Secular and race/ethnic trends in glycemic outcomes by BMI in US adults: The role of waist circumference

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Abstract

Background For the same body mass index (BMI) level, waist circumference (WC) is higher in more recent years. How this impacts diabetes and prediabetes prevalence in the United States and for different race/ethnic groups is unknown. We examined prevalence differences in diabetes and prediabetes by BMI over time, investigated whether estimates were attenuated after adjusting for waist circumference, and evaluated implications of these patterns on race/ethnic disparities in glycemic outcomes.

Methods Data came from 12 614 participants aged 20 to 74 years from the National Health and Nutrition Examination Surveys (1988-1994 and 2007-2012). We estimated prevalence differences in diabetes and prediabetes by BMI over time in multivariable models. Relevant interactions evaluated race/ethnic differences.

Results Among normal, overweight, and class I obese individuals, there were no significant differences in diabetes prevalence over time. However, among individuals with class II/III obesity, diabetes prevalence rose 7.6 percentage points in 2007-2012 vs 1988-1994. This estimate was partly attenuated after adjustment for mean waist circumference but not mean BMI. For prediabetes, prevalence was 10 to 13 percentage points higher over time at lower BMI values, with minimal attenuation after adjustment for WC. All patterns held within race/ethnic groups. Diabetes disparities among blacks and Mexican Americans relative to whites remained in both periods, regardless of BMI, and persisted after adjustment for WC.

Conclusions Diabetes prevalence rose over time among individuals with class II/III obesity and may be partly due to increasing waist circumference. Anthropometric measures did not appear to account for temporal increases in prediabetes, nor did they attenuate race/ethnic disparities in diabetes. Reasons underlying these trends require further investigation.

KEYWORDS

BMI, diabetes, race/ethnicity, trends, waist circumference

1 | INTRODUCTION

The prevalence of adult obesity remains high in the United States, but recent reports suggest that it may be leveling off.[1] Despite this plateau, waist circumference (WC) has increased over a similar timeframe to a greater extent than what would be expected given changes in body mass index (BMI).[2–6] Moreover, at the same BMI level, WC is higher in more recent years particularly at the upper end of the BMI distribution.[3,6] This pattern extends across race/ethnic groups but is especially predominant in Mexican Americans and non-Hispanic whites.[6] These findings suggest that body shape may be changing even when obesity as determined by BMI has plateaued.

One potential consequence of this pattern may be a concomitant rise in diabetes and prediabetes for the same BMI level, especially across parts of the BMI distribution where WC has been increasing over time. Recent reports have drawn attention to the alarming rise in diabetes and prediabetes in the United States, much of which has been attributable to the obesity epidemic.[7–9] However, it is unknown if diabetes and prediabetes have also been increasing at the same BMI level, if dysglycemia is higher over time only at certain parts of the BMI distribution, and if this pattern may be attributable to temporal

increases in WC. Evaluating whether increasing WC, independent of BMI, plays a role in diabetes and prediabetes trends can improve our understanding of the reasons underlying the growing diabetes epidemic in the United States.

Although dysglycemia has been shown to affect all segments of the US population, non-Hispanic blacks and Mexican Americans bear a disproportionate burden, independent of BMI.[10,11] There is also evidence that diabetes in minorities has increased over time at a faster rate than among whites and is not explained by BMI alone.[12,13] Increases in WC adjusted for BMI have not disproportionately affected blacks and Mexican Americans relative to whites.[6] Nevertheless, it is possible that minorities may be more sensitive to adverse changes in body composition given their higher prevalence of diabetes compared to whites regardless of anthropometry. This may result in even larger race/ethnic disparities in dysglycemia over time even for the same BMI level.

Using data from nationally representative health surveys spanning over a 20-year period, we examined trends in diabetes and prediabetes by BMI over time, whether these patterns were present across all race/ethnic groups, and we evaluated the implications of these trends on race/ethnic disparities in glycemic outcomes. We also investigated whether the observed patterns remained after accounting for temporal increases in WC.

