Obesity Progression Between Young Adulthood and Midlife and Incident Diabetes: A Retrospective Cohort Study of U.S. Adults

Andrew Stokes,[1] Jason M. Collins,[1] Bethany F. Grant,[2] Robin F. Scamuffa,[2] Chia-Wen Hsiao,[2] Stephen S. Johnston,[3] Eric M. Ammann,[3] JoAnn E. Manson,[4] and Samuel H. Preston[5]

OBJECTIVE

Understanding how changes in weight over the life course shape risk for diabetes is critical for the prevention of diabetes. Using data from the National Health and Nutrition Examination Survey (NHANES), we investigated the association between self-reported weight change from young adulthood to midlife and incident diabetes.

RESEARCH DESIGN AND METHODS

We categorized individuals into four weight-change groups: those who remained nonobese (stable nonobese), those who moved from an obese BMI to a nonobese BMI (losing), those who moved from a nonobese BMI to an obese BMI (gaining), and those who remained obese (stable obese). Diabetes status was determined by self-report of a prior diagnosis, and age at diagnosis was used to establish time of diabetes onset. Hazard ratios (HRs) relating weight change to incident diabetes over 10 years of follow-up were calculated using Cox models adjusting for covariates.

RESULTS

Those who were obese and lost weight exhibited a significantly lower risk (HR 0.33; 95% CI 0.14, 0.76) of diabetes compared with those with stable obesity. We also observed lower risk among those who were stable nonobese (HR 0.22; 95% CI 0.18, 0.28) and those in the gaining category (HR 0.70; 95% CI 0.57, 0.87). Further, there was evidence of an increased incidence of diabetes among obese individuals who lost weight compared with individuals who were stable nonobese; however, weight loss was rare, and the association was not statistically significant. If those who were obese had become nonobese during the 10-year period, we estimate that 9.1% (95% CI 5.3, 12.8) of observed diabetes cases could have been averted, and if the population had maintained a normal BMI during the period, 64.2% (95% CI 59.4, 68.3) of cases could have been averted.

CONCLUSIONS

The findings from this study underscore the importance of population-level approaches to the prevention and treatment of obesity across the life course of individuals.

[1]Department of Global Health, Boston University School of Public Health, Boston, MA
[2]Ethicon, Cincinnati, OH
[3]Johnson & Johnson, New Brunswick, NJ
[4]Division of Preventive Medicine, Brigham and Women's Hospital, and Department of Epidemiology, Harvard T.H. Chan School of Public Health, Boston, MA
[5]Department of Sociology and Population Studies Center, University of Pennsylvania, Philadelphia, PA

Corresponding author: Andrew Stokes, acstokes@bu.edu.

The incidence and prevalence of diabetes among adults in the U.S. has risen during the past 30 years (1,2). In 1988, 9.8% of the adult population was living with diabetes; by 2014, this prevalence had grown to 12.3% (2). These increases have important implications for U.S. mortality (3), morbidity (4), disability (5), and health care expenditures

(6). A recent study found that diabetes was responsible for a higher volume of personal medical expenditures than any other medical condition (6). A 2017 study estimated that nearly one-fifth of deaths among obese adults were attributable to diabetes (3).

A rising trend in the prevalence of obesity can account for most of the increase in diabetes between 1976 and 1980 and between 2007 and 2010 (2,7). The relationship between obesity and diabetes has been established by observational studies of BMI and diabetes status (8–11) and supported further by research into the physiologic link between body fat, insulin resistance, and type 2 diabetes (12). Alongside the effects of prevalent obesity on diabetes, prospective cohort studies have shown weight change over the life course can also have profound effects on the risk of diabetes (11,13–18). A recent study using data from the Nurses' Health Study (NHS) and the Health Professionals Follow-Up Study (HPFS) examined major health outcomes associated with weight change in adulthood. Of the outcomes considered, type 2 diabetes showed the strongest associations with weight change. A general reduction in risk was observed for weight loss in both cohorts, whereas weight gain was associated with an increased incidence of diabetes commensurate with the absolute change (11). These associations have also been observed in studies outside the U.S. and in studies focused on ethnic differences (17,19–21).

