Serum Insulin, Obesity, and the Incidence of Type 2 Diabetes in Black and White Adults

The Atherosclerosis Risk in Communities Study: 1987–1998

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OBJECTIVE — In this study, we tested the hypothesis that fasting serum insulin is higher in nonobese black adults than in white adults and that high fasting insulin predicts type 2 diabetes equally well in both groups.

RESEARCH DESIGN AND METHODS — At the baseline examination (1987–1989) of the Atherosclerosis Risk in Communities Study, fasting insulin and BMI were measured in 13,416 black and white men and women without diabetes. Participants were examined at years 3, 6, and 9 for incident diabetes based on fasting glucose and American Diabetes Association criteria.

RESULTS — Fasting insulin was 19.7 pmol/l higher among nonobese (BMI <30 kg/m^2) black women compared with white women (race and obesity interaction term, P < 0.01). There were no differences among men. Among nonobese women, the relative risk for developing diabetes was similar between racial groups: 1.4 (95% CI 1.2–1.5) and 1.3 (1.2–1.4) per 60 pmol/l increase in insulin (P < 0.01) for black and white women, respectively (interaction term, P = 0.6). Findings were similar among men. Adjusting for established risk factors did not attenuate this association.

CONCLUSIONS — Nonobese black women have higher fasting insulin levels than nonobese white women, and fasting insulin is an equally strong predictor of diabetes in both groups. These results suggest one mechanism to explain the excess incidence of diabetes in nonobese black women but do not explain the excess among black men. Future research should evaluate additional factors: genetic, environmental, or the combination of both, which might explain higher fasting insulin among black women when compared with white women.

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T he excess prevalence of type 2 diabetes among black men and women when compared with white men and women is well documented (1–3), but explanations for these findings are limited. Physical inactivity, obesity, and low socioeconomic status only partially explain the disparity (4), and the joint contribution of these and other potentially modifiable risk factors only account for about half of the excess risk (1). Both hyperinsulinemia, a marker for insulin re-

sistance, and obesity are established risk factors for type 2 diabetes (5).

In cross-sectional studies, black children and adults have higher levels of fasting insulin and a lower insulin sensitivity index than white people after adjustment for BMI (6–9). Whether this profile places black people at an increased risk of developing type 2 diabetes has not been evaluated. Recent research suggests that the elevated risk of diabetes for black people compared with white subjects is only present in individuals with lower BMIs (1,10). The objectives of this study were to explore whether a marker of insulin resistance, hyperinsulinemia, differs between black and white adults with lower BMIs and to determine whether hyperinsulinemia is an important predictor of diabetes among nonobese individuals in both racial groups.

RESEARCH DESIGN AND METHODS

Study population

This investigation was conducted in the Atherosclerosis Risk in Communities (ARIC) Study. A probability sample of black and white men and women aged 45–64 years was recruited from the metropolitan areas Forsyth County, NC, Washington County, MD, and Jackson, MS (black residents only) and from the suburbs of Minneapolis, MN. A detailed description of the response rates, study design, and methods is available (11). At baseline (1987–1989), 15,792 individuals were examined. Participants were excluded for the following reasons: neither black nor white race, missing insulin or BMI data, fasting for <8 h, prevalent diabetes, or BMI indicating underweight (<18.5 kg/m^2). A total of 13,287 participants (1,930 black women, 5,395 white women, 1,205 black men, and 4,757 white men) were included in this analysis.

Data collection

Participants underwent clinical examinations at baseline and at three subsequent examinations on 3-year cycles (examination 2: 1990–1992; examination 3: 1993–1995; examination 4: 1996–1998). All measurements were collected according to standardized protocols common to all ARIC study sites (12).

