Duration of Abdominal Obesity Beginning in Young Adulthood and Incident Diabetes Through Middle Age

The CARDIA Study

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OBJECTIVE—To examine whether the duration of abdominal obesity determined prospectively using measured waist circumference (WC) is associated with the development of new-onset diabetes independent of the degree of abdominal adiposity.

RESEARCH DESIGN AND METHODS—The Coronary Artery Risk Development in Young Adults Study is a multicenter, community-based, longitudinal cohort study of 5,115 white and black adults aged 18–30 years in 1985 to 1986. Years spent abdominally obese were calculated for participants without abdominal obesity (WC >102 cm in men and >88 cm in women) or diabetes at baseline ($n = 4,092$) and was based upon repeat measurements conducted 2, 5, 7, 10, 15, 20, and 25 years later.

RESULTS—Over 25 years, 392 participants developed incident diabetes. Overall, following adjustment for demographics, family history of diabetes, study center, and time varying WC, energy intake, physical activity, smoking, and alcohol, each additional year of abdominal obesity was associated with a 4% higher risk of developing diabetes [hazard ratio (HR) 1.04 (95% CI 1.02–1.07)]. However, a quadratic model best represented the data. HRs for 0, 1–5, 6–10, 11–15, 16–20, and >20 years of abdominal obesity were 1.00 (referent), 2.06 (1.43–2.98), 3.45 (2.28–5.22), 3.43 (2.28–5.22), 2.80 (1.73–4.54), and 2.91 (1.60–5.29), respectively; P-quadratic < 0.001.

CONCLUSIONS—Longer duration of abdominal obesity was associated with substantially higher risk for diabetes independent of the degree of abdominal adiposity. Preventing or at least delaying the onset of abdominal obesity in young adulthood may lower the risk of developing diabetes through middle age.

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I n 2010, diabetes was diagnosed in ~25.6 million adults in the United States (prevalence 11.3%) (1). Overall obesity as reflected by an increased BMI is a well-recognized risk factor for type 2 diabetes (2), and weight control is an effective strategy in the prevention of diabetes (3). Body shape or increased central obesity has also emerged as an important determinant of diabetes. In fact, abdominal obesity as reflected by an increased waist circumference (WC) has been shown to predict risk of diabetes beyond that of overall obesity (4).

Due to increases in the prevalence of overall and abdominal obesity in the United States over the last three decades, younger individuals are experiencing a greater cumulative exposure to excess adiposity over their lifetime (5–7). However, few studies have determined the impact of this chronic, long-term obesity on diabetes rates. Most (8–14), but not all (15), studies that have quantified the duration of overall obesity have found a longer duration to be independently associated with an increased risk of diabetes. With an increasing prevalence of abdominal obesity and its established role in the development of diabetes (16), it also becomes important to better understand the implications of a longer duration of abdominal obesity. However, to our knowledge, no study has examined whether the duration of abdominal obesity also contributes to the incidence of diabetes.

The current study was conducted to investigate whether the duration of abdominal obesity determined prospectively using measured WC is associated with the development of new-onset diabetes independent of the degree of abdominal adiposity during a 25-year follow-up period beginning early in adulthood in 1985 to 1986 among white and black participants of the Coronary Artery Risk Development in Young Adults (CARDIA) Study. The CARDIA Study provided an ideal setting in which to address this question because participants were recruited and followed largely during the obesity epidemic of the last three decades in the United States as well as during the period of greatest gain in adiposity during the life course (6,17). Since previous studies have shown that rates of both abdominal obesity and diabetes vary significantly by race (1,5,18), and, for a given level of adiposity, relative risk estimates for diabetes are lower among blacks compared with whites (19), we also sought to determine whether the duration of abdominal obesity differentially influences risk for diabetes between white and black men and women.

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RESEARCH DESIGN AND METHODS

Study population

CARDIA is a multicenter community-based longitudinal cohort study of the development and determinants of cardiovascular disease over time in 5,115 young adults initially aged 18–30 years in 1985 to 1986. Black and white adults were recruited from four cities in the United States (Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA) with population-based samples approximately balanced within center by sex, age (18–24 years and 25–30 years), race (white and black), and education (high school graduate or less, greater than high school graduate). To date, participants have been re-examined 2, 5, 7, 10, 15, 20, and 25 years after baseline, and retention rates across examinations were 91, 86, 81, 79, 74, 72, and 72%, respectively. All participants provided written informed consent at each examination, and institutional review boards from each field center and the coordinating center approved the study annually.

