



A Shift Toward a Plant-Centered Diet From Young to Middle Adulthood and Subsequent Risk of Type 2 Diabetes and Weight Gain: The Coronary Artery Risk Development in Young Adults (CARDIA) Study

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OBJECTIVE

To examine the associations between change in plant-centered diet quality and type 2 diabetes risk and change in body size.

RESEARCH DESIGN AND METHODS

A prospective study conducted in the U.S. enrolled adults ages 18–30 years in 1985–1986 (examination year [Y0]) and followed them through 2015–2016. We analyzed the associations between change in plant-centered diet quality over 20 years (Y0–Y20) and diabetes (Y20–30; $n = 2,534$) and change (Y0–Y20 and Y20–30) in BMI, waist circumference (WC), and weight ($n > 2,434$). Plant-centered diet quality was measured using the A Priori Diet Quality Score (APDQS); a higher score favors nutritionally rich plant foods. Cox regression models were used to assess diabetes risk, and linear regression models were used to examine change in body size.

RESULTS

During a mean follow-up of 9.3 (± 1.7) years, 206 case subjects with incident diabetes were observed. In multivariable analysis, participants with the largest increase in APDQS over 20 years had a 48% (95% CI 0.31–0.85; $P_{\text{trend}} < 0.001$) lower risk of diabetes over the subsequent 10 years compared with participants whose score remained stable. Each 1-SD increment in APDQS over 20 years was associated with lower gains in BMI (-0.39 kg/m^2 ; SE 0.14; $P = 0.004$), WC (-0.90 cm ; SE 0.27; $P < 0.001$) and weight (-1.14 kg ; SE 0.33; $P < 0.001$) during the same period, but not with subsequent changes.

CONCLUSIONS

Young adults who increased plant-centered diet quality had a lower diabetes risk and gained less weight by middle adulthood.

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Increased incidence of type 2 diabetes and obesity have been major public health problems in the U.S. Weight gain in young adulthood may lead to an earlier onset of type 2 diabetes and greater risk of heart disease and all-cause mortality (1,2). Recently, the EAT-Lancet Commission pointed to the benefits that a plant-centered diet has on health and the environment (3). Previous studies reported that a plant-centered diet was associated with a lower risk of type 2 diabetes and weight gain (4,5). However, only a few previous studies focused on change in diet quality over time (started following participants when they were middle-aged) (6–8). These studies showed that diets that generally emphasized plant foods were associated with a lower subsequent risk of type 2 diabetes and concurrent weight gain (6–8). Given the importance of the transition period from young to middle adulthood for preventing chronic disease, young adults may benefit from increasing plant-centered diet quality, as this may help to prevent type 2 diabetes and weight gain.

In the current study, diet quality was measured using the A Priori Diet Quality Score (APDQS); high index scores were characterized by higher consumption of nutritionally rich plant foods with limited consumption of meats and less healthful plant foods. Although no food is forbidden, a high score requires a variety of food choices, which in turn enhances flexibility. The APDQS embodies many principles of the 2015–2020 Dietary Guidelines for Americans and was found to predict risk of myocardial infarction, mortality, and other clinical outcomes (9–12). A previous study found a relationship between cumulative APDQS and type 2 diabetes over 30 years (11). The current study modeled both baseline and long-term change in diet quality from young to middle adulthood as exposures and incidence of type 2 diabetes and change in body size as outcomes. We hypothesized that baseline and 20-year change in plant-centered diet quality predict subsequent 10-year risk of type 2 diabetes, as well as concurrent and subsequent change in BMI, waist circumference (WC), and weight.

RESEARCH DESIGN AND METHODS

Study Design and Participants

Coronary Artery Risk Development in Young Adults (CARDIA) is a community-based, prospective cohort study of 5,115

Black and White men and women aged 18–30 years (1985–1986, examination year [Y0]) who were recruited from four U.S. cities (13). Study recruitment balanced the age, sex, race, and education of the participants within each study center. Nine examinations were conducted, with high retention over 30 years of follow-up among survivors (71% at Y30, 2015–2016). All participants provided written informed consent, and the research protocols were approved by institutional review boards at each CARDIA site.