Participants were asked to fast overnight (>8 h) before the clinic examination and to refrain from drinking alcohol and caffeine or smoking the day of the examination. Blood was drawn from seated participants and shipped to a central laboratory for assay. Serum insulin was measured by radioimmunoassay using an Insulin Kit (Cambridge Medical Diagnosis, Billerica, MA). Serum glucose was measured by a hexokinase/glucose-6-phosphate dehydrogenase method on a Coulter DACOS device. Homeostasis model assessment was calculated as the product of fasting insulin (microunits per milliliter) and fasting glucose (micromoles per liter) divided by 22.5 (13). Fasting serum insulin and homeostasis model assessment were used as markers of insulin resistance (14). Total cholesterol and triglycerides were measured in plasma with enzymatic methods. HDL cholesterol was measured after dextran-magnesium precipitation (15). Blood pressure was measured three times from seated participants; the average of the last two measurements was used in this study. Hypertension was defined as systolic blood pressure ≥140 mmHg, diastolic blood pressure ≥90 mmHg, or use of medications to lower blood pressure in the 2 weeks before the clinic examination.

BMI was calculated as the ratio of weight (kilograms) to standing height (meters) squared (kg/m^2); participants with a BMI ≥30 were classified as obese. To calculate waist-to-hip ratio, waist girth was measured at the umbilicus, and hip girth was measured as the largest diameter around the gluteal muscles.

Type 2 diabetes was defined according to the American Diabetes Association criteria (16) as any of the following: a fasting serum glucose level of 7 mmol/l (≥126 mg/dl), a nonfasting glucose level ≥11.1 mmol/l (200 mg/dl), self-reported use of medications for diabetes, or a self-reported previous physician diagnosis. Participants who were diagnosed with diabetes at examinations 2, 3, or 4 were considered to have incident diabetes.

Follow-up time was calculated as the difference between the dates of clinic examination at diagnosis and the baseline clinic examination.

Age, race, sex, education level, physical activity, and diet were assessed at baseline. Education was categorized based on the highest grade level completed, and participants with less than a high school education (grade 12) were compared with participants with a high school education or higher. Alcohol consumption was categorized as current, former, or never. Current cigarette smokers were compared with participants who smoked <100 cigarettes in a lifetime. The modified questionnaire by Baecke et al. (17) was used to assess leisure time physical activity (e.g., gardening) and sports-related physical activity (e.g., jogging) on a five-point scale from one (low) to five (high). The modified 61-item food frequency questionnaire by Willett et al. (18) was used to assess diet. Prevalent coronary heart disease was defined as a history of coronary artery bypass surgery, balloon angioplasty, or myocardial infarction based on electrocardiograph or physician diagnosis (19). Medication use was identified and defined by coding all reported medications, vitamins, and supplements used in the 2 weeks before the clinic examination.

Statistical analysis

All analyses were performed separately by sex. The distribution of baseline characteristics was compared by race using t tests (means) and χ^2 tests (proportions). The distribution of insulin was skewed so values were log-transformed for analyses and back-transformed to geometric means for presentation. BMI was divided into six categories: 18.5 to <22, 22 to <25, 25 to <28, 28 to <31, 31 to <34, and ≥34 kg/m^2. Means of fasting insulin (95% CI) by racial group and BMI category were calculated from an ANOVA model including an interaction term between race and BMI category. In a separate model, the interaction between race and obesity (BMI ≥30 kg/m^2) was examined. All models were adjusted for BMI as a continuous variable.

Incidence rates of diabetes (per 1,000 person-years) and incident rate ratios were calculated using Poisson regression. Cox proportional hazards regression models (adjusted for discrete failure time) were used to estimate the relative risk of

incident diabetes by insulin level and racial group (20). To test our a priori hypothesis that the relationship between fasting insulin and incident diabetes differed between nonobese black and white participants, an interaction term between race and fasting insulin was entered into the regression models. A significant change in the maximum likelihood χ^2 indicated statistical interaction.

The role of covariates on the relationship between fasting insulin and incident diabetes was evaluated by entering covariates into regression models. A change in the relative risk for insulin of >10% was accepted as an indication of a statistically important confounder. To account for multiple testing, statistical significance was denoted at $P < 0.01$. All analyses were performed using SAS version 8.1 (SAS Institute, Cary, NC).

