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Background: Previous research suggests non-Hispanic blacks (blacks) are more insulin resistant than non-Hispanic whites (whites). Physical activity can play an important role in reducing insulin resistance. However, it is unknown whether racial differences exist in response to exercise. Therefore, the purpose of this study was to compare metabolic responses to a single bout of exercise in blacks and age-, sex-, and body mass index-matched whites.

Methods: Whole-body insulin sensitivity, glucose storage, glucose oxidation, and respiratory exchange ratio (RER) were assessed during a hyperinsulinemic-euglycemic clamp in normoglycemic blacks (n = 11) and whites (n = 10). Outcome measures were evaluated in a sedentary control condition and 12 h after treadmill walking at 75% of maximal heart rate for 75 min.

Results: In the control condition, there were no differences in insulin sensitivity between blacks and whites ($P = 0.54$). During the clamp, glucose oxidation and insulin-stimulated RER values were significantly higher in blacks compared with whites ($P = 0.04$ and $P < 0.01$, respectively). Despite similar RER values during exercise, RER values at 60, 90, and 120 min after exercise in blacks were also significantly higher compared with whites ($P < 0.05$). After exercise, there were no significant improvements in insulin sensitivity ($P = 0.57$) or glucose storage ($P = 0.42$) in blacks or whites; however, glucose oxidation was significantly lower in both racial groups ($P < 0.05$).

Conclusions: These data suggest that insulin sensitivity is similar in blacks and age-, sex-, and body mass index-matched whites, but the glucose disposal pathways (storage *vs.* oxidation) are somewhat different. Compared with whites, blacks appear to have a greater capacity to increase glucose oxidation immediately after exercise and during insulin stimulation. **(*J Clin Endocrinol Metab* 95: E219–E223, 2010)**

For non-Hispanic blacks (blacks), diabetes risk is 2-fold higher than for non-Hispanic whites (whites) (1). This disparity in diabetes risk is associated with more severe insulin resistance (IR) (2). Several researchers have reported that increased IR in blacks is independent of body mass index (BMI) and body composition (2), suggesting that other contributing factors play a role in IR. If race affects IR, then approaches to improving insulin sensitivity may also be influenced by race.

A strong body of research demonstrates that single bouts of exercise reduce IR (3–5). Heath *et al.* (3) reported that the insulin response to oral glucose was considerably higher after 10 d of detraining in highly trained men but mostly restored after one bout of exercise. Devlin and Horton (5) reported significant improvements in insulin stimulated glucose uptake (~25%) after one bout of high-intensity exercise in obese, insulin-resistant men. Our laboratory previously reported one bout of moderate-intensity exercise improved insulin sensitivity in black women (~18%) with IR (4), but the effect was smaller than previously observed in white cohorts (3, 5).

We recognize that the intensity of exercise used in our study was lower than both of the previously mentioned studies, and our study was in women, not men. Neverthe-

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Abbreviations: ANCOVA, Analysis of covariance; BMI, body mass index; FFA, free fatty acid; IR, insulin resistance; RER, respiratory exchange ratio.

less, the question still remains: do metabolic responses to exercise explain racial differences in IR? Therefore, the primary aim of this study was to compare the metabolic responses to a single bout of exercise in blacks and age-, sex-, and BMI-matched whites. Because blacks are more insulin resistant than whites, we hypothesized that the positive metabolic effects of exercise on whole-body insulin sensitivity and carbohydrate metabolism would be smaller in blacks compared with whites.

Participants and Methods

Participants

Healthy, sedentary black (n = 11) and white (n = 10) individuals from the university community participated in this study. Race was defined as persons who self-identified as black or white and self-reported all four grandparents belonging to the same racial group as the participant. After being provided with a full description of the study, participants signed an informed consent document approved by the institutional review board.

Baseline measurements

Before completing blood measurements, total daily energy expenditure was estimated using indirect calorimetry with a metabolic hood system (ParvoMedics TrueMax 2400; Consentius Technology, Sandy, UT) (6). Body composition was measured via dual-energy x-ray absorptiometry (Lunar Prodigy; GE Lunar Corp., Madison WI) (7).

Standardized meals

All participants consumed a standardized meal provided by the investigators 10 h before blood measurements in the sedentary control and postexercise conditions. The macronutrient composition of the standardized meals was relatively low in carbohydrate (20% carbohydrate, 20% protein, 60% fat) to minimize effects of glycogen repletion on the pathways of glucose disposal (8).

