Effects of resistance exercise on glucose tolerance in normal and glucose-intolerant subjects

JAMES D. FLUCKEY, MATTHEW S. HICKEY, JILL K. BRAMBRINK, KEVIN K. HART, KURT ALEXANDER, AND BRUCE W. CRAIG

Human Performance Laboratory of Ball State University, Ball Memorial Hospital, and Medical Consultants, Muncie, Indiana 47306

Fluckey, James D., Matthew S. Hickey, Jill K. Brambrink, Kevin K. Hart, Kurt Alexander, and Bruce W. Craig. Effects of resistance exercise on glucose tolerance in normal and glucose-intolerant subjects. J. Appl. Physiol. 77(3): 1087-1092, 1994. This study was conducted to determine whether improvements in glucose tolerance could be observed after a single bout of resistance exercise in young (27.1 ± 1.2 yr) control subjects, older (53.3 ± 1.7 yr) patients with non-insulin-dependent diabetes mellitus (NIDDM), and older (50.7 ± 1.9 yr) age-matched control subjects. Each subject was screened for fitness level and any contraindications to exercise before inclusion in the study. A 75-g oral glucose tolerance test was administered 2 wk after the subjects were screened, and the subjects were familiarized with the exercise equipment. The maximum weight that could be lifted with one repetition was determined on seven Nautilus machines that utilized the upper and lower body. After a 48-h rest period, a 3-set × 10-repetition protocol based on the subject’s one repetition maximum was completed by each participant on each machine. Eighteen hours after the lifting protocol, a second oral glucose tolerance test was administered. There was no change in the pre- to post-exercise glucose levels in any of the treatment groups, but the total insulin responses (area under the curve) of the young control and NIDDM groups were significantly lower after exercise: from 9.83 ± 0.8 × 10³ to 5.38 ± 0.65 × 10³ pM in the young control group and from 9.83 ± 1.95 × 10³ to 7.77 ± 1.50 × 10³ pM in the NIDDM group. The postexercise C-peptide levels were unchanged in all groups. The decrease in insulin 18 h after exercise in the young control and NIDDM groups, with no change in insulin secretion (C-peptide data), indicates an enhanced ability to clear insulin from the blood. Whether the removal is peripheral and/or hepatic cannot be determined from these data, but the results show that resistance exercise can influence insulin action without affecting glucose tolerance.

THE BENEFICIAL EFFECTS of aerobic exercise training have been well established (1-4) and include an improvement in glucose tolerance. Recent research has shown that a single bout of moderate- to high-intensity aerobic exercise can have similar effects (10, 11, 14, 24, 28). In fact, Mikines et al. (24) demonstrated that improved glucose tolerance after a training regimen is not greater than improvements after a single aerobic exercise bout, and they suggested that the improved glucose tolerance after aerobic exercise is somewhat independent of general improvements in physical condition.

Although few investigators have examined the effects of resistance forms of training on glucose tolerance, Miller et al. (25) demonstrated that 10 wk of strength training can improve glucose tolerance in young college-aged males. They attributed the improvement to an increased muscle mass. A well-designed resistance program utilizes upper and lower body muscle actions, which are not readily evident in running or cycling activities. Therefore, whereas running may allow for prolonged work in a given muscle group, resistance exercises incorporating muscle action in the upper and lower body work a greater amount of muscle mass. Craig et al. (7) demonstrated that glucose tolerance was improved in young and elderly men after a 12-wk resistance training program. However, the effects of an acute bout of resistance exercise on glucose tolerance have not been determined. Therefore it was the purpose of this study to establish the effects of a single bout of resistance exercise on glucose tolerance in normal and glucose-intolerant individuals.

