Timing and Duration of Obesity in Relation to Diabetes

Findings from an ethnically diverse, nationally representative sample

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OBJECTIVE—The influence on diabetes of the timing and duration of obesity across the high-risk period of adolescence to young adulthood has not been investigated in a population-based, ethnically diverse sample.

RESEARCH DESIGN AND METHODS—A cohort of 10,481 individuals aged 12–21 years enrolled in the U.S. National Longitudinal Study of Adolescent Health (1996) was followed over two visits during young adulthood (18–27 years, 2001–2002; 24–33 years, 2007–2009). Separate logistic regression models were used to examine the associations of diabetes (A1C ≥6.5% or diagnosis by a health care provider) in young adulthood with *1*) obesity timing (never obese, onset <16 years, onset 16 to <18 years, onset ≥18 years) and 2) obesity duration over time (never obese, incident obesity, fluctuating obesity, and persistent obesity), testing differences by sex and race/ethnicity.

RESULTS—Among 24- to 33-year-old participants, 4.4% had diabetes (approximately half were undiagnosed), with a higher prevalence in blacks and Hispanics than whites. In multivariable analyses, women who became obese before age 16 were more likely to have diabetes than women who became obese at or after age 18 (odds ratio 2.77 [95% CI 1.39–5.52]), even after accounting for current BMI, waist circumference, and age at menarche. Persistent (vs. adult onset) obesity was associated with increased likelihood of diabetes in men (2.27 [1.41–3.64]) and women (2.08 [1.34–3.24]).

CONCLUSIONS—Diabetes risk is particularly high in individuals who were obese as adolescents relative to those with adult-onset obesity, thus highlighting the need for diabetes prevention efforts to address pediatric obesity.

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Diabetes prevalence has risen substantially over the last few decades, disproportionately affecting racial/ethnic minorities (1,2). In 2007, 24 million Americans had diabetes; nearly 6 million were undiagnosed (3). The total cost of diabetes in 2007 was estimated to be $174 billion (4), and this economic burden is likely to escalate over time. The adverse health and economic consequences combined with significant racial/ethnic disparities and high rates of undiagnosed diabetes emphasize the critical need to address this disease.

Although the links between current obesity and type 2 diabetes are clear, some research suggests that a history of obesity during childhood (5,6) and young adulthood (7) increases diabetes risk later in life. However, the differential and cumulative effects of obesity during different periods of the life span on the development of diabetes are not well documented. In particular, adolescence may be a sensitive period for the development of diabetes given the substantial decreases in insulin sensitivity associated with pubertal development (8). This period has the potential for alterations in insulin metabolism, which might increase diabetes risk later in life, and there is the potential that a longer duration of obesity might elicit additional metabolic changes that increase future diabetes risk (9,10). With obesity, pancreatic β-cells initially increase insulin release to overcome the reduced efficiency of insulin action, although over time compensation may become insufficient, ultimately leading to the development of diabetes (9,10). Nevertheless, epidemiologic research specifically examining the relationship between duration of obesity with diabetes in adulthood has yielded inconsistent results (11–16). A better understanding of the relationship of obesity timing and duration to diabetes in adulthood is needed to determine important periods for diabetes intervention, particularly in diverse ethnic groups.

In this article, we use nationally representative, longitudinal data to examine racial/ethnic differences in rates of diagnosed and undiagnosed diabetes in young adults. In addition, we capitalize upon longitudinal data to examine how adolescent-onset obesity and adult-onset obesity might differentially relate to diabetes risk in young adulthood and whether these associations differ by sex and race/ethnicity. We hypothesize that individuals who experienced adolescent- (vs. adult onset) obesity would be more likely to have diabetes in young adulthood, independent of current body size, with the highest risk among those with persistent obesity from the teen to adult years.

RESEARCH DESIGN AND METHODS

National Longitudinal Study of Adolescent Health

The National Longitudinal Study of Adolescent Health (Add Health) is a cohort of adolescents (N = 20,745; ages 11–21 years), representative of the U.S. school population in grades 7 to 12 in 1994–1995 (wave I) and followed into young adulthood. Wave II (1996; n = 14,738; ages 12–22 years) included wave I

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A slide set summarizing this article is available online.

adolescents who had not graduated from high school (including those who had dropped out of high school). Wave III (2001–2002; *n* = 15,197; ages 18–28 years) and wave IV (2008–2009; *n* = 15,601; ages 24–34 years) followed all wave I respondents regardless of wave II participation. Survey procedures have been described elsewhere (17) and were approved by the institutional review board at the University of North Carolina at Chapel Hill.

