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Healthful dietary patterns and type 2 diabetes risk among women with a history of gestational diabetes

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Abstract

Background—Type 2 diabetes (T2DM) has reached epidemic proportions. Women with gestational diabetes mellitus (GDM) are at high risk for T2DM after pregnancy. Adherence to healthful dietary patterns has been inversely associated with T2DM in the general population; however whether these dietary patterns are associated with progression to T2DM among a susceptible population is unknown.

Methods—4,413 participants from the Nurses' Health Study II cohort with prior GDM were followed from 1991–2005. The alternate Mediterranean Diet (aMED), Dietary Approaches to Stop Hypertension (DASH), and alternate Healthy Eating Index (aHEI) dietary pattern adherence scores were derived from post-GDM validated food-frequency questionnaire, with cumulative average updating every 4 years. Multivariable Cox proportional hazards models estimated the relative risk (HR) and 95% confidence intervals [95% CI].

Results—491 cases of incident T2DM were observed over 52,743 person-years. All three patterns were inversely associated with T2DM risk adjusting for age, total calories, age at first birth, parity, ethnicity, parental diabetes, oral contraceptive use, menopause, and smoking. Comparing participants with highest adherence (quartile 4) versus lowest (quartile 1), the aMED pattern was associated with 40% lower risk of T2DM (HR=0.60 [95% CI: 0.44, 0.82] p-trend=0.0019), DASH with 46% lower risk (HR=0.54 [95% CI: 0.39, 0.73] p-trend=0.0002), and

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- Patent: Harvard University has filed a provisional patent application, that has been assigned to Harvard University, listing Dr. Mozaffarian as a co-inventor to the US Patent and Trademark Office for use of trans-palmitoleic acid to prevent and treat insulin resistance, type 2 diabetes, and related conditions.

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aHEI with 57% lower risk (HR=0.43 [95% CI: 0.31, 0.59] p-trend<0.0001). Adjustment for body mass index (BMI) moderately attenuated these findings.

Conclusions—Adherence to healthful dietary patterns is associated with lower T2DM risk among women with a history of GDM. The inverse associations were partly mediated by BMI.

INTRODUCTION

Type 2 diabetes (T2DM) has become an epidemic in the United States and globally. More alarmingly, many individuals have developed complications by the time they receive the diagnosis, including cardiovascular disease, renal dysfunction, and retinopathy, underscoring the importance of identifying high risk populations in need of targeted prevention. One such high risk group is women who developed glucose intolerance during pregnancy; it is estimated that up to one-third of parous women with diabetes have a history of gestational diabetes mellitus (GDM).[1] Compared to women with a history of normoglycemic pregnancies, those with prior GDM have more than a 7-fold increased risk of developing T2DM.[2] There is limited longitudinal research following women from the time of their GDM pregnancy to the development of T2DM many years later. Further, studies of major risk factors, particularly modifiable risk factors, for the progression to T2DM among this high risk population are sparse.

Several healthful dietary patterns including the alternate Mediterranean Diet (aMED), Dietary Approaches to Stop Hypertension (DASH), and alternate Healthy Eating Index (aHEI), have been inversely associated with T2DM risk and other cardiovascular disease endpoints in the general population,[3–5] but rarely investigated among women a history of GDM. In the present study, we aim to quantify the association of adherence to these healthful dietary patterns with T2DM risk among women with a history of GDM with prospective follow-up of 16 years. Findings from the present study may help identify dietary patterns that would be crucial for post-partum and life-long dietary modifications to prevent T2DM.

SUBJECTS AND METHODS

Study Population

The study population is composed of women with a history of GDM in the Nurses' Health Study II (NHS II), an ongoing prospective cohort established in 1989 with the enrollment of 166,671 female nurses, ages 24–44 at baseline. Questionnaires are distributed every two years to update lifestyle and medical characteristics and to capture incident health outcomes. Follow-up for each questionnaire cycle is greater than 90%. This study has been approved by the institutional review board of the Partners Health Care System (Boston, MA, USA), with participants' consent implied by the return of the questionnaires.