METHODS

Subjects and Treatment Groups

Seventeen individuals not currently undergoing resistance training signed an informed consent form in accordance with the Human Subjects Institutional Review Board of Ball State University. Two groups were initially established: four female and three male patients with non-insulin-dependent diabetes mellitus (NIDDM) and four female and three male young controls. The diabetic condition of the NIDDM group had been defined by their personal physician and consisted of fasted baseline serum glucose >5.56 mM and a response to a 75 g oral glucose load of >11.1 mM at some point during a 3-h test (National Diabetes Data Group guidelines). The mean age of the NIDDM group was 53.3 ± 1.7 yr, and the time since diagnosis was 2.8 yr. The NIDDM group was prescreened for retinopathy and cardiovascular complications by an attending physician and performed a maximal exercise test to detect contraindications to exercise (American College of Sports Medicine guidelines) before inclusion in the study. An age-matched older control group consisting of three male and three female subjects was established after the NIDDM group was tested. Unfortunately only the male subjects completed the entire protocol. The young control group (27.1 ± 1.2 yr of age) was recruited from the student population of Ball State University. They were screened for contraindications to exercise with a maximal exercise test before inclusion in the study. Because it has been shown that the menstrual cycle can influence glucose tolerance (6) in young women, all young females were tested during the follicular phase of their cycle to ensure uniformity between trials. The subject characteristics and fitness parameters for all groups are given in Table 1.

Pretrial Testing

Two weeks before the start of the experimental protocol, each subject reported to the laboratory in the morning after a 12-h fast for measurement of body composition and maximal aerobic capacity (VO₂max). Percent body fat was determined with a seven-site skinfold method (19) with Lange calipers by a...
TABLE 1. Subject characteristics and glycosylated hemoglobin values

<table>
<thead>
<tr>
<th></th>
<th>Young Control (n = 7)</th>
<th>NIDDM (n = 7)</th>
<th>Age Matched Control (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, yr</td>
<td>27.1±1.2</td>
<td>53.3±1.7</td>
<td>50.7±1.9</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>67.5±5.4*</td>
<td>83.3±7.7</td>
<td>81.7±2.1</td>
</tr>
<tr>
<td>%Body fat</td>
<td>18.9±0.9</td>
<td>22.6±2.3†</td>
<td>19.3±3.0</td>
</tr>
<tr>
<td>LBM, kg</td>
<td>54.5±4.2*</td>
<td>64.0±5.1</td>
<td>65.9±2.1</td>
</tr>
<tr>
<td>V0\textsubscript{2}, ml.kg\textsuperscript{-1}.min\textsuperscript{-1}</td>
<td>44.9±9.2†</td>
<td>25.6±2.5</td>
<td>25.5±2.0</td>
</tr>
<tr>
<td>Gly Hb, %</td>
<td>7.5±0.1</td>
<td>10.8±0.7†</td>
<td>8.5±0.01</td>
</tr>
<tr>
<td>Pre</td>
<td>7.5±0.1</td>
<td>10.6±1.1†</td>
<td>7.8±0.39</td>
</tr>
<tr>
<td>Post</td>
<td>7.5±0.1</td>
<td>10.6±1.1†</td>
<td>7.8±0.39</td>
</tr>
</tbody>
</table>

Values are means ± SE. NIDDM, non-insulin-dependent diabetes mellitus; LBM, lean body mass; Gly Hb, glycosylated hemoglobin; V0\textsubscript{2}, O\textsubscript{2} uptake; Pre, preexercise; Post, postexercise. * Significantly lower than NIDDM and age-matched control. † Significantly higher than age-matched control.

Experimental Design

Two weeks after the subjects completed their graded exercise test, they were administered a 75-g oral glucose tolerance test (OGTT) to determine their ability to assimilate glucose. After these measurements, the subjects were familiarized with the exercise testing equipment and the procedures for determining their V0\textsubscript{2} max. They then completed a graded exercise test, based on a modified Naughton-Balke treadmill protocol (16), to determine V0\textsubscript{2} max with use of on-line gas analysis. The treadmill protocol was conducted at 3.0 mph with a 2.5% increase in grade every 2 min for the NIDDM group and included a 12-lead electrocardiogram that was monitored by a physician throughout the test. Termination of the test occurred when increases in O\textsubscript{2} uptake were no longer observed or when the participants stopped of their own volition. The treadmill test for the control groups was the same, except the initial speed of the treadmill was higher.

