ABSTRACT

EFFECTS OF DIFFERENT EXERCISE MODALITIES ON POSTPRANDIAL HYPERGLYCEMIA AND INSULIN SENSITIVITY IN OVERWEIGHT AND OBESE ADULTS

by Craig William Berry

Postprandial hyperglycemia (PPH) is directly associated with cardiovascular disease risk. A single bout of aerobic (AE) or resistance exercise (RE) lowers PPH in healthy adults. No studies have examined the extent to which prior exercise regulates PPH and insulin sensitivity in overweight and obese adults, and whether differences exist between exercise modalities. PURPOSE: To determine the effects of different exercise modalities on PPH and insulin responses to an oral glucose tolerance test (OGTT) in overweight and obese adults. METHODS: In a randomized, cross-over design, 11 overweight and obese adults (21.8 y; BMI = 32.3 kg/m^2) completed three separate trials. Seated rest, a single bout of AE, or a single bout of RE preceded an OGTT by 14-17 h. Blood was obtained at 30 min intervals for 180 min following the OGTT. RESULTS: A main effect for time (p<0.001), but no trial (p>0.05) or interaction (p>0.05) effect, was found for plasma glucose and insulin. Plasma glucose and insulin concentrations increased above baseline (p<0.05) through 120 min and 180 min, respectively. CONCLUSION: Acute AE and RE performed the evening prior to an OGTT does not affect PPH or insulin responses in overweight and obese adults.
EFFECTS OF DIFFERENT EXERCISE MODALITIES ON POSTPRANDIAL HYPERGLYCEMIA AND INSULIN SENSITIVITY IN OVERWEIGHT AND OBESE ADULTS

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# Table of Contents

| LIST OF TABLES | IV |
| LIST OF FIGURES | V |
| ACKNOWLEDGEMENTS | VI |

## CHAPTER

| I. INTRODUCTION | 1 |
| II. LITERATURE REVIEW | 3 |
| III. METHODS | 8 |
| IV. RESULTS | 12 |
| V. DISCUSSION | 13 |

REFERENCES | 22 |
List of Tables

1. Table 1. Participant Characteristics ................................................. 16
2. Table 2. Participants’ Dietary Intakes ................................................. 17
3. Table 3. Fasting and Postprandial Plasma Glucose and Insulin Responses ................................................. 17
# List of Figures

<table>
<thead>
<tr>
<th>Figure</th>
<th>Description</th>
<th>Page</th>
</tr>
</thead>
<tbody>
<tr>
<td>1.</td>
<td>Figure 1. Timeline of study measures</td>
<td>10</td>
</tr>
<tr>
<td>2.</td>
<td>Figure 2. Plasma glucose responses</td>
<td>18</td>
</tr>
<tr>
<td>3.</td>
<td>Figure 3. Plasma insulin responses</td>
<td>19</td>
</tr>
<tr>
<td>4.</td>
<td>Figure 4. Plasma glucose area under the curve (AUC)</td>
<td>20</td>
</tr>
<tr>
<td>5.</td>
<td>Figure 5. Plasma insulin area under the curve (AUC)</td>
<td>21</td>
</tr>
</tbody>
</table>
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Chapter 1

Introduction

**Background:** Individuals spend a majority of their day in a postprandial state. Postprandial hyperglycemia (PPH), the exaggerated and prolonged elevation in blood glucose following the ingestion of carbohydrates, is a better predictor of cardiovascular disease (CVD)-related mortality compared to fasting blood glucose levels (8). Thus, strategies aimed at lowering PPH may reduce CVD risk.

Current guidelines recommend that adults participate in 30 minutes of moderate-intensity aerobic exercise at least 5 days/week, as well as resistance exercises targeting the major muscle groups 2-3 days/week (10). Most, but not all (7, 9), studies show that acute aerobic and resistance exercise lowers PPH and improves insulin sensitivity in healthy adults (1, 24, 40). One study showed that a 60-minute bout of moderate-intensity aerobic exercise (~60% maximal oxygen consumption (VO$_2$max)) performed 17 hours prior to the ingestion of a high-carbohydrate meal decreased the postprandial area under the curve (AUC) for plasma glucose and insulin (40). Similarly, a 45-minute bout of moderate-to-high intensity aerobic exercise (~70% VO$_2$max) lowered the incremental postprandial insulin area under the curve for up to 5 days post-exercise (24). In terms of resistance exercise, a single bout of whole-body resistance exercise resulted in a downward shift in the insulin AUC and lowered the peak blood glucose and blood glucose AUC for up to 14 hours post-exercise (1). Studies examining the efficacy of acute exercise on postprandial metabolic disturbances in clinical populations are lacking.