For the 3,549 participants who attended CARDIA at Y20, exclusion criteria were: did not complete the dietary history questionnaire at Y0 and Y20 ($n = 409$); had an implausible energy intake (<800 or $>8,000$ kcal/day for men; <600 or $>6,000$ kcal/day for women; $n = 514$); or did not provide information regarding smoking status at Y0 or physical activity at Y0 or Y20 ($n = 43$). For the analysis of diabetes, participants were excluded who had diabetes in Y0–Y20 ($n = 332$) or were not examined at Y25 or Y30 (no assessment of incident type 2 diabetes, $n = 238$). For body size change, participants with missing BMI ($n = 628$), WC ($n = 631$), or weight ($n = 622$) at Y0, Y20, or Y30 or who reported bariatric surgery ($n = 81$) were excluded. The final sample number for diabetes analysis was 2,534 participants and 2,436 for BMI, 2,434 for WC, and 2,439 for weight. Comparison of the Y0 characteristics of excluded versus included participants showed that those excluded were more likely to report lower educational attainment, identify as Black race, and smoke cigarettes and to have lower APDQS and higher BMI and diabetes (Supplementary Table 1). Nevertheless, all population subgroups were well represented in those included.

Assessment of Plant-Centered Diet Quality

Diet was assessed using the interviewer-administered, validated CARDIA diet history questionnaire at Y0 and was updated at Y7 (1992–1993) and Y20 (2005–2006) (14,15). Interviewers asked 100 open-ended questions about food and beverage consumption over the past month. The frequency of consumption, unit or serving size, and preparation method were recorded. The number of food items that were collected was 950 at Y0, 1,388 at Y7, and 4,598 at Y20.

Plant-centered diet quality was measured by the APDQS, which is a hypothesis-driven index of 46 food groups. The APDQS is based on food groups classified as beneficial ($n = 20$), adverse ($n = 13$), and neutral ($n = 13$) according to their hypothesized relationship with cardiovascular disease. The scoring system of APDQS is based on quintile rankings of each of the 46 food groups (for food groups with large zero servings per day, a zero and quartiles above zero were used). The study-specific cut points derived at Y0 were applied to follow-up data, which allowed change in participants' dietary patterns to be tracked. Beneficially rated food groups were assigned points ranging from 0 (lowest quintile) to 4 (highest quintile), adversely rated food groups were assigned points ranging from 0 (highest quintile) to 4 (lowest quintile), and zero points were assigned to neutral food groups. The APDQS corresponded to the sum of the 46 component scores and had possible scores ranging between 0 and 132, with a range of 35–95 for the data collected in this study. For the current analysis, 20-year change in diet quality was subtracted from the value at Y0 from the value at Y20.

Supplementary Table 2 describes the feature of plant-centeredness in APDQS by comparing the mean intake of the 46 food groups between the extreme groups (“high initial and increased,” defined as at or above the median Y0 APDQS and quintile 5 of 20-year change vs. “low initial and decreased,” defined as below the median Y0 APDQS and quintile 1 of 20-year change). Supplementary Table 3 describes how the “high initial and increased” group made changes to their diet to obtain the highest increasing diet score over time. Notably, this group greatly increased their consumption of beneficially rated plant foods compared with the “low initial and decreased” group over a 20-year period. Subfood groups constituting the 46 food groups are shown in Supplementary Table 4.

Ascertainment of Type 2 Diabetes

Type 2 diabetes was diagnosed if fasting glucose concentration was ≥ 126 mg/dL, the 2-h postchallenge glucose concentration was ≥ 200 mg/dL (measured at Y10, Y20, and Y25), glycated hemoglobin (HbA_{1c}) was $\geq 6.5\%$ (48 mmol/mol) at Y20 and Y25, and/or use of antidiabetic medications was self-reported

(per medication bottle brought to clinic). Incident type 2 diabetes was diagnosed if this condition was first satisfied at Y25 or Y30.

Anthropometric Measurements

With participants barefoot and wearing light clothing, height was measured to the nearest 0.5 cm with a vertical ruler, weight was measured to the nearest 0.2 kg with a calibrated balance beam scale, and WC was measured to the nearest 0.5 cm with a tape in duplicate. BMI was calculated as weight divided by height squared (kg/m^2).

Assessment of Covariates

Standardized questionnaires were used to collect demographics, parental history of diabetes, smoking, and physical activity at all CARDIA exams. Physical activity level was assessed by a trained interviewer using the CARDIA physical activity history questionnaire (16). Participants were asked to report the frequency of 13 exercise activities over the previous year. The total score was the product of intensity and frequency.

