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Significant Enhancements in Glucose Tolerance and Insulin Action in Centrally Obese Subjects Following Ten Days of Training

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Objective: The objective of the study was to determine the effects of short-term exercise on glucose tolerance and insulin response to a glucose load in centrally obese individuals.

Design: 75 g oral glucose tolerance tests (OGTT) were performed prior to participation and 24 hours after the last exercise session. Exercise bouts were 40 minutes in duration and consisted of treadmill walking and cycle ergometry at 70–80% of age-predicted maximum heart rate ($APHR_{max}$).

Participants: Eleven sedentary, centrally obese men [mean (SE): Mass, 119.1 (5.4) kg; BMI, 37.7 (1.8) kg/m^{-2}; waist-to-hip ratio (WHR), 0.97 (0.01); age 31.7 (2.4) years] were studied before and after 10 days of aerobic exercise training.

Results: No significant change ($p > .05$) in body mass was noted following 10 days of exercise as compared with preparticipation [119.1 (5.4) kg versus 118.9 (5.4) kg]. Fasting plasma glucose concentration was significantly lower ($p < 0.05$) following 10 days of exercise as compared with preexercise [5.58 (0.15) mmol/L versus 5.27 (0.12) mmol/L]. No significant change ($p > 0.05$) in fasting plasma insulin concentration, however, was observed following 10 days of exercise training as compared with preexercise [276.2 (33.7) pmol/L versus 225.3 (35.9) pmol/L]. Plasma insulin concentrations at 60 minutes and 120 minutes were significantly decreased ($p < 0.05$) when comparing the preexercise to the postexercise OGTT [60: 1264.2 (88.3) pmol/L versus 1103.5 (81.1) pmol/L; 120: 1066.9 (110.5) pmol/L versus 764.1 (106.2) pmol/L]. Plasma glucose concentration at 120 minutes. was also significantly reduced ($p < 0.05$) after 10 days of exercise as compared with preexercise [6.09 (0.24) mmol/L versus 5.39 (0.22) mmol/L]. Area under the glucose curve was significantly ($p < 0.05$) reduced after 10 days of exercise as compared with preparticipation [944.6 (44.4) mmol/L/120 min versus 884.4 (43.2) mmol/L/120 min]. Area under the insulin curve was also significantly decreased ($p < 0.05$) following 10 days of exercise training as compared with preexercise [126,890 (9014.0) pmol/L/120 min versus 109,445 (7,888.9) pmol/L/120 min].

Conclusions: These data suggest that short-term exercise may improve glucose tolerance and insulin response to a glucose load in centrally obese men.

Key Words: Obesity—Exercise—Glucose intolerance—Insulin—Glucose.

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INTRODUCTION

The obese state in general, and more specifically abdominal obesity, is the result of a prolonged, positive energy balance and is known to be associated with a sedentary lifestyle. Furthermore, it has been shown to accentuate dyslipidemias insulin resistance, and the deterioration of glucose tolerance, all of which are very prevalent in centrally obese individuals. As such, abdominal obesity may be clinically more important than obesity per se since it may predispose individuals to an increased risk of developing Type 2 diabetes mellitus (T2DM) and coronary heart disease (CHD).[1–3]

Changes in glucose tolerance have also been associated with aging.[3,4] Deterioration in glucose tolerance with aging may occur as a result of either insulin resistance or impaired beta cell function.[5] It has been suggested, however, that this age-related decline in glucose tolerance may be a consequence of obesity and/or sedentary lifestyle.[6,7] Furthermore, Kohrt et al.[7] have shown that the decreased glucose tolerance in obese individuals most likely occurs as a result of insulin resistance, which is typically present in this population.

Long-term exercise training has been previously shown to improve glucose tolerance and insulin response to an oral glucose tolerance test (OGTT) in overweight and untrained individuals.[8–10] Regular exercise, however, may result in weight loss and change in adiposity.

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Holloszy et al.[11] reported a significant reduction in weight after long-term training, whereas Smutok et al.[10] reported a significant decrease in percent body fat following several months of aerobic exercise training. Weight loss has been previously shown to improve glucose tolerance and insulin sensitivity in overweight subjects.[8] As such, the observed changes in glucose tolerance and insulin response to a glucose load could be due to a possible interaction between exercise and reduction in adiposity.

