Effects of Intensity and Volume on Insulin Sensitivity During Acute Bouts of Resistance Training

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Abstract

Black, LE, Swan, PD, and Alvar, BA. Effects of intensity and volume on insulin sensitivity during acute bouts of resistance training. *J Strength Cond Res* 24(4): 1109–1116, 2010–This study evaluated the effects of various resistance exercise protocols on 24-hour postexercise insulin sensitivity. Seventeen participants with impaired fasting glucose (100–125 mg/dL) completed 4 separate bouts of resistance exercise under moderate intensity (65% 1 repetition maximum [1RM]) or high intensity (85% 1RM) conditions within the confines of single set and multiple set protocols. Intravenous fasting blood was taken at baseline and 24 hours postexercise for each exercise condition to measure fasting plasma glucose (G0) and fasting serum insulin (I0) to calculate insulin sensitivity (homeostasis model assessment-insulin resistance = (G0*I0)/405). A minimum of 3 days washout was given between each exercise protocol. A 4 x 2 factorial analysis of variance was performed to compare insulin sensitivity and fasting glucose within subjects and between treatments. All of the exercise protocols improved subsequent insulin sensitivity ($p = 0.002$) and G0 ($p = 0.001$). In comparison with single set, there was a significantly greater decrease in G0 ($p = 0.021$) 24 hours after multiple set bouts. High intensity showed significant decreases in insulin sensitivity as compared with moderate intensity protocols ($p = 0.046$). Effect size data suggest a dose response relationship between program variables of volume and intensity and 24-hour post-exercise insulin sensitivity. High-intensity protocols resulted in greater effect sizes for insulin sensitivity (0.83 multiple set; 0.53 single set) as compared with moderate-intensity protocols. The high-intensity, multiple set bout yielded the greatest treatment effect in both fasting glucose (0.61) and insulin sensitivity (0.83). Overall, single set protocols were less effective than multiple set protocols in lowering fasting blood glucose. Findings suggest a dose-response relationship between volume and intensity on insulin sensitivity and fasting blood glucose. Results indicate that resistance exercise is an effective treatment for acutely enhancing insulin sensitivity and regulating blood glucose in individuals with impaired fasting glucose.

Key Words diabetes, glucose, single set, multiple set, effect size

Introduction

Prediabetes, a condition characterized by impaired fasting glucose or impaired glucose tolerance, currently affects over 54 million US adults ages 40 to 71 (35). Furthermore, individuals with prediabetes are at an increased risk for heart disease and are likely to develop type 2 diabetes within 10 years if steps toward prevention are not taken. Past findings indicate that lifestyle modification, including exercise, healthy diet, and behavior modification, decreases risk of developing type 2 diabetes by 58% (12).

Aerobic exercise has been shown to improve insulin action, delay pancreatic exhaustion, and may slow the progression of prediabetes to type 2 diabetes (1). Research suggests that exercise-induced increases in glucose uptake may reflect the percentage and type of fiber recruitment along with the metabolic stress on active muscle fibers, all factors of intensity (13,31), and has shown that high-intensity compared with moderate-intensity aerobic exercise may have a more pronounced effect on improving insulin sensitivity in participants with type 2 diabetes. However, high-intensity aerobic exercise is often contraindicated in this population because of increased risk of physiologic complications or additional comorbidities (18,26). In addition to the medical risk, there is a lack of definitive research to substantiate that high-intensity significantly improves insulin sensitivity over less-intense bouts (4).

One of the theories as to why exercise enhances insulin sensitivity is that depleted muscle glycogen stores after a single intense exercise bout initiates increased glucose uptake in the skeletal muscle, resulting in enhanced insulin sensitivity (31). Substrate use and glycogen stores are the

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main determinants of this physiologic response. Exercise-eliciting metabolic responses that predominantly depend on carbohydrates as the main fuel source (phosphocreatine and anaerobic glycolysis systems) theoretically will produce a greater effect on insulin sensitivity. In contrast, there are studies that suggest that the key to optimal gains in insulin sensitivity with exercise training is a result of the accumulated benefit of multiple bouts of exercise, moving the focus from intensity to duration (4,21,27). Currently, the intensity and duration of exercise needed to elicit the optimal improvement insulin sensitivity is unknown. In addition, there is little data examining these dose-response relationships between resistance exercise and insulin sensitivity and fasting blood glucose (4,27,33).

