ETHNIC COMPARISONS OF SARCOPENIA AND OBESITY IN DIABETES

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Objective: To examine the association between obesity and low relative skeletal muscle mass (sarcopenia) with type 2 diabetes and poor glycemic control, and to determine whether these associations varied by ethnicity.

Design, Setting, Participants: Data from the Third National Health and Nutrition Examination Survey (NHANES III) was used (5,727 adults, 40–74 years of age: 26% Mexican Americans, 25% non-Hispanic Blacks, and 49% non-Hispanic Whites).

Main Outcome Measures: Sarcopenia was defined as a skeletal muscle [SM(kg)/height(m^2)] <1 standard deviation below the young adult mean. Obesity was defined as waist circumference >102 cm in men and >88 cm in women.

Results: The prevalence of diabetes was 40% higher in Mexican Americans than in non-Hispanic Blacks or Whites ($P<.05$). The lowest prevalence of obesity and sarcopenia were observed in Mexican Americans (except for obesity in women). Independent of ethnicity, subjects with a high waist circumference were more likely to have diabetes and poor glycemic control ($P<.05$). These associations were strongest in non-Hispanic Whites. Conversely, sarcopenia was not associated with diabetes or poor glycemic control in any ethnic group.

Conclusions: Although the prevalence of type 2 diabetes was highest in Mexican Americans, non-Hispanic Whites exhibited the highest prevalence of obesity and sarcopenia. The role of genetically based, ethnic differences in body composition and diabetes risk needs to be taken into account when developing clinical guidelines such as the waist circumference cut-points used in this study. (*Ethn Dis.* 2005;15:664–670)

Key Words: Body Composition, Chronic Disease, Type 2 Diabetes

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INTRODUCTION

Obesity, an increasingly significant health problem worldwide, is associated with insulin resistance, diabetes, and metabolic syndrome.[1] Abdominal obesity has been shown to be more strongly linked to insulin resistance, type 2 diabetes, and cardiovascular disease than total adiposity.[2] However, the association between total and abdominal obesity with diabetes may vary by race and ethnicity. Using data from the NHANES III study, Okosun found that a high waist circumference, a marker of both total and abdominal obesity,[3] accounted for ≈12% of the difference in diabetes rates between White and Black women and ≈10% between White and Mexican-American women.[4] In another study of Hispanic elders of Caribbean origin living in Massachusetts, Bermudez et al[5] found that total and abdominal obesity had a differential effect on the presence of diabetes. The prevalence of diabetes was strongly associated with total and central obesity among non-Hispanic White women but not in Mexican Americans.

Low muscle mass may impair glucose disposal since skeletal muscle is the major site of insulin-stimulated glucose uptake.[6] However, little is known about ethnic differences in skeletal muscle mass and its association with diabetes. Therefore, the objective of the present study was to examine the associations between low muscle mass (sarcopenia) and obesity with diabetes and poor glycemic control and to determine whether these associations differed by ethnicity.

METHODS

Study Population

We excluded from the total Third National Health and Nutrition Examination Survey (NHANES III) sample[7] those persons who did not have fasting measures of glucose, insulin, and glycosylated hemoglobin (A1C); individuals who did not undergo a two-hour oral glucose tolerance test; subjects who did not have measures of height, weight, and waist circumference; subjects who did not have bioelectrical impedance measures necessary to calculate skeletal muscle mass; pregnant women; subjects who were not of Mexican-American, non-Hispanic Black, or non-Hispanic White ethnicity; and those who were missing measures of one or more of the confounding variables (see below). This left a total of 5,727 subjects between 40 and 74 years of age for analysis.

Anthropometric Variables

Height was measured to the nearest 0.1 cm, and weight was measured to the nearest 0.1 kg by using standardized equipment and procedures.[7,8] Body mass index (BMI) was calculated as weight divided by height squared (kg/m^2). Waist circumference was obtained with a flexible tape to the nearest 0.1 cm while the subjects were standing in an erect position with feet together and weight evenly distributed.[7] The waist circumference measure was made at minimal respiration at the level of the iliac crest.