Sedentary control condition

Whole-body insulin sensitivity (9), glucose storage and glucose oxidation (10), and whole-body respiratory exchange ratio (RER) (10) were assessed during a hyperinsulinemic-euglycemic clamp (9) and using indirect calorimetry. Whole-body insulin sensitivity or rate of blood glucose uptake was expressed as the ratio of glucose metabolized to the prevailing plasma insulin levels [M (mg/kg^{-1} · min^{-1})/I (picomoles)] during the last 30 min of the euglycemia.

Fasting samples of glucose, insulin, free fatty acids (FFAs), and triacylglycerols were collected before the clamp with immediate glucose analysis every 5 min during the clamp. Immediately after the fasting samples, two infusions were started using a peristaltic infusion pump (Harvard Apparatus Pump 22, Holliston MA): 1) a primed (250 mU/m^{-2}) constant infusion (40 mU/m^{-2} · min^{-1}) of insulin diluted in saline containing 3% (vol/vol^{-1}) of the subject's own blood; and 2) a variable infusion of a 20% glucose solution in water, adjusted to maintain plasma glucose at approximately 90 mg/dl, and continued for 120 min. Expired breath samples along with insulin and FFAs were collected during the last 30 min of the clamp.

Exercise condition

Participants arrived at the laboratory in the evening hours and walked on a treadmill at 75% of maximal heart rate assessed via heart rate monitoring for 75 min (four 15 min bouts at the prescribed intensity accompanied by three 5 min active recovery periods at 2.0 mph). Breath samples were collected during the third 15-min bout of exercise to calculate exercise energy expenditure, exercise oxygen consumption, and exercise RER using indirect calorimetry with a metabolic cart. RER values were also measured 60, 90, and 120 min after exercise.

Postexercise condition

Twelve to 15 h after exercise, participants completed another hyperinsulinemic-euglycemic clamp (procedures identical with those described above).

Statistical analysis

Racial differences in participant characteristics as well as exercise variables were assessed using two-sample *t* tests. Main effects and interactions of race (black *vs.* white), time (fasting *vs.* during clamp), and condition (sedentary *vs.* after exercise) were assessed using a three-way repeated-measures analysis of covariance (ANCOVA) for glucose, insulin, FFAs, and RER. Main effects and interactions of race and condition were assessed using a two-way repeated-measures ANCOVA for fasting triacylglycerols, insulin sensitivity, glucose oxidation, and glucose storage.

Main effects and interactions of race and time were assessed using a two-way repeated-measures ANCOVA for exercise RER values. Sex was used as a covariate for all analyses. When significant differences across race, time, or condition were identified, *post hoc* pairwise comparisons with Bonferroni adjustments were conducted. For all analyses, $P < 0.05$ was considered significant.

Results

There were no significant differences between ethnic groups for any of the physical characteristics or exercise variables (Table 1). Compared with whites, blacks had significantly lower fasting glucose concentrations during the sedentary and postexercise conditions ($P < 0.01$). For insulin, there was a significant effect of time ($P < 0.01$), with concentrations increasing during the sedentary control and postexercise clamps in both races. For FFAs, both blacks and whites had lower concentrations during both clamps compared with fasting values ($P < 0.01$) and higher concentrations during the postexercise condition compared with the sedentary control condition ($P < 0.01$). There were no significant effects of race ($P = 0.31$) or condition ($P = 0.22$) for triacylglycerols.

For insulin sensitivity, there were no significant effects of race ($P = 0.69$) or condition ($P = 0.57$). However, for glucose oxidation, there were significant effects of race ($P =$

TABLE 1. Participant characteristics and metabolic measurements taken during the sedentary control, exercise, and postexercise conditions (mean ± SD)

