Excess type 2 diabetes in African-American women and men aged 40–74 and socioeconomic status: evidence from the Third National Health and Nutrition Examination Survey

Jessica M Robbins, Viola Vaccarino, Heping Zhang, Stanislav V Kasl

Abstract

***Objective*—To examine whether socioeconomic status (SES) explains differences in the prevalence of type 2 diabetes between African-American and non-Hispanic white women and men.**

***Design*—Cross sectional study of diabetes prevalence, SES, and other risk factors ascertained by physical examination and interview.**

***Setting*—Interviews were conducted in subjects' homes; physical examinations were conducted in mobile examination centres.**

***Participants*—961 African-American women, 1641 non-Hispanic white women, 839 African-American men and 1537 non-Hispanic white men, aged 40 to 74 years, examined in the Third National Health and Nutrition Examination Survey (NHANES III), a representative sample of the non-institutionalised civilian population of the United States, 1988–1994.**

***Main results*—Among women, African-American race/ethnicity was associated with an age adjusted odds ratio of 1.76 (95% confidence intervals 1.21, 2.57), which was reduced to 1.42 (95% confidence intervals 0.95, 2.13) when poverty income ratio was controlled. Controlling for education or occupational status had minimal effects on this association. When other risk factors were controlled, race/ethnicity was not significantly associated with type 2 diabetes prevalence. Among men, the age adjusted odds ratio associated with African-American race/ethnicity was 1.43 (95% confidence intervals 1.03, 1.99). Controlling for SES variables only modestly affected the odds ratio for African/American race/ethnicity among men, while adjusting for other risk factors increased the racial/ethnic differences.**

***Conclusions*—Economic disadvantage may explain much of the excess prevalence of type 2 diabetes among African-American women, but not among men.**

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Population Studies Center, University of Pennsylvania, 3718 Locust Walk, Philadelphia, PA 19104–6298, USA
J M Robbins

Department of Epidemiology and Public Health, Yale University, USA
V Vaccarino
H Zhang
S V Kasl

Correspondence to:
Dr Robbins
(Jessica_Robbins@pop.upenn.edu)

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Racial/ethnic disparities in the prevalence of type 2 diabetes are an important public health issue in the United States. All major racial/ethnic minority groups in the United States have higher prevalences of diabetes than non-Hispanic white people.[1 2] Diabetes prevalence is increasing in all population groups in the United States, but this increase seems to be greater in minority groups.[3] The excess prevalence of type 2 diabetes is particularly striking among African-American women, and contributed to their declining life expectancy during the 1980s.[4]

In contemporary industrialised societies, including the United States, there is an inverse relation between socioeconomic status (SES) and type 2 diabetes prevalence.[5] Given the racial/ethnic disparities in SES in the United States, SES differences could account for some of the racial/ethnic disparities in diabetes prevalence. However, several studies have concluded that racial/ethnic disparities in type 2 diabetes continue to be significant after controlling for SES as well as other risk factors.[6 7]

We tested the effects of controlling for three major SES variables—income, education, and occupational status—on the racial/ethnic inequalities in type 2 diabetes observed between African-American and non-Hispanic white women and men aged 40 to 74 years in the Third National Health and Nutrition Examination Survey (NHANES III). In addition, we examined the extent to which risk factors that are often identified as potential targets for public health intervention, including body size, physical activity, diet, and tobacco and alcohol use, mediated the association between race/ethnicity and type 2 diabetes after controlling for SES.[8]

Methods

NHANES III is the most recent in a series of surveys conducted by the National Center for Health Statistics designed to assess the health and nutritional status of the non-institutionalised civilian population of the United States. NHANES III was carried out between 1988 and 1994. African-Americans and subjects aged 60 and over were oversampled. Face to face interviews were conducted in the households selected, and all subjects were invited to participate in medical examinations conducted in mobile examination centres convenient to the subjects' residences. Medical examinations in the mobile examination centres for subjects aged 40 to 74 years included oral glucose tolerance tests, unless the subject reported use of insulin.[9]

VARIABLE DEFINITION

Race/ethnicity

Each respondent was asked whether the subject's national origin or ancestry was Mexican/Mexican-American or other Latin American or Spanish, and which of four choices (Aleut, Eskimo, or American Indian; Asian or Pacific Islander; Black; and White) best described the subject's race. Those who answered no to the first question and chose "Black" were classified as African-American; those who answered no to the first question and chose "White" were classified as non-Hispanic white. All others were excluded from these analyses.