Approximately 70% of American adults are classified as overweight (body mass index (BMI) = 25-29.9 kg·m$^{-2}$) or obese (BMI = 30-35 kg·m$^{-2}$) (5). Compared to healthy adults, overweight and obese individuals have a greater CVD risk (5) that is explained, in part, by exaggerated postprandial glucose (20, 21, 31) and insulin responses (20, 21). Thus, determining the efficacy of lifestyle strategies, such as exercise, to mitigate postprandial metabolic disturbances in overweight and obese individuals is clinically important as these disturbances are associated with CVD-related morbidity and mortality (8). Compared to 30 minutes of aerobic exercise and a non-exercise control condition, 30 minutes of whole-body resistance exercise performed prior to the consumption of a high-fat meal significantly reduced insulin AUC in obese adults without affecting PPH (17). To our knowledge, no studies have examined the
influence of acute exercise, and differences due to exercise modality, on postprandial blood glucose and insulin responses to an OGTT in overweight and obese adults.

**Purpose Statement and Hypothesis:** The primary purpose of this investigation is to determine the effects of prior exercise and different exercise modalities on postprandial glucose and insulin responses following an OGTT in overweight and obese individuals. Compared to a non-exercise control trial, we hypothesize that a single bout of aerobic or resistance exercise performed 14-17 hours prior will attenuate postprandial glucose and insulin responses to an OGTT in overweight and obese individuals.

**Significance of Study:** Our anticipated findings are timely and of public health significance given the increasing prevalence of overweight and obesity (5, 43). Findings from this investigation will provide preliminary data for future studies to determine the efficacy of various lifestyle strategies (e.g., different exercise modalities, timing of exercise, dietary strategies) to attenuate postprandial metabolic disturbances in clinical populations.
Chapter 2
Review of the Literature

Overview: Review of the pertinent literature on this topic is divided into four related categories: 1) Background on PPH and insulin sensitivity, and the relationship to CVD; 2) cross-sectional data linking impaired glucose tolerance and insulin sensitivity in overweight and obese individuals to increased risk for CVD, and; 3) the effects of exercise on PPH and insulin sensitivity.

PPH and Insulin Sensitivity: Individuals spend the majority of their day in a postprandial state, defined as the time following meal consumption. Following meal consumption, the body experiences an influx of glucose entering the bloodstream. To counteract this, the pancreatic β-cells release insulin into the bloodstream. Cellular glucose uptake cannot occur until insulin triggers the insulin receptor located on the cell membrane. The insulin receptor is composed of two α- and two β-subunits. Binding of insulin to the α-subunits triggers phosphorylation of insulin receptor substrates (IRSs), which activates specific intracellular mechanisms (23, 30, 35). One such mechanism is the translocation of GLUT-4 to the cell membrane. GLUT-4 is a transport protein that allows glucose to enter the cell membrane and be subsequently utilized for energy production and storage. It is well known that skeletal muscle contraction (i.e., exercise) increases translocation of GLUT-4 to the cell surface independent of insulin (13).

Metabolic disturbances occurring during the postprandial period potentially contribute to increased risk for the development of CVD and metabolic diseases (2, 8). One such postprandial metabolic disturbance is PPH, which is the prolonged and exaggerated elevation of blood glucose levels following the intake of a high-carbohydrate meal. In comparison to fasting blood glucose levels, postprandial blood glucose levels are a better predictor of CVD-related morbidity and mortality (8). The Fungata Diabetes Study, which followed 2,651 non-diabetic individuals over the age of 40 for 7 years, found that individuals with impaired glucose tolerance following a 75-g OGTT had a higher rate of CVD-related mortality compared to those with normal glucose tolerance, while impaired fasting glucose did not increase the risk for CVD-related mortality compared to normal fasting glucose (39).
Similarly, it has been well established that insulin resistance is associated with increased risk for the development of CVD (35). Hyperinsulinemia has been observed in patients following myocardial infarctions (15) and in those with peripheral artery disease (41).

**Insulin Resistance and Glucose Tolerance in Overweight and Obese Individuals:** According to the American Heart Association, 69% of Americans over the age of 20 are overweight or obese, with 32 percent of children falling under the same category (43). In comparison to healthy adults, individuals who are overweight and obese are at greater risk of developing CVD, which is currently the leading cause of mortality in the United States (42).