With few exceptions, investigations of the relationship between weight change and the incidence of diabetes have been limited to samples that are not nationally representative. Exceptions include two studies using the National Health and Nutrition Examination Survey (NHANES) I Epidemiologic Follow-Up Study, 1971–1992 (22,23), and two studies that monitored adolescents for one to two decades (24,25). Recent national data on the association between weight change and incident diabetes are not available, largely because the two major national cohorts with ongoing data collection, the NHANES and the National Health Interview Survey (NHIS), have not prospectively monitored individuals for incident disease status.

One opportunity not yet explored is to treat a national probability sample as a retrospective longitudinal study. NHANES has routinely asked questions about weight histories, including weight at age 25 and weight 10 years before the survey, alongside weight at the survey. It has also asked questions about the history of diabetes diagnosis. Leveraging these data provides an opportunity to investigate the relationship between weight histories, in the form of weight at age 25 and weight 10 years before the survey, and the incidence of diabetes during 10 years of follow-up before the survey.

We used this approach to examine the relationship between weight change in adulthood and diabetes incidence. We sought to determine whether obese individuals who lost weight were *1*) at a reduced risk of diabetes relative to individuals with stable obesity (the "risk reduction" hypothesis), and *2*) at an increased risk of diabetes relative to individuals who maintained a nonobese BMI over time (the "residual risk" hypothesis). This latter hypothesis predicts that individuals who have been obese are at higher risk of developing diabetes than those who have never been obese.

RESEARCH DESIGN AND METHODS

Data for this study were drawn from the NHANES, a nationally representative sample of U.S. adults. NHANES was collected periodically from the noninstitutionalized U.S. population before 1999 and continuously thereafter. Interviews were conducted at individuals' homes, and laboratory and physical examinations were performed by trained technicians using mobile examination centers (26). Institutional Review Board approval was not required for this study because the investigation was based on secondary analyses of publicly available, deidentified data.

We incorporated data on adults aged 40–74 at examination from both the NHANES III (1988–1994) and continuous NHANES (1999–2014). Self-reported weight change was assessed by participant recall of weight at age 25 and 10 years before the NHANES survey. Incident diabetes was determined from respondents affirming that a health care provider had indicated a diagnosis of diabetes. The reported age at diagnosis was used to establish the time of diabetes onset. The study design is visually depicted in Supplementary Fig. 1.

Those who reported a date of onset that was before the initiation of follow-up were considered prevalent diabetes cases

and were excluded. An individual who did not report a diagnosis of diabetes but recorded an $HbA_{1c} \geq 6.5\%$ (48 mmol/mol) at examination was considered to have undiagnosed diabetes and excluded from survival analyses. Diabetes type (type 1 or type 2) was not able to be determined. We also excluded individuals missing observations for BMI, HbA_{1c}, family history of diabetes, or education. After exclusions, a sample size of 21,554 individuals remained for analysis.

Respondents were asked to recall weight at age 25 and weight 10 years before their age at survey. Measured height at the examination was used to calculate BMI, unless the participant was 50 years or older at the time of the survey. In this case, reported height at 25 was used to calculate BMI at 25, and measured height at the examination was used to calculate BMI at 10 years before the examination. Reported height at age 25 was incorporated to account for the possibility of height decline with age. To calculate BMI, weight was converted into kilograms and height was converted to meters. BMI values for both time points were categorized into underweight, normal weight, overweight, obese, and obese II.

BMI change categories were then generated to capture weight change over the life course of an individual. The primary analysis used four BMI change categories based on BMI (kg/m^2) at age 25 and on BMI 10 years prior to the survey: stable nonobese ($BMI_{age\ 25} < 30$ and $BMI_{10\ years\ prior} < 30$), losing ($BMI_{age\ 25} \geq 30$ and $BMI_{10\ years\ prior} < 30$), gaining ($BMI_{age\ 25} < 30$ and $BMI_{10\ years\ prior} \geq 30$), and stable obese ($BMI_{age\ 25} \geq 30$ and $BMI_{10\ years\ prior} \geq 30$).

