Strength training increases insulin action in healthy 50- to 65-yr-old men

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Division of Gerontology, Department of Medicine, University of Maryland at Baltimore, and Baltimore Geriatric Research, Education and Clinical Center, Baltimore Veterans Affairs Medical Center, Baltimore 21201; and Departments of Kinesiology and of Human Nutrition and Food Systems, University of Maryland, College Park, Maryland 21218

Miller, John P., Richard E. Pratley, Andrew P. Goldberg, Patricia Gordon, Michelle Rubin, Margarita S. Treuth, Alice S. Ryan, and Ben F. Hurley. Strength training increases insulin action in healthy 50- to 65-yr-old men. *J. Appl. Physiol.* 77(3): 1122–1127, 1994.—The insulin resistance associated with aging may be due, in part, to reduced levels of physical activity in the elderly. We hypothesized that strength training increases insulin action in older individuals. To test this hypothesis, 11 healthy men 50–63 yr old [mean 58 ± 1 (SE) yr] underwent a two-step hyperinsulinemic-euglycemic glucose clamp with concurrent indirect calorimetry and an oral glucose tolerance test (OGTT) before and after 16 wk of strength training. The training program increased overall strength by 47% ($P < 0.001$). Fat-free mass (FFM; measured by hydrodensitometry) increased (62.4 ± 2.1 vs. 63.6 ± 2.1 kg; $P < 0.05$) and body fat decreased (27.2 ± 1.8 vs. 25.6 ± 1.9%; $P < 0.001$) with training. Fasting plasma glucose levels and glucose levels during the OGTT were not significantly lower after training. In contrast, fasting plasma insulin levels decreased (85 ± 25 vs. 55 ± 10 pmol/l; $P < 0.05$) and insulin levels decreased ($P < 0.05$, analysis of variance) during the OGTT. Glucose infusion rates during the hyperinsulinemic-euglycemic glucose clamp increased 24% (13.5 ± 1.7 vs. 16.7 ± 2.2 $\mu mol \cdot kg\,FFM^{-1} \cdot min^{-1}$; $P < 0.05$) during the low (20 $mU \cdot m^{-2} \cdot min^{-1}$) insulin infusion and increased 22% (55.7 ± 3.3 vs. 67.7 ± 3.9 $\mu mol \cdot kg\,FFM^{-1} \cdot min^{-1}$; $P < 0.05$) during the high (100 $mU \cdot m^{-2} \cdot min^{-1}$) insulin infusion. These increases were accompanied by a 40% increase ($n = 7$; $P < 0.08$) in nonoxidative glucose metabolism during the high insulin infusion. These results demonstrate that strength training increases insulin action and lowers plasma insulin levels in middle-aged and older men.

exercise; aging; glucose tolerance; body composition; weight lifting

THE INSULIN RESISTANCE associated with aging (5, 10, 25) may contribute to the high prevalence of impaired glucose tolerance and non-insulin-dependent diabetes mellitus in the elderly (13). Although age related, the impairment in insulin action in older individuals may not be primarily due to the aging process per se. Other factors such as physical inactivity and obesity are associated with insulin resistance (3), are more common in older individuals (11), and thus may contribute to the apparent decrease in insulin sensitivity that can be demonstrated even in otherwise healthy older individuals (3, 5, 10, 25).

To further understand the contribution of decreased levels of physical activity to the age-associated decline in insulin sensitivity, a number of recent investigations have examined the effects of exercise training in older individuals. Most studies to date have focused on aerobic exercise training, which increases insulin action, improves glucose tolerance, and decreases hyperinsulinemia in older individuals (18, 19, 28). However, because isometric contractions produce insulin-like effects on glucose uptake in isolated muscle (15), we hypothesized that strength training also increases insulin action in older individuals. Recent reports from our laboratory (26) and others (4, 23) provide some support for this hypothesis. Insulin responses to an oral glucose challenge are lower in both younger (4, 23) and older (4, 26) individuals after strength training, and, in some cases, glucose tolerance is improved as well (26). Although these findings suggest that strength training improves insulin action, this aspect was not directly measured in those studies. Thus, the purpose of the present investigation was to determine the effects of strength training on in vivo insulin action in middle-aged and older individuals.

