Intravenous Glucose Tolerance Test–Derived Glucose Effectiveness in Strength-Trained Humans

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The effect of long-term strenuous resistance training on glucose effectiveness (SG) was examined by comparing 11 strength-trained and 20 sedentary males by a minimal model approach. Lean body mass (LBM) was measured by hydrostatic weighing. The LBM in strength-trained subjects (68.7 ± 3.1 kg) was significantly larger than in sedentary subjects (65.6 ± 1.2 kg, P < .01). The glucose disappearance constant ([KG] 3.07 ± 0.45% min−1) and insulin sensitivity (SI) (17.5 ± 2.0 x 10−5 · min−1 · pmol/L−1) in strength-trained subjects were significantly higher than in sedentary subjects (2.06% ± 0.14% · min−1 and 10.3 ± 1.2 x 10−5 · min−1 · pmol/L−1, P < .05). SG in strength-trained subjects (0.024 ± 0.003 min−1) was significantly higher than in sedentary subjects (0.018 ± 0.001 min−1, P < .05). These results thus suggest that the improved glucose tolerance in strength-trained subjects was due to increased SG and SI.

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SUBJECTS AND METHODS

Subjects

Eleven strength-trained and 20 sedentary males participated in the study after provision of informed consent. Strength-trained subjects were engaged in weight lifting and/or resistance exercise more than five times per week for at least 1 year. None of the subjects had a family history of diabetes or hypertension. All had a normal response to a 75-g oral glucose tolerance test. The subjects' physical characteristics are listed in Table 1. Body composition was measured by hydrostatic weighing and corrected for residual lung volume.9 Waist girth was measured at the narrowest part of the torso while the subject was standing, and hip circumference was measured at the level of the greatest gluteal protuberance. All subjects participated in an incremental exercise test on a cycle ergometer. The work load was increased by 20 W every 4 minutes, and maximal oxygen uptake (VO\textsubscript{2} max) was determined by the leveling-off criterion,10 or greater than 8 mmol/L blood lactate. The exercise test was performed at least 1 week before the intravenous glucose tolerance test (IVGTT). Strength-trained subjects refrained from any type of exercise for 2 days before the IVGTT. On the day before the IVGTT, all subjects were provided with a supper meal containing greater than 140 g carbohydrate, greater than 30 g fat, and greater than 33 g protein.

IVGTT

The subjects reported to the laboratory at 8 AM, and an IVGTT was performed in the reclining position at 9 AM as described previously.1,11 In brief, baseline samples for glucose and insulin assays were obtained at −20, −10, and −3 minutes. At time 0, glucose (300 mg/kg body weight) was administered intravenously within 2 minutes, and subsequent blood samples were taken at 3, 4, 5, 6, 8, 10, 12, 14, 16, 19, 22, 24, 26, 28, 30, 33, 36, 40, 50, 60, 70, 80, 100, 120, 140, 160, and 180 minutes from the contralateral antecubital vein. An additional infusion of insulin (Humalin; Shionogi, Osaka, Japan) was administered (20 mU/kg) via the antecubital vein from 20 to 25 minutes after administration of glucose. The glucose disappearance constant (KG value) and the area under the insulin curve between 0 and 20 minutes after administration of glucose were calculated as previously described.1,11 SI and SG were estimated by the minimal model approach.12-14 In this analysis, fluctuations in circulating glucose levels over time are described by the following differential equations: dG(t)/dt = −p1(G(t) − Gb) − X(t)G(t) and dX(t)/dt = −p2X(t) + p3[I(t) − Ib], where G(t) is the plasma glucose concentration; I(t) is the plasma insulin concentration; G\textsubscript{b} and I\textsubscript{b} are baseline concentrations; p1, p2, and p3 are model parameters; and X(t) is the time course of peripheral insulin effects in minutes. Parameter $p1$ represents the effect of glucose per se at basal insulin to enhance net glucose disposal, and is known as SG. SG consists of a
GLUCOSE EFFECTIVENESS IN STRENGTH-TRAINED HUMANS

Table 1. Subject Characteristics

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Sedentary</th>
<th>Trained</th>
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</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>23 ± 1</td>
<td>20 ± 1</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.73 ± 0.02</td>
<td>1.70 ± 0.02</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>65.8 ± 1.6</td>
<td>73.7 ± 3.6*</td>
</tr>
<tr>
<td>WHR</td>
<td>0.84 ± 0.01</td>
<td>0.81 ± 0.01</td>
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<tr>
<td>Body mass index (kg · m⁻²)</td>
<td>22.0 ± 0.5</td>
<td>25.4 ± 0.71</td>
</tr>
<tr>
<td>LBM (kg)</td>
<td>56.6 ± 1.2</td>
<td>65.7 ± 3.11</td>
</tr>
<tr>
<td>Body fat (%)</td>
<td>14.2 ± 1.3</td>
<td>9.0 ± 0.9*</td>
</tr>
<tr>
<td>k/o₂max (mL · kg⁻¹ · min⁻¹)</td>
<td>43.2 ± 1.8</td>
<td>44.4 ± 2.0</td>
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</table>

NOTE. Values are the mean ± SE.

