

Effects of Resistance Training and Endurance Training on Insulin Sensitivity in Nonobese, Young Women: A Controlled Randomized Trial*

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ABSTRACT

We examined the effects of a 6-month randomized program of endurance training ($n = 14$), resistance training ($n = 17$), or control conditions ($n = 20$) on insulin sensitivity in nonobese, younger women (18–35 yr). To examine the possible mechanism(s) related to alterations in insulin sensitivity, we measured body composition, regional adiposity, and skeletal muscle characteristics with computed tomography. We observed no changes in total body fat, sc abdominal adipose tissue, or visceral adipose tissue with endurance or resistance training. Insulin sensitivity, however, increased with endurance training (pre, 421 ± 107 ; post, 490 ± 133 mg/min; $P < 0.05$) and resistance training (pre, 382 ± 87 ; post, 417 ± 89 mg/min; $P = 0.06$). When the glucose disposal rate was expressed per kg fat-free mass (FFM), the improved insulin sensitivity persisted in endurance-trained (pre, 10.5 ± 2.7 ; post, 12.1 ± 3.3 mg/min·kg FFM; $P < 0.05$), but not in resistance-trained (pre, 9.7 ± 1.9 ; post, 10.2 ± 1.8 mg/min·kg FFM;

$P = \text{NS}$) women. Muscle attenuation ratios increased ($P < 0.05$) in both endurance- and resistance-trained individuals, but this was not related to changes in insulin sensitivity. Moreover, the change in insulin sensitivity was not related to the increased maximum aerobic capacity in endurance-trained women ($r = 0.24$; $P = \text{NS}$). We suggest that both endurance and resistance training improve glucose disposal, although by different mechanisms, in young women. An increase in the amount of FFM from resistance training contributes to increased glucose disposal probably from a mass effect, without altering the intrinsic capacity of the muscle to respond to insulin. On the other hand, endurance training enhances glucose disposal independent of changes in FFM or maximum aerobic capacity, suggestive of an intrinsic change in the muscle to metabolize glucose. We conclude that enhanced glucose uptake after physical training in young women occurs with and without changes in FFM and body composition. (*J Clin Endocrinol Metab* 85: 2463–2468, 2000)

AEROBIC EXERCISE training can improve insulin sensitivity (1–4), whereas the role of resistance training to improve the metabolic profile has received less attention. As isometric contractions produce insulin-like effects on glucose uptake in isolated skeletal muscle (5), and skeletal muscle is the primary site of glucose disposal at euglycemia, it is reasonable to hypothesize that increasing skeletal muscle mass may be an effective intervention to improve insulin sensitivity. There is little information on the effects of resistance training on glucose disposal using clamp methodology in a controlled, randomized design. Moreover, investigators have tended to rely on nonrandomized studies and the use of oral glucose tolerance tests to estimate insulin sensitivity (6–9).

To our knowledge, no study has directly compared the effects of endurance *vs.* resistance training on insulin sensitivity using clamp methodology in women. This area of investigation is important because recent data show that despite having a normal body weight, a subset of young

women show a cluster of metabolic abnormalities that would predispose them to type 2 diabetes and related comorbidities if left untreated (10). The incidence of obesity and type 2 diabetes is increasing among women (11), which places them at high risk for the development of insulin resistance and associated comorbidities (12, 13). Clearly, preventive public health measures to prevent deterioration of the metabolic profile of younger women are needed before disease processes become established.

To address this topic, we directly compared the effects of resistance training and aerobic training on insulin sensitivity using a controlled randomized trial. Moreover, to examine potential mechanism(s) regulating training effects on insulin sensitivity, we measured changes in body composition, visceral fat, and skeletal muscle density using radiological imaging techniques, as changes in these variables are thought to be related to altered glucose disposal (14–18). We hypothesized that endurance training would increase insulin sensitivity to a greater degree than resistance training in young women, and these changes would be associated with greater reductions in intraabdominal fat and increased skeletal muscle density.

