Differential Effects of BMI on Diabetes Risk Among Black and White Americans

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OBJECTIVE — To determine whether the associations of BMI and fat distribution with diabetes risk are modified by race.

RESEARCH DESIGN AND METHODS — Data from the National Health and Nutrition Examination Survey, Epidemiologic Follow-up Study (1971–1992), were used to investigate potential interactions of BMI and fat distribution with race. Incident diabetes was defined by self-report of physician-diagnosed diabetes, hospital and nursing home discharge records, and death certificates.

RESULTS — Among the 1,531 black and 9,852 white subjects who were nondiabetic at baseline, 1,139 (10.0%) developed diabetes during 20 years of follow-up. Although the cumulative risk of diabetes increased with baseline BMI in all four race-sex groups, the sex-specific odds ratios (ORs) for black:white subjects decreased with increasing BMI. In particular, for BMI of 22 kg/m^2, the OR of diabetes for black:white individuals was 1.87 and 1.76 ($P < 0.01$) for men and women, respectively; for BMI of 32 kg/m^2, the OR decreased to 0.99 and 1.20 (NS) for men and women, respectively. Skinfold ratio was also associated with increased diabetes risk in all race-sex groups, but did not modify the association between race and diabetes.

CONCLUSIONS — These findings suggest that the effect of BMI on diabetes risk is different for black and white Americans, with a larger risk for blacks than whites at low BMI and an equivalent risk for both groups at high BMI. A lower degree of visceral adiposity among blacks at higher BMI or a greater impact of visceral adiposity among blacks at low BMI may help explain the interaction of race and BMI on diabetes risk.

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Previous research on the prevalence and incidence of type 2 diabetes in the U.S. has consistently shown the frequency of diabetes to be higher among black than among white Americans and to be higher among obese individuals and those with centralized fat distribution (1–5). Possible explanations for the observed difference in the risk of diabetes between blacks and whites include differences in the distribution, clustering, or impact of known and potential diabetes risk factors such as obesity, fat distribution, insulin resistance, hypertension, level of physical activity, diet, and perhaps a genetic susceptibility to obesity and/or diabetes (6–13). Although the exact mechanisms by which obesity and centralized fat distribution are associated with increased diabetes risk are unclear (14), recent findings have suggested that the metabolic features associated with these factors may be different for blacks and whites (15). This raises the possibility that these factors modify the association between race and diabetes risk.

Understanding the complex associations of race, obesity, and fat distribution with the development of diabetes has been the focus of considerable research. A study by Lipton et al. (16) on incident diabetes in a representative sample of U.S. adults ages 25–70 years revealed significant differences in the distribution of diabetes risk factors between black and white subjects, with blacks having multiple risk factors for this disease. In particular, the authors reported higher BMI and subscapular-to-triceps skinfold ratio (STR) among blacks and showed that blacks were at a substantially higher 16-year risk of developing diabetes than whites. The present study built on these findings using an additional 5 years of follow-up data on the same cohort. We hypothesized that a continued excess risk of diabetes would be observed in black subjects in the sample, and that the association between race and diabetes risk would be modified by BMI and fat distribution, with lower levels of these factors being more strongly associated with diabetes risk among black subjects. The rationale for the second hypothesis was based on clinical findings suggesting a smaller correlation between anthropometric measures of obesity and centralized fat distribution and metabolic risk factors among blacks than among whites (15,17,18). Our study extended these findings to investigate the association of overall body mass and centralized fat distribution with diabetes risk in black and white Americans.

RESEARCH DESIGN AND METHODS

— Baseline data are from the First National Health and Nutrition Examination Survey (NHANES I; 1971–1975), and follow-up data are from the National Health and Nutrition Examination Survey, Epidemiologic Follow-Up Study (NHEFS). The NHEFS was designed

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Abbreviations: ICD-9, *Intemational Classification of Diseases, Ninth Revision*; NHANES I, First National Health and Nutrition Examination Survey; NHEFS, National Health and Nutrition Examination Survey, Epidemiologic Follow-Up Study; OR, odds ratio; sBP, systolic blood pressure; STR, subscapular-to-triceps skinfold ratio; WHR, waist-to-hip ratio.

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

to investigate associations among clinical, nutritional, and behavioral factors assessed in NHANES I and subsequent morbidity, mortality, and hospital utilization. The NHEFS cohort includes all subjects who were ages 25–74 years during the NHANES I and who completed the baseline medical examination (n = 14,407).

