Heterogeneity in the Relationship Between Ethnicity, BMI, and Fasting Insulin

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OBJECTIVE — To determine whether the association of BMI and fasting insulin is modified by ethnicity.

RESEARCH DESIGN AND METHODS — Non-Hispanic black (black), non-Hispanic white (white), and Mexican-American men and women aged 20–80 years from the Third National Health and Nutrition Examination Survey (1988–1994) were included in this study. Linear regression models with an interaction term were used to test whether ethnicity modified the association between BMI and fasting insulin.

RESULTS — Fasting insulin was 19, 26, 20, and 19% higher in black women than white women with BMI levels of <22, 22–24, 25–27, and 28–30 kg/m^2, respectively. These differences between black and white women converged at BMI levels >30 kg/m^2. Mexican-American women had fasting insulin levels that were 17, 22, 20, and 16% higher than those of white women at BMI levels of 25–27, 28–30, 31–33, and >34 kg/m^2, respectively, but were not different in individuals with BMI levels <25 kg/m^2. Adjusting for established risk factors did not attenuate these associations in women. Differences in fasting insulin among men were not as apparent.

CONCLUSIONS — These findings suggest that the effect of obesity on insulin sensitivity is different for Americans in ethnic minorities. In black subjects, fasting insulin is higher at lean weight when compared with white and Mexican-American subjects. In Mexican-American subjects, fasting insulin is higher in overweight individuals when compared with white and black subjects. These findings are more pronounced in women than in men. This result reinforces the importance of designing prevention programs that are tailored to meet the needs of specific populations. Investigation of possible explanations for these differences seems warranted.

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Type 2 diabetes constitutes ~85% of all cases of diabetes and is more common in black (1) and Mexican-American (2) subjects than in white subjects (3). Obesity is a known risk factor for type 2 diabetes (1,2) and is more common in black and Mexican-American populations in the U.S. (4,5). The ethnic disparity in diabetes incidence is not explained entirely by higher prevalence of obesity in these minority groups. Data from the National Health and Nutrition Examination Survey (NHANES), Epidemiologic Follow-Up Study (1971–1992), revealed that at lower levels of obesity, black subjects have a higher risk of developing diabetes than white subjects (6). A study in San Antonio, Texas, revealed that the prevalence of type 2 diabetes was significantly greater in Mexican-American subjects than in white subjects, even when comparisons were made within obesity categories (7). These results suggest that although obesity contributes to type 2 diabetes in ethnic minorities, it does not by itself explain the entire excess prevalence rate.

Insulin insensitivity is also a risk factor for diabetes (8–10). Prospective studies show a relationship between decreased insulin sensitivity and subsequent development of type 2 diabetes (11). Nondiabetic black and Mexican-American subjects appear to have decreased insulin sensitivity when compared with white subjects (12). Previous studies have shown that there may be ethnic differences in secretion, sensitivity, and hepatic extraction of insulin in black and white Americans (13). Mexican-American subjects have higher insulin levels than white subjects, even after adjustment for BMI (14). Both low insulin sensitivity and obesity are established risk factors for type 2 diabetes, and previous research suggests that the relationship between obesity and insulin may be different in ethnic minorities.

The purpose of this study is to examine the relationship between BMI and fasting insulin in a large representative population-based sample. This study extends the investigation to a wider age, obesity, and ethnicity range than previous studies. Detecting effect modification is a primary aim of this study. We hypothesize that the known positive correlation between adiposity and insulin insensitivity differs by ethnicity. Our primary hypothesis is that at comparable levels of BMI, fasting insulin will be higher in ethnic minorities than in white subjects.