Subjects and Methods

For inclusion in the study, subjects were required to be premenopausal and between 18–35 yr of age with a body mass index less than 26. In addition, subjects had to be weight stable (± 2 kg) and to have had no regular participation in exercise for 6 months before the study. Exclusion criteria included a history or evidence on physical examination

Received October 14, 1999. Revision received December 29, 1999. Rerevision received April 10, 2000. Accepted April 10, 2000.

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* This work was supported by a grant from the Department of Defense (DE-950226, to E.T.P.), a postdoctoral fellowship from the American Heart Association, Maine/New Hampshire/Vermont affiliate (to R.V.D.), a grant from the Medical Research Council of Canada (to M.B.), and General Clinical Research Center Grant RR-109.

or testing of the following: 1) diabetes, 2) orthopedic limitations or history of pathological fractures, 3) hypertension ($>160/90$ mm Hg), 4) use of prescription or over the counter medications that could affect glucose metabolism (including insulin and oral hypoglycemic agents), 5) smoking, or 6) alcohol consumption of more than 15 g alcohol/day. An oral glucose tolerance test was performed in all volunteers to determine glucose tolerance according to the criteria of the National Diabetes Group (12) to exclude diabetics. This study was approved by the committee for human research at the University of Vermont, and each participant gave written informed consent before the beginning of the study.

Overview of experimental protocol

Subjects were recruited from local newspaper advertisements in the Burlington, VT, and the University of Vermont community. After determination of eligibility by telephone, volunteers were scheduled for the first screening visit. On the screening visit, an oral glucose tolerance test, medical history, physical examination, maximum oxygen consumption test, and complete blood chemistry and profile were performed. Two weeks later, participants were scheduled for an overnight visit to the General Clinical Research Center at the University of Vermont. For 3 days before the overnight visit, participants were provided with standardized diets prepared by the metabolic kitchen at the General Clinical Research Center containing 55% carbohydrate, 25% fat, and 20% protein. During the afternoon of admission, we conducted body composition and body fat distribution measurements using dual energy x-ray absorptiometry and computed tomography. The following morning, the hyperinsulinemic-euglycemic clamp was performed. After successful completion of this testing sequence, volunteers were randomly assigned to the endurance exercise, resistance exercise, or control group. An identical posttesting sequence was performed, and these tests were performed 4 ± 1 days after the last exercise session.

Recruiting and screening

Based on our advertisements, 321 women were interviewed by telephone. Of these 321 women, 105 women consented to participate in screening procedures. Of these 105 women, 78 were deemed eligible and consented to participate in pretraining testing procedures. Of these 78 women, 74 were Caucasian, 2 were of Asian descent, and 2 were of Hispanic origin. They were randomized to either endurance training, resistance training, or control conditions after completion of physiological testing.

Exercise training programs

All workouts were preceded by a 10-min warm-up, which consisted of stretching of the major muscle groups and slow walking around the track. All women were taught to monitor their heart rates (HR). HRs were verified with a Polar Heart Rate monitor (Polar Electro, Port Washington, NY). The endurance-training program consisted of two parts: 1) weeks 1–16 were an endurance base-training phase; and 2) weeks 17–28 were an interval-recovery phase. Women trained on 3 nonconsecutive days/week for 6 months (28 weeks) under the supervision of a personal trainer.

The endurance base training consisted of four phases. The first phase (first 4 weeks) began with an exercise prescription of 25 min of slow jogging. Thereafter, the aerobic training program of each 4-week phase increased by 5 min. By the fourth phase (*i.e.* 16 weeks), women were jogging for approximately 40 min. Within the phases, the exercise intensity was increased by 5% of maximum HR (HR max) each week, so that by the end of the fourth week of the fourth phase, the training was 40 min at 90% of HR max.

The second part (weeks 16–28) of the endurance training program used interval training sessions. Women followed a detailed program of specific workouts aimed at increasing exercise duration and intensity. The interval sessions consisted of 45 min of 80% HR max training on Monday, four 5-min periods at 95% HR maximum with 3-min rests on Wednesday, and 45 min at 75–80% of HR max on Friday. By the final week of training, women successfully completed 60-min sessions at 85% of HR max.

