INSULIN SENSITIVITY AFTER MAXIMAL AND ENDURANCE RESISTANCE TRAINING

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ABSTRACT

Hansen, E, Landstad, BJ, Gundersen, KT, Torjesen, PA, and Svebak, S. Insulin sensitivity after maximal and endurance resistance training. J Strength Cond Res 26(2): 327–334, 2012—The purpose of the study was to compare the effects of maximal resistance training (MRT) vs. endurance resistance training (ERT) on improvements in insulin levels and glucose tolerance in overweight individuals at risk of developing type 2 diabetes. Eighteen participants with baseline values suggesting impaired glucose tolerance were randomly assigned to 1 of 2 groups. Group 1 engaged in supervised MRT (Bernstein inverted pyramid system: 5 × 3–4, 60–85% 1 repetition maximum [1RM]), 3 d wk⁻¹ over 4 months, whereas members of group 2 acted as controls. Later, group 2 engaged in supervised ERT (3 × 12–15, 45–65% 1RM), 3 d wk⁻¹ over a 4 month period with the 2 prebaselines as controls. Both interventions consisted of 8 exercises that included the entire body. Glucose (fasting and 2-hour test), insulin and C-peptide measures were assessed from pre to post in both groups. The MRT led to reduced blood levels of 2-hour glucose (p = 0.044) and fasting C-peptide (p = 0.023) and decreased insulin resistance (p = 0.040). The ERT caused a significant reduction in the blood levels of insulin (p = 0.023) and concomitant positive effects on % insulin sensitivity (p = 0.054) and beta-cell function (p = 0.020). The findings indicate that both MRT and ERT lead to decreased insulin resistance in people with a risk of developing type 2 diabetes; MRT led to a greater increase in glucose uptake capacity (in muscles), whereas ERT led to greater insulin sensitivity, supporting the recommendation of both MRT and ERT as primary intervention approaches for individuals at a risk of developing type 2 diabetes.

KEY WORDS beta-cell function, exercise, glucose tolerance, IGT, insulin resistance, strength training

INTRODUCTION

Impaired glucose tolerance (IGT) presents an increased risk of developing type 2 diabetes, which in turn is a major risk factor in the development of metabolic syndrome and related hypertension and overweight (9,23). This risk chain presents major challenges for modern public health management (37). Added to this, IGT and obesity are important risk factors for cardiovascular disease (37). Diagnosis of type 2 diabetes and the identification of those who are at the risk of developing the disease are based on laboratory tests indicating glucose intolerance. Exercise may be helpful for people with type 2 diabetes and can postpone the development of type 2 diabetes in those at risk (5,7,13,27). Decreased 2-hour blood glucose and fasting insulin levels and a reduced risk of having IGT and type 2 diabetes have been shown to be associated with high levels of leisure time physical activity. These effects appear to be independent of the degree of abdominal obesity (8,31).

Lifestyle intervention in IGT seems to be at least as effective as drug treatment (14). Some studies show an ~50% reduction in risk of developing diabetes following lifestyle changes among people with IGT (26,35), and it is well known that daily physical activity is recommended for delaying or avoiding the development of type 2 diabetes (20,21,41). Several studies have reported improvement in glucose tolerance after resistance training (16,32,33,34), whereas others have not (24,25,40).

The uptake of glucose by muscle cells is stimulated and controlled by insulin and muscle contractions (38). The effect of exercise is increased blood flow to working muscles primarily resulting from an immediate need for glucose uptake and utilization of glucose (30). Glucose availability is important in itself, although the transport of the substrate across the cell wall is even more important. Furthermore, the
glucose protein IV (GLUT4) transports the substrate across the cell membrane and seems to be a crucial agent in glucose uptake. Resistance training has an impact on muscle mass by increasing muscle volume. Consequently, the ability of the cells to accrue muscle protein is improved through hypertrophy and the related increased rate of protein synthesis.

Conventional aerobic endurance resistance programs (2,4) include multiple bouts of low work-load training with intervals of rest in between to permit a slower heart rate and to allow muscle cells to be refilled with oxygen. To build increased muscle mass, however, it is important to induce anaerobic exercise conditions. Optimal intensities for maximal resistance training (MRT) involve a high work load with few repetitions and few bouts of exercises (2,4). The Bernstein hypothesis explains how strenuous (maximal) resistance exercise improves insulin sensitivity through anaerobic conditions in the muscle cells. The inverted pyramid resistance training system without pauses or rest between repetitions is probably the most efficient training method (3).

