Disparities in Diabetes by Education and Race/Ethnicity in the U.S., 1973–2012

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Introduction: Diabetes mellitus incidence has more than doubled in the U.S. over the past 2 decades. Not all sectors of the population have experienced the increase proportionally. The goal of this study was to determine if disparities in diabetes by education and race/ethnicity have increased over time, and if there are differences by gender and birth cohort.

Methods: Repeated cross-sectional data were used from the 1973–2012 National Health Interview Survey of adults aged 25–84 years. Logistic regression models were run and predicted probabilities were calculated to determine if disparities in self-reported diabetes by education and race/ethnicity changed over time, by gender and birth cohort (birth before 1946, 1946–1970, 1971 or after). Analyses were conducted in 2014–2015.

Results: Relationships between education or race/ethnicity and diabetes were modified by time for people born before 1971, with stronger effect modification for women than men. Inequalities in diabetes prevalence grew over time, although the magnitude of disparities was smaller for the 1946–1970 cohort. For example, in 2005–2012, the gap in diabetes prevalence for women with the highest and lowest levels of education was 12.7% for pre-1946 versus 7.9% for 1946–1970. Similar trends were seen for differences between non-Hispanic whites and non-Hispanic blacks or Hispanics. Results were inconclusive for the youngest cohort.

Conclusions: Diabetes disparities are evident. Smaller differences in later cohorts may indicate that large structural changes in society (e.g., Civil Rights movement, increased educational and economic opportunities) have benefited later generations.
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Introduction

Over the past 2 decades, the rate of newly diagnosed Type 2 diabetes mellitus (hereafter, diabetes) cases among adults in the U.S. has more than doubled.[1] People with diabetes have substantially higher mortality, with a decreased life expectancy of 14 years, compared with people without diabetes.[2,3] Societal costs for diabetes are also high: Medical expenses and loss of productivity were estimated to cost the U.S. $245 billion in 2012.[4]

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Not all populations are equally impacted by diabetes. SES and race/ethnicity are key determinants. Lower educational attainment is associated with higher diabetes incidence in the U.S.[5] From the 1970s to the early 2000s, the increase in diabetes prevalence among those with lower levels of education was more than twice that of their higher-educated counterparts.[6] Moreover, non-Hispanic blacks, Hispanics, and Asians have up to twice the prevalence of diabetes compared with non-Hispanic whites, and disparities by race/ethnicity in the U.S. are increasing.[7,8]

Evidence suggests that gender and birth cohort may modify socioeconomic disparities in diabetes. Women of lower SES have higher prevalence of diabetes and related risk markers, whereas men exhibit weaker, and sometimes no, associations between diabetes and SES.[9–11] In addition, birth cohorts may experience socioeconomic disparities in diabetes differentially,[12] resulting from social changes related to improved educational attainment and differences in the experience of racial/ethnic minorities.[13,14]

Diabetes risk is increasing,[1,15] and appears to be doing so differentially across the population.[6–9,16] Examining differences in diabetes risk by key sociodemographic characteristics, including gender, education, race/ethnicity, and birth cohort, may provide important insight into future disease burden as well as highlight which salient factors impact health disparities. The purpose of this paper is to examine changes in diabetes prevalence in the U.S. over time by education and race/ethnicity, with particular attention to gender and cohort differences.

Methods

Data came from the 1973–2012 waves of the National Health Interview Survey, a nationally representative annual survey conducted by the National Center for Health Statistics.[17] Data were downloaded from the Integrated Health Interview Surveys.[18]

Study Population

Two analytic samples were constructed to examine the relationships between education and diabetes and race/ethnicity and diabetes, respectively. Data were restricted to adults, aged 25–84 years, to allow for completion of education for most people. The education sample included data from 1973 to 2012 for all adults with complete information for education status, with the exception of data from 1976, which did not include a measurement of diabetes (n=931,207). The race/ethnicity sample was restricted to self-reported non-Hispanic white (hereafter, white); non-Hispanic black (hereafter, black); Hispanic; and Asian adults for the years 1978–2012 (n=720,575). Hispanic ethnicity and Asian race were not identified prior to 1978.

Measures

The outcome in this study was diabetes; measurements were harmonized and weighted by the Integrated Health Interview Surveys. Diabetes status was measured by asking whether a participant had ever been told they had diabetes in 1974, 1983, 1988, 1991, 1993, and 1997–2012.[19] For all other years, diabetes status was assessed by asking participants whether they had been told they had diabetes during the past 12 months or year.[20,21] Surveys from 1983, 1988, 1991, and 1993 asked both versions of the question. Because diabetes was not assessed uniformly over time, the measurement was adjusted to enhance comparison across all survey years, as described below.