Our primary inclusion criterion was that respondents were seen during wave IV (*n* = 15,601). We excluded individuals without a longitudinal sampling weight (*n* = 801), which was needed to correct for nonresponse bias and sample design (18), ultimately resulting in 14,800 eligible individuals. In addition, we excluded Native Americans (*n* = 59) because of the small sample size and individuals who were missing the following data: diabetes status at wave IV (*n* = 113), measured height and weight at two or more waves (*n* = 1,221 [measured data not available at wave I]), family history of diabetes (*n* =

Table 1—*Prevalence of each diabetes group by sociodemographic and anthropometric characteristics of the analytic sample, from the National Longitudinal Study of Adolescent Health, waves II through IV* (N = *10,481*)

	N	No diabetes	Undiagnosed diabetes*	Diagnosed diabetes*	Total diabetes
Total	10,481	95.6 (0.3)	2.1 (0.3)	2.4 (0.2)	4.4 (0.3)
Race					
White	6,218	97.2 (0.3)	0.6 (0.1)	2.2 (0.2)	2.8 (0.3)
Black	2,096	87.5 (1)	9.6 (1)	2.9 (0.5)	12.5 (1)
Hispanic	1,630	94.0 (0.8)	2.6 (0.6)	3.5 (0.7)	6.0 (0.8)
Asian	537	96.9 (1.4)	1.5 (0.7)	1.6 (1.3)	3.1 (1.4)
Women	5,486	95.4 (0.4)	1.9 (0.3)	2.6 (0.3)	4.6 (0.4)
Mean age at wave IV, years	10,481	28.6 (0.1)	29.0 (0.2)	29.0 (0.2)	29.0 (0.2)
Education at wave IV					
Not a high-school graduate	2,364	94.0 (0.6)	2.5 (0.5)	3.5 (0.5)	6.0 (0.6)
High-school graduate	4,520	95.2 (0.4)	2.2 (0.3)	2.5 (0.3)	4.8 (0.4)
Some college	2,202	97.4 (0.5)	1.2 (0.3)	1.4 (0.3)	2.6 (0.5)
College graduate	1,395	96.7 (0.8)	2.0 (0.6)	1.3 (0.4)	3.3 (0.8)
Parental history of diabetes	879	89.8 (1.2)	3.9 (0.8)	6.4 (0.8)	10.2 (1.2)
Obesity timing† from baseline to final follow-up (waves II, III, and IV)					
Never obese	6,582	97.8 (0.3)	1.1 (0.2)	1.1 (0.2)	2.2 (0.3)
Adolescent obesity (onset before age 16)	386	92.3 (0.7)	3.3 (0.5)	4.3 (0.5)	7.7 (0.7)
Adolescent obesity (onset at or after age 16 to before age 18)	441	88.6 (2.3)	5.5 (1.8)	5.9 (1.4)	11.4 (2.3)
Adult-onset obesity (at or after age 18)	3,072	90.7 (1.8)	4.5 (1.3)	4.8 (1.2)	9.3 (1.8)
Obesity duration‡ from baseline to final follow-up (waves II, III, and IV)					
Never obese	6,582	97.8 (0.3)	1.1 (0.2)	1.1 (0.2)	2.2 (0.3)
Incident obesity	2,244	93.8 (0.7)	2.7 (0.5)	3.5 (0.5)	6.2 (0.7)
Fluctuating obesity	331	96.7 (0.9)	1.0 (0.5)	2.3 (0.8)	3.3 (0.9)
Persistent obesity	1,324	86.9 (1.4)	6.1 (1)	7.0 (1)	13.1 (1.4)
Adult weight status at final follow-up (wave IV)					
Normal (BMI <25)	3,562	98.1 (0.3)	0.8 (0.2)	1.1 (0.2)	1.9 (0.3)
Overweight (25 ≤ BMI < 30)	3,036	97.4 (0.4)	1.5 (0.4)	1.1 (0.2)	2.6 (0.4)
Obesity (30 ≤ BMI < 40)	2,743	93.1 (0.7)	2.8 (0.5)	4.1 (0.6)	6.9 (0.7)
Severe obesity (BMI ≥40)	883	85.9 (1.7)	7.5 (1.2)	6.7 (1.1)	14.1 (1.7)
Missing§	257				
Adult WC at final follow-up (wave IV)					
Normal (men, <120 cm; women, <88 cm)	5,381	97.8 (0.3)	1.1 (0.2)	1.1 (0.2)	2.2 (0.3)
Elevated	4,768	93.0 (0.6)	3.2 (0.4)	3.8 (0.4)	7.0 (0.6)
Missing§	322				