Skeletal Muscle Mass

Bioelectrical impedance analysis (BIA) measurements (ohms) were ob-

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tained by using a Valhalla 1990B Bio-Resistance Body Composition Analyzer (Valhalla Medical, San Diego, Calif) with an operating frequency of 50 kHz at 800 μA. Whole-body BIA measurements were taken between the right wrist and ankle with the subject in a supine position[9] after the subjects completed a minimum six-hour fast. Whole-body muscle mass was calculated with the BIA equation of Janssen and colleagues[10] defined as: skeletal muscle mass (kg)=[([height^2/BIA resistance] * 0.401) + (sex * 3.825) + (age * −0.071)] + 5.102 where height is in cm; BIA resistance is in ohms; for sex, men=1 and women=0; and age is in years. Absolute muscle mass (kg) was normalized for height (muscle mass in kg/height in m^2) and referred to as skeletal muscle index.

Metabolic Variables

Fasting blood samples were used to measure glucose, insulin, and glycosylated hemoglobin (HbA1C). A 75-g oral glucose challenge was given, and a blood sample was drawn two hours (±15 min) later to measure glucose and insulin. Glucose level was determined with a hexokinase enzymatic method,[7] and serum insulin was measured with a Pharmacia Insulin RIA kit (Pharmacia Diagnostics AB, Uppsala, Sweden). Post–glucose challenge insulin values were available for 52% of the subjects.

Confounding Variables

Confounding variables, including age, education, and health behaviors (alcohol consumption, smoking, physical activity), were assessed by questionnaire. Age was included in the analysis as a continuous variable. Education level was used as an indicator of lifelong socioeconomic status, which unlike income or occupation, is not affected by the study outcomes. Education was divided into three categories: <8 years, 8–12 years, or >12 years. Alcohol consumption was categorized as none (0 drinks/month), moderate (1–15 drinks/month), or heavy (>15 drinks/month). Subjects were considered current smokers if they smoked at the time of the interview; previous smokers if they were not current smokers but had smoked 100 cigarettes, 20 cigars, or 20 pipes full of tobacco in their entire life; and non-smokers if they smoked less than these amounts. Based on the subject's reports of their monthly frequency of engaging in leisure time physical activities, they were grouped into none (<4 times/month), low (4–10 times/month), moderate (11–19 times/month), or high (>19 times/month) physical activity categories.

Definition of Groups and Terms

Obesity was defined as having a waist circumference >102 cm in men or >88 cm in women, in accordance with the National Institutes of Health obesity guidelines.[11] We used waist circumference rather than BMI to define obesity because waist circumference is a stronger correlate of abdominal and visceral fat than BMI. In addition, waist circumference is as strong a correlate of total fat as is BMI.[3] Previous research has shown that waist circumference adds to the predictive power of BMI in determining obesity-related health risk,[12–14] whereas BMI does not add to the predictive capacity of waist circumference in determining obesity-related health risk.[15]

Low muscle mass (or sarcopenia) was defined as having a whole body skeletal muscle mass (kg/m^2) less than one standard deviation below the mean of young (18- to 39-year-old) healthy adults. These cut-points were determined by using the NHANES III data and are established at 9.5 kg/m^2 for men and 7.0 kg/m^2 for women.[16] To consider the independent effects of obesity and sarcopenia on diabetes-related outcomes, subjects were further subdivided into four groups based on obesity and sarcopenia status: non-obese with normal muscle mass as the reference group, obese with normal muscle mass, non-obese with sarcopenia, and obese with sarcopenia.