Educational attainment was categorized as Grade 8 or less; Grade 9–11; Grade 12 (high school graduation); 1–3 years of college; and 4 years of college or higher level education. Race/ethnicity was defined as white, black, Hispanic, and Asian. Age was a continuous variable and centered at the median of 45 years. Time period was classified as 1973–1980, 1981–1988, 1989–1996, 1997–2004, and 2005–2012 in the education analytic sample, and 1978–1984, 1985–1991, 1992–1998, 1999–2005, and 2006–2012 in the race/ethnicity analytic sample. In light of the different life experiences of individuals born during the 20th century, respondents were grouped into three distinct birth cohorts: Cohort 1 was born before 1946 (e.g., The Lucky Few); Cohort 2 was born between 1946 and 1970 (e.g., Baby Boomers); and Cohort 3 was born after 1970 (e.g., Late Boomers and Generation Xers).[22] This operationalization distinguishes cohorts in qualitatively meaningful ways based on salient social and historic changes, including the Civil Rights Movement (1955–1968); immigration reforms (e.g., Bracero Era for Latinos from 1942 to 1964, reforms in 1980s); and the expansion of educational opportunities following World War II (e.g., GI Bill).[22]

Statistical Analysis

To harmonize the measurement of diabetes across survey years, "ever told" was used as the gold standard for analysis. In years where "past year" was used exclusively, diabetes status was treated as mis-specified. The consequences of a mis-specified binary outcome have been well researched.[23] To correct for outcome mis-specification, the probability someone had diabetes according to "past year" was estimated given they had diabetes according to "ever told" (sensitivity), and the probability someone did not have diabetes according to "past year" was estimated given they did not according to "ever told" (specificity). These values were used to impute outcome data for people with the missing "ever told" outcome while incorporating uncertainty in the values.[24] The authors did not impute outcome data for anyone who did not have the "past year" diabetes measurement. Estimates from the imputed data sets were then combined using standard multiple imputation techniques.[25] Five imputed data sets were used; increasing the number of imputed data sets had minimal impact on results.

A series of four models were run, by birth cohort and gender, for both analytic samples:

1. sociodemographic marker only;
2. sociodemographic marker and age;
3. sociodemographic marker, age, and period; and
4. sociodemographic marker, age, period, and an interaction between the sociodemographic marker and period to examine differences over time.

Then, predicted probabilities of diabetes were calculated (for the median age of 45 years) by cohort, gender, period, and sociodemographic marker from Model 4 and results were graphed. Analyses were run in 2014–2015 using SAS, version 9.3, and accounted for the misclassification of the outcome as well as the complex survey design of the National Health Interview Survey, including the primary sampling units, strata, and sampling weights (defined by the Integrated Health Interview Surveys) using survey procedures.

Three sensitivity analyses were conducted. First, the race/ethnicity analysis was repeated controlling for education, to confirm that results were not due to educational attainment; likewise, education results were re-examined controlling for race/ethnicity. Second, the influence of nativity (i.e., country of birth) on the race/ethnicity results for Cohort 3 was considered; nativity data were collected starting in 1997. Third, potential Type 1 diabetes cases (defined as being diagnosed before age 30 years and currently taking insulin[26]) were eliminated and analyses were rerun for Cohort 3, owing to data availability starting in 1997.

Results

The average prevalence of diabetes was 6.4% in the education analytic sample (Appendix Table 1, available online). More than 80% of participants had at least a high school education. The average prevalence of diabetes was 6.5% in the race/ethnicity analytic sample. More than three quarters of the population was white, 10.9% was black, 9.5% was Hispanic, and the remaining 3.2% was Asian. Descriptive data by period and cohort are available online (Appendix Tables 2 and 3).

Regardless of birth cohort, women with higher education had lower odds of diabetes than women with lower education (Table 1). Point estimates were similar when comparing women with the highest level of education (≥4 years of college) to women with the lowest level of education (≤Grade 8) for women born before 1946 and women born between 1946 and 1970 (Model 3: OR=0.31, 95% CI=0.28, 0.33 for Cohort 1; OR=0.33, 95% CI=0.30, 0.38 for Cohort 2). However, differences were more striking along the educational gradient for women born before 1946 (Cohort 1).

For men, the strongest inverse educational gradients for diabetes risk were seen in Cohort 2. Men born between 1946 and 1970 with ≥4 years of college had 0.42 times the odds of diabetes (Model 3, 95% CI=0.37, 0.47) compared with men with ≤Grade 8 education. This relationship was not as strong among men born before 1946, and was not statistically significant for men born after 1970. Differences along the educational gradient were observed for men in Cohorts 1 and 2, with higher education associated with lower odds of diabetes, but not for men in Cohort 3.

