Race/Ethnicity Disparities in Dysglycemia Among U.S. Women of Childbearing Age Found Mainly in the Nonoverweight/Nonobese

Jessica A. Marcinkevage, msph[1,2,3]
C.J. Alverson, ms[1]
K.M. Venkat Narayan, md[3]
Henry S. Kahn, md[4]
Julia Ruben, mph[5]
Adolfo Correa, md[6]

OBJECTIVE—To describe the burden of dysglycemia—abnormal glucose metabolism indicative of diabetes or high risk for diabetes—among U.S. women of childbearing age, focusing on differences by race/ethnicity.

RESEARCH DESIGN AND METHODS—Using U.S. National Health and Nutrition Examination Survey data (1999–2008), we calculated the burden of dysglycemia (i.e., prediabetes or diabetes from measures of fasting glucose, A1C, and self-report) in nonpregnant women of childbearing age (15–49 years) by race/ethnicity status. We estimated prevalence risk ratios (PRRs) for dysglycemia in subpopulations stratified by BMI (measured as kilograms divided by the square of height in meters), using predicted marginal estimates and adjusting for age, waist circumference, C-reactive protein, and socioeconomic factors.

RESULTS—Based on data from 7,162 nonpregnant women, representing >59,000,000 women nationwide, 19% (95% CI 17.2–20.9) had some level of dysglycemia, with higher crude prevalence among non-Hispanic blacks and Mexican Americans vs. non-Hispanic whites (26.3% [95% CI 22.3–30.8] and 23.8% [19.5–28.7] vs. 16.8% [14.4–19.6], respectively). In women with BMI <25 kg/m^2, dysglycemia prevalence was roughly twice as high in both non-Hispanic blacks and Mexican Americans vs. non-Hispanic whites. This relative increase persisted in adjusted models (PRR_{adj} 1.86 [1.16–2.98] and 2.23 [1.38–3.60] for non-Hispanic blacks and Mexican Americans, respectively). For women with BMI 25–29.99 kg/m^2, only non-Hispanic blacks showed increased prevalence vs. non-Hispanic whites (PRR_{adj} 1.55 [1.03–2.34] and 1.28 [0.73–2.26] for non-Hispanic blacks and Mexican Americans, respectively). In women with BMI >30 kg/m^2, there was no significant increase in prevalence of dysglycemia by race/ethnicity category.

CONCLUSIONS—Our findings show that dysglycemia affects a significant portion of U.S. women of childbearing age and that disparities by race/ethnicity are most prominent in the nonoverweight/nonobese.

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While national trends show that diabetes prevalence among all U.S. adults (men and women) has risen in recent years, seemingly concomitantly with rates of overweight and obesity, non-Hispanic blacks and Mexican Americans continue to be disproportionately affected, with rates almost twice those of non-Hispanic whites (1,2). This has also been the trend for impaired fasting glucose (IFG), a marker of future diabetes risk (1,2). Previous research on racial disparities of diabetes prevalence has focused on disparities for common risk factors for the disease: obesity and poverty, among others (3,4). However, findings from these studies show that there appears to be a residual effect of race/ethnicity (3,4), while controlling for the effect of BMI and social factors, with no concrete explanation as to why this might be so.

Little attention has been paid specifically to investigating factors associated with disparity in glucose levels among women in their reproductive years. However, this proves an important population to target, not only because of the woman's health needs and subsequent risk for type 2 diabetes (5), but also because of her role as a caregiver and the potential adverse consequences for her offspring if exposed to gestational hyperglycemia (6–8). We therefore conducted an analysis using U.S. national data to describe the burden of dysglycemia—diabetes, IFG, or high risk for diabetes by A1C criteria—among women of childbearing age, focusing specifically on differences by race/ethnicity. We also explored the extent to which measurements of obesity—measured by BMI and waist circumference—might modify these associations.

RESEARCH DESIGN AND METHODS

Sample population and data source

The National Health and Nutrition Examination Survey (NHANES) is an ongoing national survey conducted by the National Center for Health Statistics (NCHS) (9). It uses a complex multistage probability sample so as to represent the civilian, noninstitutionalized U.S. population. Participants of the survey complete in-home interviews followed by medical and laboratory examinations in mobile examination centers. Additionally, half of those who participate in the medical

From the [1]National Center on Birth Defects and Developmental Disabilities, Division of Birth Defects and Developmental Disabilities, Centers for Disease Control and Prevention, Atlanta, Georgia; the [2]Oak Ridge Institute for Science and Education, Oak Ridge Associated Universities, Oak Ridge, Tennessee; the [3]Program in Nutrition and Health Sciences, Graduate Division of Biological and Biomedical Sciences, Laney Graduate School, Emory University, Atlanta, Georgia; the [4]Division of Diabetes Translation, Centers for Disease Control and Prevention, Atlanta, Georgia; [5]D-Tree International, Weston, Massachusetts; and the [6]Department of Medicine, University of Mississippi Medical Center, Jackson, Mississippi.
Corresponding author: Jessica A. Marcinkevage, jmarcinkevage@cdc.gov.
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examination are asked to fast overnight for laboratory testing, comprising a nationally representative fasting subsample. Our study focuses on nonpregnant females 15–49 years of age who underwent the interview and medical and/or laboratory examinations of NHANES, combined from five survey cycles from 1999 to 2008. Pregnancy status was by self-report, confirmed with a laboratory test. For fasting measures, we included women who were part of the morning fasting session and excluded women from the fasting subsample if their fasting times were <8 h. The NCHS Research Ethics Review Board approved the surveys, and documented consent was obtained from all participants. The interview, examination, and laboratory procedures have previously been described (9).