Participants were eligible if they reported a history of GDM at baseline (1991). Women also became eligible during follow-up if they reported incident GDM through the 2001 questionnaire, as the update of GDM occurrence ceased after 2001. GDM was assessed via self-report of a physician's diagnosis, which has been previously validated against medical records (94% confirmed) in a subgroup of this population.[6] Participants were restricted from analysis if they reported chronic disease (T2DM, cardiovascular disease event, cancer) at baseline, prior to their GDM pregnancy, or before the return of their first post-GDM FFQ, a multiple birth pregnancy, missing dietary exposure information, more than 70 FFQ items left blank, or unrealistic total energy intake (<500, >3,500 kilocalories/day).

Exposure Assessment

In 1991 and every 4 years thereafter, participants complete a semi-quantitative food frequency questionnaire (FFQ).[7] The FFQ captures usual intake of several common food items over the past year and has been extensively validated.[8–10] Three dietary pattern adherence scores (aMED, DASH, and aHEI) were computed for each FFQ cycle after the first reported GDM pregnancy. Scoring methods and justification for inclusion of components have been described in detail elsewhere.[11–13] Total scores are the sum of points earned across all dietary components, with a higher score indicating greater adherence, ranging from 0–8 for aMED, 8–39 for DASH, and 2.5–87.5 for aHEI.

Briefly, to derive the aMED score, participants were assigned 1 point for being above the median of servings/day for the following components: fruit, vegetables, legumes and soy, nuts, fish and seafood, whole grains, monounsaturated-to-saturated fatty acid ratio (MUFA:SFA). Red and processed meat was scored 1 point for being below the median intake and one point for for moderate alcohol (5–15 grams/day).[14] We conducted a sensitivity analysis removing MUFA:SFA, since the primary source of MUFA in Western diets (beef and dairy) differs from the traditional Mediterranean diets (plant-based oils).[15]

To derive DASH scores women were assigned 1–5 points based on their quintile of intake (servings/day) of: fruit, vegetables, nuts, legumes and soy, red and processed meats, whole grains, low-fat dairy, sodium (milligrams).[16] Sweetened beverages were derived from quartiles of usual intake, as there was less variability. Scoring was reversed for red and processed meats, sugar-sweetened beverages, and sodium, receiving more points for less consumption.

For the aHEI pattern, points were allotted for intake of each component on a scale from 0–10, with 10 indicating adherence to the recommended levels of servings/day, 0 for the worst intake, and intermediate scores categorized proportionately for: fruit, vegetables, nuts and soy, white-to-dark meat ratio, cereal fiber (grams), alcohol, polyunsaturated-to-saturated fatty acid ratio (grams), *trans* fat (% total energy). Multivitamin use was scored giving 2.5 points for 0–4 years of use and 7.5 points for ≥5 years of use.

Outcome Assessment

Participants reporting a physician's diagnosis of T2DM are mailed a supplemental questionnaire. Confirmed cases are defined from this additional information according to the National Diabetes Data Group classification [17] as those reporting at least one of the following: ≥1 classic symptoms (excessive thirst, polyuria, unintentional weight loss, hunger) and fasting plasma glucose concentration ≥140 mg/dL (7.8 mmol/L) or random plasma glucose ≥200 mg/dL (11.1 mmol/L); no symptoms but ≥2 elevated plasma glucose concentrations on more than one occasion (fasting ≥140 mg/dL, random ≥200, 2-hour oral glucose tolerance test ≥200 mg/dL); or hypoglycemic medication use (insulin or oral hypoglycemic agent). Diagnostic criteria was changed in June 1998 to adopt a new diagnostic threshold for fasting plasma >126 mg/dL (7.0 mmol/L).[18] A subgroup validation study was conducted in a similar cohort of US female nurses, comparing our classification against medical records with high accuracy (98%).[19]

Covariate Assessment

Age was computed from date of birth to date of questionnaire return for each risk set. Body mass index (BMI; kilograms/meters^2), from self-reported height and weight, was highly correlated with measured weight among a random subset of Boston-area cohort participants (r=0.97).[20] Total physical activity was ascertained by frequency of engaging in common recreational activities, from which MET-hours per week were derived. The questionnaire-

based estimates correlated well with detailed activity diaries in a prior validation study (r=0.56).[21] Other relevant covariables captured on the biennial questionnaires included age at first birth, oral contraceptive use, months of breastfeeding, menopausal status, smoking status, self-reported race and ethnicity, and parental history of hypertension and diabetes. Parity was defined as the number of pregnancies lasting greater than 6 months.

