Effect of Exercise on Postprandial Insulin Responses in Mexican American and Non-Hispanic Women

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Postprandial insulin responses (integrated area under the curve) to an oral glucose load after a period of aerobic exercise and no exercise (control) were compared in sedentary normoglycemic Mexican American and non-Hispanic women pair-matched (n = 9) on total body fat mass (21.8 ± 3.5 kg). The age (27.4 ± 3.0 years), body mass index (BMI) (23.6 ± 1.4 kg/m^2), waist to hip ratio (WHR) (0.85 ± .02), waist circumference (83.5 ± 4.5 cm), lean mass (36.2 ± 1.5 kg), and maximal O_2 consumption ([$\dot{V}O_2$ max] 32.9 ± 1.6 mL · kg^{-1} · min^{-1}) were similar, although the centrality index (subscapular/triceps skinfolds) was significantly greater in Mexican Americans (0.88 ± 0.06 *v* 0.70 ± 0.05, $P < .01$). Exercise (treadmill walking for 50 minutes at 70% $\dot{V}O_2$ max) and control trials were performed 4 weeks apart and 5 to 12 days after the onset of menstruation. A 75-g oral glucose load was administered 15 hours after the completion of each trial, with the subjects 12 hours postprandial. Blood samples were drawn prior to glucose ingestion (fasting, 0 minutes) and at minutes 15, 30, 60, 90, 120, and 150 postingestion. The postprandial insulin response was calculated using a trapezoidal method. In Mexican Americans, significant ($P < .02$) reductions in the postprandial insulin response (exercise *v* control, 6.5 ± 1.0 *v* 8.5 ± 1.4 pmol/L · min · 10^4) and fasting insulin (exercise *v* control, 77.4 ± 7.0 *v* 88.5 ± 10.3 pmol/L) occurred after exercise compared with the control condition. In non-Hispanics, neither the postprandial insulin response (exercise *v* control, 7.2 ± 1.0 *v* 6.2 ± 0.9 pmol/L · min · 10^4) nor fasting insulin (exercise *v* control, 77.0 ± 8.2 *v* 82.9 ± 8.9 pmol/L) were significantly different between trials. The postprandial insulin response in the control trial was significantly correlated with the change in the insulin response (control minus exercise) in the 18 women ($r = .56$, $P = .01$). No trial or group differences were found for postprandial glucose and C-peptide responses. Mexican American women have a high risk of developing type 2 diabetes, and aerobic exercise may be valuable in the prevention or delay of onset of diabetes by reducing peripheral insulin resistance.

THE POSTPRANDIAL insulin response is an indirect measure of the amount of endogenous insulin secretion to remove a known quantity (typically 75 g) of orally ingested glucose. This response is quantified by calculating the integrated area under the response curve of the plasma insulin concentration over time from baseline (prior to glucose ingestion) to a predetermined endpoint, usually 120 to 150 minutes. The postprandial insulin response[1] and fasting insulin concentration are directly related to the degree of peripheral insulin resistance,[2] a state occurring in the skeletal muscle in which normal concentrations of insulin fail to adequately remove circulating glucose.[3] As a consequence of insulin resistance, additional pancreatic insulin secretion is required to achieve postprandial normoglycemia. Peripheral insulin resistance is an important determinant of blood glucose control, since skeletal muscle is the largest reservoir of postprandial glucose disposal in the human body.[4] Peripheral insulin resistance is clinically relevant because it is a precursor[4] to the development of type 2 diabetes mellitus.

Postprandial insulin responses in sedentary normoglycemic individuals are generally lower 12 to 15 hours following a single bout of aerobic exercise of 50 to 60 minutes in duration at an intensity of 70% to 80% maximal O_2 consumption ($\dot{V}O_2$max)[5-10] compared with a condition of no exercise. Although trained athletes have reduced postprandial insulin responses compared with sedentary individuals, 3 days of detraining resulted in increased postprandial insulin responses in athletes.[9] Thus, the effect of aerobic exercise to diminish the postprandial insulin response is short-lived and is largely attributed to the last bout of exercise. The magnitude of the exercise-induced reduction in the postprandial insulin response has been positively associated with the baseline (no exercise) postprandial insulin response.[11] However, the molecular mechanism for the acute exercise effect on the insulin response is unknown.