The relationship between education and diabetes was modified by time period for women and men, with variation by birth cohort. The strongest effect modification was among women born before 1971 (p=0.0196 for Cohort 1, p=0.0003 for Cohort 2 in Model 4) and men born between 1946 and 1970 (p=0.0146) (Figure 1). For women in Cohort 1, the gap in diabetes prevalence between those with the highest and lowest educational levels was 3.9% in 1973–1980 (prevalence, 2.5% for ≥4 years of college vs 6.4% for ≤Grade 8). The difference in diabetes prevalence between the highest and lowest education levels increased ~325% from the 1973–1980 period to the 2005–2012 period up to 12.7% (2005–2012 prevalence, 7.0% for ≥4 years of college vs 19.7% for ≤Grade 8). The gap was much narrower for men in Cohort 1 in the earliest time period (1.4%), but again increased (~390%), reaching 5.5% in 2005–2012 (prevalence, 8.6% for ≥4 years of college vs 14.1% for ≤Grade 8) (p=0.0757, Model 4). The trends were similar for women and men in Cohort 2, but large differences across the educational gradient only started to emerge in 1981–1988 for women and 1989–1996 for men. By 2005–2012, the difference in diabetes prevalence between the highest and lowest educational groups was 7.9% for women and 6.8% for men. Trends for Cohort 3 were less clear given the relatively young age of people in this cohort.

Disparities in diabetes prevalence between white respondents and other groups varied across cohorts for women (Table 2). When compared with whites, blacks had a higher odds of diabetes; differences were greatest for women born before 1946, and least (though still substantial) for women born after 1970. Differences in diabetes prevalence followed similar patterns across cohorts for Hispanics compared with whites, although the magnitude of the differences were not as great as those for blacks. Asian women had a higher odds of diabetes than white women for Cohort 1, but this relationship was null for Cohort 2, and opposite for Cohort 3.

Differences in diabetes prevalence between whites and other groups remained fairly constant across cohorts for men, with the exception of Hispanic men. The largest differences, as with women, were between black and white men, with ORs between 1.74 (Cohort 3) and 1.82 (Cohort 2). Hispanic men also had a higher odds of diabetes than white men across all three cohorts, although the magnitude of the association differed. The OR for diabetes ranged from 1.17 (Cohort 1) to 1.27 (Cohort 2) for Asian men compared with white men; differences were not statistically significant for Cohort 3.

As with education, the relationship between race/ethnicity and diabetes was modified by time period for women and men, with variation by birth cohort. For women and men in Cohort 1, the gap in diabetes prevalence between whites and other race/ethnicity groups grew over time (Figure 2). For example, the difference between blacks and whites was 4.0% in 1978–1984 and 9.8% in 2006–2012 for women, and 2.3% in 1978–1984 and 7.4% in 2006–2012 for men. Similar patterns were seen for Hispanic women and men compared with whites, although the magnitude of the differences was slightly less. Differences between Asians and whites only emerged in 2006–2012 for women and men in Cohort 1. For Cohort 2 women, differences between whites and blacks or Hispanics started to emerge in 1992–1998, with the greatest differences seen in 2006–2012 (p=0.0044). Asian women had lower diabetes prevalence than white women in the two earliest periods, and no differences later. The interaction between race/

Table 1. AORs for Association Between Education and Diabetes Stratified by Gender and Birth Cohort, 1973–2012 NHIS

