THE EFFECTS OF TWO BOUTS OF HIGH- AND LOW-VOLUME RESISTANCE EXERCISE ON GLUCOSE TOLERANCE IN NORMOGLYCEMIC WOMEN

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ABSTRACT

Reed, ME, Ben-Ezra, V, Biggerstaff, KD, and Nichols, DL. The effects of two bouts of high- and low-volume resistance exercise on glucose tolerance in normoglycemic women. J Strength Cond Res 26(1): 251–260, 2012—The purpose of the study was to determine the efficacy of a low-volume, moderate-intensity bout of resistance exercise (RE) on glucose, insulin, and C-peptide responses during an oral glucose tolerance test (OGTT) in untrained women compared with a bout of high-volume RE of the same intensity. Ten women (age 30.1 ± 9.0 years) were assessed for body composition, maximal oxygen uptake, and 1-repetition maximum (1RM) before completing 3 treatments administered in random order: 1 set of 10 REs (RE1), 3 sets of 10 REs (RE3), and no exercise (C). Twenty-four hours after completing each treatment, an OGTT was performed after an overnight fast. Glucose area under the curve response to an OGTT was reduced after both RE1 (900 ± 113 mmol L^{-1} min^{-1}, p = 0.056) and RE3 (827.9 ± 116.3, p = 0.01) compared with C (960.8 ± 152.7 mmol L^{-1} min^{-1}). Additionally, fasting glucose was significantly reduced after RE3 (4.48 ± 0.45 vs. 4.90 ± 0.44 mmol L^{-1}, p = 0.01). Insulin sensitivity (IS), as determined from the Cederholm IS index, was improved after RE1 (10.8%) and after RE3 (26.1%). The reductions in insulin and C-peptide areas after RE1 and RE3 were not significantly different from those in the C treatment. In conclusion, greater benefits in glucose regulation appear to occur after higher volumes of RE. However, observed reductions in glucose, insulin, C-peptide areas after RE1 suggest that individuals who may not well tolerate high-volume RE protocols may still benefit from low-volume RE at moderate intensity (65% 1RM).

KEY WORDS diabetes, insulin sensitivity, insulin, weight training, oral glucose tolerance test

INTRODUCTION

In 2007, diabetes was the fifth leading cause of death by disease in the United States, costing Americans an estimated $174 billion in medical costs, disability, work loss, and premature mortality (2). Poor insulin sensitivity (IS or insulin resistance) is believed to be one of the basic defects in the development of type 2 diabetes (T2D) and is often associated with a clustering of abnormalities such as hyperinsulinemia, impaired glucose tolerance (IGT), hypertension, and dyslipidemia (33). Insulin resistance and IS can be measured directly (4) or estimated from the glucose and insulin responses to an oral glucose tolerance test (OGTT) (9). Elevated glucose and insulin responses to an OGTT reflect a diminution of glucose tolerance and increased insulin resistance, respectively.

However, insulin resistance, and its potential progression into T2D, may be largely preventable in that the primary causes appear to be inactivity and obesity (1). It has long been recognized that improvements in the glucoregulatory response in humans may be attained by participation in an aerobic exercise (AE) training program (13). Furthermore, these metabolic benefits can be achieved even after the performance of a single bout of AE (3). Resistance exercise (RE) has been recognized in recent years as a viable alternative or complement to AE for improving glucose regulation. Comparison training studies of both modes of exercise demonstrate RE to be equally effective (29,34,37,38) or superior (8) to AE in improving IS and glucose tolerance. When performed in conjunction with AE, RE has also been shown to provide an additive effect (11) in improving glucose regulation. This suggests that RE may offer beneficial adaptations that are distinct from AE such as increases in muscular strength and fat-free mass (7,19,29) and the sparing of fat-free mass when combined with weight loss (8,25,34). These adaptations result not only in improved IS but can also reverse sarcopenia and improve functional fitness in the elderly.

Despite the abundance of evidence for the efficacy of RE as a mode of exercise that is beneficial for improving IS and glucose tolerance, little data exist on specific exercise prescriptions with respect to intensity and volume (40).
Recent findings suggest that RE protocols of a higher intensity (5,28,29,34) or volume (5,8,22,23) may translate into greater improvements in IS and glucose tolerance. However, health fitness professionals face a dilemma in that populations with the greatest need to improve glucose regulation are sometimes the least capable of tolerating RE protocols necessary to achieve improvements. Insulin resistance appears to be more pronounced in obese and elderly individuals (14,39) who are typically not physically active and have little tolerance to exercise. Consequently, there is a tendency for diminished adherence to these high-volume and high-intensity exercise protocols (12,17,31). Therefore, the establishment of an appropriate and realistic RE prescription is essential for health fitness professionals that prescribe exercise to populations either already at risk for the development of T2D (individuals with IGT), individuals that have already developed T2D, or to apparently disease-free individuals who have been inactive and are attempting to prevent the development of the disease. It would seem prudent, with respect to individuals who suffer from glucoregulatory dysfunction, to determine whether a low-volumeb, moderate-intensity RE protocol would provide a sufficient stimulus for improving the IS and glucose tolerance. A low-volume, moderate-intensity RE protocol would more likely be well-tolerated by individuals that are less inclined to participate in a RE program of higher intensity or volume.