In a review study by Gordon et al. (15), the authors claim that supervised resistance training improves glycemic control and insulin sensitivity in adults with type 2 diabetes. Results from another study indicate that resistance exercise is effective in acutely enhancing insulin sensitivity and regulating blood glucose levels in individuals with impaired fasting glucose (4). Contradictory findings call for further research to explore the mechanism(s) behind improvements in glucose tolerance after resistance training and to determine the optimal frequency, intensity, and duration of such training. Although significant improvements in insulin sensitivity in healthy individuals have been reported when resistance training was carried out 3 times a week or more and, in 1 study, only 1 session with resistance exercise improved whole-body 24-hour insulin sensitivity in healthy men (19), and these effects appear to be different in people with diabetes (10). Thus, this study focused upon overweight individuals at the risk of developing type 2 diabetes who had not yet developed the disease and aimed to compare the effects of MRT with endurance resistance training (ERT) on improvements in insulin levels (sensitivity) and glucose tolerance.

**METHODS**

**Experimental Approach to the Problem**

The sample consisted of 4 men and 14 women. The participants were randomized into 2 groups with 9 individuals in each (N = 18). Two men were randomized into each group, although the study does not analyze possible gender differences because of the small number of men. The design included 2 interventions of resistance training with controls: Group 1 performed 4 months of maximal intensity resistance training (MRT) based on the Bernstein inverted pyramid training model (group 1), and the reference group subsequently performed 4 months of conventional low-intensity ERT (group 2). Group 2 participants acted as their own control using 2 pretraining baselines. Fasting and 2-hour glucose levels of insulin were assessed according to a standard procedure before and after each intervention period.

**Subjects**

Eighteen participants volunteered for this study, all of whom were classified as IGT according to standards set by WHO (36), during their 2004 participation in a large-scale public health screening study in Norway (unpublished material) (Group 1: Males = 2; mean age = 47.5, Females = 7; mean age = 46.5. Group 2: Males = 2, mean age = 60.5; Females = 7, mean age = 44.4. Overall age ranges 33–69 years). Subject characteristics and baseline values (Table 1) were assessed using mean values (M) and standard deviations (SD), and confidence intervals (CI = 95%) for difference in means and t-scores. p values tests were used to evaluate the groups for homogeneous characteristics at prebaseline (percentage body fat, upper leg skeletal muscle area (left + right), body mass index (BMI) and body weight). The participants

<table>
<thead>
<tr>
<th>Variables</th>
<th>Mean (SD)</th>
<th>CI 95% Diff. of means</th>
<th>t-score</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Group 1 (n = 9)</td>
<td>Group 2 (n = 9)</td>
<td>Lower</td>
<td>Upper</td>
</tr>
<tr>
<td>Percentage body fat</td>
<td>39.7 (4.9)</td>
<td>36.7 (5.5)</td>
<td>-2.38</td>
<td>8.07</td>
</tr>
<tr>
<td>Skeletal muscle area</td>
<td>25,730 (3811)</td>
<td>24,473 (5,238)</td>
<td>-3,320.81</td>
<td>5,835.72</td>
</tr>
<tr>
<td>BMI</td>
<td>28.55 (4.3)</td>
<td>27.20 (4.2)</td>
<td>-2.83</td>
<td>5.58</td>
</tr>
<tr>
<td>Body weight</td>
<td>76.8 (11.6)</td>
<td>78.2 (16.9)</td>
<td>-15.94</td>
<td>13.05</td>
</tr>
</tbody>
</table>

*BMI = body mass index; CI = confidence interval.
†Percentage body fat, upper leg skeletal muscle area (left + right), BMI, and body weight at baseline.
in both groups fulfilled the overweight criterion for BMI (group 1: $M = 28.55, SD = 4.3$; group 2: $M = 27.17, SD = 4.1$). All the participants were classified as sedentary; they had partaken of no regular physical activity (3 d/wk) for at least 6 months before the start of the study. During the second intervention (group 2), consisting of conventional ERT, there were 2 dropouts. Written informed consent was obtained from each of the study participants in accordance with the Helsinki Declaration (1). The study was approved by the Regional Committee for Ethics in Medical Research (Ref. No. 4.2006.2549).