Women randomized to resistance training exercised on 3 noncon-

secutive days during the week (*e.g.* Monday, Wednesday, and Friday) under the supervision of a personal trainer. Because of the need for test specificity, one repetition maximum (1-RM) evaluation of certain exercises used in the training program provided the most direct evaluation of the training gains made over the 6-month period. The 1-RM is defined as the maximum amount of resistance that can be moved through the full range of motion of an exercise for no more than one repetition. To determine the 1-RM, each subject initially performed three to five repetitions with the lightest weight possible to assure that proper technique was used. The trainer then selected a weight and asked the subject to perform the lift. After 3–4 min of rest, the next heaviest weight was selected, and the attempt was repeated until the subject could not complete the full lift. The same number of trials, time between trials, and order of exercises were used before and after training for the 1-RM test. Tests were administered before the start of the training program, midway through the program, and after the exercise program. The following exercises were evaluated for 1-RMs: leg press, bench press, military press, and seated rows.

Training was approximately 80% of 1-RM. Each training session included a warm-up of low intensity cycling for 5 min, followed by 10 min of static stretching of all of the major muscle groups used in training. Each exercise session was individually monitored for optimal progression by two trainers. The resistance program consisted of the following exercises: 1) leg press, 2) bench press, 3) leg extensions, 4) shoulder press, 5) sit-ups, 6) seated rows, 7) tricep extensions, 8) arm curls, and 9) leg curls. The exercises provided a total body resistance training program for all of the major muscle groups of the body. The volunteer was given a target load range and attempted to keep each set ($n = 3$) within the target range by adjusting the load to allow the prescribed number ($n = 10$) of repetitions. Resting periods were 1–1.5 min between sets.

During the conduct of the training programs, 28 women dropped out of the study, yielding a dropout rate of 36%. The reasons for dropouts included 1) noncompliance with training ($n = 18$), 2) relocation ($n = 3$), 3) injury related to endurance training ($n = 3$), 4) refused posttesting ($n = 2$), 5) health problems not related to training ($n = 1$), and 6) pregnancy ($n = 1$). Thus, 51 women (17 resistance, 14 endurance, and 20 control) satisfactorily completed all pre- and posttesting procedures and the 6-month training program. The exercising women successfully completed 90% of all exercise-training sessions. Oral contraceptive use was 47% in resistance-trained women (8 of 17), 50% in endurance-trained women (7 of 14), and 50% in controls (10 of 20).

Body composition and adipose tissue distribution

Fat mass and fat-free mass (FFM) were measured by dual energy x-ray absorptiometry using a DPX-L densitometer (Lunar Corp., Madison, WI) as previously described (19). All scans were analyzed using the Lunar Corp. version 1.3 DPX-L extended analysis program for body composition. The test-retest coefficient of variation for this measurement was 1.2% for fat mass and 2% for FFM, respectively.

Visceral and sc adipose tissue areas were measured by computed tomography with a GE High Speed Advantage CT scanner (General Electric Medical Systems, Milwaukee, WI) as previously described (19). Subjects were examined in the supine position with both arms stretched above the head. The scan was performed at the L4–L5 vertebrae level using a scout image of the body to establish the precise scanning position. Visceral adipose tissue area was quantified by delineating the intraabdominal cavity at the internal most aspect of the abdominal and oblique muscle walls surrounding the cavity and the posterior aspect of the vertebral body with the computer interface of the scanner. Adipose tissue was highlighted and computed using an attenuation range from -190 to -30 Hounsfield units (HU) (20). The sc adipose tissue area was quantified by highlighting adipose tissue located between the skin and the external-most aspect of the abdominal muscle wall. The same individual analyzed all scans, and the intraclass correlation for repeated analysis of 10 scans was 0.99 in 10 women. Computed tomography was also used to measure cross-sectional areas of mid thigh muscle and adipose tissue and to characterize muscle attenuation. With the subject supine, a 5-mm cross-sectional scan of both legs was obtained, located at the midpoint between the anterior iliac crest and the top of the patella. In image analysis, areas of adipose tissue and skeletal muscle were measured by selecting the following region of interest defined by at-

tenuation values: -190 to -30 HU for adipose tissue and 0-100 HU for muscle.