The design and operation of NHANES I and the NHEFS follow-up surveys have been described previously (19–22). Briefly, the baseline medical examination included measurements of height (m), weight (kg), subscapular and triceps skinfold thickness (mm), and sitting blood pressure (mmHg). BMI was calculated from measured height and weight (kg/m^2) and was used as an indicator of relative weight. The STR was used as an indicator of centralized fat distribution (23,24). The baseline interview included questions on exercise from recreational physical activity (categorized as low, moderate, or high), educational attainment, occupation, and income. Data from three NHEFS follow-up interviews—conducted in 1982–1984, 1987, and 1992—were used. Discharge diagnoses from overnight stays in hospitals and nursing homes were collected, and, for decedents, causes of death were collected from death certificates and coded according to the *International Classification of Diseases, Ninth Revision* (ICD-9) (25).

Definition of prevalent diabetes

To estimate the cumulative incidence of diabetes, prevalent cases were excluded from the baseline sample. Two types of information helped identify prevalent cases: self-report and a urine glucose test. At baseline, 713 subjects (67.6% of all prevalent cases) answered affirmatively to the question "Has a doctor ever told you that you have diabetes?" An additional 123 subjects (11.6%) were identified who reported no diabetes at baseline, but did report diabetes at one of the three follow-up exams and indicated a diagnosis date before the baseline interview. The discrepancy was identified using data on reported year of diabetes diagnosis that were available for those subjects who reported diabetes during follow-up. The choice to use all available data to identify prevalent cases was the most conservative approach to reduce misclassification. Of the 836 subjects who reported diabetes, 168 had a positive urine glucose test and 668 did not (Table 1).

A semiquantitative urine glucose test was used at baseline with ordered results—"negative," "trace," "light," "medium," "dark," and "very dark." Subjects with an indication of "trace" or more glucose in their urine were classified as prevalent cases and excluded from the analysis; 218 subjects (20.7%) were excluded based on this criterion alone (Table 1). Thus 1,054 subjects (7.3% of the original cohort) were considered to have had prevalent diabetes. Prevalent cases were older and had higher BMI, STR, and systolic blood pressure (sBP) than nondiabetic subjects who were included in the analysis (data not shown).

Sample selection

Of the 14,407 subjects in the NHEFS cohort, 172 subjects (1.2%) were excluded from the study because they self-reported their race as being other than black or white. After exclusion of the 1,054 prevalent cases of diabetes, 812 subjects (5.6%) were excluded because they were missing all follow-up data, and 871 (6.0%) were excluded because their diabetes status could not be classified by the end of the study. Of the remaining 11,498 subjects, 86 women who were pregnant at baseline were excluded because their BMI would be misleading, and 29 subjects were excluded because they were missing anthropometric data. The sample for this analysis therefore consisted of 11,383 subjects, representing 79% of the original NHEFS cohort.

To examine the effect that excluded subjects may have had on diabetes risk estimates, characteristics of subjects included in the sample were compared with those lost to follow-up (n = 812) and those whose diabetes status was not available (n = 871). Compared with subjects who were included in the study, both excluded groups were younger, were more likely to be black, and had higher sBP and lower BMI (data not shown). Although these differences were statistically significant because of the large number of subjects, the absolute differences were small. For example, subjects included in the sample had a mean BMI of 25.5 versus 25.1 kg/m^2 for those lost to follow-up.

Ascertainment of incident cases

At each follow-up survey, information from self-report, subjects' health care facility records, and death certificates was used to identify incident diabetes (ICD-9 codes 250.0–250.9) and gestational diabetes (ICD-9 code 648) (25). No cases of gestational diabetes were found. A total of 1,139 new cases of diabetes were identified. Table 1 shows the distribution and overlap of data sources contributing to their ascertainment. The majority of cases were identified from self-report or a facility record, and 45.1% of all cases were confirmed by an overlap of two or three sources of information.

Table 1—*Prevalent and incident cases of diabetes in the NHEFS, 1971–1992, by data source*

Prevalent cases	
Self-report and urine glucose	168 (15.9)
Self-report only	668 (63.4)
Urine glucose only	218 (20.7)
Total	1,054 (100.0)
Incident cases	
One source	
Self-report	420 (36.9)
Facility record	180 (15.8)
Death certificate	25 (2.2)
Two sources	447 (39.2)
Three sources	67 (5.9)
Total	1,139 (100.0)

Data are n (%).