Statistical Analysis

The focus of this study was change in diet quality from young to middle adulthood (over 20 years, Y0–Y20). Proportional hazards regression was used to estimate hazard ratios (HRs) and 95% CIs. Of main interest was the joint association of the continuous Y0 APDQS and 20-year change in APDQS with type 2 diabetes ($P_{\text{interaction}} = 0.94$). Model 1 was adjusted for age, sex, race (White or Black), and energy intake (Y0 and 20-year change). Model 2 was further adjusted for parental history of diabetes, physical activity level (tertiles; Y0 and 20-year change), Y0 smoking (never, former, and current), education, and BMI (Y0 and 20-year change). For the smoking variable, information at Y0 was used because the time-varying smoking status variable did not contribute to predictions. We evaluated the extent to which the relationship between 20-year change in APDQS and diabetes risk was mediated by concurrent change in BMI, WC, and weight. Mediation analysis estimated the degree of the mediation effect by comparing the models with and without the mediating variables and quantified the difference in estimates between these two models: $1 - (\beta_{\text{mediator model}}/\beta_{\text{base model}}) * 100$ (17). Goodness of linear fit of the

continuous models in the joint association with Y0 and 20-year change in APDQS was assessed in two ways. First, to visually assess the shape of association of Y0 APDQS and 20-year change in APDQS (both as continuous) with diabetes, restricted cubic splines with four knots were computed. Nonlinearity was tested using likelihood ratio tests by comparing two models: 1) model with the linear term and 2) the model with the linear and cubic spline terms in model 2 (18). Second, the combined effect of Y0 (median) and 20-year change (quintiles) in APDQS on diabetes incidence was estimated. A separate analysis was conducted that examined whether Y20 APDQS predicted diabetes over 10 years. We tested for interactions between 20-year change in APDQS and age, race, sex, and education.

Next, we evaluated the associations of Y0 APDQS and 20-year APDQS change with concurrent (Y0–Y20) and subsequent (Y20–Y30) change in BMI, WC, and weight. The multivariable models were adjusted for the same covariates as the diabetes analysis, omitting parental history of diabetes. Y0 BMI, WC, or weight was also adjusted in the model. Additional analyses were performed that examined whether Y0 APDQS predicted change in BMI, WC, or weight over 30 years and whether Y20 APDQS predicted the same outcomes over 10 years in which the same covariates were adjusted but only Y0 or Y20 variables were included in the models.

Sensitivity analyses examined whether short-term change (7-year change) in APDQS was consistently associated with diabetes risk or change in body size. The same modeling strategies were applied to this analysis as were applied to the analysis of 20-year change. In addition, individual food group analyses were conducted by fitting two main distinctive APDQS food groups regarding nutritional and health value, beneficially rated plant food groups (fruit, avocado, beans/legumes, green vegetables, yellow vegetables, tomatoes, other vegetables, nuts and seeds, soy products, whole grain, and vegetable oil), and adversely rated animal food groups (high-fat meats, processed meats, organ meats, fried fish/poultry, and sauces), with adjustment for the rest of the APDQS food groups in separate multivariable analyses. SAS software version 9.4 (SAS Institute Inc., Cary, NC) was used in all

analyses. Statistical tests were two-tailed, with significance at $P < 0.05$.

RESULTS

Characteristics

We identified 206 new case subjects with type 2 diabetes during mean follow-up of $9.3 (\pm 1.7)$ years after Y20 APDQS was measured. Twenty-year changes in APDQS were inversely related to Y0 APDQS, energy intake, and current smoking within both categories of below the median and at or above the median Y0 APDQS. Twenty-year changes in APDQS were positively related to the proportion of females and Whites and 20-year change in alcohol intake (Table 1). A greater increase in APDQS over 20 years was associated with a smaller decrease in the level of physical activity. Twenty-year changes in APDQS were negatively associated with 20-year change in WC, BMI, and weight only for at or above the median Y0 APDQS.

Prediction of Incident Type 2 Diabetes

Y20 APDQS predicted type 2 diabetes risk over 10 years (HR 0.68 [95% CI 0.57–0.80] per 1-SD increment in Y20 APDQS) (model 2 in Table 2). In joint predictor models, both Y0 and 20-year APDQS change predicted diabetes. Fully adjusted analyses showed a 37% (95% CI 0.51–0.78) reduction in diabetes risk per 1-SD increment in Y0 APDQS and a 29% (95% CI 0.59–0.86) decrease in diabetes risk per 1-SD increment in change in APDQS over 20 years. These patterns were reiterated in spline analysis (Supplementary Fig. 1). Mediation effect analyses showed that 31.5% (95% CI 12.3–60.0%; $P < 0.001$) of the association between 20-year change in APDQS and diabetes risk was explained by concurrent change in BMI, WC, and weight.