Procedures

Three weeks of instruction on techniques for carrying out each exercise was compulsory for the participants. This also served to prepare the participants for the intervention period. Once the intervention commenced, they were supervised individually and strictly guided and followed up by qualified personnel at the training center where the intervention took place. The participants were encouraged through an informational letter to avoid alcohol and heavy exercise the day before working out. They were further instructed to avoid heavy meals and smoking, coffee, tea, or large amounts of sugar 2–3 hours before exercising. The participants wore lightweight clothing and shoes that were suited to the exercise. Individual logs were used to record the time of the day work outs took place and general information on health conditions and weight loads for each exercise.

Group 1 performed 8 exercises of high-intensity MRT according to Bernstein’s inverted pyramid training program, with 60–85% of 1 RM (3–4 repetitions), 3 d·wk$^{-1}$ over 4 months. To make the 3–4 repetitions manageable, the work load had to be slightly reduced with successive bouts of exercise because of the induced anaerobic conditions in the muscles being targeted. Each bout of exercise was to be followed immediately by a new bout without breaks in between, for a total of 5 bouts (~20 repetitions) in the set.

Consequently, 8 exercises were completed with work load, repetitions, and bouts of exercise according to a standard procedure for ERT at 45–65% of 1 RM (3 bouts at 12–15 repetitions for ~45 repetitions) (4,3). The break between bouts of training lasted between 30 and 60 seconds.

The 8 exercises involved the whole body and included the following: (a) abdominal muscles (total abdominal), (b) lower back, (c) press for thighs, (d) leg press, (e) chest press, (f) arm press, extension for triceps, (g) pull down for upper back, and (h) arm curl for biceps. Before the resistance exercises were carried out, the person walked for 10 minutes on a treadmill to warm up. Resistance training was followed by another 10 minutes of walking on the treadmill to cool down.

Measurements

Clinical measurements included the height and the weight of the participants, taken while they were wearing undergarments without shoes; the height was rounded off to the nearest 1.0 cm, and the weight to the nearest 0.5 kg. Also, waist circumference was measured horizontally at the height of the umbilicus to the nearest 1.0 cm, with the participants standing with their arms hanging relaxed at their sides. Four skinfold measures were taken at the biceps, triceps, subscapularis, and suprailiac. Scores were calculated using waist circumference combined with age and gender according to a formula defined by Durnin and Womersley (12) to provide a corrected index of body fat (percent). Before and after the intervention, a standard procedure was followed for collecting venous blood samples and estimating serum levels of insulin and fasting glucose and 2-hour delayed glucose level after exposure. The same procedure was used to collect samples from group 2 while they acted as a control for group 1. Serum samples were analyzed at the Central Laboratory of Levanger Hospital on an Abbot Architect ci8200 auto analyzer, applying reagents and Calibrator from Abbot. Glucose was measured using

Figure 1. Changes in blood glucose (fasting and 2-hour test), insulin and C-peptide from baseline (Pre 1) to postintervention with maximal resistance training (Post 1), compared with a control group that was later exposed to an endurance resistance training intervention (Pre 2 – Post 2).
Effects of Resistance Exercise on Insulin Sensitivity

Statistical Analyses
Analyses involved the use of the SPSS 17.0 statistical package (SPSS Inc., Chicago, IL, USA) with descriptive statistics, repeated measures analysis of variance (ANOVA), and t-tests. For the MRT intervention, differences were investigated between preintervention-postintervention (time factor), between groups to test the effects of the intervention type (group factor), and potential differences in the relationship between the groups and the time factor. These tests were run using serum insulin and glucose levels. T-tests were run for group 2. Possible effects from intervention on glucose and insulin were also assessed by the Homeostatic Model Assessment (HOMA)-2 model (see below). Statistical significance was set at $p \leq 0.05$. An effect size of 0.8 was calculated, which indicated improved glucose tolerance. The results from F-tests for the group and interaction yielded significant F-scores. This is unsurprising, as the participants’ fasting glucose levels were not impaired. The 2-hour glucose load test, in contrast, reflected an overall decrease from preintervention to postintervention ($p < 0.044$), which indicated improved glucose tolerance. The results from F-tests for the group and interaction of group and time factors were nonsignificant. For insulin, none of the F-scores were significant, whereas for