2 | MATERIALS AND METHODS

2.1 | Data

Data came from the National Health and Nutrition Examination Surveys (NHANES), a series of ongoing national surveys conducted by the National Center for Health Statistics.[14] NHANES uses a complex, multistage sample design and is intended to be nationally representative of the US noninstitutionalized population. Survey participants completed in-home interviews followed by medical and laboratory examinations in mobile examination centers. Additionally, half of those who participate in the medical examination were asked to fast overnight for laboratory testing, comprising a nationally representative fasting subsample. These analyses were restricted to this subsample. We used data from NHANES III (1988-1994) and the continuous NHANES 2007-2012 and included nonpregnant adults aged 20 to 74 years. We excluded participants with incomplete anthropometry data, as well as participants who were underweight (BMI <18.5 kg/m^2; n = 213), yielding a sample size of 12 614 across the 2 periods (NHANES III: n = 6443; 2007-2012: n = 6171). The National Center for Health Statistics ethics review board reviewed and approved the surveys, and participants gave informed consent.

2.2 | Definition of diabetes and prediabetes

Participants were classified as having diabetes if they met any of the following criteria: (1) During the in-home interview, they answered yes to the question of whether, outside of pregnancy, a doctor or other health care professional had ever told them they had diabetes; or they reported taking insulin or diabetes medicines; (2) laboratory results indicated diabetes by either a fasting plasma glucose (FPG) value ≥126 mg/dL or a glycated hemoglobin (HbA_{1c}) ≥6.5% (48 mmol/mol).[15] We defined prediabetes as having HbA_{1c} in the range of 5.7% to <6.5% (39 to <48 mmol/mol) or FPG 100 to <126 mg/dL. Fasting plasma glucose was measured using the same hexokinase enzymatic method in NHANES III and as in the continuous surveys. Hemoglobin A_{1c} 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.[16]

2.3 | Anthropometric and demographic measures

Trained personnel used a standardized protocol to collect anthropometric measurements. Height was measured in centimeters while the participant stood without shoes, and weight was measured in kilograms while the participant stood without shoes and in light clothing. BMI was calculated as weight (kg) per height (m^2). BMI was used to classify participants as normal weight (18.5-24.9), overweight (25.0-29.9), class I obesity (30.0-34.9), and class II/III obesity (≥35.0 kg/m^2). WC was measured in centimeters at the midpoint between the bottom of the ribs and the top of the iliac crest. Demographic information was collected on the basis of self-report during the in-home interviews. Race/ethnicity was categorized as non-Hispanic white, non-Hispanic black, Mexican American, and other. However, we did not present estimates for individuals in the "other" race category given the heterogeneity of this group. Other variables included age (years), sex (male and female), and highest level of education (<high school and ≥high school).

2.4 | Statistical analysis

Appropriate sampling weights for the fasting subsample were incorporated to produce national population estimates and to account for unequal probabilities of selection, nonresponse, and noncoverage. All analyses were conducted using Stata software, version 12.1 (Stata Corp, College Station, Texas). The SVY module was used in all analyses with Taylor series linearization methods to adjust for the complex survey design. Descriptive estimates were age standardized by the direct method to the year 2000 US Census population using the age groups 20 to 34 years, 35 to 64 years, and 65 to 74 years. Differences in estimates across the 2 survey periods (1988-1994 and 2007-2012) were evaluated using the *t* statistic, and a *P* value of <.05 was considered statistically significant. We used linear probability models to separately estimate prevalence differences (PDs) in diabetes and prediabetes over time. Because associations of BMI with diabetes and prediabetes were not linear across the BMI distribution, in exploratory analyses, we first used a nonparametric approach to model the BMI-outcome relationship by relying on narrowly defined BMI categories. Models were further adjusted for cubic terms for age (due to nonlinear relationship with the outcomes), sex, race/ethnicity, education, survey period, and interactions between BMI and survey period. On the basis of the observed inflection points and to improve efficiency, we

