Dietary Patterns During Adolescence and Risk of Type 2 Diabetes in Middle-Aged Women

VASANTI S. MALIK, SCD¹ TERESA T. FUNG, SCD^{1,2} ROB M. VAN DAM, PHD^{1,3,4,5} ERIC B. RIMM, SCD^{1,3,4} BERNARD ROSNER, PHD⁴ FRANK B. HU, MD, PHD^{1,3,4}

OBJECTIVE—Whether dietary habits early in life can affect risk of type 2 diabetes (T2DM) in adulthood is unknown. We evaluated the relationship between dietary patterns during adolescence and risk of T2DM in midlife.

RESEARCH DESIGN AND METHODS—We examined the 7-year incidence of T2DM in relation to dietary patterns during high school among 37,038 participants in the Nurses' Health Study II cohort, who completed a food-frequency questionnaire about their diet during high school. Dietary patterns were derived by factor analysis. Cox proportional hazards regression was used to estimate relative risk (RR) and 95% CI.

RESULTS—The prudent pattern, characterized by healthy foods, was not associated with risk of T2DM. The Western pattern, characterized by desserts, processed meats, and refined grains, was associated with 29% greater risk of T2DM (RR 1.29; 95% CI 1.00–1.66; *P* trend 0.04), after adjusting for high school and adult risk factors comparing extreme quintiles, but was attenuated after adjusting for adult weight change (1.19; 0.92–1.54). Women who had high Western pattern scores in high school and adulthood had an elevated risk of T2DM compared with women who had consistent low scores (1.82; 1.35–2.45), and this association was partly mediated by adult BMI (1.15; 0.85–1.56).

CONCLUSIONS—A Western dietary pattern during adolescence may increase risk of T2DM in later life, partly through adult weight gain. Preventive measures should be aimed at developing healthy dietary habits that begin in early life and continue through adulthood.

Diabetes Care 35:12-18, 2012

A lthough obesity is the most important risk factor for type 2 diabetes (T2DM), epidemiologic studies have identified a number of foods that can also modulate risk independent of energy balance. For example, diets low in whole grains or fiber and high in glycemic load and processed meats have been shown to increase risk (1,2). As a complementary approach to evaluating the effects of individual foods or nutrients, evaluation of dietary patterns in relation to T2DM risk has become increasingly popular. The

overall dietary pattern may affect T2DM risk more than individual components because of interactions between nutrients, and physical properties of foods (3). Dietary patterns also reflect the manner in which foods are consumed, which may better facilitate dietary change and translation of findings into dietary recommendations. Factor analysis has emerged as a useful tool for characterizing dietary patterns in nutritional epidemiology. Previous studies have shown consistent positive associations between the

Western dietary pattern and weight gain (4) and risk of T2DM (3,5).

To date, T2DM research has generally focused on identification of risk factors that operate during adulthood. However, accumulating evidence suggests that exposures operating during early life, such as maternal diet during pregnancy, and childhood and adolescent diet may affect the risk of developing chronic diseases including T2DM (6). Such a life-course approach could enhance our understanding of T2DM etiology and complement current prevention strategies. Data on adolescent diet in relation to future risk of T2DM are sparse, and, to our knowledge, no study has investigated whether particular dietary patterns at early stages in the life course can affect risk of T2DM in adulthood. Therefore, we evaluated the relation between major dietary patterns during high school and incident T2DM \sim 2 decades later in a large cohort of U.S. women. We further examined the joint effect of high school and adult dietary patterns on risk of developing T2DM.

RESEARCH DESIGN AND METHODS

Study population

The Nurses' Health Study II (NHS II) is a prospective cohort of 116,671 female registered nurses aged 24-44 years at baseline in 1989. This cohort is followed using biennial mailed questionnaires on lifestyle, diet, and medical history. The study has maintained a response rate of \geq 90% for every 2-year period (7). In 1997, participants were asked if they would be willing to complete a questionnaire about diet during high school (HS-FFQ [High School Food Frequency Questionnaire]), at which time they were 34-53 years of age. Approximately 61% of the cohort indicated willingness (n = 64,380). Comparison of these women with the entire cohort suggests that they do not differ with regard to baseline dietary intake or T2DM risk factors. This analysis is restricted to the 47,355 women who returned the HS-FFQ in 1998 (73% of those sent the questionnaire). We excluded participants if they reported implausible

From the ¹Department of Nutrition, Harvard School of Public Health, Boston, Massachusetts; the ²Department of Nutrition, Simmons College, Boston, Massachusetts; the ³Department of Epidemiology, Harvard School of Public Health, Boston, Massachusetts; the ⁴Channing Laboratory, Department of Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, Massachusetts; and the ⁵Departments of Epidemiology and Public Health and Medicine, Faculty of Medicine, National University of Singapore, Singapore. Corresponding author: Vasanti S. Malik, vmalik@hsph.harvard.edu.