Study variables

Demographic variables. Demographic information was collected on the basis of self-report during the in-home interviews. Race/ethnicity was categorized according to NHANES guidelines for comparing across survey cycles and included "non-Hispanic white," "non-Hispanic black," "Mexican American," and "other." We chose not to present estimates from women in the "other" category because of the small sample size for, and heterogeneity of, this group. We considered age as a continuous variable and dichotomized education attainment as having completed less than high school or having completed high school (or the equivalent) or more. We categorized civil status as single or married/cohabitating and the number of live births to women as 0, 1, 2, 3, or ≥ 4. The poverty-to-income ratio (PIR)—measuring the ratio of family income to the family's appropriate poverty threshold—was computed by the NCHS from the poverty threshold for the relevant calendar year, family income, and other family data provided by the respondents to measure income status (9). We present PIR classified into three categories, as suggested by Healthy People 2010 (10): PIR <1 (poor), PIR ≥1 but <2 (near poor), and PIR ≥2 (middle or high income).

Outcome variables. We defined dysglycemia as any abnormality in glucose metabolism, indicative of diabetes or high risk for diabetes (IFG or elevated A1C). Specifically, a participant was identified as having some measure of dysglycemia if she met any one of the following criteria: *1*) During the in-home interview, she responded affirmatively to the question of whether, outside of pregnancy, a doctor or other health care professional had ever told her that she had diabetes; she reported taking insulin; or she reported taking diabetes medicines. *2*) Results from her clinical examination indicated diabetes by either an FPG value ≥126 mg/dL or A1C ≥6.5% (48 mmol/mol) (11). Or *3*) results from her clinical examinations indicated a high risk for diabetes by either a FPG value between 100 and 126 mg/dL or A1C value of 5.7–6.4% (39–46 mmol/mol) (11).

Details about collection and processing of blood samples can be found in documentation on the NHANES website (9). Briefly, FPG was measured using a hexokinase enzymatic method, with a coefficient of variation of 1.3–2.2%. To account for changes to the laboratory and equipment used for measurement of glucose in 2005–2008 versus those used for 1999–2004, we converted values from 2005 to 2008 via a linear transformation to make them comparable with values from 1999 to 2004 (9). A1C was measured using whole blood at a central laboratory by a high-performance liquid chromatographic assay and standardized according to the method of the Diabetes Control and Complications Trial (12), with a coefficient of variation of 1.0–1.7% (9). We used only A1C values from 1999 to 2006 for this analysis to avoid any bias that might be introduced by the inexplicable trending higher values from 2007 to 2008 (9).

Other cardiometabolic factors and covariates. Height and weight were measured in the mobile examination centers (9), and BMI was calculated as weight in kilograms divided by the square of the height in meters. We categorized BMI according to World Health Organization definitions (13) and grouped these into three categories: under- or normal weight (BMI <25.0 kg/m^2), overweight (25–29.99 kg/m^2), and obese or morbidly obese (≥30 kg/m^2). To consider the adverse metabolic effects and increased cardiovascular mortality resulting from greater abdominal adiposity (14,15), we assessed waist circumference as an independent risk factor. High central adiposity was considered as waist circumference ≥88 cm (16). To account for low-grade inflammation and its potential association with diabetes development (16), we categorized levels of C-reactive protein (CRP), a proinflammatory marker measured in NHANES participants, with the cut point at 0.3 mg/dL or higher (16). CRP concentrations were measured by latex-enhanced nephelometry on a Behring Nephelometer (Siemans Healthcare Diagnostics, Deerfield, IL).

Statistical analysis

Women were considered eligible for the analysis if they attended the medical exam and had complete information for race/ethnicity status, pregnancy status, age, education attainment, and PIR. Statistical analyses were performed using SAS-callable SUDAAN, version 9.2 (SAS Institute, Cary, NC). The five survey cycles (1999–2000, 2001–2002, 2003–2004, 2005–2006, and 2007–2008) were merged into one dataset, and 10-year sampling weights were calculated based on guidelines recommended by NCHS for analyses that combine two or more survey cycles (9). For those variables not meeting normality assumptions (i.e., number of live births) the categorical variable was used for analyses. All analyses incorporated the correct sample weights for the subsample and complex survey design. We calculated mean levels (95% CI) for continuous variables and prevalence estimates (95% CI) for categorical variables. SEs were estimated using the Taylor series linearization method. Estimates were considered reliable if degrees of freedom were ≥12 and the relative SE ≤30% (9). We used fitted multiple logistic regression models to estimate crude and adjusted odds ratios (ORs) and prevalence risk ratios (PRRs) with accompanying 95% CIs for our outcome variable dysglycemia (17,18). Our calculation of the PRR was performed as a function of the average marginal predictions from the fitted regression models (17). We considered the following variables for confounding, based on previous literature: age, PIR, education, number of live births, waist circumference, and CRP. We elected to keep the covariates stated above in the model if they changed the full model OR by ≥10% (19). Our final models therefore adjusted for age, education, PIR, waist circumference, and CRP. We assessed model fit using the Hosmer-Lemeshow goodness of fit, Satterthwaite-adjusted *F* test (20). Correlation among independent variables was assessed via the variance inflation factor and condition indices, using accepted criteria (20); we found no presence of multicollinearity. We assessed effect modification by using fully adjusted models containing all relevant two-way interaction terms; because the interaction between race/ethnicity and BMI was significant in our models ($P < 0.05$), we present our findings stratified on BMI category. To assess possible sample

bias from the limited data of A1C, we conducted a sensitivity analysis restricted to data from 1999–2006 and arrived at the same conclusions as for the full dataset (1999–2008); therefore, we presented our findings based on the full 1999–2008 dataset.