The effects of short-term exercise on glucose tolerance and insulin response to a glucose load have not been well characterized. Some studies have reported significant improvements in glucose tolerance and/or insulin response to glucose,[12,13] whereas others have failed to show significant exercise-induced reductions in either glucose tolerance or insulin responsiveness.[14,15] These discrepancies may be related to differences in various factors, which include differences in baseline glucose tolerance and insulin responsiveness, differences in subject somatotype, and differences in exercise training protocols (i.e., intensity, duration) used. Furthermore, all of these studies used fairly vigorous or prolonged exercise regimens. Most obese individuals are sedentary and not able to participate in demanding exercise programs. As such, it may be of particular interest if beneficial effects on glucose tolerance and insulin sensitivity could be achieved at a low exercise intensity. Therefore, we investigated the effects of 10 days of moderate intensity aerobic exercise training on glucose tolerance and insulin response to an OGTT in sedentary men with abdominal obesity and normal baseline glucose tolerance.

METHODS

Recruitment of Subjects

Eleven sedentary male individuals who had a central pattern of obesity, and were between the ages of 20 and 45, volunteered to participate as subjects in this investigation. Subjects in this investigation served as their own controls. None of the subjects demonstrated diabetes or impaired glucose tolerance as defined by the American Diabetes Association[16] in response to an OGTT. The subjects' physical characteristics are presented in Table 1. All subjects gave their written consent to participate. The research protocol was approved by the University of Southern Mississippi's Institution Review Board for Human Subjects Research. Only sedentary, apparently healthy individuals who were not taking medications or hormone therapy participated in the study.

TABLE 1. *Subject characteristics (n = 11)*

Characteristics	Mean (SE)
Age (yrs)	31.7 {2.4)
Body mass (kg)	119.1 (5.4)
BMI ($kg.m^{-2}$)	37.7 (1.8)
WHR	0.97 (0.01)
Daily caloric intake ($kJ.d^{-1}$)	11,462 (2729)
% CHO	46 (2.3)
% Fat	37 (1.6)
% Protein	17 (1.0)
Caloric expenditure per exercise session (kJ)	1,837 (70.5)

BMI, body mass index; WHR, waist-to-hip ratio; CHO, carbohydrate.

Screening/Orientation

After written consent was obtained, each subject completed a medical history and activity profile questionnaire and underwent a 12-lead electrocardiogram (ECG) submaximal graded exercise test (GXT) to evaluate exercise tolerance and to rule out disorders that would preclude exercise participation. Anthropometric measurements including height, weight, body mass index (BMI), and waist-to-hip ratio (WHR) were also taken. Only sedentary, obese subjects with android-type obesity (WHR >0.95) and normal glucose tolerance as defined by the American Diabetes Association[16] qualified for participation. Each subject was instructed not to engage in any additional aerobic conditioning during the time period of the investigation. Moreover, each subject was told not to change his regular eating habits and to refrain from alcohol during the investigation.

Experimental Design

A pretest posttest design was used in this study. Subjects underwent a 10-day exercise intervention consisting of treadmill walking and ergometer cycling. Subjects exercised for 40 min/day at 70–80% of age-predicted heart rate max ($APHR_{max}$). All exercise trials were begun within 1 week after baseline fasting plasma samples were obtained and the baseline OGTT was administered. The subjects served as their own controls.