Acute benefits gained from high-intensity training include increased demand for circulating blood glucose, greater usage of muscle glycogen stores (increasing glycogen resynthesis and storage capacity), and increased daily energy expenditure (aiding in weight loss) (31), which occur independently of changes in insulin action. Uptake of glucose into the cell is mediated by a transporter protein known as GLUT4. The translocation of GLUT4 from intracellular storage sites to the cell surface is regulated by plasma insulin. Decreased insulin sensitivity within the cells, which is characteristic of diabetes, significantly reduces the impact of insulin on GLUT4 movement. However, recent studies have supported prior findings suggesting an insulin-like effect in GLUT4 transport as a result of muscle contraction (37). Early research indicated that long-term resistance training interventions enhanced the GLUT4 signaling pathway (19,30). However, data from more recent studies have shown GLUT4 to have a very short half-life, with signaling changes occurring rapidly in response to a single bout of exercise (20,28).

Gvien the weight-related limitations facing the majority of people with prediabetes, certain exercise modalities may not be appropriate (36). Weight-bearing exercise may be restricted or contraindicated because of a decreased exercise capacity caused by poor cardiovascular fitness or pre-existing musculoskeletal pain or injury. An alternative mode in which body weight is supported, such as resistance exercise, may be able to effectively improve insulin sensitivity and prevent diabetes onset without exacerbating pre-existing cardiovascular or musculoskeletal issues (22,36).

Although impaired fasting glucose (100 mg/dL – 125 mg/dL) is recognized as an independent marker of risk for developing type 2 diabetes, a clear-cut resistance exercise prescription for the treatment and prevention of type 2 diabetes is lacking (12,21). The unknown definitive effects of resistance training on glucose regulation and insulin action are currently curtailing these efforts. The purpose of this research was to measure the acute effects of 2 different resistance exercise intensities (moderate vs. high) under 2 different volumes (single set or multiple set) on 24-hour insulin sensitivity in individuals with impaired fasting glucose.

Methods

Experimental Approach to the Problem

Participants were used as their own controls in a randomized 2×2 factorial analysis of variance (ANOVA) crossover design. This design was selected to compare insulin sensitivity and fasting glucose within subjects and between treatments. Each training day tested a separate treatment condition denoted by changes in program variable combinations (intensity-high/low and volume-single/multiple sets). Measures of insulin sensitivity and fasting blood glucose 24 hours postexercise were selected as indicators of acute metabolic changes in response to single bouts of exercise.

Subjects

Seventeen participants, male and female, between the ages of 18 to 45 years were recruited from the Phoenix metropolitan area. Initial prescreening was conducted at Arizona State University in a fasting state 24 hours postexercise using a One-Touch Ultra glucometer (LifeScan, Inc., Milpitas, CA, USA) to verify fasting blood glucose levels within the range of 100 to 125 mg/dL. Volunteers were excluded from the study if their fasting blood glucose was less than100 gm/dL or greater than 125 mg/dL, they had a history of smoking, cardiovascular disease, renal or liver disease, were taking hypoglycemic or hypertensive medication, or had any conditions that would be contraindicative of exercise. Participants had little or no prior experience with strength training before the study. All participants were classified as sedentary: no regular physical activity (3 days/wk for $\geq$20 min) for at least 6 months before beginning the study. No differences in age or body mass index (BMI) were found between sexes at baseline. There were differences in body fat percentage in which higher values were recorded for females, whereas body weight was greater in males (Table 1). Written physician consent was required from each participant before beginning the study. Each participant was informed of the potential risks associated with the study and signed an informed consent before participation. The experimental methods and procedures were approved by the human subjects institutional review board of the university.

Procedures

There were a total of 2 orientation days and 4 exercise testing days over a total of 4 to 5 weeks. The first orientation day was conducted 1 week before testing for anthropometric measurements, orientation to the resistance training exercises, and dietary recall instructions. Anthropometric measurements collected included body composition (Bod Pod), height, and weight. To familiarize participants with the protocol, each person was shown a demonstration of each lift followed by instruction and practice repetitions to assure proper technique. At the end of the first session, participants were taught how to keep a 24-hour dietary recall. Using this initial recall as a reference, participants were told to closely duplicate this same recorded daily intake 24 hours before each of the subsequent 3 sessions. In addition, participants were required