RESEARCH DESIGN AND METHODS

— The Third National Health and Nutrition Examination Survey (NHANES III) study, conducted between 1988 and 1994 by the National Center for Health Statistics, was designed to collect information to assess the health status of

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Abbreviations: NHANES, National Health and Nutrition Examination Survey.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

Table 1—*Means (± SE) and proportions of covariates by ethnicity and sex: NHANES III*

	Men			Women		
	Non-Hispanic white*	Non-Hispanic black	Mexican-American	Non-Hispanic white*	Non-Hispanic black	Mexican-american
n	2,583	1,881	2,017	2,756	1,874	1,588
Age (years)	43.7 (0.5)	40.1 (0.5)†	35.6 (0.4)†‡	46.9 (0.6)	42.9 (0.4)†	39.9 (0.4)†‡
Education (years)	13.0 (0.1)	11.7 (0.1)†	9.2 (0.2)†‡	12.7(0.1)	11.9 (0.1)†	9.2 (0.2)†‡
Household income <$20,000	23.3	50.7†	53.6†	28.7	53.0†	53.4†
Weight (kg)	83.4 (0.4)	82.4 (0.4)	77.5 (0.6)†‡	68.7 (0.5)	77.0 (0.6)†	69.3 (0.5)‡
BMI (kg/m^2)	26.7 (0.1)	26.5 (0.1)	26.8 (0.2)	26.0 (0.2)	29.0 (0.2)†	28.1 (0.2)†‡
Obese (BMI >30 kg/m^2)	19.1	19.6	19.5	21.8	37.0†	34.0†
Waist-to-hip ratio	0.96 (0.00)	0.92 (0.00)†	0.96 (0.00)‡	0.86 (0.00)	0.87 (0.00)†	0.88 (0.00)†‡
% kcal from carbohydrate	48.4(0.5)	46.5 (0.3)†	49.0 (0.4)‡	50.7 (0.4)	50.2 (0.3)	51.6 (0.3)‡
Proportion not active	8.6	13.1†	21.6†‡	14.2	25.9†	28.5†
Insulin (pmol/l)	51.0 (1.1)	55.0 (1.0)†	57.3 (1.7)†	47.3 (1.0)	65.2 (1.8)†	63.2 (1.6)†
Glucose (mmol/l)	5.3 (0.0)	5.2 (0.0)†	5.4 (0.0)†‡	5.1 (0.0)	5.2 (0.0)	5.3 (0.0)†
Total cholesterol (mmol/l)	5.3 (0.0)	5.1 (0.0)†	5.1 (0.0)	5.4 (0.0)	5.2 (0.0)†	5.1 (0.0)†
Total cholesterol/HDL ratio	4.6 (0.0)	3.9 (0.0)†	4.4 (0.0)†‡	3.8 (0.1)	3.6 (0.0)†	3.9 (0.0)‡
Systolic blood pressure (mmHg)	121.0 (0.5)	124.5 (0.5)†	118.6 (0.5)†‡	116.1 (0.5)	120.1 (0.5)†	114.3 (0.3)†‡
Diastolic blood pressure (mmHg)	76.0 (0.4)	78.0 (0.5)†	74.9 (0.6)‡	70.9 (0.3)	73.6 (0.4)†	70.1 (0.35)‡
Diabetes (glucose >126 mg/dl)	2.8	2.7	3.2	2.1	3.9†	4.1†
Hypertension (140/90 mmHg or on medication)	21.1	27.3†	13.8†‡	21.4	30.6†	13.8†‡
Family history of diabetes in first-degree relatives	21.0	23.0	25.9†	22.2	31.7†	34.2†

*Referent; †$P < 0.01$ vs. non-Hispanic white; ‡$P < 0.01$, non-Hispanic black vs. Mexican-American.

the U.S. civilian noninstitutionalized population ≥2 months of age. The NHANES III sample design is similar to that of the previous NHANES, which used a stratified multistage probability design. It included oversampling of both the Mexican-American and black American populations so that the sample could produce statistically reliable health estimates for the two largest ethnic minority groups in the U.S. Detailed sampling and methodological information is available (15).