Data provided as percentages or means (SEs). All results were weighted for national representation, and the SEs were corrected for multiple stages of cluster sample design and unequal probability of selection. Diabetes status obtained in adulthood (wave IV). *Diagnosed diabetes defined as self-reported health care provider diagnosis of diabetes. Undiagnosed diabetes defined as A1C ≥6.5% with no report of a diabetes diagnosis. Percentages of diagnosed and undiagnosed diabetes do not sum to percentage of total diabetes because of rounding. †Timing of obesity was determined using the individual's age at the wave of initial obesity classification and categorized as follows: *1*) never obese; *2*) adolescent obesity (defined as initial classification of obesity before 16 years of age); *3*) adolescent obesity (between 16 and 18 years of age); and *4*) adult obesity (obesity onset at or after 18 years of age). ‡Obesity duration from adolescence to adulthood was based on obesity status at waves II, III, and IV and categorized as follows: *1*) never obese; *2*) incident obesity (nonobese at baseline and became and remained obese through last examination); *3*) fluctuating obesity (any shift in classification from obese to nonobese); and *4*) persistent obesity (obese at all waves). §Of the participants, 256 women had missing adult weight status/WC because they were pregnant at the time of measurement.

2,839), or demographic data (n = 87). Our final analytic sample included 10,481 individuals. Comparing the 15,601 eligible participants included in our analytic sample with the missing 5,120, we observed significant differences by race/ethnicity, education, age, and sex.

To assess selection bias, we used inverse probability weighting (IPW), which assigns a weight to each subject in the analytic sample so each subject accounts for himself or herself in the analysis as well as those with similar characteristics who were not selected into our final analytic sample (19). For each participant, we estimated a weight proportional to the inverse of the probability of being in our analytic sample. We used two separate multivariable, logistic models to predict diabetes at wave IV as a function of obesity (timing or duration), race/ethnicity, education, age, and sex: one model included the IPW and the second model did not include the IPW. The β coefficients from the two models were nearly identical, suggesting an absence of selection bias by race/ethnicity, education, age, and sex in our final models.

Measures

Outcome measure. At wave IV, diabetes was identified using self-reported previous diagnosis and A1C (from whole-blood spot assays collected from finger pricks, which have achieved the same level of precision and reproducibility as other standard methods of collecting blood, such as venipuncture [20]). We defined diagnosed diabetes as a "yes" response to the question, "Has a doctor, nurse, or other health provider ever told you that you have or had high blood sugar or diabetes [if female, when you were not pregnant]?" Individuals who responded "no" and had an A1C ≥6.5% were considered to have undiagnosed diabetes.

In addition, we conducted sensitivity testing of an alternative classification strategy in which we take account of medication use, given that some researchers have defined diagnosed diabetes as self-reported diabetes or self-reported use of diabetes medication (21). Yet some participants may have used diabetes medication (e.g., metformin) for the treatment of diseases other than diabetes (e.g., polycystic ovary syndrome or obesity). For this reason, we did not use diabetes medication as a criterion for diagnosed diabetes in our primary analysis. Nonetheless, we conducted a sensitivity analysis, in which diagnosed diabetes was defined as

