

Unaltered insulin sensitivity after resistance exercise bout by postmenopausal women

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ABSTRACT

CHAPMAN, J., A. W. GARVIN, A. WARD, and G. D. CARTEE. Unaltered insulin sensitivity after resistance exercise bout by postmenopausal women. *Med. Sci. Sports Exerc.*, Vol. 34, No. 6, pp. 936–941, 2002. **Purpose:** The major aims of this study were to determine whether a single session of resistance exercise would alter insulin sensitivity, glucose effectiveness, and C-peptide response to glucose challenge in a group of previously sedentary, postmenopausal women. **Methods:** Ten postmenopausal women (aged 57.5 ± 1.6 yr) were studied. Each participant underwent two frequently sampled intravenous glucose tolerance tests (FSIVGTT): without prior exercise (no exercise), and postexercise (15 h after a session of resistance exercise; three sets of 10 repetitions performed at 50%, 75%, and 100% of 10-repetition maximum for 7 exercises). Insulin sensitivity and glucose effectiveness were determined according to Bergman's minimal model procedure. In addition, C-peptide concentration and glucose disappearance were measured. **Results:** There was no significant difference between trials for insulin sensitivity, glucose effectiveness, glucose disappearance, or area under the curve (AUC) for glucose or insulin during the glucose challenge. AUC for C-peptide tended ($P = 0.059$) to be 10% higher in the postexercise versus no exercise trial, and C-peptide values were significantly ($P \leq 0.02$) higher at several time points (60, 70, 140, and 180 min) during the postexercise compared with no exercise trial. **Conclusions:** In contrast to previously reported results with young men and women after a single bout of endurance exercise, insulin sensitivity was unaltered by a single session of resistance exercise in postmenopausal women. Higher plasma C-peptide values concomitant with unchanged insulin values provide evidence that resistance exercise may have induced a slightly higher insulin secretion and a proportional increase in insulin clearance. **Key Words:** WEIGHT LIFTING, GLUCOSE TOLERANCE, C-PEPTIDE, INSULIN SECRETION

Abnormal glucose and insulin metabolism have important implications for health. Insulin resistance (a subnormal biologic effect of insulin) is associated with increased risk for hypertension (23) and atherosclerosis (15), even in the absence of diabetes. Understanding the efficacy of various modes of exercise to improve insulin sensitivity is a valuable goal.

Many studies have demonstrated that endurance exercise training can improve insulin sensitivity in people of diverse ages, both genders, and with normal, impaired, or abnormal glucose tolerance (4,7,25). In addition to adaptations after endurance training, a single session of endurance exercise can elevate insulin action in previously sedentary women and men (3,20).

A substantial body of previous research has documented that resistance exercise training can also amplify insulin action in men (8,9,16,21,30). In contrast, few studies that have assessed the influence of resistance exercise training on glucoregulation and insulin action have included women (18,26,27), and the efficacy of this mode of exercise for improving insulin sensitivity in women remains to be clearly established. This gap in knowledge is important because the prevalence of diabe-

tes is as great in women as in men (12). Understanding the effects of resistance exercise on specific aspects of health (e.g., insulin sensitivity) could help women and health professionals working with women in planning an appropriate exercise regimen. To fully understand the effects of resistance exercise training, it is useful to address the acute effects of one resistance-exercise session. Apparently, only one published study has evaluated the effect of a single resistance-exercise session on glucose and insulin dynamics (11). Although insulin sensitivity was not directly measured, insulin values during an oral glucose tolerance test (OGTT) were lower in the postexercise compared with preexercise test concomitant with unchanged glycemia. Young adult, nondiabetic people, as well as older type 2 diabetic people were studied, but because data for men and women were combined, the specific effects of acute resistance exercise on insulin and glucose dynamics in women remain uncertain.

Accordingly, the purpose of this study was to elucidate the influence of acute resistance exercise on glucoregulation and insulin sensitivity in postmenopausal women. The specific aims were to assess the influence of a single resistance exercise session on insulin sensitivity, glucose effectiveness (the efficiency by which glucose restores its own concentration independent of dynamic insulin response) (2), and plasma C-peptide response to glucose challenge (an index of insulin secretion) in a group of postmenopausal women.