RESULTS—After exclusion of women with inadequate fasting times (n = 421), our final sample totaled 7,162 women, with 2,950 eligible for the fasting analyses. Women who were excluded from the analysis because of invalid fasting times did not differ from those included in the analysis in race/ethnicity category, PIR, parity, BMI, waist circumference, or CRP values. We did notice that women who were excluded were slightly younger than women included in the analysis ($P < 0.05$) by ~2 years (mean [SD] age for included vs. excluded women, respectively: 29.8 [10.6] vs. 27.8 [10.6]). Demographic characteristics of the study population are provided in Table 1. The mean age of our population was 33 years, with Mexican American women slightly younger than the total population and both non-Hispanic white and non-Hispanic black women. Most women surveyed in the total population were married or cohabitating, with at least a high school education, and had had at least one live birth. Compared with non-Hispanic whites, both non-Hispanic blacks and Mexican Americans were more likely to be near poor or below the poverty line (55.4 and 65.9 vs. 30.2%, respectively) and less likely to have attained a high school degree or greater (71.0 and 50.7 vs. 84.6%, respectively; $P < 0.01$ for each comparison).

More than 50% of U.S. women of childbearing age were overweight, obese, or morbidly obese. Prevalence of obesity was significantly higher in non-Hispanic black and Mexican American women compared with non-Hispanic white women (prevalence estimates: 47.0% [95% CI 44.6–49.5], 36.3% [33.1–39.6], and 28.0% [25.8–30.3] for non-Hispanic black, Mexican American, and non-Hispanic white women, respectively; $P < 0.01$ for each comparison) (Fig. 1). Additionally, high central adiposity (waist circumference ≥88 cm) affected almost 50% of all women in this population, with proportions reaching 58% or greater for both non-Hispanic blacks and Mexican

Table 1—*Population statistics for nonpregnant U.S. women of childbearing age: NHANES 1999–2008*

	n‡	Total population ($n_{weighted}$ = 59,465,044)	Non-Hispanic white ($n_{weighted}$ = 39,758,373)	Non-Hispanic black ($n_{weighted}$ = 7,853,867)	Mexican American ($n_{weighted}$ = 5,109,137)
Social factors					
Age (years)†	7,162	33.1 (32.7–33.4)	33.5 (33.1–34.9)	32.6 (32.1–33.2)[a]	31.3 (30.8–31.8)[b]
Education level	7,162				
<High school	2,711	21.2 (19.8–22.7)	15.4 (13.7–17.2)	29.0 (25.8–32.5)[b]	49. 4 (45.5–53.2)[b]
≥High school	4,451	78.8 (77.3–80.2)	84.6 (82.8–86.3)	71.0 (67.6–74.2)[b]	50.7 (46.8–54.5)[b]
Civil status	7,069				
Married/cohabitating	3,132	56.9 (55.1–58.6)	61.8 (59.8–63.8)	34.8 (32.0–37.7)[b]	61.1 (58.3–63.9)
PIR†	7,162	2.8 (2.7–2.9)	3.1 (3.0–3.2)	2.1 (2.0–2.2)[b]	1.9 (1.8–2.0)[b]
PIR <1, below poverty line	1,881	18.2 (16.7–19.8)	13.0 (11.1–15.1)	29.1 (25.7–32.7)[b]	33.0 (29.8–36.4)[b]
1 ≤ PIR < 2, near poor	1,797	20.7 (19.5–22.0)	17.2 (15.4–19.0)	26.3 (23.4–29.4)[b]	32.9 (30.3–35.6)[b]
PIR ≥2, middle or high income	3,484	61.1 (59.1–63.1)	69.8 (67.0–72.5)	44.6 (41.3–48.0)[b]	34.2 (31.2–37.0)[b]
Number of live births†	3,822	2.1 (2.1–2.2)	2.0 (2.0–2.1)	2.3 (2.2–2.4)[b]	2.5 (2.4–2.6)[b]
0	245	6.5 (6.1–7.8)	6.6 (6.1–8.4)	7.7 (7.0–10.2)*	3.0 (2.5–4.7)*,[b]
1	1,005	25.0 (23.3–26.8)	25.7 (23.2–28.3)	23.3 (20.8–25.9)	23.2 (19.9–26.8)
2	1,211	36.0 (34.1–37.8)	37.9 (35.4–40.4)	32.7 (29.5–36.0)[a]	27.3 (23.9–31.1)[b]
3	854	22.3 (20.8–23.9)	22.2 (20.2–24.3)	20.8 (17.9–23.9)	27.5 (25.0–30.3)[b]
≥4	507	10.2 (9.0–11.6)	7.6 (6.1–9.4)	15.6 (13.3–18.3)[b]	19.0 (16.1–22.2)[b]
Cardiometabolic factors					
BMI (kg/m^2)†	7,027	27.6 (27.3–27.9)	27.0 (26.6–27.5)	30.7 (30.3–31.2)[b]	28.7 (28.2–29.1)[b]
BMI <25 kg/m^2 (under- or normal weight)	3,065	44.6 (42.5–46.7)	48.9 (45.9–52.0)	26.4 (24.3–28.7)[b]	32.8 (30.1–35.7)[b]
25 ≤ BMI <30 kg/m^2 (overweight)	1,747	24.6 (23.2–26.2)	23.1 (21.3–24.9)	26.6 (24.3–29.0)	30.9 (28.7–33.2)[b]
BMI ≥30 kg/m^2 (obese or morbidly obese)	2,215	30.8 (29.1–32.6)	28.0 (25.8–30.3)	47.0 (44.6–49.5)[b]	36.3 (33.1–39.6)[b]
Waist circumference (cm)†	6,900	90.5 (89.8–91.2)	89.7 (88.7–90.7)	95.8 (94.8–96.7)[b]	92.7 (91.8–93.6)[b]
≥88 cm (high)	3,331	48.8 (46.7–50.9)	46.1 (43.3–48.9)	62.4 (60.1–64.6)[b]	58.4 (55.0–61.8)[b]
CRP (mg/dL)†	6,696	0.4 (0.4–0.5)	0.4 (0.4–0.4)	0.6 (0.5–0.6)[b]	0.5 (0.5–0.6)[b]
>0.3 (elevated)	2,435	37.1 (35.5–38.8)	35.4 (33.1–37.7)	44.6 (41.9–47.4)[b]	44.5 (41.1–47.9)[b]
FPG (mg/dL)†	2,950	93.3 (92.5–94.1)	92.1 (91.1–93.1)	95.2 (92.6–97.8)[a]	97.1 (94.8–99.5)[b]
A1C (%)†	5,764	5.2 (5.2–5.3)	5.2 (5.1–5.2)	5.5 (5.4–5.5)[b]	5.4 (5.3–5.4)[b]
Any level of dysglycemia present^	2,954	19.0 (17.2–20.9)	16.8 (14.4–19.6)	26.3 (22.3–30.8)[b]	23.8 (19.5–28.7)[b]