Socioeconomic status

Education was categorised based on the subject's reported number of years of education. Categories used were: 0–8 years (less than high school); 9–11 years (some high school); 12 years (high school graduate); and 13 or more years (some college). Years of education were unknown for 26 subjects; they were excluded from analyses including education.

Income was ascertained by asking respondents to select one of 28 categories as best representing total combined family income for the past 12 months. Each subject's income (taken as the midpoint of the selected range) was then divided by the applicable federal poverty line (which varies by calendar year and family size) to arrive at the poverty income ratio.[10] For these analyses, poverty income ratio was categorised as: less than 1.000 (that is, below the federal poverty line); 1.000–1.999; 2.000–2.999; 3.000–4.999; 5.000+; and missing. The "missing" category included 373 subjects, 7.5% of the sample.

Occupational status scores were assigned based on the subject's reported usual occupation. The scores assigned to each subject were the mean Duncan socioeconomic index scores calculated for the major occupational grouping (as defined by the US Census Bureau in 1980) into which the subject's usual occupation fell.[11] These scores were then modelled as tertiles. Subjects who did not report an occupation (127 women and 99 men) were excluded from analyses using this variable. For women, alternative models using head of household occupation or including a category of "no occupation" were examined; the results were similar to those presented.

Type 2 diabetes

Subjects who reported use of anti-diabetic medications were classified as having prevalent type 2 diabetes, except that subjects who reported a physician diagnosis of diabetes and use of insulin prior to age 40 were classified as type 1 diabetics and excluded from the study. Oral glucose tolerance tests with a 75 g oral load were conducted to ascertain diabetes status for the remaining subjects. Subjects examined in the morning were instructed to fast for 12 hours before the examination, and standard World Health Organisation criteria were used (fasting plasma glucose greater than or equal to 7.8 mmol/l or two hour plasma glucose greater than or equal to 11.1 mmol/l). Subjects who scheduled afternoon or evening examinations were instructed to fast for six hours before the examination, and a modified two hour plasma glucose criterion (greater than or equal to 13.9 mmol/l) suggested by the National Diabetes Data Group was used.[9] Some analyses were repeated using the 1997 American Diabetes Association criteria for diabetes.[12]

Covariables

A number of variables that are known or suspected risk factors for type 2 diabetes were examined as possible mediators of the relations between race/ethnicity and type 2 diabetes. These included three measures of adiposity: body mass index (weight in kilograms divided by the square of height in metres); waist–hip ratio (the ratio of waist circumference to hip circumference); and self reported weight at age 25, as a measure of lifetime adiposity. For 158 (3.2 per cent) of the subjects included in these analyses, weight or height measurements were not obtained, and values were imputed by the National Center for Health Statistics using stratum specific regression models. Sex specific tertiles were used to model each of these variables. Physical activity was assessed through a series of questions regarding walking ("In the past month, how often did you walk a mile or more at a time without stopping?") and recreational exercise during the past month. Each activity was assigned an intensity rating (defined as metabolic work rate divided by resting metabolic rate) based on standard classifications.[13] The authors calculated a total exercise score by multiplying the number of times that each activity was reported by its intensity rating and summing the total for all activities; the log of the score was used in analyses. Dichotomous variables for whether or not the subject engaged in any vigorous physical activity (defined as an intensity rating $\geqslant$ 6.0) during the preceding month and whether they reported being more active or less active than 10 years previously were also included in analyses controlling for physical activity. Dietary variables (based on 24 hour dietary recall) included total kilocalories consumed and saturated fat and total fat as percentages of total kilocalories. Cigarette smoking (ever smoker, current smoker, number of cigarettes smoked currently, and lifetime pack years of smoking) and alcohol use (any in past 12 months, frequency of drinking, and quantity consumed on each occasion) were also examined as potential mediators.