Statistical Analysis

Baseline characteristics were derived from the questionnaire period in which participants first reported a GDM pregnancy. Dietary pattern adherence scores were updated as the cumulative average of all scores since GDM to reduce random within-person error and to represent long-term usual intake.[22] Updating ceased if a participant reported incident chronic disease (cardiovascular disease, cancer) to reduce reverse causation. Missing exposure data was carried forward from the most recent post-GDM FFQ for which data were captured. Follow-up time was computed from the date of GDM diagnosis to the date of T2DM diagnosis, death, or return of the 2005 questionnaire, whichever came first. We computed pair-wise Pearson correlations between scores to assess overlap of the exposures.

Cox proportional hazards models stratified by time since GDM diagnosis were used to estimate the relative risks (HR) and 95% confidence intervals [95% CI] for associations between dietary pattern adherence and risk of incident T2DM. Scores were analyzed continuously for a 1 interquartile range (IQR) increase in adherence, and categorically in quartiles, with the lowest adherence (Q1) as the reference group. Chi^2 tests for trend across quartiles were computed by modeling the median scores of each quartile as a continuous variable. Our first multivariable model adjusted for age and total energy intake. Additional multivariable models further adjusted for possible confounders including parity, age at first birth, race and ethnicity, parental history of diabetes, oral contraceptive use, menopausal status, smoking status, physical activity, and subsequently, BMI. Alcohol intake was also included in DASH models, as this was not a component of the score and is a potential lifestyle confounder. BMI was included in the model separately as it is also a plausible intermediate between exposure and outcome. The proportion of the associations mediated by BMI was estimated with a SAS macro developed by Spiegelman and colleagues (Harvard School of Public Health, Mediate SAS: www.hsph.harvard.edu/faculty/donna-spiegelman/software/mediate/). This computes the magnitude (%) of mediation, as well as the 95% CI and p-value for significance.[23] Time-varying lifestyle covariables were updated biennially. Categorical covariables included an indicator variable for missing data, if necessary.

Secondary analyses assessed effect modification by overweight status (BMI: <25 vs. ≥25), age (<35 vs. ≥35) parental history of diabetes (none vs. either parent), and physical activity (MET-hours per week: 0–15.4 vs. ≥15.5). P-values for heterogeneity were derived from 1 degree of freedom −2 log likelihood ratio test chi^2 statistics, comparing the main effects model with and without the addition of the multiplicative interaction terms.

An analysis of each patterns' components was also conducted, modeling all components simultaneously to assess a 1 point increase in total score by a given dietary factor. This was performed to assess whether the contribution of any one individual component or components explained the association observed between total scores and diabetes risk. In an additional sensitivity analysis to address the potential of screening bias, we restricted cases to those reporting at least 1 classic diabetic symptom at the time of diagnosis. SAS version 9.1 was used for all analyses (SAS Institute Inc., Cary, NC, USA).

RESULTS

Overall 4,413 participants with a history of GDM met our inclusion criteria, contributing 52,743 person-years of observation. On average, participants in the fourth quartile (highest adherence) of each pattern score had a lower BMI, consumed more alcohol, had a higher percent of total calories from carbohydrates and a lower percent from animal fat, were less likely to be current smokers, and were less likely to have a parental history of diabetes (Table 1). The dietary pattern adherence scores were significantly correlated with one another ($p<0.0001$). During 16 years of observation, 491 (11.1%) participants developed T2DM. Mean time from GDM to development of T2DM was 13.8 years (median=13.5, range=2.0–27.6). Mean age at diagnosis of T2DM was 46.5 years (median=46.7; range=32.4–59.8).