Therefore, the purposes of this study are to (a) examine the effectiveness of a low-volume, moderate-intensity RE protocol on the insulin and glucose responses to an OGTT in sedentary, normoglycemic women and (b) compare those results with a higher-volume RE protocol of equal intensity.

**METHODS**

**Experimental Approach to the Problem**

To address the question as to whether a low-volume, moderate-intensity RE protocol could affect OGTT plasma glucose, insulin, and C-peptide responses, volunteers completed 3 randomly assigned treatments: rest (C), low volume (RE1), and high volume (RE3) with participants serving as their own controls. The decision to collect plasma glucose and insulin samples permitted the determination of glucose tolerance and the estimation of insulin resistance. C-peptide samples were also collected to evidence pancreatic secretion of insulin. Treatments were separated by approximately 1 month to ensure that blood samples were collected during the follicular phase of the participants’ menstrual cycle and to provide an ample washout period between treatments. A repeated-measures multivariate analysis of variance (MANOVA) was employed for statistical analysis. This design was chosen to detect within-subject and between-treatment changes of multiple dependent variables. The protocols were designed to recruit major muscle groups, involve exercises that are primarily multijoint and that require a minimum of learning, and mimic the range of RE volumes that exists in the majority of the literature investigating glucose metabolic health (10–30 sets). The 10 REs performed, RE loads, repetitions per set, and rest intervals between sets were held constant for both protocols, permitting the investigation of the effect of RE volume on IS and glucose tolerance.

**Subjects**

Ten women between the ages of 18 and 40 years participated in the study. The participants were screened for abnormal fasting blood glucose concentrations and excluded from the study if glucose concentrations exceeded 5.55 mmol L⁻¹. Other criteria for participant exclusion from the study included smoking, postmenopausal, signs or symptoms of cardiovascular disease, diabetes, pregnancy, irregular menstrual cycle, and participation in 30 minutes of moderate activity ≥2 times per week. The participants had little to no prior experience with RE training. None of the women in the study had participated in regular physical activity within the 6 months before the start of the study. Furthermore, 6 of the participants had been sedentary for the previous 2 years. The participants were instructed to remain sedentary for the duration of the study other than undergoing each RE treatment. The mean VO2 max (30.2 ml·kg⁻¹·min⁻¹) confirmed the untrained status of the participants. The study was approved by the Texas Woman’s University Institutional Review Board (IRB). The participants completed a medical questionnaire and informed consent in accordance with the Texas Woman’s University IRB guidelines.

**Height, Body Mass, and Body Composition Measurements**

Body mass, body mass index (BMI), and body fat percentage (BF%) were measured prestudy and poststudy to detect any significant changes in body composition over time that might influence the results. Body mass was measured on an electronic balanced scale (Tanita BWB-800, Tanita Corporation, Arlington Heights, IL, USA) calibrated to 0.1 kg. The height was measured to nearest 0.1 cm using a wall-mounted stadiometer (Perspective Enterprises, Kalamazoo, MI, USA). Height and body mass were measured with participants wearing t-shirts, exercise shorts, and in bare feet. Body composition was determined by the Food and Drug Administration-approved dual energy x-ray absorptiometer, the Lunar DPX-IQ DXA scanner (Lunar Radiation Corp., Madison, WI, USA). The mean BMI (29.5 kg·m⁻²) and BF% (42.3%) revealed that the participants tended to be overweight or obese, indicative of the participants’ unfit status. The descriptive physical data are summarized in Table 1.

**Oral Glucose Tolerance Test**

The OGTTs were performed between 7:30 and 11:30 AM after a 12- to 14-hour overnight fast and 24 hours after each RE treatment or rest. A catherter (20 gauge × 1 in.; Becton-Dickinson, Sandy, UT, USA) was inserted into an antecubital vein and kept patent with 1 ml of a dilute sterile saline-heparin solution (10 U/ml). Before each blood sampling, 3 ml of venous blood was removed via the catheter and...
TABLE 1. Participant descriptive statistics (n = 10).