Exercise Trials

Each of the 10 consecutive exercise sessions consisted of 40 minutes of continuous exercise. The exercise sessions were performed on a Quinton Model Q55 motor-driven treadmill (Quinton, Bothell, WA, U.S.A.) with a model 3000 controller and on a Monark cycle ergometer (Monark Exercise AB, Vansbro, Sweden). Workload (grade/speed, resistance/pedal frequency) were adjusted to elicit 70–80% of $APHR_{max}$. This training protocol has been previously described.[17] Heart rate (HR) and blood pressure were monitored before, during, and after each session. HR was monitored to ensure that the subjects were exercising at the prescribed intensity. In addition, metabolic data were collected for 3–5 minutes at the 12-, 22-, and 32-minute marks during the exercise sessions for the purpose of determining energy expenditure during exercise. Metabolic data were recorded using a MedGraphics Cardiopulmonary Exercise System CPX (Med Graphics, St. Paul, MN, U.S.A.) and Compaq Data Acquisition Computer (Compaq, Houston, TX, U.S.A.). Prior to each exercise session, the ventilatory flow meter and gas (CO_2 and O_2) analyzers were manually calibrated against known concentrations of CO_2 and O_2. Subjects breathed through a two-way 2700 Hans-Rudolph (Hans-Rudolph, Inc., Kansas City, MO, U.S.A.) valve held in place by a 2785 Hans-Rudolph head support. The subject's diets were assessed during the 10 days of exercise by analysis of a 3-day dietary recall.

Blood Samples

Glucose tolerance was assessed on two separate occasions, prior to and at the conclusion of the study, using an OGTT. The postexperimental OGTT was administered within 24 hours of the last exercise session. Both OGTTs were performed after a 12-hour overnight fast. A Teflon intravenous (i.v.) catheter was placed in an antecubital vein from which five blood draws were taken. The i.v. line was kept patent by a regulated primary i.v. set microdrip with a 0.9% NaCl solution. A baseline sample was obtained immediately prior to the ingestion of Medi-Cola (8D, Franklin Lakes, NJ, U.S.A.) carbonated glucose tolerance test beverage containing 75 g of glucose. Following the ingestion of the glucose solution, blood draws were taken at 30, 60, 90, and 120 minutes. All the samples were collected in Vacutainer EDTA K_3 (15%) 7 mL blood collection tubes.

Baseline samples were used to determine fasting glucose and insulin levels. The samples taken during the 2-hour time period were used to evaluate the area under the insulin and glucose curves during the OGTT. Blood samples were immediately centrifuged in an International Clinical Centrifuge (International Equipment Co., Needham Heights, MA, U.S.A.) at 3,000 r.p.m. for 20 minutes. Following centrifugation, the plasma was immediately separated and stored in cryovials at −80° C until analyzed. Plasma glucose was analyzed using a Kodak Ektachem DT60 system (Eastman-Kodak Co., Rochester, NY, U.S.A.). Insulin levels were determined using a commercially available radioimmunoassay (RIA) kit (ICN, Costa Mesa, CA, U.S.A.). The mean intraassay coefficient of variation for insulin was 8.98%. The mean interassay coefficient of variation was 9.56%. To control for changes in blood volume with exercise, hematocrit (Hct) was determined pre- and posttraining by a microcapillary technique.[18]

Statistical Analysis

Differences between initial and final glucose and insulin concentrations during the OGTT were evaluated with a one-way analysis of variance (ANOVA) with repeated measures. Areas under the glucose and insulin curves during the OGTT were determined using a trapezoidal model. The difference between the initial and final OGTT glucose and insulin areas were analyzed with a Wilcoxon *t* test. The difference between the initial and final hematocrit (Hct) was also evaluated with a Wilcoxon *t* test. Statistical significance was accepted at the $p < 0.05$ level of confidence for all variables.

RESULTS

Diet was recorded during the 10 days of exercise by a 3-day dietary recall. Diet composition was analyzed using a computer analysis program by Ohio Distinctive Software (Ohio Distinctive, Columbus, OH, U.S.A.) (Table 1). Average caloric intake was 11,462 (2,729) kJ/day. Diet composition consisted of 46% CHO (carbohydrates), 37% fat, and 17% protein. Average energy expenditure per exercise session was 1,837 (70.5) kJ (Table 1).

There was no significant ($p > 0.05$) change in body mass following 10 days of exercise as compared with preparticipation [119.1 (5.4) kg versus 118.9 (5.4) kg]. Hct was not significantly ($p > 0.05$) different after 10 days of training as compared with preparticipation [44.6 (0.8) % versus 43.8 (1.0) %]. This suggests that venous blood variables (i.e., glucose and insulin) were not influenced by changes in vascular volume.