Models and variables	Female: Cohort 1 (before 1946), OR (95% CI)	Female: Cohort 2 (1946–1970), OR (95% CI)	Female: Cohort 3 (after 1970), OR (95% CI)	Male: Cohort 1 (before 1946), OR (95% CI)	Male: Cohort 2 (1946–1970), OR (95% CI)	Male: Cohort 3 (after 1970), OR (95% CI)
Model 1						
Education						
≤Grade 8	1.00	1.00	1.00	1.00	1.00	1.00
Grade 9–11	0.78 (0.74, 0.82)	0.80 (0.71, 0.91)	1.16 (0.77, 1.74)	0.93 (0.86, 0.99)	0.72 (0.63, 0.82)	1.20 (0.69, 2.07)
Grade 12	0.57 (0.54, 0.60)	0.55 (0.50, 0.61)	0.98 (0.70, 1.38)	0.84 (0.80, 0.89)	0.64 (0.58, 0.72)	1.32 (0.80, 2.20)
1–3 years of college	0.56 (0.53, 0.60)	0.56 (0.50, 0.62)	0.89 (0.64, 1.23)	0.96 (0.90,1.03)	0.68 (0.60, 0.76)	1.35 (0.81, 2.26)
≥4 years of college	0.36 (0.33, 0.38)	0.34 (0.31, 0.38)	0.44 (0.30, 0.62)	0.66 (0.62, 0.72)	0.45 (0.40, 0.50)	0.76 (0.45, 1.28)
Model 2						
Education						
≤Grade 8	1.00	1.00	1.00	1.00	1.00	1.00
Grade 9–11	0.91 (0.87, 0.97)	0.99 (0.87, 1.11)	1.24 (0.82, 1.86)	1.10 (1.02, 1.18)	0.90 (0.78, 1.03)	1.30 (0.75, 2.27)
Grade 12	0.69 (0.65, 0.72)	0.61 (0.56, 0.68)	1.02 (0.73, 1.44)	1.05 (0.99, 1.11)	0.73 (0.65, 0.81)	1.41 (0.85, 2.35)
1–3 years of college	0.65 (0.60, 0.69)	0.55 (0.49, 0.61)	0.92 (0.66, 1.27)	1.16 (1.08, 1.24)	0.67 (0.59, 0.75)	1.45 (0.86, 2.42)
≥4 years of college	0.43 (0.40, 0.47)	0.34 (0.30, 0.38)	0.44 (0.30, 0.64)	0.82 (0.76, 0.89)	0.42 (0.37, 0.47)	0.77 (0.46, 1.30)
Age (centered)	1.038 (1.036, 1.039)	1.082 (1.079, 1.086)	1.10 (1.08, 1.12)	1.047 (1.045, 1.049)	1.099 (1.095, 1.103)	1.12 (1.10, 1.15)
Model 3						
Education						
≤Grade 8	1.00	1.00	1.00	1.00	1.00	1.00
Grade 9–11	0.80 (0.76, 0.84)	0.99 (0.87, 1.11)	1.24 (0.82, 1.86)	0.97 (0.90, 1.04)	0.90 (0.78, 1.04)	1.31 (0.75, 2.27)
Grade 12	0.54 (0.51, 0.57)	0.61 (0.56, 0.68)	1.02 (0.73, 1.44)	0.82 (0.77, 0.87)	0.73 (0.55, 0.81)	1.41 (0.85, 2.35)
1–3 years of college	0.46 (0.43, 0.49)	0.54 (0.49, 0.60)	0.91 (0.66, 1.27)	0.81 (0.76, 0.88)	0.66 (0.59, 0.75)	1.44 (0.86, 2.42)
≥4 years of college	0.31 (0.28, 0.33)	0.33 (0.30, 0.38)	0.44 (0.31, 0.63)	0.58 (0.54, 0.63)	0.42 (0.37, 0.47)	0.77 (0.46, 1.30)

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Table 1. AORs for Association Between Education and Diabetes Stratified by Gender and Birth Cohort, 1973–2012 NHIS (*continued*)

	Female			Male		
Models and variables	**Cohort 1 (before 1946), OR (95% CI)**	**Cohort 2 (1946–1970), OR (95% CI)**	**Cohort 3 (after 1970), OR (95% CI)**	**Cohort 1 (before 1946), OR (95% CI)**	**Cohort 2 (1946–1970), OR (95% CI)**	**Cohort 3 (after 1970), OR (95% CI)**
Age (Centered)	1.020 (1.018, 1.022)	1.070 (1.066, 1.074)	1.09 (1.07, 1.12)	1.026 (1.024, 1.029)	1.082 (1.077, 1.086)	1.11 (1.08, 1.14)
Period						
1973–1980	1.00	1.00		1.00	1.00	
1981–1988	1.29 (1.21, 1.36)	0.91 (0.68, 1.22)		1.34 (1.25, 1.43)	0.82 (0.65, 1.04)	
1989–1996	1.56 (1.47, 1.66)	0.98 (0.76, 1.28)		1.62 (1.51, 1.75)	0.77 (0.61, 0.97)	
1997–2004	2.45 (2.32, 2.60)	0.99 (0.81, 1.21)	1.00	2.85 (2.67, 3.03)	1.02 (0.83, 1.25)	1.00
2005–2012	3.62 (3.40, 3.86)	1.39 (1.13, 1.72)	1.23 (0.99, 1.53)	3.86 (3.59, 4.16)	1.41 (1.14, 1.76)	1.36 (1.02, 1.81)

NHIS, National Health Interview Survey.

ethnicity and period was not statistically significant for men in Cohort 2 or women or men in Cohort 3.

The first sensitivity analysis mutually controlled for education and race/ethnicity. Education results controlling for race/ethnicity showed similar point estimates in Model 3, and similar patterns for the interaction models between education and period. For race/ethnicity, Model 3 ORs were slightly attenuated, but the general patterns and statistical significance held. The exception was Asian men, who had a higher odds of diabetes compared with whites across all three cohorts after adjusting for education. Patterns were the same in the interaction models.