Survival Analysis

Two hypotheses, which are referred to as the "risk reduction" hypothesis and the "residual risk" hypothesis, were tested using Cox proportional hazard models predicting the rate of incident diabetes across the four BMI change categories over the time period. A total of 1,877 cases of incident diabetes were recorded during follow-up, with individuals contributing 208,061 person-years, or a rate of 9.02 cases per 1,000 person-years. Models included adjustment for race/ethnicity, sex, educational attainment, family history of diabetes, and age at the start of follow-up. To test the risk reduction hypothesis, the stable obese weight-change category

was used as the reference category to which all other weight-change categories were compared. To test the residual risk hypothesis, the same model was used, with the stable nonobese category chosen as the reference category. To determine whether the time period studied had an effect on the results observed, a sensitivity analysis was run excluding NHANES 1988–1994 data (Supplementary Table 2). No meaningful differences were observed, so the primary model with both time periods (1988–1994 and 1999–2014) was preserved.

A secondary analysis was implemented in which those who maintained a normal BMI during the time period were treated as a separate category of BMI change. In doing so, those originally in the stable nonobese category were categorized into two new groups: *1*) "stable normal" ($BMI_{age\ 25} < 25$ and $BMI_{10\ years\ prior} < 25$), as described; and *2*) "max overweight," comprising those who were never obese but were overweight ($BMI \geq 25$ and $BMI < 30$) at least once at age 25 or 10 years before the survey. Stable normal was used as the reference category for this model.

Hypothetical Scenarios
Estimates of the percentage of diabetes cases that could be averted under four hypothetical scenarios were calculated using the following equation for the population-attributable fraction (PAF):

$$PAF = \sum_{i=0}^{k} pd_i \left(\frac{HR_i - 1}{HR_i} \right)$$

where pd_i is the proportion of total incident cases in the sample observed in the *i*th BMI trajectory category, and HR_i is the hazard ratio associated with that BMI trajectory (27). To calculate the attributable fractions, we treated individuals with the BMI trajectory exposure of interest as if they were hypothetically part of a different level of exposure based on the respective scenario. In doing so, the PAFs represent the fraction of cases that would be eliminated if people with a particular BMI trajectory were redistributed to another trajectory and experienced the same relative risks as individuals in that new trajectory.

For clarity, we referred to the four scenarios used in PAF calculations with the names *1*) "weight loss," *2*) "weight maintenance," *3*) "partial prevention,"

and *4*) "comprehensive prevention" (Table 3):

1. The weight loss scenario is a hypothetical scenario in which individuals who were obese in young adulthood lost down to a nonobese BMI at midlife.

2. The weight maintenance scenario estimates what would have happened if individuals who gained weight—from nonobese in young adulthood to obese by midlife—had not gained this weight, but instead had maintained their nonobese BMI during the time period.

3. Partial prevention asks what if the total population had a nonobese BMI from young-adulthood that was maintained through midlife.

4. Comprehensive prevention examines what would happen if the total population had a normal BMI from young adulthood through midlife. The PAF calculation for this scenario used estimates generated for the stable normal category from our secondary analysis, because those who were stable normal approximate the ideal weight-change category for the period from young adulthood to midlife.

Examination sample weights were used for all estimates and analyses. Stata 14 software (StataCorp) was used for statistical analysis and data management. The Stata package punafcc was used to generate PAFs (28). A two-sided *P* value of <0.05 was used to determine statistical significance.

RESULTS
Table 1 reports characteristics of the sample with weighted estimates and unweighted sample sizes stratified by weight-change category. The mean age of the sample was 43.8 years at baseline, and 50.2% were men. The mean BMI was 23.6 kg/m^2 at age 25, 26.6 kg/m^2 at 10 years before the survey, and 27.8 kg/m^2 at the end of follow-up. The study sample was 78.9% non-Hispanic white, 9.0% non-Hispanic black, and 8.5% Hispanic. Of the individuals in the study, 52.6% reported having more than a high school education. A family history of diabetes was reported by 43.5%, ranging from 54.9% among those who lost weight to 41.1% among those who maintained a stable nonobese BMI.

Regarding life-course weight change, 79.4% of the population was stable

nonobese, 1.1% reported losing from an obese BMI to a nonobese BMI, 14.6% reported gaining weight, and 4.9% remained stable obese between young adulthood and midlife.