Cardiorespiratory fitness

Maximum aerobic capacity ($\text{VO}_{2\text{max}}$) was determined from an incremental exercise test on a treadmill to volitional exhaustion, as previously described (21, 22). After an initial 3-min warm-up, the speed was held constant, and the grade was increased by 2.5% every 2 min. The criteria for achieving a $\text{VO}_{2\text{max}}$ were 1) a respiratory exchange ratio greater than 1.0, 2) a HR at or above the age-predicted maximum, and 3) no further increase in oxygen consumption with an increasing workload. At least two of these criteria were met by all volunteers. Test-retest conditions for nine individuals (on two occasions, tested 1 week apart) yielded an intraclass correlation of 0.94 and a coefficient of variation of 3.8% in our laboratory.

Insulin sensitivity

We measured insulin sensitivity by the hyperinsulinemic-euglycemic clamp technique as described by DeFronzo *et al.* (23) and as previously reported in our laboratory (10, 24). Briefly, a Teflon catheter was inserted into the antecubital vein for the infusions of insulin and dextrose. Another Teflon catheter was retrogradely placed into the dorsal vein of the contralateral hand and used for the blood draws during the clamp procedure. This hand was placed in a hot box and warmed to 50 °C for arterialization of blood. At 0 min, a continuous infusion of insulin was started at a constant rate of 40 mU/m² body surface area·min. At the same time, a variable infusion of 20% dextrose was started to maintain fasting glycemia at $\pm 5\%$ (80 ± 4.4 mg/dL in endurance-trained women, 80 ± 6.4 mg/dL in resistance-trained women, and 81 ± 6.2 mg/dL in controls). Blood samples for glucose measurement were taken every 5 min for insulin measurement at -30, -10, 0, 30, 60, 70, 90, 105, and 120 min of the clamp. The insulin levels attained during the last 30 min of the clamp (90-120 min) before training were 75 ± 23 $\mu\text{U/mL}$ in endurance-trained women 74 ± 21 $\mu\text{U/mL}$ in resistance-trained women, and 76 ± 20 $\mu\text{U/mL}$ in controls ($P = \text{NS}$). After training, insulin levels were 76 ± 28 $\mu\text{U/mL}$ in endurance-trained women, 72 ± 22 $\mu\text{U/mL}$ in resistance-trained women, and 75 ± 23 $\mu\text{U/mL}$ in controls (mean \pm SD). The insulin-stimulated glucose disposal rate (M-value) was calculated as the average glucose infusion rate (milligrams per min) during the last 30 min of the 120-min clamp. Hepatic glucose production has previously been shown to be fully suppressed with the insulin dose used in our study to induce hyperinsulinemia (25).

Biochemical analyses

Plasma glucose concentrations were measured using the glucose oxidase method with an automated glucose analyzer (YSI, Inc., Yellow Springs, OH). Serum insulin was measured by a double antibody RIA (Diagnostics Products, Los Angeles, CA). The coefficient of variation for glucose measurement using the glucose oxidase method is less than 1.9%. The coefficient of variation for serum insulin measurement by the doubly antibody RIA method is less than 5%.

Statistical analysis

Differences in physical characteristics among groups at baseline were examined using a one-way ANOVA. A 2×3 repeated measures ANOVA was used to detect changes with time within the treatment condition (pre/post) and among groups (endurance *vs.* resistance *vs.* control). The repeated measures factor was the repeated tests during the exercise programs. Pearson product-moment correlation coefficients were used to examine the association between variables. Significance was accepted at $P < 0.05$.