Received 25 February 2011 and accepted 11 October 2011.

DOI: 10.2337/dc11-0386

This article contains Supplementary Data online at http://care.diabetesjournals.org/lookup/suppl/doi:10 .2337/dc11-0386/-/DC1.

^{© 2012} by the American Diabetes Association. Readers may use this article as long as the work is properly cited, the use is educational and not for profit, and the work is not altered. See http://creativecommons.org/ licenses/by-nc-nd/3.0/ for details.

daily caloric intakes of <500 or $\geq 3,500$ kcal/day for current intake levels (n =2,920) and <500 or $\geq 5,000$ kcal/day for high school intake levels (n = 1,181), or left \geq 10 items blank (*n* = 225). In addition, we excluded participants if they had a confirmed diagnosis of diabetes (n = 502)T2DM, n = 42 type 1 diabetes) or death (n = 1) before return of the HS-FFQ in 1998, were missing the date of diagnosis of diabetes (n = 616), or had a history of diabetes (n = 137), gestational diabetes (n = 1,875), cancer except nonmelanoma skin cancer, or cardiovascular disease (n =2,818) reported in 1999. After exclusions, a total of 37,038 participants remained for the analysis.

Assessment of high school and adult dietary patterns

High school dietary intake was assessed using the HS-FFQ, a 124-item FFQ that asked participants about their diet during high school. Subjects were asked how often they consumed a specified amount of each item using nine responses ranging from "never or less than once per month" to "six or more per day." Categories on the HS-FFQ included main dishes, bread and cereals, fruits, vegetables, snack foods, and dairy. The HS-FFQ was designed to include foods that were consumed when these women were in high school. Nutrient intakes on the HS-FFQ were computed by multiplying the frequency of consumption of each unit of food by the nutrient content of the specified portions and then summing the contributions. Nutrient values were obtained from the U.S. Department of Agriculture and food manufacturers. Recall of adolescent diet among NHS II participants and a similar cohort was reproducible (8,9), and the HS-FFQ has shown reasonable validity (9,10).

Adult dietary patterns were assessed by ~131-item FFQs administered in 1991, 1995, 1999, and 2003. These FFQs assessed usual dietary intake during the past year, and food items and response categories were similar to the HS-FFQ. The validity of FFQs similar to those used in the NHS II was reasonable (11).

To identify dietary patterns, food items on the FFQs were first aggregated into 37 food groups for the HS-FFQ and 40 food groups for the NHS II FFQs, on the basis of similarities in nutrient profile and food preferences. Similar food groupings were used previously (5). Factor analysis with the orthogonal rotation procedure varimax was applied to the food groups to generate dietary patterns (12).

Briefly, factor analysis aggregates correlated variables, and each obtained factor (dietary pattern) is a linear combination of all food groups that are weighted by their factor loadings and explains as much variation in the food groups as possible (12). Food groups with positive loadings contribute to the dietary pattern, whereas those with negative loadings are inversely associated. Food groups with absolute factor loadings ≥ 0.30 were considered as significantly contributing to the pattern (12). Similar to previous studies, our analysis retained two factors, labeled "prudent" and "Western" patterns, on the basis of eigenvalues (>1), Scree tests (a graphical representation of eigenvalues >1), and interpretability of the factors (5). Scree tests allowed us to identify two major patterns with the largest eigenvalues (>2.75). Dietary pattern scores were calculated by summing the standardized intake of food groups, weighted by the factor loading of the food groups (12). These scores were used to rank participants according to the degree to which they conformed to a given dietary pattern. The reproducibility and validity of dietary patterns derived by this method were reasonable (13). Because of potential overreporting or underreporting on the FFQs, dietary patterns were adjusted for total energy intake using the residual method (14).

Outcome assessment

Women reporting a new diagnosis of diabetes on any of the biennial questionnaires were sent supplementary questionnaires to confirm the self-report. We used the American Diabetes Association criteria for diagnosis (fasting glucose levels ≥7 mmol [126 mg/dL]). In substudies of the NHS, 98% of self-reported diabetes cases documented by the same supplementary questionnaire were confirmed by medical record review (15).