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre</th>
<th>Post</th>
</tr>
</thead>
<tbody>
<tr>
<td>MRT group 1 ($n = 9$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting (mmol·L$^{-1}$)</td>
<td>5.40 (0.75)</td>
<td>5.29 (0.63)</td>
</tr>
<tr>
<td>2 h (mmol·L$^{-1}$)</td>
<td>8.11 (2.26)</td>
<td>7.24 (2.50)</td>
</tr>
<tr>
<td>Insulin (pmol·L$^{-1}$)</td>
<td>60 (54)</td>
<td>52 (46)</td>
</tr>
<tr>
<td>C-peptide (pmol·L$^{-1}$)</td>
<td>842 (349)</td>
<td>769 (369)</td>
</tr>
<tr>
<td>Waitinglist control group</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Fasting (mmol·L$^{-1}$)</td>
<td>5.23 (0.60)</td>
<td>5.29 (0.56)</td>
</tr>
<tr>
<td>2 h (mmol·L$^{-1}$)</td>
<td>6.91 (1.71)</td>
<td>6.31 (1.21)</td>
</tr>
<tr>
<td>Insulin (pmol·L$^{-1}$)</td>
<td>49 (32)</td>
<td>49 (32)</td>
</tr>
<tr>
<td>C-peptide (pmol·L$^{-1}$)</td>
<td>804 (330)</td>
<td>746 (304)</td>
</tr>
</tbody>
</table>

*CI = confidence interval; Fasting = fasting glucose; 2 h = 2-hour glucose test; MRT = maximal resistance training.

Results

Subject Characteristics
Mean values for all dependent variables and all assessment times are illustrated in Figure 1. Scores for means, SDs, and CI 95% are given in Table 2 for fasting and 2-hour serum glucose levels and for insulin and C-peptide for the 2 intervention groups. According to the diagnostic guidelines from WHO (37), a person has IGT when the fasting glucose level is between 6.1 and 7.0 mmol/L or when 2HrPPG is between 7.75 and 11.1 mmol/L. Thus, the study groups fulfill at least 1 criterion for IGT. Mean scores for fasting C-peptide levels were around 800 pmol/L, which is well below the upper limit for normal range (220–1,400 pmol/L). Individual spot plot values for pre and post glucose (fasting and 2-hour) and insulin scores are presented in Figure 2 and for the MRT and the ERT.

Insulin Sensitivity, Fasting Glucose, and 2-Hour Blood Glucose
Results from the 2-way repeated ANOVAs (Table 3) indicated that fasting glucose scores showed no effect with the intervention. Neither pre-to-post changes (Time factor), difference between the groups (Group factor) nor the time by group interaction yielded significant F-scores. This is unsurprising, as the participants’ fasting glucose levels were not impaired. The 2-hour glucose load test, in contrast, reflected an overall decrease from preintervention to postintervention ($p < 0.044$), which indicated improved glucose tolerance. The results from F-tests for the group and interaction of group and time factors were nonsignificant. For insulin, none of the F-scores were significant, whereas for
C-peptide, the time factor yielded a significant change because of overall reduced scores postintervention \((p < 0.023)\) (Table 3).

**Effect Sizes**
The effects of MRT and ERT on fasting glucose and insulin levels were assessed using the HOMA-2 model. These results are presented in Table 5. Although the participants were selected on the basis of tests showing impaired glucose tolerance (IGT), they showed no signs of insulin resistance, impaired insulin sensitivity, or beta cell function. The MRT improved the initially acceptable insulin resistance level, whereas no significant changes were found in insulin sensitivity or beta cell function. The MRT reduced blood levels of 2-hour glucose \((p = 0.044)\) and fasting C-peptide \((p = 0.023)\).