Results

Table 1 shows physical characteristics for endurance-training, resistance-training, and control subjects before and after training. There were no differences among the three groups in baseline physical characteristics, suggesting a successful randomization. As expected, endurance-trained individuals increased their absolute $\text{VO}_{2\text{max}}$ by 29% ($P < 0.01$), whereas no changes were noted in resistance-trained and control subjects. Similar results were obtained when $\text{VO}_{2\text{max}}$ data were expressed per kg BW. Body weight and body mass index increased in resistance-trained individuals (both $P < 0.05$) relative to those in the other two groups. Fat mass, as measured by dual energy x-ray absorptiometry, showed no change in endurance-trained, resistance-trained, or control women. FFM showed no change in endurance-trained women or controls, but increased in resistance-trained women (2 kg; $P < 0.001$). As expected, resistance-trained individuals increased their 1-RM for leg press (29%), bench press (39%), military press (29%), and seated rows (27%; data not shown in table form). There was no increase in $\text{VO}_{2\text{max}}$ in the resistance-trained group, and there was no change in strength in the endurance-trained group.

Figure 1 shows pre- and posttraining values for absolute values of insulin sensitivity and indexed per kg FFM. Insulin sensitivity increased in both endurance-trained (pre, 421 ± 107 ; post, 490 ± 133 mg/min; $P < 0.05$) and resistance-trained (pre, 382 ± 87 ; post, 417 ± 89 mg/min; $P = 0.06$) women, with no change in controls (pre, 470 ± 139 ; post, 480 ± 168 mg/min). When data were expressed per kg FFM, the improvement in glucose disposal persisted in endurance-trained women (pre, 10.5 ± 2.7 ; post, 12.1 ± 3.3 mg/kg FFM·min; $P < 0.05$), whereas no significant change was noted in resistance-trained (pre, 9.7 ± 1.9 ; 10.2 \pm 1.8 mg/kg FFM·min) and controls (pre, 11.4 ± 2.8 ; post, 11.8 ± 3.5 mg/kg FFM·min). The improvement in $\text{VO}_{2\text{max}}$ was not related ($r = 0.02$; $P =$

TABLE 1. Changes in characteristics of younger women before and after training

Physical characteristic	Endurance training (n = 14)		Resistance training (n = 17)		Control (n = 20)	
	Pre	Post	Pre	Post	Pre	Post
Age (yr)	29 \pm 5		28 \pm 3		28 \pm 4	
$\text{VO}_{2\text{max}}$ (L/min)	2.1 \pm 0.5	2.7 \pm 0.5 ^a	2.1 \pm 0.4	2.2 \pm 0.3	2.2 \pm 0.5	2.3 \pm 0.4
Ht (cm)	163 \pm 5		164 \pm 7		165 \pm 7	
BW (kg)	59 \pm 5	59 \pm 5	58 \pm 6	60 \pm 6 ^b	60 \pm 7	61 \pm 8
BMI (kg/m ²)	22 \pm 2	22 \pm 2	22 \pm 2	23 \pm 2 ^b	22 \pm 2	22 \pm 2
DEXA measures						
Fat mass (kg)	16 \pm 5	15 \pm 4	16 \pm 4	17 \pm 4	17 \pm 6	17 \pm 6
Fat-Free mass (kg)	40 \pm 4	40 \pm 4	39 \pm 4	41 \pm 3 ^a	39 \pm 4	40 \pm 3

Values are the means \pm SD. BMI, Body mass index; Pre/Post, 6 months of endurance or resistance training.

^a $P < 0.001$.

^b $P < 0.05$.