Mexican American women have a twofold to fivefold higher prevalence of type 2 diabetes relative to the general US population.[12,13] Several related physiological factors contribute to the increased diabetes risk in Mexican American women, including greater peripheral insulin resistance,[14] hyperinsulinemia, greater upper-body and total adiposity,[15] and an androgenic hormonal profile.[16] Peripheral insulin resistance in Mexican American women is evidenced by their 30% larger postprandial insulin response area[15] and 17% higher fasting insulin levels compared with non-Hispanics.[17] Although a substantial amount of research documents the greater risk of diabetes in Mexican American women, little is known about the acute effects of exercise on the postprandial insulin response in this population. Therefore, the purpose of this study was to compare the acute effects of a single bout of aerobic exercise on the postprandial insulin response and fasting insulin in sedentary normoglycemic Mexican American and non-Hispanic white women. The objectives were (1) to compare the postprandial insulin and accompanying glucose and C-peptide responses and the fasting insulin concentration in Mexican American and non-Hispanic women, (2) to evaluate the exercise effect on these responses in the two groups, and (3) to identify significant correlates of the baseline (no exercise) postprandial insulin response in women.

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SUBJECTS AND METHODS

Subjects

Seventeen Mexican American and 18 non-Hispanic women aged 18 to 41 years provided informed written consent to participate in the study and completed all aspects of the protocol. From this pool of participants, the women were pair-matched on total body fat mass based on dual-energy x-ray absorptiometry ([DEXA] Lunar DPX; Lunar, Madison, WI). Nine matched pairs were identified. Body fat mass was selected as the matching variable due to the association between adiposity and insulin resistance, as well as epidemiological data indicating a fourfold greater prevalence of obesity in Mexican American women versus non-Hispanic women.[18] Participants were apparently healthy, had no personal history of diabetes, and did not engage in regular (≥two times per week) aerobic or strength training during the study and for 3 months prior to the study. Normoglycemia (capillary whole-blood glucose < 5.6 mmol/L)[19] was verified by fingertip blood-sample screenings on two separate occasions after an overnight fast. The whole-blood glucose concentration was analyzed using a 2300 Stat Plus Glucose Analyzer (Yellow Springs Instruments, Yellow Springs, OH). Ethnic classification was based on the San Antonio Heart Study algorithm.[20] Participant characteristics are listed in Table 1 The study was approved by the Human Subjects Review Committee of Texas Woman's University.

Study Design

At least 1 week following a $\dot{V}O_2$max test, the participants completed the first of two randomly ordered trials (exercise or control), each with an exercise condition and an oral glucose tolerance test (OGTT). The two exercise conditions were a single bout of submaximal aerobic exercise and a control trial with no exercise. The control trial was used to establish a baseline postprandial insulin response. Each trial was completed during the follicular phase of the menstrual cycle (5 to 12 days after the self-reported onset of menses). Approximately 4 weeks separated the first and second trials. The exercise was performed in the laboratory between 3 and 7 PM. For the control trial, all participants reported to the laboratory between 3 and 7 PM but did not exercise. The participants returned to the laboratory 12 to 18 hours after each exercise condition and 12 to 15 hours after the last meal for an OGTT. The aerobic exercise consisted of walking at approximately 70% $\dot{V}O_2$max for 50 minutes on a motorized treadmill. Expired respiratory gas was collected and analyzed for a 5-minute period at the start of exercise and during minutes 20 to 25 and 40 to 45. The walking pace was 93.8 m/min (3.5 mph), with the elevation adjusted until 70% $\dot{V}O_2$max was reached. The treadmill elevation was 0.5% to 12.0% during the exercise protocol. Short practice workloads were performed before beginning the 50-minute exercise protocol to verify the intensity, and treadmill-elevation adjustments were made as necessary. The intensity of the practice workloads was less than the desired intensity of 70% $\dot{V}O_2$max. Within approximately 4 minutes from the start of the practice workload, the participants were ramped up to 70% $\dot{V}O_2$max. The practice time was not included in the total exercise time.

Table 1. Subject Characteristics

Characteristic	Non-Hispanic (n = 9)	Mexican American (n = 9)
Age (yr)	26.4 ± 2.6	28.4 ± 3.2
Height (cm)	165.0 ± 3.7	158.8 ± 1.8
Weight (kg)	63.4 ± 5.4	61.0 ± 4.7
BMI (kg/m^2)	23.0 ± 1.0	24.2 ± 1.8
WHR	0.83 ± 0.02	0.87 ± 0.03
Waist circumference (cm)	82.2 ± 4.5	84.8 ± 4.6
Centrality index	0.70 ± 0.05*	0.88 ± 0.06
Lean mass (kg)†	37.5 ± 1.9	34.8 ± 1.2
Fat mass (kg)†	21.3 ± 3.7	22.4 ± 3.4
Body fat (%)†	33.0 ± 2.6	36.3 ± 2.8
$\dot{V}O_2$max (mL · kg^{-1} · min^{-1})	34.2 ± 1.9	31.7 ± 1.3

NOTE. Values are the mean ± SE.
*$P < .01$.
†Based on DEXA.