<table>
<thead>
<tr>
<th>Variable</th>
<th>M</th>
<th>SD</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (y)</td>
<td>30.1</td>
<td>9.0</td>
<td>18.0–40.0</td>
</tr>
<tr>
<td>Body mass (kg)</td>
<td>83.4</td>
<td>25.8</td>
<td>52.8–126.4</td>
</tr>
<tr>
<td>Height (m)</td>
<td>1.67</td>
<td>0.06</td>
<td>1.54–1.74</td>
</tr>
<tr>
<td>Body mass index (kg·m⁻²)</td>
<td>29.5</td>
<td>8.6</td>
<td>19.6–43.7</td>
</tr>
<tr>
<td>Body fat %</td>
<td>42.3</td>
<td>12.2</td>
<td>19.8–56.9</td>
</tr>
<tr>
<td>VO₂max (ml·kg⁻¹·min⁻¹)</td>
<td>30.2</td>
<td>6.1</td>
<td>22.8–39.6</td>
</tr>
<tr>
<td>Fasting glucose (mmol·L⁻¹)</td>
<td>4.89</td>
<td>0.44</td>
<td>4.50–6.06</td>
</tr>
<tr>
<td>Fasting insulin (pmol·L⁻¹)</td>
<td>1.3 × 10²</td>
<td>1.3 × 10²</td>
<td>0.43–5.02 × 10²</td>
</tr>
</tbody>
</table>

discarded. Venous samples (7 ml) were collected prior to and at 15, 30, 60, 90, 120 and 150 min after ingesting a 75 g glucose solution (TruGlu, Fisher Scientific, Pittsburg, PA, USA). The samples were placed in blood collection tubes containing 7.5 mg of ethylenediamine tetraacetic acid (Sherwood Medical, St. Louis, MO, USA) and centrifuged at 1,800 rpm for 8 minutes (24). Plasma samples were immediately stored at −80°C for future analysis of insulin (Insulin enzyme-linked immunosorbent assay [ELISA] DSL-10-1600: Diagnostics Systems Laboratory, Inc., Webster, TX, USA), C-peptide (C-peptide of insulin ELISA DSL-10-7000: Diagnostics Systems Laboratory, Inc.), and plasma glucose. Plasma glucose was analyzed manually in duplicate by using a 2300 STATplus+ glucose/lactate analyzer (Yellow Springs Instruments, Yellow Springs, OH, USA). The coefficient of variation for the insulin, C-peptide, and glucose assays was 3.60, 4.14, and 0.70%, respectively. Before the performance of the assays, changes in plasma volume from C to RE1 and RE3 were calculated (15). No significant differences (+2.42%, p = 0.528) were observed between the C treatment and the RE1 treatment plasma volumes. However, a significant reduction in plasma volume was found from the C treatment to the RE3 (−8.45%, p = 0.016). Therefore, the RE3 plasma insulin, glucose, and C-peptide concentrations were adjusted before performing any statistical analysis. No adjustment was made for the RE1 treatment.

![Figure 1](https://example.com/figure1.png)

**Figure 1.** Timeline for the experimental design sequence.

Resistance Exercise Protocol
Three treatments were administered in random order: 1 set of 10 REs (RE1), 3 sets of 10 REs (RE3), and no exercise (C). Each separate RE treatment was composed of 3 bouts. Bout 1 consisted of the determination of a 1RM for each of the REs and was performed on a Friday. The protocol for determining the 1-repetition maximum (27) was conducted for each RE except for back raises and abdominal crunches. Bout 2 was performed 3 days later (Monday) and consisted of the performance of 1 of the 3 treatments. Its inclusion was designed to serve as a practice bout and also to minimize muscle damage, muscle soreness, and elevated plasma creatine kinase that may occur subsequent to the third bout (16). Short-term insulin resistance is a potential consequence of muscle damage after unaccustomed eccentric exercise (30). Bout 3 was performed on a Friday morning (4 days after bout 2 and 24 hours before the OGTT) and duplicated the treatment that was performed in bout 2 on the previous Monday. Both experimental protocols were approximately 65% of the participant’s 1RM and were performed for 10 repetitions on all sets. Both RE1 and RE3 bouts were designed to emulate the lower and upper ranges of volume, respectively, that one would expect to observe in typical RE bouts. A timeline of the experimental design sequence is shown in Figure 1.