NHANES III data were collected via standardized questionnaires administered by bilingual interviewers and examiners at participants' homes and laboratory tests conducted at NHANES mobile examination centers. Of the 40,600 people invited to participate, 86% completed the home questionnaire, and 78% completed both the medical examination and the home questionnaire (n = 33,199). Ethnicity was self-reported. Respondents who considered themselves black (non-Hispanic), Mexican or Mexican-American, or white (non-Hispanic) are included in this analysis. Briefly, the medical examination included measurements of height (m) and weight (kg). BMI was calculated from measured height and weight (kg/m^2) and was used as an indicator of obesity. The interview included questions on exercise from recreational physical activity, educational attainment, income, family history, and diet. Physical activity was assessed by times per month the participant engaged in various activities. Family history questions included history of diabetes in first-degree relatives. Diet was assessed via a food frequency questionnaire and 24-h dietary recall. Percent of calories from carbohydrate was calculated from 24-h recall data.

Blood samples were obtained after a 12-h fast to measure insulin, glucose, and cholesterol. The determination of serum insulin was made by radioimmunoassay (Pharmacia Diagnostics) (16). Insulin resistance studies, using more invasive measures such as the euglycemic clamp tests, are not feasible in large epidemiologic studies such as NHANES. Several alternative indexes have been suggested, including fasting insulin, fasting glucose, homeostasis index (homeostasis model assessment, which is a ratio of glucose to insulin), and the oral glucose tolerance test. We use fasting insulin because it has been shown to be a reliable surrogate marker for insulin resistance for patients with normal and impaired glucose tolerance (17,18).

Diabetes was defined as a fasting glucose level >126 mg/dl. Because oral hypoglycemic agents and insulin would affect the outcome variable, participants on these medications were excluded. Participants with a self-reported history of diabetes but with normal glucose levels and not on medications were considered nondiabetic in this analysis.

The sample for our analysis included 12,699 participants. There were 15,132 participants aged over 20 years and under 80 years who identified themselves as white, black, or Mexican-American. We used 20 years as our lower age cut point because insulin insensitivity may be harder to detect in children using fasting insulin alone (19,20). We used 80 years as our upper age cut point because aging is associated with changes in insulin metabolism (21), and after this age, fasting insulin is not a well-studied risk factor