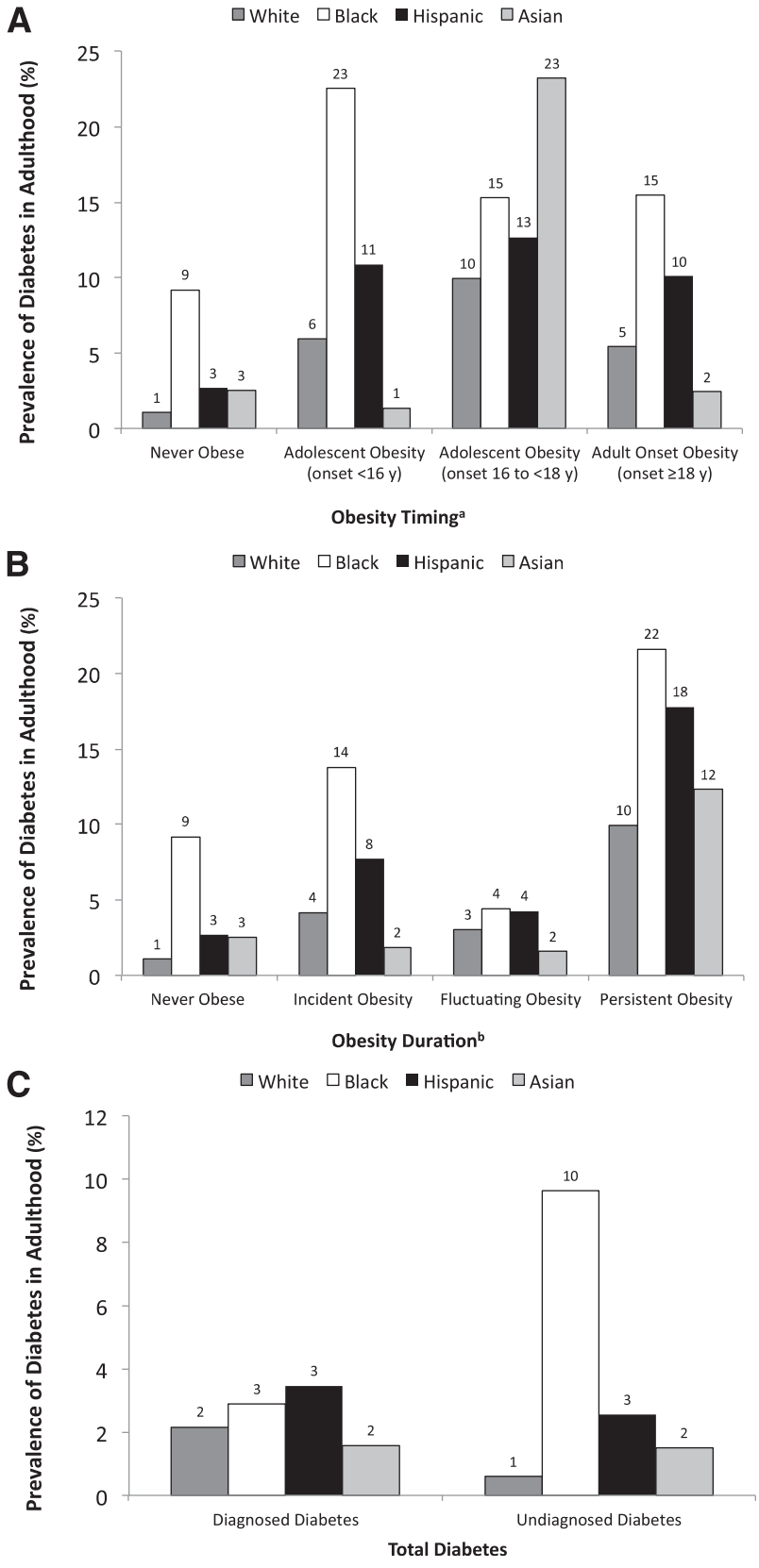

Figure 1—*Prevalence of diabetes in adulthood by obesity timing* (A)*, obesity duration from adolescence to adulthood* (B)*, and diagnosed and undiagnosed diabetes stratified by race/ethnicity* (C)*, from wave IV (2007–009) of the Add Health study.* A*: Timing of obesity was determined using the individual's age at the wave of initial obesity classification and categorized as follows:* 1*) never obese;* 2*) adolescent obesity (onset before 16 years of age);* 3*) adolescent obesity (onset at or after*

self-reported diagnosis (using the survey question described above) and the use of diabetes medication (recorded either by an interviewer when medication containers were available or by participant recall). In the sensitivity analysis, undiagnosed diabetes was defined as no self-report of diabetes, no use of diabetes medication, and an A1C ≥6.5%.

Main exposures. Height and weight were measured in waves II through IV during in-home surveys using standardized procedures. As recommended by expert panels, obesity was defined for adolescents as a BMI equal to or greater than the 95th percentile of the age- and sex-specific Centers for Disease Control and Prevention growth reference or BMI ≥30 kg/m^2 and as a BMI ≥30 kg/m^2 for adults (22). For women who were pregnant at time of measurement (wave II: n = 74; wave III: n = 152; wave IV: n = 256), BMI was coded as missing.

Obesity timing was determined using individual's age at the wave of initial obesity classification and categorized as follows: *1*) never obese; *2*) young adolescent obesity (initial classification of obesity before age 16 years); *3*) adolescent obesity (between the ages of 16 and 18 years); and *4*) adult obesity (obesity onset at or after the age of 18 years). Duration of obesity from adolescence to young adulthood was based on obesity status at waves II, III, and IV and categorized as as follows: *1*) never obese; *2*) incident obesity (nonobese at baseline and became and remained obese at last examination); *3*) fluctuating obesity (any shift in classification from obese to nonobese); and *4*) persistent obesity (obese at all waves). We selected adult-onset obesity and incident obesity as our referent categories because of our primary interest in understanding the effect of obesity timing and duration on diabetes risk among obese individuals. We include findings for individuals who were never obese to allow comparison with results of other studies.