RESULTS — At baseline, a higher proportion of black women had less than a high school education, their mean caloric intake was higher, their physical activity levels were lower, and their prevalence of hypertension was nearly double that of white women (Table 1). Black women were twice as likely to be obese than white women, and mean fasting insulin was significantly higher. The same characteristics differed significantly between black and white men, with the notable exceptions of BMI and insulin.

Fasting insulin by race and BMI

In each category of BMI, black women had higher fasting insulin than white women (race and BMI category interaction term, $P = 0.0003$) (Fig. 1A). In individuals with BMIs from 22 to 31 kg/m^2, differences in mean insulin were 15–20 pmol/l higher in black women than in white women. In the highest BMI category (≥34 kg/m^2), means of insulin converged (absolute difference 5.8 pmol/l, black versus white) and 95% CIs overlapped, suggesting little difference between racial groups. Differences in fasting insulin when comparing obese (BMI ≥30 kg/m^2) and nonobese (BMI <30 kg/m^2) black and white women were also highly significant (race and obesity interaction term, $P < 0.0001$). Mean fasting insulin was 19.7 pmol/l higher among nonobese black women and 7.5 pmol/l higher among obese black women compared with white women.

Table 1—Baseline covariates by race and sex

Covariate	Women			Men		
	Black	White	$P*$	Black	White	$P*$
n	1,930	5,395	—	1,205	4,757	—
Age (years)	52.9 ± 5.7	53.8 ± 5.7	<0.0001	53.7 ± 6.0	54.7 ± 5.7	<0.0001
Education (% with less than high school education)	37.0	15.3	<0.0001	42.5	17.3	<0.0001
Current alcohol drinker (%)	23.2	62.4	<0.0001	51.6	70.0	<0.0001
Current smoker (%)	25.5	25.0	0.6461	38.4	24.4	<0.0001
% Calories from carbohydrates	50.9 ± 9.3	49.6 ± 9.4	<0.0001	48.8 ± 9.3	47.5 ± 9.1	<0.0001
% Calories from total fat	31.9 ± 6.3	32.7 ± 6.9	<0.0001	31.9 ± 6.2	33.5 ± 6.8	<0.0001
Dietary fiber (g)	16.1 ± 8.3	17.3 ± 7.9	<0.0001	16.1 ± 8.2	17.7 ± 8.2	<0.0001
Total kcal	1,567.7 ± 755.4	1,497.6 ± 569.0	0.0002	1,772.7 ± 779.1	1,794.6 ± 727.3	0.3740
Physical activity sport score†	2.1 ± 0.7	2.4 ± 0.8	<0.0001	2.3 ± 0.7	2.7 ± 0.8	<0.0001
Leisure-time physical activity score†	2.1 ± 0.6	2.5 ± 0.5	<0.0001	2.1 ± 0.6	2.4 ± 0.5	<0.0001
BMI (kg/m^2)	30.2 ± 6.5	26.2 ± 5.2	<0.0001	27.2 ± 4.7	27.2 ± 3.9	0.9614
Obese (BMI >30 kg/m^2)	44.4	20.6	<0.0001	24.3	20.1	0.0015
Waist-to-hip ratio	0.895 ± 0.08	0.886 ± 0.08	<0.0001	0.935 ± 0.05	0.966 ± 0.05	<0.0001
Insulin (pmol/l)	104.6 ± 74.2	66.9 ± 50.2	<0.0001	80.6 ± 59.2	79.3 ± 56.3	0.5581
Glucose (mg/dl)	98.2 ± 10.1	96.7 ± 8.7	<0.0001	99.4 ± 10.1	100.8 ± 8.9	<0.0001
Homeostasis model assessment*	67.6 ± 52.6	42.4 ± 34.9	<0.0001	52.6 ± 41.4	52.1 ± 39.6	0.2249
Total cholesterol (mmol/l)	3.9 ± 1.4	4.0 ± 1.5	0.1615	5.4 ± 1.1	5.5 ± 1.0	0.7606
HDL cholesterol (mmol/l)	1.5 ± 0.5	1.5 ± 0.4	0.0110	1.3 ± 0.4	1.1 ± 0.3	<0.0001
Triglycerides (mmol/l)	1.1 ± 0.6	1.4 ± 0.8	<0.0001	1.3 ± 0.9	1.6 ± 1.0	<0.0001
Fibrinogen (mg/dl)	297.2 ± 59.4	323.0 ± 69.3	<0.0001	293.0 ± 61.8	305.0 ± 68.9	<0.0001
Hypertension‡	52.4	23.9	<0.0001	51.8	26.0	<0.0001
Prevalent coronary heart disease§	1.9	1.6	0.2828	4.3	8.0	<0.0001