The REs were performed as a circuit in the following order: dual-axis seated row, prone leg curl, dual-axis seated chest press, seated leg press, crunches, dual-axis pulldown, prone leg curl, dual-axis overhead press, seated leg press, and 45° back raise machine (Cybex International, Inc., MA, USA). The exercise order was designed to maximize recovery for each muscle group while limiting local muscular fatigue. Exercises 1–4 and 6–9 alternated upper and lower body exercises (4 upper body and 4 lower body). Additionally, exercises alternated between extensor and flexor groups for both the upper and lower body. Exercises 5 and 10 of each circuit targeted the lumbar spine extensors or flexors. Each set of RE in the circuit was initiated on a 2-minute interval (set 1 started at 0 minutes, set 2 started at 2 minutes, set 3 started at 4 minutes, etc.). The RE1 and RE3 bouts required 19 and 59 minutes to complete, respectively. Each RE bout was preceded by a...
10-minute warm-up on a treadmill. Each treatment was administered approximately 26–32 days apart and timed to occur during the participant’s follicular phase of the menstrual cycle (4–11 days after the onset of menstruation) to avoid the possibility of an elevated insulin response (6,19).

**Dietary Considerations**

The participants were instructed to maintain a 3-day dietary record before each OGTT and to consume a minimum of 150 g of carbohydrates. Photocopies of the food records were supplied after the initial OGTT, and the volunteers were instructed to repeat the 3-day diet before subsequent OGTTs. Diet records were analyzed for macronutrient composition (Nutritionist IV; N-Squared Computing, San Bruno, CA, USA) and verified for compliance to the above-mentioned guidelines by a registered dietician. Each participant was provided the same meal the night before each OGTT. The participants were permitted to consume water during the procedure, but the volume consumed after the initial OGTT was documented and held constant for the remaining 2 treatments.

**Statistical Analyses**

Paired samples $t$-tests were performed prestudy and poststudy on body composition measures. Area under the curve (AUC), relative to the participant’s baseline value, was calculated from the OGTT for glucose, insulin, and C-peptide. Repeated MANOVA was used to analyze glucose, insulin, and C-peptide areas under the curve and the Cederholm IS index (9) for each treatment. Time points for glucose, insulin, and C-peptide were also analyzed by repeated-measures MANOVA. The Bonferroni correction was used to adjust for multiple comparisons. The Cederholm index was chosen to estimate IS in this study because of its use of OGTT glucose and insulin concentration values, mean glucose and insulin values, and an anthropometric measure (body mass). The Cederholm IS index has been referenced to and has demonstrated a significant correlation ($r = 0.58, p = 0.0001$) with euglycemic-hyperinsulinemic clamp-derived IS in women with normal glucose tolerance (32). Furthermore, it has been reported that IS indices that have a higher degree of concordance with the clamp procedure are those indices that used OGTT glucose and insulin concentrations and some type of anthropometric measurement (32).

The Cederholm IS index equation is shown below, where 75,000 is the glucose load during the OGTT (75,000 mg), $G_t$ represents glucose (mmol·L$^{-1}$·min$^{-1}$) concentration at $t$ time, $I_{mean}$ and $G_{mean}$ represent mean insulin (mIU·L$^{-1}$·min$^{-1}$) and glucose (mmol·L$^{-1}$·min$^{-1}$) concentrations, respectively, during the OGTT. Glucose space is calculated as 0.19 $\times$ body mass (kilograms). The factor 120 was used to convert 2 hours into minutes. The factor 180 was used to transform glucose values from mmol·L$^{-1}$ to mg·dl$^{-1}$ (9).

$$
Cer = \left( \frac{75,000}{180} \right) \times \frac{1}{\Delta t} \int_0^{\Delta t} G_t dt + \left( \frac{120}{180} \right) \times \frac{1}{\Delta t} \int_0^{\Delta t} I_{mean} dt
$$

**Table 2.** Insulin, glucose, and C-peptide areas ($n = 10$).*

<table>
<thead>
<tr>
<th>Variable</th>
<th>$M$</th>
<th>$SD$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Insulin (pmol·L$^{-1}$·min$^{-1}$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>$9.4 \times 10^4$</td>
<td>$7.1 \times 10^4$</td>
</tr>
<tr>
<td>RE1</td>
<td>$7.4 \times 10^4$</td>
<td>$4.6 \times 10^4$</td>
</tr>
<tr>
<td>RE3</td>
<td>$7.5 \times 10^4$</td>
<td>$6.2 \times 10^4$</td>
</tr>
<tr>
<td>Glucose (mmol·L$^{-1}$·min$^{-1}$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>960.8</td>
<td>152</td>
</tr>
<tr>
<td>RE1</td>
<td>900.5</td>
<td>113</td>
</tr>
<tr>
<td>RE3</td>
<td>827.9</td>
<td>116</td>
</tr>
<tr>
<td>C-peptide (nmol·L$^{-1}$·min$^{-1}$)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>$2.5 \times 10^5$</td>
<td>$1.0 \times 10^2$</td>
</tr>
<tr>
<td>RE1</td>
<td>$2.2 \times 10^5$</td>
<td>$0.7 \times 10^2$</td>
</tr>
<tr>
<td>RE3</td>
<td>$2.2 \times 10^5$</td>
<td>$1.0 \times 10^2$</td>
</tr>
</tbody>
</table>