Second, in examining the role of nativity, differences between Hispanic and white women and men were greater after adjustment for nativity. Asian men also had a higher odds of diabetes compared with white men after adjustment, although the lower odds of diabetes for Asian women compared with white women was attenuated. Interactions between race/ethnicity and period remained statistically non-significant.

Lastly, when probable Type 1 diabetes cases were eliminated, higher education was more protective against diabetes for both genders, and differences between white men and women and other racial/ethnic groups were more pronounced. The exception was that the lower odds of diabetes among Asian compared with white women was attenuated. Interactions between education and period and race/ethnicity and period were not statistically significant. Sensitivity analysis results are available upon request.

Discussion

Using a large, nationally representative U.S. sample with data from 1973 to 2012, results indicated that disparities in diabetes prevalence are increasing over time for women and men, by education and race/ethnicity. Importantly, the gap between those of highest and lowest education, and between non-Hispanic whites and other race/ethnic groups, varied by birth cohort. For people born before 1946, a time of racial segregation and limited access to higher education, diabetes disparities increased more over time than for individuals born between 1946 and 1970, which corresponded to a period of increasing access and equality. The story remains untold for the youngest cohort, born after 1970, although diabetes disparities persist. Regardless of cohort, disparities by education and race/ethnicity were greater among women than men.

Relationships between education or race/ethnicity and diabetes were modified by time for women and men, with variation by birth cohort. As diabetes prevalence

