

Secular Changes in the Age-Specific Prevalence of Diabetes Among U.S. Adults: 1988–2010

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OBJECTIVE—To examine the age-specific changes of prevalence of diabetes among U.S. adults during the past 2 decades.

RESEARCH DESIGN AND METHODS—This study included 22,586 adults sampled in three periods of the National Health and Nutrition Examination Survey (1988–1994, 1999–2004, and 2005–2010). Diabetes was defined as having self-reported diagnosed diabetes or having a fasting plasma glucose level ≥ 126 mg/dL or $\text{HbA}_{1c} \geq 6.5\%$ (48 mmol/mol).

RESULTS—The number of adults with diabetes increased by 75% from 1988–1994 to 2005–2010. After adjusting for sex, race/ethnicity, and education level, the prevalence of diabetes increased over the two decades across all age-groups. Younger adults (20–34 years of age) had the lowest absolute increase in diabetes prevalence of 1.0%, followed by middle-aged adults (35–64) at 2.7% and older adults (≥ 65) at 10.0% (all $P < 0.001$). Comparing 2005–2010 with 1988–1994, the adjusted prevalence ratios (PRs) by age-group were 2.3, 1.3, and 1.5 for younger, middle-aged, and older adults, respectively (all $P < 0.05$). After additional adjustment for body mass index (BMI), waist-to-height ratio (WHtR), or waist circumference (WC), the adjusted PR remained statistically significant only for adults ≥ 65 years of age.

CONCLUSIONS—During the past two decades, the prevalence of diabetes increased across all age-groups, but adults ≥ 65 years of age experienced the largest increase in absolute change. Obesity, as measured by BMI, WHtR, or WC, was strongly associated with the increase in diabetes prevalence, especially in adults < 65 .

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Diabetes leads to microvascular complications and increased risk of cardiovascular disease morbidity and mortality. Unfortunately, the prevalence of diabetes in the U.S. has increased over the past 2 decades (1), paralleled by increasing obesity, aging, and a combination of changes in personal lifestyle, environmental conditions, population demographic characteristics, and improved survival of persons with diabetes

(2,3). It is less clear whether the prevalence of diabetes (diagnosed and undiagnosed) has increased to the same degree across all age-groups and what role the presence of obesity plays in the prevalence of diabetes across age categories. Having a better understanding of the diabetes burden and changes over time across age categories of the U.S. population is essential for the delivery of primary and secondary prevention

interventions, planning of health services, and allocation of limited health care resources.

The U.S. National Health and Nutrition Examination Survey (NHANES) is an ongoing, national, multiple-phase, cross-sectional survey that contains information on self-reported diabetes status and laboratory measurements of blood glucose levels, thus allowing the examination of trends in both diagnosed and undiagnosed diabetes. In this study, we examined changes in the age-specific total diabetes prevalence among U.S. adults and the association of these changes with body mass index (BMI), waist-to-height ratio (WHtR), and waist circumference (WC) from 1988 to 2010 NHANES data.

RESEARCH DESIGN AND METHODS

Population and data collection

NHANES is designed to represent the U.S. noninstitutionalized civilian population. The NHANES III was conducted from 1988 to 1994. Beginning in 1999, NHANES became a yearly survey, with data released every 2 years. For this study, we divided the continuous NHANES administration into two segments of 6 years each (NHANES 1999–2004 and NHANES 2005–2010). NHANES uses a complex, multistage sample design. Participants are selected for a home interview and then invited to participate in a medical examination in a mobile center. One-half of sampled households in the survey are randomly assigned to a morning fasting blood collection. Detailed exclusion criteria, phlebotomy collection and processing, and body measurement instructions are discussed in the NHANES online documents (4).

We included all adults with self-reported diagnosed diabetes in the interview sample and adults without diagnosed diabetes selected for the morning medical examination session ($n = 23,972$). Among adults without diagnosed diabetes, we excluded those who were pregnant ($n = 637$), without a fasting

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plasma glucose (FPG) level ($n = 705$), and without a glycated hemoglobin A_{1c} (HbA_{1c}) value ($n = 44$). The final analytic sample included 22,586 adults (7,950 for 1988–1994, 6,756 for 1999–2004, and 7,880 for 2005–2010) among whom 1,441 had missing values on body weight ($n = 625$), body height ($n = 631$), and WC ($n = 1,353$). We used the full analytic sample for estimating crude or demographically adjusted prevalence, and the full analytic sample with 10 sets of multiple imputed BMI, WHtR, and WC values for BMI-, WHtR-, and WC-related analyses.