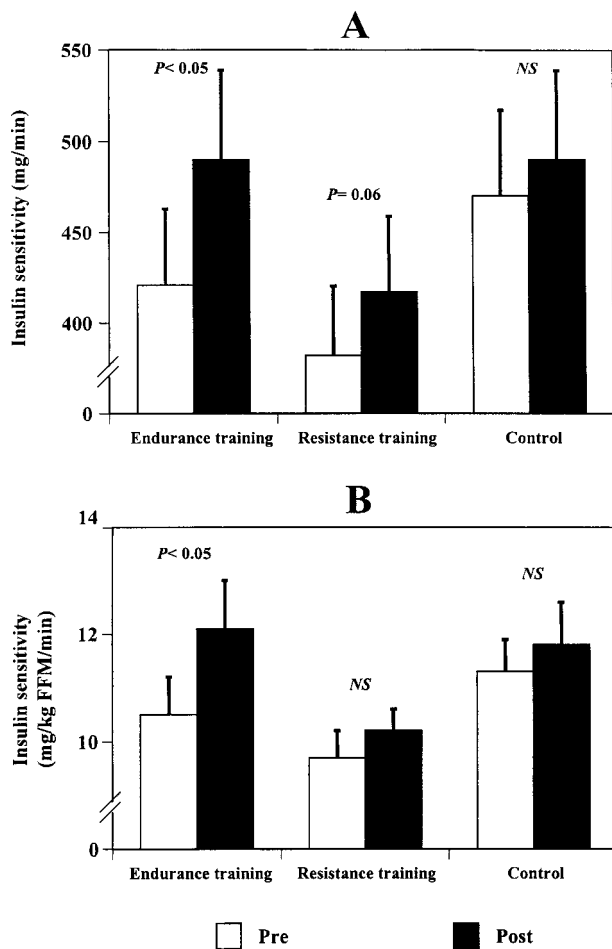


FIG. 1. Changes in insulin sensitivity before and after endurance training and resistance training and in control conditions. A, Values expressed on an absolute basis; B, values indexed per kg FFM. Values are the mean \pm SE. *, $P < 0.05$.

NS) to increased insulin sensitivity in the endurance-trained group.

Table 2 shows changes in abdominal adiposity, thigh adipose content, and lean tissue content before and after training. As expected for nonobese young women, baseline areas of sc adipose tissue and visceral adipose tissue were low. No significant changes were noted in sc or visceral adipose tissue in any group, as measured by computed tomography. Skeletal muscle characteristics, as estimated from computed tomography, are also shown in Table 2. We estimated quantities of midthigh fat area, thigh muscle area, and muscle attenuation values because of their reported relationship to insulin sensitivity (14, 15). Midthigh fat and muscle areas did not change in response to endurance or resistance training. On the other hand, we noted an altered composition in computed tomographic imaging in terms of higher mean attenuation values (HU) for both endurance-trained ($P < 0.05$) and resistance-trained ($P < 0.001$) individuals, suggesting a reduction in skeletal muscle lipid content. Changes in muscle attenuation in endurance-trained and resistance-trained individuals, however, were not related ($r = 0.24$; $P = \text{NS}$) to improved insulin sensitivity.

Discussion

Insulin resistance is linked with physical inactivity, increased visceral fat, and alterations in skeletal muscle characteristics. Moreover, we have shown the presence of these obesity-related phenotypes even in normal weight, apparently healthy, young women (10). Thus, interventions to improve or prevent the deterioration of the metabolic profile in this population have significant public health interest. The major findings are that both endurance and resistance training improve glucose disposal in young women, although by different mechanisms. An increase in the quantity of FFM from resistance training contributes to increased glucose disposal, probably from a mass effect, without altering the intrinsic capacity of the muscle to respond to insulin. On the other hand, endurance training enhances glucose disposal independent of changes in FFM, fat mass, or VO_2max , suggestive of an intrinsic change in the ability of the muscle to metabolize glucose.

Our experimental and methodological approaches lend credibility to our findings. Volunteers were randomly assigned to treatment conditions to control for known and unknown sources of experimental bias and subject self-selection. Moreover, the use of a control group decreases the influence of a placebo effect, and the application of euglycemic/hyperinsulinemic clamps and radiological imaging techniques provide direct measures of insulin sensitivity, body composition, and regional fat.