Several confounders were controlled in the analyses. Age (in years) was controlled in all analyses. Time of year was controlled because there was evidence in the literature that season and/or ambient temperatures could affect the results of oral glucose tolerance testing.[14–21] Both time of day (categorised as morning, afternoon, or evening) and reported fasting times (categorised as low—less than six hours for morning examinees or less than 4.5 hours for afternoon and evening examinees, average, or high—13 or more hours) were controlled as well. Day of the week (weekend versus

weekday) was controlled because of its potential association with patterns of diet, physical activity, and stress that are associated with glycaemic control. The recorded number of minutes between the initial and the "two hour" venipunctures (which ranged from 86 to 150) was controlled as well.

Data analysis

All analyses (except the descriptive data shown in table 1) use sample weights that adjust both for differential probabilities of selection and for non-response.[22] The association between race/ethnicity and type 2 diabetes was assessed using logistic regression models in SUDAAN, version 7.5.2, which uses generalised estimating equations to adjust for complex sample designs.[23] Three sets of models were examined: models adjusted only for examination related confounders and age; models in which one or more of the SES variables was included in addition; and models in which potentially mediating risk factors were also controlled.

Multicollinearity and stability of results were examined (pages 257–258).[24] A number of sensitivity analyses were conducted, such as excluding subjects whose diabetes status was ascertained by non-standard methods (afternoon and evening examinees), excluding imputed data for body mass index, and using head of household occupation or a separate category for "no occupation" in analyses of occupational status in women. There were no meaningful differences in the results observed.

Results

STUDY SAMPLE

Of the 6282 African-American and non-Hispanic white subjects aged 40 to 74 years interviewed in NHANES III, 749 did not complete an examination in the mobile examination centre. Overall response rates were higher for African-Americans than for non-Hispanic whites, and were inversely associated with SES, but did not vary with self reported health status. Subjects who reported previously diagnosed diabetes were only slightly less likely than others to be examined (85% as compared with 89%), and SES differences in rates of examination were small. Fifteen subjects who reported a physician diagnosis of diabetes and use of insulin before age 40 were excluded as possible type 1 diabetics. Another 540 subjects (who did not report use of anti-diabetic medications) failed to complete the oral glucose tolerance test and were excluded from these analyses. Among those who completed physical examinations, African-Americans were less likely to complete the oral glucose tolerance test, but within racial/ethnic strata there was little difference in income, education, or self reported health status between those who completed the oral glucose tolerance test and those who did not.

The resulting study sample included 2602 women (961 African-Americans and 1641 non-Hispanic whites) and 2376 men (839 African-Americans and 1537 non-Hispanic whites). The characteristics of the study sample are summarised in table 1. African-American women and men had less education, lower incomes, and lower occupational status than non-Hispanic white women and men. African-American women had a less favourable distribution of most of the established risk factors for type 2 diabetes. African-American men had a more favourable distribution of body sizes than non-Hispanic white men, but reported significantly less exercise. There were also significant racial/ethnic differences in several of the examination related variables. There was a higher prevalence of type 2 diabetes among

Table 1 Characteristics of the study population aged 40–74 years by sex and race/ethnicity, Third National Health and Nutrition Examination Survey, 1988–1994