*RE = resistance exercise; RE1 = 1 set of 10 REs; RE3 = 3 sets of 10 REs.

†Significant difference from C.
The 3-dagy food intake record of carbohydrates, fats, and proteins was analyzed by a MANOVA with repeated measures for any differences. A Pearson product-moment correlation analysis was used to determine the relationships between exercise-induced changes in insulin or glucose AUC and the following: resting treatment glucose and insulin AUC, $\dot{V}O_2$max, BF%, and total volume lifted per kilogram of lean body mass. The significance for each analysis was set at an $\alpha$ level of 0.05. All statistical analyses were performed using SPSS 15.0 for Windows.

### RESULTS

#### Glucose, Insulin, and C-peptide Area Under The Curve

Paired samples t-tests on the volunteers’ prestudy and poststudy body composition measures confirmed that no significant changes occurred over the course of the study. Plasma glucose AUC was reduced 6.2 and 13.8% after RE1 and RE3, respectively. However, these changes were significant only after RE3 ($p = 0.01$) and not after RE1 ($p = 0.056$). No significant difference between RE3 and RE1 was detected (Figure 2). Insulin AUC was reduced by 21.0 and 19.5% after RE1 and RE3, respectively, but neither changes reached significance ($p = 0.072$). Likewise, reductions in C-peptide RE1 (11.8%) and RE3 (13.1%) failed to reach significance ($p = 0.083$). The values for glucose, insulin, and C-peptide AUC are given in Table 2.

#### Insulin Sensitivity Index

Significant treatment differences ($p = 0.004$) were detected for IS as determined by the Cederholm IS index (9). Relative to the C treatment, IS increased after RE1 and RE3 by 10.8 and 26.1%, respectively (Figure 3). The IS index after RE3 ($p = 0.009$), but not after RE1 ($p = 0.148$), was significantly higher than that of C. The IS after RE3 was not different from that after RE1 ($p = 0.306$).

#### Glucose, Insulin, and C-peptide Time Points

Pairwise comparisons identified that RE3 plasma glucose concentrations were significantly lower than that of C at 0 minutes ($p = 0.010$), 30 minutes ($p = 0.014$), 60 minutes ($p = 0.004$), 90 minutes ($p = 0.007$), 120 minutes ($p = 0.018$), and 150 minutes ($p = 0.008$). Additionally, RE3 was significantly lower than RE1 at 120 minutes ($p = 0.003$). No RE1 glucose time points were significantly different from those of C. Although all the participants in this study qualified as normoglycemic during pre-study screening, one participant’s C treatment fasting glucose was within the impaired fasting glucose (IFG) range (5.6–6.9 mmol/L). Furthermore, 6 participants had C treatment glucose values within the IGT range ($p = 0.083$). The values for glucose, insulin, and C-peptide AUC are given in Table 2.

### Table 3. Effect size data (n = 10), *

<table>
<thead>
<tr>
<th>Measure</th>
<th>RE1</th>
<th>RE3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose AUC</td>
<td>0.45</td>
<td>0.97</td>
</tr>
<tr>
<td>Insulin AUC</td>
<td>0.33</td>
<td>0.29</td>
</tr>
<tr>
<td>C-peptide AUC</td>
<td>0.35</td>
<td>0.30</td>
</tr>
<tr>
<td>Insulin sensitivity</td>
<td>0.51</td>
<td>1.00</td>
</tr>
<tr>
<td>Effect size magnitude (10)</td>
<td>0.2: Small</td>
<td>0.5: Moderate</td>
</tr>
</tbody>
</table>

*AUC = area under the curve; RE = resistance exercise; RE1 = 1 set of 10 REs; RE3 = 3 sets of 10 REs.*
(7.8–11.0 mmol L\(^{-1}\)) at multiple OGTT time points. The number of participants with plasma glucose values within the IGT range for one or more time points after RE1 and RE3 was reduced to 4 and 2, respectively.