*P < .05.

A non-insulin-dependent component and a basal insulin component. The basal insulin component of SG (BIE) can be calculated as the product of Ib and SI: BIE = Ib × SI. Therefore, the contribution of the non-insulin-dependent component (SG at zero insulin [GEZI]) is the difference between total SG and BIE: GEZI = SG − (lb × SI). The ratio p3/p2 defines the SI index, which represents the increase in the net glucose disappearance rate, which in turn depends on the increase in insulin above basal. The minimal model program was written in Pascal (Borland International, Scotts Valley, CA) on a Macintosh IIcx computer (Apple Computer, Cupertino, CA) as described previously.

Analytical Methods

The glucose oxidase method was used to measure plasma glucose concentrations in triplicate (Glucose B-test; Wako Pure Chemical, Osaka, Japan). The measurement error for glucose was assumed to be "white," with a Gaussian of zero mean and a coefficient of variation of 1.5%. The immunoreactive insulin level was measured in duplicate using a Phadeseph insulin radioimmunoassay kit (Shionogi, Osaka, Japan).

Statistics

The statistical comparison between strength-trained and sedentary subjects was performed by Student’s t test. Differences were considered statistically significant at P less than .05.

RESULTS

Body Composition and Vo₂max

LBM in strength-trained subjects was larger than in sedentary subjects. The percent fat in strength-trained subjects was lower than in sedentary subjects. Vo₂max was similar between strength-trained and sedentary subjects. The percent body fat and the waist to hip ratio (WHR) were slightly lower in strength-trained subjects than in sedentary subjects, but the difference was not statistically significant (Table 1).

IVGTT

Basal glucose and insulin concentrations were similar in strength-trained and sedentary subjects. Plasma glucose and insulin concentrations during the IVGTT are illustrated in Fig 1. KG was significantly higher in strength-trained subjects versus sedentary subjects. The integrated area of plasma insulin above the basal level during the first 20 minutes of the IVGTT was smaller in strength-trained subjects versus sedentary subjects, but the difference was not statistically significant (Table 2).

Minimal Model Analysis

SI was significantly higher in strength-trained subjects than in sedentary subjects (Table 2). SG in strength-trained subjects was significantly higher than in sedentary subjects. However, the non-insulin-dependent component of SG (GEZI) was similar between strength-trained and sedentary subjects. The insulin-dependent component of SG (BIE) was higher in strength-trained subjects versus sedentary subjects. SG was not found to be significantly associated with LBM (r = .05, P = .91) or body fat (r = −.12, P = .61).

DISCUSSION

In this study, we used the minimal model approach to assess the effect of resistance training on SG. Strength-trained subjects had a higher SG than sedentary subjects. Our data also
The role of LBM as a determinant of glucose effectiveness has been suggested by Maki and Abraira\(^4\) from several lines of evidence. Chronic undernutrition may be responsible, in part, for the depression of SG in anorexia nervosa\(^3\) and hepatic cirrhosis. Cirrhotic patients also demonstrated a significantly reduced 24-hour urinary creatine excretion normalized to height, and the decrease in SG was strongly correlated with the creatine excretion to height ratio, suggesting that reduced skeletal muscle mass may be responsible for the depression of SG in hepatic cirrhosis. Consistently, an increase in LBM due to resistance training was accompanied by an increase in glucose effectiveness in the present study, although contradictory results\(^8\) exist, as already discussed. However, the increase in SG was not greater in strength-trained subjects versus endurance-trained subjects with normal LBM. SG estimated 16 hours and 1 week after the endurance training bout was 0.028 ± 0.003 and 0.030 ± 0.004 min\(^{-1}\), respectively. Furthermore, the variation in SG values among control subjects was not correlated with LBM, at least in our 29 subjects (R\(^2\) = .001, P = .91; Y. Higaki, J. Fujitani, T. Kagawa, et al, unpublished data, September 1997). Taken together, factors other than skeletal muscle mass and LBM exist as a major determinant of SG in the general population and athletes.

We have shown that SG in endurance-trained subjects with a lower percentage of body fat (10.3% ± 0.8%) was higher than in sedentary subjects.\(^1\) In this study, the percent body fat in strength-trained subjects (9.0% ± 0.9%) was also lower than in sedentary subjects. Therefore, reduced body fat might reflect an improvement of SG, as well as SI. However, there is no significant correlation between SG and body fat. Further, Clausen et al\(^2\) observed in 380 young healthy caucasians that glucose effectiveness was not significantly associated with any measure of body fat. These results suggest that low body fat may not be a determinant of glucose effectiveness.

In conclusion, we have shown that resistance training induces an increase in muscle mass and results in an enhancement of SG and SI.

ACKNOWLEDGMENT

We are grateful to Shionogi Biomedical Laboratory, Osaka, Japan, for assistance in this study.

REFERENCES