Data are means ± SD unless otherwise indicated. *Insulin × glucose/22.5; †Baecke index; ‡systolic/diastolic blood pressure >140/90 mmHg, antihypertensive medication use, or diagnosis of hypertension; §coronary heart disease; myocardial infarction, balloon angioplasty, bypass surgery, electrocardiographic changes, or physician diagnosis. P values were from χ^2 test of proportions and t test of means.

Mean fasting insulin was similar among black and white men at each BMI category (Fig. 1*B*). Neither the interaction term for race and the six-level BMI category nor the term for race and obesity was significant (BMI categories, $P = 0.2620$; obesity, $P = 0.5120$). Therefore, all modeling of the relationship between fasting insulin and diabetes was restricted to women.

Incident diabetes
Over an average of 8.7 years of follow-up (SD 1.9), 750 women (10.2%) and 729 men (12.4%) were diagnosed with diabetes. In the full sample of women, the incidence rate ratio was double among black women compared with white women. This pattern was equally strong in nonobese women but less so in obese women (Table 2). The magnitude of the racial difference was slightly smaller among all men and nonobese men compared with women, and the rate of diabetes did not differ between obese black and white men.

Among nonobese women, the risk of developing diabetes was 1.37 (95% CI 1.22–1.54) for a 1-SD increase in insulin (60 pmol/l) for black women and 1.32 (1.24–1.41) for white women. This association was homogeneous by racial group ($\chi^2 = 0.24$, $P = 0.6273$). Similarly, the association between insulin and incident diabetes did not differ between black (1.21 [95% CI: 1.02–1.44]) and white (1.33 [1.26–1.40]) nonobese men ($\chi^2 = 1.16$, $P = 0.2811$). There was a positive monotonic relationship between fasting insulin and the relative risk of developing diabetes for both black and white women (Fig. 2*A*) and men (Fig. 2*B*).

In the absence of interaction between race and insulin among nonobese women and men, racial groups were pooled. We evaluated demographic (race, age, and education), dietary (% total calories from carbohydrates, dietary fiber), and physical activity as potential confounders of the relationship between insulin and incident diabetes. No variables were confounders according to the criteria of a 10% change

in either sex. The pooled relative risk of developing diabetes did not change by >1% with the inclusion of any of the covariates entered singly or in groups into the models for women (1.31 [95% CI 1.26–1.36]) or men (1.31 [1.24–1.38]) per SD increase in insulin.

CONCLUSIONS — In this population-based sample, nonobese black women, previously thought to be at a lower risk for developing diabetes (compared with their obese counterparts), demonstrated evidence of insulin resistance. Among nonobese women, the magnitude of association between hyperinsulinemia and diabetes was statistically equivalent between black and white women.