Waist circumference (WC), obtained at wave IV, was measured midway between the lowest rib and the superior border of the iliac crest at end-expiration. We defined central obesity according to the criteria set by the Third Report of the National Cholesterol Education Program Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (men, ≥40 inches; women, ≥35 inches). For women who were pregnant at the time of measurement (n = 256), WC was coded as missing.

Control measures. Covariates were measured using traditional survey methods and included age; sex; education (less than high school, high school degree, some college, college or more); smoking (nonsmoker, former smoker, current smoker); parental history of diabetes (mother or father [yes/no]); and race/ethnicity (white, black, Hispanic, Asian).

Statistical analysis

Statistical analyses were conducted using STATA (release 12.0, Stata Corp., College Station, TX). To account for the stratified sampling strategy and clustered sampling design and nonresponse bias (18), longitudinal sample weights and survey analysis techniques were used in all analyses. Percentages were calculated for categorical variables, whereas means were calculated for continuous variables. We used an F statistic to test statistical differences between individuals with diagnosed diabetes, undiagnosed diabetes, and no diabetes. Given the low prevalence of diabetes in our cohort, we collapsed categories of undiagnosed and diagnosed diabetes together for multivariable analyses. Furthermore, 57 individuals with diabetes at wave IV (by self-report, A1C diagnosis, or both) also self-reported diabetes at wave III. Because diabetes diagnosis could result in purposeful changes in BMI resulting from diagnosis, use of medications to improve A1C, or both, we conducted a sensitivity analysis, excluding individuals classified with diabetes before wave IV (results not shown); we found almost identical results.

We used two separate multivariable logistic regression models to examine the association between obesity timing and duration with prevalent diabetes. Effect measure modification by sex was examined using interaction terms and Wald tests (criterion, $P < 0.15$). We did not find evidence of modification by sex for the association between obesity duration and prevalent diabetes (P = 0.35), but we did find evidence of modification by sex for the association between obesity timing and prevalent diabetes (P = 0.03). For comparability across timing and duration models, we included interaction by sex terms in all models. Given observed racial/ethnic disparities for whites and blacks, we also used race interaction terms to determine whether the relationships of obesity duration (P = 0.0002) and timing (P = 0.0003) with diabetes varied in whites versus blacks. The low prevalence of diabetes precluded our ability to examine racial/ethnic disparities in Hispanics and Asians and to examine a three-way interaction between our main obesity exposures of race/ethnicity and sex. The final model predicted odds of diabetes (undiagnosed and diagnosed) by obesity (timing or duration) and included confounders that met change-in-estimate criterion (>10% change in main effect coefficient) or conceptual rationale: age, sex (in race-stratified models), race/ethnicity (in sex-stratified models), education, and parental history of diabetes (smoking was not included because it did not meet change in estimate criterion). The final models were repeated with control for current BMI, current WC, and age at menarche (women) to assess changes in estimates for obesity timing and duration once current body size and menarche were considered.

RESULTS—Approximately 4% of the sample had diabetes; half of these cases were undiagnosed (Table 1). Furthermore, the prevalence of diagnosed and undiagnosed diabetes varied significantly by race/ethnicity, with blacks and Hispanics disproportionately affected. Among whites, 2.2% had diagnosed diabetes and 0.6% had undiagnosed diabetes. In contrast, among blacks, 2.9% had diagnosed diabetes and 9.6% had undiagnosed diabetes. Among Hispanics, 3.5% had diagnosed diabetes and 2.6% had undiagnosed diabetes.

By young adulthood 3,899 of 10,481 participants (37.2%) had been classified as obese at least once in the 13-year study period, and only a small percentage fluctuated in obesity status (Table 1).

age 16 to before age 18); and 4) adult obesity (onset at or after the age of 18 years). B: *Obesity duration from adolescence to adulthood was based on obesity status at waves II, III, and IV and categorized as follows:* 1) *never obese;* 2) *incident obesity (nonobese at baseline and became and remained obese through last examination);* 3) *fluctuating obesity (any shift in classification from obese to nonobese); and* 4) *persistent obesity (obese at all waves). Prevalence of diabetes was based on total diabetes, which includes* (C) *both diagnosed diabetes (self-reported health care provider diagnosis of diabetes) and undiagnosed diabetes (A1C ≥6.5% with no reported diabetes diagnosis) unless otherwise noted.*

Diabetes prevalence during young adulthood was highest among individuals who were persistently obese from adolescence into young adulthood.

Total diabetes prevalence differed across categories of obesity timing (Fig. 1*A*) and duration (Fig. 1*B*), and blacks and Hispanics were disproportionately affected. Although the prevalence of diagnosed diabetes did not differ by race/ethnicity, there were differences in rates of undiagnosed diabetes, which was highest in blacks (Fig. 1*C*).