Analytic strategy

NHANES I oversampled certain groups, such as older adults, low-income individuals, and women of childbearing age. Although the sampling design can be adjusted by using weights that allow calculation of national prevalence estimates based on the NHEFS (26), there has been controversy concerning use of these weights. Many epidemiological studies using NHEFS data have failed to mention them (27–30), whereas others have done weighted and unweighted analyses, presenting unweighted results only (31–33). In the latter studies, unweighted results were generally consistent with weighted ones. Because the goal of this study was to examine the association between risk factors and diabetes risk and not to provide national estimates, unweighted results are presented.

BMI, STR, and sBP were examined across race-sex strata, using generalized linear models to test for differences in age-adjusted means between groups. The distribution of categorical variables was evaluated across race-sex strata using the χ^2 test of association. Educational attainment (less than a high school education versus at least a high school education) was used as a proxy for socioeconomic status. Although data on occupation and income were also available, 3.6% of the data were missing for

Table 2—*Baseline characteristics of nondiabetic subjects in the NHEFS (n = 11,383)*

	Black men	White men	*P* value	Black women	White women	*P* value
n	591	4,059		940	5,793	
Age (years)						
25–34	88 (14.9)	773 (19.1)	0.001	226 (24.0)	1,506 (26.0)	0.001
35–44	72 (12.2)	667 (16.4)		265 (28.2)	1,360 (23.5)	
45–54	117 (19.8)	757 (18.6)		116 (12.3)	898 (15.5)	
55–64	76 (12.9)	633 (15.6)		94 (10.1)	676 (11.7)	
65–74	238 (40.2)	1,229 (30.3)		239 (25.4)	1,353 (23.3)	
BMI (kg/m^2)*	25.5 ± 4.9	25.6 ± 4.0	NS	27.7 ± 6.8	24.9 ± 5.1	<0.001
Median	24.9	25.5		26.9	23.8	
90th percentile	31.9	30.5		35.9	31.9	
STR†	1.6 ± 0.6	1.4 ± 0.5	<0.001	1.0 ± 0.3	0.8 ± 0.3	<0.001
Median	1.5	1.3		0.9	0.8	
90th percentile	2.4	2.1		1.4	1.2	
sBP (mmHg)*	145 ± 27	135 ± 21	<0.001	140 ± 29	131 ± 24	<0.001
Education (years)†			0.001			0.001
<12	4,036 (68.2)	1,724 (42.5)		560 (59.6)	2,063 (35.6)	
12	110 (18.6)	1,169 (28.8)		256 (27.2)	2,322 (40.1)	
>12	64 (10.8)	1,141 (28.1)		111 (11.8)	1,386 (23.9)	
Activity level†			0.001			0.001
Low	305 (51.6)	1,392 (34.3)		623 (66.3)	2,600 (44.9)	
Moderate	184 (31.1)	1,653 (40.7)		226 (24.0)	2,308 (39.8)	
High	102 (17.3)	1,010 (24.9)		89 (9.5)	882 (15.2)	

Continuous variables (BMI, STR, sBP) are expressed as age-adjusted means ± SD. Categorical variables (age, education, activity level) are *n* (%). For continuous variables, *P* value is for the test of difference in sex-specific means of the variable of interest. For categorical variables, *P* value is for the χ^2 test of association within sex. *Reported means are age-adjusted; †percentages do not add to 100.0 because of missing data.

income and 44.3% were missing for occupation. Moreover, unlike income and occupation, baseline education is likely to change the least over time among adults. Physical activity was examined in descriptive analyses, but was not used in multivariate models as most subjects were sedentary at baseline, which limited the variation in this measure. Using only age and race as predictors of diabetes, we used the logistic model to calculate age-adjusted odds ratios (ORs) and 95% CIs for the association between race and diabetes risk in the whole sample and separately by sex. Cumulative incidence rates of diabetes were standardized within race-sex-BMI strata by the direct method, using the age distribution of the whole sample as the standard.