Determination of $\dot{V}O_2$max

$\dot{V}O_2$max was determined by open-circuit spirometry during an incremental treadmill protocol with a computer-interfaced Ametek OCM-2 oxygen uptake system (Ametek, Pittsburgh, PA). The treadmill elevation began at 5% and was increased 5% every 2 minutes while the speed was maintained at 93.8 m/min (3.5 mph). Expired oxygen and carbon dioxide were analyzed by an Ametek oxygen (S-3A/I) and carbon dioxide (CD-3A) analyzer, respectively. Heart rate was continuously monitored using a Quinton Q4500 12-lead electrocardiogram (Quinton Instruments, Seattle, WA). The participants met at least two of the following criteria for determination of $\dot{V}O_2$max: an increase in oxygen consumption of 150 mL/min or less between the last two stages of work, a maximal heart rate within 10 beats of age-predicted maximum, and a respiratory exchange ratio of 1.1 or greater during the last stage of work.

Glucose Tolerance Testing

All OGTTs commenced between 7:30 and 9:30 AM with the participants 12 to 15 hours postabsorptive upon arrival at the laboratory. A catheter was inserted into an antecubital vein. Basal (0 minutes) blood samples were drawn for analysis of glucose, insulin, C-peptide, free fatty acids (FFAs), and sex hormone–binding globulin (SHBG). The participants then drank a 75-g glucose solution (Tru-Glu 100; Fisher Scientific, Pittsburgh, PA). Additional blood samples were drawn at 15, 30, 60, 90, 120, and 150 minutes after glucose ingestion. Between blood samples, the catheter was kept patent with 1 mL dilute sterile saline-heparin solution (10 μU/mL). Before drawing each sample, 1 mL venous blood mixed with the saline-heparin solution was removed via the catheter and discarded. It was assumed that this amount of heparin does not play a role in FFA release. All samples were gently mixed and then centrifuged at 4°C at 2,500 rpm for 8 minutes. A small amount of plasma was immediately analyzed for glucose with a 2300 Stat Plus Glucose/Lactate Analyzer. The remaining plasma was stored at −80°C for future analysis of insulin, C-peptide, FFA, and SHBG. Plasma insulin and C-peptide were analyzed by radioimmunoassay (RIA) using ICN Biomedicals (Costa Mesa, CA) Double Antibody I^{125} RIA Kits and Diagnostic Products (Los Angeles, CA) Double Antibody C-Peptide Kits. The intraassay and interassay coefficients of variation (CVs) for insulin were 4.8% and 6.9%, respectively. For C-peptide, the intraassay and interassay CVs were 7.0% and 11.3%, respectively. FFA levels were analyzed using the Wako (Osaka, Japan) in vitro enzymatic calorimetric method. The intraassay and interassay CVs for the FFA analysis were 2% and 5%, respectively. SHBG was analyzed by immunoradiometric assay (Farmos, Oulunsalo, Finland). Intraassay and interassay CVs for SHBG were 8.8% and 11.3%. A 10 detector gamma counter (RIASTAR 5410; Packard Instruments, Meriden, CT) was used to complete the assays of insulin, SHBG, and C-peptide. For each assay, all samples from the same individual were analyzed using kits with the same lot numbers.

Dietary Records

The participants were instructed to maintain a diet of at least 150 g carbohydrate/d for the 3 days prior to each OGTT and to record their dietary intake on these days. Photocopies of the food records prior to the first trial were provided, and participants were asked to repeat the diet prior to the next trial. Food models (Nasco, Fort Atkinson, WI) were used to facilitate the estimation of portion size. Dietary records were

verified during an interview with one of the researchers (C.J.), and were analyzed for macronutrient composition using computer software (Nutritionist IV; N-Squared Computing, San Bruno, CA). The participants were asked to refrain from eating for 2 hours before reporting to the laboratory for submaximal exercise. Furthermore, each participant was provided with the same evening meal (approximately 720 kcal containing 50% carbohydrate, 20% protein, and 30% fat) following each trial.