Multivariable analysis showed that the likelihood of diabetes for individuals with adolescent obesity relative to those with obesity onset at or after 18 years of age differed by sex ($P = 0.03$) (Table 2). Women with onset of obesity during early adolescence (before 16 years of age) were at more than a twofold higher risk of diabetes in early adulthood (odds ratio [OR] 2.77 [95% CI 1.39–5.52]) than women with obesity onset at or after 18 years of age, even after controlling for current BMI (2.26 [1.07–4.76]), current WC (2.91 [1.45–5.86]), or age at menarche (2.46 [1.11–5.48]). In contrast, men who had onset of obesity between 16 and 18 years of age had slightly higher odds of diabetes than those with onset at or after 18 years of age, but this association was not statistically significant. Men who had onset of obesity before 16 years of age had similar odds of diabetes in young adulthood as men with obesity onset at or after age 18 years.

Although individuals who were never obese were less likely to have diabetes during young adulthood than those who became obese during young adulthood (Table 3), this relationship was significantly attenuated in blacks compared with whites ($P = 0.0003$). Persistent obesity conferred a higher likelihood of diabetes during young adulthood than incident obesity in whites, but not blacks, even after controlling for current BMI (OR 2.02 [95% CI 1.16–3.51]) and current WC (2.30 [1.38–3.82]). The estimates for blacks were attenuated after controlling for current BMI (1.36 [0.77–2.40]) but became statistically significant after controlling for current WC (1.75 [1.01–3.03]). Limitations of the sample sizes precluded multivariable analyses for Hispanics and Asians. However, unadjusted analyses suggest that the relationship between duration of obesity and prevalent diabetes in Hispanics was similar to that in whites. In sensitivity analyses, including diabetes medication as a criterion for diagnosed diabetes yielded a similar pattern of results, albeit with larger confidence intervals around the estimates (results not shown).

Table 2—*Adjusted OR (95% CIs)* from separate logistic regression models predicting prevalent diabetes in young adulthood (wave IV, 2007–2009) as a function of obesity timing and obesity duration, stratified by sex, from the National Longitudinal Study of Adolescent Health, waves II through IV (N = 10,481)*

	Total	Men	Women
Obesity timing†			
Never obese	0.34 (0.26–0.44)	0.31 (0.20–0.46)	0.37 (0.25–0.54)
Adolescent obesity (onset before age 16)	1.70 (0.99–2.94)	0.90 (0.39–2.06)	2.77 (1.39–5.52)
Adolescent obesity (onset at or after age 16 to before age 18)	1.62 (0.99–2.66)	1.51 (0.78–2.91)	1.72 (0.96–3.10)
Adult-onset obesity (at or after age 18)	Referent	Referent	Referent
Obesity duration (waves II, III, and IV)‡			
Never obese	0.40 (0.29–0.55)	0.39 (0.25–0.63)	0.41 (0.27–0.62)
Fluctuating obesity	0.58 (0.30–1.13)	0.30 (0.11–0.80)	0.93 (0.40–2.19)
Persistent obesity	2.17 (1.55–3.05)	2.27 (1.41–3.64)	2.08 (1.34–3.24)
Incident obesity	Referent	Referent	Referent

*Adjusted logistic regression model predicting odds of diabetes (total diabetes, which includes both diagnosed diabetes and self-reported health care provider diagnosis of diabetes) and undiagnosed diabetes (A1C ≥6.5% with no reported diabetes diagnosis) by obesity (timing or duration), age, race/ethnicity, education, and parental history of diabetes. †Timing of obesity was determined using the individual's age at the wave of initial obesity classification and categorized as follows: *1*) never obese, *2*) adolescent obesity (defined as initial classification of obesity before 16 years of age), *3*) adolescent obesity (defined as initial classification of obesity between 16 and 18 years of age), and *4*) adult obesity defined as obesity onset at or after 18 years of age. Significant effect measure modification by race/ethnicity (Wald test, $P = 0.03$). ‡Obesity duration from adolescence to adulthood was based on obesity status at waves II, III, and IV and categorized as follows: *1*) never obese, *2*) incident obesity (nonobese at baseline and became and remained obese through last examination), *3*) fluctuating obesity (any shift in classification from obese to nonobese), and *4*) persistent obesity (obese at all waves). Effect measure modification by race/ethnicity not statistically significant but is shown by strata for comparability (Wald test, $P = 0.35$).