Table 1. Subject characteristics and baseline measures.*†

Measure	Baseline measures		
	Men ($n = 12$)	Women ($n = 5$)	Combined ($n = 17$)
Age (yr)	28 ± 11.3	32.4 ± 9.8	29.9 ± 9.6
BMI (kg/m^2)	31.3 ± 5.7	28.2 ± 5.07	30.4 ± 5.6
Weight (kg)	95.2 ± 16.9	75.6 ± 18.6§	90.0 ± 18.8
Body fat‡	27.8 ± 7.7	39.9 ± 10.3§	30.9 ± 10.6
Mean fasting glucose (mg/dL)			
Pre-exercise	99.3 ± 8.4	93.2 ± 6.3	97.2 ± 8.2
Postexercise	98.5 ± 8.7	91.5 ± 5.9	96.4 ± 8.0
Mean fasting insulin (UIU/mL)			
Pre-exercise	12.8 ± 1.8	7.4 ± 3.1	10.8 ± 1.3
Postexercise	11.1 ± 6.3	6.3 ± 2.5	9.4 ± 2.1
Mean HOMA			
Pre-exercise	2.6 ± 1.8	1.7 ± 0.7	2.3 ± 1.6
Postexercise	2.1 ± 1.5	1.3 ± 0.4	1.7 ± 1.2

*HOMA = homeostasis model assessment.
†Data are means ± *SD*.
‡Measured using BOD POD (Mdoel 1SD-06-M), computed by way of Siri model (%fat = [4.95/Db − 4.50]*100).
§Significant sex difference, $p < 0.05$.

to adhere to a 12-hour overnight fast preceding each blood draw. Participants were not permitted to exercise in between sessions. Caffeine consumption was restricted 24 hours before all blood draws because previous studies have shown that it may interfere with normal substrate use.

Each participant attended a second orientation day at which they were further familiarized with the lifting techniques. During this session, a 5 repetition maximum test was conducted for the bench press, leg press, squat, lat pull-down, and shoulder press. Test values were used to determine appropriate weights for each successive exercise session (2). There was no weight progression for any of the exercises throughout the study.

The resistance exercise protocol design comprised multijoint exercises targeting each of the major muscle groups. Lower-body exercises included squats, horizontal leg press, lunges, and step-ups. Upper-body exercises included bench press, lat pull-downs, shoulder press, and upright rows. Free-weight variations were used (except with the lat pull-down and the horizontal leg press) because of the additional demands placed upon secondary stabilizer muscles, enhancing overall muscle contraction. Moderate-intensity protocols were determined at 65% of participants estimated 1 repetition maximum. At this intensity, participants performed 12 to 15 repetitions for all 8 exercises. The high-intensity protocol was set at 85% of the estimated 1 repetition maximum, requiring participants to complete 6 to 8 repetitions of each exercise. Volume was determined by number of sets performed for each exercise. Single and multiple (4) set designs were paired with moderate and high intensity to create the 4 protocols.

For each exercise testing day, the participant reported to the laboratory for an intravenous blood draw to evaluate baseline measures of serum insulin and blood glucose. Then, the participants consumed a high-glycemic pre-exercise meal consisting of a bagel and orange juice (6 oz) to minimize transit time of glucose from ingestion into the blood stream. Participants were given 20 minutes between the meal and beginning the resistance exercise portion of the session. Participants performed 1 of 4 randomly selected resistance exercise protocols each testing day lasting between 30 and 55 minutes. Twenty-four hours after exercise session, participants reported to the research laboratory for a second fasting blood draw. The 24-hour postexercise blood draw examined plasma glucose and serum insulin levels.

Participants participated in a maximum of 2 exercise sessions per week for consecutive weeks until all 4 protocols had been completed. A minimum washout of 72 hours was used between test days to prevent a carryover effect from previous testing (5). All resistance exercise sessions were conducted in a climate-controlled setting in a weightlifting laboratory.

Measurement

Anthropometry. Height (cm) and weight (kg) were determined at baseline to calculate BMI as weight (kg)/height (m^2). Height was measured using a wall-mounted stadiometer. Weight was taken on a calibrated digital scale.

Body Composition Assessment. Fat mass, lean mass, and body density were measured using whole body air displacement

Table 2. Effect sizes of standardized differences.*

Magnitude	Difference in means
Trivial	<0.2
Small	0.2–0.5
Moderate	0.5–0.8
Large	>0.8

*Adapted from Cohen, *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.), 1988.

plethysmography (Bod Pod) (Life Measurements, Concord, CA, USA). Body composition analysis was estimated with subjects in a fasted state using the Siri (1961) model (%fat = [4.95/Db – 4.50]*100) designated for general population using predicted lung volume values.