Exercise Protocol

Forty-eight hours after the preexercise OGTT, each subject was familiarized with the resistance equipment, and each lifting occasion. For familiarization purposes, the weight pins were removed from the machines to eliminate resistance during this learning experience. The participants were asked to complete one set of 15 repetitions on each machine and were instructed on the appropriate lifting and breathing techniques. After 48 h the subjects returned to the weight room for determination of their 1 RM on each of the machines. A warm-up set of 10–12 repetitions was completed and was followed by a 1-min rest period. On the basis of the relative effort of the warm up, the investigator incrementally increased the resistance of the machine until the subject’s 1 RM was reached. Only one repetition was completed on each attempt, and weight was added in 4.5-kg increments until the subject was unable to complete the full range of the lift. The 1 RM was reached in three to seven attempts.

The lifting protocol occurred 72 h after the 1 RM determination. The participants reported to the laboratory at 12:00 P.M. and completed resistance exercises based on a Delorme-Watkins lifting protocol (9), which consisted of three sets of lifts. Briefly, the weight required for a 10 RM was calculated from the measured 1 RM for each machine (10 RM = 75% of 1 RM). In the first two sets of lifts, the subjects utilized 50 and 75% of the calculated 10 RM and used the 10 RM for the third set. All three sets were completed on each machine before the subjects moved to the next, and the machines were used in the order listed previously. The subjects rested for 70 s between sets and for 2 min between lifting stations. The subjects were required to complete 10 repetitions per set but could stop at volitional fatigue or if the lifting technique was compromised. The lifting data are provided in Table 2.

Pre- and Post-OGTT

The subjects reported to the laboratory at 6:30 A.M. after a 12-h fast. A 22-gauge Aquavene (Baxter, Deerfield, IL) catheter was inserted in an antecubital vein by a trained phlebotomist. A three-way stopcock for blood sampling was attached to the catheter, and patency was maintained with a saline drip. Once the catheter was established, the subjects were seated in a semirecumbent position. After 30 min, a 5-ml blood sample was taken and a D-glucose drink (75 g) was administered. Additional 5-ml blood samples were taken 15, 30, 45, 60, 90, 120, and 180 min after consumption of the glucose drink.

Total Response Calculations

The total response for the pre- and postexercise glucose, insulin, and C-peptide represents the area under the curve for each of these measurements. The areas were determined using a best-fit polynomial regression analysis and calculated integrals.

Serum Analysis

Upon removal, the blood was centrifuged at 1,160 g for 20 min, and the serum was removed and stored at −20°C until it was analyzed for glucose, insulin, and C-peptide. Plasma volume shifts were calculated as described by Hill and Costill (19), and the hormonal data were adjusted accordingly. Serum glucose was measured spectrophotometrically using hexokinase kits (Sigma Chemical, St. Louis, MO). The insulin and C-peptide levels of the samples were measured using radioimmunoassay double-antibody kits (Diagnostic Products, Webster, TX). The intra-assay coefficient of variation was 2.8% for insulin and 4.8% for C-peptide. Kits (Sigma Chemical) were used to measure the total number of repetitions, total weight lifted, and total volume lifted.