METHODS

Subjects. Healthy nonsmoking 50- to 65-yr-old men were recruited to take part in this study. All subjects provided written informed consent according to the guidelines of the Institutional Review Boards of the University of Maryland and Francis Scott Key Medical Center before participation. None of the subjects exercised regularly, and all had been weight stable (±2.5 kg) for at least 6 mo before enrollment. Subjects underwent a thorough medical screening including a history and physical examination, a fasting blood profile, a graded exercise treadmill test, and a 2-h oral glucose tolerance test (OGTT) before entering the study. Individuals with significant abnormalities (including diabetes or other endocrine disorders, hypertension, evidence of cardiovascular disease, and orthopedic limitations) on screening were excluded. Eleven men, ranging in age from 50 to 63 yr [mean 58 ± 1 yr (SE)], entered the study.

Body composition. Body mass index was calculated by dividing weight by height squared (in kg/m^2). Body density was determined by hydrostatic weighing and was corrected for residual lung volume, which was measured by the closed-circuit oxygen dilution method (29) using a mass spectrometer (model 2000, Airspec, Kent, UK). Body fat and fat-free mass (FFM) were calculated from body density values (2). The waist-to-hip ratio, an index of the pattern of regional fat distribution, was calculated by dividing the minimal circumference of the abdomen by the circumference of the buttocks at the maximal gluteal protuberance.

Maximal aerobic power. Maximal oxygen consumption ($\dot{V}O_{2\,max}$) was determined during a treadmill test to subjective exhaustion as previously described (20). Expired air was collected in meteorological balloons at 1-min intervals during exercise. Fractional concentrations of oxygen and carbon dioxide in the expired gases were measured by mass spectrometry (model 2000, Airspec), and gas volumes were measured with a 120-liter Tissot spirometer. To establish that a true $\dot{V}O_{2\,max}$ was obtained, at least two of the following three criteria were met

for all $\dot{V}O_{2\,max}$ tests: *1*) a plateau (<2 $ml \cdot kg^{-1} \cdot min^{-1}$ increase) in oxygen uptake with increasing workload, *2*) a respiratory quotient of >1.10, and *3*) a heart rate within 10 beats/min of predicted maximum.

Dietary control. Subjects met individually with a nutritionist who instructed them in a diet that followed American Heart Association recommendations (1), and the subjects were weight stable on this diet for 6 wk before initial testing. They were instructed to maintain this dietary pattern as well as their baseline body weight during training. Compliance was monitored by analyzing 7-day food records before and after training and 24-h dietary recalls during the training phase (Nutritionist III, Silverton, OR). In addition, weekly weights were reviewed. All subjects were provided isocaloric weight-maintaining diets for 3 days before the hyperinsulinemic-euglycemic glucose clamp and for an additional 2 days before the OGTT. These diets, which were based on calculated energy requirements and 7-day food records, also followed American Heart Association recommendations (1) and provided ~50–55% of calories as carbohydrate, 15–20% as protein, and 30% as fat. All metabolic testing was performed in the morning after a 12-h overnight fast. Studies conducted after the strength-training intervention were performed 22–24 h after the last exercise session.

OGTT. After two baseline blood samples 10 min apart were obtained for measurement of glucose and insulin, subjects drank a lemon-flavored solution containing 40 g glucose/m^2 body surface area. Blood samples were collected at 30-min intervals for an additional 2 h to determine glucose and insulin responses. Plasma glucose levels were measured using the glucose oxidase method (Yellow Springs Instruments, Yellow Springs, OH). Aliquots of plasma were frozen at −70°C until measurement of insulin levels by radioimmunoassay (31). Samples obtained at baseline and after training were measured in a single insulin assay to eliminate interassay variation. The intra-assay coefficient of variation for this assay is 8%. Mean values of the fasting glucose and insulin samples were used in subsequent statistical analyses.