Blood Glucose and Insulin Sensitivity. Venous blood samples were taken from the antecubital vein in the morning between 6:00 AM and 9:00 AM. The fasting blood draw from each testing day was used as the baseline for the ensuing bout of resistance exercise. Blood samples were spun in a centrifuge for 15 minutes and refrigerated. Special attention was paid to assure that the integrity of the blood samples were not compromised by having the samples delivered daily to an internationally certified laboratory for analysis. Fasting plasma glucose (G0) and insulin concentrations (11) were determined using glucose oxidase assays and enzyme immunoassays (Roche, Indianapolis, IN, USA), respectively.

Insulin sensitivity was calculated from the homeostasis model assessment (HOMA)-IR equation developed by DeFronzo et al. (11) in 1979. This equation estimates insulin sensitivity from fasting glucose and fasting insulin values and maintains a correlation value with the clamp method of $r = 0.60$ (11).

Statistical Analyses

Normality of variables was assessed by the Shapiro-Wilk test in this 4-way (randomized) repeated measures design. Differences of pre-exercise insulin sensitivity were compared with 24-hour postexercise within subjects and between treatments using a 2 x 2 factorial ANOVA. The factors used in subject comparisons included treatment and time (pre- and 24-hr postexercise). Statistical analyses were performed using SPSS 15.0 for Windows (Chicago, IL, USA). Results are expressed as the mean ± *SD*, with significance shown at $p \leq 0.05$. In the case of a significant treatment effect, paired *t*-tests were conducted to determine significant differences between individual measures using the Bonferroni post hoc test.

Effect sizes (ES) were calculated according to Cohen's *d* (1969) and the standardized mean difference. Effect size represents the difference between pre- and post-treatment means divided by the variability among the sample (29). The purpose of this analysis was to increase the applicability of the research to the health care professional. Results are

Table 3. Changes in metabolic indices pre- and postexercise.*†

Intensity (by volume)	Pre	Post	Δ
65% 1RM (single set)			
Mean fasting glucose (mg/dL)	96.0 ± 8.8	94.4 ± 9.2	−1.6 ± 0.6
Mean fasting insulin (UIU/mL)	9.8 ± 7.5	12.3 ± 7.5	2.5 ± 0.0
Mean HOMA	1.9 ± 1.06	1.45 ± 0.69	−0.45 ± 0.37
65% 1RM (multiple set)			
Mean fasting glucose (mg/dL)	97.3 ± 7.7	94.3 ± 9.9	−3.0 ± 2.2[b]
Mean fasting insulin (UIU/mL)	9.6 ± 4.5	8.4 ± 3.3	−1.2 ± 1.2
Mean HOMA	2.27 ± 0.96	1.98 ± 0.80	−0.29 ± 0.18
85% 1RM (single set)			
Mean fasting glucose (mg/dL)	96.4 ± 8.3	94.8 ± 5.5	−1.6 ± 2.8
Mean fasting insulin (UIU/mL)	10.4 ± 6.8	7.6 ± 5.4	−2.8 ± 1.4
Mean HOMA	2.05 ± 0.81	1.58 ± 1.50	−0.47 ± 0.69[a]
85% 1RM (multiple set)			
Mean fasting glucose (mg/dL)	99.1 ± 9.8	96.4 ± 8.7	−2.7 ± 1.1‡[b]
Mean fasting insulin (UIU/mL)	11.0 ± 8.2	8.1 ± 2.9	−2.9 ± 5.3‡[a]
Mean HOMA	2.41 ± 1.30	1.59 ± 0.72	−0.82 ± 0.58‡

*1RM = 1 repetition maximum; HOMA = homeostasis model assessment.
†Data are means ± *SD*.
‡Significant difference between pre- and postmeasures, $p = 0.05$.
[a]Significant difference between corresponding-volume groups at 65% intensity ($p < 0.05$).
[b]Significant difference between corresponding-intensity, single set group ($p < 0.05$).

Table 4. Effect size data.

	Single set		Multiple set	
Measure	A (65% 1RM)	B (85% 1RM)	C (65% 1RM)	D (85% 1RM)
Fasting glucose	0.1761	0.1992	0.3865	0.7216
Fasting insulin	−0.3359	0.42	0.2513	0.366
HOMA	−0.2898	0.4158	0.3399	0.8342

*1RM = 1 repetition maximum; HOMA = homeostasis model assessment.

reported for fasting glucose and insulin sensitivity across all 4 protocols. Magnitude of gain was determined according to previously established ES scale shown in Table 2 (8,9).