Survival Analysis
Figure 1 shows cumulative incidence curves by time in study for each weight change group. Compared with stable obese individuals, those who maintained a stable nonobese BMI from young adulthood through midlife had the lowest risk (HR 0.22; 95% CI 0.18, 0.28) of developing diabetes during the 10 years of follow-up (Table 2). Those who reported losing from an obese BMI to a nonobese BMI over their life course had 0.33 (95% CI 0.14, 0.76) times the risk of developing diabetes as those who maintained a stable obese BMI during the time period. Individuals who reported gaining from a nonobese BMI to an obese BMI had 0.70 (95% CI 0.57, 0.87) times the risk of developing incident diabetes as those who were stable obese. When the reference category was changed to the stable nonobese (Table 2), no significant difference (HR 1.47; 95% CI 0.65, 3.36) was observed in the risk of onset diabetes between those reporting losing from an obese BMI to a nonobese BMI and those who maintained a stable nonobese BMI over the time period.

In the secondary analysis (Supplementary Table 1), we observed that those who gained from a nonobese to an obese BMI during the period had 5.77 (95% CI 4.63, 7.18) times the rate of incident diabetes compared with those who remained in the normal BMI range. Those who remained obese at both times had 8.07 (95% CI 6.28, 10.38) times the rate compared with the stable normal category. Finally, those who were overweight at either time point (max overweight) showed increased incidence of diabetes (HR 2.65; 95% CI 2.12, 3.31) relative to those who were stable normal weight.

Hypothetical Scenarios
PAFs were calculated from the four scenarios that are outlined in the RESEARCH DESIGN AND METHODS and represented in Table 3. In the weight loss scenario, if those who were obese lost to a nonobese BMI, 9.1% (95% CI 5.3, 12.8) of observed diabetes cases could have been averted (Table 3). In the weight maintenance scenario, if those who gained weight

Table 1—Life course obesity progression and onset diabetes in adults ages 40–75*

	Stable nonobese[b] n (%) = 16,454 (79.4)	Gaining[b] n (%) = 3,719 (14.6)	Stable obese[b] n (%) = 1,154 (4.9)	Losing[b] n (%) = 227 (1.1)	Total N = 21,554
Age (years), μ (95% CI)[a]	43.6 (43.2, 43.9)	46.2 (45.7, 46.7)	40.9 (40.1, 41.7)	43.7 (42.2, 45.1)	43.8 (43.5, 44.1)
Sex, n (%)					
Male	8,483 (49.7)	1,836 (50.8)	601 (54.8)	133 (62.6)	11,053 (50.2)
Female	7,971 (50.3)	1,883 (49.2)	553 (45.2)	94 (37.4)	10,501 (49.8)
Race, n (%)					
Non-Hispanic					
White	8,176 (79.4)	1,757 (77.5)	517 (76.5)	114 (78.3)	10,554 (78.9)
Black	3,388 (8.3)	923 (11.3)	348 (13.9)	45 (7.8)	4,704 (9.0)
Hispanic	4,008 (8.4)	951 (9.0)	260 (7.7)	63 (8.4)	5,282 (8.5)
Other	882 (3.9)	88 (2.2)	29 (1.9)	5 (5.4)	1,004 (3.5)
Education, n (%)					
Less than high school	4,687 (18.2)	1,193 (22.0)	357 (19.6)	81 (24.2)	6,318 (18.9)
High school or equivalent	4,161 (28.0)	956 (30.0)	299 (31.4)	56 (29.4)	5,472 (28.5)
More than high school	7,606 (53.8)	1,570 (48.0)	498 (48.9)	90 (46.4)	9,764 (52.6)
BMI (kg/m^2), μ (95% CI)					
At age 25[c]	22.5 (22.4, 22.6)	25.2 (25.0, 25.3)	34.4 (34.1, 34.8)	33.4 (32.5, 34.2)	23.6 (23.5, 23.7)
10 years prior[d]	24.6 (24.5, 24.7)	33.7 (33.5, 33.9)	38.0 (37.4, 38.6)	26.6 (26.0, 27.3)	26.6 (26.5, 26.7)
At survey[e]	26.2 (26.1, 26.3)	33.2 (33.0, 33.4)	37.3 (36.7, 37.8)	29.5 (28.4, 30.6)	27.8 (27.7, 28.0)
Family history of diabetes[f]	7,058 (41.1)	1,950 (51.9)	672 (54.7)	114 (54.9)	9,794 (43.5)