No significant treatment differences occurred in plasma insulin concentration at any time point. The mean fasting insulin concentration for C was 1.3 \(\times\) 10\(^2\) pmol L\(^{-1}\), falling within the normal range of fasting insulin values of 0.39–1.9 pmol L\(^{-1}\) \(\times\) 10\(^2\) (18). Additionally, RE1 and RE3 plasma C-peptide concentrations were significantly lower than those of C at 150 minutes \((p = 0.011\) and \(p = 0.019\), respectively).

**Effect Size of the Treatments**

Effect size (ES) data, the standardized measure of the magnitude of the difference between treatment means, were calculated for IS and the AUC for glucose, insulin, C-peptide using Cohen’s \(d\) (10). The effect size for changes in glucose AUC and IS from C to RE3 were large, whereas the changes in glucose AUC and IS from C to RE1 were moderate. The ES for insulin and C-peptide changes from C to both RE1 and RE3 were small to moderate, with RE1 ES being slightly larger than RE3. Effect size data are given in Table 3.

**Dietary Records**

A repeated-measures MANOVA detected no treatment differences in total calories consumed either in the 3 days \((F = 2.11, p = 0.072)\) or in 1 day \((F = 0.526, p = 0.826)\) before the OGTTs. Additionally, no significant treatment differences existed between any dietary macronutrient percentages in either the 3-day or 1-day intake before any of the OGTTs.

**Correlations**

Insulin AUC during the C treatment was significantly correlated to changes in insulin AUC from C to RE1 \((r = 0.905, p = 0.0001)\) and changes in glucose AUC from C to RE1 \((r = 0.649, p = 0.042)\). However, there were no significant relationships between C insulin AUC and changes in glucose and insulin AUC from C to RE3. Additionally, C glucose AUC was significantly correlated to changes in insulin AUC from C to RE3 \((r = 0.853, p = 0.002)\), changes in glucose AUC from C to RE1 \((r = 0.731, p = 0.016)\) and changes in glucose AUC from C to RE3 \((r = 0.649, p = 0.042)\). No other meaningful significant correlations were detected.

**Discussion**

The primary purpose of this study was to explore the effectiveness of a low-volume, moderate-intensity RE protocol on the insulin, glucose, and...
C-peptide responses to an OGTT in sedentary, normoglycemic women, and to compare those results with a higher-volume RE protocol of equal intensity. There were 2 significant findings in this study. After the RE3 treatment, significant reductions in glucose AUC (13.8%) and significant increases in IS (26.1%) were observed. It is noteworthy to mention that 24 hours after the performance of RE1 (1 set of 10 REs at 65% 1RM), the participants in this study realized (a) reductions in the AUC for plasma glucose, insulin, and C-peptide by 6.2, 21.0, and 11.8%, respectively and (b) an increase of 10.8% in IS. However, these changes did not achieve significant levels.

The significant reduction in glucose AUC after the RE3 treatment was somewhat unexpected and is in contrast to the majority of the published data. The decreased glucose response suggests that the higher volume RE bout was sufficient to improve glucose tolerance in these women. Although examples of RE-induced decrements in glucose AUC are unusual in normoglycemic individuals, it is not unprecedented. Several studies have reported significant or near-significant reductions in glucose AUC after RE. However, none were single-bout studies and, in all but one, the study participants were either type 2 diabetic (19,38), IGT (38), or had IFG (37). The mean fasting glucose concentration after the C treatment in this study was within the normal range (4.89 ± 0.44 mmol L⁻¹). However, the individual OGTT time points for glucose during the C treatment (and to a lesser extent RE1 and RE3) revealed that several participants’ values were in the upper end of the range of normoglycemia or were mildly IGT. The elevated C glucose time point values observed in some of the participants may explain why the reduction after RE3 was observed. A significant relationship was detected between C treatment glucose AUC and changes in AUC from C to both RE1 (r = 0.731, p = 0.016) and RE3 (r = 0.649, p = 0.042), indicating that the participants with the highest C treatment AUC experienced the greatest reduction in AUC after the RE treatments. A similar relationship (r = 0.77, p < 0.05) was found between initial glucose AUC and change in AUC to a single bout of RE in women with T2D (19). Exercise-induced reductions in glucose in populations with higher glucose values such as T2D and IGT are more plausible because they are initially elevated and any reduction would bring glucose concentration closer to normal levels (19).

Typically, improvement in glucose tolerance in apparently healthy populations after an RE intervention is manifested by an attenuated insulin response with no changes in glucose, suggesting improved IS (29,38). The participants in this study experienced a 21.0 and 19.5% decline in insulin AUC after the RE1 and RE3 treatments, respectively. However, these declines failed to reach statistical significance despite attenuated insulin responses being observed in 8 and 9 out of 10 participants after both the RE1 and RE3 treatments, respectively.