Model selection

Using the logistic model, we examined the effects of BMI and STR on diabetes risk and the possibility that race modifies the effects of these variables. To further investigate this interaction, we calculated the ORs of diabetes associated with race (black:white) at various levels of BMI. Multivariate models were selected by the likelihood ratio test, and the Hosmer-Lemeshow test (34) was used to evaluate the goodness-of-fit of the final models. This method is based on the χ^2 distribution and tests the null hypothesis that the fit of a binary response model is adequate. Therefore, a small χ^2 statistic and a large *P* value for this test indicated good model fit. Regression diagnostics were performed to identify outlying observations and to determine if individual observations influenced the regression coefficients for race, BMI, or STR.

The initial models included BMI, STR, race, race × BMI, race × STR, age, sBP, and education. We also tested for quadratic effects for continuous variables. Models were run separately for men and women; age, sBP, and education were considered potential confounders. Variables from the initial model were selected for inclusion in the final model at the $P < 0.05$ level of significance, except for age, which was forced into the final model for both men and women.

RESULTS

— Baseline characteristics of the study sample are shown in Table 2. Black men were represented more in the oldest age group and were older than white men overall ($P = 0.001$). Black women had greater BMI and higher STR than white women. Among men, however, only STR was significantly different. Black men and women had higher age-adjusted sBP compared with whites (both $P < 0.001$), with black men having the highest sBP. Black subjects reported significantly less schooling and physical activity than whites ($P = 0.001$). Thus at baseline there was a clustering of diabetes risk factors among black subjects.

During the first 10 years of follow-up, ~50% of all incident cases were identified; during the each of the two subsequent 5-year follow-up segments, ~25% of the remaining cases were identified. The crude 20-year cumulative incidence of diabetes in this sample was 10.0%, but, as expected, race- and sex-specific rates varied greatly. The age-adjusted OR of diabetes for blacks:whites was 2.1 (CI 1.8–2.4). Black women had more than twice the age-adjusted risk of white women (OR = 2.5, CI 2.1–3.0). Similarly, black men were at greater risk than white men (OR = 1.5, CI 1.1–1.9).

A comparison of baseline characteristics of subjects who did and did not develop diabetes revealed no age differences among blacks who developed diabetes and those who did not, whereas significant age differences were noted among whites ($P < 0.001$) (Table 3). As expected, in each race-sex stratum, subjects who developed diabetes had

Table 3—*Comparison of baseline characteristics of subjects who did and did not develop diabetes, NHEFS, 1971–1992 (1,383)*

Variable	Black men			White men			Black women			White women		
	Diabetic	Nondiabetic	*P*	Diabetic	Nondiabetic	*P*	Diabetic	Nondiabetic	*P*	Diabetic	Nondiabetic	*P*
n	80	511		377	3,682		180	760		502	5,291	
Age (years)	54.9	53.4	NS	54.4	51.0	<0.001	47.6	47.7	NS	51.0	47.2	<0.001
BMI (kg/m^2)	27.9	25.2	<0.001	28.5	25.4	<0.001	31.3	26.9	<0.001	30.2	24.5	<0.001
STR	1.69	1.61	NS	1.60	1.37	<0.001	1.12	0.97	<0.001	0.96	0.76	<0.001
sBP (mmHg)	142	146	NS	142	135	<0.001	146	139	0.003	143	130	<0.001
<12 years of education	70	70	NS	53	42	0.001	67	59	0.036	53	34	0.001
Low physical activity	53	51	NS	41	34	0.001	72	65	NS	58	44	0.001

Continuous variables (BMI, STR, sBP) are mean baseline values. Categorical variables (age, education, activity level) are %. *P* for continuous variables are for the *t* test of difference in means between diabetic and nondiabetic subjects within race-sex strata. *P* for categorical variables are for the χ^2 test of association comparing diabetic with nondiabetic subjects within race-sex strata.

higher baseline BMI than those who did not ($P < 0.001$, all groups). Among women and white men, there were also significant differences in STR, with subjects who developed diabetes having a higher STR ($P < 0.001$). Significant differences in sBP between diabetic and nondiabetic subjects were observed among white men and women and black women, but not among black men. Subjects who developed diabetes had less education and were less physically active at baseline than those who did not.

Table 4 shows that the age-adjusted cumulative incidence of diabetes was greater with increasing BMI in all race-sex groups. Blacks were at higher risk of diabetes at all levels of BMI compared with whites. However, at lower BMI, the relative risk (RR) of diabetes for black:white subjects was much larger than at higher levels of BMI. For example, at BMI <20 kg/m^2, the adjusted RR was 2.83 and 3.13 for men and women, respectively, but at BMI ≥32 kg/m^2, the magnitude of this association decreased to 1.14 and 1.09, respectively. No similar trend was observed for STR, indicating that this variable did not modify the association between race and diabetes risk.