0195-9131/02/3406-0936/\$3.00/0

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Submitted for publication August 2001.

Accepted for publication October 2001.

METHODS

Subjects. Ten postmenopausal women (aged 57.5 ± 1.6 yr; 9.7 ± 1.6 yr since last menstrual period) were studied. Subjects were sedentary, defined as engaging in less than 20 min of exercise twice weekly. They did not use nicotine and had no history of hypertension, based on self-report. All participants signed an informed consent that was consistent with *MSSE* guidelines and had been approved by the University of Wisconsin-Madison Institutional Review Board.

Experimental design. Participants were studied for insulin and glucose dynamics under control (no exercise) and treatment (postexercise) conditions. Subjects reported to the University of Wisconsin Sports Medicine Clinic on five separate occasions. During visit 1, informed consent and physician's release forms were collected, a medical history was taken, resting blood pressure measured, and anthropometry (skin-folds, body mass index, and waist-to-hip ratio) was assessed. Participants then underwent a familiarity session with the resistance equipment. Visit 2 (2–26 d after visit 1) consisted of the initial (no exercise) frequently sampled intravenous glucose tolerance test (FSIVGTT) at 8:00 a.m. Visit 3 (2–16 d after visit 2) consisted of a one-repetition maximum (1-RM) test on each of the seven resistance machines. Beginning at 5:00 p.m. on visit 4 (3–5 d after visit 3), the women exercised using the resistance machines. The next morning at 8:00 a.m. (visit 5), the postexercise FSIVGTT was performed. The postexercise FSIVGTT was at least 7 d after the no exercise FSIVGTT.

Resting blood pressure. Resting blood pressure was measured via auscultation using a stethoscope and mercury sphygmomanometer after the subject had been sitting quietly for at least 5 min.

Diet. Subjects recorded their last meal on the evening before the no exercise FSIVGTT, and they were provided with a copy of this record and asked to replicate that meal on the evening prior the postexercise FSIVGTT. In addition, the subjects were asked to fast overnight 12–14 h before each FSIVGTT. A subgroup of four subjects recorded their daily intake in a food diary for 2 d before each FSIVGTT. Dietary analysis was performed using the Diet Balancer program, version 1.0 (Nutridata Software Corp., Wappingers Falls, NY).

Anthropometry. Three skin-fold sites (triceps, suprailium, and thigh) were measured using Harpenden (Bothel, WA) skin-fold calipers to determine body density of the women (17), which was in turn used to estimate percent fat (29). In addition, body mass index (BMI, $\text{kg}\cdot\text{m}^{-2}$ = $\text{weight}\cdot\text{height}^{-2}$) and waist-to-hip ratio (WHR) were determined.

Exercise. The exercise familiarity session consisted of instruction and participation with the seven exercise stations: chest press, row, leg press, biceps curl, triceps extension, leg extension, and leg curl (Cybex, Medway, MA). This exercise sequence was maintained throughout the study. Proper form and breathing were demonstrated before participants were fitted comfortably in each machine (seat

adjustments recorded so they could be duplicated for the entire study) and allowed to practice by performing one set of 15 repetitions with no added resistance on each machine.

The 1-RM test was used to estimate the most weight a subject could lift on each of the seven resistance exercise machines. Subjects performed a general warm-up of 5–10 min using a cycle ergometer or treadmill, followed by 10 min of static stretching. Next, approximately 12 repetitions of the lightest weight of the particular resistance machine were performed. Then the weight was increased to an appropriate level, one repetition performed, and the load was increased by 6.25- or 12.5-lb increments, except for the leg press (10- or 20-lb increments). Subjects performed one repetition on each attempt (with 3–5 min of rest between attempts) until they were unable to complete the full range of the lift. The last successful attempt was considered the 1-RM.