In the National Health and Nutrition Examination Survey (NHANES) I, Epidemiologic Follow-Up Study, the incidence of diabetes is only elevated in nonobese (BMI <30 kg/m^2) black adults compared with white adults, but also the risk of diabetes is equivalent between races among
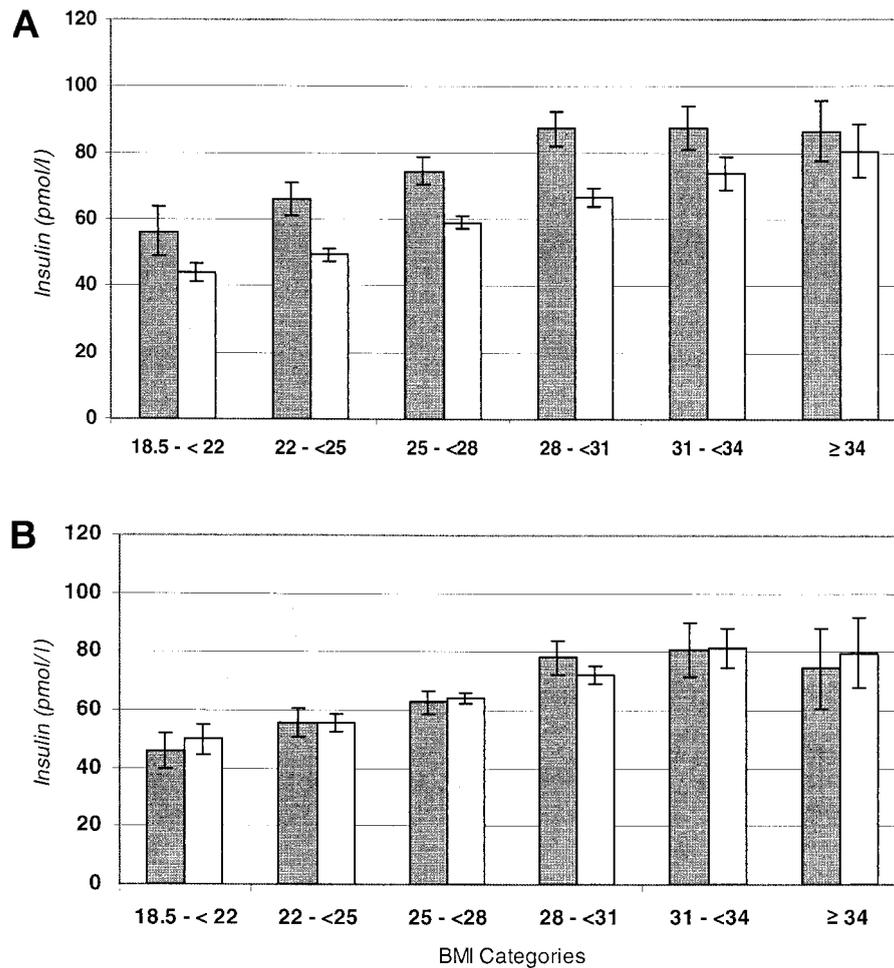
Figure 1—*Adjusted* means and 95% CIs of insulin by racial group: the ARIC Study, 1987–1989. A: Women (P value from race × BMI interaction term = 0.0003). B: Men (P value from race × BMI category interaction term = 0.2620). *Adjusted for BMI (per 1 unit).* ■*, Black subjects;* □*, white subjects.*

the people who are not obese (BMI ≤30 kg/m^2) (10). The authors suggest that greater visceral adiposity at a lower BMI in black adults may explain this association. However, previous research in this sample (21) and others (6) does not support this theory. In our sample, waist-to-hip ratio was smaller among black men and women compared with their white counterparts at lower body weights (data not shown). This finding suggests that another mechanism may be responsible for the racial disparity in diabetes incidence at lower body weights.

Previous research detected higher insulin among black children compared with white children (6–9,22). In a study of 73 black and white children (5–10 years old), fasting and postchallenge insulin was higher among black children even after total body fat, intra-abdominal fat, and subcutaneous fat were controlled

(7). In the Insulin Resistance and Atherosclerosis Study, black adults aged 40–69 years demonstrated evidence of insulin resistance when compared with white adults at a similar body weight, independent of diabetes status (9,22). Lovejoy et al. (6) confirmed these results in a small (*n* = 59) sample of black and white women. Thus, in this large population, evidence of insulin resistance at a lower body weight in black women confirms previous research.

Each of the above-mentioned studies reports effects in both men and women. The sample presented by Lovejoy et al. (6) was restricted to women, whereas Gower et al. (7) and Karter et al. (9) demonstrated differences in both sexes, and Haffner et al. (22) controlled for sex in their analysis. We did not have an a priori hypothesis that the relationship between body weight and insulin would differ by

sex. Rather, our decision was prompted by racial differences in energy expenditure, metabolism, and other factors associated with high insulin that may be more pronounced among women.