Added specificity is shown in Table 3, in which individuals who started with a higher plant-centered diet quality in young adulthood and increased the most over 20 years (at or above median Y0 APDQS and quintile 5 of 20-year change) had a 95% (95% CI 0.01–0.44) lower subsequent 10-year risk of diabetes compared with those who had a lower diet quality in young adulthood and remained stable (<median Y0 APDQS and quintile 2 of 20-year change) over 20 years. However, these results should be cautiously interpreted due to the small sample size of this category. The cumulative incidence of diabetes

Table 1—Characteristics of the participants according to 20-year change* in APDQS†, stratified by Y0 APDQS (n = 2,534)

Characteristics	Below the median Y0 APDQS			At or above the median Y0 APDQS		
	20-year change in APDQS			20-year change in APDQS		
	Quintile 1 (n = 126)	Quintile 3 (n = 252)	Quintile 5 (n = 377)	Quintile 1 (n = 363)	Quintile 3 (n = 288)	Quintile 5 (n = 112)
APDQS						
Y0	57.4 ± 4.6	53.9 ± 6.3	51.1 ± 7.4	78.7 ± 9.3	73.7 ± 7.3	69.7 ± 5.3
20-year change*	−7.9 ± 3.9	6.6 ± 1.8	23.7 ± 6.1	−9.6 ± 5.7	6.5 ± 1.7	21.3 ± 4
Y0 age, years	24.6 ± 3.5	24.6 ± 3.7	23.7 ± 3.8	25.9 ± 3.3	25.9 ± 3.1	25.6 ± 3.2
Female, n (%)	55 (43.7)	146 (57.9)	215 (57)	215 (59.2)	166 (57.6)	74 (66.1)
White race, n (%)	44 (34.9)	80 (31.8)	141 (37.4)	262 (72.2)	241 (83.7)	93 (83)
Highest grade of education, years	14.7 ± 2.6	15 ± 2.6	15.7 ± 2.6	16.5 ± 2.4	16.8 ± 2.4	17 ± 2.3
Parental history of diabetes, n (%)	36 (28.6)	80 (31.8)	106 (28.1)	82 (22.6)	53 (18.4)	28 (25)
Physical activity, EU‡						
Y0	401 ± 331	344 ± 269	382 ± 278	483 ± 327	486 ± 285	451 ± 248
20-year change*	−148 ± 305	−81.3 ± 248	−20.6 ± 283	−151 ± 310	−71.8 ± 285	16.2 ± 274
Y0 current smoker, n (%)	43 (34.1)	71 (28.2)	79 (21)	86 (23.7)	63 (21.9)	19 (17)
Alcohol intake, drinks/day						
Y0	1.08 ± 1.68	0.64 ± 1.28	0.46 ± 0.84	1.1 ± 1.43	0.94 ± 1.12	1.04 ± 1.44
20-year change*	−0.67 ± 1.72	−0.05 ± 1.65	0.31 ± 1.17	−0.26 ± 1.49	0.1 ± 1.41	0.23 ± 1.45
Total energy intake, kcal/day						
Y0	3,253 ± 1,573	2,892 ± 1,425	2,873 ± 1,283	2,658 ± 1,146	2,507 ± 1,108	2,466 ± 1,061
20-year change*	−488 ± 1,473	−634 ± 1,334	−578 ± 1,324	−325 ± 1,126	−251 ± 1,014	−141 ± 1,104
Case subjects with incident type 2 diabetes (Y20–Y30), n (%)	19 (15.1)	31 (12.3)	28 (7.4)	26 (7.2)	16 (5.6)	2 (1.8)
BMI, kg/m ²						
Y0	25.2 ± 5.6	24.6 ± 5	23.9 ± 4.4	23.9 ± 3.9	23.3 ± 3.8	23.1 ± 4.5
20-year change*	5.3 ± 4	5.7 ± 6.4	5.3 ± 4.3	4.6 ± 4.5	3.8 ± 4	4.1 ± 11
WC, cm						
Y0	80.1 ± 11.9	77.8 ± 10.7	76 ± 9.8	76.1 ± 9.7	75.5 ± 9.4	74.5 ± 9.9
20-year change*	14.8 ± 8.6	15.6 ± 12	14.4 ± 9.3	13.4 ± 10.3	11.5 ± 9.7	9.4 ± 7.8
Weight, kg						
Y0	74.1 ± 17.1	71.0 ± 15.1	69.0 ± 14.2	69.0 ± 14.0	68.4 ± 13.6	66.4 ± 13.2
20-year change*	15.8 ± 11.2	16.5 ± 15.1	15.7 ± 11.8	13.7 ± 13.1	11.3 ± 11.5	9.4 ± 10.2

Data are mean ± SD unless otherwise indicated. Median cut point of Y0 APDQS was 64. *The 20-year change was calculated by subtracting the Y0 values from the follow-up values at Y20. †Total score sums the 46 components (possible scores 0–132, with a range of 35–95 in these data), with higher scores representing a nutritionally rich, plant-centered diet. A one-point increment represents a one-category shift in the presumed favorable direction. ‡EU is exercise units, a physical activity score derived from the CARDIA physical activity history.

corresponding to these two categories was 12.2% and 1.8%, respectively, which is an ~5.8-fold difference in the incidence rate. Similar, but weaker, inverse association was observed for 7-year change in APDQS in relation to risk of diabetes in the subsequent 23-year period (Supplementary Tables 5 and 6). We found no difference in the association by stratified variables, including age, race, sex, and education ($P_{\text{interaction}} > 0.05$ for each). In analyses of individual food groups, beneficially rated plant foods were inversely associated with diabetes (HR 0.45 [95% CI 0.26–0.78] for Y0 and HR 0.84 [95% CI 0.60–1.17] for 20-year change), whereas adversely rated animal foods were positively associated with diabetes (HR 2.26 [95% CI 1.18–4.30] for Y0 and HR 2.25 [95% CI 1.23–4.13] for 20-year change).