subsequently categorized BMI using the standard cut-points to define obesity and reestimated PDs in diabetes and prediabetes over time in adjusted models. We also evaluated heterogeneity by race/ethnicity by including the relevant interaction terms. To investigate whether PDs in diabetes and prediabetes over time were attenuated after accounting for the higher mean WC over time within the BMI categories, we subsequently added WC as a continuous variable to the models using a quadratic specification since it improved the model fit. In sensitivity analyses, we substituted WC with a continuous measure of BMI to evaluate whether increases in diabetes and prediabetes over time were merely a function of a higher mean BMI within the BMI categories. Finally, some studies have shown that there is more power to predict cardiovascular risk outcomes when using a composite measure that combines BMI and WC, over using the 2 measures independently.[17] Thus, we also evaluated whether a composite measure of BMI and WC (created by classifying individuals into mutually exclusive BMI and WC category combinations using established cutoffs for BMI and WC[18]) identified diabetes and prediabetes in 2007-2012 as well as in 1988-1994.

3 | RESULTS

Table 1 presents age-standardized mean BMI and mean WC by BMI categories across survey periods for participants in the analytic sample and by race/ethnicity. In general, mean BMI, but to a much greater extent, mean WC, increased over time within BMI categories. However, the magnitude of WC increase was largest among individuals with class II/III obesity. For the most part, these patterns held across race/ethnic groups; though within most BMI categories, mean WC was similar or lower among blacks and Mexican Americans than among whites. Age-standardized glycemic characteristics are presented in Table 2. Prevalence of normoglycemia declined considerably over time in the United States, in conjunction with an increase in dysglycemia that was driven largely by a rise in prediabetes prevalence over time. However, this large increase in prediabetes appeared to occur to the greatest extent among whites, resulting in much smaller race/ethnic disparities in prediabetes by 2007-2012. Nevertheless, blacks and Mexican Americans experienced increases in diabetes prevalence twice as large as that for whites, resulting in a widening of diabetes disparities over time.

Figure 1A and 1B depicts diabetes and prediabetes prevalence over time, respectively, across narrowly defined categories of BMI. All estimates are based on multivariate linear probability models adjusting for age, sex, race/ethnicity, education, survey year, BMI, and BMI*survey year (*P*-interaction = .0067). Across normal, overweight, and class I obese values, there were no statistically significant differences in diabetes prevalence over time (Figure 1A). However, among individuals with a BMI roughly greater than 36 kg/m^2, diabetes prevalence was significantly higher over time. For prediabetes, although the BMI*survey year interaction did not reach statistical significance (*P* = .06), estimates suggested that across the distribution of BMI values in the normal, overweight, and class I obesity range, but not in the class II/III obese range, prediabetes prevalence was significantly higher in 2007-2012 than in 1988-1994 (Figure 1B). Patterns were qualitatively similar for men and women though estimates for diabetes were larger for men (not shown).

In Table 3, we show PDs in diabetes (top panel) and prediabetes (bottom panel) across the 2 survey periods by BMI estimated from linear probability models. To improve efficiency, BMI was introduced into the models as a categorical variable using the broader, standard cutoffs. Model 1 included adjustment for the same sociodemographic covariates as in Figure 1A and 1B. Consistent with the pattern observed in Figure 1A, among class II/III obese adults, diabetes prevalence was 7.6 percentage points higher in 2007-2012 than in 1988-1994, whereas for normal, overweight, and class I obese adults, there was little difference in diabetes prevalence over time. Adding WC to the model resulted in a partial attenuation of the PD across all BMI categories, including the class II/III obese, though the PD for this group remained large (PD = 5.4%, 95% CI, −0.18 to 11.0) (model 2). Replacing WC with a continuous measure of BMI in model 3 did not result in a similar degree of attenuation (not shown). Findings for prediabetes were similar to the patterns observed in Figure 1B. Adjusting for WC resulted in only a slight attenuation of the PDs, which nevertheless remained large and statistically significant (lower panel, model 2). Even though research has shown that a composite measure of BMI and WC does a better job of identifying risk than either measure independently,[17] we nevertheless observed the same deterioration in predictive power over time. Even with a cross-categorized composite measure of BMI and WC, we still observed residual time trends for diabetes and prediabetes similar to the ones from our main analyses (Table S1). There was no evidence that the secular trends in diabetes and prediabetes by BMI differed for any of the race/ethnic groups (all *P*-interaction > .3). However, estimates suggested that the PD in diabetes over time among individuals with class II/III obesity was largest for Mexican Americans (PD = 8.4%, 95% CI, 0.8-15.9) and smallest for blacks (PD = 6.6, 95% CI, −1.4 to 14.6) (Table S2, top panel, model 1). Inclusion of WC partially attenuated diabetes PD estimates for all race/ethnic groups (model 2). Using the same model, it was also evident that disparities in diabetes prevalence among blacks and Mexican Americans relative to whites were present across all BMI categories, remained in both periods, and persisted even after adjustment for WC (Figure 2).