Statistical analysis

Dietary pattern scores were divided into quintiles, and Cox proportional hazards regression was used to estimate incidence rate ratios comparing each quintile to the lowest. Person-time of follow-up was calculated for each participant from 1997 until June 2005, or date of T2DM diagnosis, or death. Test for trends across quintiles was conducted by modeling the median value for each category as a continuous variable. In the analysis of high school dietary patterns, multivariate models were adjusted for BMI at age 18

Malik and Associates

years (<18.5, 18.5 to <25, 25 to <30, or \geq 30 kg/m²), total energy intake in high school (quintiles, kcal/day), smoking between ages 15 and 19 years (none or 1-4, 5-14, or 15+ cigarettes/day), and high school physical activity (quintiles, metabolic equivalents [Mets]/week). We also adjusted for adult risk factors, including current smoking status (never, past, current: 1-14 cigarettes/day, ≥ 15 cigarettes/ day), current physical activity (quintiles, Mets/week), history of T2DM in parents or siblings (yes, no), oral contraceptive use (never, past, current), postmenopausal hormone use (never, ever), total energy (quintiles, kcal/day), and alcohol (0, 0.1-4.9, 5–9.9, \geq 10 g/day). Sensitivity tests were conducted by additionally adjusting for current prudent or Western dietary pattern and weight change since age 18 years, which are potential mediators. Effect modification of the association between high school dietary pattern and T2DM by BMI at age 18 years, and physical activity during high school, was also assessed. Interaction tests were performed by including a product term with the respective stratification variable and the median score of dietary pattern quintiles as a continuous variable and examining Wald *P* values. The interaction between high school and adult dietary patterns was evaluated by modeling a cross-product term using median values of tertiles.

To evaluate current dietary patterns in relation to T2DM risk, the cumulative average of patterns obtained from the 1991, 1995, 1999, and 2003 FFQ cycles were used, and multivariate models were adjusted for adult risk factors described above. Current BMI (<23, 23-24.9, 25-26.9, 27-28.9, 29-30.9, 31-32.9, 33-34.9, 35-36.9, 37-38.9, 39-40.9, 41-42.9, 43-44.9, \geq 45 kg/m²) and hypertension (yes, no) were additionally added to models. Cumulative averages were used to reduce random within-person variation and best reflect long-term dietary intake; however, a parallel analysis using baseline patterns was also evaluated. The correlation between high school and adult dietary pattern scores was evaluated by Spearman correlation coefficients.

To assess the joint effect of high school and adult Western dietary pattern on T2DM risk, dietary pattern scores were categorized into tertiles of low, medium, and high and cross-classified into a single variable, which was evaluated categorically using "low-low" as the reference in the multivariate model. All statistical tests were two-sided and performed using

Table 1—Age-adjusted high school and baseline characteristics by quintile of energy-adjusted dietary patterns among NHS II participants during high school