### Table 3. F-scores and \(p\) values for effects of MRT on blood levels of fasting glucose, 2-hour glucose test, insulin, and C-peptide from preintervention to postintervention (Time), compared with prevalue and postvalues in a control group that was later exposed to an ERT intervention \((N = 18)\).*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Time (T)</th>
<th>Group (G)</th>
<th>(T \times G)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>(F)</td>
<td>(p)</td>
<td>(F)</td>
</tr>
<tr>
<td>Fasting</td>
<td>0.15</td>
<td>n.s.</td>
<td>1.36 n.s.</td>
</tr>
<tr>
<td>2 h</td>
<td>4.97</td>
<td>0.044</td>
<td>1.50 n.s.</td>
</tr>
<tr>
<td>Insulin</td>
<td>2.37</td>
<td>n.s.</td>
<td>2.36 n.s.</td>
</tr>
<tr>
<td>C-peptide</td>
<td>6.35</td>
<td>0.023</td>
<td>0.04 n.s.</td>
</tr>
</tbody>
</table>

*CI = confidence interval; Fasting = fasting glucose; 2 h = 2-hour glucose test; MRT = maximal resistance training; ERT = endurance resistance training; n.s. = not significant.

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Figure 2. Changes in spot plot values from pre intervention to post intervention for glucose (fasting and 2-hour) and insulin scores for each individual in the MRT (upper panel) and ERT (lower panel) groups.
The ERT caused a significant reduction in the blood levels of insulin (\(p = 0.023\)) (Table 4) and concomitant positive effects on insulin resistance (\(p = 0.03\)), % insulin sensitivity (\(p = 0.054\)), and beta-cell function (\(p = 0.020\)) (Table 5). These findings indicate that both MRT and ERT decrease insulin resistance in people at a risk of developing type 2 diabetes, whereas ERT yields additional positive effects. The spot plot (Figure 2) presents responders and nonresponders as measured by fasting and 2-hour glucose levels and insulin resistance at the individual level. Responses varied from nonresponse to varying degrees of positive response to MRT and ERT.

**DISCUSSION**

The development of insulin resistance and subsequent type 2 diabetes is a gradual process that spans many years and includes risk factors such as increased BMI and a sedentary lifestyle. High levels of leisure time physical activity are associated with decreased 2-hour blood glucose and fasting insulin levels and a reduced risk of having IGT and type 2 diabetes. In the present investigation, we compared the effects of MRT and ERT on people with impaired 2-hour blood glucose. Interestingly, MRT improved the initially acceptable insulin resistance, whereas no significant changes were found in insulin sensitivity or beta cell function. The improved insulin resistance may explain the ameliorated 2-hour blood glucose tolerance although this conclusion may be spurious in light of the fact that an improvement was also observed in the control group (Table 3). Several studies have reported an improvement in glucose tolerance after resistance training in body builders (39) and in patients with type 2 diabetes (15). This study is unique in describing the positive effect of MRT in ordinary overweight people with baseline IGT.

The ERT decreased fasting levels of insulin, which relate to improved insulin resistance, increased % insulin sensitivity, and decreased % beta-cell function. Many studies describe these effects (15). Most of the insulin-mediated glucose disposal takes place in the skeletal muscles, and increased amounts of intramyocellular lipid have been associated with insulin resistance and linked to decreased activity of mitochondrial oxidative phosphorylation. Several studies show reduced activity of oxidative enzymes in the skeletal muscles of those with type 2 diabetes (6,28). The reductions are independent of muscle fiber type and are accompanied by

### Table 4. Mean (SD) of blood levels of fasting glucose, 2-hour glucose test, insulin, and C-peptide pre and post ERT intervention (N = 7).†‡

<table>
<thead>
<tr>
<th>Variables</th>
<th>Pre Mean (SD)</th>
<th>CI 95%</th>
<th>Post Mean (SD)</th>
<th>CI 95%</th>
<th>T-score</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting (mmol L⁻¹)</td>
<td>5.24 (0.58)</td>
<td>4.70</td>
<td>5.78</td>
<td>5.23 (0.48)</td>
<td>4.79</td>
<td>5.67</td>
</tr>
<tr>
<td>2 h (mmol L⁻¹)</td>
<td>6.23 (1.38)</td>
<td>4.96</td>
<td>7.50</td>
<td>6.50 (1.61)</td>
<td>5.00</td>
<td>7.99</td>
</tr>
<tr>
<td>Insulin (pmol L⁻¹)</td>
<td>53 (34)</td>
<td>21.19</td>
<td>84.80</td>
<td>32 (19)</td>
<td>14.16</td>
<td>49.84</td>
</tr>
<tr>
<td>C-peptide (pmol L⁻¹)</td>
<td>737 (324)</td>
<td>436.61</td>
<td>1,037.39</td>
<td>716 (342)</td>
<td>399.26</td>
<td>1,032.74</td>
</tr>
</tbody>
</table>

*CI = confidence interval; Fasting = fasting glucose; 2 h = 2-hour glucose test; MRT = maximal resistance training; ERT = endurance resistance training; n.s. = not significant.