We originally hypothesized that endurance training would improve insulin sensitivity to a greater degree than resistance training due to a greater reduction in total fat and visceral fat. The physiological basis underlying our hypothesis is derived from several lines of evidence. First, endurance training may preferentially reduce visceral fat (26). Second, lower levels of visceral fat are associated with higher levels of insulin sensitivity and an improved metabolic profile (14–17, 27, 28). This hypothesis, however, was only partially supported by our findings in the present investigation. That is, endurance training improved insulin sensitivity to a greater degree than resistance training when expressed on an absolute basis or indexed per kg FFM. However, no change in total body fat, intraabdominal fat, or sc abdominal fat was found in endurance-trained women. Although it has been suggested that exercise training leading to a reduction in body fat is a prerequisite to improve glucose disposal (29), our findings as well as others (30) refute this assertion. Our results suggest that a vigorous program of endurance training improves glucose disposal independent of a reduction in total and regional body fat in nonobese young women.

It is possible that the volume of endurance exercise used in this study was inadequate to significantly modify total or regional body fat in young women who are not restricting energy intake. Indeed, it is possible that increased energy expenditure is compensated for by a greater energy intake, thus blunting any detectable change in total or regional body fatness (31, 32). Another potential reason underlying the absence of changes in body fatness is the potential of a ceiling effect. That is, it is difficult to reduce total or visceral fat in young women whose baseline levels are already low. This concept is supported by the findings of Wilmore and colleagues (33). They found only a small

TABLE 2. Changes in abdominal adiposity, thigh adipose, and lean tissue content in younger women before and after training

Physical characteristic	Endurance training (n = 14)		Resistance training (n = 17)		Control (n = 20)	
	Pre	Post	Pre	Post	Pre	Post
CT scan measures						
SAT area (L4-L5, cm ²)	194 ± 86	193 ± 80	186 ± 74	186 ± 85	147 ± 66	210 ± 95
VAT area (L4-L5, cm ²)	40 ± 11	41 ± 13	36 ± 17	36 ± 13	36 ± 13	41 ± 15
Thigh fat area (cm ²)	98 ± 34	90 ± 24	108 ± 32	102 ± 38	98 ± 29	101 ± 31
Thigh muscle area (cm ²)	108 ± 11	114 ± 14	109 ± 19	115 ± 16	119 ± 16	113 ± 17
Muscle attenuation (HU)	49 ± 3	51 ± 1 ^a	50 ± 2	52 ± 1 ^a	48 ± 2	48 ± 2

Values are the means ± SD.

^a $P < 0.05$.

reduction in intraabdominal fat (-3.1 ± 0.7 cm²; mean ± SE) in 299 overweight young women after an endurance training program similar to the one conducted in this investigation. This small decrement in intraabdominal fat, compared to the absence of changes in our study, probably reflects their greater baseline intraabdominal values in their overweight cohort (67 ± 45 cm²) compared to our nonobese women (40 ± 11 cm²). Unfortunately, no measure of insulin sensitivity was reported in their investigation, thus rendering the effects of a reduction in intraabdominal fat on insulin sensitivity unknown. It is likely that the volume of physical activity performed in the present study may be more beneficial in preventing increases in total and regional fat with advancing age rather than in promoting fat loss (34, 35).

As insulin-mediated glucose disposal occurs mainly in muscle, one would hypothesize that an increase in the skeletal muscle mass component of FFM would augment glucose disposal. Our data support this suggestion, as the absolute change in glucose disposal (milligrams per min) was related to the increase in FFM ($r = 0.48$; $P < 0.05$) after resistance training. There was no change, however, in glucose disposal when indexed per kg FFM. We interpret this finding to suggest that improved insulin sensitivity probably reflects a mass effect without altering the intrinsic capacity of the muscle to respond to insulin. The failure of resistance training to enhance insulin sensitivity per kg FFM could be due to the inability of resistance exercise to increase muscle capillary density (36) or to change muscle fiber types in an insulin-sensitive direction (37).