	Quintile of prudent pattern			Quintile of Western pattern		
	1 (lowest)	3	5	1 (lowest)	3	5
High school factors						
Total physical activity (Mets/week)	50.3	50.9	59.3	55.3	51.0	51.2
Television viewing (h/week)	3.9	3.8	3.5	3.6	3.8	4
BMI at age 18 years (kg/m^2)	21.1	21.0	21.3	21.1	21.1	21.4
Smoking (%)	25	23	21	20	22	29
Smoking (cigarettes/day)	3.0	2.8	2.9	3.0	2.8	3.0
Oral contraceptive use (%)	24	21	20	20	21	26
Multivitamin use (%)	8.0	8.0	9.0	9.0	8.0	8.0
Dietary intake						
Total energy (kcal/day)	2,858	2,612	2,878	2,807	2,595	2,913
Vitamin D intake (IU/day), including supplement	324	351	377	390	349	271
Alcohol (g/day)	1.3	0.96	0.84	0.91	0.91	1.4
Glycemic load	170	170	177	175	170	169
Cereal fiber (g/day)	5.4	5.7	6.6	6.3	5.6	5.3
Total carbohydrate (g/day)	306	308	327	322	308	298
Total protein (g/day)	104	107	111	110	106	102
Trans fat (g/day)	7.6	7.3	6.3	6.7	7.3	7.9
Total fat (g/day)	127	125	116	118	125	132
Polyunsaturated fat:saturated fat ratio	0.40	0.42	0.49	0.44	0.41	0.44
Processed meat (servings/day)	0.67	0.55	0.47	0.46	0.54	0.83
Red meat (servings/day)	1.02	0.90	0.85	0.88	0.91	1.07
Vegetable intake (servings/day)	2.3	2.8	5.5	4.0	2.8	2.4
Fruit intake (servings/day)	1.2	1.4	2.5	2.1	1.4	1.1
Better-quality grains (servings/day)	0.26	0.32	0.79	0.66	0.28	0.18
Refined grains (servings/day)	2.6	2.4	2.3	2.2	2.3	3.0
Coffee (servings/day)	0.19	0.17	0.25	0.21	0.17	0.19
Sugar-sweetened beverages (servings/day)	0.75	0.37	0.25	0.37	0.40	0.77
Low-fat dairy (servings/day)	0.57	0.77	1.17	1.24	0.60	0.32
High-fat dairy (servings/day)	2.19	1.86	1.69	1.85	2.0	1.96
Adult baseline characteristics						
Age (years)	42.1	42.3	42.1	41.9	42.3	42.4
Weight change since age 18 years (kg)	12.6	12.0	11.4	11.1	12.1	13.8
BMI (kg/m ²)	25.7	25.4	25.4	25.2	25.5	26.4
Height (inches)	64.9	65	65	65.0	65.0	64.8
Physical activity (Mets/week)	17.3	18.8	25.0	22.0	18.8	16.5
Postmenopausal hormone use (%)	17.0	16.0	15.0	15.0	16.0	19.0
Current oral contraceptive use (%)	8.0	9.0	9.0	8.0	9.0	8.0
Current smoker (%)	10.0	9.0	7.0	7.0	9.0	13.0
Family history of diabetes (%)	16.0	16.0	15.0	14.0	16.0	17.0
Hypertension (%)	7.0	6.0	6.0	6.0	6.0	8.0
Dietary factors						
Calories per day (kcal)	1,795	1,763	1,875	1,844	1,746	1,804
Alcohol intake (g/day)	3.3	3.5	3.7	3.5	3.6	3.5
Coffee (servings/day)	1.6	1.6	1.7	1.6	1.6	1.6
Carbonated soft drinks (servings/day)	0.53	0.42	0.34	0.38	0.43	0.56
Trans fat (g/day)	1.6	1.5	1.3	1.4	1.5	1.7
Polyunsaturated fat:saturated fat ratio	0.50	0.51	0.57	0.53	0.51	0.51
Glycemic load	123	123	126	125	123	120
Cereal fiber (g/day)	5.9	6.1	6.5	6.4	6.1	5.6
Processed meat (servings/dav)	0.23	0.21	0.16	0.17	0.20	0.26
Red meat (servings/day)	0.57	0.53	0.46	0.49	0.53	0.61
Vegetables (servings/day)	3.0	3.2	4.7	4.0	3.2	3.0
Fruit (servings/day)	1.1	1.2	1.7	1.5	1.2	0.99

Means are shown for continuous variables, and row percentage for dichotomous variables.

SAS version 9 for UNIX (SAS Institute, Cary, NC).

RESULTS—Two major dietary patterns were identified from the HS-FFQ (Supplementary Table 1). The pattern labeled prudent" was characterized by a high consumption of vegetables, fruit, legumes, fish, and better-quality grains and low consumption of snacks and soda. In contrast, the pattern labeled "Western" was characterized by a high consumption of desserts, snacks, processed meat, red meat, French fries, and refined grains and low consumption of vegetables, fruit, and fish. Similar factor loadings were observed for the two adult dietary patterns, also labeled "prudent" and "Western," with some variation in the 2003 patterns (Supplementary Table 2). High school and current baseline characteristics of the study population according to quintile of high school dietary patterns are shown in Table 1. Women with higher prudent pattern scores during high school were more physically active, less likely to smoke, had healthier dietary habits during high school and in adulthood, and gained less weight since age 18 years than women with a lower score for this pattern. Women

with higher Western pattern scores during high school were less physically active, more likely to smoke, and tended to have less favorable dietary habits during high school and in adulthood, gained more weight since age 18, and were more likely to have hypertension or a family history of diabetes. The correlation between high school and adult dietary pattern scores was 0.49 for the prudent pattern and 0.40 for the Western pattern.