	Women		*Men*	
	African-American (n=961)	*Whites (n=1641)*	*African-American (n=839)*	*Whites (n=1537)*
Education (mean, y)	11.0	12.2***	10.2	12.5***
Poverty income ratio (mean)	2.0	3.4***	2.3	3.6***
Duncan SEI score (mean)	29.5	36.4***	27.0	36.0***
Age (mean)	54.2	57.8**	55.4	58.1**
Body mass index (mean)	30.3	27.4***	26.9	27.4*
Waist–hip ratio (mean)	0.91	0.89***	0.97	1.00***
Weight at age 25 (mean, kg)	60.4	57.7***	73.9	74.8*
Exercise score (mean)	98.5	110.2***	95.0	114.6**
Total kilocalories (mean, 24 hour)	1565	1643**	2104	2350***
Ever smoked cigarettes (%)	46.0	48.9	74.5	74.6
Current cigarette smoker (%)	27.9	20.8*	43.1	25.2***
Consumed 12+ alcohol drinks/year (%)	24.2	36.4***	54.1	56.3
Examination time (%)				
Morning	48.3	50.7	50.7	50.5
Afternoon	28.0	31.7*	27.0	31.6
Evening	18.1	14.4	18.6	15.1
Fasting time (%)				
0–6 hours	10.2	6.5*	13.2	8.2***
6≤13 hours	46.1	54.4***	42.9	53.2***
13+ hours	30.8	32.6	32.1	31.7
Weekend examination (%)	31.8	27.2***	28.5	28.2***
Time to two hour venipuncture (mean, min)	120.3	119.9	119.6	119.5
Season of examination (%)				
Summer	22.3	36.9***	21.9	34.0***
Winter	27.3	18.6***	29.1	21.7***
Spring/Autumn	50.5	44.5*	49.1	44.3
Diabetic (%)	20.1	13.2***	18.9	13.9**

*0.01<p<0.05 for racial/ethnic differences. **0.001<p<0.01 for racial/ethnic differences. ***p<0.001 for racial/ethnic differences.

African-American women and men than among non-Hispanic white women or men.

KEY POINTS
- Excess type 2 diabetes prevalence among African-Americans is greater among women than men.
- The excess type 2 diabetes prevalence among African-American women is explained by differences in income and body size.
- Gender differences in the associations between race/ethnicity, body size, and type 2 diabetes parallel those between socioeconomic status, body size, and type 2 diabetes.
- Investigators should consider controlling for seasonal variations in glycaemia, which seem to have a substantial impact on diabetes prevalence in NHANES III.

WOMEN

Among women (table 2), when only age and examination variables were controlled, African-American race/ethnicity was associated with a 76 per cent excess type 2 diabetes prevalence (odds ratio 1.76, 95% confidence intervals (95% CI) 1.21, 2.57). When race/ethnicity and education were evaluated together, the odds ratio for African-American race/ethnicity was reduced to 1.59 (95% CI 1.09, 2.31). Controlling for occupational status produced almost identical results (odds ratio 1.58, 95% CI 1.09, 2.28). When poverty income ratio rather than education or occupational status was added to the model, the excess prevalence of type 2 diabetes among African-American women was reduced to 42 per cent (odds ratio 1.42, 95% CI 0.95, 2.13). The addition of education and/or occupational status to models including poverty income ratio was not significant and did not substantially change the results (data not shown).

When body size variables were controlled, race/ethnicity was not significantly associated with type 2 diabetes if any one of the three SES variables was included in the model. Including poverty income ratio in the model almost completely eliminated the excess prevalence associated with African-American race/ethnicity, (odds ratio 1.08, 95% CI 0.68, 1.72). When each of the other potentially mediating variables was examined individually (data not shown), there was no impact on the association between race/ethnicity and diabetes. Models controlling for all the mediating variables further reduced the odds ratios associated with African-American race/ethnicity (table 2).

When we repeated our analyses using the 1997 American Diabetes Association criteria for type 2 diabetes, based solely on fasting glucose values, the excess prevalence of diabetes among African-American women was noticeably greater than in the analyses using World Health Organisation criteria, which are based on both fasting and two hour glucose values. Despite this, when both poverty income ratio and other risk factors were controlled, the odds ratio associated with African-American race/ethnicity was no longer statistically significant (1.27, (95% CI 0.72, 2.25) data not shown).