Of the 5,115 participants, we excluded those who had diabetes or an unknown diabetes status at baseline (n = 240), were abdominally obese [>102 cm in men and >88 cm in women (20)] or missing WC at baseline (n = 387), were pregnant during any examination (n = 206), had bariatric surgery during follow-up (n = 33), were transgender (n = 2), or were missing the measurement of WC during follow-up (n = 155). The remaining 4,092 participants formed the sample population for analysis.

Clinical measurements

Standardized protocols for data collection were used across study centers and examinations. Participants were asked to fast for at least 12 h before each examination and to avoid smoking or engaging in heavy physical activity for at least 2 h.

Anthropometry

WC was measured with a tape in duplicate to the nearest 0.5 cm around the minimal abdominal girth identified laterally midway between the iliac crest and the lowest portion of the rib cage and anteriorly midway between the xiphoid process and the umbilicus. Weight and height were measured with participants wearing light examination clothes and no shoes. Body weight was measured to the nearest 0.2 kg with a calibrated balance-beam scale. Height was measured with a vertical ruler to the nearest 0.5 cm. BMI was calculated as weight in kilograms divided by height in meters squared.

Duration of elevated WC

The primary exposure variable of interest was duration of elevated WC. The sum of years spent abdominally obese during the 25-year follow-up was calculated for participants without abdominal obesity at baseline. Onset of abdominal obesity was considered to be the time between the examination first measured as abdominally obese and the prior examination year. The algorithm accounted for fluctuations in WC above and below the threshold of abdominal obesity during follow-up. For participants who developed diabetes, the number of years spent abdominally obese were determined up until the examination when incident diabetes was identified. For those who did not develop diabetes, years of abdominal obesity were summed until the last known follow-up examination. The age at first appearance of abdominal obesity was defined as the age at the examination year prior to the examination at which abdominal obesity was identified.

Diabetes assessment

Blood was drawn by venipuncture and processed at the central laboratory according to a standard protocol. Glucose was assayed at baseline (i.e., year 0) using the hexokinase ultraviolet method by American Bio-Science Laboratories (Van Nuys, CA) and at years 7, 10, 15, 20, and 25 using hexokinase coupled to glucose-6-phosphate dehydrogenase by Linco Research (St. Louis, MO). Glucose values at follow-up were recalibrated to year 0 glucose values. Glycated hemoglobin A_{1c} was measured using the Tosoh G7 high-performance liquid chromatography method (Tosoh Bioscience) at years 20 and 25. Diabetes was determined based on a combination of measured fasting glucose levels ($\geq$7.0 mmol/L, $\geq$126 mg/dL) at examination years 7, 10, 15, 20, or 25; self-report of oral hypoglycemic medications or insulin (all examinations); a 2-h postload glucose $\geq$11.1 mmol/L ($\geq$ 200 mg/dL) at examination years 10, 20, and 25; or a glycated hemoglobin $A_{1c} \geq$6.5% at years 20 and 25 (21). The incidence of diabetes over 25 years was determined among participants who did not have diabetes at baseline based on fasting glucose levels and report of medications.

Other measurements

Standard questionnaires were used to maintain consistency in the assessment of demographic (age, sex, race, and education) and behavioral (physical activity, cigarette smoking, and alcohol use) information across all CARDIA examination visits. Education was represented as years of schooling. The CARDIA Physical Activity History questionnaire was used to query the amount of time per week spent in 13 categories of leisure, occupational, and household physical activities over the past 12 months (22). Activity was expressed in exercise units; a total activity score of 300 exercise units approximates Department of Health and Human Services recommendations of $\geq$150 min of moderate-intensity activity per week. Cigarette smoking status was classified as ever or never based upon information collected at each examination. Total daily alcohol consumption was calculated from an interviewer-administered questionnaire. Energy intake was measured with the interviewer-administered, validated CARDIA dietary history at years 0, 7, and 20 (23). Extreme values of energy intake (high: >8,000 kcal/day in men and >6,000 kcal/day in women; low: <800 kcal/day in men and <600 kcal/day in women) were excluded as unreliable. Diabetes diagnosed in an immediate family member (mother, father, sister, or brother) was also queried at years 0, 5, 10, and 25. Among women, number of pregnancies and menopausal status were obtained by self-report.