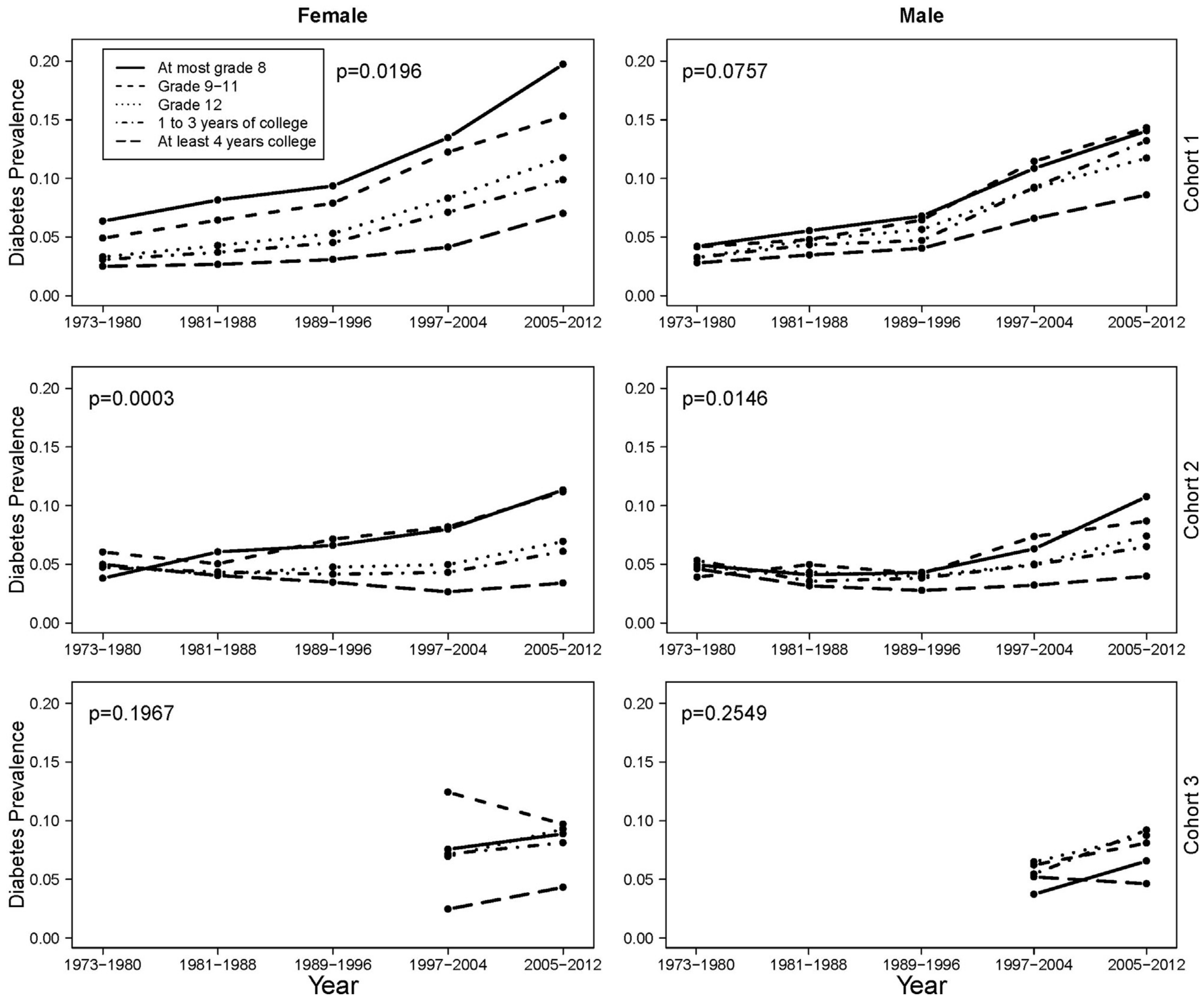

Figure 1. Disparities in diabetes by education according to gender and birth cohort, 1973–2012.

increased over time, disparities also increased for Cohorts 1 and 2. Potential explanations for these findings include secular changes and cohort differences. The diabetes epidemic is largely fueled by the obesity epidemic; disparities in BMI have increased over time, differentially impacting women, communities of color, and individuals with less education.[27–29] When examining the differences between Cohorts 1 and 2, the accumulation of disadvantage might explain the larger diabetes-related disparities for Cohort 1. Among blacks and Hispanics born prior to 1946, pervasive structural disadvantage, including racial and economic discrimination, may have created conditions that jeopardized health over the life course.[30,31] Later cohorts of racial minorities and women have made socioeconomic gains, including the expansion of educational and occupational opportunities. These individuals came of age during the social changes of the 1960s (i.e., Civil Rights and Feminist Movements), thereby reaping the sociopolitical gains of a transformed racial and gender system.[32,33] Similar work on the importance of social and political change in advancing health have been documented elsewhere.[34] For instance, Krieger and colleagues[35] offer evidence that the abolition of legalized segregation in the form of Jim Crow laws affected premature death among younger cohorts of black Americans. These results hint at the importance of broad social changes in influencing health disparities.

The reasons behind the gender differences in diabetes risk across education and race/ethnicity remain unclear. Although the positive association between health and education is well established, the intersections of gender, education, and health are less well understood.[36] An important and emerging avenue of explanation lies in

Table 2. AORs for Association Between Race/Ethnicity and Diabetes Stratified by Gender and Birth Cohort, 1978–2012 NHIS

Models and variables	Female			Male		
	Cohort 1 (before 1946), OR (95% CI)	**Cohort 2 (1946–1970), OR (95% CI)**	**Cohort 3 (after 1970), OR (95% CI)**	**Cohort 1 (before 1946), OR (95% CI)**	**Cohort 2 (1946–1970), OR (95% CI)**	**Cohort 3 (after 1970), OR (95% CI)**
Model 1						
Race/ethnicity						
Non-Hispanic white	1.00	1.00	1.00	1.00	1.00	1.00
Non-Hispanic black	2.30 (2.17, 2.43)	1.81 (1.68, 1.94)	1.75 (1.45, 2.11)	1.66 (1.55, 1.79)	1.70 (1.57, 1.85)	1.75 (1.35, 2.26)
Hispanic	2.09 (1.95, 2.24)	1.72 (1.60, 1.86)	1.12 (0.92, 1.35)	1.66 (1.53, 1.80)	1.60 (1.47, 1.75)	1.23 (0.98, 1.55)
Asian	1.37 (1.19, 1.58)	1.05 (0.89, 1.23)	0.47 (0.30, 0.75)	1.33 (1.16, 1.53)	1.38 (1.20, 1.59)	1.24 (0.83, 1.85)
Model 2						
Race/ethnicity						
Non-Hispanic white	1.00	1.00	1.00	1.00	1.00	1.00
Non-Hispanic black	2.48 (2.34, 2.63)	1.93 (1.79, 2.07)	1.75 (1.45, 2.11)	1.78 (1.66, 1.91)	1.84 (1.69, 2.01)	1.74 (1.34, 2.27)
Hispanic	2.27 (2.12, 2.44)	1.82 (1.69, 1.96)	1.11 (0.92, 1.34)	1.78 (1.64, 1.93)	1.77 (1.62, 1.93)	1.23 (0.98, 1.55)
Asian	1.43 (1.24, 1.64)	0.99 (0.84, 1.17)	0.46 (0.29, 0.72)	1.37 (1.20, 1.57)	1.30 (1.12, 1.50)	1.18 (0.79, 1.77)
Age (centered)	1.041 (1.039, 1.043)	1.083 (1.079, 1.086)	1.10 (1.08, 1.12)	1.047 (1.044, 1.049)	1.099 (1.095, 1.103)	1.12 (1.09, 1.15)
Model 3						
Race/ethnicity						
Non-Hispanic white	1.00	1.00	1.00	1.00	1.00	1.00
Non-Hispanic black	2.45 (2.32, 2.59)	1.91 (1.78, 2.06)	1.74 (1.45, 2.10)	1.78 (1.66, 1.92)	1.82 (1.67, 1.98)	1.74 (1.34, 2.26)
Hispanic	2.10 (1.96, 2.24)	1.78 (1.66, 1.92)	1.11 (0.91, 1.34)	1.61 (1.49, 1.75)	1.70 (1.56, 1.86)	1.22 (0.97, 1.54)
Asian	1.24 (1.08, 1.43)	0.98 (0.83, 1.15)	0.45 (0.29, 0.72)	1.17 (1.03, 1.34)	1.27 (1.10, 1.47)	1.18 (0.79, 1.76)
Age (centered)	1.029 (1.027, 1.031)	1.073 (1.068, 1.077)	1.09 (1.07, 1.11)	1.029 (1.026, 1.032)	1.083 (1.078, 1.088)	1.11 (1.08, 1.14)
Period						
1978–1984	1.00	1.00		1.00	1.00	