The mechanism by which insulin resistance in overweight and obese populations occurs is thought to be due to a decrease in insulin-stimulated glucose transport via GLUT-4 in adipocytes and skeletal muscle cells and lowered hepatic glucose output (34). Insulin resistance results from defects in pancreatic β-cell function leading to hyperinsulinemia during the postprandial state (19, 21, 23, 38). Multiple studies have aimed to determine other mechanisms by which insulin sensitivity is lower in obese individuals. One mechanism proposed is through disruptions of the signaling pathways in insulin receptor cells via protein tyrosine phosphatases (PTPs), which terminate signaling propagated through tyrosyl phosphorylation events. These PTPs experience increased expression in the adipose tissues of obese individuals (11). Expression of various signaling pathways in skeletal tissues is also reduced in morbidly obese individuals (12), although GLUT-4 expression and action seems to remain stable and normal (37).

Obesity is closely associated with several comorbidities, including reduced insulin sensitivity and impaired glucose tolerance (6, 22, 38). Insulin resistance states, such as hypertension and diabetes, are common in overweight and obese individuals. Such insulin resistant states, as well as hyperglycemia, are associated with decreased endothelial vasodilation and coronary artery disease (CAD) (Steinberg). With the recent rise in obesity in the United States, there has also been an increase in the rate of CVD (43) which can be explained in part by impaired glucose tolerance and insulin resistance. Therefore, it is necessary to assess potential lifestyle mechanisms, such as exercise to help combat these occurrences.

**Effects of Exercise on Insulin Resistance and PPH:** Physical activity is an effective lifestyle intervention for the reduction of CVD-risk (10). The American College of Sports Medicine
(ACSM) currently recommends that adults participate in moderate-intensity aerobic exercise for at least 30 minutes a day for 5 days a week, or vigorous-intensity aerobic exercise for at least 20 minutes a day for 3 days a week, in addition to resistance-training exercise of major muscle groups for 2-3 days per week (10). It has been well established that exercise, both resistance and aerobic, provides a means for the promotion of glucose tolerance and insulin sensitivity. During exercise, skeletal muscle tissue is the primary means for peripheral glucose uptake (3, 16). GLUT-4 translocation and glucose uptake are stimulated by muscular contractions that occur during exercise (16, 36). Specifically, acute bouts of exercise triggers vast improvements on glucose uptake and insulin sensitivity in human populations (13). This provides a potential lifestyle mechanism for individuals with a lowered insulin sensitivity, such as overweight and obese populations, to combat such instances.

**Resistance Exercise:** Multiple studies have examined the effects of prior resistance exercise on postprandial hyperglycemia and insulin sensitivity the following day. In one study, 10 healthy, strength trained young men (age = 24 ± 2 years) performed a bout of resistance exercise approximately 14 hours prior to the ingestion of a carbohydrate-rich meal (1g carbohydrate per kg body weight). Blood samples taken every 15 minutes over a 2-hour period following meal consumption indicated that postprandial blood glucose and insulin levels were significantly lower (P <0.05) compared to a non-exercise trial (1). Similar findings were observed by Miller et.al following a 10-week strength training program in young college-aged males (29). Another study looked at these effects of resistance and endurance training in healthy, non-obese or diabetic young women. Results showed that while insulin sensitivity increased with both resistance and endurance training following the last training session, this phenomenon did not persist in the resistance training group when the glucose disposal rate was expressed per kg fat-free mass (33).

Acute bouts of resistance exercise also promote insulin sensitivity and glucose tolerance in healthy individuals. In a study focusing on the effects of a single bout of resistance exercise on insulin sensitivity and glucose tolerance in three different groups - young control subjects, older patients with non-insulin dependent diabetes mellitus (NIDDM), and older age-matched control subjects – participants completed a pre-OGTT, followed 48 hours later by a resistance exercise bout consisting of 3 10-rep sets on 7 different machines, followed again by a post-OGTT 18
hours later. While there were no changes in the pre-to-post glucose responses in any of the groups, the total insulin responses in the young control and the NIDDM groups were significantly lower post-exercise, showing that insulin sensitivity can be improved without affecting glucose tolerance (9). In comparison to a non-exercise trial, 30 minutes of resistance exercise also lowers insulin levels by 30% in overweight and obese individuals following a high-fat meal 14 hours post-exercise (18). However, in another study examining the effects of a single bout of resistance exercise on insulin sensitivity and glucose tolerance in postmenopausal women, no significant differences in insulin and glucose levels were observed between the non-exercise and exercise trials (7).