Type 2 diabetes was defined according to the American Diabetes Association (ADA) guidelines as having a fasting plasma glucose level ≥126 mg/dL or post–glucose challenge values ≥200 mg/dL.[17] Individuals with diagnosed diabetes (other than gestational diabetes and if diabetes was diagnosed prior to age 25) and those who reported using hypoglycemic agents were also considered to have type 2 diabetes. Poor glycemic control and hyperinsulinemia were defined per ADA guidelines as having an A1C level >6.5%[18] and a fasting insulin level of >25 mU/mL,[19] respectively.

Statistical Analysis

The Intercooled Stata 7 program (Stata Corporation, College Station, Tex) was used to weigh the sample to be representative of the population and to take into account the complex sampling strategy of the NHANES III design. Differences in subject characteristics were compared between Mexican Americans, non-Hispanic Blacks and Whites by analysis of variance and Scheffe post-hoc comparisons. Prevalence rates of type 2 diabetes and diabetes indicators across ethnic groups were compared by using χ^2 statistics.

Univariate associations using Pearson correlation coefficient were carried out to determine the association between body composition variables. Bivariate and multiple logistic regression analyses were used to examine the associations between body composition classification with diabetes, poor glycemic control, and hyperinsulinemia within each ethnic group. Dummy variables were created to compute odds ratios (OR) and 95% confidence intervals (CI) for these factors. Two models are presented: Model 1 represents OR adjusted for the confounding effects of age and sex, while Model 2 shows OR adjusted for education, alcohol consumption, smoking, and physical activity in addition to age and sex. The logistic regression models were employed to determine if the patterns of associations between the body composition and diabetes outcomes varied by ethnicity. Furthermore, logistic regression analysis was also used to examine the independent and combined effects of obesity and muscle on diabetes risk, with waist circumference and skeletal muscle index being entered into the regression model as continuous variables. For this analysis the OR were computed for each unit increase in waist circumference (cm) and skeletal muscle index (kg/m^2).

RESULTS

Study Population

Table 1 shows the characteristics of the study sample.

Diabetes Indicators by Diabetes Status and Ethnicity

Few significant ethnic differences were seen in diabetes indicators among diabetic men and women (Table 2). However, non-Hispanic Whites tended to have the most favorable indicators. Similarly, some ethnic differences in diabetes indicators were seen in non-diabetic men and women (Table 2). However, non-Hispanic Whites without diabetes also had the most favorable indicators, with the exception of basal and two-hour glucose levels in White men and two-hour glucose level in White women.

Logistic Regression Models

Positive univariate associations across ethnic groups were found between waist circumference and BMI (r values 0.87–0.96, $P<.05$), BMI and skeletal muscle index (r values 0.65–0.74, $P<.05$), and between waist circumference and skeletal muscle index (r values 0.46–0.60, $P<.05$). Within each ethnic group, bivariate logistic regression models were used to determine the association between obesity status and sarcopenia status with diabetes, poor glycemic control, and hyperinsulinemia. There were significant associations for obesity and nonsignificant associations for sarcopenia (data not shown) independent of race and diabetes indicators. The lack of an association with sarcopenia may be reflected by the fact that muscle is positively associated with waist circumference. Thus, on average, individuals with a low skeletal muscle mass or sarcopenia had lower waist circumference values.

Therefore, we looked at four obesity $\times$ sarcopenia subgroups: 1) non-obese/normal muscle (reference group); 2) obese/normal muscle; 3) non-obese/sarcopenic; and 4) obese/sarcopenic to examine the associations between body composition classification with diabetes-related outcomes for each ethnic group (Table 3). Within each ethnic group, individuals with obesity but a normal muscle mass were more likely to have diabetes, poor glycemic control (A1C levels >6.5%), and hyperinsulinemia (basal insulin >25 mU/mL) than those in the reference group ($P<.05$). In contrast, the non-obese individuals with sarcopenia were not more likely to have any of these conditions (diabetes, poor