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Table 2. AORs for Association Between Race/Ethnicity and Diabetes Stratified by Gender and Birth Cohort, 1978–2012 NHIS *(continued)*

	Female			Male		
Models and variables	**Cohort 1 (before 1946), OR (95% CI)**	**Cohort 2 (1946–1970), OR (95% CI)**	**Cohort 3 (after 1970), OR (95% CI)**	**Cohort 1 (before 1946), OR (95% CI)**	**Cohort 2 (1946–1970), OR (95% CI)**	**Cohort 3 (after 1970), OR (95% CI)**
1985–1991	1.13 (1.05, 1.21)	0.94 (0.76, 1.16)		1.24 (1.13, 1.35)	0.92 (0.73, 1.15)	
1992–1998	1.40 (1.30, 1.50)	0.87 (0.67, 1.13)		1.54 (1.40, 1.69)	0.87 (0.69, 1.11)	
1999–2005	1.83 (1.71, 1.95)	0.98 (0.82, 1.18)	1.00	2.52 (2.33, 2.73)	1.19 (0.96, 1.47)	1.00
2006–2012	2.40 (2.23, 2.57)	1.25 (1.02, 1.52)	1.16 (0.95, 1.42)	3.08 (2.82, 3.37)	1.52 (1.21, 1.90)	1.20 (0.91, 1.58)

NHIS, National Health Interview Survey.

examining these relationships through an intersectional frame.[37] Intersectionality considers the simultaneous interactions among different aspects of social identities (i.e., race, class, and gender), and how these identities operate as a system of oppression and domination that (in)directly influences life experiences.[38] For example, researchers examining the intersection of gender, race, and class and diabetes risk find, as reported here, that disparities are multidimensional.[9] By examining these relationships through an intersectional frame, researchers may gain a broader understanding of the social, cultural, and political contexts that affect health.

Limitations

Diabetes was self-reported. Though not ideal, undiagnosed diabetes has decreased over time, and no differences in undiagnosed diabetes exist among racial/ethnic groups since the late 1990s, although disparities in undiagnosed diabetes by education persist.[39,40] Therefore, results likely underestimate differences by education. Owing to data limitations, analyses did not exclude gestational diabetes, nor separate out Type 1 from Type 2 diabetes. However, a sensitivity analysis eliminating probable cases of Type 1 diabetes showed larger emerging disparities for education and race/ethnicity. Immigrant health may also play an important role in racial/ethnic patterns, particularly for Asians and Hispanics. When nativity was accounted for, differences between Hispanics and Asians compared with whites were greater; therefore, the main results may underestimate differences. The National Health Interview Survey did not oversample black and Hispanic households until 1995, and did not oversample Asian households until 2006; oversampling improved precision in later years.[41] Although age was adjusted for, the oldest cohort in particular will only have healthy survivors; in that regard, results may again underestimate the importance of education and race/ethnicity disparities over time. In addition, prevalence was predicted at age 45 years. Because age, period, and cohort are not simultaneously identifiable, age was fixed. Social and environmental conditions affecting disparities likely changed over time, but the biological effect of age was likely more consistent. Although this approach required extrapolation for some period/cohort groups, this was controlled for using a parsimonious age effect (i.e., continuous). Quadratic and cubic age specifications were also investigated. However, the increased complexity produced more erratic estimates of prevalence, so the more parsimonious age effect was used in the final models.