Hyperinsulinemic-euglycemic glucose clamp. Insulin action was measured using a two-step modification of the hyperinsulinemic-euglycemic glucose clamp technique of DeFronzo et al. (7). Briefly, an intravenous catheter was inserted into an antecubital vein for infusion of insulin, glucose, and potassium. A second catheter was inserted into a dorsal hand vein for blood sampling. The hand was then placed in a warming box thermostatically controlled at 70°C to arterialize the blood and was allowed to equilibrate for 30 min before baseline samples for glucose and insulin were obtained. Insulin (Humulin-R, Eli Lilly, Indianapolis, IN) was administered as a primed continuous infusion at a rate of 20 $mU \cdot m^{-2} \cdot min^{-1}$ for 90 min. This infusion was immediately followed by a second primed continuous infusion of insulin at a rate of 100 $mU \cdot m^{-2} \cdot min^{-1}$ for an additional 90 min. Potassium chloride was simultaneously infused at a rate of 4 mmol/h to prevent hypokalemia. Plasma glucose levels were measured (Beckman Instruments, Fullerton, CA) at 5-min intervals during the clamp and were maintained at basal levels with a variable infusion of 20% glucose, which was adjusted according to a computerized algorithm. Samples were obtained at 10-min intervals during the clamps for subsequent measurement of plasma insulin by radioimmunoassay (31). The higher insulin infusion was not completed in one subject after training because of problems with intravenous access.

Oxygen consumption and carbon dioxide production were measured before and during the clamp by indirect calorimetry (open-circuit dilution technique) using a metabolic cart (model 2900, Sensormedics, Yorba Linda, CA) calibrated before each test with use of standard gases of known concentrations. After insertion of the intravenous catheters, subjects rested comfortably in the supine position for at least 30 min before a 30-min baseline measurement was obtained. Measurements were also obtained during the last 30 min of each insulin infusion. Complete indirect calorimetry measurements were obtained in only seven individuals before and after training because of technical difficulties.

Calculations. Mean glucose infusion rates (GIR) were calculated from *minutes 60* to *90* of the 20 $mU \cdot m^{-2} \cdot min^{-1}$ insulin infusion and from *minutes 150* to *180* of the 100 $mU \cdot m^{-2} \cdot min^{-1}$ insulin infusion. Mean plasma insulin levels were also calculated during these intervals. On the basis of earlier reports (10), hepatic glucose output is ~75% suppressed during the low insulin infusion. Thus, GIR at this level of hyperinsulinemia represents the net effect of insulin on increasing insulin-mediated glucose uptake and suppressing hepatic glucose output. However, during the high insulin infusion hepatic glucose output should be almost completely suppressed in these healthy subjects. Therefore, at the high dose the GIR closely approximates the rate of tissue glucose disposal. Rates of glucose oxidation were calculated from measurements of oxygen consumption and carbon dioxide production with use of standard equations (9) and estimates of protein oxidation derived from 24-h urine urea nitrogen measurements obtained 1 day before the clamp. Nonoxidative glucose metabolism was calculated as the difference between total glucose disposal and glucose oxidation during the high insulin infusion. Because total glucose disposal cannot be approximated by GIR during the low insulin infusion, nonoxidative glucose disposal was not calculated at this dose.

Strength testing and training. Strength testing and training were performed on Keiser K-300 pneumatic variable-resistance machines. Subjects were familiarized with the equipment during at least four low-intensity exercise sessions before initial strength testing. Strength, indexed as the three repetition maximum (3 RM), was measured in six major muscle groups (leg press, leg extension, chest press, latissimus pull down, upper back row, and military press) before and after training.

Training sessions began with a 3-min warm-up period of low-intensity cycling followed by 10 min of static stretching exercises. The strength-training program consisted of the following 14 exercises: seated leg press, seated chest press, leg curl, latissimus pull down, leg extension, military press, hip abduction, hip adduction, upper back row, seated triceps extension, lower back, abdominal crunch, seated dumbbell curls, and supine abdominal crunches. The first three to four repetitions of each exercise were performed at ~90% of the 3 RM, after which resistance was gradually reduced without interruption of the normal cadence of exercise to permit subjects to complete 15 repetitions. A 1- to 2-min rest was allowed between exercises. One set of all exercises was performed in each session except for the lower body exercises, which were repeated at the end of the first circuit. Each training session lasted ~1 h. Subjects trained three times a week for 16 wk.

Statistical analyses. All data were analyzed with commercial statistical software packages (SAS, Cary, NC, and Statview II, Abacus Concepts, Berkley, CA). Plasma insulin levels from the OGTT were log transformed to yield a normal distribution before parametric analyses. All other data were normally distributed. The effects of strength training on the major dependent variables were tested with paired t tests. The effects of training on glucose and insulin responses to the OGTT and glucose oxidation during the clamp were tested with repeated-measures analysis of variance models. $P < 0.05$ was considered significant. Values are expressed as means ± SE.