4 | DISCUSSION

Obesity as defined by BMI is a major contributor to glucose dysregulation. However, given studies that have shown increased central adiposity for the same BMI in more recent years, we investigated the implications of this trend on diabetes and prediabetes prevalence over time. Among individuals with class II/III obesity—the group that experienced the largest temporal increases in mean WC—diabetes prevalence in 2007-2012 was higher than in 1988-1994. Adjustment for WC partially attenuated this estimate. While diabetes prevalence did not increase across the BMI groups at the lower end of the distribution, prediabetes prevalence did rise considerably, though there was little attenuation after adjusting for

TABLE 1 Age-standardized mean BMI and mean waist circumference by BMI categories among US adults aged 20 to 74 years by NHANES survey period

	All Race/Ethnic Groups			Non-Hispanic Whites			Non-Hispanic Blacks			Mexican Americans		
	1988-1994	2007-2012	Δ[a]	1988-1994	2007-2012	Δ[a]	1988-1994	2007-2012	Δ[a]	1988-1994	2007-2012	Δ[a]
N	6443	6171		2432	2567		1888	1245		1856	1071	
Normal: BMI 18.5 to 24.9 kg/m^2												
Mean BMI, kg/m^2	22.3 (0.05)	22.4 (0.06)	0.1	22.3 (0.05)	22.4 (0.07)	0.1	22.3 (0.08)	22.5 (0.07)	0.2*	22.8 (0.08)**	22.9 (0.1)**	0.1
Mean WC, cm	81.5 (0.3)	83.1 (0.2)	1.6*	81.6 (0.3)	83.5 (0.3)	1.9*	80.3 (0.3)**	82.1 (0.3)**	1.8*	83.1 (0.4)**	83.9 (0.5)	0.8
Overweight: BMI 25 to 29.9 kg/m^2												
Mean BMI, kg/m^2	27.1 (0.04)	27.4 (0.04)	0.3*	27.1 (0.05)	27.4 (0.06)	0.3*	27.3 (0.06)**	27.6 (0.09)	0.3*	27.4 (0.06)**	27.5 (0.07)	0.1
Mean WC, cm	94.5 (0.2)	96.0 (0.2)	1.5*	95.1 (0.2)	96.7 (0.2)	1.6*	92.8 (0.2)**	94.0 (0.3)**	1.2*	93.9 (0.3)**	95.3 (0.4)**	1.4*
Class I obesity: BMI 30-34.99 kg/m^2												
Mean BMI, kg/m^2	31.9 (0.07)	32.2 (0.05)	0.3*	31.9 (0.10)	32.2 (0.08)	0.3*	32.1 (0.07)	32.2 (0.09)	0.1	32.0 (0.1)	32.1 (0.1)	0.1
Mean WC, cm	104.8 (0.3)	107.1 (0.3)	2.3*	105.3 (0.3)	108.4 (0.4)	3.1*	103.4 (0.4)**	104.1 (0.5)**	0.7	103.7 (0.4)**	105.5 (0.5)**	1.8*
Class II/III obesity: BMI > =35 kg/m^2												
Mean BMI, kg/m^2	39.4 (0.2)	40.3 (0.3)	0.9*	39.1 (0.3)	40.3 (0.4)	1.2*	40.0 (0.3)**	41.1 (0.41)	1.1*	39.0 (0.3)	39.7 (0.3)	0.7
Mean WC, cm	119.0 (0.6)	123.1 (0.6)	4.1*	119.8 (0.8)	124.4 (0.9)	4.6*	117.6 (0.8)	121.6 (0.9)**	4*	116.3 (1.1)**	121.1 (1.2)**	4.8*

Abbreviations: BMI, body mass index; NHANES, National Health and Nutrition Examination Survey; WC, waist circumference.