Results of logistic regression analyses relating the association of baseline characteristics to diabetes incidence are presented in Table 5. Our final models were complex and included linear and quadratic terms for BMI, a term for race, and an interaction term for race*BMI. The OR for skinfold ratio was expressed as a 0.2-unit increase, since this was more easily interpreted than a 1-unit increase in this measure. The interaction between race and BMI presented in Table 5 was calculated for BMI = 25. For all levels of BMI among men, low education, older age at baseline, and higher STR were predictive of diabetes risk. However, the association between race and diabetes risk was modified by BMI ($P = 0.02$). To quantify the magnitude of the interaction, we defined specific levels of BMI in our logistic models and calculated the black:white OR at these levels. Accordingly, at BMI = 22, the OR of diabetes for black:white men was 1.87 ($P < 0.01$), but at BMI = 32, the association decreased to 0.99 (NS). The Hosmer-Lemeshow test (34) indicated that this model fit the data well ($P = 0.19$). Although some outlying observations were detected, model diagnostics indicated that no individual observations influenced the regression coefficients for race, BMI, or STR. The association between race and diabetes risk was not modified by STR among men.

Although there were some differences in the structure of the final model for women, the associations of BMI and STR with dia-

Table 4—*Age-adjusted, BMI-specific cumulative incidence, and relative risk of diabetes, NHEFS, 1971–1992*

	Black			White			
	Diabetic (*n*)	Nondiabetic (*n*)	Cumulative incidence (%)	Diabetic (*n*)	Nondiabetic (*n*)	Cumulative incidence (%)	Relative risk
BMI (kg/m^2)							
Men							
<20	7	50	0.065	6	248	0.023	2.83
20 to <22	4	90	0.055	17	412	0.035	1.57
22 to <24	7	85	0.051	25	698	0.032	1.59
24 to <26	17	92	0.163	56	756	0.065	2.50
26 to <28	9	72	0.097	84	794	0.093	1.04
28 to <30	10	51	0.163	78	403	0.160	1.01
30 to <32	9	29	0.239	46	202	0.182	1.31
≥32	17	42	0.319	65	169	0.278	1.14
Women							
<20	5	99	0.050	10	758	0.016	3.13
20 to <22	6	79	0.085	19	1,051	0.022	3.86
22 to <24	9	93	0.083	49	1,085	0.037	2.24
24 to <26	14	103	0.114	65	801	0.074	1.54
26 to <28	22	109	0.173	69	556	0.108	1.60
28 to <30	22	75	0.236	61	378	0.140	1.68
30 to <32	30	66	0.292	59	267	0.183	1.59
≥32	72	136	0.330	170	395	0.302	1.09

Age distribution of the study sample used as standard population. Relative risk is black:white ratio.

Table 5—*Logistic regression analysis of the effects of baseline characteristics on diabetes risk, NHEFS, 1971–1992*

Variable	β coefficient	*P* value	OR (95% CI)
Men (n = 4,611)			
Race (black vs. white)	0.44	<0.01	1.55 (1.14–2.12)
Age (per 10 years)	0.01	<0.01	1.11 (1.03–1.20)
BMI (kg/m^2) (per unit)			
Black	0.20	<0.001	1.14 (1.08–1.20)
White	0.20	<0.001	1.22 (1.18–1.25)
STR (per unit)	0.38	<0.001	1.08 (1.04–1.12)
Education (≥12 years vs. <12 years)	0.25	0.02	1.28 (1.03–1.61)
BMI × race	−0.06	0.02	
BMI^2	−0.003	0.01	
Women (n = 6,663)			
Race (black vs. white)	0.45	<0.001	1.57 (1.20–2.06)
Age (per 10 years)	−0.008	0.12	0.99 (0.89–1.13)
BMI (kg/m^2) (per unit)			
Black	0.18	<0.001	1.16 (1.11–1.19)
White	0.18	<0.001	1.20 (1.16–1.23)
STR (per unit)	1.22	<0.001	1.25 (1.17–1.34)
sBP (per 5 mmHg)	0.006	<0.01	1.03 (1.01–1.05)
Education (≥12 years vs. <12 years)	0.41	<0.001	1.51 (1.25–1.81)
Race × BMI	−0.04	0.02	
BMI^2	−0.004	<0.001	
STR^2	−0.44	0.02	