Results

Subject Characteristics

There were no significant differences in baseline fasting glucose levels between males and females across each of the 4 exercise protocols ($p = 0.804$). A total of 17 participants were selected for this study. During the testing period, 2 subjects were unable to complete all 4 protocols (1 because of injury unrelated to testing and 1 because of a change in work schedule). Thus, all data represent a total of 15 individuals.

Insulin Sensitivity and Fasting Blood Glucose

Pre- and postexercise and change values for fasting glucose, fasting insulin, and insulin sensitivity (HOMA) are shown in Table 3. All exercise protocols improved subsequent insulin sensitivity ($-.62 \pm 1.02$, $p = 0.002$), fasting glucose (-4.87 ± 0.14, $p = 0.025$), and fasting insulin (-2.25 ± 2.14, $p = 0.001$). There was no significant between-subjects interaction by bout for insulin sensitivity, fasting glucose, or fasting insulin.

Significant differences were found when intensity protocols were combined and grouped into single set or multiple set bouts. In comparison with single set protocols, repeated measures ANOVA revealed a significantly greater decrease in fasting glucose ($p = 0.021$) after multiple set bouts. There was also a significant within-subjects interaction showing greater decreases in fasting insulin ($p = 0.046$) after high-intensity bouts when controlling for load. Load was calculated by multiplying the weight lifted by the number of repetitions and sets completed. Repeated measures ANOVA revealed no statistical significance ($p < 0.05$) between groups across all other measures.

Effect Sizes

Effect sizes computed for fasting glucose, fasting insulin, and HOMA across all 4 protocols are shown in Table 4. According to Cohen's d and the standardized mean difference scale (Appendix D), high-intensity protocols produced moderate to large ES. Low-intensity protocols produced trivial to moderate ES. When grouped according to intensity, high-intensity protocols resulted in greater ES for fasting insulin (Multiple Set (MS) = 0.67; Single Set (SS) = 0.47) and insulin sensitivity (MS = 0.83; SS = 0.53) as compared with moderate-intensity protocols for both multiple set and single set protocols. High-intensity, multiple set bout yielded the greatest treatment effect in all 3 metabolic variables: fasting glucose (0.61), fasting insulin (0.67), and insulin sensitivity (0.83).

Discussion

In this study, a 4-way crossover design was used to compare the acute effects of moderate-intensity resistance exercise versus high-intensity exercise within the confines of a single set and a multiple set protocols on the 24-hour postexercise insulin sensitivity in individuals with elevated fasting blood glucose levels. It was hypothesized that all forms of resistance exercise would improve 24-hour postexercise insulin sensitivity uptake as compared with baseline in individuals with elevated blood glucose. Findings from this study indicated that 24-hour postexercise fasting glucose, fasting insulin, and insulin sensitivity were improved in individuals with impaired fasting glucose. More specifically, high intensity significantly improved insulin sensitivity, whereas higher volume achieved with multiple sets significantly reduced fasting blood glucose. In addition, ES calculated for individual bouts suggest a possible dose-response relationship between participant workload and magnitude of change in fasting glucose and insulin sensitivity. Significant improvements after all bouts indicate that resistance exercise is an effective modality for improving insulin sensitivity 24 hours postexercise.

Although exercise is known to be efficacious for those with diabetes, an optimal prescription for the treatment and prevention of type 2 diabetes does not exist. It is also unknown whether resistance training is as effective as aerobic exercise for improving glucose tolerance and insulin resistance. Before the current study, little research has directly compared the high-intensity resistance exercise with the American College of Sports Medicine recommendations of low- to moderate-intensity protocols (10). In addition, there has been a lack of research looking at the health related benefits

resulting exclusively from resistance exercise. Past intervention trials are difficult to compare in terms of specific improvements from resistance exercise because many have combined aerobic and resistance training without comparing a controlled condition. These designs are not conducive for differentiating gains achieved through each of the separate modes of training, yet greater improvements in glucose uptake acquired through combined training as compared with aerobic training only imply an additive benefit from resistance exercise (23).