Data are percent (95% CI) unless otherwise indicated. ‡Unweighted n. †Mean (95% CI). [a]P value < 0.05 compared with non-Hispanic whites. [b]P value < 0.01 compared with non-Hispanic whites. *<12 df and relative SE >30%; presenting adjusted CI (9). ^Self-report of diabetes or taking diabetes medicines from the interview, FPG ≥100 mg/dL, or A1C ≥5.7% (39 mmol/mol) from laboratory measures.

Americans compared with 46% for non-Hispanic whites (Table 1).

We observed higher FPG, A1C, and CRP levels among non-Hispanic blacks and Mexican Americans compared with non-Hispanic whites (Table 1). Almost 20% of all nonpregnant U.S. women of childbearing age had some measure of dysglycemia. Higher proportions of dysglycemia were seen among minority groups compared with non-Hispanic whites: 26.3% (95% CI 22.3–30.8) in non-Hispanic blacks and 23.8% (19.5–28.7) in Mexican Americans vs. 16.8% (14.4–19.6) in non-Hispanic whites.

In stratified analyses (Table 2 and Supplementary Table 1), within normal to underweight women, we saw higher prevalence of dysglycemia among both non-Hispanic black and Mexican American women compared with non-Hispanic white women. For this BMI category, in our unadjusted models, prevalence of dysglycemia was twice as high in both non-Hispanic blacks and Mexican Americans compared with non-Hispanic whites. These observations held when models were adjusted for age, socioeconomic factors, waist circumference, and CRP levels, though the estimates were slightly attenuated (PRR_{adj} 1.86 [95% CI 1.16–2.98] for non-Hispanic blacks and 2.23 [1.38–3.60] for Mexican Americans) (Table 2). Lower education attainment was also significantly associated with higher prevalence of dysglycemia within this BMI category, as was having a waist circumference ≥88 cm. Among overweight women, we observed that non-Hispanic blacks had ~1.5 times the prevalence of dysglycemia compared with non-Hispanic whites; this disparity persisted after adjusting for age, socioeconomic factors, waist circumference, and CRP (PRR_{adj} 1.55 [1.03–2.34]), with the same effect for both lower education attainment and waist circumference observed as seen in the normal and underweight women. We found no significant differences in dysglycemia prevalence between overweight Mexican American women and overweight non-Hispanic white women. Additionally, among obese and morbidly obese women, we did not observe any differences in dysglycemia by race/ethnicity status in either crude or adjusted models (Fig. 2).

CONCLUSIONS—Using nationally representative data collected over 10 years and representing >50 million women, we found that almost 1 in 5 U.S. women of childbearing age were affected by some form of dysglycemia. Additionally, more than one-half of all U.S. women of childbearing age were overweight or obese, and nearly 50% had high central adiposity. We also observed the disproportionate burden of dysglycemia among racial and ethnic minorities compared with non-Hispanic whites, with prevalence estimates in minorities ~1.5 times those in non-Hispanic whites. When stratified by BMI category, we continued to see disparities in dysglycemia prevalence by race/ethnicity status, though this was restricted to distinct BMI categories. Within the normal-to-underweight group, both non-Hispanic blacks and Mexican Americans had almost twice the prevalence of dysglycemia versus non-Hispanic whites. In the overweight group, only non-Hispanic blacks had increased prevalence, at almost 1.5 times that of non-Hispanic whites.

Hyperglycemia among women of childbearing age poses a risk not only to the woman as she progresses through various life stages (5) but also to her fetus if she becomes pregnant (6–8). These effects could impact the long-term health of her child, including increased risk of obesity and type 2 diabetes later in life (21,22). However, little attention has been paid to diabetes and measures of dysglycemia specifically among nonpregnant women of childbearing age. Most estimates from U.S. data for this particular population subgroup are derived from analyses using broad age ranges, as well as pregnant and nonpregnant women. Previously reported prevalence estimates range between 3 and 7.5% for self-reported diabetes and 8 and 23% for clinical measures of IFG (1,2), with higher estimates for both measures in minority groups and higher age categories. Our results for overall dysglycemia (which includes diabetes and prediabetes measures, including IFG) are comparable with these estimates, despite our younger cohort. This is of note, since diabetes and dysglycemia in general increase with age (23), and highlights the importance of focusing interventions on this younger age-group.