Data are weighted means or weighted % (standard error).

All estimates were age standardized using the direct method to the 2000 US Census population using the age groups 20 to 34, 35 to 44, 45 to 64, and 65 to 74 years.

[a]This indicates difference comparing estimates in 2007-2012 to 1988-1994 within race/ethnic groups.

*P < .05, comparing difference in estimates in 2007-2012 to 1988-1994 within each race/ethnic group.

**P < .05, comparing difference in estimates among non-Hispanic blacks and Mexican Americans to non-Hispanic whites within each survey period.

TABLE 2 Age-standardized glycemic characteristics of US adults aged 20 to 74 years by NHANES survey cycle

	All Race/Ethnic Groups			Non-Hispanic Whites			Non-Hispanic Blacks			Mexican Americans		
	1988-1994	2007-2012	Δ[a]	1988-1994	2007-2012	Δ[a]	1988-1994	2007-2012	Δ[a]	1988-1994	2007-2012	Δ[a]
N	6443	6171		2432	2567		1888	1245		1856	1071	
Normoglycemia	61.9 (1.1)	46.1 (1.0)	−15.8*	64.3 (1.4)	48.9 (1.3)	−15.4*	50.4 (1.4)**	38.3 (1.8)**	−12.1*	49.2 (1.5)**	37.1 (1.6)**	−12.1*
Prediabetes	30.7 (0.9)	42.9 (0.9)	12.2*	29.2 (1.2)	42.4 (1.3)	13.2*	36.5 (1.5)**	44.4 (1.9)	7.9*	37.2 (1.5)**	44.8 (1.9)	7.6*
Diabetes	7.4 (0.5)	11.0 (0.6)	3.6*	6.5 (0.6)	8.7 (0.6)	2.2*	13.1 (0.8)**	17.3 (1.2)**	4.2*	13.6 (0.9)**	18.2 (1.3)**	4.6*
Any dysglycemia	38.1 (1.1)	53.9 (1.0)	15.8*	35.7 (1.4)	51.1 (1.3)	15.4*	49.6 (1.4)**	61.7 (1.8)**	12.1*	50.9 (1.5)**	62.9 (1.6)**	12*

Abbreviation: NHANES, National Health and Nutrition Examination Survey.

Data are weighted % (standard error).

All estimates were age standardized using the direct method to the 2000 U.S. Census population using the age groups 20 to 34, 35 to 44, 45 to 64, and 65 to 74 years.

[a]This indicates difference comparing estimates in 2007-2012 to 1988-1994 within race/ethnic groups.

*$P < .05$, comparing difference in estimates in 2007-2012 to 1988-1994 within each race/ethnic group.

**$P < .05$, comparing difference in estimates among non-Hispanic blacks and Mexican Americans to non-Hispanic whites within each survey period.

mean WC. These secular trends were observed within all race/ethnic groups.

A few studies have examined trends across a broad range of cardiovascular disease risk factors by BMI.[19,20] The most recent of these also noted that among individuals with class II/III obesity (BMI ≥ 35 kg/m^2), prevalence of not only diabetes but also hypertension and dyslipidemia increased between 1999-2002 and 2007-2010.[19] Another study that evaluated trends over a much earlier timeframe (1976-1980 through 1999-2000) did not find evidence of a significant change in diabetes prevalence by BMI category,[21] suggesting that these adverse trends reflect a more recent phenomenon. However, these studies did not consider the role of WC in potentially accounting for these observed patterns nor was there consideration of potential race/ethnic differences.

A higher mean WC over time among individuals with class II/III obesity may be at least part of the reason why diabetes prevalence increased over time in this group in the US overall and within race/ethnic groups. Adjustment for a continuous measure of BMI instead of WC did not have the same impact on estimates, suggesting that a change in fat distribution may be playing a role. While our study did not address the causes underlying this potential shift in body composition, others have speculated that factors such as higher sedentary activity, diets high in sugar and refined carbohydrates, sleep deprivation, and some pharmaceuticals may underlie greater accumulation of abdominal fat for a given BMI.[22] Similar factors have also been implicated in BMI-independent pathways to diabetes.[23–25] Individuals with BMI extremes constitute an increasing proportion of the US population, and such BMI extremes are especially prevalent among minorities.[1,26] As a result, these trends present a cause for concern because of the potential to further exacerbate race/ethnic disparities in diabetes and related outcomes.