Aerobic Exercise: Similarly, recent findings have supported the benefits that prior aerobic exercise can have on insulin sensitivity and glucose tolerance. As mentioned previously, endurance training in healthy, non-obese or diabetic young women showed that insulin sensitivity increased and persisted for 3-5 days following the last exercise session (33). 30 minutes of acute aerobic exercise also lowers plasma glucose and insulin levels 14 hours post-exercise following a high-fat meal in comparison to a non-exercise (18). Weiss et. Al examined the effects of 60 minutes of aerobic exercise on postprandial glucose and insulin levels (40). In this study, participants completed two trials – a 60-minute aerobic exercise trial and a non-exercise trial. Subjects ingested a high-sugar meal 48-hours after the non-exercise trial, and 17-hours post-exercise. In comparison to the non-exercise trial, glucose and insulin areas under the curve were shifted downward 17-hours post-exercise.

A separate study in nine moderately trained middle-aged adults showed that five consecutive days of aerobic exercise (45 min at 73% of their VO2max) lowered blood glucose responses to an OGTT (24). In this study, five days of exercise were followed by An OGTT (75g glucose) was performed immediately after exercise on the fifth and final day, and 1, 3, 5, and 7 days thereafter. Blood glucose levels were significantly lower on days 1 and 3 compared to days 5 and 7. The plasma insulin area under the curve was initially higher immediately post exercise, but were significantly lowered on days 1 and 3, and returned to normal levels after 5 days of inactivity. These findings support that aerobic exercise lowers postprandial blood glucose and insulin levels for 1-3 days following exercise. However, unchanged glucose and insulin levels
with a hyperinsulinemic euglycemic clamp have also been reported in healthy, endurance-trained men following an acute bout of aerobic exercise (28).

While these studies show that both acute aerobic and resistance exercise increase insulin sensitivity and glucose tolerance for 14-17 hours post-exercise, subjects were healthy and recreationally active. Such populations are not at increased risk for the development of CVD or metabolic diseases. There is an increased need for studies focusing on the effects of different exercise modalities on overweight and obese populations to determine the efficacy of lifestyle strategies to attenuate PPH and hyperinsulinemia, which may lower CVD risk.

**Purpose Statement and Hypothesis:** The primary purpose of this investigation is to determine the effects of prior exercise and different exercise modalities on postprandial glucose and insulin responses following an OGTT in overweight and obese individuals. Compared to a non-exercise control trial, we hypothesize that a single bout of aerobic or resistance exercise performed 14-17 hours prior will attenuate PPH responses induced by an OGTT and increase insulin sensitivity in overweight and obese individuals.
Chapter 3
Methodology

**Study Design:** The protocol for this study was approved by the Institutional Review Board at Miami University and written informed consent was obtained from all participants. Participants completed three randomized study trials in a cross-over design: 1) acute aerobic exercise, 2) acute resistance exercise, and 3) a control (non-exercise) trial. Each study trial consisted of two visits to the laboratory. The first visit occurred in the late afternoon/early evening and was preceded by a 2-hour fast and no exercise for 48 hours. Following the first visit, participants left the laboratory and returned the next morning (14-17 hours later) after an overnight fast (>10 hours). During each morning visit, blood was collected prior to and at 30-minute intervals following the ingestion of an OGTT.

**Participants:** Twelve (n = 12) overweight and obese men and premenopausal women were recruited to participate in this study. A phone screening was conducted to determine initial eligibility. Participants were required to meet the following inclusion criteria: 18-50 years of age, non-smokers, BMI ≥25 kg/m², alcohol intake <3 drinks/day or <10 drinks/week, not taking any anti-hypertensive, lipid-lowering, and/or anti-diabetic medications, not taking any dietary supplements, no musculoskeletal injuries or physical limitations affecting ability to exercise, weight stable (±2 kg) past 3 months, no history or symptoms of cardiovascular, pulmonary, metabolic, or neurological disease, and <2 days/week of moderate-intensity physical activity over past 6 months.