Exercise intensity is known to greatly impact physiologic functions contributing to glucose regulation and insulin sensitivity independent of changes in insulin. The clear dose-response relationship between exercise intensity and insulin sensitivity shown in this study is supported by previous research describing the efficacy of higher-intensity protocols for improving insulin sensitivity and fasting glucose control (7,14,17,22). The results from this study reveal that high-intensity exercise up to 85% 1 repetition maximum improves insulin sensitivity significantly more than moderate intensity in both multiple and single set designs. A number of studies using aerobic exercise have provided analogous evidence to the current study regarding intensity, suggesting consistent improvements in glycemic control are seen exclusively at intensities between 70% and 90% of 1 repetition maximum regardless of modality (6,21,25).

The inverse relationship between volume and intensity when considering the body's natural capacity to do work provided the rationale for examining single versus multiple set protocols in this study. Higher volume in the current study produced greater changes in 24-hour postexercise fasting glucose when intensity was held constant. This suggests that volume has an additive effect on fasting glucose independent of intensity. In addition, these data support the use of multiple set resistance exercise protocols. One mechanism proposed to explain this additive effect is that of caloric deficit at any given intensity. Greater energy demands incurred by higher volumes require more blood glucose and muscle glycogen usage for fuel resulting in greater overall muscle glycogen depletion.

To aid in the interpretation of the clinical importance of the study results ES were calculated. Effect sizes provide clinicians and health fitness professionals with tangible values to compare intervention efficacy. These values can help optimize the design of exercise treatment and prevention programs for individuals with impaired fasting glucose. In the case of predominantly 1-directional (positive) physiologic responses, identification of the protocol eliciting the greatest gain provides a vital step in understanding the effectiveness of the trial. When comparing high- and low-intensity exercise protocols, high intensity yielded greater improvements in fasting insulin and insulin sensitivity. The high-intensity, multiple set bout was shown to have the greatest ES. This finding is supported by studies demonstrating a relationship between improved metabolic control and number and magnitude of muscle contractions.

To make proper recommendations for exercise prescription, the specific length of the treatment effect must be determined. This study was designed to identify the acute changes acquired from a single session of exercise. It was not a training study. There were no significant differences in baseline values after each 3-day washout period, suggesting there was no carryover training effect between the exercise session and that repeating exercise bouts daily, or perhaps every other day is necessary to regulate blood glucose in those with impaired fasting glucose. This recommendation is supported by reports demonstrating that when the exercise stimulus is removed, improvements in insulin sensitivity deteriorate to levels shown before exercise (5,17,32). The acute nature of the insulin response to exercise may be caused in part by the short half-life of GLUT4, which is largely responsible for improved glucose uptake during exercise (12). Studies examining short-term exercise training ($<$7 days) have shown increases in insulin action before any recorded change in body composition (3). Thus, a minimum recommendation of 2 sessions per week, without a concomitant weight loss diet, has been suggested as enough to sustain enhanced insulin sensitivity and fasting glucose in individuals with impaired fasting glucose (22,34).

Despite the wealth of support for integrating higher-intensity resistance exercise in clinical populations to enhance glucose clearance, moderate-intensity aerobic rather than high-intensity resistance exercise is often prescribed for fear of exacerbating comorbidities such as coronary heart disease (36). However, high-intensity protocols have been shown to be safe and effective in improving metabolic measures as compared with control groups of dietary or pharmacologic intervention. In addition, resistance training has been shown to increase adherence particularly in obese individuals and persons with musculoskeletal or orthopedic limitations, the population for which glucose control is also recommended (5,7,14,17,36).

This study contributes considerably to the emerging research surrounding the effectiveness of resistance training for persons with prediabetes. Although identified by the American Diabetes Association as being at elevated risk for developing type 2 diabetes, research on this subpopulation remains scarce. Moreover, this study is the first to provide a direct comparison of moderate- and high-intensity resistance exercise on acute changes in insulin sensitivity in the aforementioned population.

Practical Applications

In summary, this study indicates that resistance exercise is efficacious for lowering 24-hour fasting blood glucose levels and improving insulin sensitivity in individuals with impaired fasting glucose. Higher volume, as achieved through multiple set protocols, was shown to be the most effective for acute regulation of blood glucose in individuals with impaired fasting glucose. Also, resistance exercise decreased -our fasting glucose without a concomitant change in insulin. This

supports the theory of increased glucose uptake independent of insulin action after exercise. The bulk of the previous research suggests that acute rather than chronic exercise is responsible for the beneficial effects on postexercise glucose control. In concert with current findings, whole-body resistance exercise may incur greater muscle contraction and fiber recruitment than aerobic exercise of the same caliber intensity. Also, the weight supported feature of performing resistance exercise may protect against joints in those overweight and untrained individuals who may have pain or limitations associated with weight bearing exercise. In addition, muscular strength has been shown to be inversely proportional to indices of the metabolic syndrome independent of cardiorespiratory fitness levels (24), providing further justification for using resistance exercise programs. Previous studies have also provided substantial evidence of higher participant compliance rates in resistance exercise programs compared with aerobic exercise programs (14,16). Thus, resistance exercise is clearly supported as an exercise modality for people with impaired fasting glucose. These preliminary results suggest that high-intensity, multiple set resistance exercise programs may emerge as the prescription of choice for individuals with impaired fasting glucose.