All three dietary pattern adherence scores were strongly and inversely associated with T2DM risk after adjusting for age and total energy intake (Model 1) (Table 2). Adjustment for other confounders (Model 2) did not substantially change the findings; however, adjustment for BMI (Model 3) partially attenuated the effect estimates for all three dietary patterns. Breastfeeding did not impact results. Independent of BMI, a 1 IQR increase in score adherence to the aMED dietary pattern was associated with a 15% lower diabetes risk (IQR=2.0; HR=0.84 [95%CI: 0.73, 0.96] p=0.014), a 10% lower risk for DASH (IQR=7.0; HR=0.86 [95% CI: 0.73, 1.03] p=0.097), and aHEI with a 17% lower risk (IQR=15.0; HR=0.77 [95% CI: 0.64, 0.93] p=0.0073). BMI was estimated to mediate 41% (19–63%, p=0.0003) of the association between aMED pattern adherence and diabetes risk, 39% (15–64%, p=0.0018) of the DASH pattern, and 50% (28–72%, $p<0.0001$) of the aHEI pattern.

Tests for heterogeneity did not suggest effect modification by parental history of diabetes, age, BMI, or physical activity level, for all three dietary patterns. When we assessed the association between a 1-point increase by an individual pattern component in the multivariable model, including all other components simultaneously, several factors within the dietary patterns trended towards an inverse association with incident T2DM. For the aMED pattern this included vegetable intake, fish and seafood, and moderate alcohol consumption. For DASH, vegetables, lower red and processed meat, and decreased sugar-sweetened beverages were inverse. For the aHEI pattern this included vegetables, an increased white-to-dark meat ratio, cereal fiber, moderate alcohol consumption, and long-term multivitamin use. In addition, removing the MUFA:SFA ratio from the aMED score produced similar results, and inclusion of only symptomatic T2DM cases gave similar effect estimates although statistical power was reduced.

DISCUSSION

In this large prospective cohort of 4,413 women with a history of GDM, we found that adherence to healthful dietary patterns, in particular the alternate Healthy Eating Index, is inversely associated with progression to T2DM. The significant association persisted even after the adjustment of other risk factors of T2DM.

Direct comparisons of dietary patterns were not performed for several reasons. First, we utilized previously published scoring methods to estimate adherence to each of the patterns, which produce substantially different scales; thus, differences in precision of exposure measurement may partially explain differences in observed effect estimates. Second, all scores were significantly and inversely associated with T2DM risk, with broadly overlapping 95% confidence intervals. Analyses to detect minor differences might therefore be statistically underpowered. Finally, there are several common components shared by the

dietary patterns, suggesting that in general, an overall healthful dietary pattern may be beneficial for the prevention of T2DM.

Our findings are consistent with previous findings between diet and T2DM in the general population;[24] however, we are unaware of previous studies investigating healthful dietary patterns and T2DM risk among the high risk population of women with a history of GDM. The recent Diabetes Prevention Program clinical trial enrolled individuals with impaired glucose tolerance at baseline, including 350 women with a history of GDM.[25] GDM participants in the lifestyle intervention (diet and physical activity advice) experienced a reduced risk of T2DM compared to placebo (53% vs. 49%). Inference from this intervention study was limited by the relatively small sample size (GDM N= 350, T2DM cases n=122). Moreover, the intervention effect was not specific to dietary modifications only.

Plausible biological mechanisms may explain the observed associations between healthful dietary patterns and the delay or prevention of progression of GDM to T2DM. Evidence suggests that women with prior GDM have diminished β-cell function. Thus, factors that increase insulin sensitivity may minimize β-cell compensation, preserving their capacity over time.[26,27] Carbohydrate quality, vegetables, fruit, low red and processed meat intake, and low saturated fat are common characteristics between the dietary patterns included in this analysis. Carbohydrate quality, reflected by intake of whole grains and cereal fiber, may mitigate β-cell demands by blunting intestinal glucose absorption and downstream insulin burden.[28] Glycemic index and glycemic load are measures of this insulin rise after glucose uptake and have been associated with chronic hyperglycemia and hyperinsulinemia.[29] Vegetables and fruit are high in micronutrients such as magnesium, antioxidants like vitamins C and E, phytochemicals, and fiber, leading to reductions in free radical-induced oxidative stress, a pathology correlated with pancreatic tissue damage.[30,31] Fish and seafood was inversely associated with diabetes risk as a component of the aMED dietary pattern in our study, and is a source of omega-3 polyunsaturated fatty acids, vitamin D, protein, and selenium. Current evidence between polyunsaturated fatty acids and diabetes risk is mixed. Inverse associations in the observational literature have largely not been supported by short-term trials of fatty acid supplementation and markers of insulin resistance;[32] thus, it is unclear which elements of seafood might benefit diabetes risk.