TABLE 2. Total number of repetitions, total weight lifted, and total volume lifted

<table>
<thead>
<tr>
<th></th>
<th>Young Control (n = 7)</th>
<th>NIDDM (n = 7)</th>
<th>Age-Matched Control (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Two repetitions</td>
<td>28.3±0.3</td>
<td>28.3±0.3</td>
<td>28.1±0.1</td>
</tr>
<tr>
<td>TW</td>
<td>6,630±680</td>
<td>6,045±521</td>
<td>6,690±361</td>
</tr>
<tr>
<td>TW/LBM</td>
<td>137.3±7.2*</td>
<td>96.6±6.2</td>
<td>101.4±5.1</td>
</tr>
</tbody>
</table>

Values are means ± SE. TW, total volume lifted (kg); LBM, lean body mass (kg). * Significantly higher than NIDDM and age-matched control.
sure glycosylated hemoglobin spectrophotometrically in the pre-OGTT blood sample of each OGTT after column filtration and separation.

Statistical Analysis

The group and treatment differences were analyzed with a two-way analysis of variance (ANOVA). Pre- to postexercise differences were tested with a one-way ANOVA. Significant differences were analyzed using a Scheffé F test post hoc analysis of significance. Significance was set at P < 0.05.

RESULTS

Subject Characteristics

The body weight, LBM, and $\bar{V}O_2\text{max}$ were significantly lower in the young control group than in the NIDDM and age-matched control groups (Table 1), and except for lower percent body fat, the characteristics of the age-matched control and NIDDM groups were nearly identical. The pre- and postexercise percent glycosylated hemoglobin was significantly lower in the young control than in the NIDDM group. Glycosylated hemoglobin was slightly higher in the age-matched control than in the young control group but still significantly lower than in the NIDDM group.

Performance Data

The total number of repetitions completed at each exercise station was relatively constant, so only the mean ± SE of all stations is given in Table 2. All subjects were able to complete the 10 repetition requirement in the first two sets but could complete only ~8 lifts in the last set. A comparison of the total weight (repetitions x sets x mass) lifted per exercise session showed that the NIDDM group was able to lift less weight than either of the control groups. When the total weight lifted was expressed as a function of the LBM (TW/LBM), it was evident that the young control group was able to lift significantly more weight than the NIDDM or age-matched control group.

Glucose Tolerance Data

Glucose response. The resting glucose levels of the young and age-matched control groups were unchanged by the exercise protocol, and the pre- and postexercise levels were significantly lower than those attained by the NIDDM group (Table 3). In response to a 75-g load, the young control group cleared significantly more glucose than the NIDDM group throughout the OGTT, and glucose clearance in the NIDDM group was significantly different from that in the age-matched control group at 45 and 60 min. However, a single bout of resistance exercise did not alter the glucose response of the young control group. The NIDDM group showed a delay in the time required to reach a peak in glucose levels in comparison to the control groups and maintained elevated levels of glucose throughout the rest of the OGTT. Exercise did not alter the glucose response of the NIDDM group. The response of the age-matched control group was well within the normal range but indicated that this group was more glucose intolerant than the young control group. As in the young control and NIDDM groups, no exercise effect was demonstrated in the age-matched control group.

Insulin response. The resting insulin levels were significantly higher in the NIDDM group than in either of the control groups before and after exercise (Table 4). The insulin response of the young control group peaked 45 min before exercise and was significantly different from the response of the NIDDM group at 45, 120, and 180 min. After exercise the total insulin levels were significantly lower in the young control group than in the NIDDM group (from 6.93 ± 0.80 x 10^3 to 5.38 ± 0.65 x 10^3 pM), but the time to peak insulin was unchanged. The peak insulin response of the NIDDM group before exercise was 120 min but shifted to 90 min after the exercise session. The total insulin response was significantly lower after exercise in the NIDDM group (from 9.83 ± 1.95 x 10^3 to 7.77 ± 1.50 x 10^3 pM) but was not significantly different from the response of the young control group. The insulin response was lowest in the age-matched control group and significantly different from the response of the NIDDM group between 120 and 180 min. Unlike the other two groups, exercise had no effect on the total insulin response in the age-matched control group.