CONCLUSIONS—Taking advantage of nationally representative data, we observed that 4.4% of our study population, aged 24–34 years and measured in 2007–2009, had diabetes. These estimates are slightly higher than estimates from individuals enrolled in the National Health and Nutrition Examination Survey 2003–2006, which report that 2.5% of individuals 20–39 years of age have diabetes (1). From SEARCH, a population-based study of diabetes, approximately 150,000 children and adolescents were estimated to have diabetes diagnosed by a physician (23). Our nationally representative estimates suggest that approximately 309,209 young adults between the ages of 24 and 34 have diagnosed diabetes; thus, the transition from adolescence to young adulthood is a sensitive period for the development of diabetes. In addition, our estimates suggest approximately 265,624 U.S. young adults have A1C ≥6.5% but have not been told by a health care professional that they have diabetes, and a significantly higher proportion of these individuals are black. These results are particularly concerning in that racial/ethnic minorities may be at greater risk of certain diabetes-related complications (e.g., end-stage renal disease, lower limb amputations, and arterial stiffness [as a marker of cardiovascular disease]) than whites (24–26). Furthermore, our findings underscore the need for individual awareness of diabetes risk, access to health care, and physicians to screen for diabetes in at-risk populations.

Although current obesity is associated with diabetes risk (7,9,10) and some research suggests that adolescent obesity is associated with diabetes during young adulthood (27,28), history of obesity may provide additional information about future risk. Obesity during adolescence may be more deleterious for insulin resistance and diabetes than obesity during other periods of the life span. There are notable changes in insulin sensitivity during pubertal development, and the adolescent

Table 3—*Adjusted ORs (95% CIs)* from separate logistic regression models predicting prevalent diabetes in young adulthood (wave IV, 2007–2009) as a function of obesity timing and obesity duration, stratified by race/ethnicity, from the National Longitudinal Study of Adolescent Health, waves II through IV* (N = *8,314*)†

	Whites	Blacks
Obesity timing‡		
Never obese	0.21 (0.14–0.31)	0.60 (0.41–0.89)
Adolescent obesity (onset before age 16)	1.43 (0.68–3.00)	2.25 (1.01–4.98)
Adolescent obesity (onset at or after age 16 to before age 18)	1.90 (0.92–3.92)	1.06 (0.51–2.17)
Adult-onset obesity (at or after age 18)	Referent	Referent
Obesity duration (waves II, III, and IV)§		
Never obese	0.26 (0.16–0.41)	0.65 (0.41–1.02)
Fluctuating obesity	0.70 (0.31–1.57)	0.28 (0.07–1.17)
Persistent obesity	2.37 (1.43–3.94)	1.69 (0.96–2.98)
Incident obesity	Referent	Referent

*Adjusted logistic regression model predicting odds of diabetes: total diabetes, which includes both diagnosed diabetes (self-reported health care provider diagnosis of diabetes) and undiagnosed diabetes (A1C ≥6.5% with no reported diabetes diagnosis) by obesity (timing or duration); age; race/ethnicity; education; sex; and parental history of diabetes. †Hispanics and Asians are excluded because of the small sample sizes. ‡Timing of obesity was determined using the individual's age at the wave of initial obesity classification and categorized as follows: *1*) never obese, *2*) adolescent obesity (defined as initial classification of obesity before 16 years of age), *3*) adolescent obesity (defined as initial classification of obesity between 16 and 18 years of age), and *4*) adult obesity defined as obesity onset at or after age 18 years. Significant effect measure modification by race/ethnicity (Wald test, $P = 0.0003$). §Obesity duration from adolescence to adulthood was based on obesity status at waves II, III, and IV and categorized as follows: *1*) never obese, *2*) incident obesity (nonobese at baseline and became and remained obese through last examination), *3*) fluctuating obesity (any shift in classification from obese to nonobese), and *4*) persistent obesity (obese at all waves). Significant effect measure modification by race/ethnicity (Wald test, $P = 0.0002$).

period is one of increased insulin resistance, particularly for women (8). However, duration of obesity likely plays a role as well. We addressed this issue by examining adolescent obesity onset before 16 years of age as well as onset at or after age 16 to before age 18. We observed a significantly higher prevalence of diabetes in young adulthood among women who became obese before the age of 16 years compared with those who became obese during young adulthood, even after controlling for current size and age at menarche. Our results suggest that the influence of early-onset obesity (before 16 years of age) may be particularly deleterious for future diabetes risk for women and could potentially be related to biological factors associated with being obese during pubertal maturation. Disentangling sex differences related to the association between obesity and diabetes risk is an area with great potential for future research.