Research focusing specifically on nondiabetic women of childbearing age shows disproportionate levels of obesity and other clinical characteristics of the metabolic syndrome, including IFG, among both non-Hispanic blacks and Hispanics compared with non-Hispanic whites (24). Since these data excluded diabetic women, our study is the first to investigate diabetes and prediabetes measures specifically among women of childbearing age. Other findings from national data have shown variations in diabetes prevalence by BMI group. A recent study (25) looking at 30 years of NHANES data shows variation in racial/ethnic disparities of diabetes by BMI group: in normal and overweight individuals, minority groups experience a greater increase in diabetes prevalence than whites over the time period studied, but in obese and severely obese groups this disparity is less pronounced. Though this recent study includes a larger

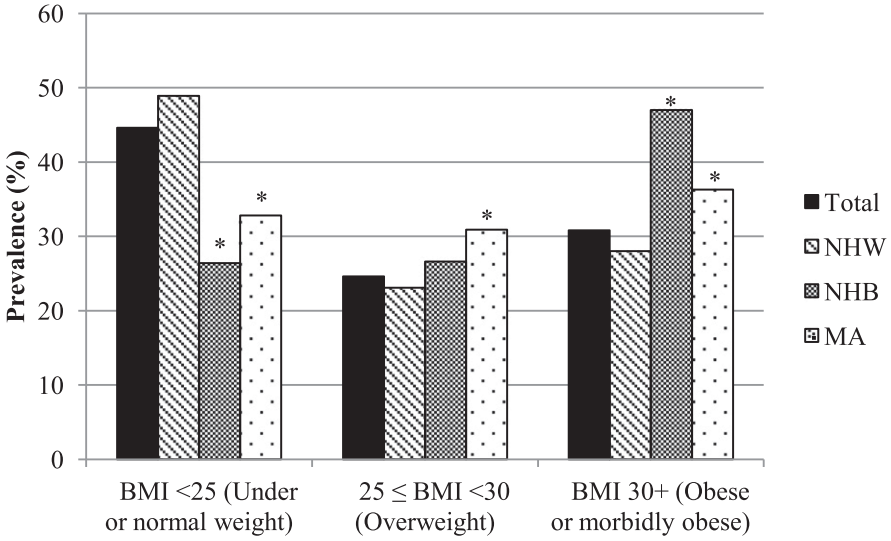

Figure 1—*Prevalence of under/normal weight, overweight, and obese (by BMI cutoffs) among nonpregnant U.S. women of childbearing age (15–49 years) for total population and by race/ethnicity, NHANES 1999–2008. *P ≤ 0.05 vs. non-Hispanic white. MA, Mexican American; NHB, non-Hispanic black; NHW, non-Hispanic white.*

Table 2—*Crude PRRs and PRR_{adj} (95% CI) for dysglycemia in nonpregnant U.S. women of childbearing age (15–49 years) by BMI category[2]: NHANES 1999–2008*

	Under- or normal-weight BMI (n = 1,118; $n_{weighted}$ = 23,213,172)		Overweight BMI (n = 640; $n_{weighted}$ = 12,462,018)		Obese or morbidly obese (n = 830; $n_{weighted}$ = 16,222,072)	
	Crude model	Adjusted model[1]	Crude model	Adjusted model[1]	Crude model	Adjusted model[1]
Race/ethnicity						
Non-Hispanic white	Reference category	Reference category	Reference category	Reference category	Reference category	Reference category
Non-Hispanic black	1.81 (1.09–3.01)	1.86 (1.16–2.98)	1.54 (1.06–2.24)	1.55 (1.03–2.34)	0.99 (0.75–1.30)	1.06 (0.81–1.39)
Mexican American	2.12 (1.37–3.30)	2.23 (1.38–3.60)	1.41 (0.87–2.29)	1.28 (0.73–2.26)	0.94 (0.72–1.23)	0.99 (0.74–1.32)
Education						
≥High school		Reference category		Reference category		Reference category
<High school		2.24 (1.34–3.77)		1.71 (1.04–2.81)		1.09 (0.87–1.36)
Waist circumference (cm)						
≤88		Reference category		Reference category		Reference category
>88		2.30 (1.21–4.38)		1.89 (1.00–3.57)		1.21 (0.30–4.80)
PIR						
≥Middle class		Reference category		Reference category		Reference category
Near poor		0.76 (0.44–1.33)		1.20 (0.67–2.14)		1.00 (0.75–1.33)
Below poverty line		0.78 (0.42–1.46)		0.69 (0.30–1.63)		1.21 (0.95–1.59)
CRP (mg/dL)						
≤0.3		Reference category		Reference category		Reference category
>0.3		1.30 (0.78–2.15)		0.94 (0.60–1.48)		1.41 (1.02–1.96)

[1]Adjusted for age (continuous), waist circumference (<88 or ≥88 cm), education (<high school or ≥high school), PIR (<1, poor; ≥1 but <2, near poor; or ≥2, middle or high income), and CRP (≤0.3 or >0.3 mg/dL). [2]Normal and underweight: BMI <25.0 kg/m^2, overweight: BMI 25–29.99 kg/m^2, and obese or morbidly obese: BMI ≥30 kg/m^2.

age-group (20–74 years old) as well as men and women together, it corroborates some of our observations presented here.