Fasting plasma glucose was significantly ($p < 0.05$) decreased following 10 days of exercise as compared with preexercise [5.58 (0.15) mmol/L versus 5.27 (0.12) mmol/L]. There were no significant ($p > 0.05$) changes in plasma glucose concentrations at the 30-, 60-, and 90-minute time points during the OGTT [30: 9.36 (0.50) mmol/L versus 9.07 (0.51) mmol/L; 60: 9.02 (0.59) mmol/L versus 8.32 (0.62) mmol/L; 90: 7.29 (0.44) mmol/L versus 6.76 (0.42) mmol/L], when comparing the pretraining to posttraining OGTT. Plasma glucose concentration was significantly ($p < 0.05$) decreased at the 120-minute time point during the OGTT after 10 days of training as compared with preparticipation [6.09 (0.24) mmol/L versus 5.39 (0.22) mmol/L]. Pretraining and posttraining glucose tolerance curves are presented in Figure 1. Area under the glucose curve was significantly ($p < 0.05$) reduced after 10 days of exercise as compared with preparticipation [944.6 (44.4) mmol/L/120 min versus 884.4 (43.2) mmol/L/120 min]. Means and SE for fasting glucose and glucose concentrations during the OGTT (30, 60, 90, 120 minutes) are presented in Figure 1.

Fasting plasma insulin showed a 17.0 (7.5) % reduction following 10 days of training as compared with pre-

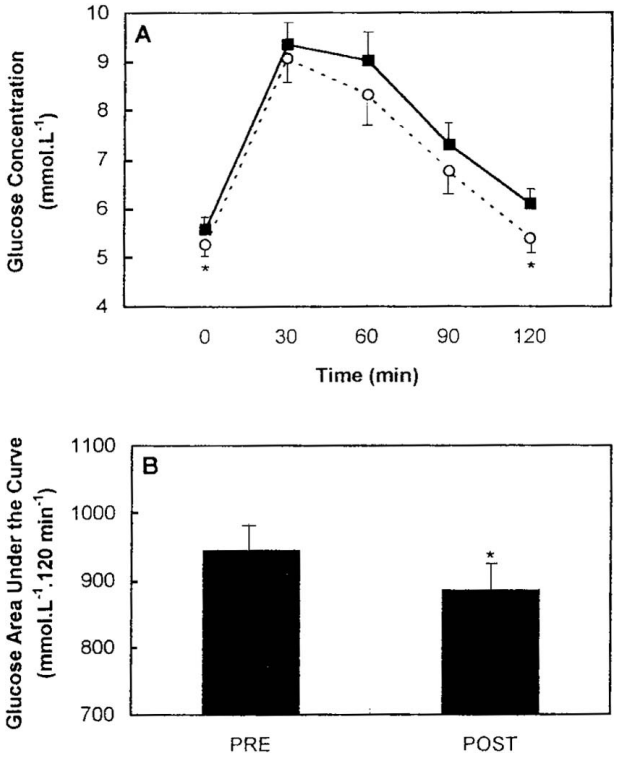

FIG. 1. Glucose tolerance. **A:** Plasma glucose concentration during 75 g oral glucose tolerance test before (solid lines) and after (dashed lines) 10 days of exercise. **B:** Glucose area under the curve before and after training. Values are means ±SE. *$p < 0.05$.

participation [276.2 (33.7) pmol/L versus 225. 3 (35.9) pmol/L]. This change however, did not attain statistical significance ($p > 0.05$). There were no significant ($p > 0.05$) changes in plasma insulin concentrations at the 30- and 90-minute time points during the OGTT [30: 1153.7 (68.9) pmol/L versus 1,101.4 (91.1) pmol/L; 90: 1,140.1 (125.6) pmol/L versus 949.3 (101.9) pmol/L], when comparing the pretraining to posttraining OGTT. Plasma insulin concentrations at the 60-minute and 120-minute time points during the OGTT were significantly ($p < 0.05$) reduced following the 10 days of exercise training as compared with preexercise [60: 1,264.2 (88.3) pmol/L versus 1,103.5 (81.1) pmol/L; 120: 1,066.9 (110.5) pmol/L versus 764.1 (106.2) pmol/L]. Pretraining and posttraining insulin areas under the curves are presented in Figure 2. Insulin response to a glucose load was significantly ($p < 0.05$) reduced following 10 days of exercise when comparing the preexercise OGTT to the postexercise OGTT [126,890 (9,014.0) pmol/L/120 min versus 109,445 (7,888.9) pmol/L/120 min]. Means and SE for fasting insulin and insulin responses to a glucose load during the OGTT (30, 60, 90, 120 minutes) are presented in Figure 2.