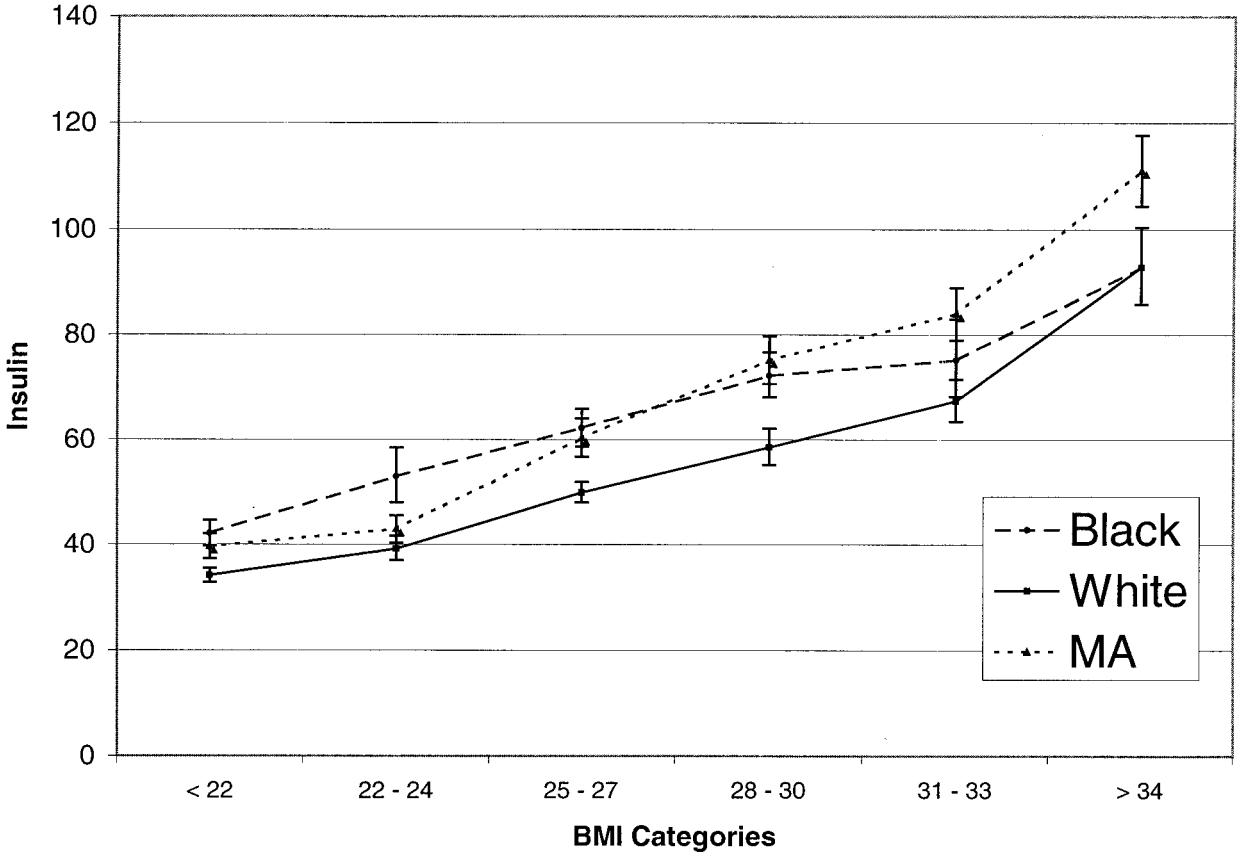

Figure 1—*Means and 95% CIs of insulin by ethnicity: women. MA, Mexican-American.*

(22,23). We excluded data for participants on insulin or diabetes medications (n = 1,386) and for women who were currently pregnant or were pregnant within the last 6 months (n = 621). Missing data were as follows for the outcome and predictor variables: BMI (n = 26) and serum insulin (n = 402).

Data analysis

Primary analyses using linear models were carried out in SUDAAN to adjust for the complex sample design of NHANES III. All analyses incorporated sampling weights that adjusted for unequal probabilities of selection. Analyses were also performed using SAS version 8.1 (SAS Institute, Cary, NC). Analyses were run separately for men and women. Means (SD) and proportions of baseline characteristics were compared by racial group using t tests and χ^2 tests, respectively.

The primary outcome variable was fasting insulin level. Because the distribution of fasting insulin was skewed, the fasting insulin was log-transformed for analyses and back-transformed to geometric means for presentation. The predictor variables were ethnicity and BMI, and an interaction term for ethnicity and BMI. The relationship between BMI and insulin was linear up to a BMI of 40 kg/m^2 (data not shown). For illustration in graphs, BMI was categorized into six arbitrary levels: <22, 22–24, 25–27, 28–30, 31–33, and ≥34 kg/m^2. The following potential confounding factors were included as covariates in the linear models: age, years of education, physical activity, percent of calories from carbohydrate, and family history of diabetes in a first-degree relative. BMI was modeled continuously in these linear models.

Means of fasting insulin (95% CI) by racial group and BMI category were calculated from an ANOVA model using an interaction term between race and BMI category. When undertaking multiple testing, findings of heterogeneity of effects may be due only to chance; therefore, we evaluated the presence of interaction at a stringent criterion of $P <$ 0.01. To compare the differences between means of fasting insulin for black, Mexican-American, and white participants at each BMI category, percentage differences were calculated by subtracting the mean fasting insulin level of the referent group from the mean fasting insulin of the comparison group and dividing by the mean fasting insulin of the comparison group.

RESULTS — As expected, risk factors for diabetes and insulin insensitivity were more prevalent in ethnic minority populations (Table 1). In particular, black and Mexican-American women had fewer years of formal education, were more likely to report a family history of diabetes, had greater measures of adiposity, and were less likely to be active. A similar disparity prevailed in men, with the notable exception of adiposity, which was similar among men of the three ethnicities. Mexican-American men and women obtained a greater percentage of their calories from carbohydrate than black subjects. Total cholesterol was highest in white subjects, and both systolic and diastolic blood pressures were highest in black subjects. Black and Mexican-