Diabetes has been shown to be associated with obesity (26,27). However, in our results we see discordance in dysglycemia and obesity, with differences by race/ethnicity among women not considered obese by standard clinical measures. Others before us have noted differential effects of BMI on diabetes risk between black and white Americans (4). This highlights the fact that obesity, as measured by either BMI or waist circumference, does not explain all disparities by race/ethnicity in impaired glucose metabolism in a clinical setting or at a population level. One possible explanation for the disparity within nonobese subjects is a differential β-cell function between race/ethnicity groups. Results from clinical studies have shown decreased insulin sensitivity in African American women compared with European American women (28,29), with differential responses by race/ethnicity in insulin sensitivity and β-cell responsiveness according to level of body fat (30) and location of body fat depots (31,32). Although obesity alone causes a state of insulin resistance, it is possible that the pancreatic response is different in the presence of adipose tissue within different race/ethnicity groups, which may help to explain our observation by BMI category.

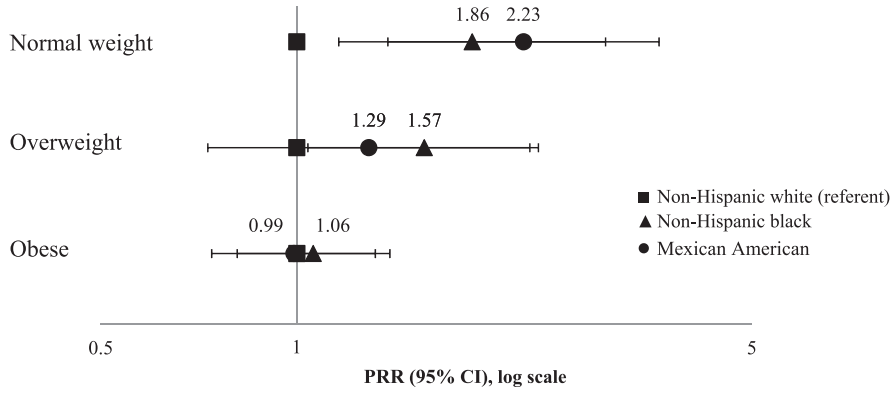

Figure 2—*PRR_{adj} (95% CI) for dysglycemia in different race/ethnicities in nonpregnant U.S. women of childbearing age, stratified by BMI category. Adjusted for age (continuous), waist circumference (<88 or ≥88 cm), education (<high school or ≥high school), PIR (<1, poor; ≥1 but <2, near poor; or ≥2, middle or high income), and CRP (≤0.3 or >0.3 mg/dL). Under or normal weight: BMI <25.0 kg/m^2 (n = 1,118; $n_{weighted}$ = 23,213,172); overweight: BMI 25–29.99 kg/m^2 (n = 640; $n_{weighted}$ = 12,462,018), and obese or morbidly obese: BMI ≥30 kg/m^2 (n = 830; $n_{weighted}$ = 16,222,072).*

We focused our attention on variations within BMI categories. However, BMI has been criticized as a crude measure for obesity, since it does not discriminate between lean muscle and body fat and therefore might not account for individuals with normal-weight obesity (i.e., normal BMI but high body fat). Clinical studies among women with a normal BMI showed that as body fat increased, so did prevalence of metabolic syndrome and dyslipidemia (33). Furthermore,

among those women with normal-weight obesity, insulin sensitivity was significantly decreased. Additionally, the researchers found an increased risk of cardiovascular mortality among these women, suggesting that classifying a person as "normal" based on BMI alone might mask the effects of that person's body fat content. However, the body fat measures used in these clinical studies did not account for the distribution of fat—specifically, the location of the fat stores. Although visceral adipose has been linked to increased diabetes incidence (14), clinical studies have shown that African American women have less visceral adipose stores compared with European American women, even after periods of significant weight gain and loss (34,35). We attempted to account for the increased risk posed by central adiposity by adjusting for waist circumference category using a clinically relevant measure and still noticed differences in dysglycemia prevalence by race/ethnicity among women with a normal BMI. The use of these clinical indices alone may not identify some women at increased risk for impaired glucose tolerance.

Differences in dysglycemia by race/ethnicity may also be due to factors unrelated to glucose control and could be a result of genetic or ancestral differences, particularly related to A1C variation. Several epidemiological studies have reported higher A1C values in African Americans vs. whites, independent of FPG levels (36,37). Results from the Diabetes Prevention Program show that among individuals with impaired glucose tolerance, both blacks and Hispanics had higher A1C levels than whites, even after adjusting for cofactors such as age, sex, education, BMI, blood pressure, and insulin resistance (38). Previous research estimates that genetic factors might explain >50% of variation in A1C (39) and supports the exploration for a genetic loci unique to A1C. However, recent research from the Atherosclerosis Risk on Communities (ARIC) study shows a small contribution of genetic ancestry relative to social and metabolic factors in explaining A1C variation among African Americans, indicating that ancestral genetic differences might not explain significantly the observed race/ethnicity differences in A1C (40). More research is warranted to investigate the role of genetic factors in these specific associations.