Definition of diabetes

Adults were classified as having diagnosed diabetes if they answered yes to the question, “Other than during pregnancy (for women aged ≥ 20 years), have you ever been told by a doctor or health professional that you have diabetes or sugar diabetes?” FPG level was measured with the same hexokinase enzymatic method in both the NHANES III and the continuous NHANES 1999–2010. HbA_{1c} was measured by high-performance liquid chromatography, as used in the Diabetes Control and Complications Trial (4). To make FPG levels comparable across surveys, we used crossover regression equations recommended by the National Center for Health Statistics (5); we did not use crossover equations for HbA_{1c} across surveys (6). Adults without diagnosed diabetes but with an FPG level of ≥ 126 mg/dL (7.0 mmol/L) or $HbA_{1c} \geq 6.5\%$ (48 mmol/mol) were classified as having undiagnosed diabetes. Therefore, diabetes was defined as having either diagnosed or undiagnosed diabetes.

Covariates

The anthropometric data were collected by trained health technicians in accordance with the NHANES Anthropometry Procedures Manual (7). No changes to these measurements have been made to the NHANES since 1988. All survey participants were eligible for the body measurement component. Body weight (kg) was measured on a floor scale. Height (m) was measured with a wall-mounted stadiometer. Standing WC was measured just above the uppermost lateral border of the ilium. BMI was equal to weight divided by height squared. BMI (kg/m^2) was assigned to six categories for description of characteristics as follows: underweight (< 18.5), normal weight (18.5–24.9), overweight (25.0–29.9),

obese I (30.0–34.9), obese II (35.0–39.9), and obese III (≥ 40).

Although BMI has been used to monitor overall obesity trends (8), WHtR and WC were used as surrogates of distribution of adipose tissues, especially abdominal fat deposition (9). In multivariate models, we used BMI, WHtR, and WC separately.

We analyzed age in years as a continuous and as a categorical variable (younger adults 20–34, middle-aged adults 35–64, and older adults ≥ 65) based on the results of the Joinpoint model (10). The study also included the following self-reported demographic variables: sex, race/ethnicity (non-Hispanic white, non-Hispanic black, Mexican American, other Hispanic, and other), and highest education level attained (less than high school, high school graduate, and more than high school).

Statistical methods

To account for the differences in the probability of selection and the design of the survey and to provide nationally representative estimates, all analyses were weighted to the U.S. population and analyzed with the survey module of Stata 12.1 (StataCorp LP, College Station, TX) according to NHANES analytic guidelines (11). Initial sampling weights were chosen on the basis of interview sampling weights for adults with diagnosed diabetes and morning FPG sampling weights for adults without diagnosed diabetes. Poststratification procedures involved adjustment of sampling weights so that they sum to the population sizes within each stratum, thereby reducing bias as a result of nonresponse and underrepresented groups in the population. Thus, after excluding the adults without measurements of FPG or HbA_{1c} and pregnant women among sampled adults without diagnosed diabetes, we reweighted the sample of adults without diagnosed diabetes to sum the weights of individuals to the total interview weight of all adults without diagnosed diabetes according to the provided poststrata of the U.S. noninstitutionalized population (12). There was no reweighting for adults with diagnosed diabetes; the sum of sample weights of those with and without diabetes added to the total noninstitutionalized U.S. population for each survey period.

We used multiple imputation (MI) technology with chained equations to impute the missing values for BMI ($n = 704$),

WC ($n = 1,353$), and WHtR ($n = 1,412$). The imputation model of missing values included all dependent and independent variables of logistic models plus sampling design variables (primary sampling unit, stratum). Ten sets of multiple imputed data, which is much > 100 times the largest fraction of missing information (0.0048×100), were generated to provide an adequate level of reproducibility of the MI analysis. The MI module with the survey prefix command of Stata was used for MI data analysis.