The coefficients and ORs for race, BMI, and the interaction between race and BMI are expressed at a BMI of 25. For women, the coefficient for STR is expressed at a STR of 0.8. The final models for men and women excluded 30 and 70 subjects, respectively, because of missing data for education or blood pressure. The OR for BMI represents the effect of a 1.0 increase in BMI from 25 to 26 kg/m^2. The OR for STR among men represents the effect of any 0.2-U increase. For women, the OR for STR represents a 0.2-U increase from 0.8 to 1.0.

betes risk were similar to those observed for men. BMI modified the association between race and diabetes risk, with black women at higher risk of diabetes relative to white women at lower levels of BMI (P = 0.02). As was observed for men, STR did not modify the association between race and diabetes risk. However, a significant quadratic relationship between STR and diabetes risk was identified. By defining specific levels of BMI in the model, it was observed that at BMI = 22, the OR for black:white women was 1.76 ($P < 0.01$), but the magnitude of this association decreased to 1.20 for BMI = 32, and diabetes risk was not significantly different between black and white women at this level of BMI. No influential observations were noted, and the Hosmer-Lemeshow test indicated that the model provided a good fit for the data (P = 0.69).

Using the methods described above, the models were used to calculate the odds of diabetes at varying levels of BMI, as well as the OR of diabetes for blacks relative to whites. Figure 1*A* shows that the odds of diabetes increased with increasing BMI for all race-sex groups, but were higher among blacks at most levels of BMI. Figure 1*B* demonstrates that the sex-specific OR for diabetes among blacks relative to whites decreased with increasing BMI. Thus the modification by BMI of the association between race and diabetes risk narrowed the gap of diabetes risk between blacks and whites as BMI increased.

CONCLUSIONS — Our data indicated that the association between race and diabetes risk was modified by BMI, with a larger risk for blacks than whites at low BMI and an equivalent risk at high BMI. Although the interaction between race and BMI implies a relative decrease in the risk of diabetes associated with higher levels of BMI in blacks, black subjects still experienced a higher risk of diabetes at most levels of BMI. This could be due to the effect of BMI overwhelming the effect of race at higher levels of BMI or to higher levels of BMI having a greater relative impact on diabetes risk among whites. Alternatively, a lower degree of visceral adiposity among blacks at higher BMI or a greater impact of visceral adiposity among blacks at lower levels of BMI may help explain the interaction of race and BMI on diabetes risk. Unfortunately, differences in visceral adiposity cannot be detected by the anthropometric measures available in this data set.

Because metabolic factors associated with the deposition of intra-abdominal fat are more likely to influence diabetes risk than anthropometric features or BMI per se (17,35), the question remains as to which anthropometric measures best represent the metabolic features that enhance the risk of diabetes. This is an important issue in large epidemiological studies such as NHEFS because computed tomography and magnetic resonance data are frequently unavailable (36).

Mechanisms by which accumulation of abdominal fat may cause abnormalities of glucose and lipid metabolism have been described (17). However, significant ethnic differences in the deposition of abdominal fat for similar waist-to-hip ratios (WHRs) have been shown in black and white women (37). Differences in circumference measures have also been shown between black and white women of similar BMI (38). A possible link between these findings and our observation that BMI modifies the association between race and diabetes risk is that the impact of abdominal fat on diabetes risk may differ in blacks and whites.

The hypothesis of a differential association between measures of centralized fat distribution and metabolic risk factors for diabetes in blacks and whites is supported by findings from other studies. In the Insulin Resistance and Atherosclerosis Study (17), waist circumference was positively related to fasting insulin in subjects with normal and impaired glucose tolerance, but the association was significantly weaker among blacks than among whites, indicating ethnic variation in metabolic characteristics associated with this anthropometric measurement. This finding also suggests that waist circumference may not adequately represent abdominal fat in blacks. Lovejoy et al. (18) showed that black women had two seemingly incongruous characteristics: less visceral fat and lower insulin sensitivity, adjusted for BMI and WHR. This finding suggests a greater impact of visceral fat among black women.