The resistance exercise session was patterned after that described by Fluckey et al. (11). In brief, subjects performed a general warm-up of 5–10 min using a cycle ergometer or treadmill, followed by 10 min of static stretching. The resistance exercise session consisted of three sets of 10 repetitions on each resistance machine. The weight used for each set of each exercise was determined from the individual subject's 10-RM on each machine. The 10-RM was estimated as 75% of 1-RM (11). The first set was 50% of 10-RM, the second set was 75% of 10-RM, and the third set was 100% of 10-RM. Subjects were allowed 70-s rest between sets and 2-min rest between exercise stations. Subjects were allowed to stop before the completion of 10 repetitions if their exercise technique was compromised or at volitional fatigue.

Frequently sampled intravenous glucose tolerance test (FSIVGTT). Subjects reclined comfortably throughout the procedure. An indwelling catheter was placed in an antecubital arm vein of each arm. One catheter was used for blood collection, whereas the glucose bolus was injected into the other catheter. After the placement of the catheters, 30 min was allowed before initial blood sampling. Basal samples were collected in Gray top Vacutainer tubes (Fisher Scientific, Hampton, NH) containing a glycolytic inhibitor (sodium fluoride) and anticoagulant (potassium oxalate) before (15, 10, 5, and 1 min) the glucose injection. Glucose (50% dextrose injectable, Abbott Laboratories, North Chicago, IL) ($300 \text{ g}\cdot\text{kg}^{-1}$ of body weight; 50% aqueous solution) was steadily injected from time zero to 1 min. Postinjection samples were collected as previously described during the next 180 min (1). Blood samples were centrifuged, and resultant plasma was stored at -80°C until analyzed.

Biochemical analyses. Plasma insulin was measured using a radioimmunoassay with human insulin as the standard (Linco, St. Charles, MO). Plasma C-peptide was measured by radioimmunoassay with human C-peptide as the standard (Linco). Plasma glucose concentration was determined by glucose oxidase method (YSI model 2300 Stat Plus Glucose and L-Lactate Analyzer, Yellow Springs, OH). Plasma hemoglobin concentration was

TABLE 1. Physical characteristics of the subjects.

	Mean ± SE
Age (yr)	57.5 ± 1.6
Weight (kg)	70.4 ± 4.6
Height (cm)	161.5 ± 2.7
Body mass index (kg·m ⁻²)	27.0 ± 1.7
Body fat (%)	29.1 ± 2.7
Waist circumference (cm)	84.1 ± 3.5
Hip circumference (cm)	106.3 ± 3.3
WHR	0.79 ± 0.02
Systolic blood pressure (mm Hg)	123 ± 4
Diastolic blood pressure (mm Hg)	74 ± 3

spectrophotometrically analyzed (Sigma, St. Louis, MO; Procedure 525). Plasma creatine kinase activity was measured by spectrophotometric assay (Procedure 45-UV; Sigma).

Calculations. Plasma glucose, plasma insulin, and sampling time after glucose injection were entered into MINMOD for each FSIVGTT. The MINMOD program computed insulin sensitivity (S_I) and glucose effectiveness (S_G), and acute insulin response to glucose (AIR_g) (2). Glucose disappearance constant (K_g) was calculated as the least square regression of the natural logarithm of the glucose concentration at the time points 10–30 min after the intravenous glucose challenge. The trapezoid method was used to calculate area under the curve (AUC) for glucose, insulin, and C-peptide.

Statistical analyses. Statistical analyses were done using SigmaStat, version 2.03 (San Rafael, CA). Data for the no exercise and the postexercise trials were compared using a two-tailed, paired *t*-test ($P < 0.05$). The change in insulin AUC on the day after a single resistance-exercise session that was reported by Fluckey et al. (11) was used to estimate the adequate sample size ($N = 10$ at $\beta = 0.7$ and $\alpha = 0.05$). When data did not pass the Kolmogorov-Smirnov normality test, a two-tailed, paired Wilcoxon signed rank test was used. Correlations were performed using the Pearson product moment technique.

RESULTS

Subject characteristics. Characteristics of the participants are summarized in Table 1.