We used the Joinpoint Regression Program version 3.5.2 (National Cancer Institute, Bethesda, MD) to find age-specific inflection points (i.e., joinpoints, breakpoints, or knots) in the prevalence of diabetes by 5-year age-groups (10). On the basis of the logarithm of diabetes prevalence, Joinpoint regression analysis detected two inflection points for age (shown in parentheses) for each NHANES time period as follows: 1988–1994 (35 and 60), 1999–2004 (45 and 65), and 2005–2010 (35 and 65). Because the association of age with diabetes prevalence was not linear across the whole age range, piecewise regression incorporating inflection points for change was appropriate for fitting a model of age, as a continuous variable, and diabetes prevalence.

From the inflection points of the entire sample from 1988 to 2010, we divided the combined survey sample of 1988–2010 into three adult age-groups in years: younger (20–34), middle-aged (35–64), and older (≥ 65). We examined absolute change (i.e., prevalence difference [PD]) and relative change (i.e., prevalence ratio [PR]) in diabetes prevalence between two different survey periods for these three age-groups. The prevalence, PR, and 95% CIs were estimated by predicted margins from logistic regression models. Sex, race/ethnicity, education level, and year of survey were included in analyses as categorical variables; age and BMI were treated as either continuous or categorical in various analyses; and WHtR and WC were used as continuous variables. We tested the interaction terms of age with BMI, WHtR, and WC (all $P > 0.05$) as well as without these terms in the analyses. We calculated PD and PR for comparison between extreme survey time periods of diabetes. The Taylor series linearization method was used to estimate SEs as a default method; the delta method was used to estimate SEs of combinations of estimates, such as PR. An estimate with a two-sided $P < 0.05$ or a 95% CI

Changing diabetes prevalence

that did not include a null value was considered statistically significant.

RESULTS—Across the three NHANES time periods (1988–1994, 1999–2004, and 2005–2010), mean age, BMI, WHtR, WC, and attained education level increased (all overall $P < 0.05$) (Table 1). The crude prevalence of diabetes changed from 8.4% (95% CI 7.7–9.1%) in 1988–1994 to 12.1% (11.3–13.1%) in 2005–2010, with a relative increase of 44.8% (28.3–61.3%) between the two survey periods. There was less change of prevalence of undiagnosed diabetes ($P = 0.053$). The change in education levels reflected the demographics change in the U.S. (Table 1).

The mean (SE) values among the six BMI groups (<18.9, 18.9–24.9, 25.0–29.9, 30.0–34.9, 35.0–39.9, ≥ 40) of all survey participants were 17.6 (0.1), 22.2 (0.1), 27.3 (0.1), 32.2 (0.1), 37.2 (0.1), and 45.1 (0.4), respectively, for those

20–34 years of age; 17.5 (0.1), 22.6 (0.1), 27.4 (0.1), 32.2 (0.1), 37.2 (0.1), and 45.3 (0.3) for those 35–64; and 17.4 (0.1), 22.6 (0.1), 27.4 (0.1), 31.9 (0.1), 36.9 (0.1), and 44.0 (0.3) for those ≥ 65 . The mean values of anthropometric measurements by the three age-groups (20–34, 35–64, and ≥ 65) were 26.5 (0.1), 28.6 (0.1), and 27.8 (0.1), respectively, for BMI; 89.8 (0.3), 97.7 (0.2), and 99.3 (0.3) for WC; and 0.529 (0.002), 0.577 (0.001), and 0.603 (0.002) for WHtR.

The estimated number (in millions) of adults with diabetes grew from 14.9 (95% CI 13.3–16.4) in 1988–1994 to 26.1 (23.8–28.3) in 2005–2010, resulting in an increase of 11.2 prevalent cases (a 75.5% [52.1–98.9%] increase). Younger adults contributed 5.5% (2.5–8.4%), middle-aged adults contributed 52.9% (43.4–62.3%), and older adults contributed 41.7% (31.9–51.4%) of the increased number of cases. In each survey time period, the number of adults with

diabetes increased with age until ~60–69 years; thereafter, it decreased (Fig. 1). The gap between NHANES 1988–1994 and NHANES 2005–2010 for each age-group represents the increase in the number of prevalent diabetes cases; the largest increase of cases occurred in middle-aged and older adults.

After adjusting for sex, race/ethnicity, and education after age 34, the prevalence of diabetes was higher with each successive time period, and this increase was most pronounced among those ≥ 65 (Fig. 2). In each time period, prevalence peaked between 60 and 69 years of age.