†T-scores and \(p\) values are given for predifferences to postdifferences.

### Table 5. Estimation of IR, %S, and %B by Homa-2.†‡

<table>
<thead>
<tr>
<th>Variable</th>
<th>Before maximal resistance (median)</th>
<th>After maximal resistance (median)</th>
<th>(p)</th>
<th>(n)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Maximal resistance training (N = 9)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR</td>
<td>0.78</td>
<td>0.53</td>
<td>0.04</td>
<td>9</td>
</tr>
<tr>
<td>%S</td>
<td>128</td>
<td>190</td>
<td>0.10</td>
<td>9</td>
</tr>
<tr>
<td>%B</td>
<td>67</td>
<td>69</td>
<td>0.16</td>
<td>9</td>
</tr>
<tr>
<td>Endurance resistance training (N = 7)</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>IR</td>
<td>0.72</td>
<td>0.48</td>
<td>0.03</td>
<td>7</td>
</tr>
<tr>
<td>%S</td>
<td>137</td>
<td>207</td>
<td>0.05</td>
<td>7</td>
</tr>
<tr>
<td>%B</td>
<td>79</td>
<td>56</td>
<td>0.02</td>
<td>7</td>
</tr>
</tbody>
</table>

*IR = insulin resistance; %S = insulin sensitivity; %B = beta-cell insulin production.

†Significance calculated by Wilcoxon paired samples test.
visual evidence of damaged mitochondria. In most studies, the reduced oxidative enzyme activity is explained by decreases in mitochondrial content. Thus, evidence of functional impairment in mitochondria in type 2 diabetes is not convincing. Rather, these impairments in oxidative function and mitochondrial morphology could reflect the sedentary lifestyle of subjects with diabetes.

Studies of insulin-resistant offspring of parents with type 2 diabetes have provided important insights into the earliest metabolic defects in type 2 diabetes. These defects include reductions in basal adenosine triphosphate (ATP) production and an attenuated response to insulin stimulation. The decreased basal ATP production does not affect overall lipid or glucose oxidation and, to our knowledge, no study linking changes in oxidative activity and insulin sensitivity in type 2 diabetes has been published. Thus, evidence of functional impairment of mitochondria in type 2 diabetes is not convincing, and intervention studies estimating the degree of correlation between changes in insulin resistance and changes in mitochondrial function in type 2 diabetes are needed. Accordingly, specific effects of regular physical training and muscular work on mitochondrial function and plasticity in type 2 diabetes remain an important area of research (29).

Effects of resistance training should be further explored in light of the encouraging findings from this study. Around 70–80% of insulin-mediated glucose disposal takes place in skeletal muscle. Several studies show that physical activity may double, if not triple, insulin-mediated glucose disposal in skeletal muscle within the norm of insulin concentrations in plasma (11). The mechanism behind this phenomenon involves capillary proliferation and an increase in GLUT4 (42), an increase in insulin sensitive muscle fibers, possible changes in phospholipid composition of sarcolemma, an increase in glycogenic and oxidative enzyme activity and an increase in glycogen synthase activity (18).

**Practical Applications**

The current results support a relationship between resistance exercise and insulin resistance in people with an increased risk of developing type 2 diabetes. Both MRT and ERT decrease insulin resistance. Although MRT had an impact on insulin sensitivity caused by increased glucose uptake, ERT decreased insulin resistance by increasing insulin sensitivity and concomitantly decreasing beta cell activity. If the aim is to increase glucose uptake capacity in skeletal muscles, one should recommend MRT and, on the other hand, if the aim is to also increase insulin sensitivity, one should recommend ERT. Consequently, these results provide some support to the recommendation of both MRT and ERT as a primary prevention approach to type 2 diabetes.

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**References**

Effects of Resistance Exercise on Insulin Sensitivity