Increases in healthful dietary factors may also incur a benefit by replacing harmful food options. For example, substitution of red meat servings with several other foods (nuts, fish, whole grains, poultry) was associated with lower T2DM risk in a previous analysis conducted among initially healthy women.[33] Constituents of red and processed meat include heme iron, a pro-oxidant that may lead to increased oxidative stress and damage similar to that described above.[34] Nitrosamines in processed red meats are created during digestion and with certain cooking methods, and have been associated with insulin resistance, decreased insulin secretion, and diabetes in animal studies.[35] Finally, the mechanism by which moderate alcohol consumption may prevent diabetes is unclear, although consumption has been associated with improved insulin sensitivity and HDL concentration, and suppressed gluconeogenesis.[36]

Although several macronutrients, micronutrients, and individual foods have been associated with diabetes risk, assessment of dietary patterns offers a comprehensive and complimentary approach of the association between diet and disease. Additionally, dietary patterns may be more applicable to public health interventions, as people tend to consume complex and diverse meals rather than individual components in isolation. Analyzing food patterns also accounts for any interactions or synergistic effects between individual foods or nutrients. Other strengths of this analysis include our adjustment for several confounding lifestyle factors. With the exception of BMI, changes in the effect estimates after adjustment for

several well-known diabetes risk factors were minimal; thus, it seems unlikely that unmeasured or residual confounding would account for the observed associations. Exposure assessment by validated FFQ was an additional strength of this analysis and cumulative update of repeated exposure measures reduced misclassification from random within-person error and better represented long-term intake. Long-term prospective follow-up eliminates recall bias, and allowed us to observe participants from GDM exposure to incident T2DM. With almost 500 cases there was adequate statistical power.

There are some limitations to this analysis. First, since the majority of our study population is Caucasian Americans, we are unable to ascertain whether our findings are similar across other race and ethnic groups. However, the relative homogeneity of our population advantageously reduces potential sources of unmeasured confounding. Second, screening bias is a concern when there is a potential for more health-conscious women to regularly see a physician, thus increasing their chance of receiving a medical diagnosis. Similar results were seen when we restricted cases to symptomatic T2DM, minimizing concerns for this bias.

In summary, we observed significant inverse associations between increased adherence to the aMED, DASH, and aHEI dietary patterns and incident T2DM, in this large prospective study of women at high risk with a history of GDM and long-term follow-up. Identifying post-partum modifiable risk factors and increased education is crucial for the early prevention of T2DM among this high risk population. The novel study suggests that public health efforts targeting women with a history of GDM may consider encouraging diets rich in whole grains, fruit and vegetables, protein sources such as white meat, seafood, nuts and legumes, and a reduced intake of red and processed meats and sugar-sweetened beverages.

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Abbreviations

GDM	gestational diabetes mellitus
aMED	alternate Mediterranean Diet
DASH	Dietary Approaches to Stop Hypertension
aHEI	alternate Healthy Eating Index
NHS II	Nurses' Health Study II
FFQ	food frequency questionnaire
BMI	body mass index

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Table 1

Baseline[1] characteristics by dietary pattern adherence score quartiles among women with a history of GDM