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References

1. American Diabetes Association. Standards of medical care for patients with diabetes mellitus (Position Statement). *Diabetes Care* 25: S33-S49, 2002.
2. Baechle, TR and Earle, RW. *The Essentials of Strength and Conditioning Research* (2nd ed). Champaign, IL: Human Kinetics, 2000.
3. Black, SE, Mitchell, E, Freedson, PS, Chipkin, SR, and Braun, B. Improved insulin action following short-term exercise training: role of energy and carbohydrate balance. *J Appl Physiol* 99: 2285–2289, 2005.
4. Borghouts, LB, Backx, K, Mensink, MF, and Keizer, HA. Effect of training intensity on insulin sensitivity and evaluated by insulin tolerance test. *Eur J Appl Physiol Occup Physiol* 80: 461–466, 1999.
5. Boule, NG, Weisnagel, SH, Lakka, TA, Tremblay, A, Bergman, RN, and Rankinen, T. Effects of exercise training on glucose homeostasis: the HERITAGE family study. *Diabetes Care* 28: 108–114, 2005.
6. Braith, RW and Stewart, KJ. Resistance exercise training: its role in the prevention of cardiovascular disease. *Circulation* 113: 2642–2650, 2006.
7. Castandeda, C, Layne, JE, Munoz-Oriazns, L, Gordon, PL, Walsmith, J, and Foldvari M. A randomized controlled trial of resistance exercise training to improve glycemic control in older adults with type 2 diabetes. *Diabetes Care* 25: 2335–2341, 2002.
8. Cohen, J. *Statistical Power Analysis for the Behavioral Sciences* (2nd ed.). Hillsdale, NJ: L. Erlbaum Associates, 1969.
9. Cohen, J. *Statistical Power Analysis for the Behavioral Sciences* (1st ed.). New York: Academic Press, 1988.
10. Davis, JK. Resistance training and type 2 diabetes. *J Strength Cond* 29: 42–48, 2007.
11. DeFronzo, RA, Tobin, JD, and Andres, R. Glucose clamp technique: a method for quantifying insulin secretion and resistance. *Am J Physiol* 237: E214–E223, 1979.
12. Diabetes Prevention Program Research Group. The Diabetes Prevention Program (DPP): description of lifestyle intervention. *Diabetes Care* 25: 2165–2171, 2002.
13. DiPietro, L, Dziura, J, Yeckel, CW, and Neufer, PD. Exercise and improved insulin sensitivity in older women: evidence of the enduring benefits of higher intensity training. *J Appl Physiol* 100: 142–149, 2006.
14. Dunstan, DW, Daly, RM, Owen, N, Jolley, D, DeCourten, M, Shaw, and J, Zimmet P. High intensity resistance training improves glycemic control in older patients with type 2 diabetes. *Diabetes Care* 25: 1729–1736, 2002.
15. Dunstan, DW, Daly, RM, Owen, N, Jolley, D, Vulikh, E, Shaw, J, and Zimmet, P. Home-based resistance training is not sufficient to maintain improved glycemic control following supervised training in older individuals with type 2 diabetes. *Diabetes Care* 28: 3–9, 2005.
16. Ewart, CK, Stewart, B, Gillialan, LB, and Kelemen, MH. Self-efficacy mediates strength gains during circuit weight training in men with coronary artery disease. *Med Sci Sports Exerc* 18: 531–540, 1986.
17. Fennicchia, LM, Kanaley, JA, Azevedo, JL, Miller, CS, Weinstock, RS, Carhart, RL, and Ploutz-Snyder, LL. Influence of resistance exercise training on glucose control in women with type 2 diabetes. *Metab Clin Exp* 53: 284–289, 2004.
18. Fiatarone, M, O'Neill, EF, Ryan, ND, Clements, KM, Solares, GR, Nelson, ME, Roberts, SB, Kehayias, JJ, Lipsitz, LA, and Evans, WJ. Exercise training and nutritional supplementation for physical frailty in very elderly people. *New Engl J Med* 330: 1769–1775, 1994.
19. Friedman, JE, Sherman, WM, Reed, MJ, Elton, CW, and Dohm, GL. Exercise-training increases glucose transporter protein GLUT4 in skeletal muscle of obese Zucker rats. *FEBS Lett* 16: 13–16, 1990.
20. Holloszy, J. Exercise-induced increases in muscle insulin sensitivity. *J Appl Physiol* 99: 338–343, 2005.
21. Houmard, JA, Tanner, CJ, Slentz, CA, Duscha, BD, McCartney, JS, and Kraus, WE. Effect of the volume and intensity of exercise training on insulin sensitivity. *J Appl Physiol* 96: 101–106, 2004.
22. Ibanez, J, Izquierdo, M, Arguelles, I, Forga, L, Larrion, JL, Garcia-Unciti, M, Idoate, F, and Gorostiaga, E. Twice-weekly progressive resistance training decreases abdominal fat and improves insulin sensitivity in older men with type 2 diabetes. *Diabetes Care* 28: 662–667, 2005.
23. Jurca, R, Lamonte, MJ, Church, TS, Earnest, CP, Fitzgerald, SJ, Barlow, CE, Jordan, AN, Kampert, JB, and Blair, SN. Associations of muscle strength and fitness with metabolic syndrome in men. *Med Sci Sports Exerc* 36: 1301–1307, 2004.
24. Jurca, R, Lamonte, MJ, Barlow, CE, Kampert, JB, Church, TS, Blair, SN. Association of muscular strength with incidence of metabolic syndrome in men. *Med Sci Sports Exerc* 37: 1849–1855, 2005.
25. Koval, JA, Maezono, K, Patti, ME, Pendergrass, M, DeFronzo, RA, and Mandarino, LJ. Effects of exercise and insulin insulin signaling proteins in human skeletal muscle. *Med Sci Sports Exerc* 31: 998–1004, 1999.
26. McCartney, N. Acute responses to resistance training and safety. *Med Sci Sports Exerc* 31: 31–37, 1998.
27. O'Donovan, G, Kearney, EM, Nevill, AM, Woolf-May, K, and Bird, SR. The effects of 24 weeks of moderate- or high-intensity exercise on insulin resistance. *Eur J Appl Physiol* 95: 522–528, 2005.
28. Ren, J, Semenkovich, C, Gulve, E, Gao, J, and Holloszy, J. Exercise induces rapid increases in GLUT4 expression, glucose transport capacity, and insulin-stimulated glycogen storage in muscle. *J Biol Chem* 269: 14386–14401, 1994.