Although mean WC also increased over time within BMI groups <35 kg/m^2, there were no secular changes in diabetes prevalence. However, prediabetes prevalence increased dramatically for individuals in this BMI range. This is consistent with another study that documented an overall 21% increase in prediabetes in the United States from 1999 to 2010, despite adjustment for BMI, and an especially concerning trend among individuals with normal BMI.[9] In our study, we explored whether greater central fat for same BMI might explain these patterns but noted only a very mild attenuation of estimates after adjustment for WC. Reasons behind this dramatic increase in prediabetes remain unclear, though similar to diabetes, lifestyle and environmental factors may be contributing to BMI-independent pathways. Importantly, this concerning trend of increasing glucose dysregulation particularly among individuals with lower BMI values points to the potential for underestimation of metabolic consequences if relying on anthropometric measures alone for risk prediction. Prediabetes is associated with an increased risk of developing diabetes and a higher prevalence cardiovascular risk factor clustering.[27] Thus, additional research will be necessary to identify the modifiable factors underlying this rise in prediabetes.

The overall trends in diabetes and prediabetes by BMI extended to all race/ethnic groups. Nevertheless, diabetes disparities among blacks

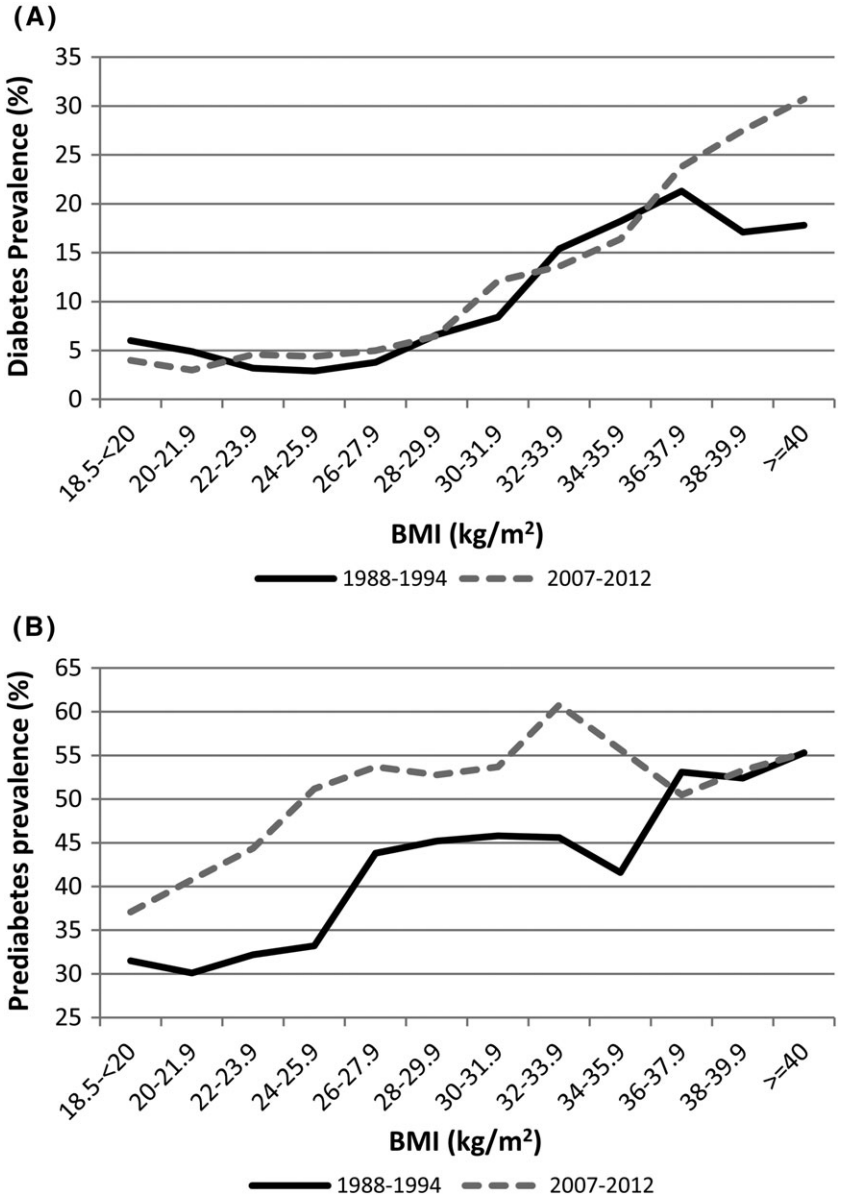

FIGURE 1 Adjusted prevalence of diabetes (A) and prediabetes (B) by BMI over time among US adults aged 20 to 74 years. Linear probability model further adjusted for age, age^2, age^3, sex, race/ethnicity, and education. BMI, body mass index

and Mexican Americans compared to whites within BMI categories were large and present across the 20+year timeframe. Moreover, because Mexican Americans in the class II/III obese category experienced a slightly greater increase in diabetes prevalence over time than corresponding whites, the potential exists for disparities to worsen over time. Other studies have noted similar race/ethnic disparities in diabetes that persist even among individuals with normal BMI.