Statistical analysis

All analyses were performed overall and according to the four race-sex strata. Participant characteristics were described using means and proportions. We calculated the incidence rate of diabetes (number of incident cases per person time at risk) per 1,000 person-years according to the duration of abdominal obesity. Follow-up time at risk was calculated as the difference between baseline and the examination when incident diabetes was identified. For participants who did not develop diabetes, follow-up time was censored at the last known follow-up examination. Multivariable Cox proportional hazards regression models were used to estimate the hazard ratio (HR) and 95% CI for incident diabetes according to the duration of abdominal obesity. Duration of abdominal obesity was included as a time-dependent variable in analyses in one of two exposure forms:

first as a continuous variable assuming a linear dose-response association and second as a six-level categorical variable (i.e., 0, 1–5, 6–10, 11–15, 16–20, and >20 years). Analyses were adjusted for baseline age, maximum years of education, family history of diabetes (yes/no), CARDIA field center, and the following time-dependent covariates: WC (cm), energy intake (average kcal), smoking status (never/ever), alcohol use (average mL/day), physical activity (average exercise units), and, among women in models stratified by race, number of pregnancies and postmenopausal status (yes/no). To test for the presence of a quadratic trend, we added a squared duration of abdominal obesity term to the multivariable models that also included a linear term. Potential effect modification by race in sex-stratified models was evaluated by testing the statistical significance of a multiplicative interaction term including race and duration of abdominal obesity as a categorical variable in models that also included lower-order terms.

We also performed a sensitivity analysis to determine the influence of missing WC values (18.8% of all measurements) on the association between the duration of abdominal obesity and incident diabetes. Multiple imputation was used to impute missing WC using the sequential regression imputation approach that is implemented in the software package IVEware (24). Five datasets were generated using all available WC data on the sample of 4,092 participants. Each data set was analyzed separately, and results from the five analyses were combined using Rubin's rules (25).

Tests of statistical significance were two-tailed, with an α level of 0.05. A type 1 error rate of 0.10 was set for tests of multiplicative interaction. SAS version 9.2 (SAS Institute, Cary, NC) was used to perform all analyses.

RESULTS—Of the 4,092 eligible participants without abdominal obesity or diabetes at baseline, 48.8% were black, and 50.0% were women. The characteristics of participants overall and according to race and sex group are shown in Table 1. At baseline, mean values of glucose were well below the therapeutic threshold across all race-sex groups. About one-third of participants had a family history of diabetes with a slightly higher proportion observed among black as compared with white adults.

The percentage of participants who developed abdominal obesity, mean age at onset, mean duration, and the proportion of participants in each 5-year category of abdominal obesity overall and according to race and sex are shown in Table 2. The occurrence of abdominal obesity during follow-up was significantly higher among black women (57.5%) than all other race-sex groups ($P < 0.001$). Compared with all other groups, black women also developed abdominal obesity at a younger age (mean 34.5 years), resulting in a longer duration of exposure (mean 7.6 years) ($P < 0.001$ for both). Additional comparisons showed the proportion (34.7 vs. 28.4%) and duration (4.4 vs. 3.3 years) of abdominal obesity were higher among white women as compared with black men ($P < 0.01$ for both). On average, black

men developed abdominal obesity at a younger age than white men (36.1 vs. 38.4 years; $P < 0.001$).

During 85,096 person-years of follow-up, 392 participants developed incident diabetes, a rate of 4.6/1,000 person-years (Table 3). Fig. 1 displays the incidence of diabetes per 1,000 person-years according to the duration of abdominal obesity in 5-year increments overall and according to race and sex. The incidence of diabetes was higher with a longer duration of abdominal obesity peaking at 11–15 years for black men and women, 16–20 years for white men, and >20 years for white women.

A longer duration of exposure to abdominal obesity was associated with incident diabetes (Table 3). Overall, with each additional year of abdominal obesity, the multivariable adjusted HR for diabetes was 4% higher [HR 1.04 (95% CI: 1.02–1.07)] and was similar when a lower cut point was used to define abdominal obesity (≥94 cm for men and ≥80 cm for women) [1.04 (1.01–1.06)]. Similar results were also observed in a sensitivity analysis that imputed missing values using all available measures of WC during follow-up on the sample of 4,092 participants [1.03 (1.01–1.06)]. However, we found that a quadratic model best represented the risk for diabetes associated with the duration of abdominal obesity. Compared with those who did not develop abdominal obesity (zero years of abdominal obesity), each 5-year higher category of abdominal obesity was associated with a substantially higher risk for diabetes that peaked at ~11–15 years of abdominal obesity (Table 3). WC was also