Prediction of Change in Measures of Body Size

The mean increase in BMI, WC, and weight over 30 years was 6.1 kg/m² (± 5.2), 18.6 cm (± 12), and 17.1 kg (± 15.0), respectively. Y0 APDQS predicted a smaller increase in body size over 30 years; the adjusted $\beta \pm$ SE per 1 SD was -0.64 ± 0.13 kg/m² ($P < 0.001$) for BMI, -1.58 ± 0.3 cm for WC ($P < 0.001$), and -1.62 ± 0.36 kg for weight ($P < 0.001$).

In models that simultaneously adjusted for Y0 APDQS and 20-year change in APDQS, 20-year change in APDQS was associated with a smaller concurrent increase in BMI (-0.39 kg/m²; SE 0.14; $P = 0.004$), WC (-0.90 cm; SE 0.27; $P < 0.001$), and weight (-1.14 kg; SE 0.33; $P < 0.001$), and Y0 APDQS more strongly predicted change in BMI (-0.65 kg/m²; SE 0.14; $P < 0.001$), WC (-1.35 cm; SE 0.29;

$P < 0.001$), and weight (-1.65 kg, SE 0.36; $P < 0.001$) than 20-year change in APDQS (Table 4). Y0 APDQS, but not 20-year change in APDQS, was associated with a lower increase in BMI (-0.37 kg/m²; SE 0.11; $P < 0.001$), WC (-0.77 cm; SE 0.22; $P < 0.001$), and weight (-0.61 kg; SE 0.25; $P = 0.014$) in the subsequent period. The results were similar for 7-year change in APDQS (Supplementary Table 7).

CONCLUSIONS

The current study showed that increased plant-centered diet quality over 20 years, beginning in young adulthood, was associated with a lower risk of type 2 diabetes by middle adulthood. The current study extends a previous analysis of the same database, which showed that the cumulative average APDQS was associated with the risk of type 2 diabetes

Table 2—HR (95% CI) of incident type 2 diabetes according to quintile of 20-year change, Y0, or Y20 APDQS (N = 2,534)

	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5	Each 1-SD (13-point) increment*	P for trend†
Joint predictor model (both APDQS variables in one model)							
20-year change in APDQS predicting type 2 diabetes in the subsequent 10 years							
Range	Less than -2	-2 to 3	4-9	10-16	≥17		
N of case subjects/N at risk (unadjusted cumulative incidence rate)	45/489 (9.2)	40/488 (8.2)	47/540 (8.7)	44/528 (8.3)	30/489 (6.1)		
Model 1: basic model‡	1.37 (0.89-2.12)	1 (reference)	0.97 (0.63-1.48)	0.83 (0.54-1.28)	0.50 (0.31-0.82)	0.66 (0.55-0.79)	<0.001
Model 2: fully adjusted model‡	1.30 (0.84-2.01)	1 (reference)	1.02 (0.66-1.55)	0.90 (0.58-1.40)	0.52 (0.31-0.85)	0.71 (0.59-0.86)	<0.001
Y0 APDQS predicting type 2 diabetes in the subsequent 10 years, after a 20-year lag							
Range	<53	53-59	60-66	67-76	≥77		
N of case subjects/N at risk (unadjusted cumulative incidence rate)	72/533 (13.5)	40/457 (8.8)	41/504 (8.1)	33/548 (6.0)	20/492 (4.1)		
Model 1: basic model‡	1 (reference)	0.56 (0.38-0.84)	0.50 (0.33-0.76)	0.39 (0.24-0.61)	0.24 (0.13-0.43)	0.59 (0.49-0.72)	<0.001
Model 2: fully adjusted model‡	1 (reference)	0.59 (0.40-0.88)	0.53 (0.35-0.81)	0.41 (0.25-0.66)	0.31 (0.17-0.58)	0.63 (0.51-0.78)	<0.001
Single predictor models							
Y20 predicting type 2 diabetes over 10 years							
Range	<60	60-67	68-74	75-82	≥83		
N of case subjects/N at risk (unadjusted cumulative incidence rate)	67/494 (13.6)	63/509 (12.4)	33/519 (6.4)	26/497 (5.2)	17/515 (3.3)		
Model 1: basic model§	1 (reference)	0.96 (0.68-1.35)	0.50 (0.33-0.76)	0.44 (0.27-0.70)	0.28 (0.16-0.50)	0.63 (0.54-0.73)	<0.001
Model 2: fully adjusted model	1 (reference)	0.97 (0.69-1.38)	0.59 (0.38-0.91)	0.57 (0.35-0.93)	0.33 (0.18-0.61)	0.68 (0.57-0.80)	<0.001