[10,11] However, the inclusion of WC in our models did not attenuate race/ethnic differences. On the contrary, our descriptive analyses showed that mean WC within BMI categories among blacks and Mexican Americans was often similar or lower than among whites. Why blacks and Mexican Americans have more diabetes even at the lower end of the BMI distribution, and even after adjusting for WC, is unknown. Hypotheses related to differences in body composition, diets high in sugar, low levels of physical activity, genetic predisposition, and differential insulin sensitivity and beta cell function by degree and location of body fat have been posited, but a definitive explanation remains elusive.[28–30] Given the fast growth of minorities, and especially of Mexican Americans,[31] identifying the reasons underlying this disparity will be essential to stem the diabetes epidemic in the US overall and in these high-risk populations.

Although the diagnostic criteria for diabetes and prediabetes have changed over time, we relied on a combination of self-reported status (for diabetes) as well as measured FPG and HbA_{1c} to define our outcomes. Therefore, improved screening and detection was not a key driver of our findings given the consistency of our definitions across the study timeframe. However, one limitation was that we did not differentiate diabetes by type since NHANES does make this distinction. While type 1 diabetes is on the rise in the United States,[32] the proportion of the adult population with type 1 is likely to be too small to meaningfully alter our findings. Waist circumference is also a relatively crude indicator of central adiposity and is measured with error, especially in individuals with class II/III

TABLE 3 Multivariate adjusted prevalence difference of diabetes and prediabetes by BMI and NHANES survey period for US adults aged 20 to 74 years

	BMI:18.5 to 24.9 kg/m^2	BMI:25 to 29.9 kg/m^2	BMI: 30 to 34.99 kg/m^2	BMI ≥ 35 kg/m^2
DIABETES				
Model 1				
Survey period				
1988-1994	Ref	Ref	Ref	Ref
2007-2012	0.03 (−1.5, 1.5)	1.1 (−0.7, 2.8)	1.5 (−2.3, 5.4)	7.6 (1.7, 14.4)
Model 2				
Survey period				
1988-1994	Ref	Ref	Ref	Ref
2007-2012	−0.09 (−1.6, 1.4)	0.71 (−1.1, 2.5)	0.76 (−3.2, 4.7)	5.4 (−0.18, 11.0)
PREDIABETES				
Model 1				
Survey period				
1988-1994	Ref	Ref	Ref	Ref
2007-2012	12.6 (9.0, 16.1)	11.2 (7.2, 15.3)	10.8 (4.5, 17.2)	3.0 (−5.4, 11.4)
Model 2				
Survey period				
1988-1994	Ref	Ref	Ref	Ref
2007-2012	11.2 (7.6, 14.8)	10.5 (6.4, 14.5)	10.4 (4.0, 16.7)	3.0 (−5.5, 11.6)

Abbreviations: BMI, body mass index; NHANES, National Health and Nutrition Examination Survey.