Table 2 demonstrates the unadjusted and adjusted prevalence of diabetes by the three time periods and three age-groups. Within each age-group, both unadjusted and sociodemographically adjusted prevalence increased across time periods. By 2005–2010, versus 1988–1994, the prevalence difference of diabetes adjusted for sociodemographic characteristics was

Table 1—Characteristics of estimated U.S. adults aged ≥ 20 years by survey years: U.S. NHANES, 1988–2010

	1988–1994 (n = 7,950)	1999–2004 (n = 6,756)	2005–2010 (n = 7,880)	P value
Age (year)	44.1 (43.3–45.0)	45.0 (44.2–45.9)	46.5 (45.7–47.3)	< 0.001
Age-group (%)				
20–34 years	35.0 (32.5–37.6)	29.9 (27.8–32.1)	27.7 (26.0–29.5)	< 0.001
35–64 years	48.5 (46.2–50.8)	54.1 (52.3–56.0)	55.0 (53.3–56.7)	
≥ 65 years	16.5 (14.9–18.2)	15.9 (14.7–17.2)	17.3 (16.0–18.7)	
Sex (%)				
Male	47.6 (46.1–49.2)	47.8 (46.8–48.9)	48.2 (46.9–49.4)	0.827
Female	52.4 (50.8–53.9)	52.2 (51.1–53.2)	51.8 (50.6–53.1)	
Race/ethnicity (%)				
Non-Hispanic white	76.3 (73.0–79.4)	72.3 (68.5–75.8)	69.8 (66.2–73.2)	0.062
Non-Hispanic black	10.9 (9.5–12.6)	11.2 (9.1–13.6)	11.4 (9.7–13.3)	
Mexican American	5.1 (4.2–6.1)	7.1 (5.5–9.1)	8.3 (6.7–10.3)	
Other	7.6 (5.8–10.1)	9.4 (7.0–12.4)	10.5 (8.7–12.6)	
Highest education level (%)				
Less than high school	24.4 (22.2–26.8)	20.9 (19.3–22.6)	18.4 (16.8–20.1)	< 0.001
High school graduate	33.4 (31.4–35.3)	26.0 (24.1–28.1)	24.2 (22.6–25.9)	
More than high school	42.2 (39.5–44.9)	53.1 (50.6–55.6)	57.4 (54.7–60.1)	
WC (cm)	91.8 (91.2–92.5)	96.1 (95.4–96.7)	98.2 (97.6–98.8)	< 0.001
WHtR	0.55 (0.54–0.55)	0.57 (0.56–0.57)	0.58 (0.58–0.59)	< 0.001
BMI (kg/m ²)	26.5 (26.3–26.8)	28.0 (27.8–28.3)	28.7 (28.5–28.9)	< 0.001
BMI group (%)				
<18.9	2.0 (1.5–2.5)	1.9 (1.4–2.4)	1.8 (1.3–2.2)	< 0.001
18.9–24.9	43.5 (41.6–45.5)	34.3 (32.6–36.0)	30.7 (29.2–32.2)	
25.0–29.9	32.5 (30.9–34.0)	33.9 (31.9–35.9)	33.0 (31.6–34.4)	
30.0–34.9	14.0 (12.7–15.3)	17.8 (16.6–19.0)	19.0 (17.8–20.2)	
35.0–39.9	5.3 (4.5–6.1)	7.3 (6.4–8.1)	9.2 (8.1–10.2)	
≥ 40	2.7 (2.1–3.2)	4.9 (4.1–5.7)	6.4 (5.7–7.0)	
Undiagnosed diabetes (%)	3.3 (2.8–3.8)	3.1 (2.6–3.6)	3.9 (3.4–4.4)	0.053
Diabetes (diagnosed + undiagnosed) (%)	8.4 (7.7–9.1)	9.8 (9.1–10.6)	12.1 (11.3–13.1)	< 0.001

Data are mean or % (95% CI).