**Screening Visit:** Following the initial phone screening, individuals determined to be eligible for the study were scheduled for an in-person screening visit after an overnight fast and no exercise for 48 hours. During this visit, participants were given a detailed explanation of the study, signed an informed consent document, and completed a health history and physical activity questionnaire. The International Physical Activity Questionnaire (IPAQ) was administered to estimate participants’ physical activity levels. Next, height and waist circumference were measured using standard procedures. Body mass and composition were measured using bioelectrical impedance analysis (InBody 770, Cerritos, CA, USA). Heart rate and blood pressure were recorded two times following 5 minutes of seated rest using an automated blood pressure monitor (Omron HEM907XL, Bonnockburn, IL, USA). A fasted blood sample was
obtained from an antecubital vein for the determination of blood glucose and lipids (Cholestech). Participants then completed a graded exercise test to volitional fatigue on a motor driven treadmill to determine maximal oxygen consumption (VO2max). Expired air was collected and analyzed using a calibrated Parvomedics True One 2400 Metabolic System (ParvoMedics, Sandy, UT, USA). Participants must have met two of the following criteria to determine if VO2max was achieved: RER ≥ 1.1, ±10 bpm of age-predicted maximum HR, RPE ≥ 18 (6 to 20 scale), and plateau of VO2 with an increase in work-rate (27).

**Familiarization Visit:** Approximately 1 week following the screening visit, participants reported to our laboratory for a familiarization session with the resistance exercise equipment and determination of 10-repetition maximum (RM) in the following exercises: seated leg press, seated chest press, seated leg curl, lat pulldown, seated shoulder press, and seated row exercises. Participants were instructed to refrain from strenuous physical activity for 48 hours prior to the familiarization visit. Following a 5-minute warm-up on the treadmill, an initial set of 10 repetitions was performed with a light load. The participant then performed successive sets separated by 90 seconds with increasing loads until the study personnel determined the 10-RM load. Each participant’s 10-RM load was determined within 3-5 sets.

**Study Trials:** Participants completed three randomized study trials in a cross-over design: 1) acute aerobic exercise, 2) acute resistance exercise, and 3) a control (non-exercise) trial. The first study trial occurred approximately 1 week following the familiarization visit. Study trials were separated by ≥1 week. Each study trial was conducted over 2 days and consisted of a late afternoon/early evening visit and a morning visit 14-17 hours later (**Figure 1**). During the late afternoon/early evening visit, participants completed either aerobic or resistance exercise or a non-exercise control visit.

A 5-minute warm-up on the treadmill at a self-selected speed preceded each acute exercise bout. During the acute aerobic exercise bout, participants completed 30 minutes of continuous treadmill exercise at approximately 60% VO2max. During the acute resistance exercise bout, participants completed six exercises in the following order: seated leg press, seated chest press, seated leg curl, lat pulldown, seated shoulder press, and seated row exercises. Each exercise was performed for 3 sets of 10 repetitions per set, with the load corresponding to the previously determined 10-RM load. The resistance exercise bout lasted approximately 30 minutes. A 5-minute cool-down on the treadmill at a self-selected speed concluded each acute
exercise bout. The non-exercise control trial consisted of 40 minutes of quiet rest in the laboratory. Heart rate and rating of perceived exertion (RPE) were assessed at rest during each trial, at 10-minute intervals during the control trial, at 5-minute intervals during the aerobic exercise trial, and following each set during the resistance exercise trial.

Participants reported back to the laboratory the next morning (14-17 hours later) after an overnight fast. Body mass and composition were re-assessed, and a flexible catheter was inserted into a forearm vein following 15 minutes of supine rest. A baseline blood sample was obtained, immediately followed by ingestion of an OGTT (1 g/kg body mass; TRUTOL, Fisher Diagnostics). Blood samples were obtained at 30-minute intervals during the 3-hour postprandial period.

Figure 1. Timeline of study measures. Blood samples were obtained 14-17 hours following no exercise (control), aerobic exercise, or resistance exercise, and at 30-minutes intervals following a 1g/kg OGTT.