Because the finding that race/ethnicity was not an independent risk factor for type 2 diabetes among women after SES variables or other risk factors were controlled differs from the results of other studies,[6 7 25 26] additional analyses were conducted to explore possible explanations for this discrepancy. Because the other studies did not control for the examination related variables included here, we repeated the analyses without controlling for these variables (time of day, time of year, day of week, reported length of fast, and the recorded number of minutes between the administration of the glucose solution and the "two hour" blood draw). The apparent risks associated with African-American race/ethnicity were substantially greater in all cases. In the model adjusted for age only, the odds ratio associated with African-American race/ethnicity was 2.16 (95% CI 1.62, 2.89).

Further exploration showed that in this study, subjects examined during the summer were less likely to have type 2 diabetes than those examined during the rest of the year. This reduced prevalence of type 2 diabetes among subjects examined in the summer was found in all four regions of the country, and among both African-Americans and non-Hispanic white subjects. African-Americans were significantly less likely than non-Hispanic white subjects to be examined in the summer (table 1), both in the South (where a majority of African-American subjects lived, and very few examinations were conducted in the summer) and in other regions of the country. The timing of the examination was therefore an important confounder in analyses examining the association between race/ethnicity and type 2 diabetes.

Table 2 Effect of race/ethnicity on type 2 diabetes prevalence in women aged 40–74 years, Third National Health and Nutrition Examination Survey, 1988–1994

Control variables	*OR* (95% CI*)*	*p Value*
Confounders only†		
African-American race/ethnicity only	1.76 (1.21, 2.57)	0.00
+ socioeconomic status†		
Controlled for education	1.59 (1.09, 2.31)	0.01
Controlled for PIR*	1.42 (0.95, 2.13)	0.08
Controlled for Duncan SEI*	1.58 (1.09, 2.28)	0.01
+ body size‡		
Controlled for education + body size	1.31 (0.84, 2.04)	0.22
Controlled for PIR* + body size	1.08 (0.68, 1.72)	0.74
Controlled for Duncan SEI* + body size	1.36 (0.85, 2.17)	0.19
+ other mediators§		
Controlled for education + mediators	1.25 (0.77, 2.04)	0.36
Controlled for PIR* + mediators	1.04 (0.62, 1.73)	0.88
Controlled for Duncan SEI* + mediators	1.30 (0.76, 2.22)	0.33

*OR, odds ratio; CI, confidence intervals; PIR, Poverty income ratio; SEI, socioeconomic index. †Adjusted for examination related variables (time of day, season of year, weekend/weekday, reported length of fast,and number of minutes between fasting and two hour venipuncture) and for age. ‡Adjusted for variables noted above plus body size variables (body mass index, waist–hip ratio, and reported weight at age 25). §Adjusted for variables noted above plus physical activity (exercise score, more/less active than 10 years ago, and intense exercise 1+ times per month), diet (total kilocalories and percent of kilocalories from fat), cigarette smoking (current, ever, current frequency, and lifetime pack years), and alcohol consumption (current, frequency of drinking, and usual amount consumed).

MEN

African-American race/ethnicity was associated with a 43 per cent excess prevalence of type 2 diabetes among men in models adjusted only for age and confounders (table 3, odds

Table 3 Effect of race/ethnicity on type 2 diabetes prevalence in men aged 40–74 years, Third National Health and Nutrition Examination Survey, 1988–1994

Control variables	*OR* (95% CI*)*	*p Value*
Confounders only†		
African-American race/ethnicity only	1.43 (1.03, 1.99)	0.03
+ socioeconomic status†		
Controlled for education	1.30 (0.89, 1.88)	0.16
Controlled for PIR*	1.41 (1.01, 1.96)	0.04
Controlled for Duncan SEI*	1.48 (1.00, 2.19)	0.04
+ body size‡		
Controlled for education + body size	1.65 (1.13, 2.41)	0.01
Controlled for PIR* + body size	1.78 (1.25, 2.54)	0.00
Controlled for Duncan SEI* + body size	1.89 (1.27, 2.81)	0.00
+ other mediators§		
Controlled for education + mediators	1.79 (1.27, 2.53)	0.00
Controlled for PIR* + mediators	1.86 (1.33, 2.61)	0.00
Controlled for Duncan SEI* + mediators	2.00 (1.40, 2.86)	0.00