While our analysis uses robust, nationally representative survey data, there are some limitations to our study. NHANES data are based on a cross-sectional survey; consequently, there is no way to assess causality. Also, because of possible disclosure risks, for the 2007–2008 survey cycle only pregnancy status information for women aged 20–44 years was available. We therefore may have missed some nonpregnant women aged 15–19 and 45–49 years in the survey. We also restricted our analyses to only three race/ethnicity categories; small sample size and wide heterogeneity of a fourth category ("other") did not allow for reliable comparison with the other defined categories for race/ethnicity. Additionally, fasting measures are based on one FPG value, and fasting state is based on the participants' self-report. For clinical diagnoses, it is recommended that the subject be retested in the presence of an abnormal result; we did not have this opportunity. Therefore, it is likely that some prevalence estimates from the use of FPG values might be overestimated. Also, diagnosed diabetes is by self-report; however, we are able to use laboratory values for diabetes measures to help eliminate any biases of self-report. Finally, we are missing values of A1C from the 2007–2008 NHANES cycle. NCHS released a statement in March 2012 noting an increase in the proportion of A1C values between 5.7 and 6.4% (39–48 mmol/mol) and subsequent shift to the right (increased values) of A1C distribution in NHANES 2007–2010 compared with 1999–2006. However, after extensive investigation, the specific source for this observation is currently unknown (9). Since our analyses are focused on those persons with higher A1C—particularly, 5.7% (39 mmol/mol) and above—inclusion of these data from 2007–2008 may have biased our results; therefore, we chose not to include these data in our analyses. Because of this, some individuals may have been misclassified on their status of dysglycemia. To test how this might affect our results and subsequent conclusions from our findings, we conducted a sensitivity analysis using only data from 1999–2006. We observed no significant changes to our conclusions using this data subset and therefore presented results from the full dataset, 1999–2008. A breakdown of our outcome showed that 8.3% of our population were categorized as having dysglycemia by A1C criteria, 16.2% by FPG criteria, and 2.7% by interview response (i.e., with diagnosed diabetes). Within the whole study sample, 3.0% were categorized as having dysglycemia by meeting both A1C and FPG criteria, 1.3% by A1C criteria alone, and 8.4% by FPG criteria alone. However, the fact that we were able to include both laboratory measures for glucose and A1C, as well as a self-report of doctor-diagnosed diabetes, adds to the robustness of our study.

In summary, we found that approximately one in five of the nation's nonpregnant women of childbearing age is affected by some form of dysglycemia. This corresponds to almost 9 million U.S. women between the ages of 15 and 49 years, with a greater burden among minorities compared with non-Hispanic whites. While our findings confirm the presence of disparities in dysglycemia prevalence by race/ethnicity, contrary to previous literature we find this difference is explained not by obesity but, rather, by differences within normal to underweight groups. These findings suggest that special attention should be paid specifically to the disparities among nonobese individuals both in clinical practices and in development of public health programs and interventions.

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J.A.M. assisted with project conception, conducted the statistical analysis, reviewed the data and results, and wrote the manuscript. C.J.A. researched the data, led the statistical analysis, provided statistical consult, and reviewed and edited the manuscript. K.M.V.N. reviewed the results, contributed to the discussion, and reviewed and edited the manuscript. H.S.K. contributed to the data analysis, reviewed the results, contributed to the discussion, and reviewed and edited the manuscript. J.R. researched data, contributed to the discussion, and reviewed and edited the manuscript. A.C. assisted with project conception, reviewed the results, contributed to the discussion, and reviewed and edited the manuscript. A.C. is the guarantor of this work and, as such, had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis.