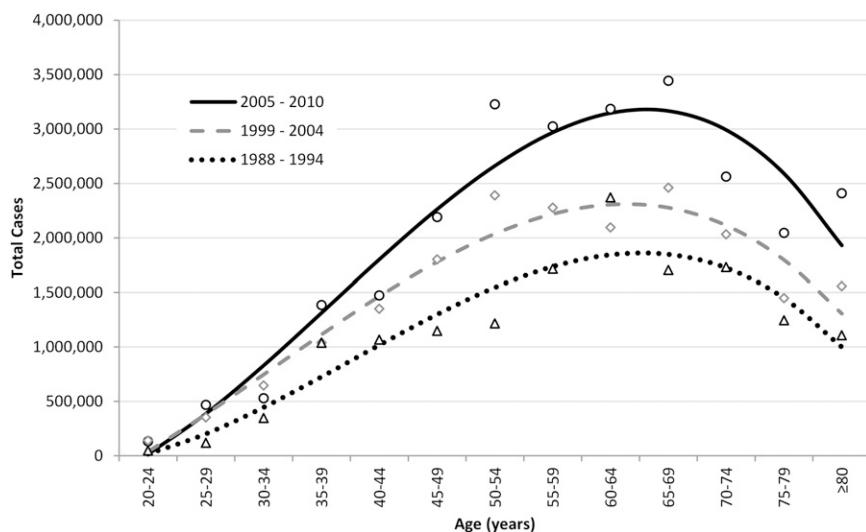


Figure 1—Total number of adults ≥ 20 years of age with diabetes by survey year: U.S. NHANES, 1988–2010. Each dot of 1988–1994 (Δ), 1999–2004 (\diamond), and 2005–2010 (\circ) represents the total number of adults with diabetes within a 5-year age-group. Each line for 1988–1994 (dotted), 1999–2004 (dashed), and 2005–2010 (solid) represents the smoothed trend line of the total number of adults with diabetes derived from a cubic polynomial regression by age-group (AgeGrp) from 1 (20–24 years) to 13 (≥ 80 years) for each time period. For 1988–1994, $-3,893.6 \cdot \text{AgeGrp}^3 + 52,915 \cdot \text{AgeGrp}^2 + 53,590 \cdot \text{AgeGrp} - 85,885$; for 1999–2004, $-3,254.7 \cdot \text{AgeGrp}^3 + 30,451 \cdot \text{AgeGrp}^2 + 274,991 \cdot \text{AgeGrp} - 263,749$; and for 2005–2010, $-5,520.1 \cdot \text{AgeGrp}^3 + 68,979 \cdot \text{AgeGrp}^2 + 204,097 \cdot \text{AgeGrp} - 253,127$.

much higher among older adults (10.0%) than among younger adults (1.0%) and middle-aged adults (2.7%) (all $P < 0.001$). The adjusted PRs (2005–2010 vs. 1988–1994) were 1.5, 1.3, and 2.3

for older, middle-aged, and younger adults, respectively; there were no statistically significant differences in the PRs of diabetes among these three age-groups. After additional adjustment for BMI,

WHtR, or WC as a continuous variable in separate models, the PDs and PRs between the first and last survey period were attenuated; especially after adjusting for WHtR or WC, both PD and PR remained statistically significant only among adults ≥ 65 years of age (Table 2).

CONCLUSIONS—From 1988 to 2010, the prevalence of diagnosed and undiagnosed diabetes increased by 45%, and the total number of persons with diabetes increased by almost 75%. Whereas diabetes prevalence increased by a similar magnitude across age-groups in relative terms, by far the greatest absolute increases in prevalence were observed in U.S. adults ~ 65 years of age. From 1988 to 2010, obesity, as measured by BMI, WHtR, or WC, explained most of the increase of diabetes prevalence among adults < 65 years of age but only part of the increase among those ≥ 65 .

Adjusting for BMI, WHtR, or WC attenuated the increase in diabetes prevalence, especially among younger and middle-aged adults, reiterating the prior observation that obesity is a major determinant of diabetes trends in the population (13). Definitions of obesity measurement remain controversial (14). Between BMI and WHtR, two major clinical indicators of obesity, BMI is the most commonly used (15), but WHtR might be a better predictor of cardiometabolic risk factors and diabetes (9,16,17). In the present study, abdominal obesity as measured by WHtR or WC accounted for slightly more variation in change of diabetes prevalence than did BMI ($P < 0.001$).