In a national sample, the proportion of physically inactive women was higher among black and Hispanic women than among white women (23). Weyer et al. (24) found that sleeping metabolic rate and 24-h energy expenditure was lower among black women compared with white women; less noticeable differences were detected among men. The striking excess of obesity among U.S. black women compared with white women (25) and the absence of such a difference between black and white men (26) may be a manifestation of these differences.

Interpreting differences in disease risk by race is complicated because of the close relationship between socioeco-

Table 2—Incidence of type 2 diabetes by race and obesity

	Black	White	Nonobese Black	Nonobese White	Obese Black	Obese White
Women						
Individuals at risk (*n*)	1,929	5,395	1,063	4,267	866	1,128
Person-years of follow-up	16,000	47,966	9,022	38,384	6,978	9,582
Incident cases of diabetes	315	435	118	229	197	206
Incidence rate per 1,000 person-years (95% CI)	19.6 (15.5–24.9)	9.1 (8.3–10.0)	13.0 (9.1–18.5)	6.0 (5.2–6.8)	28.2 (20.3–39.3)	21.5 (18.8–24.6)
Incidence rate ratio (black vs. white) (95% CI)	2.17 (1.87–2.50)	—	2.18 (1.74–2.72)	—	1.31 (1.08–1.60)	—
P	<0.0001		<0.0001		0.0063	
Men						
Individuals at risk (*n*)	1,205	4,757	908	3,797	297	960
Person-years of follow-up	9,775	40,839	7,458	33,164	2,317	7,674
Incident cases of diabetes	178	561	99	326	79	235
Incidence rate per 1,000 person-years (95% CI)	18.0 (14.0–23.1)	13.7 (12.6–14.9)	13.0 (9.3–18.2)	9.8 (8.8–11.0)	34.1 (23.3–50.0)	30.6 (26.9–34.8)
Incidence rate ratio (black vs. white) (95% CI)	1.31 (1.10–1.55)	—	1.33 (1.06–1.66)	—	1.11 (0.86–1.44)	—
P	0.0018		0.0141		0.4088	

Obesity = BMI $\geq$30 kg/m^2.

nomic status and race (27). In this sample, there were marked socioeconomic differences as measured by education between black and white participants (Table 1). Less educated individuals may be more likely to engage in detrimental health behaviors, which may place them at increased risk for disease. However, previous research has shown that the racial disparity in diabetes cannot be explained fully by differences in socioeconomic status (4). Similarly, in this study, adjustment for education, physical activity, and dietary components does not attenuate the relationship between race or high fasting insulin and diabetes. Any epidemiologic study is limited in its ability to accurately measure socioeconomic status; thus, the possibility of residual confounding remains. However, given the consistency of these results across other study populations (6–9,22), it is equally plausible that there is a real biologic or genetic basis for sus-

ceptibility to insulin resistance in black women that should be investigated.

Primary strengths of this study include the large population sample, the longitudinal design, and the ability to assess the risk of developing diabetes among individuals with a combination of baseline risk factors. However, some potential limitations may affect these results. This epidemiologic study was restricted to a surrogate marker of insulin resistance: fasting insulin. In previous research, fasting insulin was highly correlated with Bergman's insulin sensitivity index (Spearman's $r = \sim 0.6$) in people free of diabetes (14). At the time of insulin measurement, our sample was restricted to individuals without diabetes.

The possibility of geographic confounding by race arises in this study because the majority of black participants (89%) are from one study site: Jackson, MS. To address this concern, we conducted a secondary analysis that re-

stricted comparisons to the Forsyth County, NC, site, which includes both black ($n = 374$, 59% women) and white ($n = 3,148$, 54% women) participants. The direction of the association was similar for all analyses, although the ability to ascertain statistical significance was limited because of sample size.