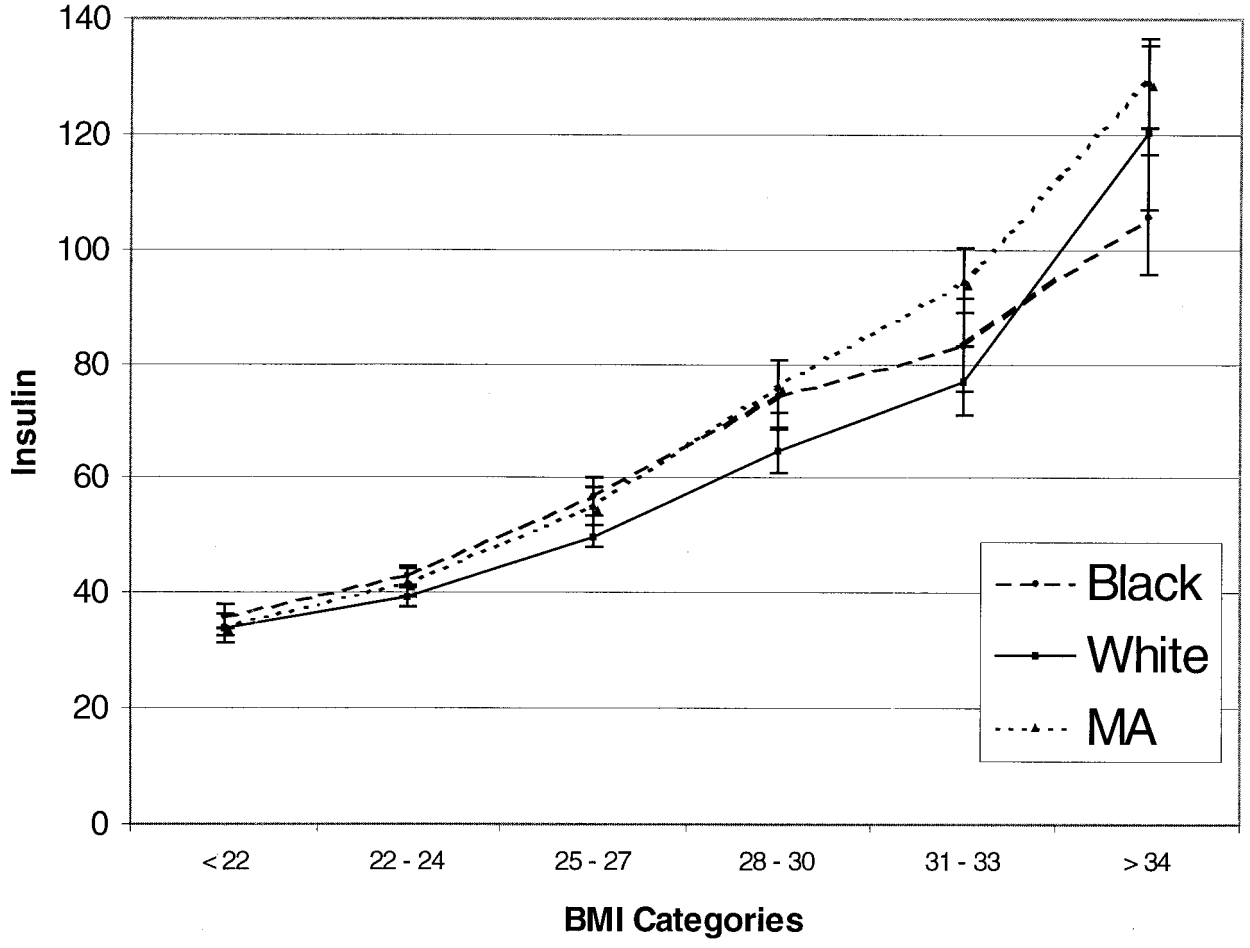

Figure 2—*Means and 95% CIs of insulin by ethnicity: men. MA, Mexican-American.*

American subjects had higher mean fasting insulin levels than white subjects, not adjusting for obesity.