During 290,703 person-years of follow-up, we confirmed 550 cases of T2DM. As shown in Table 2, the prudent pattern during high school was not associated with risk of T2DM. However, higher Western pattern scores during high school were associated with increased risk of T2DM. After adjusting for age, BMI at age 18 years, total energy intake during high school, and physical activity and smoking during high school, participants in the highest quintile of Western pattern score had a 62% greater risk of T2DM than participants in the lowest quintile (relative risk [RR] 1.62; 95% CI 1.27–2.07; P trend <0.0001). This association persisted after adjustment for adult risk factors (physical activity, family history of diabetes, smoking,

postmenopausal hormone use, oral contraceptive use, total energy intake, and alcohol use) (1.39; 1.08–1.78; *P* trend: 0.006). Additional adjustment for adult Western pattern score weakened the association (1.29; 1.00–1.66; *P* trend: 0.04), and the association became nonsignificant after adjustment for weight change since age 18 years.

The association between high school Western dietary pattern and T2DM was modified by BMI at age 18 years (*P* for interaction: 0.04), with the association being stronger among individuals with BMI at age 18 years of $<25 \text{ kg/m}^2$ than individuals who were overweight at age 18 years (Supplementary Table 3). There were no other interactions observed.

In our analysis of adult dietary patterns, a higher score for the prudent pattern was associated with reduced risk of T2DM after adjusting for age (RR for highest vs. lowest quintile: 0.52; 95% CI 0.40–0.67; *P* trend: <0.0001) (Supplementary Table 4). This association dissipated after adjustment for potential confounders. In contrast, a higher adult Western pattern score was associated with a substantially higher risk for T2DM. The association persisted after adjustment

	Quintile of dietary pattern							
	1	2	3	4	5	P trend		
Prudent pattern								
Number of cases	163	111	87	96	93			
Person-years	87,127	51,607	51,222	50,983	49,764			
Model 1	1.00	1.14 (0.89–1.45)	0.89 (0.68–1.15)	1.01 (0.78–1.30)	1.03 (0.80–1.33)	0.95		
Model 2	1.00	1.23 (0.96-1.57)	0.96 (0.74-1.25)	1.05 (0.81-1.36)	1.03 (0.80-1.34)	0.96		
Model 3	1.00	1.23 (0.96-1.58)	0.95 (0.73-1.24)	1.03 (0.79–1.32)	0.98 (0.76-1.27)	0.64		
Adjustment for adult risk factors								
Model 4	1.00	1.26 (0.99–1.62)	1.00 (0.76-1.31)	1.14 (0.88–1.47)	1.15 (0.89–1.50)	0.39		
Model 5	1.00	1.26 (0.99-1.62)	1.03 (0.78-1.34)	1.20 (0.92-1.55)	1.25 (0.95-1.64)	0.14		
Model 6	1.00	1.29 (1.00-1.66)	1.00 (0.76-1.31)	1.19 (0.91–1.55)	1.27 (0.96-1.67)	0.14		
Western pattern								
Number of cases	132	82	90	111	135			
Person-years	86,983	51,267	51,449	51,293	49,711			
Model 1	1.00	1.06 (0.80-1.40)	1.10 (0.84–1.45)	1.36 (1.06–1.76)	1.68 (1.32-2.14)	< 0.0001		
Model 2	1.00	1.12 (0.84-1.47)	1.15 (0.88-1.51)	1.39 (1.08-1.80)	1.58 (1.24-2.01)	< 0.0001		
Model 3	1.00	1.13 (0.85–1.49)	1.17 (0.89–1.54)	1.42 (1.10-1.83)	1.62 (1.27-2.07)	< 0.0001		
Adjustment for adult risk factors								
Model 4	1.00	1.13 (0.85–1.49)	1.12 (0.85–1.47)	1.30 (1.00-1.68)	1.39 (1.08–1.78)	0.006		
Model 5	1.00	1.12 (0.84–1.48)	1.08 (0.82-1.42)	1.23 (0.95-1.60)	1.29 (1.00-1.66)	0.04		
Model 6	1.00	1.13 (0.85–1.50)	1.10 (0.83–1.46)	1.21 (0.93–1.57)	1.19 (0.92–1.54)	0.14		

Table 2—RR of T2DM among NHS II participants (1997–2005) according to quintile of energy-adjusted high school dietary pattern

Data are RR (95% CI) unless otherwise indicated. Model 1: adjusted for age. Model 2: adjusted for age, BMI at age 18 years, and high school total calories. Model 3: additional adjustment for high school smoking and physical activity. Model 4: additional adjustment for adult risk factors: physical activity, family history of diabetes, smoking status, postmenopausal hormone use, oral contraceptive use, total energy intake, and alcohol. Model 5: additional adjustment for baseline adult prudent or Western dietary pattern. Model 6: additional adjustment for weight change since age 18 years to baseline.