Body Composition Measurements

The body mass index (BMI) was derived from weight measurement to the nearest 0.1 lb using a Detecto-Medic scale (Detecto Scales, Brooklyn, NY) and self-reported height. The waist to hip ratio (WHR) was derived from the waist circumference measured at the level of the umbilicus and the hip circumference measured at the level of the greater trochanter with the participant in the standing position at the end of a normal expiration. These anatomical landmarks for measuring WHR were selected because they were used in the San Antonio Heart Study on a large number of Mexican American and non-Hispanic individuals.[15] Duplicate measures were made using a nonstretchable, spring-loaded fiberglass tape. The average of the duplicate measures was used in data analysis. The waist circumference was also used independently as an indicator of visceral abdominal fat.[21] Skinfold measurements for determining the centrality index (subscapular/triceps) were made with Lange calipers (Cambridge Scientific Instruments, Cambridge, MA) as described by Pollock et al.[22] Three readings within 1.0 mm were averaged. Adipose and lean mass measurements were determined by DEXA (Lunar DPX) total-body scans.

Calculations and Statistics

All data represent the nine pairs of women (N = 18) matched by fat mass. The postprandial response areas for glucose, insulin, and C-peptide were calculated using the trapezoidal rule. Repeated-measures multivariate ANOVAs (MANOVAs) were used to analyze the postprandial response areas and time-point data for glucose, insulin, and C-peptide. Basal (0 minutes of the OGTT) FFA and SHBG concentrations were analyzed using a repeated-measures MANOVA, and group comparisons of body composition and fitness variables were tested using Hotelling's T^2 test. Dietary data were analyzed by repeated-measures MANOVA. Significant relationships between the baseline postprandial insulin response (control trial) and the following variables were tested using Pearson product-moment correlations: change in insulin response (change = control trial response − exercise response), SHBG, BMI, centrality index, WHR, and waist circumference. Significance for each analysis was set at a P level of .05 or less. Values are presented as the mean ± SE. Statistical calculations were made using BMDP statistical software (Berkeley, CA).

RESULTS

Body Composition, $\dot{V}O_2max$, and Submaximal Exercise

The groups had a similar BMI, WHR, waist circumference, fat mass, percent body fat, and lean mass, but the Mexican Americans had a significantly ($P < .01$) greater centrality index. $\dot{V}O_2max$ was similar between groups (Table 1). Both groups achieved approximately 70% $\dot{V}O_2max$ during submaximal exercise (non-Hispanic *v* Mexican American, 69% ± 1% *v* 73% ± 2%) and had similar submaximal heart rates (non-Hispanic *v* Mexican American, 165 ± 6 *v* 169 ± 5 bpm).

Postprandial Responses of Insulin, Glucose, and C-Peptide

The group-by-trial interaction was significant ($P = .01$) for the postprandial insulin response. Compared with the control trial, the postprandial insulin response was 23% lower following exercise ($P = .02$) in Mexican Americans, whereas no significant ($P > .05$) difference in control and exercise postprandial insulin responses was found for non-Hispanics (Table 2). Neither the glucose nor the C-peptide postprandial response was significantly different between groups or trials. However, the postprandial glucose and C-peptide responses from the control trial were approximately 14% greater in Mexican Americans compared with non-Hispanics.

Fasting insulin (0 minutes of the OGTT) was significantly lower (13%, $P = .01$) in Mexican Americans after exercise versus the control trial (Table 2). Several other time points under the insulin curve were also different between trials. In Mexican Americans, insulin concentrations at minutes 15, 60, and 150 were significantly ($P \leq .05$) higher after the control trial compared with exercise (Fig 1). In non-Hispanics, the insulin concentration at 15 minutes was significantly ($P < .05$) higher after the exercise trial compared with the control trial. At 60 minutes, the non-Hispanic insulin concentration tended to be greater ($P = .06$) after exercise compared with the control trial. Group differences in insulin time points were significant at minute 60 of the control trial (Mexican Americans > non-Hispanics, $P = .01$) and minute 15 of the exercise trial (Mexican Americans < non-Hispanics, $P = .04$). Plasma glucose and C-peptide concentrations at each time point of the OGTTs, including fasting values, were similar between groups and trials (Figs 2 and 3 and Table 2).