Blood Analysis: Whole blood was collected at each time point into evacuated tubes. Blood glucose was measured using the Contour Next blood glucose monitoring system (Ascensia Diabetes Care, Parsippany, NJ). Plasma was obtained by centrifugation (2000 x g, 15 minutes, 4°C) and transferred to cryogenic vials, which were stored at -80°C until analyses were completed. Plasma glucose was measured using a commercially available clinical assay (Pointe Scientific, Canton, MI, USA) on a microplate reader (BioTek Instruments, Synergy HT,
Plasma insulin was measured via enzyme-linked immunosorbent assay (ALPCO, Salem, NH). Due to issues with the intravenous line, blood was not collected during three postprandial trials (8% of total trials). For these trials (one control, one aerobic, and one resistance trial), whole blood was collected from the fingertip and glucose analyzed using the Contour Next blood glucose monitoring system (Ascensia Diabetes Care, Parsippany, NJ). This monitoring system has been validated against other techniques to determine blood glucose concentration (4, 32). Insulin and glucose area under the curve (AUC) were measured using the trapezoidal method. The homeostatic model assessment of insulin resistance (HOMA-IR) was calculated to evaluate insulin resistance (26). The Matsuda Index (MI), a measure of whole body insulin sensitivity that is strongly correlated with insulin sensitivity determined via the hyperinsulinemic-euglycemic clamp method, was calculated (25).

**Statistical Analyses:** One-way ANOVA was used to evaluate between trial differences in fasting plasma glucose and insulin. Two-way repeated-measures ANOVA were used to evaluate differences in insulin and glucose levels due to time, trial, and their interaction. If the presence of significant main or interaction effects, pairwise differences within and between groups were evaluated using LSD post-hoc tests. One-way ANOVA was used to evaluate AUC between trials. An $\alpha$-level of $P \leq 0.05$ was considered statistically significant for all analyses.
Chapter 4

Results

Participants and Dietary Intakes: Eleven (n = 3 males, 8 females) overweight and obese participants completed the study (Table 1). Participants were normotensive, obese on the basis of BMI, and physically inactive (<1 d/wk of moderate-intensity physical activity). Energy and nutrient intakes did not differ between trials (Table 2), indicating that participants had similar dietary intakes the day before each trial.

Study Trials: Body mass did not differ between trials (97.2 kg, 97.9 kg, and 98.4 kg for trials 1, 2, and 3, respectively; P>0.05). All trials (exercise and control) were performed between 3:00 and 6:00 p.m., which corresponded to 15.3 ± 1.0 h prior to the OGTT performed the next morning. Participants completed the 30 min of treadmill exercise at 58.7 ± 5.5% VO_{2}\text{max}.

Average RPE did not differ between the aerobic and resistance exercise trials (12.6 ± 1.8 and 13.5 ± 1.1 for aerobic and resistance exercise, respectively; P>0.05), indicating similar intensity between the acute exercise bouts.

Postprandial glucose and insulin responses: Fasting plasma glucose and insulin did not differ between trials (Table 3). A significant main effect for time (P<0.001), but no trial (P = 0.49) or interaction (P = 0.33) effect, was found for plasma glucose concentrations. Plasma glucose remained elevated above baseline through 120 min during the three trials (Figure 2) Compared to baseline, plasma glucose increased by 29-66%, 22-55%, and 25-63% in the control, aerobic, and resistance trials, respectively. A significant main effect for time (P<0.01), but no trial (P = 0.26) or interaction (P = 0.25) effect, was found for plasma insulin concentrations. Plasma insulin remained elevated above baseline (P<0.05) through 180 min during the three trials (Figure 3). Change from baseline was calculated for plasma glucose and insulin but findings did not differ from absolute values. Glucose AUC (Figure 4), insulin AUC (Figure 5), HOMA-IR, and the Matsuda Index (Table 3) did not differ between trials.
Chapter 5

Discussion

To our knowledge, our study is the first to examine the influence of different exercise modalities on changes in postprandial glucose tolerance and insulin sensitivity following an OGTT in overweight and obese young adults. Sedentary, overweight and obese individuals were chosen for this study as they are at increased CVD risk and constitute a large percentage of the US population (43). Further, obese individuals exhibit exaggerated postprandial glucose (20, 21, 31) and insulin responses (20, 21), suggesting that acute exercise may attenuate postprandial metabolic disturbances to a greater extent compared to previous observations in healthy individuals (1, 24, 40). The primary finding of our study was that acute aerobic or resistance exercise performed the evening prior to an OGTT did not significantly alter fasting or postprandial glucose and insulin responses in overweight and obese young adults.