*Source: NHANES III (1988–1994) and NHANES (1999–2014); sample weighted estimates. [a]Age at start of follow-up. [b]Stable nonobese: BMI$_{age\ 25}$ <30 and BMI$_{10\ years\ prior}$ <30; losing: BMI$_{age\ 25}$ ≥30 and BMI$_{10\ years\ prior}$ <30; gaining: BMI$_{age\ 25}$ <30 and BMI$_{10\ years\ prior}$ ≥30; stable obese: BMI$_{age\ 25}$ ≥30 and BMI$_{10\ years\ prior}$ ≥30. [c]Self-reported BMI at age 25. [d]Self-reported BMI 10 years before survey. [e]Self-reported BMI at survey. [f]Self-reported family history of diabetes.

during the period had not gained weight, 23.5% (95% CI 21.8, 25.1) of the observed cases would have been averted. In the partial prevention scenario, maintaining a nonobese BMI between young adulthood and midlife would have prevented 34.5% (95% CI 32.4, 36.6) of diabetes cases. In the comprehensive prevention scenario, if the total population had a normal BMI from young adulthood through midlife, 64.2% (95% CI 59.4, 68.3) of cases would have been averted.

CONCLUSIONS

The category with the highest risk of incident diabetes consisted of people who were obese during both young adulthood

and midlife. Being nonobese at age 25 or 10 years before the survey was advantageous relative to remaining obese throughout this period. Those who lost from an obese BMI to a nonobese BMI benefited from a reduction in the rate of incident diabetes compared with those who remained obese (HR 0.33; 95% CI 0.14, 0.76). Likewise, those who had been nonobese at age 25 but became obese by a period 10 years before the survey had lower risks than those who were obese throughout the period (HR 0.70; 95% CI 0.57, 0.87) (Table 2). These results are consistent with other evidence based on nonnational sources that duration of obesity predicts the incidence of diabetes (29).

We found some evidence in support of the residual risk hypothesis. Those who were obese at age 25 and became nonobese had an HR of 1.47 (95% CI 0.65, 3.36) relative to those who maintained a nonobese BMI throughout the period. However, the 95% CIs were wide because weight loss from an obese BMI to a nonobese BMI was rare, representing only 1.1% of the total population (Table 1).

We used our estimates of the risks associated with weight change to explore the potential effect of weight loss interventions and prevention initiatives targeting weight gain. The weight loss scenario was designed to approximate a comprehensive weight loss intervention targeting individuals with obesity at age 25. We estimated that if all those who were obese at age 25 lost to a nonobese BMI by midlife, 9.1% (95% CI 5.3, 12.8) of observed incident cases of diabetes could be averted. Preventing weight gain in the population after age 25, represented by the weight maintenance scenario, was associated with a 23.5% (95% CI 21.8, 25.1) reduction in diabetes cases in the population. In total, we found that 64.2% (95% CI 59.4, 68.3) of diabetes cases during this time period could be averted if all individuals in the population maintained a weight in the normal range between early adulthood and midlife.

Table 2—Risk reduction and residual risk: HRs for obesity progression and incident diabetes*

Hypothesis	BMI change	HR[†]	95% CI	P value
Risk reduction	Stable nonobese	0.22	0.18, 0 0.28	<0.001
	Losing	0.33	0.14, 0.76	0.009
	Gaining	0.70	0.57, 0.87	0.002
	Stable obese	1.00 (Ref)	—	—
Residual risk	Stable obese	4.45	3.59, 5.51	<0.0001
	Gaining	3.13	2.69, 3.63	<0.0001
	Losing	1.47	0.65, 3.36	0.355
	Stable nonobese	1.00 (Ref)	—	—