	Black	White
Participant characteristics		
Gender (female/male)	7/4	6/4
Age (yr)	26.0 ± 7.2	23.4 ± 8.3
Height (in.)	66.4 ± 3.4	66.8 ± 3.7
Weight (kg)	81.6 ± 9.5	81.4 ± 22.2
BMI (kg/m^2)	28.9 ± 4.4	28.0 ± 6.3
Body composition (percent fat)	34.5 ± 12.7	35.5 ± 8.4
Fat-free mass (kg)	49.4 ± 10.1	47.9 ± 10.5
RMR (kcal/d)	1602.1 ± 354.0	1625.7 ± 427.2
PAS	1.5 ± 0.8	1.8 ± 0.6
Sedentary control condition		
Fasting glucose (mg/dl)	82.3 ± 5.0	88.6 ± 5.9^{a**,b**,d**}
Fasting insulin (pmol/liter)	60.6 ± 32.4	68.8 ± 18.6^{b**}
Fasting FFAs (mEq/liter)	0.45 ± 0.16	0.51 ± 0.16^{b**,c**}
Fasting triacylglycerols (mg/dl)	78.0 ± 69.4	110.9 ± 59.3
During clamp		
Glucose (mg/dl)	88.8 ± 2.1	89.5 ± 3.2
Insulin (pmol/liter)	492.6 ± 178.2	474.0 ± 195.0
FFAs (mEq/liter)	0.18 ± 0.16	0.13 ± 0.11
Insulin sensitivity [M (mg/kg · min)/I(pmol)]	0.10 ± 0.06	0.10 ± 0.05
Glucose oxidation (g/kg · min)	3.2 ± 1.2	1.8 ± 1.2^{a*,c*}
Glucose storage (g/kg min)	3.8 ± 2.0	5.5 ± 2.3
Exercise condition		
Energy expenditure (kcal)	508.0 ± 112.5	476.7 ± 134.4
Oxygen consumption (ml/kg · min)	20.67 ± 3.2	20.5 ± 3.8
Heart rate (bpm)	135.4 ± 12.9	132.9 ± 7.2
RER during exercise	0.88 ± 0.04	0.87 ± 0.03
RER 60 min after exercise	0.79 ± 0.07	0.74 ± 0.03^{a**,d*,e**}
RER 90 min after exercise	0.78 ± 0.05	0.74 ± 0.04^{a**,d*,e**}
RER 120 min after exercise	0.84 ± 0.05	0.74 ± 0.03^{a**,d*,e**}
Postexercise condition		
Fasting glucose (mg/dl)	80.5 ± 5.2	89.5 ± 5.0
Fasting insulin (pmol/liter)	57.6 ± 30.6	70.2 ± 31.8
Fasting FFAs (mEq/liter)	0.64 ± 0.15	0.67 ± 0.16
Fasting triacylglycerols (mg/dl)	74.1 ± 49.7	88.4 ± 32.4
During clamp		
Glucose (mg/dl)	88.5 ± 2.2	90.6 ± 2.6
Insulin (pmol/liter)	517.8 ± 241.8	528.6 ± 213.6
FFAs (mEq/liter)	0.41 ± 0.14	0.44 ± 0.13
Insulin sensitivity [M (mg/kg · min)/I(pmol)]	0.12 ± 0.07	0.10 ± 0.06
Glucose oxidation (g/kg/min)	2.7 ± 1.4	1.6 ± 1.7
Glucose storage (g/kg · min)	5.3 ± 2.6	4.6 ± 1.6

The values presented are means ± SD. Glucose, insulin, and and FFA values during the clamp represent the mean concentration during the last 30 min of the clamp. RMR, Resting metabolic rate; PAS, physical activity score.

* Statistically significant difference, $P < 0.05$, preceded by a letter.

** Statistically significant difference, $P < 0.001$, preceded by a letter.

[a] Race: black *vs.* white.

[b] Time: fasting concentrations *vs.* concentrations during the clamp.

[c] Condition: sedentary control *vs.* after exercise.

[d] Interaction: race × time.

[e] Time: exercise RER *vs.* postexercise RER.

0.04) and condition ($P = 0.046$), with significantly higher rates in blacks compared with whites. Both groups, however, had a significant decrease in oxidation rates during the postexercise condition. There were no significant effects of ethnicity ($P = 0.39$) or condition ($P = 0.42$) for glucose storage.

RER values significantly increased during both clamps compared with fasting conditions in both races ($P < 0.01$, Fig. 1). Blacks, however, had a significantly greater increase in insulin-stimulated carbohydrate metabolism compared with whites ($P = 0.01$). During exercise, RER values were similar between races; however, postexercise

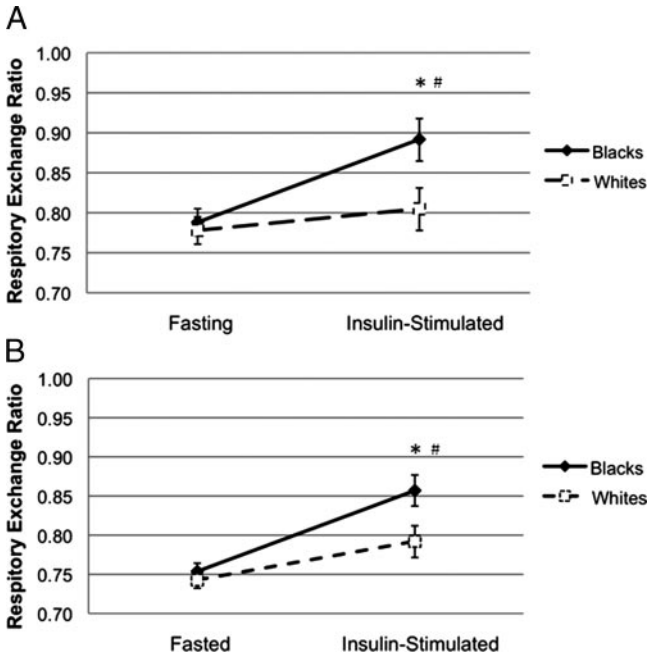

FIG. 1. Fasting and insulin-stimulated RER values during the sedentary control (A) and postexercise conditions (B). *, Significant effect of race at $P < 0.01$; #, significant effect of time at $P < 0.01$.