* A one-point increment represents a one-category shift in the presumed favorable direction. †Model 1: Y0 APDQS and 20-year change in APDQS were fitted simultaneously in the model. Covariates included age (Y0), sex, race (White or Black), and total energy intake (Y0 and 20-year change). ‡Model 2: model 1 plus parental history of diabetes (yes or no), physical activity level (Y0 and 20-year change), smoking status (Y0; never, former, and current), highest grade of education achieved during follow-up, and BMI (Y0 and 20-year change). §Model 1: age (Y20), sex, race (White or Black), and total energy intake (Y20). ||Model 2: model 1 plus parental history of diabetes (yes or no), physical activity level (Y20; tertiles), smoking status (Y20; never, former, and current), highest grade of education achieved during follow-up, and BMI (Y20). ¶IP for interaction = 0.94, tested with the multiplicative term of 20-year change in APDQS (continuous) and Y0 APDQS (continuous) in model 2.

Table 3—Subsequent 10-year HR (95% CI) of incident type 2 diabetes according to joint classification of Y0 APDQS and 20-year change in APDQS (n = 2,534)

	20-year change in APDQS				
	Quintile 1	Quintile 2	Quintile 3	Quintile 4	Quintile 5
Below the median of Y0 APDQS (n = 1,272)					
Range	Less than -2	-2 to 3	4-9	10-16	≥17
N of case subjects/N at risk (unadjusted cumulative incidence rate)	19/126 (15.1)	23/189 (12.2)	31/252 (12.3)	35/328 (10.7)	28/377 (7.4)
Unadjusted	1.27 (0.69-2.33)	1 (reference)	1.03 (0.60-1.76)	0.87 (0.52-1.47)	0.60 (0.34-1.04)
Model 1: basic model*	1.21 (0.66-2.23)	1 (reference)	1.00 (0.59-1.72)	0.93 (0.55-1.57)	0.62 (0.36-1.08)
Model 2: fully adjusted model†	1.22 (0.66-2.24)	1 (reference)	1.03 (0.60-1.77)	1.00 (0.59-1.71)	0.72 (0.41-1.27)
At or above the median of Y0 APDQS (n = 1,262)					
Range	Less than -2	-2 to 3	4-9	10-16	≥17
N of case subjects/N at risk (unadjusted cumulative incidence rate)	26/363 (7.2)	17/299 (5.7)	16/288 (5.6)	9/200 (4.5)	2/112 (1.8)
Unadjusted	0.58 (0.33-1.02)	0.45 (0.24-0.85)	0.44 (0.23-0.83)	0.37 (0.17-0.79)	0.14 (0.03-0.60)
Model 1: basic model*	0.70 (0.39-1.24)	0.57 (0.30-1.07)	0.58 (0.30-1.12)	0.47 (0.22-1.04)	0.19 (0.04-0.80)
Model 2: fully adjusted model†	0.77 (0.43-1.37)	0.68 (0.36-1.30)	0.79 (0.40-1.53)	0.69 (0.31-1.54)	0.05 (0.01-0.44)

Median cut point of Y0 APDQS was 64. *Model 1: age (Y0), sex, race (White or Black), and total energy intake (Y0 and 20-year change). †Model 2: model 1 plus parental history of diabetes (yes or no), physical activity level (Y0 and 20-year change; tertiles), smoking status (Y0; never, former, and current), highest grade of education achieved during follow-up, and BMI (Y0 and 20-year change).

over a 30-year period, with an HR of 0.55 that compared quartile 4 versus quartile 1 (11). In the current study, participants with the greatest increase in plant-centered diet score over 20 years had a 48% lower risk of type 2 diabetes over the next 10 years as compared with those with a stable score. Furthermore, individuals who had a higher diet quality in young adulthood and increased over time showed a greater decrease in risk for

diabetes. Increased diet quality over 20 years was associated with lower gains in BMI, WC, and weight.