RESULTS

Strength and body composition. The strength-training program was well tolerated. Attendance at scheduled

TABLE 1. *Strength (3 RM values) before and after strength training*

	Before Training	After Training
Upper body	162±8	266±11*
Lower body	413±26	580±37*
Total	575±30	846±42*

Values are means ± SE in kg. 3 RM, 3 repetition maximum. Upper body exercises include chest press, back row, shoulder press, and latissimus pull down; lower body exercises include leg press and leg extension; and total is sum of upper and lower body exercises. * $P < 0.001$.

training sessions was >95%, and all of the subjects completed the intervention. Training resulted in substantial increases in strength in both arms and legs (Table 1). Upper body strength (sum of 3 RM of 4 upper body exercises) increased 64% ($P < 0.001$), and lower body strength (sum of 3 RM of 2 lower body exercises) increased 40% ($P < 0.001$). Overall strength (total 3 RM) increased 47% ($P < 0.001$).

Strength training increased FFM by a mean of 1.2 kg ($P < 0.05$; Table 2) and decreased body fat from 27.2 to 25.6% ($P < 0.001$) but did not significantly change body mass. There were no significant changes in $\dot{V}O_{2\,max}$ or in the pattern of regional body fat distribution, indexed by the waist-to-hip ratio, with strength training (Table 2).

Analysis of food records indicated that subjects maintained the prescribed dietary pattern before, during, and after the training program. There were no significant differences in total energy consumed or percentage of calories derived from carbohydrate, fat, or protein with training (Table 3).

Glucose tolerance. At baseline, eight subjects had normal glucose tolerance tests and three were nondiagnostic (24). None of the subjects was impaired or diabetic. After training, all subjects were normal. However, in the group as a whole, strength training did not significantly lower fasting plasma glucose levels (5.3 ± 0.2 vs. 5.1 ± 0.1 mmol/l; NS) or glucose levels during the OGTT ($F = 2.0$, NS; Fig. 1). In contrast, both fasting plasma insulin levels (85 ± 25 vs. 55 ± 10 pmol/l; $P < 0.05$) and insulin levels during the OGTT were significantly lower after strength training ($F = 4.9$, $P < 0.05$; Fig. 2).

Insulin action. Mean plasma glucose levels during the hyperinsulinemic-euglycemic glucose clamps were comparable before and after training (4.9 ± 0.3 vs. 5.1 ± 0.2 mmol/l; NS). Plasma insulin concentrations also were similar before and after training during both the low insulin infusion (320 ± 25 vs. 335 ± 20 pmol/l; NS) and the high insulin infusion (1,705 ± 155 vs. 1,660 ± 95 pmol/l; NS; Fig. 3).

TABLE 2. *Body composition and $\dot{V}O_{2\,max}$ before and after strength training*

	Before Training	After Training
Weight, kg	85.8±2.8	85.4±3.2
BMI, kg/m^2	26.9±1.0	26.9±1.1
Body fat, %	27.2±1.8	25.6±1.9*
FFM, kg	62.4±2.1	63.6±2.1†
Waist-to-hip ratio	0.92±0.02	0.92±0.02
$\dot{V}O_{2\,max}$, ml · kg FFM^{-1} · min^{-1}	40.9±1.7	41.4±1.5

Values are means ± SE. BMI, body mass index; FFM, fat-free mass; $\dot{V}O_{2\,max}$, maximal oxygen consumption (maximal aerobic power). * $P < 0.001$; † $P < 0.05$.

TABLE 3. *Diet composition before and after strength training*

	Before Training	After Training
Energy consumed, kJ/24 h	9,627±368	9,782±247
Carbohydrate, %	52±1	51±1
Fat, %	30±1	30±1
Protein, %	18±1	19±1

Values are means ± SE. Distribution of macronutrients is expressed as percentage of total energy intake. There were no significant differences.

The mean GIR necessary to maintain euglycemia was higher after strength training at both physiological and supraphysiological levels of hyperinsulinemia (Fig. 4). During the 20 mU · m^{-2} · min^{-1} insulin infusion the mean GIR was 24% higher (13.5 ± 1.7 vs. 16.7 ± 2.2 μmol · kg FFM^{-1} · min^{-1}; $P < 0.05$), and during the 100 mU · m^{-2} · min^{-1} insulin infusion the mean GIR was 22% higher (55.7 ± 3.3 vs. 67.7 ± 3.9 μmol · kg FFM^{-1} · min^{-1}; $P < 0.05$). Nine of 11 subjects showed improvement at the lower insulin dose, and 9 of 10 subjects showed improvement at the higher insulin dose.