Exercise measures. Absolute strength (kg lifted) was measured via 1-RM testing on all seven resistance machines. All of the women completed 10 repetitions in the first set for all seven of the exercise machines. During the second set, a single woman completed only seven repetitions on the leg extension machine; for the other six machines, all 10 women completed ten repetitions. During the third set, everyone completed 10 repetitions on four machines (row, leg press, triceps extension, and seated leg curl). Eight, six, and two women completed 10 repetitions for the chest press, biceps curl, and leg extension machines, respectively.

Diet. No statistically significant differences were found between the no exercise and postexercise trials for total kcal consumed per day (1594.1 ± 220.2 , no exercise; 1699.6 ± 198.4 , postexercise) or percentage of each macronutrient:

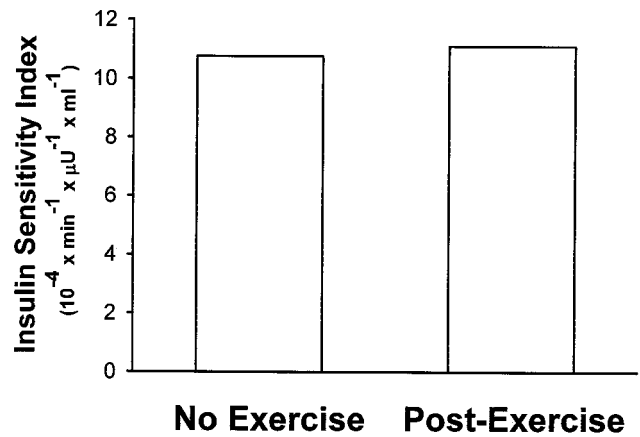


FIGURE 1—Insulin sensitivity (S_I) for the no exercise and postexercise conditions. Values are medians ($P = 0.49$, paired, two-tailed Wilcoxon signed rank test).

protein ($16.2 \pm 1.9\%$ vs $16.6 \pm 2.3\%$), carbohydrate ($56.1 \pm 10.8\%$ vs $53.9 \pm 8.0\%$), or fat ($27.7 \pm 8.8\%$ vs $29.5 \pm 2.8\%$).

Metabolic measures. Each participant had normal fasting plasma glucose, and none were identified as having impaired glucose tolerance or diabetes based on the criteria established by the American Diabetes Association (10). No significant differences between no exercise and postexercise values were found for glucose or insulin concentrations at any individual time points (including basal measures before glucose infusion) or AUC, S_G , AIR_g , or K_g (Table 2). In addition, no significant effect of exercise was found for S_I values, which were compared with a Wilcoxon signed rank test because S_I values did not pass the normality test (Fig. 1). No significant correlation was found between S_I values (postexercise; or delta exercise calculated as postexercise – no exercise) and exercise performance during the exercise session (pounds \times repetitions; or pounds \times repetitions \div lean body mass). C-peptide AUC tended to be 10% higher for the postexercise compared with no exercise condition ($P = 0.059$; Fig. 2). No significant correlation was found between C-peptide AUC and exercise performance during the exercise session. C-peptide values were significantly greater in the postexercise compared with no exercise trial at 60 ($P = 0.015$), 70 ($P = 0.004$), 140 ($P = 0.01$), and 180 ($P = 0.02$) min (Fig. 2). Plasma creatine kinase was not significantly different ($P = 0.946$) for the no exercise (26.6 ± 4.2 U·mL⁻¹) compared with the postexercise (26.9 ± 4.3) condition. Creatine kinase (no exercise, postexercise, or

TABLE 2. Metabolic measures.