Table 1. Descriptive characteristics of the study population

	Men			*Women*		
	Mexican Americans (n=775)	**Non-Hispanic Blacks (n=677)**	**Non-Hispanic Whites (n=1372)**	**Mexican Americans (n=693)**	**Non-Hispanic Blacks (n=748)**	**Non-Hispanic Whites (n=1462)**
Age (y)	50.9 ± 9.2	52.6 ± 10.0*	54.0 ± 10.2†	51.6 ± 9.3	52.2 ± 9.9*	54.7 ± 10.2†
Years of education	8.2 ± 5.0	11.0 ± 3.6*	13.0 ± 3.1†	8.2 ± 4.6	11.6 ± 3.1*	12.6 ± 2.7†
BMI (kg/m^2)	28.3 ± 4.4	26.9 ± 4.9*	27.5 ± 4.6†	29.6 ± 5.6	30.1 ± 7.2	27.1 ± 6.1†
Waist circumference (cm)	99.8 ± 11.1	95.5 ± 12.9*	100.2 ± 11.9†	96.1 ± 12.2	97.8 ± 15.7	91.8 ± 14.4†
Skeletal muscle mass (kg)	30.7 ± 4.0	31.4 ± 4.5*	32.4 ± 4.6†	19.1 ± 2.8	20.8 ± 3.6*	19.8 ± 3.3†
Skeletal muscle index (kg/m^2)	10.7 ± 1.2	10.2 ± 1.2*	10.4 ± 1.2†	7.8 ± 1.1	7.8 ± 1.2	7.5 ± 1.2†
Type 2 diabetes (%)	23.1	14.4*	14.7 *	27.6	15.9*	14.7 *
Abdominal obesity (%)	41.6	29.4*	43.1†	74.7	73.6	56.7†
Sarcopenia (%)	13.4	25.8*	22.5*	19.7	25.0	31.0†
Sarcopenia + abdominal obesity (%)	2.1	3.0	4.9*	9.9	11.5	11.9

Mean ± SD.
Comparisons of Mexican Americans, non-Hispanic Blacks, and non-Hispanic Whites using ANOVA (continuous variables) and χ^2 (prevalence scores) analysis.
* $P<.05$ vs Mexican Americans within same sex.
† $P<.05$ vs Mexican Americans and non-Hispanic Blacks within same sex.

Table 2. Diabetes indicators by diabetes status and ethnicity in men and women

	Diabetics			*Non-Diabetics*		
	Mexican Americans	**Non-Hispanic Blacks**	**Non-Hispanic Whites**	**Mexican Americans**	**Non-Hispanic Blacks**	**Non-Hispanic Whites**
Men	(*n*=201)	(*n*=111)	(*n*=238)	(*n*=574)	(*n*=566)	(*n*=1134)
Glycosylated hemoglobin (%)	7.04 ± 1.99	7.04 ± 1.93	6.64 ± 1.73*	5.43 ± 0.44	5.53 ± 0.61*	5.29 ± 0.43‡
Poor glycemic control (A1C>6.5%) (%)	43.3	50.7	40.7 †	1.2	3.3*	0.4‡
Basal glucose level (mg/dL)	150 ± 72	145 ± 65	141 ± 57	98 ± 10	95 ± 10*	96 ± 9‡
2h glucose level (mg/dL)	281 ± 100	267 ± 98	259 ± 87*	118 ± 34	113 ± 35*	115 ± 33
Basal insulin level (mU/mL)	18.9 ± 13.6	17.2 ± 15.9	17.9 ± 13.6	12.9 ± 13.9	10.8 ± 7.7*	10.0 ± 6.1‡
Hyperinsulinemia (basal insulin >25 mU/mL) (%)	19.9	15.9	17.6	6.9	5.7	3.2‡
2h insulin level (mU/mL)§	72.1 ± 53.3	58.5 ± 57.0	67.2 ± 45.7	67.8 ± 62.8	50.3 ± 46.9*	49.2 ± 41.3*
Women	(*n*=216)	(*n*=134)	(*n*=260)	(*n*=477)	(*n*=614)	(*n*=1202)
Glycosylated hemoglobin (%)	6.96 ± 1.97	7.57 ± 2.44*	6.25 ± 1.60‡	5.36 ± 0.42	5.56 ± 0.53*	5.24 ± 0.42‡
Poor glycemic control (A1C>6.5%) (%)	42.3	52.6	23.8 ¶	0.3	1.3*	0.3
Basal glucose level (mg/dL)	141 ± 69	157 ± 89	126 ± 56‡	95 ± 9	94 ± 10*	93 ± 8*
2h glucose level (mg/dL)	287 ± 101	281 ± 118	260 ± 84*	130 ± 32	118 ± 33*	121 ± 34*
Basal insulin level (mU/mL)	17.7 ± 12.5	17.7 ± 12.6	15.7 ± 12.8	12.8 ± 8.6	12.6 ± 9.2	9.1 ± 5.8‡
Hyperinsulinemia (basal insulin >25 mU/mL) (%)	18.9	15.5	15.2	6.3	6.3	2.5‡
2h insulin level (mU/mL)§	84.2 ± 54.6	77.1 ± 67.2	83.2 ± 80.5	91.9 ± 87.9	70.0 ± 60.2*	52.4 ± 41.5‡