*OR, odds ratio; CI, confidence intervals; PIR, Poverty income ratio; SEI, socioeconomic index. †Adjusted for examination related variables (time of day, season of year, weekend/weekday, reported length of fast, and number of minutes between fasting and two hour venipuncture) and for age. ‡Adjusted for variables noted above plus body size variables (body mass index, waist–hip ratio, and reported weight at age 25). §Adjusted for variables noted above plus physical activity (exercise score, more/less active than 10 years ago, and intense exercise 1+ times per month), diet (total kilocalories and percent of kilocalories from fat), cigarette smoking (current, ever, current frequency, and lifetime pack years), and alcohol consumption (current, frequency of drinking, and usual amount consumed).

ratio 1.43, 95% CI 1.03, 1.99). The addition of education to the model reduced this slightly (odds ratio 1.30, 95% CI 0.89, 1.88), but poverty income ratio and occupational status did not substantially change the association.

In a reversal of the effects seen with women, when potential mediators were added to the model, the association between race/ethnicity and type 2 diabetes prevalence was strengthened, and there was a significant excess prevalence of diabetes associated with African-American race/ethnicity, regardless of which of the SES variables was controlled. Body size variables were again the important added variables, but among the men central obesity (as measured by waist-hip ratio) was clearly most important (data not shown), accounting for about 80 per cent of the difference in odds ratios produced by controlling for all of these variables.

We also tested these results when examination related confounders were not controlled (data not shown). As with the results among women, the association between African-American race/ethnicity and type 2 diabetes prevalence was inflated, with an age adjusted odds ratio of 1.66 (1.24, 2.23). The effects of the addition of SES and other variables to the models were essentially the same as in the models described above.

Discussion

Two studies based on earlier data investigated the question of whether SES accounts for the excess prevalence of type 2 diabetes in African-Americans and concluded that it does not.[6 7] Neither of these studies presented gender specific analyses, and neither examined income adjusted for family size as a measure of SES. Davey Smith *et al* found that among the volunteers screened for the Multiple Risk Factor Intervention Trial, excess mortality from diabetes among African-American men was substantially explained by area level income, and completely explained when other risk factors were controlled.[27]

A previous analysis of data from NHANES III, the same dataset examined here, concluded that African-American race/ethnicity was significantly associated with increased prevalence of type 2 diabetes in women 25–64 years of age after controlling for SES.[26] Several different analyses were performed. There are a number of methodological differences that could explain why the findings of Winkleby *et al* were different from those reported here for women. The most important seems to be that we controlled for the season of the year when the examinations were conducted, as well as time of day, day of the week, and fasting times. Winkleby *et al* excluded subjects who reported fasting times of less than eight hours and did not include any other examination related variables in their analyses. Season of the year was an important confounder of the association between race/ethnicity and diabetes prevalence in the data we analysed. When examination related variables were not controlled in our analyses, the association between African-American race/ethnicity and type 2 diabetes prevalence was inflated, although still not statistically significant after adjustment for poverty income ratio and mediating variables.

The finding that income, modelled as poverty income ratio, is more strongly associated with type 2 diabetes prevalence and a more important factor in explaining the excess prevalence of type 2 diabetes among African-American women than other SES variables is consistent with other studies, including that of Winkleby *et al*.[26] Years of education may be less effective as a control for socioeconomic differences between African-Americans and white subjects because most of the African-Americans surveyed were raised in the South and attended segregated schools characterised by inadequate resources, undertrained teachers, and truncated school years. Years of education in such schools provided educational opportunities grossly unequal to those afforded to white subjects.[28]