	No Exercise	Postexercise
S_G (min ⁻¹)	0.022 ± 0.006	0.019 ± 0.004
AIR_g (μU·mL ⁻¹)	149.4 ± 28.9	141.3 ± 17.1
K_g (%·min ⁻¹)	1.52 ± 0.30	1.59 ± 0.31
Basal glucose (mg·dL ⁻¹)	78.9 ± 3.1	80.0 ± 2.5
Basal insulin (μU·mL ⁻¹)	6.0 ± 0.7	5.7 ± 0.5
Glucose AUC (mg·dL ⁻¹ ·min)	19,078 ± 788	19,163 ± 1056
Insulin AUC (μU·mL ⁻¹ ·min)	1743 ± 248	1720 ± 146
C-peptide AUC (ng·mL ⁻¹ ·min)	345.0 ± 45.7	380.1 ± 42.1

Values are mean ± SE; S_G , glucose effectiveness; AIR_g , acute insulin response to glucose; K_g , glucose disappearance constant; AUC, area under the curve.

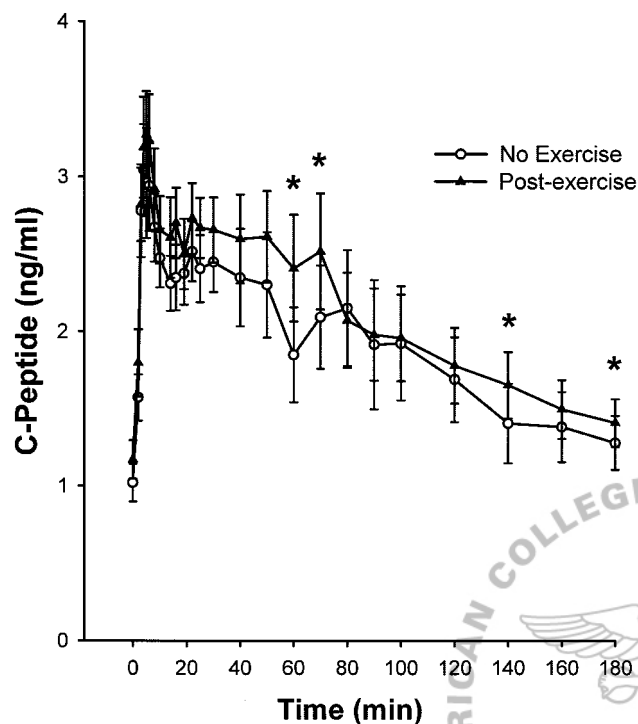


FIGURE 2—Plasma C-peptide concentration during no exercise and postexercise FSIVGTT. Values are mean \pm SE; * significantly different from no exercise value at corresponding sample time (60 min, $P = 0.015$; 70 min, $P = 0.004$; 140 min, $P = 0.01$; 180 min, $P = 0.02$).

postexercise – no exercise) was not significantly correlated to S_I (no exercise, postexercise, or postexercise – no exercise).

Hemolysis can lead to reduced plasma insulin (5,22). Blood was collected using tubes containing sodium fluoride/potassium oxalate, which has been reported to elicit moderate hemolysis accompanied by an ~28% lower insulin values compared with serum (5). We replicated this comparison and found 35% lower values for plasma sampled in tubes containing oxalate compared with serum sampled at the same time. The degree of hemolysis induced by sodium fluoride/potassium oxalate is undetectable by visual inspection, but it can be assessed by plasma hemoglobin concentration (5). Plasma hemoglobin ($g \cdot L^{-1}$) was not significantly different ($P = 0.412$) for no exercise compared with postexercise samples, indicating prior exercise did not alter the amount of hemolysis. Recalculation of S_I and AIR_G using insulin values adjusted for the 35% decrease results in proportional changes in each value (35% decrease in S_I and 35% increase in AIR_G). However, the interpretation of the results is unchanged, because as with unadjusted values, no significant effect of postexercise versus no exercise was found. S_G and K_G values were unaffected because insulin values are not used for their calculation, and sodium fluoride/potassium oxalate does not lower glucose concentration (28). Hemolysis also does not affect plasma C-peptide values (22).

DISCUSSION

The major aims of this study were to characterize in postmenopausal women the influence of a single resistance

exercise session on subsequent insulin sensitivity, glucose effectiveness (S_G), and plasma C-peptide response to a glucose challenge. Insulin sensitivity and S_G were unchanged by exercise, but C-peptide concentration was higher in the postexercise condition.