Mean ± SD.
Comparisons of Mexican Americans, non-Hispanic Blacks, and non-Hispanic Whites by diabetes status by using ANOVA.
* $P<.05$ vs Mexican Americans within the same diabetes status.
† $P<.05$ vs non-Hispanic Blacks within same diabetes status.
¶ $P<.05$ vs Mexican Americans and non-Hispanic Blacks within same diabetes status.
§ Data for two-hour (2h) plasma insulin levels were only available for 52% of the study participants.

Table 3. Odd ratios for diabetes, poor glycemic control, and hyperinsulinemia by ethnicity and body composition classification

		Non-Obese, Normal Muscle	**Obese, Normal Muscle**	**Non-Obese, Sarcopenic**	**Obese, Sarcopenic**
Mexican Americans		(*n*=146)	(*n*=1212)	(*n*=566)	(*n*=309)
Diabetes	Model 1	1.00	2.63 (1.75, 3.95)*	1.79 (1.06, 3.01)*	1.56 (0.83, 2.96)
	Model 2	1.00	2.57 (1.72, 3.85)*	1.90 (1.13, 3.18)*	1.51 (0.79, 2.88)
Poor glycemic control	Model 1	1.00	2.37 (1.39, 4.04)*	1.06 (0.46, 2.44)	1.05 (0.42, 2.62)
	Model 2	1.00	2.28 (1.33, 3.91)*	1.15 (0.51, 2.61)	0.97 (0.38, 2.46)
Hyperinsulinemia	Model 1	1.00	7.43 (3.52, 15.70)*	0.49 (0.11, 2.08)	2.38 (0.84, 6.76)
	Model 2	1.00	7.26 (3.43, 15.37)*	0.52 (0.12, 2.27)	2.48 (0.87, 7.10)
Non-Hispanic Blacks		(*n*=405)	(*n*=651)	(*n*=259)	(*n*=110)
Diabetes	Model 1	1.00	2.41 (1.58, 3.66)*	0.73 (0.42, 1.25)	1.52 (0.83, 2.80)
	Model 2	1.00	2.41 (1.56, 3.73)*	0.78 (0.45, 1.36)	1.57 (0.84, 2.94)
Poor glycemic control	Model 1	1.00	2.99 (1.85, 4.84)*	0.70 (0.35, 1.42)	1.06 (0.45, 2.50)
	Model 2	1.00	3.08 (1.87, 5.07)*	0.76 (0.37, 1.56)	1.08 (0.45, 2.58)
Hyperinsulinemia	Model 1	1.00	5.38 (2.56, 11.31)*	0.14 (0.03, 0.62)*	2.84 (0.84, 9.63)
	Model 2	1.00	5.43 (2.53, 11.66)*	0.14 (0.03, 0.63)*	2.76 (0.78, 9.71)
Non-Hispanic Whites		(*n*=747)	(*n*=727)	(*n*=199)	(*n*=126)
Diabetes	Model 1	1.00	3.94 (2.75, 5.63)*	1.48 (0.96, 2.28)	3.62 (2.32, 5.62)*
	Model 2	1.00	3.67 (2.56, 5.27)*	1.46 (0.96, 2.27)	3.35 (2.12, 5.28)*
Poor glycemic control	Model 1	1.00	7.92 (4.26, 14.74)	1.33 (0.56, 3.16)	4.96 (2.26, 10.87)*
	Model 2	1.00	6.78 (3.56, 12.80)*	1.29 (0.54, 3.09)	4.27 (1.88, 9.68)*
Hyperinsulinemia	Model 1	1.00	24.96 (9.34, 66.65)*	0.59 (0.10, 3.45)	5.07 (1.37, 18.78)*
	Model 2	1.00	19.60 (7.32, 52.43)*	0.59 (0.10, 3.45)	3.68 (1.01, 13.48)*