Previous findings have seemed to indicate that common anthropometric indexes of body fat distribution are imperfect proxies for the high-risk metabolic characteristics associated with abdominal fat deposition and may therefore inadequately represent

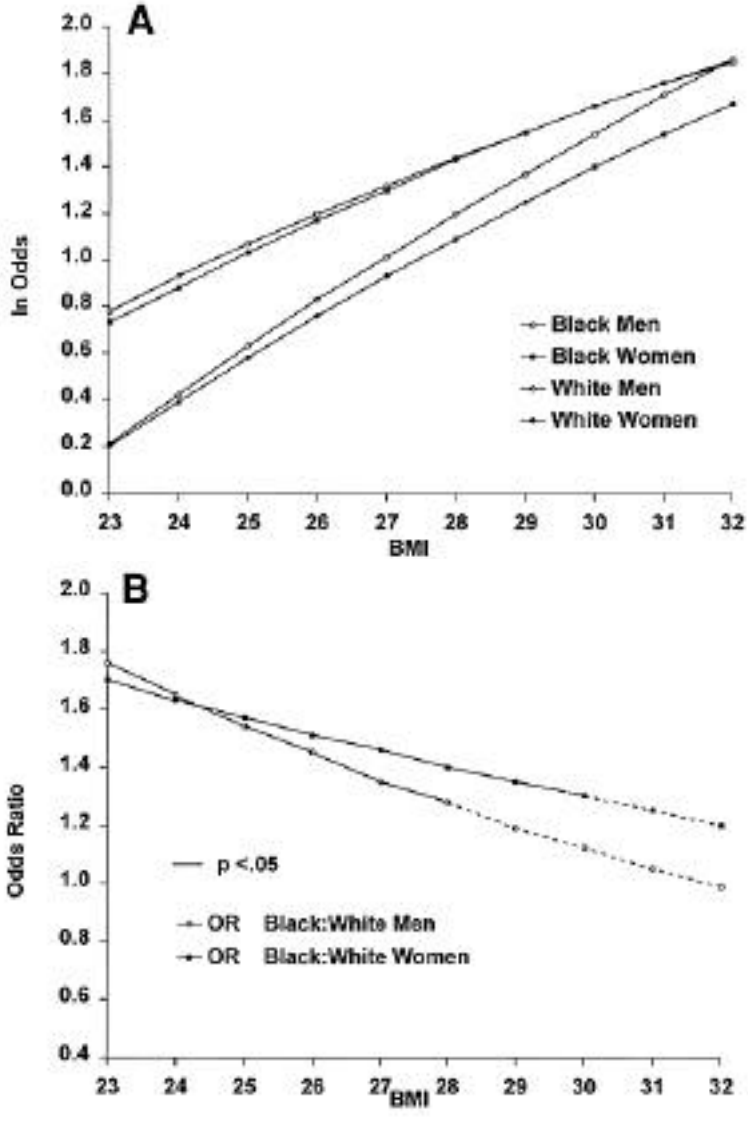

Figure 1—A*: Adjusted log-odds of diabetes in relation to baseline BMI by sex and race. Note the greater odds of diabetes among black men and women at lower BMI. B: Adjusted OR of diabetes in relation to BMI (blacks relative to whites) by sex. Note the decline in relative diabetes risk in blacks (decreasing OR) with increasing BMI. Solid line segments indicate levels of BMI at which the OR for race is significantly different from 1.0.*

the underlying diabetes risk profile in different ethnic groups. Although our data did not include circumference measures, we did have STR. Although skinfolds have been shown to correlate well with circumference measures and diabetes risk factors (38,39), our results did not indicate modification by STR of the association between race and diabetes risk. However, our findings did indicate that STR measures an important aspect of regional fat distribution with respect to diabetes occurrence, as it was found to be a strong independent predictor of disease risk over the 20 years of the study. This was consistent with previous findings that STR and WHR might reflect different aspects of body fat distribution, each with its own effect on metabolism (38). Thus, although STR is useful to consider when describing diabetes risk in general, it may not be a sufficiently sensitive proxy for differential metabolic consequences of centralized fat distribution between black and white Americans.

This study differs in several important ways from an earlier one that used the same data source to study diabetes incidence (16). First, an additional 5 years of data were analyzed, making the follow-up of this cohort 21 years and yielding 375 new cases of diabetes. Second, a more conservative definition of baseline diabetes was used to reduce misclassification of baseline disease status. This resulted in exclusion of more prevalent cases. Third, the interaction of both BMI and fat distribution with race was examined.