Data are prevalence differences and 95% CI obtained from linear probability models.

Model 1 includes age, age^2, age^3, sex, race/ethnicity, education, BMI categories, survey period, and BMI categories*survey period.

Model 2: Model 1 + waist circumference (quadratic term).

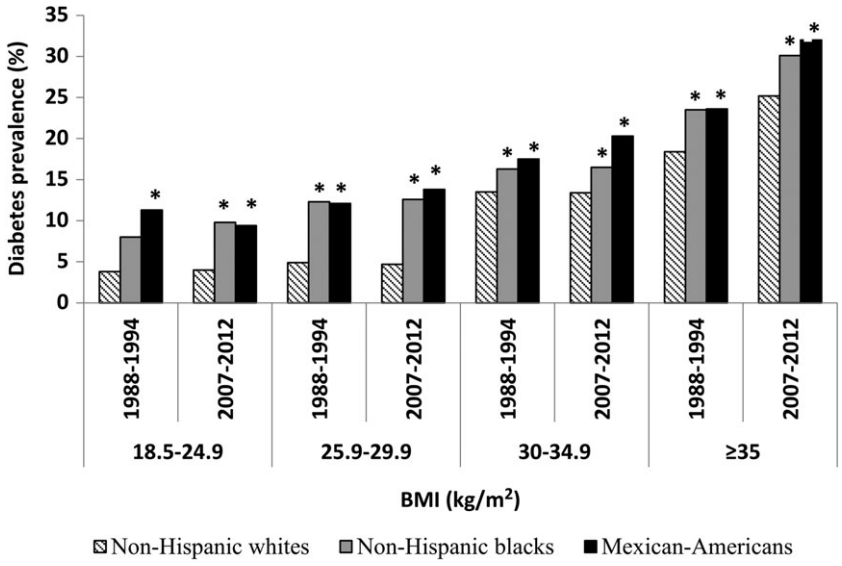

FIGURE 2 Adjusted prevalence of diabetes by race/ethnicity within BMI categories across NHANES survey periods: 1988-1994 to 2007-2012, US adults aged 20 to 74 years. Prevalence estimates obtained from a linear probability model further adjusted for age, age^2, age^3, sex, education, and waist circumference. *P < .05, comparing each race/ethnic group to non-Hispanic white referent within BMI categories and within survey period. BMI, body mass index; NHANES, National Health and Nutrition Examination Surveys

obesity. However, we do not expect this to impact our findings since this error would need to be differential over time to bias our findings. Finally, this analysis relied on cross-sectional data with only 1-time measurements of FPG and HbA_{1c} and BMI and WC. Intraindividual variability may have led to misclassification of diabetes and prediabetes and mismeasurement of adiposity in some instances. Furthermore, because of the cross-sectional design, we were unable to eliminate residual confounding and the potential role of other unmeasured factors that may have contributed to the patterns we report. Nevertheless, the goal of this study was to characterize how diabetes and prediabetes prevalence by BMI changed over time and to evaluate how estimates were impacted after

accounting for secular increases in mean WC. Additional research will be necessary to determine the causal mechanisms underlying the patterns we report.

In summary, we showed that diabetes prevalence increased over time among individuals with class II/III obesity with some attenuation after adjustment for WC. Although all race/ethnic groups were affected, the somewhat steeper increases among Mexican Americans have the potential to worsen race/ethnic disparities especially given the already high rates of obesity among Mexican Americans. This pattern was compounded by the fact that diabetes prevalence was higher for both blacks and Mexican Americans relative to whites, even at the lower end of the BMI distribution. Finally, we also documented a dramatic increase in prediabetes over time across all race/ethnic groups, which was especially apparent among normal weight individuals and which persisted even with adjustment for WC. Although obesity has been considered one of the primary contributors to the current epidemic of diabetes in the United States, it will be important to more carefully consider the role of other risk factors given the prediabetes prevalence among the normal weight and given the greater diabetes prevalence among minorities irrespective of anthropometry.

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CONFLICTS OF INTERESTS

None declared.

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SUPPORTING INFORMATION

Additional Supporting Information may be found online in the supporting information tab for this article.