Adolescent diet patterns and risk of diabetes

for potential confounders (RR 2.14; 95% CI 1.58–2.88; *P* trend: <0.0001), but disappeared after adjustment for BMI (1.24; 0.91–1.69; *P* trend: 0.23). Additional adjustment for hypertension did not change the associations (0.85, 0.64– 1.12, and 1.20, 0.88–1.62, for prudent and Western patterns, respectively). Similarly, results were obtained from the analysis using baseline dietary patterns (data not shown).

As shown in Fig. 1, individuals who had high Western dietary pattern scores in adulthood and during high school had the greatest risk of T2DM compared with individuals with low scores (RR 1.82; 95% CI 1.35–2.45). The strength of the association for the Western pattern during high school appeared to decrease with lower levels of adult Western pattern. Adjustment for current BMI attenuated this association (1.15; 0.85–1.56) (not shown).

CONCLUSIONS—In this large prospective cohort study of U.S. women, we identified two major dietary patterns during high school. Greater adherence to the prudent pattern, characterized by a high intake of healthy foods, was not associated with risk of T2DM, which is consistent with previous studies in our cohorts (3,5). However, greater adherence to the Western pattern, characterized by a high intake of desserts, processed meat, and refined grains, was associated with a higher risk of T2DM independent of other high school and adult risk factors. To our knowledge, this is the first study that has examined adolescent dietary patterns in relation to risk of T2DM decades later.

The association between Western patterns and T2DM appeared to be partly mediated through weight gain. In this cohort, greater adherence to a Western dietary pattern was previously associated with weight gain, whereas a prudent pattern was shown to facilitate weight maintenance (4). Our findings—that adjustment for weight change since age 18 years and current BMI attenuated the associations between high school and current Western dietary patterns with T2DM—support a role for adiposity as a mediator. This result is supported by our finding that both high school dietary



Figure 1—Joint analysis between adult and high school Western dietary pattern score in relation to T2DM risk. Tertiles (low, medium, high) of high school and adult Western dietary pattern score were cross-classified into a single categorical variable and evaluated for risk of T2DM using the low-low category as the reference. High levels of high school Western dietary pattern are depicted by black bars, medium levels by gray bars, and low levels by white bars. Data are adjusted for age, total calories, family history of diabetes, smoking status, physical activity, oral contraceptive use, hormone replacement therapy, and alcohol. The current pattern—high school patterns [RR (95% CI)] were as follows: low-low (ref), 1.00; medium-low, 0.95 (0.66–1.37); high-low, 1.55 (1.10–2.19); low-medium, 0.94 (0.61–1.44); medium-medium, 1.12 (0.78–1.62); high-medium, 1.45 (1.02–2.05); low-high, 0.94 (0.58–1.53); medium-high, 1.28 (0.88–1.85); and high-high, 1.82 (1.35–2.45). Categories were defined as tertiles. Current median Western pattern scores were as follows: low, -0.53; medium, -0.05; and high, 0.44. High school median Western pattern scores were as follows: low, -0.43; medium, 0.02; and high, 0.42.

patterns predicted weight change since age 18 years (P < 0.0001) (not shown). The interaction between high school Western dietary pattern and BMI at age 18 years suggests that this pattern may also affect future risk of T2DM in previously normalweight (BMI $< 25 \text{ kg/m}^2$) girls, although the mechanism is not clear and should be interpreted cautiously. Dietary components in the pattern that may affect T2DM risk include higher saturated fat (16), glycemic load (1), heme iron (17), and advanced glycation end products (18) from a higher consumption of processed meat, refined grains, and desserts. It is most likely a combination of dietary factors that was responsible for the robust associations. Higher scores for the Western pattern have been correlated with higher fasting insulin, and C-peptide levels (19) and inflammatory markers (20).