FFA and SHBG

In both groups, the plasma FFA concentration was significantly ($P = .01$) elevated in the exercise trial (non-Hispanic *v* Mexican American, 0.53 ± 0.05, *v* 0.61 ± 0.09 mEq/L) compared with the control trial (non-Hispanic *v* Mexican American, 0.43 ± 0.06 *v* 0.50 ± 0.07 mEq/L). Neither the group nor the trial effect on basal SHBG was significant (control: non-

Table 2. Fasting Concentrations and Postprandial Responses of Glucose, Insulin, and C-Peptide in Pair-Matched (n = 9) Non-Hispanic and Mexican American Women

Parameter	Group	Control†	Exercise
Fasting concentration			
Glucose (mmol/L)	NH	4.66 ± 0.1	4.57 ± 0.1
	MA	4.88 ± 0.1	4.80 ± 0.1
Insulin (pmol/L)	NH	82.91 ± 8.9	77.03 ± 8.2
	MA	88.49 ± 10.3*	77.38 ± 7.0
C-peptide (nmol/L)	NH	647.4 ± 86.8	636.3 ± 92.8
	MA	689.2 ± 112.8	625.8 ± 92.2
Postprandial responses			
Glucose (mmol/L · min · 10^2)	NH	8.7 ± 0.5	9.0 ± 0.6
	MA	10.1 ± 0.5	9.8 ± 0.4
Insulin (pmol/L · min · 10^4)	NH	6.2 ± 0.9	7.2 ± 1.0
	MA	8.5 ± 1.4*	6.5 ± 1.0
C-peptide (pmol/L · min 10^5)	NH	3.2 ± 0.3	3.6 ± 0.4
	MA	3.7 ± 0.4	3.5 ± 0.4

NOTE. Values are the mean ± SE.
Abbreviations: NH, non-Hispanic; MA, Mexican American.
*$P < .02$ for MA control *v* exercise.
†No exercise.

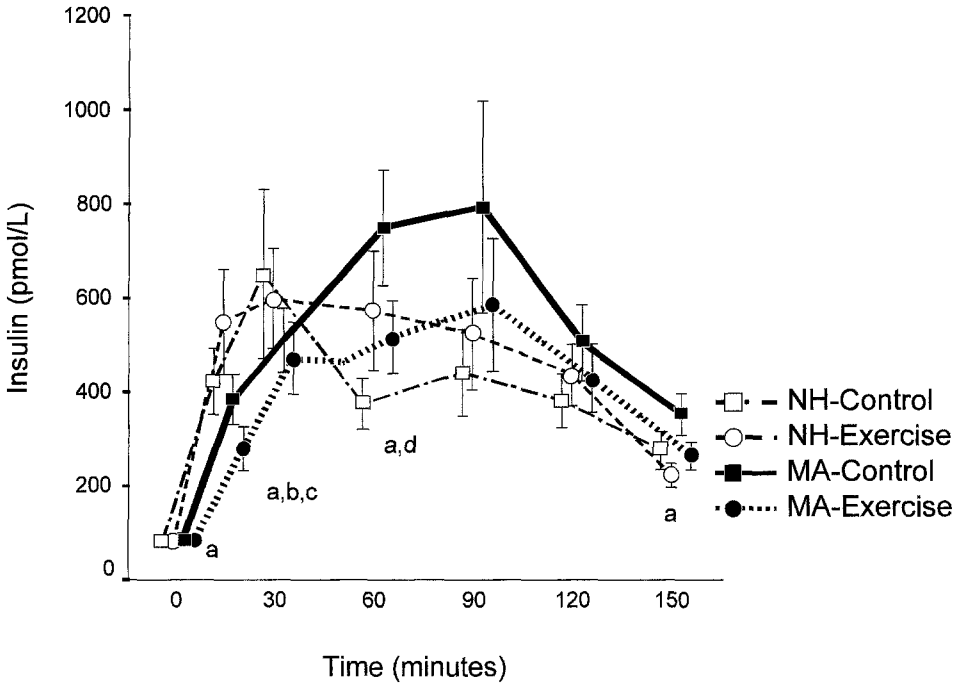

Fig 1. Time course of the postprandial insulin response to an oral glucose load following a single bout of aerobic exercise and no exercise (control) in Mexican American (MA) and non-Hispanic (NH) women. Significant differences ($P \leq .05$): [a]MA, control *v* exercise; [b]MA *v* NH, exercise; [c]NH, control *v* exercise; [d]MA *v* NH, control.

Hispanic *v* Mexican American, 56.24 ± 9.58 *v* 40.83 ± 6.00 nmol/L; exercise: non-Hispanic *v* Mexican American, 51.54 ± 6.53 *v* 43.64 ± 6.66 nmol/L).