The absence of exercise-induced changes in glucose measures in the current study was expected based on results found in most earlier studies of acute exercise. Basal glucose and AUC for glucose did not differ between the no exercise and postexercise condition, in agreement with findings reported after acute resistance exercise in either young nondiabetic or older type 2 diabetic people (11). There was no difference in K_G between trials in the current study, which was apparently the first to assess the influence of acute resistance exercise on K_G . Neither mild (60 min at 47% $\dot{V}O_{2max}$) nor hard (36 min at 82% $\dot{V}O_{2max}$) exercise on a cycle ergometer influenced K_G on the following day in young men, but exhaustive exercise (60-min mild exercise followed by 3-min intervals of hard exercise separated by 2-min rest intervals until fatigue) increased K_G (13). S_G values were similar for the no exercise and postexercise conditions, indicating that this insulin-independent mechanism for lowering blood glucose was not altered by acute resistance exercise. This result was consistent with the absence of an effect on S_G on the day after one bout of mild, hard, or exhaustive exercise on a cycle ergometer (13).

Plasma C-peptide concentration was higher at several time points during the FSIVGTT from the postexercise compared with no exercise trial. Because C-peptide and insulin are secreted in equimolar amounts and C-peptide clearance is relatively slow (24), higher C-peptide concentration postexercise suggests a greater insulin secretion rate compared with the no exercise condition. However, it is prudent to recognize plasma C-peptide concentration is an indirect marker of insulin secretion. The absence of altered plasma insulin levels concomitant with higher C-peptide levels in the postexercise compared with no exercise trial is consistent with greater insulin clearance after acute resistance exercise. Fluckey et al. (11) reported a reduction in insulin concentration concomitant with no change in C-peptide concentration after a single resistance-exercise bout (data for men and women were pooled), which also suggests that insulin clearance was increased, although their results indicate unaltered insulin secretion. Joseph et al. (18) studied women and men (54–71 yr old) who performed weight-lifting exercise for 12 wk, followed by 72 h of inactivity; half of the subjects also received chromium supplementation whereas the remaining subjects received a placebo. By focusing on the placebo-treated group of women, insulin AUC during an oral glucose tolerance test (OGTT) was virtually identical when assessed before compared to after training (18). C-peptide AUC did not differ significantly in the pretraining condition compared to 72 h after the last training bout. It is unclear which differences in experimental design among these studies might be important for their differing results for insulin and C-peptide dynamics.

No significant exercise effect on insulin sensitivity or insulin AUC was found. A very similar exercise protocol

produced a decreased insulin response, concomitant with unaltered glucose AUC, during an OGTT in nondiabetic (27 yr old) and diabetic (53 yr old) men and women (11). These findings, in contrast with those in the current study, suggested that insulin sensitivity may have been increased. Several aspects of the experimental design need to be considered when interpreting these studies, including 1) age and 2) gender.

First, with regard to age, it is well established that acute endurance exercise can increase insulin sensitivity in young adults (3,13). In addition to the present study, there are apparently only four other published studies involving middle-aged or older people and the insulin response to a glucose challenge on the day after acute exercise. Men (53 yr) with mild type 2 diabetes were found to have no change in AUC for glucose or insulin (during an OGTT) the day after 60 min of endurance exercise (25). Sedentary men (~50 yr) had unchanged AUC for glucose or insulin (during an OGTT) the day after 40 min of treadmill running (14). Walking combined with arm ergometry for 55 min by older (69 yr) men and women produced no change in AUC for glucose or insulin (during an OGTT) on the following day (6). As discussed above, Fluckey et al. (11) reported a decrease in insulin AUC on the day after a bout of resistance exercise. Including the present study, four of five studies have not found evidence for altered insulin levels with a glucose challenge in middle-aged or older people on the day after a single exercise session.