Because hyperinsulinemia is an equally strong predictor of diabetes in both racial groups, increased susceptibility to insulin resistance in black women may be one explanation for the excess incidence of diabetes in nonobese black women. However, hyperinsulinemia among nonobese black men is not a likely explanation for the excess risk in that group compared with white men; sex differences in the relationship between obesity and insulin resistance should be investigated. Future research should evaluate whether additional factors—genetic, environmental, or the combination of both—explain higher fasting insulin lev-
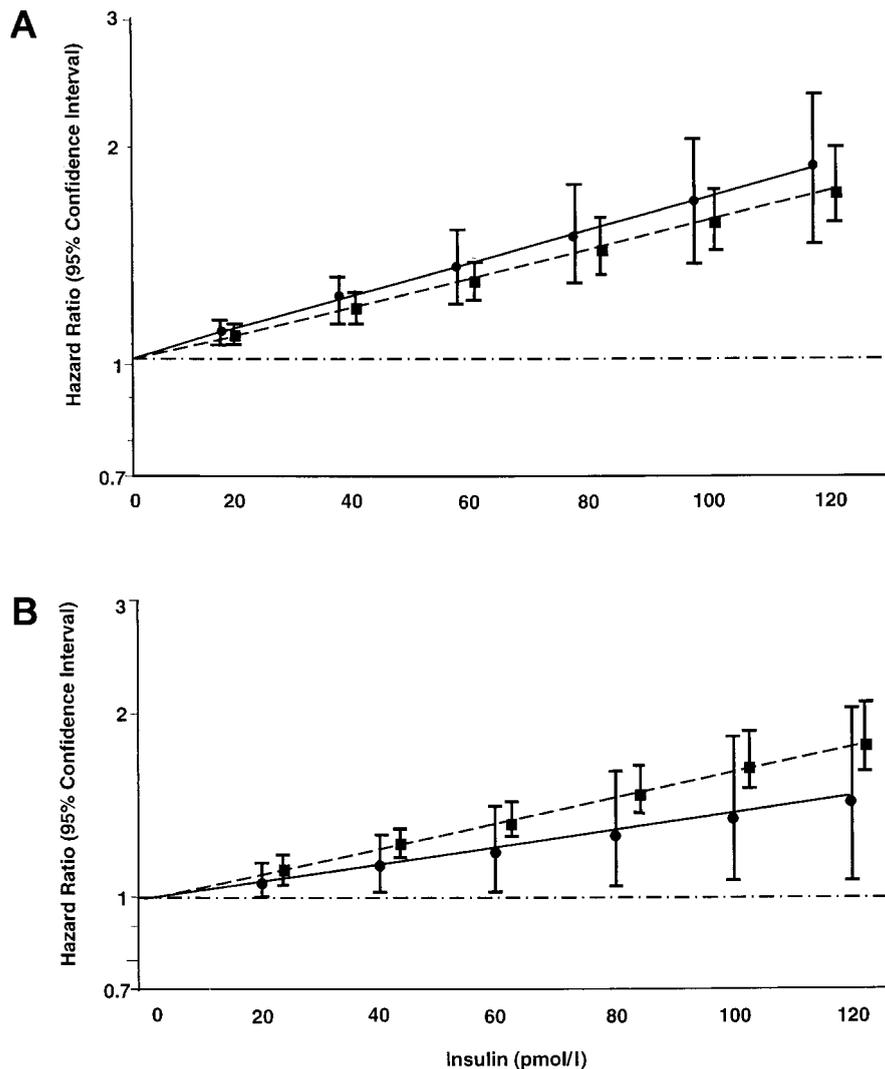
Figure 2—*Adjusted* hazard ratios and 95% CIs of incident type 2 diabetes by fasting insulin among nonobese women (A) and men (B). *Adjusted for BMI. P value for race × insulin interaction term for women = 0.6273 and men = 0.2811. ■, white subjects; ●, black subjects.*

els among black women compared with white women, independent of obesity, and whether insulin-sensitizing therapies and behavioral modifications are effective in reducing the burden of type 2 diabetes among black women.

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