Odds ratios with 95% confidence intervals.
Model 1: Adjusted for age and sex.
Model 2: Adjusted for age, sex, education, physical activity, smoking, and alcohol consumption.
* $P<.05$ vs low waist, high muscle (reference) group.

glycemic control, or hyperinsulinemia) by comparison to the reference group (exception: diabetes in Hispanics). Non-Hispanic Whites with both obesity and sarcopenia were more likely to have diabetes, poor glycemic control, and hyperinsulinemia than non-Hispanic Whites without obesity or sarcopenia. Conversely, the ORs for the obese, sarcopenic group were not significantly increased in Hispanics and non-Hispanic Blacks.

To determine if the results were influenced by the obesity and sarcopenia cut-points employed, all logistic regression analyses were repeated by using sex-specific tertiles, in order to determine the cut-points for waist circumference and skeletal muscle index. Similar results were obtained by using the tertile approach (data not shown).

Finally, Table 4 shows the results of the logistic regression in which waist circumference alone (index of obesity), skeletal muscle index alone (index of sarcopenia), or the combination of waist circumference and skeletal muscle index were used as continuous variables to predict diabetes, poor glycemic control, and hyperinsulinemia within ethnic groups. Without exception, both waist circumference alone and skeletal muscle index alone were strong positive predictors of diabetes, poor glycemic control, and hyperinsulinemia in each of the ethnic groups. Because the units for waist circumference and skeletal muscle index are different, the magnitudes of the OR for waist circumference and skeletal muscle index presented in Table 4 are not directly comparable. For example, in Mexican Americans the OR for diabetes was 1.04 for waist circumference and 1.29 for skeletal muscle index. Thus, for every 1.0 cm increase in waist circumference (a relatively small increase) the odds of metabolic syndrome increased by 4% and for every 1.0 kg/m^2 increase in skeletal muscle index (a relatively large increase) the odds of metabolic syndrome increased by 29%. When both waist circumference and skeletal muscle index were included in the same regression model, waist circumference remained a positive predictor of diabetes, poor glycemic control, and hyperinsulinemia in all three ethnic groups. However, when both waist circumference and skeletal muscle index were included in the same regression model, skeletal muscle index was no longer a predictor of diabetes indicators in Mexican Americans or non-Hispanic Blacks and remained a positive predictor of poor glycemic control and hyperinsulinemia in non-Hispanic Whites.

DISCUSSION

We observed that the prevalence of diabetes and poor glycemic control was highest among minority groups living in the United States, specifically in Mexican-American men and women. This ethnic group also exhibited the lowest prevalence of low muscle mass or sarcopenia. Independent of ethnicity, obesity (as determine by waist circumference) was significantly associated with an increased likelihood of diabetes and poor glycemic control, while sarcopenia (low muscle mass) was not a positive predictor of diabetes or poor glycemic control. The associations for obesity were strongest in non-Hispanic Whites.