The relationship between obesity and insulin level by ethnicity is shown for women in Fig. 1. Insulin level increases with increasing BMI in all groups, but in each category of BMI, black women had higher fasting insulin levels than white women. Fasting insulin was 19, 26, 20, and 19% higher among black women compared with white women with BMI levels of <22, 22–24, 25–27, and 28–30 kg/m^2, respectively. Means of insulin converged in the highest category, and the 95% CIs overlap. Mexican-American women have fasting insulin levels that are 17, 22, 20, and 16% higher than those of white women at BMI levels of 25–27, 28–30, 31–33, and >34 kg/m^2, respectively. Mean insulin levels converged between Mexican-American and white women at BMI levels <25 kg/m^2.

Differences in mean fasting insulin between black and white men at each BMI category were similar to those of women, but of a smaller magnitude (Fig. 2). Among men, the interaction term for race and the six-level BMI category was above the a priori determined 0.01 significance level (Table 2).

Although the pattern among men and women is similar, the 95% CIs overlap to a larger degree. Thus, a trend was observed in men, but the interactive relationship was stronger in women. In secondary analyses, we excluded individuals with diabetes and replicated the analyses. These results did not change (data not shown). Therefore, all etiologic modeling of the relationship between fasting insulin and ethnicity was restricted to women.

Etiologic modeling (Table 2), adjusted for established risk factors, did not attenuate this association in women. Each variable was entered separately along with all first- and second-order interaction terms. The inclusion of each separate variable did not significantly attenuate the significance of the interaction term. Additionally, the parameter estimates for both black and Mexican-American subjects did not change markedly. When age is entered in the model, the parameter estimate for the ethnicity (Mexican-American) and BMI interaction term appears to be attenuated. However, when graphically represented (not shown), the effect is the same in all three age categories.

CONCLUSIONS — In this population-based sample representative of the U.S., black and Mexican-American women have higher fasting insulin at similar levels of adiposity when compared with their white counterparts, whereas there were smaller and statistically insignificant differences among men. Compared with white women, there is a larger risk for black women at lean weight and

Table 2—*Linear regression modeling of the relationship between insulin and covariates*

Model	Parameters	*F* value 2 degrees of freedom	*P* value for ethnicity × BMI	Parameter estimate for ethnicity (black) × BMI	Parameter estimate for ethnicity (Mexican-American) × BMI
Model 1: Insulin = ethnicity + BMI + ethnicity × BMI					
1	Ethnicity × BMI interaction term				
	Women	14.23	<0.0001	−0.010	0.005
	Men	4.54	0.01	−0.002	0.008
All of the below modeled for women only					
2	Model 1 + diabetes (binary)	5.09	0.006	−0.021	0.021
3	Model 1 + education (binary, <high school vs. >high school)	11.94	<0.0001	−0.011	0.007
4	Model 1 + % calories from carbohydrates (highest quartile vs. rest)	8.54	0.0002	−0.009	0.003
5	Model 1 + age (in 20-year age groups)	11.72	<0.0001	−0.018	−0.001
6	Model 1 + income (family income <$20,000 vs. >$20,000	12.63	<0.0001	−0.010	0.000
7	Model 1 + physical activity (no physical activity vs. any)	16.32	<0.0001	−0.019	0.006
8	Model 1 + family history of diabetes	11.71	<0.0001	−0.007	0.021

Modeling in SAS. Models 2–8 contain covariate and all first- and second-order interaction terms. Interaction term contains 2 degrees of freedom. BMI was modeled continuously.

equivalent risk at obese weight, whereas in Mexican-American women, compared with white women, overweight has a larger effect on increasing fasting insulin. These findings suggest that the effect of obesity on insulin sensitivity is different for men and women and for Americans in ethnic minorities.

Obesity is thought to be an important determinant of insulin levels. The results of this study suggest that ethnicity may also contribute to fasting insulin levels. Ethnic variation in levels of fasting insulin presumably depends on an interaction of environmental factors and genetic factors that influence obesity and insulin sensitivity. Previous studies confirm higher levels of insulin in black subjects than in white subjects, starting in childhood (24). Similar results have also been reported in the Mexican-American population (25). Our study confirms in a large representative sample that these ethnic minorities have higher levels of fasting insulin at similar levels of obesity.