In our study, women who had high Western pattern scores both in adolescence and in adulthood had the greatest risk of T2DM, suggesting that adhering to a Western dietary pattern during adolescence may affect risk of T2DM beyond that imparted by high Western pattern scores in adulthood, possibly by persistence of an unhealthy dietary pattern through the life course. Little is known about how diet during early life may affect risk of T2DM years later. Greater adult height, which may represent adequate childhood nutritional status, has been associated with decreased risk of T2DM. possibly by increasing childhood IGF-1 levels (21,22), although this relation has not been fully established. It is possible that greater adherence to a Western pattern during adolescence increases risk of T2DM in adulthood via a cumulative effect on physiological processes underlying disease development. Findings from the Bogalusa Heart Study indicate that high blood pressure, hyperinsulinemia, and dyslipidemia begin to cluster during childhood and can predict adult cardiovascular risk factors (23). This clustering of risk factors has been linked to unhealthy dietary habits during childhood (24). It may also be possible that greater adherence to the Western dietary pattern is associated with an unhealthy lifestyle, which may track through life, affecting risk of T2DM. Despite adjusting for potential confounders, residual confounding cannot be ruled out. The validity of adult recall of adolescent diet 15-35 years earlier has not been established, since it is difficult to disentangle the effects of waning memory with age, influence of current diet on recall of past diet, and temporal changes in diet (8,9). However, lack of an interaction with age at return of the HS-FFQ suggests that fading memory of high school diet with age may not be a significant factor in these data, although this effect is difficult to isolate (P for interaction: 0.89 for prudent pattern and 0.43 for Western pattern; Supplementary Table 5). The validity of the HS-FFQ was assessed by administering it to 80 young adults who had completed three 24-h diet recalls and two similar FFQs 10 years earlier while in high school (10). The average nutrient correlation between the HS-FFQ and 24-h recalls was 0.45 (range 0.16-0.68) and between the HS-FFQ and FFQs was 0.58 (0.40-0.88) (10). Validity of the HS-FFQ was also assessed by comparing the nurses' self-reports with those of their mothers (9). The mean nutrient correlation between the mothers' and nurses' self-reports was 0.40 (0.13-0.59) and for foods 0.30 (0.10–0.61) (9). Other studies that have examined the validity of diet recalled 11-24 years have reported moderate correlations for food (range 0.29–0.4) and nutrients (0.23–0.59) (8), which are similar to those reported for the HS-FFO. Survey methodology shows that provision of a clear definition of the reference period (i.e., high school) enhances recall (25). These findings suggest that the HS-FFQ provides a reasonable record of adolescent diet, although some attenuation of results can be expected because of nondifferential measurement error. There are also limitations in dietary pattern analysis such as subjectivity in grouping foods and determining the number of factors to retain. However, dietary patterns have generally been consistent in our cohorts. The reason why current patterns from the 2003 FFQ were slightly different from previous years may be due to temporal changes in the diet. Exclusion of 2003 FFQ data from the cumulative average did not change our results.

The prospective design of our study, in which cases were ascertained after return of the HS-FFQ, limits the likelihood of recall bias caused by knowledge of disease status. An important strength of dietary pattern analysis is the ability to detect the combined effect of foods, since the physical properties of foods and interactions between nutrients may affect glucose homeostasis.

Our findings confirmed those from previous studies that have found positive associations between the Western dietary pattern and risk of T2DM. In addition, we showed that greater adherence to this pattern during adolescence was associated with increased risk of T2DM independently and jointly with adult Western patterns. Women who had high Western pattern scores during adolescence and in adulthood had the greatest risk of T2DM compared with individuals with consistent low scores, suggesting that persistence of a Westerntype dietary pattern into adulthood may have particularly adverse effects on T2DM risk, possibly through weight gain. For optimal prevention of T2DM, a healthy diet should be adopted in early life and maintained throughout the life course.

Acknowledgments—This work was supported by National Institutes of Health grants CA50385 and DK58845.

No potential conflicts of interest relevant to this article were reported.

V.S.M. designed and conducted the analysis, wrote the manuscript, had primary responsibility for the final content, and is the guarantor of this study. T.T.F. reviewed the analysis and edited the manuscript. R.M.v.D. and E.B.R. helped with project design and edited the manuscript. B.R. provided statistical support. F.B.H. helped with project design and interpretation of data and edited the manuscript.

Parts of this study were presented at the Scientific Sessions of the American Heart Association, Atlanta, Georgia, 22–25 March 2011.

References

- Salmerón J, Manson JE, Stampfer MJ, Colditz GA, Wing AL, Willett WC. Dietary fiber, glycemic load, and risk of noninsulin-dependent diabetes mellitus in women. JAMA 1997;277:472–477
- 2. van Dam RM, Willett WC, Rimm EB, Stampfer MJ, Hu FB. Dietary fat and meat intake in relation to risk of type 2 diabetes in men. Diabetes Care 2002;25: 417–424
- 3. van Dam RM, Rimm EB, Willett WC, Stampfer MJ, Hu FB. Dietary patterns and risk for type 2 diabetes mellitus in U.S. men. Ann Intern Med 2002;136: 201–209
- 4. Schulze MB, Fung TT, Manson JE, Willett WC, Hu FB. Dietary patterns and changes in body weight in women. Obesity (Silver Spring) 2006;14:1444–1453
- 5. Fung TT, Schulze M, Manson JE, Willett WC, Hu FB. Dietary patterns, meat intake, and the risk of type 2 diabetes in women. Arch Intern Med 2004;164:2235–2240
- Michels KB. Early life predictors of chronic disease. J Womens Health (Larchmt) 2003; 12:157–161