It is likely that the timing of our insulin sensitivity values measured relative to the last bout of exercise (4 ± 1 days) may partially reflect a detraining response on insulin sensitivity. That is, insulin sensitivity decreases as a function of time once the individual stops endurance training. We would suggest, however, that our selection of the time period to measure insulin sensitivity was reasonable, given that previous studies (30, 38, 39) showed a sustained effect of exercise training on insulin sensitivity measured 4–7 days after the last exercise bout. The magnitude of increase in resistance-trained (9%) and endurance-trained (16%) individuals was comparable to the 11% and 13% increases reported by Hughes and colleagues (30) and Tonino (38), respectively. These increases in glucose disposal, however, are less than those reported by other investigators (24–28%) (40, 41) who measured insulin sensitivity 48 h after the last exercise bout, when the residual effects of exercise are still intact. Volunteers in these studies, however, were not randomly assigned to treatment

conditions, nor did these investigators consider the effects of resistance training on insulin sensitivity.

We also considered the hypothesis that changes in lipid content within the skeletal muscle may predict changes in insulin sensitivity in women undergoing exercise training. This hypothesis is based on recent data showing that fat deposition within muscle may be an important aspect of body composition that is linked to insulin resistance (14, 15, 18). We used computed tomographic imaging to examine skeletal muscle at the level of the midthigh. We noted an increase in the attenuation values in endurance- and resistance-trained women, which most likely reflects a decrease in skeletal muscle fat content. However, we noted no relation between the improved glucose disposal and increased muscle attenuation values in endurance-trained or resistance-trained women ($r = 0.24$; $P = \text{NS}$). Thus, it is likely that other mechanisms are operative. For example, several investigators have suggested that the long-term regulation of the number and function of glucose transporters (42, 43), capillary proliferation (44), and the number of IIa (red glycolytic) fibers that have a higher GLUT-4 content and are more insulin responsive (45) are implicated in the improved insulin sensitivity in response to chronic exercise.

We identified only three reports in the literature (6, 46, 47) that examined the effects of both endurance and resistance training on proxy measures of insulin sensitivity. These studies, however, are not directly comparable to the present investigation because of differences in age, sex, initial metabolic characteristics of the volunteers, and experimental design differences. Two of these studies (6, 46) were performed in older men with untreated abnormal glucose regulation. Moreover, volunteers self-selected their mode of exercise, which raises questions regarding the biases introduced with subject self-selection. Both of these studies used an oral glucose tolerance test and found that endurance and resistance training reduced plasma glucose and insulin responses to an equivalent oral glucose load, suggestive of improved glucose tolerance and insulin sensitivity. On the other hand, Eriksson and colleagues (47) examined older men and women in a 6-month nonrandomized endurance-training study and found no discernible effect on insulin sensitivity, as measured by an iv glucose tolerance test. In the same study they used a 10-week circuit training program and found improved insulin sensitivity (23%) in eight males, as assessed with a euglycemic/hyperinsulinemic clamp technique. We suggest that additional randomized studies, such as our own,

using similar methodologies and in different populations, are needed to confirm our findings.

In summary, enhanced glucose uptake after physical training in young women occurs with and without changes in FFM and body composition. Two different mechanisms appear to be operative. Improved insulin sensitivity in resistance-trained women is probably due to a mass effect (*i.e.* increased FFM), whereas endurance training enhances glucose disposal independent of changes in FFM or $\text{VO}_{2\text{max}}$, suggestive of an intrinsic change in the muscle to metabolize glucose. We conclude that both endurance and resistance training programs are effective interventions to enhance glucose disposal in young, nonobese women.

Acknowledgments

We thank all participants for their time and effort in this study. We also thank Ms Denise DeFalco-McGeein, R.N.C., N.P., for her clinical management of our volunteers. Appreciation is extended to the administration and staff at the Racquets Edge Health Club in Essex, VT. We thank Travis Beckett and Kristan Kinamin for training our volunteers. Appreciation is expressed to Drs. Michael J. Toth and André Tchernof for their thoughtful comments on the paper.

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