When adjusted for BMI, WHtR, or WC, the increase in prevalence of diabetes among older adults from 1988–1994 to 2005–2010 was smaller in both relative and absolute terms but still remained significantly large. There are several potential reasons for this difference between adults ≥ 65 years of age and the other two age-groups. Prevalence is a function of both incidence and mortality. The U.S. studies for this time period of investigation suggested that although incidence was increasing (18), mortality was decreasing (19). Because adults ≥ 65 years of age have higher death rates than younger people and have been shown to have significant declines in all-cause mortality over time, older adults have benefited most from decreasing death rates. Hence, these main drivers of prevalence have a differential impact by age. The roles of

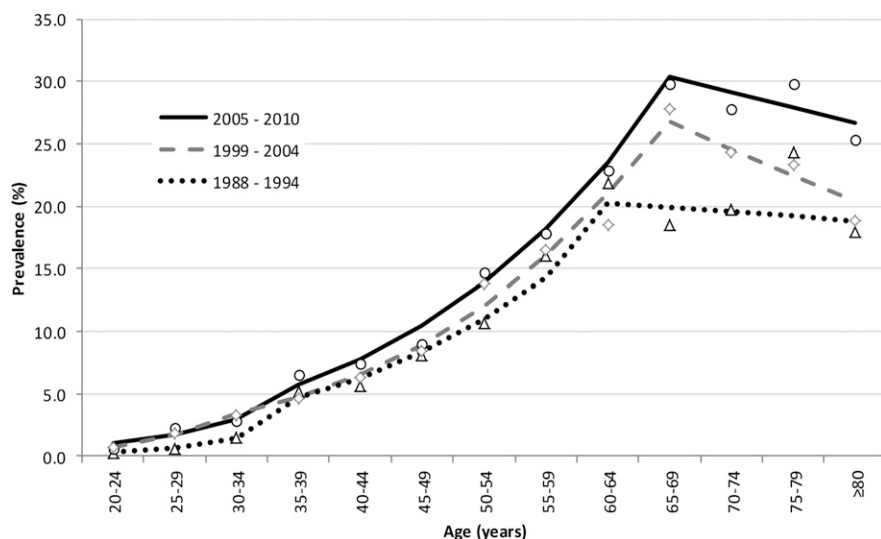


Figure 2—Age-specific unadjusted and sex-, race/ethnicity-, and education-adjusted prevalence of diabetes in adults ≥ 20 years of age by survey year: U.S. NHANES, 1988–2010. Each dot of 1988–1994 (Δ), 1999–2004 (\diamond), and 2005–2010 (\circ) represents the unadjusted prevalence within a 5-year age-group. Each line for 1988–1994 (dotted), 1999–2004 (dashed), and 2005–2010 (solid) represents the sex-, race/ethnicity-, and education-adjusted predicted prevalence of the midpoint of the 5-year age-group from piecewise regression, with age as a continuous variable.

Changing diabetes prevalence

Table 2—Prevalence of diabetes among adults aged ≥ 20 years by age-group: U.S. NHANES, 1988–2010†