*Source: NHANES III (1988–1994) and NHANES (1999–2014). [†]Adjusted for education, race, sex, family history of diabetes, and age at start time.
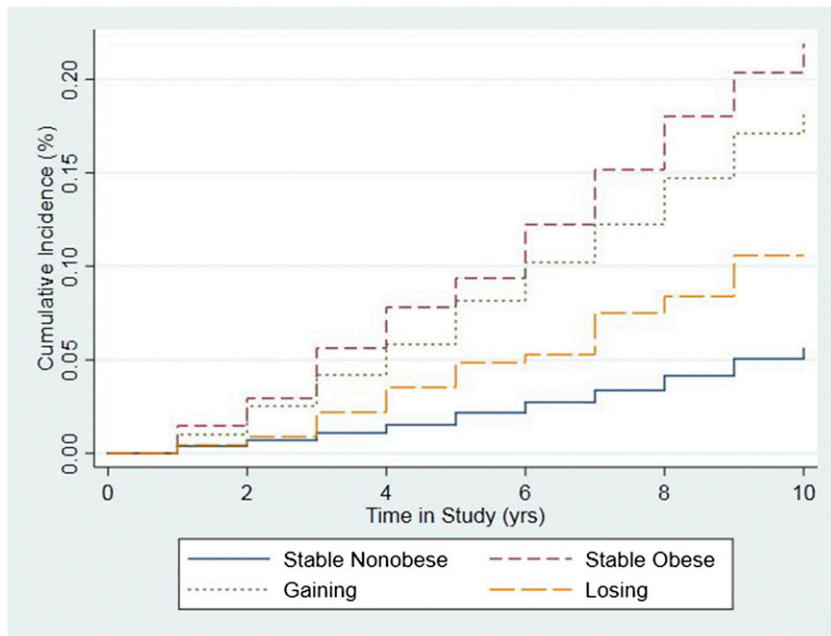
across the life course. BMI observations at two earlier points in the life course were used to study the incidence of diabetes during a subsequent decade of exposure. Although many other studies have looked at weight change and incident diabetes, the nonrepresentative samples used in previous studies may not be generalizable because covariate distributions can differ, with some of those covariates serving as key effect modifiers of the association between weight change and incident diabetes. As a nationally representative survey, results using NHANES are more broadly generalizable than those from other cohorts like the NHS and the HPFS, which rely on data gathered in disproportionately white populations. Although the focus of NHANES is not on ethnic differences, our findings agree with those from multiethnic studies that more fully consider racial/ethnic differences such the Multiethnic Cohort Study performed in Hawaii and Los Angeles (17,30).

Figure 1—Cumulative incidence curve for the Cox model. Source: NHANES III (1988–1994) and NHANES (1999–2014). Stable nonobese: $BMI_{age\ 25} < 30\ kg/m^2$ and $BMI_{10\ years\ prior} < 30$; losing: $BMI_{age\ 25} \geq 30$ and $BMI_{10\ years\ prior} < 30$; gaining: $BMI_{age\ 25} < 30$ and $BMI_{10\ years\ prior} \geq 30$; stable obese: $BMI_{age\ 25} \geq 30$ and $BMI_{10\ years\ prior} \geq 30$.

Our results are consistent with several previous studies that examined weight change in adulthood (11,13,14,16–18). A recent study by Zheng et al. (11) found that increasing levels of weight gain were strongly associated with incident diabetes and that moderate weight loss was associated with reduced risk. Among women in the NHS, those who gained ≥20 kg of weight from early to middle adulthood showed 10.51 times the incidence rate of diabetes compared with those who lost or gained <2.5 kg (11). Similar effects were observed among men in the HPFS. We also found a similar effect from weight gain in our study, with those gaining into the obese category having 5.77 times the incidence rate of diabetes

compared with those who maintained a normal BMI (Supplementary Table 2). In an earlier analysis of the HPFS, an elevated BMI of ≥23 at age 21 was associated with diabetes onset later in adulthood, and a weight gain of ≥2.5 kg in adulthood was associated with an elevated risk of diabetes (13). These findings support the results from our study that those who lost weight during the study period did not fully reduce their risk of diabetes relative to those who maintained a nonobese or normal BMI.