The failure of the participants in this study to experience a significant decrease in plasma insulin concentration is in contrast to the findings of the bulk of the literature. In nondiabetic individuals, the insulin response can vary over a very wide range of values (4). It is possible that the high variability of the insulin data may have prevented the RE-induced reduction in insulin response observed in this study to reach significance. This is evident from the wide range of individual C treatment insulin AUC values (3.8 × 10⁻² to 2.8 × 10⁰ pmol L⁻¹ min⁻¹) and an SD that is 75% of the mean insulin AUC (9.4 × 10⁰ ± 7.1 × 10⁰ pmol L⁻¹ min⁻¹). Two potential sources for the high variability in insulin and C-peptide values may have been the wide range of body composition (BMI: 19.6–43.7 kg m⁻²) and age (18.0–40.0 years) values of the volunteers. Both body adiposity (39) and age (135) have been shown to impact glucose tolerance and IS. Although participation in the study was restricted to those individuals with fasting glucose concentrations <5.55 mmol L⁻¹ at screening, no restrictions were made with respect to body composition or age (volunteers were required to be premenopausal). The mean BMI in this study (29.5 ± 8.6 kg m⁻²) would imply that the participants tended to be overweight or obese. However, 4 of the 10 participants had a BMI >30.0 kg m⁻², and 5 were relatively lean (BMI < 270 kg m⁻²). Only 1 participant in this study displayed baseline fasting insulin values above the normal range. Nonetheless, no changes in insulin or C-peptide AUC were related to any body composition measure. Likewise, although the age range was large (18–40 years), the participants in this study were still relatively young (30.1 ± 9.0 years). Subsequently, no significant or near-significant relationships were detected between age and any fasting concentrations or changes in glucose, insulin, and C-peptide AUC.

Two other single-bout RE studies have employed an OGTT to measure glucose and insulin responses. Both of these studies administered an OGTT within 24 hours after the RE bout and used RE volumes similar to those in the RE3 treatment in this study. Fluckey et al. (20) reported a significant decline of 21 and 22% in insulin AUC in type 2 diabetic and healthy young controls, respectively, with no change in glucose AUC after a single bout of RE. These reductions in insulin AUC are comparable with that in this study for both RE1 and RE3; however, the variability in the data from Fluckey et al. was much less (11.5 and 19.8% of the mean vs that in this study). The total volume lifted by each of the 3 groups in the Fluckey et al. study was approximately 6,000 kg, compared with 3,569 and 10,608 kg for the RE1 and RE3 treatments, respectively, in this study. However, the RE intensity was relatively lower (3 sets each of 10 exercise performed at 50, 75, and 100% of the participant’s 10RM) compared with the RE3 treatment (3 sets of 10 repetitions for 10 exercise all performed at ≈ 95% of each participant’s 10RM) in this study. However, Fenicchia et al. (19) reported no change in insulin AUC in diabetic women after a single-bout of RE of a volume similar to that of the RE3 treatment in this study. The authors in this study suggested that the failure of exercise to reduce insulin could be attributed to the oral hypoglycemic medications prescribed to the patients in the study.
C-peptide, an indirect measure of pancreatic secretion of insulin, was measured in this study because it has been demonstrated that physical training blunts insulin secretion (36). No significant changes in C-peptide AUC in response to RE were observed in this study. This finding is in agreement with the findings of the only other single-bout RE study in which C-peptide was measured (20). However, reductions in C-peptide concentration have been reported 24 hours after 3 consecutive days of treadmill walking (24).

The limitations of this study are the relatively small number of participants and large SDs of AUC for both insulin and C-peptide, which likely precluded finding significant differences in these measures. It should be noted that the trends for both areas (insulin, \( p = 0.072 \); C-peptide, \( p = 0.083 \)) indicate that RE may decrease insulin secretion and thus provide some benefits in IS. Particularly noteworthy is that the observable increase in IS after RE1 (11.8%) and insulin AUC (21.0%) indicate that small volumes of moderate RE may be beneficial for the improvement in glucose regulation and these reductions after RE3 relative to RE1 rival those observed after RE3 (Table 2).