	20–34 years	35–64 years	≥ 65 years	All‡
Unadjusted				
1988–1994	0.83 (0.50–1.15)	9.96 (8.59–11.32)	19.77 (17.72–21.82)	8.79 (7.99–9.59)
1999–2004	1.91 (1.14–2.68)	10.16 (9.12–11.20)	23.67 (21.12–26.21)	9.88 (9.09–10.66)
2005–2010	1.90 (1.45–2.34)	12.25 (10.92–13.59)	28.16 (25.85–30.47)	11.72 (10.87–12.57)
PD (2005–2010 vs. 1988–1994)	1.07 (0.52–1.62)***	2.29 (0.38–4.21)*	8.39 (5.30–11.48)***	2.93 (1.76–4.10)***
PR (2005–2010 vs. 1988–1994)	2.29 (1.24–3.34)*	1.23 (1.01–1.45)*	1.42 (1.24–1.61)***	1.33 (1.18–1.49)***
Adjusted for sex, race/ethnicity, education				
1988–1994	0.81 (0.49–1.14)	9.88 (8.55–11.20)	18.58 (16.58–20.59)	8.49 (7.76–9.23)
1999–2004	1.83 (1.08–2.57)	10.46 (9.44–11.48)	23.70 (21.19–26.22)	9.97 (9.20–10.74)
2005–2010	1.85 (1.41–2.29)	12.62 (11.29–13.95)	28.54 (26.26–30.82)	11.91 (11.08–12.74)
PD (2005–2010 vs. 1988–1994)	1.04 (0.49–1.58)**	2.74 (0.88–4.61)**	9.96 (7.04–12.88)*	3.42 (2.32–4.52)**
PR (2005–2010 vs. 1988–1994)	2.28 (1.21–3.32)*	1.28 (1.06–1.49)*	1.54 (1.34–1.73)*	1.40 (1.25–1.56)***
Adjusted for sex, race/ethnicity, education, BMI‡				
1988–1994	1.13 (0.69–1.58)	10.25 (8.93–11.56)	20.96 (18.88–23.04)	9.44 (8.67–10.21)
1999–2004	1.96 (1.17–2.74)	9.70 (8.86–10.54)	24.16 (21.79–26.52)	9.91 (9.18–10.64)
2005–2010	1.80 (1.39–2.21)	11.00 (9.87–12.12)	27.35 (25.16–29.55)	11.12 (10.36–11.88)
PD (2005–2010 vs. 1988–1994)	0.67 (0.06–1.28)*	0.75 (–0.97 to 2.47)	6.39 (3.56–9.23)***	1.68 (0.62–2.73)**
PR (2005–2010 vs. 1988–1994)	1.59 (0.86–2.32)	1.07 (0.90–1.25)	1.30 (1.15–1.46)***	1.18 (1.06–1.30)**
Adjusted for sex, race/ethnicity, education, WC‡				
1988–1994	1.39 (0.85–1.93)	10.52 (9.18–11.85)	19.17 (17.21–21.14)	9.83 (9.05–10.60)
1999–2004	2.17 (1.29–3.05)	9.58 (8.74–10.43)	21.30 (19.10–23.50)	9.90 (9.15–10.65)
2005–2010	1.96 (1.51–2.42)	10.52 (9.44–11.61)	23.86 (21.92–25.81)	10.84 (10.08–11.59)
PD (2005–2010 vs. 1988–1994)	0.57 (–0.14 to 1.28)	0.00 (–1.72 to 1.73)	4.69 (2.15–7.23)***	1.01 (–0.04 to 2.06)
PR (2005–2010 vs. 1988–1994)	1.41 (0.77–2.05)	1.00 (0.84–1.16)	1.24 (1.09–1.39)**	1.10 (0.99–1.22)
Adjusted for sex, race/ethnicity, education, WHtR‡				
1988–1994	1.47 (0.90–2.04)	10.53 (9.21–11.85)	17.73 (15.95–19.52)	9.77 (8.99–10.55)
1999–2004	2.34 (1.41–3.28)	9.73 (8.87–10.59)	19.82 (17.63–21.91)	9.94 (9.20–10.68)
2005–2010	2.05 (1.57–2.52)	10.66 (9.59–11.73)	22.18 (20.31–24.05)	10.84 (10.10–11.58)
PD (2005–2010 vs. 1988–1994)	0.58 (–0.17 to 1.33)	0.13 (–1.57 to 1.82)	4.45 (2.09–6.81)***	1.07 (0.03–2.11)*
PR (2005–2010 vs. 1988–1994)	1.39 (0.76–2.03)	1.01 (0.85–1.17)	1.25 (1.10–1.40)**	1.11 (1.00–1.22)

Data are prevalence (%), prevalence difference (PD, %), or prevalence ratio (PR) (95% CI). †1,440 adults without BMI, WC, or WHtR values used 10 sets of MI values. ‡The prevalence for all adults ≥ 20 years of age had additional adjustments of three age-groups. * $P < 0.05$. ** $P < 0.01$. *** $P < 0.001$.

incidence and mortality in diabetes on the increasing prevalence of diabetes may still be controversial. A Canadian population-based study demonstrated that from 1995 to 2005, the increasing prevalence of self-reported diabetes was due to both increasing incidence and decreasing mortality (20). The Danish National Diabetes Register reported that from 1995 to 2006, mortality rates in the diabetic population decreased 4% per year compared with 2% per year in the nondiabetic population (21). An Italian population-based study showed that from 2000 to 2007, the incidence rate of diabetes did not change, but mortality decreased yearly by 3.0% (3). A Finnish study showed that in young adults who were 15–39 years of age between 1992 and 2001, the incidence of

both type 1 and type 2 diabetes increased on average by 3.9 and 4.3%, respectively (22). In each of these three reports, undiagnosed diabetes could not be identified; hence, the true prevalence and incidence of total diabetes would have been underestimated.