Controlling only for age and examination related confounders, the excess prevalence of diabetes among African-American men was substantially less than that among women. This has been observed in previous studies,[29–31] although there are also conflicting findings.[7] Controlling for SES substantially reduced the excess prevalence of diabetes among African-American women, but not men. Additional adjustment for possible risk factors for diabetes had diametrically opposite effects among women and men on the association between race/ethnicity and diabetes, reducing the excess prevalence of diabetes among African-American women, while substantially increasing it among African-American men. This reflected the different racial/ethnic distribution of these risk factors by gender. African-American men in NHANES III had lower body mass index, waist–hip ratio, and weights at age 25 than non-Hispanic white men, while African-American women had substantially higher values for all of these body size variables than non-Hispanic white women. Our finding of a significant association between race/

ethnicity and diabetes among men after adjustment for risk factors is consistent with the findings of other investigators.[6 7 25]

Several investigators have observed sex differences in the effects of different risk factors on type 2 diabetes[7 8 25] and related metabolic variables.[32 33] Sex ratios in type 2 diabetes prevalence vary widely between populations, providing further evidence that environmental risk factors affect the sexes differently.[34] There are also substantial sex differences in the effects of diabetes, most importantly on coronary heart disease.[35]

While the literature on race/ethnicity, SES and health has rarely focused on sex differences, there is evidence that the health risks associated with both African-American race/ethnicity and low SES operate through different pathways in women and men. Obesity is a particularly important risk factor for diabetes that is associated with both African-American race/ethnicity and low SES among women, but not men.[36 37] There is also some evidence that stress, which is widely hypothesised to play an important part in the associations between race/ethnicity, SES, and health,[38 39] has different psychological and metabolic effects on women and men.[40] The relative unimportance of SES as a risk factor for type 2 diabetes among men has also been observed in studies of Mexican Americans.[41 42]

A number of limitations of this analysis should be emphasised. Despite extensive efforts in NHANES III to maximise response rates, substantial non-response did occur.[22] Non-response bias occurs where the association being examined varies by response status. There is no obvious reason to anticipate such a pattern. None the less, the potential for non-response bias cannot be discounted.

There are important elements of SES that were not examined in this study. Direct measures of childhood SES or SES through the lifecourse and measures of economic assets, including home ownership, were not available. Because African-Americans have far fewer economic assets than non-Hispanic white people at the same levels of income or education, controlling for these variables might further reduce the apparent excess of type 2 diabetes among African-American women. It is also possible that these aspects of SES would impact on type 2 diabetes prevalence among men. In addition, the cross sectional nature of the NHANES III data does not permit us to exclude the possibility of reverse causality, with diabetes prevalence impacting family income.

By limiting the diagnosed cases defined as type 2 diabetes to those reporting use of anti-diabetic medications and excluding those who were diagnosed and initiated insulin use before age 40, this study may have excluded some type 2 cases. Because the numbers of diagnosed diabetes cases excluded was small, this is unlikely to have had any important effect on the results.

This is the only study of which we are aware in which variables relating to the timing and conditions of oral glucose tolerance tests have been so extensively controlled. Controlling for these variables—especially the time of year in which the examination was conducted—results in conclusions different from those found by other investigators, raising the question of whether there was uncontrolled confounding in the previous studies. We would argue that future epidemiological studies should attempt to control for these variables, unless examination of the data shows that they are not confounders in the given study. Our findings also underline the sex differences in the impact of a number of different risk factors on type 2 diabetes, and the importance of examining sex specific models.

The results of this analysis suggest that the well established finding of higher prevalence of type 2 diabetes among African-American women than among non-Hispanic white women in the United States may reflect the economic disadvantage suffered by African-Americans, rather than any race specific genetic susceptibility. They underscore that the failure to find socioeconomic or environmental explanations for factors in the explanations for racial/ethnic disparities in health in any given analysis is not meaningful evidence that these differences are genetic in origin. The tools we use to measure the effects of SES on health are limited.[3] Residual confounding may explain the persistence of race/ethnicity effects in many analyses.[43] The health effects of stressors related to racial and ethnic discrimination are another, largely unexplored possible cause of health inequalities.[39 44 45] The elimination of racial/ethnic inequalities in type 2 diabetes and other health outcomes in the United States may require not the tools of the biomedical laboratory, but those of social, political, and economic action to reduce economic inequalities and social injustice.

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