- 7. Colditz GA, Manson JE, Hankinson SE. The Nurses' Health Study: 20-year contribution to the understanding of health among women. J Womens Health 1997;6:49–62
- Frazier AL, Willett WC, Colditz GA. Reproducibility of recall of adolescent diet: Nurses' Health Study (United States). Cancer Causes Control 1995;6: 499–506
- 9. Maruti SS, Feskanich D, Colditz GA, et al. Adult recall of adolescent diet: reproducibility and comparison with maternal reporting. Am J Epidemiol 2005; 161:89–97
- Maruti SS, Feskanich D, Rockett HR, Colditz GA, Sampson LA, Willett WC. Validation of adolescent diet recalled by adults. Epidemiology 2006;17:226– 229
- 11. Salvini S, Hunter DJ, Sampson L, et al. Food-based validation of a dietary questionnaire: the effects of week-to-week variation in food consumption. Int J Epidemiol 1989;18:858–867
- 12. Hatcher LA. Step-By-Step Approach to Using SAS for Factor Analysis and Structural Equation Modeling. Cary, NC, SAS Institute, 1994
- 13. Hu FB, Rimm E, Smith-Warner SA, et al. Reproducibility and validity of dietary patterns assessed with a food-frequency questionnaire. Am J Clin Nutr 1999;69: 243–249
- Willett WC, Stampfer MJ. Implications of total energy for epidemiologic analysis. In Nutritional Epidemiology. New York, NY, Oxford University Press, 1998, p. 273–301
- Manson JE, Rimm EB, Stampfer MJ, et al. Physical activity and incidence of non-insulin-dependent diabetes mellitus in women. Lancet 1991;338:774– 778
- Hu FB, van Dam RM, Liu S. Diet and risk of type II diabetes: the role of types of fat and carbohydrate. Diabetologia 2001;44: 805–817
- 17. Rajpathak S, Ma J, Manson J, Willett WC, Hu FB. Iron intake and the risk of type 2 diabetes in women: a prospective cohort study. Diabetes Care 2006;29: 1370–1376
- Cai W, Gao QD, Zhu L, Peppa M, He C, Vlassara H. Oxidative stress-inducing carbonyl compounds from common foods: novel mediators of cellular dysfunction. Mol Med 2002;8:337–346
- Fung TT, Rimm EB, Spiegelman D, et al. Association between dietary patterns and plasma biomarkers of obesity and cardiovascular disease risk. Am J Clin Nutr 2001;73:61–67
- 20. Lopez-Garcia E, Schulze MB, Fung TT, et al. Major dietary patterns are related to plasma concentrations of markers of inflammation and endothelial dysfunction. Am J Clin Nutr 2004;80:1029– 1035

Adolescent diet patterns and risk of diabetes

- 21. Asao K, Kao WH, Baptiste-Roberts K, Bandeen-Roche K, Erlinger TP, Brancati FL. Short stature and the risk of adiposity, insulin resistance, and type 2 diabetes in middle age: the Third National Health and Nutrition Examination Survey (NHANES III), 1988– 1994. Diabetes Care 2006;29:1632–1637
- 22. Bray I, Gunnell D, Holly JM, Middleton N, Davey Smith G, Martin RM. Associations of childhood and adulthood height and the

components of height with insulin-like growth factor levels in adulthood: a 65-year follow-up of the Boyd Orr cohort. J Clin Endocrinol Metab 2006;91:1382–1389

- 23. Bao W, Srinivasan SR, Wattigney WA, Berenson GS. Persistence of multiple cardiovascular risk clustering related to syndrome X from childhood to young adulthood: the Bogalusa Heart Study. Arch Intern Med 1994;154:1842–1847
- 24. Berenson GS, Srinivasan SR, Nicklas TA. Atherosclerosis: a nutritional disease of childhood. Am J Cardiol 1998;82:22T–29T
- 25. Tourangeau R. Remembering what happened: memory errors and survey reports. In *The Science of Self-Report: Implications for Research and Practice.* Stone AA, Turkkan JS, Bachrach CA, Jobe JB, Kurtzman HS, Cain VS, Eds. Mahwah, NJ, Lawrence Erblaum Associates, 2000, p. 29–48