The global data showed that increased consumption of plant-centered diets predicted a 23.6% reduction in deaths (11.6 million/year) and an 80% reduction in greenhouse gas emissions (3). Our findings are consistent with a meta-analysis of nine prospective studies that showed an inverse association between

the baseline level of a healthy plant-centered diet and risk of type 2 diabetes (4). Two prospective studies investigated the relationship between change in diet quality (measured using dietary guideline-based indices) and the subsequent diabetes risk in middle-aged adults (6,19). One of these studies found that 4-year increase in diet quality (assessed using the Alternate Healthy Eating Index-2010 [AHEI-2010] score) was associated with an 11% lower subsequent 4-year risk of diabetes for every 10% increment in the score (6). Similar to our findings, this study demonstrated that the association between change in diet quality and subsequent risk of diabetes was modestly explained by change in weight (32%). In contrast, the other study found no association between change in diet quality (assessed using the Health Eating Index-2015 [HEI-2015] and the AHEI-2010) over 6 years and risk of type 2 diabetes over the following 18 years (19). Few data exist regarding long-term change in plant-centered diet quality and subsequent risk of type 2 diabetes in young adults. Our data support dietary patterns that emphasize plant foods, but also allow for some lean meats and low-fat dairy, as suggested by the 2015-2020 Dietary Guidelines for Americans (9). The APDQS strongly correlated with HEI-2015 (age-sex-adjusted correlation coefficient was 0.73 in CARDIA participants) and predicted clinical outcomes (10-12). Based on our data and previous studies of individual foods, we propose making

Table 4—Adjusted mean change* in BMI and WC per 1-SD increment in 20-year change, Y0, or Y20 APDQS

Each 1-SD (13-point) increment	Concurrent 20-year change (Y0-Y20)			Subsequent 10-year change (Y20-Y30)		
	β	SE	P value	β	SE	P value
BMI, kg/m² (n = 2,436)						
20-year change (Y0-20)†	-0.39	0.14	0.004	-0.17	0.10	0.09
Y0‡	-0.65	0.14	<0.001	-0.37	0.11	<0.001
Y20‡	—	—	—	-0.24	0.09	0.008
WC, cm (n = 2,434)						
20-year change (Y0-20)†	-0.90	0.27	<0.001	-0.10	0.21	0.64
Y0‡	-1.35	0.29	<0.001	-0.77	0.22	<0.001
Y20‡	—	—	—	-0.31	0.19	0.10
Weight, kg (n = 2,439)						
20-year change (Y0-20)†	-1.14	0.33	<0.001	-0.05	0.23	0.84
Y0‡	-1.65	0.36	<0.001	-0.61	0.25	0.014
Y20‡	—	—	—	-0.20	0.21	0.32

*Multivariable linear models are reported. Model was adjusted for current age, sex, race (White or Black), total energy intake (Y0 and 20-year change), smoking status (Y0; never, former, and current), physical activity level (Y0 and 20-year change; tertiles), and highest grade of education achieved during follow-up. †Joint predictor models. Each model included Y0 APDQS and 20-year change in APDQS. Depending on outcome variables, the following variables were also included in the model: Y0 BMI for concurrent 20-year change in BMI, Y20 BMI for subsequent 10-year change in BMI, Y0 WC for concurrent 20-year change in WC, and Y20 WC for subsequent 10-year change in WC, Y0 weight for concurrent 20-year change in weight, and Y20 weight for subsequent 10-year change in weight. ‡Single predictor models.

nutritionally rich plant foods the central feature of a diet. Nevertheless, modest amounts of dairy, fish/seafood, poultry, and eggs may be flexibly incorporated into the diet while achieving reduced diabetes risk. A meta-analysis of prospective studies reported that total dairy (whole and low fat) was inversely associated with incident type 2 diabetes (20); particularly, risk of diabetes decreased by 14% with increased yogurt intake, up to 80 g/day. There was no association between egg consumption and type 2 diabetes risk comparing the highest versus lowest intake in meta-analysis, but modestly increased risk was observed for more than three eggs per week only among U.S. populations (21). Another meta-analysis study shows no evidence of an increased risk of type 2 diabetes with intake of fish, shellfish, and poultry (22,23). Additional studies are needed to clarify to what extent individual animal products may partially affect the risk of diabetes.