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References

1. Cowie CC, Rust KF, Byrd-Holt DD, et al. Prevalence of diabetes and impaired fasting glucose in adults in the U.S. population: National Health And Nutrition Examination Survey 1999-2002. Diabetes Care 2006; 29:1263–1268
2. Cowie CC, Rust KF, Byrd-Holt DD, et al. Prevalence of diabetes and high risk for diabetes using A1C criteria in the U.S. population in 1988-2006. Diabetes Care 2010;33:562–568
3. Link CL, McKinlay JB, Robbins JM, Vaccarino V, Zhang H, Kasl SV. Disparities in the prevalence of diabetes: is it race/ethnicity or socioeconomic status? Results from the Boston Area Community Health (BACH) survey. Ethn Dis 2009;19: 288–292
4. Resnick HE, Valsania P, Halter JB, Lin X, Black SA. Differential effects of BMI on diabetes risk among black and white Americans. Diabetes Care 1998;21:1828–1835
5. Owens MD, Beckles GL, Ho KK, Gorrell P, Brady J, Kaftarian JS. Women with diagnosed diabetes across the life stages: underuse of recommended preventive care services. J Womens Health (Larchmt) 2008; 17:1415–1423
6. Correa A, Gilboa SM, Besser LM, et al. Diabetes mellitus and birth defects. Am J Obstet Gynecol 2008;199:237.e1–237.e9.
7. Metzger BE, Lowe LP, Dyer AR, et al.; HAPO Study Cooperative Research Group. Hyperglycemia and adverse pregnancy outcomes. N Engl J Med 2008;358:1991–2002
8. Bo S, Menato G, Signorile A, et al. Obesity or diabetes: what is worse for the mother and for the baby? Diabetes Metab 2003; 29:175–178
9. *National Health and Nutrition Examination Survey 1999–2008*. Centers for Disease Control and Prevention, National Center for Health Statistics, Hyattsville, MD, 2011
10. U.S. Department of Health and Human Services (HHS). *Healthy People 2010: Understanding and Improving Health*. 2nd ed. Washington, D.C., U.S. Government Printing Office, 2000
11. American Diabetes Association. Executive summary: Standards of medical care in diabetes—2012. Diabetes Care 2012;35 (Suppl. 1):S4–S10
12. Little RR, Wiedmeyer HM, England JD, et al. Interlaboratory standardization of measurements of glycohemoglobins. Clin Chem 1992;38:2472–2478
13. World Health Organization. *Obesity: Preventing and Managing the Global Epidemic. Report of a WHO consultation*. Geneva, World Health Org., 2000 (Tech. Rep. Ser., 2000;894:i–xii, 1–253)
14. Langenberg C, Sharp SJ, Schulze MB, et al.; InterAct Consortium. Long-term risk of incident type 2 diabetes and measures of overall and regional obesity: the EPIC-InterAct case-cohort study. PLoS Med 2012;9:e1001230
15. Klein S, Allison DB, Heymsfield SB, et al.; Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; American Society for Nutrition; American Diabetes Association. Waist circumference and cardiometabolic risk: a consensus statement from Shaping America's Health: Association for Weight Management and Obesity Prevention; NAASO, The Obesity Society; the American Society for Nutrition; and the American Diabetes Association. Am J Clin Nutr 2007; 85:1197–1202
16. Grundy SM, Cleeman JI, Daniels SR, et al. Diagnosis and Management of the Metabolic Syndrome: An American Heart Association/National Heart, Lung, and Blood Institute Scientific Statement: Executive Summary. Circulation 2005;112:e285–e290
17. Graubard BI, Korn EL. Predictive margins with survey data. Biometrics 1999;55:652–659
18. Bieler GS, Brown GG, Williams RL, Brogan DJ. Estimating model-adjusted risks, risk differences, and risk ratios from complex survey data. Am J Epidemiol 2010;171: 618–623
19. Kleinbaum DG, Klein M. *Logistic Regression: A Self-Learning Text*. 2nd ed. New York, NY, Springer-Verlag, 2002
20. Regression with SAS [article online], 2003. Available from http://www.ats.ucla.edu/stat/sas/webbooks/reg/default.htm. Accessed 5 February 2013
21. Dabelea D, Pettitt DJ. Intrauterine diabetic environment confers risks for type 2 diabetes mellitus and obesity in the offspring, in addition to genetic susceptibility. J Pediatr Endocrinol Metab 2001;14:1085–1091
22. Pettitt DJ, Knowler WC. Long-term effects of the intrauterine environment, birth weight, and breast-feeding in Pima Indians. Diabetes Care 1998;21(Suppl. 2):B138–B141
23. Cowie CC, Harris MI, Silverman RE, Johnson EW, Rust KF. Effect of multiple risk factors on differences between blacks and whites in the prevalence of non-insulin-dependent diabetes mellitus in the United States. Am J Epidemiol 1993;137:719–732
24. Ramos RG, Olden K. The prevalence of metabolic syndrome among US women of childbearing age. Am J Public Health 2008;98:1122–1127
25. Zhang Q, Wang Y, Huang ES. Changes in racial/ethnic disparities in the prevalence of Type 2 diabetes by obesity level among US adults. Ethn Health 2009;14:439–457
26. Ford ES, Williamson DF, Liu S. Weight change and diabetes incidence: findings from a national cohort of US adults. Am J Epidemiol 1997;146:214–222
27. Mokdad AH, Bowman BA, Ford ES, Vinicor F, Marks JS, Koplan JP. The continuing epidemics of obesity and diabetes in the United States. JAMA 2001;286:1195–1200
28. Chandler-Laney PC, Phadke RP, Granger WM, et al. Age-related changes in insulin sensitivity and β-cell function among European-American and African-American women. Obesity (Silver Spring) 2011; 19:528–535
29. Hyatt TC, Phadke RP, Hunter GR, Bush NC, Muñoz AJ, Gower BA. Insulin sensitivity in African-American and white women: association with inflammation. Obesity (Silver Spring) 2009;17:276–282
30. Chandler-Laney PC, Phadke RP, Granger WM, et al. Adiposity and β-cell function: relationships differ with ethnicity and age. Obesity (Silver Spring) 2010;18:2086–2092
31. Ingram KH, Lara-Castro C, Gower BA, et al. Intramyocellular lipid and insulin resistance: differential relationships in European and African Americans. Obesity (Silver Spring) 2011;19:1469–1475
32. Lawrence JC, Newcomer BR, Buchthal SD, et al. Relationship of intramyocellular lipid to insulin sensitivity may differ with ethnicity in healthy girls and women. Obesity (Silver Spring) 2011;19:43–48
33. Romero-Corral A, Somers VK, Sierra-Johnson J, et al. Normal weight obesity: a risk factor for cardiometabolic dysregulation and cardiovascular mortality. Eur Heart J 2010;31:737–746
34. Lara-Castro C, Weinsier RL, Hunter GR, Desmond R. Visceral adipose tissue in women: longitudinal study of the effects of fat gain, time, and race. Obes Res 2002; 10:868–874
35. Weinsier RL, Hunter GR, Gower BA, Schutz Y, Darnell BE, Zuckerman PA. Body fat distribution in white and black women: different patterns of intraabdominal and subcutaneous abdominal adipose tissue utilization with weight loss. Am J Clin Nutr 2001;74:631–636
36. Bleyer AJ, Hire D, Russell GB, et al. Ethnic variation in the correlation between random serum glucose concentration and glycated haemoglobin. Diabet Med 2009;26:128–133
37. Ziemer DC, Kolm P, Weintraub WS, et al. Glucose-independent, black-white differences in hemoglobin A1c levels: a cross-sectional analysis of 2 studies. Ann Intern Med 2010; 152:770–777
38. Herman WH, Ma Y, Uwaifo G, et al.; Diabetes Prevention Program Research Group. Differences in A1C by race and ethnicity among patients with impaired glucose tolerance in the Diabetes Prevention Program. Diabetes Care 2007;30:2453–2457
39. Snieder H, Sawtell PA, Ross L, Walker J, Spector TD, Leslie RD. HbA(1c) levels are genetically determined even in type 1 diabetes: evidence from healthy and diabetic twins. Diabetes 2001;50:2858–2863
40. Maruthur NM, Kao WH, Clark JM, et al. Does genetic ancestry explain higher values of glycated hemoglobin in African Americans? Diabetes 2011;60:2434–2438