DISCUSSION

This investigation examined the effects of a 10-day exercise regimen, without concomitant mass loss, on glucose tolerance and insulin response to a glucose load in sedentary, centrally obese males. The results demonstrated 1) a significant reduction in the area under the glucose curve during an OGTT, 2) a significant reduction in the area under the insulin curve during an OGTT, and 3) clinically favorable changes in fasting concentrations of glucose and insulin.

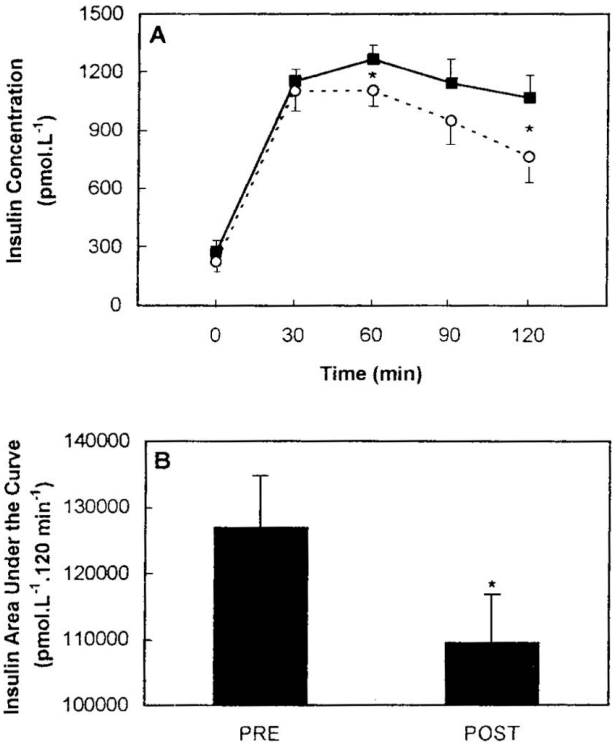

FIG. 2. Insulin responsiveness. **A:** Plasma insulin responses to a 75 g oral glucose tolerance test before (solid lines) and after (dashed lines) 10 days of exercise. **B:** Insulin area under the curve before and after training. Values are means ±SE. *$p < 0.05$.

Regular exercise training has been previously shown to improve glucose tolerance and insulin response to a glucose load in untrained individuals.[8,10,11] Chronic exercise training, however, may result in significant loss of body mass or change in body composition. Loss of body mass has been frequently linked to significant changes in fasting insulin,[8,19] fasting glucose,[8] glucose tolerance, and insulin responses to an OGTT.[8] As such, the observed changes in glucose tolerance and insulin responsiveness following endurance training may have been due, in part, to reductions in mass and/or adiposity.

Previous studies, which have investigated the effects of short-term exercise training on glucose tolerance in sedentary, obese individuals with normal baseline glucose tolerance, have not reported improvements in glucose tolerance.[15,20,21] For example, Gavigan et al.[20] observed no significant change in glucose tolerance in obese men following 7 days of exercise for 60 minutes at 66% of $VO_{2\ max}$, whereas Kang et al.[15] reported no change in glucose tolerance in obese, nondiabetic men after 1 week of exercise at both higher (70% $VO_{2\ peak}$) and lower (50% $VO_{2\ peak}$) exercise intensities. These findings are in contrast to those findings of the present investigation in which 10 days of exercise training resulted in a significant improvement in glucose tolerance in obese men, as evidenced by a decrease in the area under the glucose curve. Interestingly, only Rogers et al.[13] also reported a significant improvement in glucose tolerance following a short-term exercise program (i.e., 7 days). It should be noted, however, that the subjects in this investigation were either impaired glucose tolerant (IGT) or Type 2 diabetes mellitus (T2DM). There are several possible explanations for the discrepancies among studies regarding the effects short-term training on glucose tolerance in the obese state. These include differences in body mass/somatotype, baseline glucose tolerance, exercise dosage, duration, and possibly sample size.