Previous reports using NHANES III data show that within normal weight, overweight, and class I obese BMI categories, American women of all ethnicities with abdominal obesity (defined as a waist circumference >88 cm) were more likely to have diabetes and metabolic syndrome abnormalities than

Table 4. Odds ratios for diabetes, poor glycemic control, and hyperinsulinemia by using prediction models with waist circumference (WC) alone, skeletal muscle index (SMI) alone, or both WC and SMI

	WC Alone OR (95% CI)*	SMI OR (95% CI)*	WC + SMI	
			WC OR (95% CI)*	SMI OR (95% CI)*
Mexican Americans				
Diabetes	1.04 (1.02–1.05)†	1.29 (1.13–1.49)†	1.03 (1.10–1.05)†	1.09 (0.91–1.29)
Poor glycemic control	1.04 (1.02–1.06)†	1.29 (1.08–1.55)†	1.04 (1.02–1.06)†	1.04 (0.84–1.30)
Hyperinsulinemia	1.09 (1.07–1.11)†	1.81 (1.51–2.18)†	1.08 (1.05–1.10)†	1.19 (0.93–1.53)
Non-Hispanic Blacks				
Diabetes	1.04 (1.02–1.05)†	1.32 (1.15–1.51)†	1.03 (1.02–1.05)†	1.05 (0.90–1.23)
Poor glycemic control	1.04 (1.03–1.06)†	1.48 (1.27–1.72)†	1.04 (1.02–1.05)†	1.15 (0.96–1.37)
Hyperinsulinemia	1.06 (1.04–1.07)†	1.67 (1.42–1.96)†	1.05 (1.03–1.06)†	1.20 (0.98–1.48)
Non-Hispanic Whites				
Diabetes	1.04 (1.03–1.05)†	1.38 (1.23–1.53)†	1.04 (1.03–1.05)†	1.09 (0.95–1.25)
Poor glycemic control	1.06 (1.05–1.08)†	1.64 (1.40–1.91)†	1.05 (1.03–1.07)†	1.23 (1.00–1.50)†
Hyperinsulinemia	1.12 (1.10–1.14)†	2.61 (2.20–3.10)†	1.09 (1.07–1.11)†	1.62 (1.32–1.98)†

* Odds ratios (95% confidence intervals). WC and SMI were included in the regression model as continuous variables and the OR were computed for each unit increase in WC (cm) and SMI (kg/m^2). The OR were adjusted for age, sex, education, physical activity, smoking, and alcohol consumption.

† Significantly greater odds ($P<.05$).

those with normal waist circumference.[13] Similar findings were observed by Okosun et al[20] with the same data set. These investigators showed that in men and women of all ethnic groups, the risk for metabolic syndrome abnormalities was strongly associated with increased waist circumference independent of BMI. Taken together, these results indicate that waist circumference measurements should be considered as a clinical variable for assessing metabolic health risk, particularly as it relates to hypertension, dyslipidemia, diabetes, and cardiovascular disease.

Waist circumference cut-points employed in this study were developed with data from a group composed primarily of non-Hispanic Whites.[21] Current evidence suggests that waist circumference adds to the predictive power of BMI in determining metabolic health risk.[12–15] However, within a given BMI category the ability of the waist circumference cut-points employed here to predict obesity-related comorbidities is higher in non-Hispanic Whites compared to Hispanics and non-Hispanic Blacks.[22] Nonetheless, newly published findings indicate that within a given ethnic group, the optimal waist circumference cut-points for predicting coronary heart disease risk are similar in different ethnic groups.[23]