Prior observational studies demonstrated that adherence to plant-centered dietary patterns at baseline was associated with lower weight gain (5,24). Likewise, a meta-analysis of 12 clinical trials found that vegetarian diets were associated with weight reduction in the general population (25). However, the duration of the studies in the meta-analysis were short (median 18 weeks), and the association was weaker for subjects with longer follow-up (≥ 1 year) than for subjects with shorter follow-up (< 1 year), which complicates the interpretation of the long-term effects. Pooled data from three U.S. prospective cohort studies showed that increased diet quality at each 4-year interval over 20 years (assessed using the AHEI-2010, the Alternate Mediterranean Diet, and the Dietary Approaches to Stop Hypertension) was associated with a lower concurrent weight gain (-0.47 kg, -0.23 kg, and -0.42 kg, respectively, for each 1-SD increment in these indices) (7). Later, the same U.S. prospective cohort studies described the relationship between change in plant-based diet indices (PDI) over 4 years and concurrent weight change during the same period (8). The study found that each 1-SD increment in the healthful PDI (positive scores were assigned to nutritious plant foods and negative scores to animal foods) was associated with 0.68 kg less weight gain, whereas each 1-SD

increment in the unhealthful PDI (positive score assigned to highly processed plant foods and negative scores to animal foods) was associated with 0.36 kg more weight gain. However, these studies relied on self-reported weight measurements as opposed to the preferred objective measurements and may bias the relationship due to misclassification effects (7,8). Another study showed that increased diet quality (measured using the Alternate Mediterranean Diet score) was associated with a lower ectopic fat gain in the following 6 years (26). Similarly, increase in plant-centered diet quality (assessed using the APDQS) was associated with less weight gain from adolescence to early adulthood (12).

Some methodological uniqueness of the APDQS merits further discussion. Previous studies were mostly based on self-identified vegetarians (dichotomous form) or rare eaters of meats rather than on total diet quality (4). However, this simplified definition of a plant-centered diet overlooks the remaining parts of an individual's diet and is likely to result in misclassification bias. The way that the APDQS is structured allows flexible choice by providing a wide range of options and emphasizing variety (46 groups). Diversified food groups were equally weighted in the APDQS, with a maximum of four points; therefore, points need to come from many food groups to achieve a higher score. In contrast, the HEI-2015 and the AHEI-2010 each rely on a small number of food groups (≤ 13) and are limited by the fact that individuals can get many points from single food groups and would not lose many points for eating less healthy foods.

The health benefits of plant-centered diets may relate to food synergy, defined as the concerted action of nutrients and bioactive compounds in individual foods, in meals and over time (27). This theory states that it is challenging to disentangle one factor from various food constituents to understand the protective effects of plant-centered diets. Nevertheless, constituents may be conceptually important. Fruits, vegetables, whole grains, and nuts contain abundant and varied amounts of antioxidants (vitamins A, C, and E and β -carotene), dietary fibers, and polyphenols (28,29). Antioxidants may protect against progressive pancreatic β -cell impairment and endothelial dysfunction, which may plausibly reduce diabetes risk (30,31). Polyphenols have antioxidant, anti-inflammatory,

and antiplatelet properties and also lead to improvements in endothelial function, insulin sensitivity, and blood pressure (32). Dietary fiber in plant foods (soluble and insoluble) contributes to increased postprandial satiety, suppressed subsequent hunger, and decreased energy intake (33). Soluble fiber may also attenuate the rapid rise in the postprandial glucose response after a meal (34). All of these factors may partially explain the beneficial effect of a plant-centered diet on diabetes and weight control.

Strengths of the current study include its prospective design with high participant retention over a long follow-up, standardized and repeated assessment of covariates, detailed assessment of diet through the diet history interviewer, and collecting objective information on case subjects with incident type 2 diabetes and anthropometric measurements. Change analysis can strengthen temporality assertions and eliminate within-person confounding. Although change analysis is more challenging to interpret compared with baseline analysis, in particular because of the regression to the mean phenomenon, explicit consideration of baseline levels tends to resolve this difficulty. Our study also has several limitations. Observational studies cannot establish causal relationships, partly because of unmeasured or residual confounding. However, we adjusted for potential confounders for the association between diet and diabetes and body size. Selection bias may have occurred, but some participants had to be excluded in order to analyze the 20-year change in diet quality and preserve the quality of the data. The potential fluctuations in dietary pattern over time merit further investigation. In the current study, change in diet quality was estimated from two points in examination years (Y0 and Y20) and thus may not have fully captured change in dietary pattern at the middle point during the study follow-up. Thus, potential misclassification may attenuate the association toward the null. However, prior CARDIA analysis showed that CARDIA participants had stable dietary patterns over 20 years, with tracking coefficients of 0.57 for Whites and 0.43 for Blacks (between Y0 and Y20 APDQS) (35). Moreover, analysis of short-term change in APDQS corroborated the main findings of the association between 20-year change in APDQS and diabetes and change in

body size. We caution that differences in culture, race/ethnicity, and period of life can influence participant behaviors and may limit the generalizability of the results.

In conclusion, these findings provide evidence that young adults who consumed a plant-centered, high-quality diet and increased their diet quality over time had a lower risk of type 2 diabetes and smaller increases in body size as they aged through middle age. Our study suggests that eating a plant-centered diet may reduce early risk of type 2 diabetes and obesity.

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