C-peptide response. The resting C-peptide levels were significantly lower in the control groups than in the NIDDM group before and after exercise (Table 5). The only significant difference between the control groups and the NIDDM group occurred at 180 min, when the response was significantly lower in the control groups than in the NIDDM group. The peak level of C-peptide in the young control group occurred at 60 min both before

### Table 3. Glucose response to oral glucose tolerance test

<table>
<thead>
<tr>
<th>Time, min</th>
<th>Young Control* (n = 7)</th>
<th>NIDDM (n = 7)</th>
<th>Age-Matched Control* (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre Post</td>
<td>Pre Post</td>
<td>Pre Post</td>
</tr>
<tr>
<td>0</td>
<td>4.7±0.2 5.0±0.1</td>
<td>7.9±0.9 8.1±0.8</td>
<td>4.8±0.1</td>
</tr>
<tr>
<td>15</td>
<td>5.7±0.4 6.2±0.2</td>
<td>9.0±1.0 9.6±1.1</td>
<td>6.1±0.9</td>
</tr>
<tr>
<td>30</td>
<td>6.7±0.4 6.7±0.3</td>
<td>9.1±1.8 11.4±1.3</td>
<td>8.4±0.8</td>
</tr>
<tr>
<td>45</td>
<td>6.2±1.1 7.2±0.4</td>
<td>10.0±2.2 12.4±1.4</td>
<td>9.8±0.8</td>
</tr>
<tr>
<td>60</td>
<td>6.1±0.7 5.8±1.1</td>
<td>12.6±1.3 13.3±1.6</td>
<td>9.1±0.3</td>
</tr>
<tr>
<td>90</td>
<td>5.4±0.7 4.4±1.0</td>
<td>14.0±1.3 14.7±1.7</td>
<td>8.0±0.5</td>
</tr>
<tr>
<td>120</td>
<td>5.1±0.7 4.6±0.9</td>
<td>14.0±1.5 14.1±1.8</td>
<td>5.2±1.1</td>
</tr>
<tr>
<td>180</td>
<td>4.3±0.4 4.0±0.8</td>
<td>11.8±1.6 11.4±1.6</td>
<td>3.8±0.5</td>
</tr>
<tr>
<td>Area†</td>
<td>1,002.11±94.5</td>
<td>2,171.6±222.6</td>
<td>1,131.7±50.1</td>
</tr>
</tbody>
</table>

Values are means ± SE in mM. * Pre and Post values and total significantly lower than NIDDM. † Area under the curve.
and after exercise. The total C-peptide response after exercise was well above the preexercise level in the young control group but did not represent a significant rise. The peak C-peptide response of the NIDDM group occurred at 120 min before exercise but 15 min earlier (90-min point) after exercise. The total C-peptide was higher in the age-matched older control than in the NIDDM group than in either of the control groups before and after exercise but did not represent a significant difference. The total C-peptide response to exercise was lower in the age-matched older control than in the young control or NIDDM group but was not significantly different.

**DISCUSSION**

The significant decline in insulin response 18 h after exercise in the young control and NIDDM groups, coupled with their C-peptide data, demonstrates an increased insulin clearance after resistance exercise in these individuals. This increased clearance of insulin from the blood, however, did not translate into improved glucose tolerance 18 h after exercise. Prior research dealing with exercise training in animal (1) and human (2–4) models has also shown a drop in insulin levels with little or no change in blood glucose levels. The most common interpretation of these results is that exercise increases insulin sensitivity, because less hormone is required to clear glucose after training. Increased insulin sensitivity was originally linked to an increase in insulin receptor number (26). Prior research has shown that exercise training can significantly elevate the insulin receptor number in adipose tissue (8), but there is no experimental evidence to show that a single bout of exercise can alter insulin receptor number or activity. Bonen et al. (6) showed a decrease in insulin receptor number in muscle when exercise intensity was >69% of the subject’s VO2 max, and it is possible that the insulin decrease observed in this study represents an increased insulin turnover. However, it seems highly unlikely that an insulin turnover would still be evident 18 h after exercise.