With regard to gender, insulin sensitivity is increased after acute endurance exercise in young men (3,13) and young women (20). With brief (7 consecutive days) endurance training, both men and women groups who were young (~21 yr) or older (~60 yr), had increased insulin sensitivity (7). The relative improvements with endurance training were as great for women as for men. Taken together, previous studies have demonstrated that young women respond to endurance exercise, acute or chronic, with enhanced insulin action. The similar responses to endurance exercise training in women compared with men, both young and older, do not eliminate the possibility for a gender difference in response to acute resistance exercise.

As described above, apparently only one published study has assessed the influence of acute resistance exercise on glucose and insulin dynamics, and in that study data from men and women were pooled (11). AUC for insulin during an OGTT was lower on the day after a bout of resistance exercise compared with the no exercise condition in people either without diabetes (27 yr old) or with type 2 diabetes (53 yr old). The present study was apparently the first to provide data on acute resistance exercise and insulin sensitivity for women (when not pooled with data for men), regardless of age. However, several studies have described the influence of chronic resistance exercise and insulin action in older women. Among the published research that assessed glucoregulation in response to resistance-exercise training, only three studies were identified that reported women's data separately from men's data. Obese, postmenopausal women (50–65 yr old) underwent a 16-wk resistance-training program with a hyperglycemic clamp

performed before training and 24 h after the last training session (27). Glucose utilization (M) was not significantly different between the pretraining and posttraining condition, although M tended to be greater for the pretraining condition (27). Neither first-phase nor second-phase insulin secretion significantly differed between the pretraining versus posttraining hyperglycemic clamp. However, the 100- to 120-min insulin response was significantly lower (16%) in the posttrained compared with pretrained condition. The slope of the relationship between glucose utilization and insulin concentration in compartment 3 (considered to represent extracellular insulin) did not change in the posttrained versus pretrained hyperglycemic clamp. Thus, the resistance exercise had a modest effect on insulin dynamics, but the results do not suggest a marked improvement in insulin sensitivity. Focusing on the results from the placebo groups studied by Joseph et al. (18), insulin values for combined data from older women and men were lower during the posttraining (72 h after 12 wk of resistance training) compared with pretraining oral glucose tolerance test. Data were also presented separately for women and men: men had apparently lower insulin values posttraining whereas women did not. Ryan et al. (26) found that glucose disposal during a hyperinsulinemic-euglycemic clamp in a combined group of older (65–74 yr old) men and women tended to increase after 6 months of weight-lifting exercise. There was a trend for a relatively greater improvement in men (13%) compared with women (3%), but this difference did not attain statistical significance. Taken together with the results of the present study, it appears that resistance exercise (acute or chronic) does not markedly affect insulin sensitivity in nondiabetic, postmenopausal women. In contrast, endurance exercise training has been shown to improve insulin action in older women (7,20).

Acute eccentric exercise can result in muscle damage, which in turn has been linked to insulin resistance (19). In this context, it is possible that the resistance exercise performed in the present study may have induced muscle damage, which, in turn, might have prevented an increase in insulin sensitivity. However, plasma creatine kinase activity, an indirect indicator of muscle damage, did not differ between the no exercise and postexercise conditions. The creatine kinase results must be interpreted with caution because a direct assessment of muscle damage was not made using histochemical analysis of muscle biopsy samples.

In conclusion, no significant effects of resistance exercise were found for glucose measurements (basal, AUC, K_G , or S_G). These findings were as anticipated based on previous results with other modes of exercise and subject groups, but it was important to confirm this expectation in postmenopausal women after resistance exercise. Significantly higher plasma C-peptide values at several time points after exercise, together with unaltered insulin levels, raise the possibility of a small increase in insulin secretion that was balanced by enhanced insulin clearance. The absence of an acute resistance exercise effect on insulin sensitivity may be related to age and/or gender. Clarification of the precise causes for the apparent lack of improved insulin sensitivity

after resistance exercise by postmenopausal women is an important goal for further study.

We appreciate the technical assistance of Scott Baum in performance of glucose assay and the assistance of Angela Cirilli and

Jenelle Detheredge during the exercise protocols. This research was supported by an award from the Virginia Horne Henry Fund (GDC and AW).

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