Dietary Records

Mean carbohydrate consumption for the 3 days prior to each OGTT was at least 150 g/d. Non-Hispanics consumed significantly ($P < .05$) more kilocalories and carbohydrates prior to the control versus the exercise trial. Before the control trial, non-Hispanics consumed significantly ($P < .05$) more fat than Mexican Americans. Fat accounted for 35% of total kilocalories in non-Hispanics during the control trial, compared with 30% of total kilocalories for Mexican Americans in the same trial ($P < .05$) (Table 3).

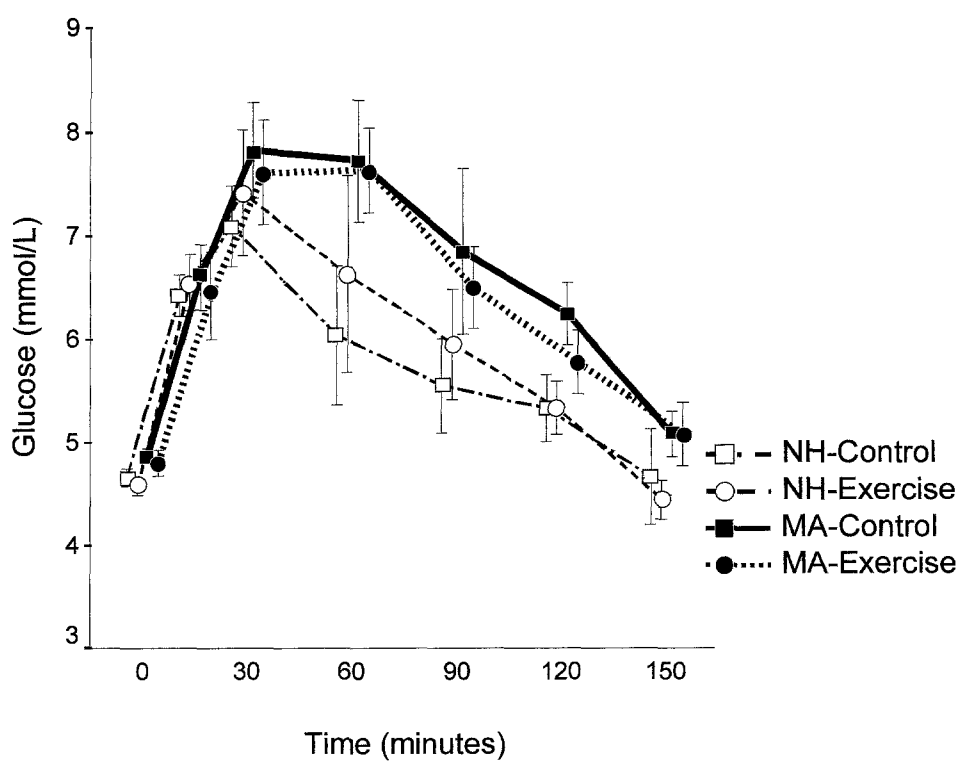

Fig 2. Time course of postprandial glucose response to an oral glucose load following a single bout of aerobic exercise and no exercise (control) in Mexican American (MA) and non-Hispanic (NH) women.