Table 1—*Characteristics of 4,092 participants included in the current study overall and according to race and sex: the CARDIA Study*

Variable	Total population (n = 4,092)	Black participants		White participants	
		Men (n = 973)	Women (n = 1,025)	Men (n = 1,075)	Women (n = 1,019)
Age (years), year 0	24.8 (3.6)	24.1 (3.8)	24.2 (3.8)	25.4 (3.4)	25.5 (3.4)
WC (cm), year 0	75.9 (8.8)	79.1 (7.7)	72.1 (7.4)	82.0 (7.5)	70.0 (6.2)
BMI (kg/m^2), year 0	23.5 (3.4)	24.0 (3.4)	23.8 (4.0)	23.9 (3.0)	22.3 (3.0)
Maximum education (years)	15.3 (2.6)	14.1 (2.3)	14.7 (2.2)	16.0 (2.8)	16.3 (2.6)
Physical activity (exercise units), year 0	433.3 (302.8)	533.8 (338.9)	280.3 (231.2)	508.2 (300.3)	411.8 (265.1)
Never smoking [n (%)], year 0	2,326 (56.8)	528 (54.3)	622 (60.7)	619 (57.6)	557 (54.7)
Alcohol (mL/day), year 0	12.5 (21.8)	16.8 (28.8)	5.4 (13.1)	18.6 (24.5)	8.9 (13.9)
Total calories (kcal), year 0	2,859.4 (1,349.7)	3,661.4 (1,550.0)	2,476.1 (1,150.8)	3,230.1 (1,244.6)	2,109.1 (792.8)
Glucose (mmol/L), year 0	4.5 (0.5)	4.6 (0.4)	4.4 (0.4)	4.7 (0.4)	4.5 (704)
Family history of diabetes [n (%)]	1,359 (33.2)	350 (36.0)	441 (43.0)	288 (26.8)	280 (27.5)

Data are mean (SD) or n (%).

Table 2—*Descriptive characteristics on the occurrence of abdominal obesity* during follow-up overall and according to race and sex: the CARDIA Study*

		Black participants		White participants	
Variable	Total population (n = 4,092)	Men (n = 973)	Women (n = 1,025)	Men (n = 1,075)	Women (n = 1,019)
Abdominal obesity during follow-up [n (%)]‡·§	1,570 (38.4)	276 (28.4)	589 (57.5)	351 (32.7)	354 (34.7)
Age at onset of abdominal obesity (years)†·‡·¥	36.3 (7.9)	36.1 (7.8)	34.5 (7.7)	38.4 (7.7)	37.4 (7.6)
Years of abdominal obesity‡·§	4.7 (7.2)	3.3 (6.2)	7.6 (8.1)	3.7 (6.3)	4.4 (7.1)
5-year higher duration of abdominal obesity [n (%)]					
0 years	2,522 (61.6)	697 (71.6)	436 (43.3)	724 (67.4)	665 (65.3)
1–5 years	426 (10.4)	89 (9.2)	127 (12.4)	114 (10.6)	96 (9.4)
6–10 years	386 (9.4)	68 (7.0)	143 (14.0)	98 (9.1)	77 (7.6)
11–15 years	317 (7.8)	54 (5.6)	124 (12.0)	65 (6.1)	74 (7.3)
16–20 years	273 (6.7)	40 (4.1)	120 (11.6)	45 (4.2)	68 (6.7)
>20 years	168 (4.1)	25 (2.6)	75 (7.5)	29 (2.7)	39 (3.8)

Data are mean (SD) or n (%). *Abdominal obesity defined as a WC >102 cm for men and >88 cm for women (20). †Among those who developed abdominal obesity during follow-up. ‡Black women significantly different than all other race-sex groups (P < 0.001). §White women significantly higher than black men (P < 0.01). ¥Black men significantly younger than white men (P < 0.001).

associated with diabetes risk [1.04 (1.03–1.05/cm)].

In general, a quadratic trend in the risk for diabetes associated with the duration of abdominal obesity that peaked at ~11–15 years was observed in all race-sex groups except white women, in whom a significant linear trend was noted (Table 3). With each additional year of abdominal obesity, an 8% higher risk for diabetes was observed in white women [HR 1.08 (95% CI 1.02–1.15)]. These associations did not vary significantly between white and black men or women (P-interaction >0.2 and 0.1, respectively).