Basal rates of glucose oxidation measured by indirect calorimetry were not significantly different after strength training (6.9 ± 2.7 vs. 8.5 ± 3.3 μmol · kg FFM^{-1} · min^{-1}; NS). Glucose oxidation increased from the low dose to the high dose of insulin infusion ($F = 19.4$, $P < 0.001$) from 15.3 ± 2.2 to 21.7 ± 2.0 μmol · kg FFM^{-1} · min^{-1} before training and from 19.8 ± 2.1 to 24.5 ± 1.6 μmol · kg FFM^{-1} · min^{-1} after training, but there was no effect of training ($F = 1.7$, NS). Nonoxidative glucose disposal during the 100 mU · m^{-2} · min^{-1} insulin infusion increased in six of the seven men in whom it was measured by an average of 40% (34.9 ± 4.2 vs. 48.9 ± 4.7 μmol · kg FFM^{-1} · min^{-1}; $P < 0.08$).

DISCUSSION

The results of this study demonstrate that strength training increases in vivo insulin action in sedentary

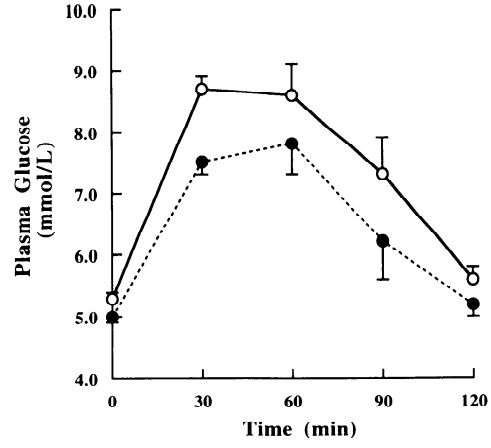

FIG. 1. Plasma glucose responses to oral glucose tolerance test (40 g/m^2 body surface area) before (○) and after (●) 16-wk strength-training program. There were no significant differences in fasting glucose levels or in glucose levels in response to oral glucose challenge.

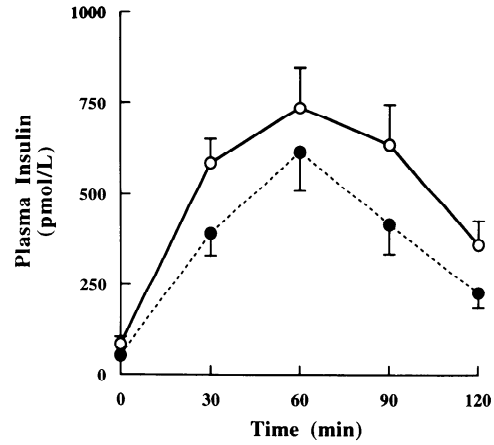

FIG. 2. Plasma insulin responses to oral glucose tolerance test (40 g/m^2 body surface area) before (○) and after (●) 16-wk strength-training program. Fasting plasma insulin levels were lower after training (85 ± 25 vs. 55 ± 10 pmol/l; $P < 0.05$), as were insulin levels in response to oral glucose challenge ($F = 4.9$, $P < 0.05$, analysis of variance).

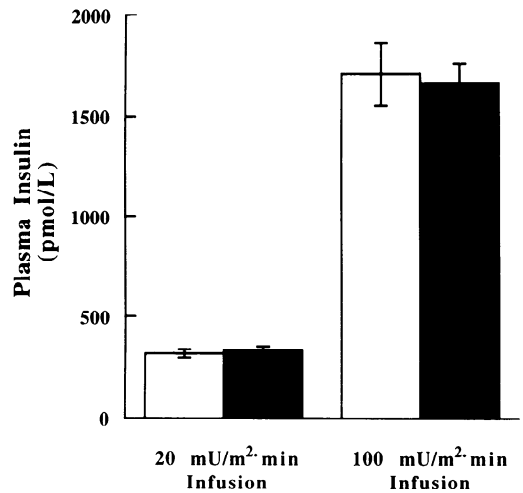

FIG. 3. Steady-state plasma insulin levels during last 30 min of 20 mU·m^{-2}·min^{-1} and 100 mU·m^{-2}·min^{-1} insulin infusions before (open bars) and after (solid bars) 16-wk strength-training program. There were no significant differences with training in insulin levels attained at either low or high dose.

middle-aged and older individuals. In these men, glucose disposal during the hyperinsulinemic-euglycemic glucose clamp increased by >20% at both physiological and supraphysiological levels of hyperinsulinemia after 16 wk of strength training. In addition, fasting insulin levels and insulin levels during the OGTT were significantly lower after training.