29. Rhea, MR. Determining the magnitude of treatment effects in strength training research through the use of the effect size. *J Strength Cond Res* 18: 918–920, 2004.

30. Rodnick, KJ, Holloszy, JO, Mondon, CE, and James, DE. Effects of exercise training on insulin-regulatable glucose-transporter protein levels in rate skeletal muscle. *Diabetes* 39: 1425–1429, 1990.

31. Rose, AJ and Richter, EA. Skeletal muscle glucose uptake during exercise: how is it regulated? *Physiology* 20: 260–270, 2005.

32. Ross, R. Does exercise without weight loss improve insulin sensitivity? *Diabetes Care* 36: 944–945, 2003.

33. Ross, R, Freeman, J, and Janssen, I. Exercise alone is and effective strategy for reducing obesity and related comorbidities. *Exerc Sport Sci Rev* 28: 165–170, 2000.

34. Shaibi, GQ, Cruz, ML, Ball, GD, Weighensberg, MJ, Salem, GJ, and Crespo, NC, et al. Effects of resistance training on insulin sensitivity in overweight Latino adolescent males. *Med Sport Sci Rev* 28: 165–170, 2006.

35. Norris, SL, Kansagara, D, Bougatsos, C, and Fu, R. Screening adults for type 2 diabetes: A review of the evidence for the U.S. preventive services task force. *Ann Intern Med* 148: 855-868, 2008.

36. Willey, K and Fiatarone Singh, M. Battling insulin resistance in elderly obese people with type 2 diabetes. *Diabetes Care* 26: 1580–1588, 2003.

37. Yaspelkis, B. Resistance training improves insulin signaling and action in skeletal muscle. *Exerc Sport Sci Rev* 34: 42–46, 2006.