CONCLUSIONS—In this multicenter, community-based, longitudinal cohort

Table 3—*Adjusted HRs and 95% CI for incident diabetes* according to duration of abdominal obesity† during follow-up overall and according to race and sex: the CARDIA Study*

		Black participants		White participants	
	Total population (n = 4,092)	Men (n = 973)	Women (n = 1,025)	Men (n = 1,075)	Women (n = 1,019)
Cases of diabetes	392	120	140	82	50
Person-years of follow-up	85,096	18,782	20,819	23,049	22,446
Incidence of diabetes (per 1,000 person-years)	4.6	6.4	6.7	3.6	2.2
HR (95% CI) per year of abdominal obesity‡	1.04 (1.02–1.07)	1.00 (0.96–1.04)	1.05 (1.01–1.09)	1.07 (1.02–1.11)	1.08 (1.02–1.15)
HR (95% CI) according to duration of abdominal obesity in 5-year increments					
0 years	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)	1.00 (referent)
1–5 years	2.06 (1.43–2.98)	1.85 (1.01–3.39)	1.48 (0.71–3.08)	2.46 (1.14–5.32)	3.04 (1.06–8.69)
6–10 years	2.43 (1.65–3.58)	1.69 (0.84–3.38)	2.03 (0.99–4.16)	3.35 (1.51–7.43)	2.80 (0.93–8.40)
11–15 years	3.45 (2.28–5.22)	2.05 (0.95–4.44)	3.42 (1.62–7.26)	5.27 (2.22–12.50)	2.26 (0.64–8.04)
16–20 years	2.80 (1.73–4.54)	1.04 (0.40–2.73)	2.96 (1.27–6.87)	4.69 (1.69–13.02)	3.75 (1.00–14.11)
>20 years	2.91 (1.60–5.29)	1.01 (0.31–3.26)	2.87 (1.03–8.00)	1.99 (0.36–11.01)	11.61 (2.59–52.13)
P-quadratic trend	<0.001	0.008	0.06	0.006	>0.2