In addition, the unequal change of exposure level and the effect of mortality and incident diabetes risk factors by age-group may also explain this difference in the secular change of prevalence between age-groups. Generally, adults gain weight until about age 60, followed by weight loss (23), which when unintentional, may be a significant indicator of frailty among older adults (24). Therefore, it is plausible that obesity among older adults may not be as strong a risk factor as among

younger adults according to the present study. On the other hand, the presence and treatment of comorbid conditions may increase diabetes risk among older adults. For example, the Women's Health Initiative and other studies reported an increased risk of diabetes among persons taking statins (25,26). Additionally, medications used to treat arthritis or chronic obstructive pulmonary disease could increase the patient's risk of insulin resistance or diabetes (27,28). However, we need to cautiously interpret the correlation of the three anthropometric obesity measures with diabetes, especially among older frail adults. As we expected, adults 35–64 years of age had the highest mean value of BMI, whereas those ≥ 65 had the highest mean values of WC and WHtR. These surrogates

cannot represent the actual adiposity level exactly, which could be a potential source of residual confounding.

In the present study, the PR of diabetes between the first and last time period of the survey became statistically nonsignificant after adjusting for BMI, WHtR, or WC for younger and middle-aged adults only. Recent analyses of data from locally and nationally representative surveys suggested that obesity prevalence trends measured by BMI reach a plateau (15,29,30), which raises questions about whether a similar plateau might be anticipated for the prevalence of diabetes. Future research as well as close surveillance monitoring of incident and prevalent diabetes that follow population-level plateaus in obesity prevalence may be particularly helpful in explaining further the age-specific differences in increasingly prevalent diabetes over time.

Misclassification might occur as a result of recall error or change over time in diagnostic criteria for diabetes. In 1997, the American Diabetes Association (ADA) lowered the FPG level for diagnosing diabetes from 140 to 126 mg/L and encouraged the use of FPG as the main diagnostic test rather than the long-time standard oral glucose tolerance test (31). In 2009, the ADA recommended the use of HbA_{1c} for the diagnosis of diabetes (32). The increase in the prevalence of diagnosed diabetes over the three time periods may be partly a result of the changes in the diagnostic criteria and increase in screening. The frequency of glucose testing might also affect the prevalence of self-reported diabetes. However, in the present study, we used both diagnosed and undiagnosed diabetes, and this approach may have minimized the impact of changes of diagnostic criteria and frequency of glucose testing.

This study has a few limitations. First, we could not distinguish between type 1 and type 2 diabetes. Second, the study was inherently limited by the nature of cross-sectional national surveys; therefore, we were unable to show a causal relationship and to evaluate the lifetime effect of obesity exposure. Finally, although self-reported diabetes is a valid measure of diabetes presence (33–35), we relied on a one-time measurement of FPG level or HbA_{1c} to define undiagnosed diabetes, whereas retesting is suggested for diabetes diagnosis in clinical settings. This could have caused some degree of discrepancy with clinical settings, but there are no reasons to believe that the

misclassification rate of diabetes between clinical settings and surveys would change over time. We speculate that the incremental change in the prevalence of diabetes in the U.S. population relates to the decrease in fatality among diabetic individuals, the aging of the population, and the increase in incident diabetes (36).

During the past two decades, the prevalence of diabetes increased across all ages, with the most prominent change among adults ≥ 65 , which accounts for 42% of the growth in number of prevalent cases. Obesity, as measured by BMI and WHtR, is strongly associated with the increase in diabetes prevalence, especially in younger and middle-aged adults, and is generally congruent with the existing obesity epidemic as a driving force for the increase in diabetes prevalence for all age-groups and reinforces the call for prevention efforts. The increased prevalence among older adults, likely the result of an aging population and successes in diabetes control (37), underscores the urgent need for planning and delivering quality health care for this growing segment of the population (38).

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