This study had several limitations, most of which were related to the fact that the NHEFS was not designed to examine diabetes incidence. Despite our efforts to identify all prevalent and incident cases of diabetes, it is likely that these were still underidentified in this data set (40–42), since the sample was not screened for diabetes and no data on glucose homeostasis were collected. To help address the issue of undiagnosed diabetes at baseline, urine glucose data were used to reduce misclassification of prevalent cases, and data from health care facilities and death certificates supplemented incident cases of diabetes that were ascertained from self-report. Implicit in our results was an underlying assumption about the accuracy of self-reported diabetes among the 36.9% of cases for whom only self-report data were available. This assumption was supported by a previous study that showed exceptional agreement between self-report of diabetes and medical records reflecting diagnosis of the disease, suggesting that individuals who know that they have diabetes report it accurately when asked (43).

To investigate the possibility that self-reported cases of diabetes might be misclassified cases of type 1 diabetes, we examined the characteristics of subjects who were age 30 years or younger at baseline and who self-reported diabetes between baseline and first follow-up (mean time, 10 years). There were 30 such subjects, only 4 of whom reported insulin use at first follow-up. One subject was diagnosed at age 32 years, one at age 33, and two at age 36. The mean BMI for this group was 32.1. From these data, we concluded that these cases were likely to be type 2 diabetes. In support of this was a recent report showing a very small prevalence of adult-onset IDDM in a national sample (44). Thus, although we use the term "diabetes" to describe incident cases throughout this report, our data suggest that this term is virtually equivalent to the term "type 2 diabetes."

Data collection for the NHEFS (1971–1992) included the period during which the National Diabetes Data Group and the World Health Organization first formalized criteria for the diagnosis and classification of diabetes (45,46). This data

set does not permit evaluation of a possible cohort effect related to the change in diagnostic criteria for diabetes across different segments of follow-up. However, because most cases of diabetes are identified during physician visits and without the oral glucose tolerance test, there was consistency with the interview question that asked specifically about physician-diagnosed diabetes. A more difficult question is whether the establishment of formal diagnostic and classification criteria led to changes in physician practice patterns that influenced the number or characteristics of cases identified during the study. Our findings must be further evaluated against the most recent changes in diagnostic criteria and screening recommendations (47). Application of the new criteria to this data set would likely have increased the number of prevalent and incident cases, since the definition of disease is set at lower fasting glucose levels and the new recommendations call for routine screening to begin at age 45 years, or at younger ages for individuals in high-risk groups.

Despite these limitations, our findings regarding ethnic differences in the effects of BMI over 20 years of follow-up were consistent with previous findings. Moreover, the longitudinal aspect of this study was one of its most notable strengths. The follow-up of this diverse cohort spanned 20 years and provided an excellent opportunity to investigate evidence of ethnic differences in the effects of BMI and fat distribution that has been gathered from smaller studies.

Although the Cox proportional hazards model is often used in cohort studies such as the NHEFS, this model was not selected for two reasons. First, 486 of the incident cases (42.6%) were missing a year of diagnosis. Second, the year of diagnosis is not a meaningful clinical end point, since the onset of diabetes may precede its clinical diagnosis by many years (48). Furthermore, year of diabetes diagnosis is partly a function of frequency of screening, which may reflect access to adequate health care. Given these methodological considerations, use of the Cox model to determine risk of diabetes using time-to-diagnosis would not provide additional insight on race, BMI, or fat distribution in relation to diabetes occurrence. In contrast, the logistic model allowed for use of all the data and did not base inferences on time–to–end point.

In summary, this study showed that over 20 years, approximately 10% of a large American cohort developed diabetes, and that the association between race and 20-year diabetes risk was modified by BMI, supporting the idea of ethnic differences in the effects of BMI in black and white Americans. These data highlight a potential paradox of differential BMI-associated diabetes risk in black and white Americans: compared with whites, blacks tend to be at higher risk of diabetes across most levels of BMI, yet the magnitude of risk among blacks relative to whites appears to be greatest at lower levels of BMI. An improved understanding of how these differences may affect long-term diabetes risk among nondiabetic individuals, especially lean blacks, may prove helpful in designing prevention programs that are tailored to meet the needs of individual risk groups.

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