*Incident diabetes defined as a self-report of oral hypoglycemic medications or insulin at examination years 2, 5, 7, 10, 15, 20, and 25; fasting glucose levels ≥7.0 mmol/L (≥126 mg/dL) at examination years 7, 10, 15, 20, or 25; a 2-h postload glucose ≥11.1 mmol/L (≥200 mg/dL) at examination years 10, 20, and 25; or a glycated hemoglobin A_{1c} ≥6.5% at years 20 and 25 (21). †Abdominal obesity defined as a WC >102 cm for men and >88 cm for women (20). ‡Adjusted for baseline age (years), race-sex (except in stratified models), maximum years of education, family history of diabetes (yes/no), study center (Birmingham, AL; Chicago, IL; Minneapolis, MN; and Oakland, CA), and time varying WC (cm), energy intake (average kcal/day), smoking status (never/ever), alcohol intake (average mL/day), physical activity (average exercise units), and, among women in analyses stratified by race, number of pregnancies and postmenopausal status (yes/no).
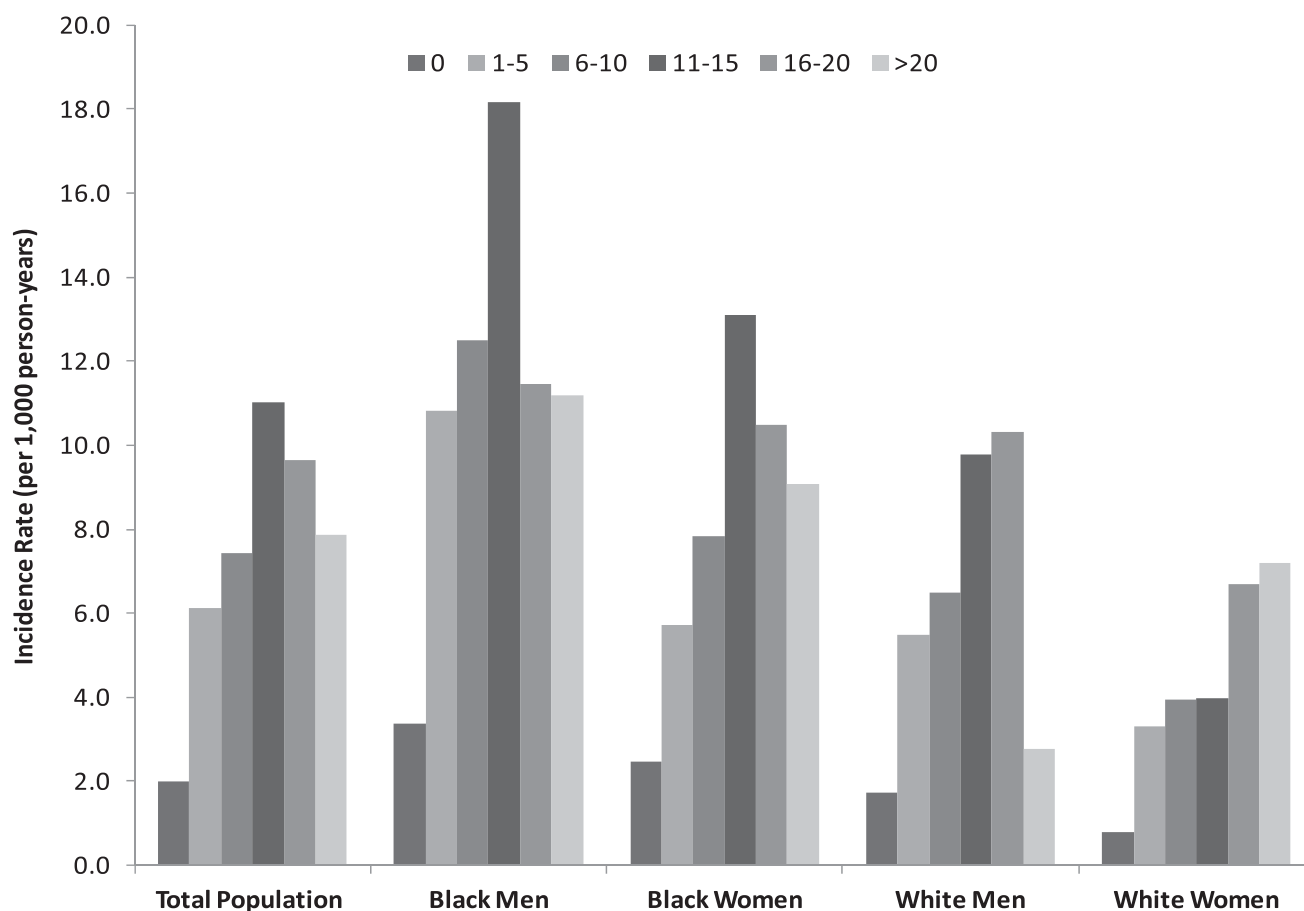
Figure 1—*Incidence of diabetes (per 1,000 person-years) according to duration of abdominal obesity in 5-year increments through year 25 (2010 to 2011) overall and according to race and sex: the CARDIA Study. See footnote of Table 3 for definition of incident diabetes and abdominal obesity.*

study of adults recruited and followed largely during the obesity epidemic over the last three decades in the United States, we found the duration of abdominal obesity starting in young adulthood and into middle age to be an important predictor of new-onset diabetes independent of the degree of abdominal adiposity, physical activity, energy intake, family history of diabetes, and a number of other potential confounding factors. Overall, each additional year of abdominal obesity was associated with a 4% higher risk of developing diabetes later in life. However, we found this risk appeared to peak with ~11–15 years of abdominal obesity, but remained elevated for >20 years. This finding suggests that the longer duration of exposure to excess abdominal adiposity as a result of the obesity epidemic and an earlier age at onset will have important implications on diabetes incidence rates in the United States.

To the best of our knowledge, the current study is the first to determine whether the duration of abdominal obesity

is associated with the development of diabetes. Previous studies examining the influence of the duration of excess adiposity on diabetes risk have measured only overall obesity (8–15). In order to quantify the duration of obesity, most of these studies have relied upon the recall of body weight at previous ages or the self-reported duration of obesity, methods that may be susceptible to a considerable amount of measurement error (9,10,13–15). However, in the current study, we used repeated assessments of WC collected every 2–5 years for a maximum of 25 years beginning early in adulthood to objectively determine the duration of abdominal obesity during follow-up. We found the duration of abdominal obesity was associated with the development of diabetes independent of the degree of abdominal adiposity. Thus, we suggest that future studies interested in estimating the cumulative exposure to excess abdominal adiposity over the life course measure not only the degree of abdominal adiposity, but also its duration.