RER values (at 60, 90, and 120 min) were significantly higher in blacks compared with whites ($P < 0.01$).

Discussion

Previous research has consistently demonstrated that blacks are more insulin resistant compared with whites (2). This greater IR has been linked to an increased risk for type 2 diabetes in blacks (11). However, racial differences in insulin sensitivity have not been observed in studies that have carefully matched the physical characteristics of their black and white participants. Berk *et al.* (12) reported similar levels of insulin sensitivity derived from an iv glucose tolerance test in healthy black and white women matched for age, BMI, and body composition. Hannon *et al.* (13) and Winnick *et al.* (14) confirmed these findings in both healthy children and adults with type 2 diabetes, respectively. Taken together, these results do not support the idea that blacks are inherently more insulin resistant compared with whites and suggest that higher IR reported in blacks may be more related to sociocultural and environmental factors.

In the present study, one bout of exercise did not improve insulin sensitivity or glucose storage in either blacks or whites. These findings are consistent with those of Black *et al.* (15), who demonstrated that refeeding calories after exercise attenuates improvements in insulin action in overweight white participants. Holtz *et al.* (8) also confirmed these findings, using a carbohydrate-restricted meal. However, it is possible that the relatively low intensity and moderate energy expenditure reported in the present study was insufficient to elicit the significant changes in insulin sensitivity reported after intense glycogen-depleting exercise (5). Nevertheless, the exercise bout used was designed for an overweight, sedentary population, and this type of exercise is well known to have long-term benefits to metabolic health and disease risk. Furthermore, because 65% of the American population is overweight or obese and 50% of adults in the United States do not meet recommended levels of physical activity, our overweight, sedentary study population is representative and results can be generalized to the public if confirmed in a larger, free-living population.

To our knowledge, this study is the first to assess the impact of a single bout of exercise on carbohydrate metabolism in blacks and whites. Despite similar rates of carbohydrate oxidation reported during exercise in the present study, blacks derived more calories from carbohydrate compared with whites immediately after exercise (~1–2 h). It is unclear why carbohydrate oxidation was higher in our black participants after exercise; however, previous research suggests ethnic differences in fatty acid flux during an epinephrine infusion, a condition similar to exercise, may play a role (16).

Insulin-stimulated carbohydrate metabolism was significantly higher in blacks during the sedentary control and postexercise clamps, even though carbohydrate metabolism was similar in both races during fasting conditions. These results combined with the postexercise data suggest that in our overweight, sedentary participants, blacks were more metabolically flexible, having a greater capacity to switch from primarily fat to carbohydrate metabolism.

Previous research examining ethnic differences in metabolic flexibility are inconsistent. Weyer *et al.* (17) reported significantly higher 24-h respiratory quotients in blacks compared with whites. Chitwood *et al.* (18) and Nicklas *et al.* (19) confirmed these findings in lean and obese black and white individuals, respectively. In contrast, Berk *et al.* (12) did not observe racial differences in fasting fat oxidation or insulin-stimulated carbohydrate oxidation during a pancreatic euglycemic clamp.

It is unclear why our results differ from those of previous studies. However, it is possible that differences in energy intake or composition between studies could have altered energy and/or macronutrient balance and therefore the response to insulin stimulation. Furthermore, sociocultural influences on behavioral factors such as habitual dietary intake may have also played a role. Nevertheless, under the conditions used in the present study blacks, appeared to demonstrate greater metabolic flexibility during a clamp, com-

pared with their white counterparts. However, the greater metabolic flexibility in our black participants does not appear to be related to greater insulin sensitivity. This is somewhat paradoxical because lack of metabolic flexibility has been previously associated with insulin resistance (20). This discrepancy is likely due to different methodologies used (whole body *vs.* limb RER) between studies.

In conclusion, the major effect of previous exercise was to decrease insulin-stimulated glucose oxidation in both black and white participants. However, in this population of overweight sedentary blacks and whites, blacks appear to have a greater capacity to increase glucose oxidation immediately after exercise and during insulin stimulation compared with whites.

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