A more current definition (31) of increased insulin sensitivity indicates that it is the insulin concentration required to shift the one-half maximal insulin response to the left. This definition accounts for receptor and postreceptor control (28-30) and fits well with the work of Devlin et al. (10, 11), who demonstrated a significant increase in glucose disposal and a significant decrease in insulin response 18 h after exercise in subjects who performed a single aerobic exercise session to exhaustion at 85% of their VO2 max. Some of the animal research conducted over the last few years (13, 17, 18) suggests that the continued glucose uptake observed by Devlin et al. (10, 11) is linked to an exercise-induced reduction in muscle glycogen that accompanies high-intensity activity. However, the animal work of Wallberg-Henriksson et al. (31) and Young et al. (35) indicates that the prolonged aftereffects of exercise on glucose uptake are independent of glycogen resynthesis. This has been supported in human studies (23, 34). Mikines et al. (23) observed an increase in insulin responsiveness of whole body glucose uptake during a hyperinsulinemic euglycemic clamp after exercise that did not reflect an increase in glycogen deposition. In another study, using high- (80% VO2 max) and low-

**TABLE 4. Insulin response to oral glucose tolerance test**

<table>
<thead>
<tr>
<th>Time, min</th>
<th>Young Control* (n = 7)</th>
<th>NIDDM (n = 7)</th>
<th>Age-Matched Control* (n = 3)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Pre</td>
<td>Post</td>
<td>Pre</td>
</tr>
<tr>
<td>0</td>
<td>35.4±5.8*</td>
<td>24.9±4.6*</td>
<td>87.8±19.6</td>
</tr>
<tr>
<td>15</td>
<td>157.5±31.8</td>
<td>150.8±27.6</td>
<td>140.4±30.5</td>
</tr>
<tr>
<td>30</td>
<td>269.9±36.2</td>
<td>229.1±30.7</td>
<td>273.8±37.0</td>
</tr>
<tr>
<td>45</td>
<td>421.4±51.1*</td>
<td>397.6±57.9*</td>
<td>188.1±20.2</td>
</tr>
<tr>
<td>60</td>
<td>379.0±65.7</td>
<td>361.2±47.3</td>
<td>313.1±70.0</td>
</tr>
<tr>
<td>90</td>
<td>286.8±42.6</td>
<td>174.4±33.5</td>
<td>415.5±104.9</td>
</tr>
<tr>
<td>120</td>
<td>218.7±26.2*</td>
<td>141.0±29.0*</td>
<td>449.6±105.9</td>
</tr>
<tr>
<td>180</td>
<td>84.8±12.8*</td>
<td>51.6±18.0*</td>
<td>262.5±61.9</td>
</tr>
<tr>
<td>Area‡</td>
<td>6.30±0.80†</td>
<td>5.38±0.65</td>
<td>9.83±1.95†</td>
</tr>
</tbody>
</table>

Values are means ± SE in pM. * Significantly different from NIDDM. † Significantly higher than Post. ‡ Area under the curve (pM × 10).
pared the glucose tolerance of well-trained athletes and the present findings. However, the work of King et al. (33) support physically trained rats after insulin infusion (33) and in trained athletes after glucose administration (32) and in the data from these studies (23, 34) and the results presented here support the concept that a single bout of exercise can affect insulin action without influencing glucose tolerance.

Reductions in postexercise insulin in endurance-trained athletes after glucose administration (32) and in physically trained rats after insulin infusion (33) support the present findings. However, the work of King et al. (20) provides evidence that reduced insulin secretion, but not clearance, is responsible for lower postexercise insul in responses. Using a hyperglycemic clamp, they found lower insulin and C-peptide levels in subjects who exercised on the day before they were tested. Mikes et al. (24) also demonstrated lower insulin and C-peptide levels after exercise, regardless of whether the exercise was 1 h or 1 day before the hyperglycemic clamp. These two studies give an effective argument against insulin clearance but are based on the significant reduction in C-peptide observed after exercise. Insulin and C-peptide are secreted from the pancreas in equimolar amounts (15), but only insulin is extracted by the liver (22), so plasma C-peptide is a good indicator of B-cell activity. Therefore the lower C-peptide release after exercise observed by King et al. and Mikes et al. is an indication of a decrease in insulin secretion. By use of the same logic, reduced plasma insulin in conjunction with unaltered C-peptide levels suggests that insulin secretion was not affected by the exercise protocol used in the present study.