Previous studies of the effects of strength training on glucose tolerance have suggested that strength training increases insulin sensitivity. In some cases glucose tolerance improved with strength training (26), whereas in others it did not change (4, 23). However, in most (4, 23, 26) but not all studies (12), insulin responses were lower after training. The results of the present study are similar. Although plasma glucose levels were not significantly lower, fasting plasma insulin levels and insulin responses to the oral glucose challenge fell significantly with strength training. The decreases in plasma insulin levels in these men were comparable to reductions that we (26) and others (4) have reported in older individuals as well

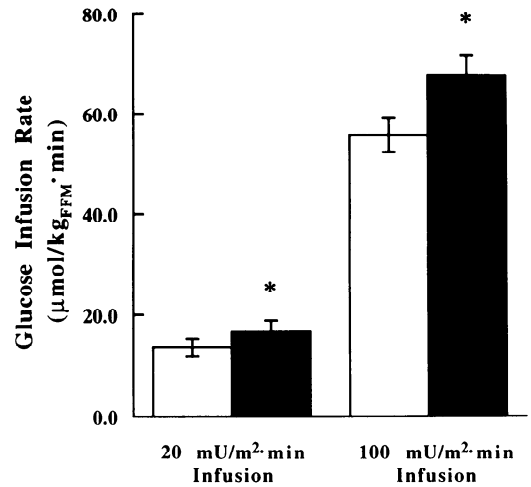

FIG. 4. Glucose infusion rates during last 30 min of 20 mU·m^{-2}·min^{-1} and 100 mU·m^{-2}·min^{-1} insulin infusions before (open bars) and after (solid bars) 16-wk strength-training program. Glucose infusion rates increased 24% (13.5 ± 1.7 vs. 16.7 ± 2.2 μmol·kg fat-free mass^{-1}·min^{-1}; * $P < 0.05$) during low insulin infusion and 22% (55.7 ± 3.3 vs. 67.7 ± 3.9 μmol·kg fat-free mass^{-1}·min^{-1}; * $P < 0.05$) during high insulin infusion.

as to changes that have been reported for younger individuals (4, 23). The differences among studies with respect to the effect of training on glucose tolerance may relate to subject selection. In the present study, changes in fasting and 2-h plasma glucose levels were related to initial levels ($r = 0.86$, $P < 0.001$ and $r = 0.59$, $P < 0.08$, respectively) such that individuals with higher initial glucose levels had relatively larger decreases with training. It is possible that the relatively normal glucose tolerance of this group precluded our finding a significant decrease in plasma glucose levels with strength training. This possibility may also explain why an earlier study from our laboratory, which included individuals with impaired glucose tolerance, was able to demonstrate an improvement in glucose tolerance (26). Similar considerations may apply to differences among studies with respect to changes in plasma insulin levels with training.

Although improvements in insulin and glucose responses to an OGTT provide indirect evidence that strength training increases insulin action, they do not, by themselves, prove that this is the case. The hyperinsulinemic-euglycemic glucose clamp technique used in the present study overcomes many of the limitations inherent in an OGTT. Using this technique, it was possible to measure increases in insulin action at both physiological and supraphysiological insulin levels after strength training. Thus, these results provide direct evidence supporting the hypothesis that strength training increases insulin action.

Although this is the first demonstration that strength training increases insulin action, an earlier cross-sectional study reported increased rates of glucose disposal in young bodybuilders compared with age- and weight-matched sedentary control subjects (30). In contrast to the results of the present study, glucose disposal rates were not significantly higher in the bodybuilders after correction for the large differences in FFM between groups. In addition to differences in study design and subject selection, these two studies differ with respect to

the duration and type of training. Thus, further investigations are necessary to resolve these disparate findings.