Obesity duration may also provide more information about diabetes risk beyond current obesity (29–31). Prolonged duration of obesity may result in additional metabolic changes, leading to the development of hyperglycemia and diabetes (10,32). The epidemiologic literature is mixed: whereas most studies report a positive association between obesity duration and diabetes (11,13,15,16), others report null associations (12,14). In our study, individuals with persistent obesity had the highest likelihood of diabetes during young adulthood, even compared with individuals with adult-onset obesity. Yet we observed attenuated risk in blacks relative to whites that must be put in context with the higher proportion of diabetes in nonobese blacks relative to that in whites, which is consistent with findings from another large multiethnic study (33). Given that, on average, blacks have lower adiposity at a similar BMI than whites (34), we speculate that the observed ethnic difference in the likelihood of diabetes is attributed to other factors beyond obesity. However, further research is needed to understand these racial/ethnic disparities.

Our study has several limitations. First, Add Health uses A1C measurements and self-report to classify diabetes. Second, in our regression models we collapsed all categories of diabetes. The resulting heterogeneous group includes those with type 1 or type 2 diabetes and individuals with undiagnosed diabetes (either type 1 or 2 diabetes). It is unfortunate that data limitations precluded us from disentangling individuals with type 1 and type 2 diabetes. The inclusion of individuals with type 1 diabetes may have attenuated our results, although there is increasing evidence that suggests a link between obesity and type 1 diabetes (35,36). The small proportion of individuals with undiagnosed diabetes precluded separate multivariable analyses with this group. However, the inclusion of individuals with undiagnosed diabetes is not likely to change our results because these individuals would not have had a purposeful change in their weight as a result of diabetes diagnosis.

Given the schedule of examinations, we were unable to determine exact age at the onset of obesity or the exact duration of obesity. Instead, we know only the age of a given individual at each wave and that the individual became obese before the wave II survey, became obese between the waves of Add Health, or was not obese at the time of the wave IV survey. In some cases ($n = 586$), we did not know whether an individual became obese before or after 18 years of age (e.g., individuals who were 16.5 years and nonobese at wave II and 21.5 years and obese at wave III). For our main analyses, these individuals were categorized with adult-onset obesity. We conducted a sensitivity analysis whereby we reclassified these individuals as having adolescent-onset obesity and found larger estimated effect sizes in identical multivariable analyses, suggesting that our results using the original categorization may be conservative estimates.

We take advantage of rich longitudinal data from a racially and ethnically diverse population followed over a period of increased risk for weight gain, but the small number of individuals with diabetes precluded our ability to fully interrogate differences in Hispanics and Asians in multivariable analyses. Nonetheless, our descriptive analyses indicate that Hispanics have a higher prevalence of diabetes during young adulthood than whites, regardless of differences in onset and duration of obesity, which is consistent with findings from National Health and Nutrition Examination Survey cross-sectional data, which examined concurrent BMI and diabetes (37). Our longitudinal data also show that the association between duration of obesity and diabetes may be different in blacks versus whites. To better understand the potentially differential effect of obesity on diabetes by race/ethnicity, future research is needed using

large, longitudinal, population-based studies of ethnically diverse children.

Using a large, ethnically diverse, nationally representative cohort followed over a 13-year time span, we examined the relationship between obesity during the transition from adolescence to young adulthood, a particularly sensitive period for development of obesity (38,39), and diabetes risk in young adulthood as classified by A1C, according to the most recent recommendations (40). Most research examining the relationship between obesity and diabetes has been limited to exclusively pediatric or adult populations, thus precluding comparisons of adolescent- versus adult-onset obesity in the same individuals followed over time. Furthermore, the few studies (27,28) examining the association between obesity in the transition from adolescence to young adulthood and diabetes have used self-report, recalled weight, or somatotype silhouettes (28) and have been conducted in relatively homogenous populations (28).

In conclusion, we observed high rates of undiagnosed diabetes in our young adult population, which is a significant public health concern. These findings emphasize the need for awareness, screening, and access to care, especially among at-risk populations. We found that adolescent obesity (vs. adult onset) obesity and persistent obesity from adolescence to young adulthood conferred the greatest likelihood of diabetes in young adulthood. Our findings suggest that, in addition to current obesity, information about timing and duration of obesity are needed to fully understand and predict diabetes risk. Finally, efforts to prevent diabetes must address pediatric obesity.

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N.S.T. and P.G.-L. designed the study. N.S.T., A.S.R., and P.G.-L. contributed to data analysis and wrote the manuscript. N.S.T., A.S.R., and P.G.-L. are the guarantors of this work and, as such, had full access to all the data in the study and take responsibility for the integrity of the data and the accuracy of the data analysis.

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