Our study had several strengths. Using a retrospective cohort design, we were able to take advantage of a large, nationally representative cohort of U.S. adults to estimate associations between weight change and incident diabetes

An additional strength of the current study is that by conditioning on survival in the 10-year period between the second weight observation and baseline, any effects of illness-associated weight loss are likely to be substantially mitigated. The prior literature has generally not accounted for potential reverse causal pathways in investigating associations between weight change and incident outcomes (15–18). Some prior studies conditioned on survival for several years subsequent to assessment of weight status; however, there is evidence that illness-induced weight loss can occur with substantial lag times, suggesting that the measures taken in those studies were not sufficient to address the bias (31,32). Finally, by updating height data in our BMI calculations for older adults, we accounted for

Table 3—Population attributable fractions for population counterfactuals*

Scenario	PAF (%)	95% CI	Definition
1 Weight loss†‡	9.1	5.3, 12.8	If those who maintained an obese BMI, instead lost to a nonobese BMI between young adulthood and midlife
2 Weight maintenance†‡	23.5	21.8, 25.1	If those who gained weight, instead remained nonobese between young adulthood and midlife
3 Partial prevention‡	34.5	32.4, 36.6	If the total population had a nonobese BMI from young adulthood that was maintained through midlife
4 Comprehensive prevention§	64.2	59.4, 68.3	If the total population had a normal BMI from young adulthood through midlife

*Source: NHANES III (1988–1994) and NHANES (1999–2014). †Weight loss and weight maintenance represent fraction of cases prevented if one trajectory's risk were substituted for another, as described. ‡HR_i from Table 2. §HR_i from Supplementary Table 1.

any age-associated height loss that these individuals might have experienced.

This study had several limitations. First was our reliance on historic, self-reported weight measures. Although prior studies have shown self-reported weight is a strongly correlated predictor of actual weight (33), our use of historic self-report likely introduced error. However, evidence has demonstrated that historic weight measures can have high agreement with measured weight (34,35).

A second limitation was the use of recalled height to account for age-related height decline. Error could have been introduced when relying on self-reported height at age 25 in adults older than age 50 to calculate BMI at age 25 (36). Evidence suggests a relatively high level of agreement between height recall and historically measured height among older adults (37,38). A validation study in an older population ($\mu_{age} = 61.0 \pm 2.9$ years) in which recalled historic height was compared with measured historic height at age 45 demonstrated high correlation ($r = 0.944$) (37). Furthermore, Must et al. (38) found high correlation between measured height in high school and recalled height among an elderly population aged 71–76 years. In light of the evidence, we opted to use recalled height with the older population in an attempt to mitigate underestimation of height due to age-related height decline.

A third limitation is that we could not adjust for physical activity or diet because recall data on these variables were not collected. The results may thus partly reflect the effects of physical activity and dietary factors over the life course.

Another limitation is that members of the relevant cohorts who had died before the survey were not represented in the retrospective data set. Their experience might have differed from that of survivors in ways that affected the estimated relationship between obesity and diabetes. Furthermore, our report relied on self-reported data on diabetes status, which may have missed people who had not been diagnosed with the condition. However, recent evidence from the NHANES indicates that the proportion of people with undiagnosed diabetes is relatively low in the U.S.; thus, relying on self-reported diabetes status is unlikely to represent a major source of bias in estimates (39). We further accounted for undiagnosed cases in the analysis by excluding individuals with HbA$_{1c}$ values that exceeded the relevant threshold. As a result, we expect any error introduced from the use of self-report diagnosis to be small. Finally, the study did not account for weight cycling between reported weight measures or distinguish subtypes of diabetes.

Conclusion

In testing the risk reduction hypothesis, we found those who lost weight between young adulthood and midlife showed statistically significant reductions in risk for diabetes onset compared with those who remained obese. When considering the residual risk hypothesis, those who had been obese at age 25 but had subsequently become nonobese had a higher risk of developing diabetes than those who remained nonobese throughout their life course, but the difference was not statistically significant. A large percentage of the observed diabetes cases could have been averted with effective intervention and prevention efforts in young adulthood.

This study used a novel application of NHANES survey data to explore the associations and implications of weight change from young adulthood through midlife and demonstrated the viability of using historic self-reported weight data for longitudinal analyses. We show that remaining or becoming obese raises the risk of incident diabetes relative to remaining nonobese. The findings from a national sample underscore the importance of developing policies and programs that reduce the prevalence of obesity.

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