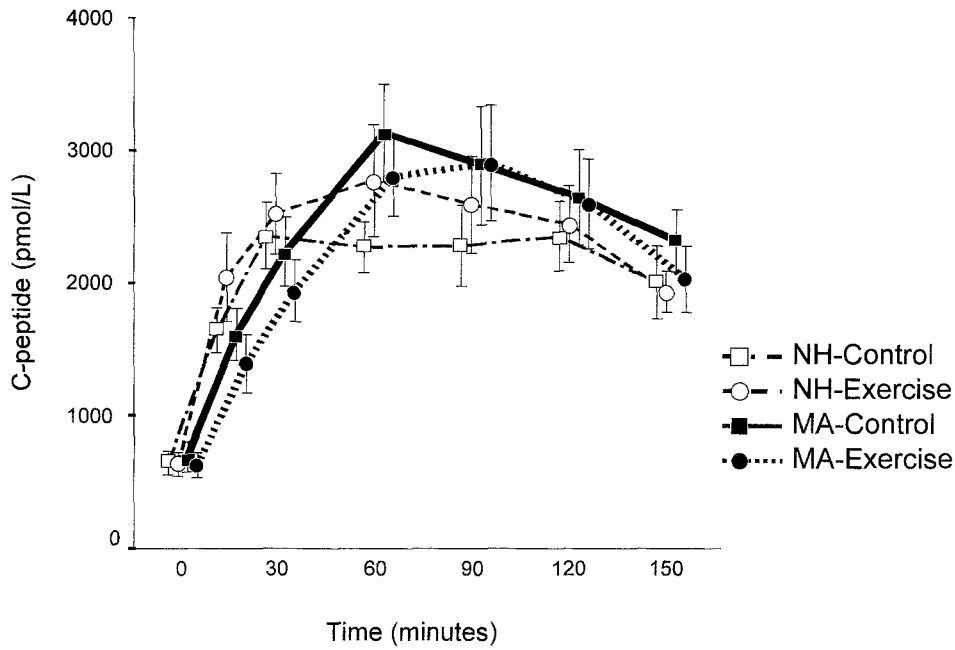

Fig 3. Time course of postprandial C-peptide response to an oral glucose load following a single bout of aerobic exercise and no exercise (control) in Mexican American (MA) and non-Hispanic (NH) women.

Correlations

The groups were combined (N = 18) for correlational analysis. Significant correlates ($.05 < P < .001$) of the baseline postprandial insulin response (control trial) were the change in insulin response ($r = .56$), SHBG ($r = -.61$), BMI ($r = .73$), centrality index ($r = .56$), waist circumference ($r = .66$), and WHR ($r = .54$). The change in insulin response was defined as the difference between the postprandial insulin response in the control and exercise trials (change = control − exercise response).

Table 3. Dietary Composition of Pair-Matched (n = 9) Non-Hispanic and Mexican American Women During the 3 Days Prior to the OGTT

			% of Total Kilocalories	
Parameter	Control‡	Exercise	Control	Exercise
Kilocalories				
NH	1,992 ± 185*	1,731 ± 174		
MA	1,558 ± 76	1,565 ± 104		
Carbohydrate (g)				
NH	269 ± 24*	227 ± 22	51	52
MA	221 ± 12	212 ± 11	56	54
Protein (g)				
NH	65 ± 8	66 ± 7	14	15
MA	61 ± 4	57 ± 5	16	14
Fat (g)				
NH	76 ± 9†	64 ± 7	35†	33
MA	52 ± 4	56 ± 7	30	32

NOTE. Values are the mean ± SE.
Abbreviations: NH, non-Hispanic; MA, Mexican American.
*$P < .05$ for NH control *v* exercise.
†$P < .05$ for NH *v* MA, control.
‡No exercise.

DISCUSSION

This is the first report of a significant reduction in postprandial insulin responses in Mexican American women following a single bout of aerobic exercise compared with a nonexercised (control) condition. In non-Hispanic women pair-matched to the Mexican Americans based on fat mass, a significant exercise effect on the postprandial insulin response was not evident. The postprandial insulin response and fasting insulin level were used as indirect measures of peripheral insulin resistance.

To appreciate the effects of exercise on postprandial insulin responses in both groups, it is necessary to first examine the baseline responses represented by the control trial. Baseline postprandial insulin responses in Mexican American women were 30% greater than in non-Hispanics. Although this difference was not statistically significant, it is consistent with previous epidemiological data.[15] Baseline postprandial C-peptide and glucose responses also tended to be greater (14%) in the Mexican Americans. Therefore, in the current study, women in each ethnic group had typical postprandial responses to the oral glucose challenge in the absence of previous exercise.

In the Mexican American women, both the postprandial insulin response and fasting insulin concentration were significantly reduced (23% and 13%, respectively) after exercise compared with the control condition. Together, these findings suggest improved insulin action following an acute bout of aerobic exercise in Mexican American women. In both groups, postprandial C-peptide responses were similar in the control and exercise trials, suggesting that the reduced postprandial insulin response after exercise in the Mexican Americans may be attributed to enhanced hepatic insulin extraction rather than reduced pancreatic insulin secretion.[23] Both decreased hepatic insulin extraction and enhanced insulin secretion have been recognized as contributors to hyperinsulinemia and type 2

diabetes risk in Mexican Americans.[24] Therefore, exercise may be useful in controlling or preventing hyperinsulinemia by acute enhancement of hepatic insulin clearance.

One explanation for the reduced postprandial insulin response following exercise in Mexican Americans is their relatively greater baseline postprandial insulin response. The baseline postprandial insulin response (no exercise) has been positively associated with the decrease in insulin response 1 day after aerobic exercise in sedentary normoglycemic elderly men and women.[11] Significant associations between the baseline postprandial insulin response and the change in this response due to exercise were also demonstrated in the current study for the combined groups ($r = .56$).