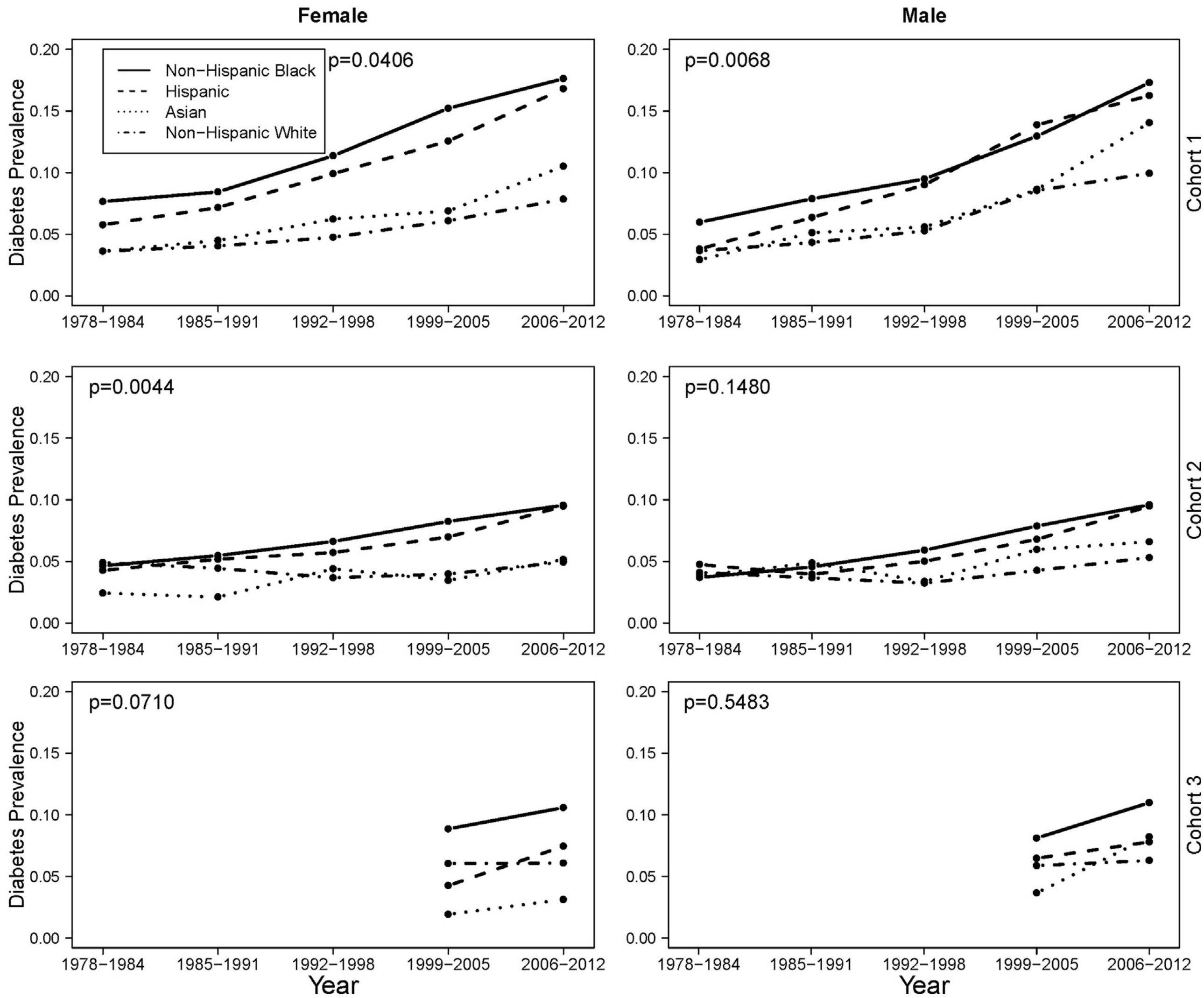

Figure 2. Disparities in diabetes by race/ethnicity according to gender and birth cohort, 1978–2012.

Conclusions

Increasing disparities in diabetes prevalence by education and race/ethnicity are evident for people born before 1971, with larger disparities for women than men. Smaller differences for later birth cohorts suggest that societal changes have enhanced socioeconomic gains for minorities and women, which may have downstream effects at reducing health disparities. Results reported here support population-level strategies that target the social determinants of health.

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NLF designed the study, supervised the analyses, and drafted and revised the manuscript. AKH assisted with data interpretation, and drafted and revised the article. Y-HW conducted the analyses and assisted in drafting and revising the article. ADL assisted with data interpretation and revising the article. ACM supervised the analyses, provided biostatistical expertise, and assisted in drafting and revising the article. All authors approved the final manuscript.

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Appendix

Supplementary data

Supplementary data associated with this article can be found at http://dx.doi.org/10.1016/j.amepre.2016.06.019.