Prior studies examining the effects of acute exercise on postprandial blood glucose and insulin responses have produced conflicting results. Some studies have shown that acute aerobic or resistance exercise performed 14-17 hours prior to a high-carbohydrate challenge lowers PPH and postprandial insulin responses in healthy adults (1, 24, 40). However, others have shown no significant effect of acute resistance exercise performed the prior evening on postprandial glucose and insulin responses (7, 9). Relative to pre-exercise measures, one study (9) found no differences in blood glucose responses to a 75g OGTT ingested 18 hours following an acute bout of resistance exercise in seven young healthy adults, three healthy older adults, and seven non-insulin dependent diabetics. Interestingly, postprandial insulin responses measured as insulin AUC were significantly lower following resistance exercise in young adults and diabetics, highlighting that acute resistance exercise lowers blood insulin responses independent of changes in PPH (9). In a separate study, compared to a non-exercise control trial, no differences in blood glucose and insulin responses to an intravenous glucose tolerance test were found after an acute bout of whole-body resistance exercise performed 15 hours prior in 10 overweight postmenopausal women (7). The authors attributed their findings to age and/or sex differences that may have influenced the amount of skeletal muscle utilized during the exercise bouts. Skeletal muscle contraction during exercise increases GLUT-4 translocation to the cell membrane, resulting in greater uptake of blood glucose (16, 36). This effect has been shown to
persist for up to 22 hours post-exercise (14). Men have a greater skeletal muscle mass compared to women and skeletal muscle mass decreases with age. Thus, it is possible that age and/or sex differences in skeletal muscle mass resulted in decreased GLUT-4 translocation in overweight postmenopausal women (7), thereby mitigating any beneficial influence of acute exercise on postprandial glucose tolerance and insulin sensitivity.

While many studies have examined the effects of acute exercise on postprandial responses in healthy individuals (1, 24, 40), few have examined populations at increased risk for chronic disease. Overweight and obese individuals are at a greater risk for the development of CVD, which can partially be explained by impairments in glucose tolerance and insulin sensitivity (20, 21, 31). The insulin signaling pathways present in skeletal muscle have been shown to be interrupted with increases in adipose tissue, resulting in impaired GLUT-4 activation (11). Indeed, previous research has shown a decrease in insulin-stimulated GLUT-4 activation and subsequent glucose transport in overweight and obese individuals compared to healthy individuals (34). Increased skeletal muscle activity during exercise may mitigate impairments attributed to increased adipose tissue, highlighting exercise as an lifestyle strategy to attenuate exaggerated PPH responses that may contribute to the development of CVD (8).

We are aware of only one previous study that examined the effects of acute exercise, as well as different exercise modalities, on postprandial responses in overweight and obese individuals. Compared to a non-exercise control condition, Ho et al. (17) found no significant different in blood glucose AUC responses to a high-fat meal consumed 14 hours after performing 30 minutes of aerobic exercise, 30 minutes of resistance exercise, or 30 minutes combined exercise in 18 middle-aged overweight and obese individuals (58.7 y; BMI = 31.7 kg·m⁻²). Interestingly, while insulin levels were not significantly different in any of the exercise conditions compared to the non-exercise control condition, insulin AUC was significantly lower following resistance exercise compared to aerobic exercise. In the present study, insulin AUC was lower in the resistance exercise trial compared to the aerobic exercise and control trials, albeit not significantly. Thus, it is possible that acute resistance exercise is more effective at improving postprandial insulin sensitivity relative to aerobic exercise of similar duration, perhaps due to differences in exercise intensity between modalities and/or increased muscle mass utilization. Future research is warranted to determine the effects of different exercise modalities.
matched for intensity and duration on postprandial metabolic disturbances in clinical populations.

**Limitations:** We examined young overweight and obese adults in the present study. Future studies are needed to determine if age, and thus greater duration of excess body mass, influence the effect of acute exercise on postprandial glucose tolerance and insulin sensitivity. Additionally, participants in the current study performed acute exercise for a shorter duration compared to previous studies in healthy adults (1, 24, 40). We purposefully chose to perform acute aerobic and resistance exercise for 30 min as this duration meets current recommendations (10) and is more representative of that performed by physically-inactive adults.

**Conclusion:** We found that performing an acute bout of aerobic or resistance exercise the prior evening did not significantly lower postprandial glucose or insulin responses in overweight and obese adults. These data suggest that the lasting benefits of exercise on glucose tolerance and/or insulin sensitivity observed in healthy adults (1, 24, 40) do not necessarily extend to individuals at heightened risk for chronic disease. Future studies should be conducted to evaluate the efficacy of strategies (e.g., different exercise modalities, timing of exercise, dietary strategies) to attenuate the deleterious health effects of postprandial metabolic disturbances. Additionally, future research should examine whether age and sex influence the effects of acute exercise on postprandial metabolism.
TABLE 1. Participant Characteristics.