To the best of our knowledge, this is the first study to show an increase in insulin clearance after a single exercise session, but the data are supported by the work of Oshida et al. (27), who compared the metabolic clearance of glucose and insulin before, during, and after 1 yr of training. Using a euglycemic clamp technique, they measured insulin action and demonstrated that steady-state insulin levels were significantly decreased after 1 yr of training. The metabolic clearance rate of insulin, on the other hand, was increased by 87% after training. Unfortunately, Oshida et al. did not measure C-peptide responses. Working with young subjects, Wirth et al. (32) also observed a blunted insulin response to a glucose load and attributed it to enhanced insulin clearance. They compared the glucose tolerance response of trained and untrained individuals and found the insulin response to be significantly lower in the trained than in the untrained subjects, whereas the C-peptide response of the two groups was identical. They concluded that the reduced insulin response was due to an increase in peripheral clearance of insulin in the trained subjects.

At odds with the data presented here and by others (27, 32), Kirwan et al. (21) found significantly higher C-peptide levels 12 h after a single bout of exhaustive exercise in untrained human subjects. Using a hyperglycemic clamp, they showed significantly higher insulin levels in the early phase of the clamp (0—10 min) but no change in insulin in the later phase (15—180 min). On the basis of the elevated C-peptide output, they concluded that insulin secretion was elevated during the later stages of the clamp but that insulin levels remained unchanged because of increased hepatic clearance. The results presented in the present study show that resistance exercise can reduce the insulin response to a glucose load given 18 h after exercise and that this effect is not due to alterations in insulin secretion. Unfortunately, even through these data clearly demonstrate that insulin clearance has been enhanced by resistance exercise, they do not indicate how insulin is removed from the system.

Although the young control and NIDDM groups showed a significant decrease in insulin response after exercise, the insulin levels of the age-matched older control group did not change. The unresponsiveness of this group is puzzling but could represent a lack of conditioning; VO_{2\text{max}} was significantly lower in the age-matched than in the younger control group (Table 1). In a recent study (20), no effect on insulin clearance could be demonstrated with a euglycemic clamp in subjects who had undergone 14 days of inactivity. However, if conditioning were the answer, similar responses would be expected in the NIDDM group, in that VO_{2\text{max}} was lower in the NIDDM than in the young control group. In addition, it has been suggested that improvements in glucose tolerance are independent of physical condition (24). At present, we cannot explain why the age-matched control group did not respond to exercise.

In summary, the data presented here indicate that a single resistive exercise session can significantly enhance insulin clearance in young control and NIDDM subjects and that the effects are still evident 18 h after exercise. This increased clearance is not accompanied by an improvement in glucose tolerance and provides evidence that resistance exercise can influence insulin action independently of glucose tolerance.

The authors thank Mitch Whaley for assistance in recruiting subjects for the study, Joseph Russo for technical assistance in data collection, and the Nautilus Corporation for funding the study.

Present addresses: J. D. Fluckey, Noll Laboratory, Pennsylvania State University, University Park, PA 16802; M. Hickey, Human Performance Laboratory, East Carolina University, Greenville, NC 27858-4353; K. Hart, Cardiology Dept., Tulane University, New Orleans, LA 70118.

Address for reprint requests: B. W. Craig, Human Performance Laboratory, Ball State University, Muncie, IN 47306.

Received 10 May 1993; accepted in final form 21 March 1994.

REFERENCES