Diabetes may be compounded by an imbalance between increased total and abdominal obesity[24] and reduced muscle mass.[25] Skeletal muscle is the target tissue for ≈80% of insulin-mediated glucose uptake,[6] and low muscle mass may lead to or result from insulin resistance and poor glycemic control. Contrary to this hypothesis, in the present study we found that Mexican-American men and women had the lowest prevalence of sarcopenia even though they had the highest prevalence of diabetes and poor glycemic control. Ethnic differences in muscle mass and the association of low muscle mass to metabolic risk have not been previously studied. In a cross-sectional study of 54 Mexican-American women between 20–75 years of age and 56 White women of similar age and socioeconomic status, ethnicity was found to be associated with modestly higher amounts of abdominal adiposity and slightly lower amounts of fat-free mass (muscle, viscera, and bone) measured by dual-energy X-ray absorptiometry.[26] Our findings using NHANES III population-based data extend from those of Casas and colleagues in that a measure of skeletal muscle mass was used[10] rather than a measure of total fat-free mass.

We found that non-Hispanic Whites exhibited the highest prevalence of obesity and sarcopenia, despite having the lowest prevalence of diabetes. To further assume a health risk at the individual level based on these observations (derived from aggregate or group-level data), would be an ecological fallacy.[27] Nonetheless, aggregate data are often easier to obtain, and at times can offer valuable insight into associations that occur at the individual level.

The present study was not designed to address potential mechanisms associated with ethnicity-related differences in body composition in terms of diabetes and poor glycemic control. However, our findings suggest that, in addition to the more widely studied socioeconomic and demographic factors,[28] genetic factors[29] may also play an important role in explaining the increased risk of diabetes in Hispanics. The challenge is to identify the appropriate combination of genetic and environmental factors for a given ethnic group and to design effective targeted interventions to reduce the risk factors associated with chronic conditions such as diabetes. We tested a resistance training intervention in Hispanics with diabetes that was safe and effective in improving glycemic control.[30] However, more research is needed in this area.

Some limitations of the present study need to be addressed. First, the cross-sectional design of NHANES III limits our ability to draw inferences about causal pathways. A second limitation is the potential bias due to survey non-response and missing values for some of the variables. Previous NHANES reports, however, have shown little bias due to non-response.[31] In addition, response bias may have been from the confounding variables (alcohol use, smoking, physical activity, and education) that were assessed by questionnaire. Third, the sample size for Mexican-American and non-Hispanic Blacks is smaller than that for non-Hispanic Whites, which may have limited our statistical power. Finally, the applicability and arbitrary nature of the cut-points used for waist circumference and muscle mass should be considered. Waist circumference estimates were established because they corresponded to a BMI of 30 kg/m^2 and not because they represented the best cut-points for predicting health risk. Similarly, the muscle mass cut-points were based on the population distribution in young adults and are not health based.

In conclusion, this study corroborates the finding that a high waist circumference is associated with metabolic health risk independent of ethnicity. Surprisingly, low muscle mass (or sarcopenia) was not associated with an increased likelihood of diabetes and poor glycemic control. This finding suggests that interventions aimed at preventing and/or treating diabetes should focus on reducing total and abdominal fat. The role of genetically based, ethnic differences in body com-

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position and diabetes risk should be taken into account when developing clinical guidelines such as the waist circumference cut-points used in this study.

ACKNOWLEDGMENTS

Carmen Castaneda, MD, PhD is a recipient of the Brookdale National Fellowship and the International Life Sciences Institute Future Leader Award. The results of this study were presented in part at the 2nd International Academy of Nutrition and Aging Congress in Albuquerque, New Mexico in July 2003.

This work was supported in part by the US Department of Agriculture, under agreement No. 58-1950-4-401 (CC), and a Canadian Institutes of Health Research Postdoctoral Fellowship (IJ). These sources had no role in the design of the study but had oversight of aspects of the conduct and reporting of this study. Any opinions, findings, conclusion, or recommendations expressed in this publication are those of the author(s) and do not necessarily reflect the view of the US Department of Agriculture.

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