In the present long-term study, we found the frequency of developing abdominal obesity was highest among black women. In addition, on average, black women developed abdominal obesity ~2 years earlier than black men and 3 to 4 years earlier than white men and women. As a result, black women experienced the longest duration of exposure to abdominal obesity of all race-sex groups. In contrast, men, either black or white, generally tended to have the lowest rates of abdominal obesity. These findings are consistent with recent data from the National Health and Nutrition Examination Survey showing a substantially higher prevalence of abdominal obesity among black women in the United States (5). Despite a longer duration of abdominal obesity and a higher incidence of diabetes among black women, we found little to no difference in the association between the duration of abdominal obesity and risk for diabetes between white and black men and women. Future studies are needed to confirm that black men and women

do not have a smaller relative risk for diabetes associated with abdominal obesity than their white counterparts.

Overall, we found the risk for diabetes appeared to peak during the first 11–15 years of abdominal obesity, but remained elevated for >20 years. In general, this quadratic trend was confirmed in all race-sex groups except white women, among whom the risk for diabetes peaked with >20 years of abdominal obesity. This finding suggests that the adverse metabolic effects of excess abdominal adiposity on diabetes risk, although elevated over the course of two decades, may begin to plateau after 11–15 years. Although the suspected mechanism to support this finding remains elusive, it may be possible that the number of individuals susceptible to the metabolic derangements of prolonged abdominal obesity decreases after approximately the first decade. A portion of the remaining abdominally obese individuals may be able to avoid hyperglycemia in the face of prolonged obesity by being either less responsive to the secretions of excess adipose tissue or their adipose tissue may not possess the same secretory properties of those abdominally obese individuals who develop diabetes. Additional studies are needed to confirm this finding.

It is generally well-accepted that excess adiposity, particularly abdominal adiposity, can have deleterious metabolic effects, thereby increasing the risk of developing diabetes. Expanded fat stores, a hallmark of obesity, results in enhanced lipolysis, leading to increased circulating free fatty acids, and promotes peripheral and hepatic insulin resistance (26). The normal pancreatic β-cell response to obesity-associated insulin resistance is compensatory insulin hypersecretion in order to maintain normoglycemia (27). Over time with prolonged obesity, β-cell dysfunction results in low insulin response and an inability to maintain glycemia near normal (28). Thus, the early stages of abdominal obesity may be largely characterized by the development of insulin resistance, whereas a prolonged duration of abdominal obesity may promote progressive deterioration in β-cell function, leading to the development of overt diabetes. Additional mechanisms that may explain, at least in part, the association between a longer duration of abdominal obesity and the development of diabetes, include sustained expression and secretion of hormones, cytokines, and adipokines from intra-abdominal fat located in close proximity to the portal circulation (29).

Strengths of our study include a community-based sampling method; a biracial cohort; extensive data on potential confounders; a large sample size well balanced with respect to age, sex, race, and education that increased precision and permitted simultaneous adjustment and stratification by multiple variables; repeat assessments of WC, glycemia, and potential confounding factors over a long follow-up period; a high retention rate; and the standardized data collection protocols and rigorous quality control of the CARDIA Study. Nevertheless, at least three limitations deserve mention. First, WC was used as an estimate of central obesity even though it does not distinguish between subcutaneous and visceral fat. Nevertheless, WC has been shown to be strongly correlated with visceral fat and offers widespread appeal due to its relative ease of measurement (30). Second, our estimation of the duration of abdominal obesity during follow-up was based on the measurement of WC every 2–5 years. It is likely that a more frequent number of assessments would have led to a more accurate estimation of the duration of abdominal obesity during follow-up; however, to the extent that there was random misclassification due to this assessment schedule, we may have underestimated the true association between the duration of abdominal obesity and diabetes risk in our cohort. Third, since our study collected data over a 25-year period, some participants were missing at least one eligible measurement of WC. However, we noted similar results between our multiple imputed datasets and our primary dataset that did not account for missing WC values.

In conclusion, our findings indicate that a longer duration of abdominal obesity is associated with a substantially higher risk for diabetes in adults independent of the degree of abdominal adiposity. This information is critical to understanding the consequences of a greater prevalence and cumulative exposure to excess adiposity over the life course. Our findings suggest that preventing or at least delaying the onset of abdominal obesity in young adulthood may substantially reduce the risk of developing diabetes into middle age.

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