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<tr>
<td>Resting heart rate (bpm)</td>
<td>77 ± 12.0</td>
</tr>
<tr>
<td>VO₂max (ml·kg·min⁻¹)</td>
<td>31.7 ± 5.8</td>
</tr>
<tr>
<td>TC (mg·dL⁻¹)</td>
<td>173.6 ± 38.4</td>
</tr>
<tr>
<td>HDL-C (mg·dL⁻¹)</td>
<td>57.5 ± 19.5</td>
</tr>
<tr>
<td>LDL-C (mg·dL⁻¹)</td>
<td>90.3 ± 33.7</td>
</tr>
<tr>
<td>TG (mg·dL⁻¹)</td>
<td>118.8 ± 80.2</td>
</tr>
<tr>
<td>Glucose (mg·dL⁻¹)</td>
<td>92.3 ± 8.6</td>
</tr>
</tbody>
</table>

Data are mean ± SD (n = 11). Abbreviations: BMI, body mass index; DBP, diastolic blood pressure; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; SBP, systolic blood pressure; TC, total cholesterol; TG, triglycerides; VO₂max, maximal oxygen consumption.
### TABLE 2. Participants’ dietary intakes

<table>
<thead>
<tr>
<th>Variable</th>
<th>Resistance</th>
<th>Aerobic</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total energy (kcal/d)</td>
<td>2218 ± 843</td>
<td>2153 ± 744</td>
<td>2131 ± 781</td>
</tr>
<tr>
<td>Carbohydrate (%)</td>
<td>44.9 ± 7.1</td>
<td>45.8 ± 7.6</td>
<td>43.5 ± 6.6</td>
</tr>
<tr>
<td>Protein (%)</td>
<td>15.8 ± 5.7</td>
<td>15.1 ± 3.8</td>
<td>15.6 ± 4.2</td>
</tr>
<tr>
<td>Fat (%)</td>
<td>38.7 ± 6.7</td>
<td>38.3 ± 6.3</td>
<td>39.8 ± 5.2</td>
</tr>
<tr>
<td>Saturated Fat (g/d)</td>
<td>32.4 ± 16.6</td>
<td>30.2 ± 12.2</td>
<td>30.2 ± 13.4</td>
</tr>
<tr>
<td>Cholesterol (mg/d)</td>
<td>221.1 ± 158.6</td>
<td>201.0 ± 155.4</td>
<td>205.3 ± 158.7</td>
</tr>
</tbody>
</table>

Data are means ± SD, n = 11. Dietary intakes were determined from food records for the 1-day preceding each intervention trial.

### TABLE 3. Fasting and postprandial plasma glucose and insulin responses.

<table>
<thead>
<tr>
<th>Variable</th>
<th>Resistance</th>
<th>Aerobic</th>
<th>Control</th>
</tr>
</thead>
<tbody>
<tr>
<td>Fasting glucose (mg/dL)</td>
<td>95.4 ± 8.8</td>
<td>96.1 ± 8.3</td>
<td>95.5 ± 8.9</td>
</tr>
<tr>
<td>Fasting insulin (µU/ml)</td>
<td>9.7 ± 6.6</td>
<td>10.1 ± 7.0</td>
<td>10.0 ± 6.8</td>
</tr>
<tr>
<td>Matsuda Index</td>
<td>4.0 ± 2.1</td>
<td>3.5 ± 1.6</td>
<td>3.7 ± 2.0</td>
</tr>
<tr>
<td>HOMA-IR</td>
<td>2.3 ± 1.6</td>
<td>2.4 ± 1.7</td>
<td>2.4 ± 1.6</td>
</tr>
</tbody>
</table>

Data are means ± SD, n = 11. Abbreviations: AUC, area under the curve; HOMA-IR, Homeostatic Model Assessment of Insulin Resistance.
Figure 2. Plasma glucose responses for the control and exercise trials (n = 11, mean ± SE).

*P<0.01 from baseline.
Figure 3. Plasma insulin responses for the control and exercise trials (n = 11, mean ± SE).

*P<0.01 from baseline.
Figure 4. Plasma glucose area under the curve (AUC) for control and exercise trials. (n = 11, mean ± SE).
Figure 5. Plasma insulin area under the curve (AUC) for control and exercise trials. (n = 11, mean ± SE).
References