The improvements in glucose metabolism seen in this study are similar to improvements reported after aerobic exercise training in older individuals. For example, increases in insulin sensitivity ranging from 13 to 36% (18, 19, 28) have been reported after 3–6 mo of aerobic exercise training. It has been speculated that the improvements seen with aerobic exercise training are related to increases in $\dot{V}O_{2\,max}$ and decreases in body fat, particularly of upper body depots (21). These mechanisms are unlikely to account for the increase in insulin action observed in the present study, since neither $\dot{V}O_{2\,max}$ nor the waist-to-hip ratio changed significantly with strength training. Although body fat decreased from 27.2 to 25.6% in these men, this decrease, too, is an unlikely explanation for the improvement in insulin action. On the basis of data obtained in our laboratory in a comparable group of subjects (3), a decrease in body fat of this magnitude would be expected to increase glucose disposal by <2% during the high insulin infusion.

Most of the glucose administered during a clamp is metabolized in muscle (6). However, it is doubtful that the improvements demonstrated in the present study are due solely to the increase in FFM. Glucose disposal increased significantly at both physiological and supraphysiological insulin levels even after the correction for FFM in these older men. Furthermore, even if the average increase in FFM of 1.2 kg were entirely attributable to an increase in muscle cell mass, this would represent only an ~5% increase in total muscle mass (assuming muscle to be ~40% of FFM) and would be quantitatively insufficient to account for the 20% increase in glucose disposal that was observed. Thus, although the increase in muscle mass with strength training may contribute to increased rates of glucose disposal, it is unlikely to be the major factor explaining the improvement in insulin action.

Oxidative glucose metabolism measured during a hyperinsulinemic-euglycemic glucose clamp is not different in aerobically trained individuals and sedentary control subjects (8). Thus, the higher rates of total body glucose disposal observed in aerobically trained subjects are primarily due to increases in nonoxidative glucose disposal (8). It appears that the effect of strength training is similar. Oxidative glucose metabolism was not altered by strength training. On the other hand, nonoxidative glucose metabolism tended to be higher after strength training, although this increase did not reach statistical significance (perhaps because of the small sample size).

Glucose disposal through nonoxidative pathways during glucose clamp experiments is largely due to glycogen synthesis and is directly related to the fractional activity of glycogen synthase in skeletal muscle (8). The activity of this enzyme complex is higher in aerobically trained individuals (8). It is possible that glycogen synthase activity is also increased by strength training, although it has not been reported. It is known, however, that glycogen depletion, which stimulates the activity of glycogen synthase, occurs with acute bouts of resistive exercise (27) and that strength training significantly increases skeletal muscle glycogen levels (22). Therefore, the effects of strength training on glycogen storage and utilization are consistent with an increase in the activity of glycogen synthase.

Glucose disposal is directly related to levels of the insulin-stimulatable glucose transport protein GLUT-4 in skeletal muscle (8). Levels of this protein are higher in endurance-trained individuals (8, 17). Thus, it is conceivable that strength training increases insulin action by increasing skeletal muscle GLUT-4 levels. Although the effects of strength training on skeletal muscle GLUT-4 levels have not been investigated, skeletal muscle GLUT-4 levels were unchanged despite a worsening of insulin responses to OGTT in 12 young strength-trained individuals who detrained for 14 days (16). Unfortunately, the effect of detraining on the subcellular localization of GLUT-4, an important determinant of transporter activity, was not addressed in that study.

Acute bouts of aerobic exercise increase insulin action and decrease insulin responses to an oral glucose challenge independently of training (14). From the results of the present study it cannot be determined whether the increase in insulin action with strength training is truly a training effect or whether the results are due to a residual effect of the last bout of exercise. However, lower insulin responses to oral glucose were demonstrated 48–72 h after the last exercise session in earlier studies (4, 23), which suggests that the effect of strength training is a true training adaptation and not simply an acute effect of exercise.

In summary, we have demonstrated that strength training increases insulin action and decreases insulin levels in sedentary older individuals. These improvements appear to be independent of changes in $\dot{V}O_{2\,max}$, body composition, and body fat distribution. The effects of strength training on insulin action are similar to those produced by aerobic exercise training. This finding, when considered with the early observation that isotonic and isometric contractions are capable of stimulating glucose transport to comparable degrees in isolated muscle preparations (15), suggests that resistive and aerobic exercises increase insulin action by a common mechanism. Additional studies focusing on the changes in skeletal muscle glucose transport and metabolism produced by strength training are required to further understand the effects of this form of exercise. In addition to providing insights into the basic mechanisms regulating glucose and insulin metabolism, these studies also may lead to more effective intervention strategies aimed at reversing the insulin resistance of aging.

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