balance during very low calorie dieting. Metabolism 1988;37:758–65.
23. Metropolitan Life Insurance Company. 1983 Height and weight tables. Stat Bull Metropol Insur Co 1984;64:2–9.
24. Perkins KA, Epstein LH, Stiller R, Marks BL, Jacob RG. Acute effects of nicotine on resting metabolic rate in cigarette smokers. Am J Clin Nutr 1989;50:545–50.
25. Bisdee JT, James WPT, Shaw MA. Changes in energy expenditure during the menstrual cycle. J Nutr 1989;61:187–99.
26. National Research Council. Recommended dietary allowances. 10th ed. Washington, DC: National Academy Press, 1989.
27. Wadden TA, Stunkard AJ. Controlled trial of very-low-calorie diet, and behavior therapy and their combination in the treatment of obesity. J Consult Clin Psychol 1986;54:482–8.
28. Heshka S, Yang M-U, Wang J, Burt P, FX Pi-Sunyer. Weight loss and change in resting metabolic rate. Am J Clin Nutr 1990;52:981–6.
29. Danforth E Jr. The role of thyroid hormones and insulin in the regulation of energy metabolism. Am J Clin Nutr 1983;38:1006–17.
30. Danforth E, Burger A. The role of thyroid hormones in the control of energy. Clin Endocrinol Metab 1984;13:581–5.
31. Wilmore J. A simplified method for determination of residual lung volumes. J Appl Physiol 1969;27:96–100.
32. Goldman RF, Buskirk ER. Body volume measurement by underwater weighing: description of a method. In: Brozek J, Henschel A, eds. Techniques for measuring body composition. Washington, DC: National Academy of Sciences, 1961:78–9.
33. Siri WE. Body composition from fluid spaces and density: analysis of methods. In: Brozek J, Henschel A, eds. Techniques for measuring body composition. Washington, DC: National Academy of Sciences, 1961:223–44.
34. Heymsfield SB, Lichtman S, Baumgartner RN, et al. Body composition of humans: comparisons of two improved four-compartment models that differ in expense, technical complexity, and radiation exposure. Am J Clin Nutr 1990;52:52–8.
35. Presta E, Segal KR, Gutin B, Harrison GG, Van Itallie TB. Comparison in man of total body electrical conductivity and lean body mass derived from body density: validation of a new body composition method. Metabolism 1983;32:524–7.
36. Segal KR, Gutin B, Presta E, Wang J, Van Itallie TB. Estimation of human body composition by electrical impedance methods: a comparative study. J Appl Physiol 1985;58:1565–71.
37. Gurney JM, Jelliffe DB. Arm anthropometry in nutritional assessment: nomogram for rapid calculation of muscle circumference and cross-sectional muscle and fat areas. Am J Clin Nutr 1973;26:912–5.
38. Beck AT, Ward CH, Mendelson M, Mock J, Erbaugh J. An inventory for measuring depression. Arch Gen Psychiatr 1961;4:561–9.
39. American College of Sports Medicine. Guidelines for exercise testing and prescription. Philadelphia: Lea and Febiger, 1986.
40. Reilly T. The energy cost of and mechanical efficiency of circuit weight training. J Hum Mov Stud 1983;9:39–45.
41. Goldberg AL, Etlinger JD, Goldspink DF, Jablecki C. Mechanism of work-induced hypertrophy in skeletal muscle. Med Sci Sports Exerc 1975;7:248–61.
42. Garrow JS. Treat obesity seriously: a clinical manual. London: Churchill Livingstone, 1981.
43. Ross R, Pedwell H, Rissanen J. Response of total and regional lean tissue and skeletal muscle to a program of energy restriction and resistance exercise. Int J Obes 1995;19:781–7.
44. Fisler JS, Drenick EJ, Blumfield DE, Swenseid ME. Nitrogen economy during very low calorie reducing diets: quality and quantity of dietary protein. Am J Clin Nutr 1982;35:471–86.
45. Sweeney ME, Hill JO, Heller PA, Baney R, DiGirolamo M. Severe vs moderate energy restriction with and without exercise in the treatment of obesity: efficiency of weight loss. Am J Clin Nutr 1993;57:127–34.
46. Svendsen OI, Hassager C, Christiansen C. Effect of an energy-restrictive diet, with or without exercise, on lean tissue mass, resting metabolic rate, cardiovascular risk factors, and bone in overweight postmenopausal women. Am J Med 1993;95:131–9.
47. Foster GD, Wadden TA, Peterson FJ, Letizia KA, Bartlett SJ, Conill

AM. A controlled comparison of three very-low-calorie diets: effects on weight, body composition, and symptoms. Am J Clin Nutr 1992;55:811–7.
48. Byrne A, Byrne DG. The effect of exercise on depression, anxiety and other mood states: a review. J Psychosom Res 1993;37:565–74.
49. Leibel RL, Rosenbaum M, Hirsch J. Changes in energy expenditure resulting from altered body weight. N Engl J Med 1995;332:621–6.
50. Broeder CE, Burrhus KA, Svanevik LS, Wilmore JH. The effects of either high-intensity resistance exercise or endurance training on resting metabolic rate. Am J Clin Nutr 1992;55:802–10.
51. Grande F. Energy expenditure of organs and tissues. In: Kinney JM, Lense E, eds. Assessment of energy metabolism in health and disease. Columbus, OH: Ross Laboratories; 1980:88–92.
52. Whatley JE, Gillespie WJ, Honig J, Walsh MJ, Blackburn AL, Blackburn GL. Does the amount of endurance exercise in combination with weight training and a very-low-energy diet affect resting metabolic rate and body composition? Am J Clin Nutr 1994;59:1088–92.