There has been much controversy recently in the study of race/ethnicity as a risk factor. Some argue that researchers use race as a surrogate for many other factors that are not measured. Despite the fact that we have considered and controlled for socioeconomic status, its multidimensional nature precludes us from excluding residual confounding by socioeconomic status. Studies have shown that mortality rates are still higher for black subjects than for white subjects, even when income is examined by category (26), and that disease risk factors are higher among black and Mexican-American women than among white women of comparable socioeconomic status (27). Ethnic designation is an important predictor of health status and susceptibility to disease (28–30).

The observed differences in fasting insulin by race were much stronger among women than men. Previous studies have reported differences in insulin metabolism between women and men with normal glucose tolerance, impaired glucose tolerance, and type 2 diabetes (31). Black women have a much higher prevalence of obesity and diabetes than black men (32). The combined presence of sensitivity to insulin's antilipolytic action with resistance to insulin's glucoregulatory action found in nondiabetic obese African-American women compared with men may contribute to the higher prevalence of obesity and type 2 diabetes in this group (33). Black-white differences in energy metabolism also seem to be sex specific, with lower energy expenditure observed particularly in black women (34). Mexican-American women are also more likely to have impaired glucose tolerance (35) and diabetes (27) than their male counterparts. These apparent sex differences in metabolism are not explained by differing levels of obesity and may explain the stronger effects seen in women.

Environmental causes that influence obesity may differ in different ethnic groups. We examined dietary carbohydrate intake and physical activity, but these factors may have been inadequately assessed in this large population-based sample. Black women may have different nutrition (32) and physical activity (36) influences on obesity than white subjects, although this has not been well studied (37). Black women may also have lower resting energy expenditure than white subjects, which may also influence obesity (38). One study comparing Mexican, Mexican-American, and white subjects suggested that genetic influences predominate in determining insulin levels, whereas environmental factors influence obesity (39). It is perceived that the environment is important in risk factor changes, although genetic characteristics may moderate the impact of these factors (40). Definitive comparisons of ethnic minorities on environmentally mediated aspects of obesity, including physical activity and nutrition, might contribute to models of obesity and may provide important new etiologic insights as well as indicate possible avenues of intervention. The NHANES III study is one of the most comprehensive national surveys to date. Extensive and complete data are available from both the home survey and medical examination. Response rates were high,

and there were minimal missing data. Unlike many surveys, NHANES III represents a sample of the U.S. population, and therefore results are generalizable. It also included oversampling of ethnic minorities to ensure that large numbers would be available for analyses.

Despite these strengths, there are several limitations to NHANES III. First, it is based on a cross-sectional survey design that does not allow one to draw inferences about causal pathways. Second, there are limitations to several of the variables we used, including BMI, physical activity, and diet. Although exact methods exist for measuring visceral adipose tissue accumulation, such as magnetic resonance imaging and computerized tomography, NHANES III did not use these more exact methods. However, studies have found that BMI is a preferred clinical and epidemiologic surrogate because of its association with adiposity, low cost, and ease of measurement (41). In addition, NHANES III lacks good measures of physical activity and does not measure physical activity at work. As a result, this covariate may not be adequately controlled for in our analyses. The validity of food frequency questionnaires in minority populations has also been debated (42).

In summary, we found evidence that at comparable levels of obesity, women of ethnic minorities, compared with white subjects, are more likely to have higher fasting insulin levels. This may indicate a predisposition to insulin insensitivity even at lower weights in these high-risk populations. Understanding epidemiologic data regarding differences between various ethnic groups should help reduce morbidity and mortality in minority populations. To address these differences in insulin metabolism, lifestyle and public health policy interventions are needed, such as those that influence food consumption patterns and stimulate physical activity to effect maintenance of lower weight in these groups. In addition, these findings should stimulate further research in this area to discern the pathway for these ethnic differences in insulin metabolism.

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