Despite the fact that changes in fasting glucose concentration and glucose tolerance typically have not been seen in sedentary individuals in response to short-term exercise training, improved fasting insulin concentration and/or insulin responsiveness as evidenced by the decrease in the area under the insulin curve during the OGTT[12,20,13–15,17,21,22] are common observations. We observed a 17% decrease in fasting plasma insulin concentration in response to 10 days of exercise training. Although not statistically significant, the magnitude of this change may be of clinical importance. Our findings are consistent with those by Cononie et al.[12] and Gavigan et al.,[20] who reported reductions in fasting plasma insulin concentrations of 15% and 27%, respectively. Furthermore, consistent with the findings of these studies mentioned above,[12,20] the present investigation resulted in a

12% decrease in insulin area under the curve during the OGTT.

This reduction in the insulin area under the curve during the OGTT can be explained primarily by reduced insulin concentrations at the 60-, 90-, and 120-minute time points of the OGTT. Specifically, plasma insulin concentrations were 11% lower at 60 minutes, 13% lower at 90 minutes, and 29% lower at 120 minutes. This exercise-induced reduction in insulin concentration observed during the latter portion of the OGTT has been reported in previous investigations as well.[12,13,15]

Subjects in the current investigation had elevated fasting insulin concentrations. This is not surprising since hyperinsulinemia is very prevalent in the obese state.[3,23] The high insulin levels demonstrated by these subjects are likely indicative of insulin resistance at the cells.[24,25] As such, the observed improvement in glucose tolerance at lower insulin levels is consistent with greater insulin sensitivity.

The mechanisms responsible for these clinically significant changes cannot be elucidated from this study since our study was not designed to address such questions. The improvement in glucose tolerance and the greater insulin sensitivity observed following chronic exercise training are mainly due to a decrease in adiposity and an increase in aerobic capacity.[11] The favorable changes in glucose tolerance and insulin action observed in this study, however, may be explained by changes in hexokinase and GLUT-4 (glucose transporter-4) total protein following training. Previous studies have shown significant increase in hexokinase and GLUT-4 total protein following similar training protocols.[26]

The present investigation did not result in any changes in body mass. Furthermore, as it has been shown by others,[13] the exercise dosage used in the present investigation likely does not elicit increases in aerobic capacity. It is reasonable, then, to speculate that the changes observed in glucose tolerance and insulin sensitivity were due to this acute training protocol and not due to adaptive responses associated with chronic training. Further research in this area should investigate the effects of this exercise protocol on muscle glycogen depletion since muscle glycogen depletion during exercise facilitates subsequent insulin stimulated glucose disposal.

These favorable changes in glucose tolerance and insulin response to a glucose load may help offset the deterioration in glucose tolerance associated with obesity and a sedentary lifestyle. Furthermore, these changes were observed following exercise training of moderate intensity and duration. The exercise dosages used in previous investigations have involved rather high intensity exercise and/or exercise of long duration. This type of higher intensity/longer duration exercise probably is not appropriate for the average, sedentary person and may be particularly difficult for obese individuals. It should be noted, however, that to enhance glucose tolerance and insulin sensitivity in obese individuals, exercise prescriptions should advocate daily exercise sessions until exercise-induced decreases in adiposity and increases in aerobic capacity occur.

Finally, the present investigation found fasting plasma glucose concentration to be significantly reduced following 10 days of exercise training. In addition, a substantial reduction (17%) in fasting insulin was also observed. These reductions in circulating levels of glucose and insulin were salutary changes following this exercise program and may be protective against coronary heart disease. Furthermore, since the prevalence of insulin resistance and its manifestations are higher among obese subjects than among those of normal weight, more exercise training studies should be performed on T2DM subjects.

In summary, 10 days of exercise resulted in clinically significant improvement in glucose tolerance and insulin response to a glucose load in sedentary centrally obese adult men with normal baseline glucose tolerance. Moreover, these improvements were observed without a concomitant reduction in body mass suggesting that these effects were not mediated by changes in body mass or adiposity. Finally, the present investigation has shown that these beneficial changes in glucose homeostasis can be attained at an appropriate exercise dosage for this population.

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