The reduced postprandial insulin response following exercise in the Mexican American women is consistent with previous research in young sedentary adults, although the lack of change in this response in non-Hispanics was somewhat surprising. The groups exercised at the same intensity and had similar elevations in postexercise FFA levels. Using the identical exercise protocol with an ethnically mixed group of female participants,[5] the postprandial insulin response was significantly decreased (15%) and the glucose response was unchanged after exercise compared with a nonexercised condition. Young et al[6] reported a 45% decrease in the postprandial insulin response following an oral glucose challenge in untrained males (18 to 29 years) 14 hours after a 40-minute bout of exercise at 80% $\dot{V}O_2$max. In sedentary males and females,[9] the postprandial insulin response to an intravenous glucose load was 20% lower 16 hours after exercise (60 minutes of bicycle ergometry at 70% $\dot{V}O_2$max) compared with a nonexercised condition.

The absence of an exercise effect on reducing the postprandial insulin response as observed in the non-Hispanic women is also supported in the literature.[11] It has been proposed that non-obese normoglycemic individuals who are already in a state of adequate insulin sensitivity will not benefit additionally from a single bout of exercise[25] if the glucose transport rate is already near-optimal.[26] Instead, consecutive days of exercise may be required to confer additional insulin response benefits for non-obese normoglycemics with adequate insulin sensitivity.[11] The Mexican American women in the current study were relatively insulin-resistant based on their tendency for a higher baseline fasting insulin concentration and postprandial response compared with the non-Hispanics. Thus, the single bout of exercise was a sufficient stimulus to change the postprandial insulin response in Mexican Americans but not in non-Hispanics. In fact, seven of the nine Mexican American women were "responders" (ie, reduced postprandial insulin response after exercise), as opposed to five of nine non-Hispanic responders.

An alternative explanation for the lack of an exercise effect on the postprandial insulin response in non-Hispanics is the lack of stringent dietary compliance. The non-Hispanic women consumed significantly more kilocalories and carbohydrates during the 3 days prior to the control trial compared with the exercise trial, although total energy from carbohydrate was consistent between trials (51% rest *v* 52% exercise). It is unclear as to why these dietary differences occurred within this group, since the order of the trials was randomized to reduce bias in energy consumption. Reduced glycogen stores may enhance insulin-stimulated glucose uptake,[27] but this condition was unlikely because carbohydrate consumption was over 200 g before each glucose tolerance test. Either caloric restriction or a single bout of exercise can result in reduced postprandial insulin responses in untrained individuals.[9] The relative caloric restriction of the non-Hispanics during the exercise trial would tend to decrease, not elevate, the postprandial insulin response. Although it is doubtful that dietary noncompliance had a measurable effect on the postprandial insulin response of the non-Hispanics, dietary issues remain as an explanation for the lack of change in response in this group.

Nearly 90% of the ethnic differences in postprandial insulin responses between Mexican American and non-Hispanic women have been attributed to overall adiposity, regional fat distribution, and an androgenic hormonal profile.[28] The groups were matched for fat mass and had similar lean mass, waist circumference, WHR, and SHBG levels. Despite these similarities in body composition and androgenicity, the centrality index remained significantly different between groups. Furthermore, the centrality index was positively associated ($r = .56$, $P = .016$) with the baseline postprandial insulin response in 18 women studied. The centrality index is an indicator of subcutaneous fat deposition on the trunk (subscapular skinfold) relative to the upper limbs (triceps skinfold). The centrality index and WHR were shown to be significant independent predictors of type 2 diabetes among Mexican American[17] and non-Hispanic[29] women. The pattern of a greater centrality index in Mexican American compared with non-Hispanic women despite a similar BMI and WHR in the present study is consistent with previous reports.[28,30] Based on the centrality index, the Mexican American women included in this report may have an increased risk of diabetes, despite the fact that they were normoglycemic.

The results of this study indicate a reduced peripheral insulin resistance following acute exercise in normoglycemic sedentary Mexican American women. With moderate-intensity exercise, hepatic insulin clearance was enhanced, whereas pancreatic insulin secretion was not significantly reduced. The exercise effect on the postprandial insulin response may be attributed to the relatively greater baseline (no exercise) postprandial insulin response in Mexican American women compared with non-Hispanic women. Further research to evaluate acute and chronic exercise effects on insulin resistance in Mexican American women is warranted, since this population is at high risk of developing type 2 diabetes and must be targeted for disease prevention strategies.

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