Furthermore, in observing the individual responses for each volunteer (Figures 4 and 5), insulin and C-peptide RE3 responses were surprisingly higher than those in RE1 in 3 of the 4 and in all 4 obese participants, respectively. The significant relationship between C insulin AUC and changes from C to RE1, and not C to RE3, may imply that individuals with poorer glucose tolerance may have some volume-dependent intolerance to RE. The obese volunteers in this study may have exhibited elevated insulin and C-peptide concentrations after RE3 relative to RE1 as a consequence of skeletal muscle microtrauma resulting from the unaccustomed volume (16,30). This provides support for the premise that higher volumes of RE may not be well tolerated and possibly counterproductive in certain populations such as those who are sedentary and obese.

The attenuated areas under the curve observed in both glucose and insulin in this study suggest that the participants may have experienced an improvement in IS. The use of the Cederholm IS index on the data reinforced this contention. The observable increase in IS after RE1 (10.8%) punctuates the potential for low doses of moderate-intensity RE to benefit individuals seeking to improve glucose regulation. The significant increase in IS after RE3 (26.1%) is comparable with that of other studies employing similar volumes of RE. In the only other single-bout RE study examining IS, the participants, performing a protocol of a similar volume to that of this study, significantly increased IS by 13 \( \pm \) 5% (26). Changes in IS in response to RE training programs of 4–16 weeks have also been reported in most (23,29,35) but not all (25) studies. Ishii et al. (23), reported a 48% increase in IS, which was twofold that of any other resistance training study reporting improvements in IS (29,35). It should be noted that the participants in the Ishii study performed 66–114% more volume per week than did participants in the other RE training studies (23,29,35), lending support to the notion that a higher volume of RE may tend to yield greater benefits, albeit volume in this case is defined as per week and not per session.

Although the focus of this study precludes the identification of specific mechanisms underlying the exercise-induced changes observed, it is reasonable to infer that the reduced plasma glucose AUC was most likely because of 2 factors. The primary sources of adenosine triphosphate (ATP) during RE are derived from high-energy phosphagen breakdown, glycogenolysis, and lipolysis of intramyocellular lipids. Depletion of intramuscular energy substrates has a beneficial effect on glucose uptake. The greater improvements observed after RE3 suggest greater substrate use. Secondly, the increase in glucose uptake most likely resulted from a protracted postexercise increase in IS that may persist for approximately 48 hours after exercise (21).

In summary, this is the first study to examine the effects of a single bout of low-volume, moderate-intensity RE on OGTT plasma insulin, glucose, and C-peptide responses relative to a higher volume RE protocol of like intensity. Both glucose tolerance and IS were significantly improved only after the high-volume, moderate-intensity RE protocol. Although the data from this study confirm previous research findings that higher doses of RE tend to yield greater improvements in glucoregulatory control, this principle may be counterproductive in certain populations who are untrained, obese, and have a low exercise tolerance. Furthermore, the trends observed in plasma glucose, insulin, C-peptide concentrations after low-volume, moderate-intensity RE reflect that low-volume RE performed at 65% 1RM may be of benefit to these same populations.

**Practical Applications**

Performing RE is a well-documented approach for preventing insulin resistance and T2D. However, program adherence is a major obstacle for many individuals who are obese and have never participated in a structured exercise program. Therefore, it is essential to establish a prescription for appropriate RE volume that will be sufficient to elicit improvements in glucose regulation while minimizing the attrition rate in these individuals. Based on the findings in this study, health fitness professionals, to maximize both benefits and compliance, should consider the following with respect to prescribing RE volume. Performing higher volumes of RE will typically maximize improvements in glucose tolerance and IS. Therefore, practitioners should typically seek to progressively increase RE total volume for most individuals seeking to improve glucoregulatory control. Conversely, although a systematic increase in total volume may tend to improve glucose tolerance and IS in many individuals, an alternative strategy may need to be considered for individuals who are obese and have been previously inactive. Examination of the individual insulin and C-peptide responses of the obese participants only revealed that the high-volume (30 sets) bout provided no additional observable benefits compared with the low-volume (10 sets) bout. Additionally, the 30-set protocol
tended to be counterproductive (elevated insulin and C-peptide responses relative to the 10-set protocol) for the obese participants. Practitioners, rather than increasing volume, may consider alternative methods for introducing a progressive overload into a RE design such as increasing resistance, increasing repetitions, or shortening rest intervals. Practitioners should be cognizant that even very low-volumes (10 total sets) of moderate-intensity (65% 1RM) RE may provide substantial benefits to untrained, obese individuals who may respond adversely to a higher-volume RE protocol. These types of bouts may last as little as 20 minutes, making it considerably easier for unfit clients or patients to maintain compliance. Furthermore, individuals with the greatest need for improvements in glucoregulation, who may be more likely to have poor exercise tolerance, tend to experience the greatest improvements from the low-volume protocol.

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