	aMED		DASH		aHEI	
	Q1	**Q4**	**Q1**	**Q4**	**Q1**	**Q4**
n	**957**	**1,077**	**1,067**	**1,118**	**1,113**	**1,159**
	Mean (Standard Deviation)					
Diet Score	1.5 (0.6)	6.6 (0.7)	17.0 (2.1)	30.0 (2.2)	25.5 (4.0)	52.4 (6.7)
Age	37.3 (4.8)	38.1 (4.8)	37.9 (4.9)	38.1 (4.8)	37.3 (4.8)	38.3 (4.8)
BMI (kilograms/meters^2)	28.0 (7.1)	26.2 (6.0)	28.0 (7.0)	26.2 (5.9)	28.2 (7.1)	25.6 (5.6)
BMI at 18	21.8 (3.9)	21.1 (3.1)	21.6 (3.8)	21.2 (3.1)	21.8 (3.9)	21.0 (3.1)
Age at Index GDM	32.2 (4.9)	32.9 (4.9)	32.6 (4.9)	33.0 (4.9)	31.9 (4.8)	33.0 (4.9)
PA (MET-hours/week)	13.1 (18.4)	21.5 (24.3)	12.9 (18.1)	22.3 (24.4)	12.8 (18.2)	23.0 (26.4)
Alcohol (grams/day)	1.6 (4.6)	3.1 (4.5)	1.9 (4.4)	2.7 (4.9)	0.9 (3.6)	3.9 (5.0)
Total Energy (kcal/day)	1610 (510)	2220 (540)	1680 (540)	2170 (540)	1600 (490)	2230 (570)
Carbs (%kcal/day)	47 (8)	52 (7)	47 (8)	53 (7)	47 (7)	52 (7)
Protein (%kcal/day)	19 (4)	19 (3)	19 (4)	20 (3)	19 (4)	19 (3)
MUFA (%kcal/day)	13 (2)	12 (2)	14 (2)	11 (2)	14 (2)	11 (3)
SFA (%kcal/day)	13 (2)	10 (2)	13 (3)	10 (2)	13 (2)	10 (2)
Animal Fat (%kcal/day)	21 (5)	15 (4)	21 (5)	15 (4)	21 (5)	15 (4)
trans Fat (grams/day)	3.5 (1.8)	3.6 (1.7)	3.8 (1.8)	3.2 (1.4)	3.7 (1.8)	3.3 (1.5)
Glycemic Load	105 (45)	155 (45)	110 (45)	150 (45)	100 (40)	155 (45)
	n (Percent)					
Caucasian	872 (91.1)	970 (90.1)	963 (90.3)	1015 (90.8)	1014 (91.1)	1054 (90.9)
Parity						
1	199 (20.8)	200 (18.6)	218 (20.4)	230 (20.6)	234 (21.0)	239 (20.6)
2	421 (44.0)	492 (45.7)	487 (45.6)	492 (44.0)	491 (44.1)	514 (44.4)
3	213 (22.3)	258 (24.0)	218 (20.4)	264 (23.6)	241 (21.7)	270 (23.3)
4+	105 (11.0)	95 (8.8)	118 (11.1)	101 (9.0)	124 (11.1)	105 (9.1)
Smoking Status						
Never	608 (63.5)	726 (67.4)	667 (62.5)	745 (66.6)	741 (66.6)	744 (64.2)

	aMED		DASH		aHEI	
	Q1	Q4	Q1	Q4	Q1	Q4
n	**957**	**1,077**	**1,067**	**1,118**	**1,113**	**1,159**
	Mean (Standard Deviation)					
Past	199 (20.8)	274 (25.4)	209 (19.6)	304 (27.2)	206 (18.5)	324 (28.0)
Current	150 (15.7)	74 (6.9)	191 (17.9)	67 (6.0)	165 (14.8)	88 (7.6)
Oral Contraceptive Use						
Never	100 (10.5)	151 (14.0)	100 (9.4)	162 (14.5)	118 (10.6)	161 (13.9)
Ever	855 (89.3)	926 (85.9)	963 (90.3)	956 (85.6)	992 (89.2)	997 (86.0)
Parental History of Diabetes	254 (26.5)	271 (25.2)	274 (25.7)	219 (23.3)	315 (28.3)	276 (23.8)

aMED=alternate Mediterranean Diet; DASH=Dietary Approaches to Stop Hypertension; aHEI=alternate Healthy Eating Index; BMI=body mass index; PA=physical activity; MET-hours=metabolic equivalent hours; kcal=kilocalories; MUFA=monounsaturated fatty acids; SFA=saturated fatty acids; n=number of participants; Q=quartile

^Baseline = 1991 for prevalent GDM, and year of index pregnancy for incident GDM

Table 2

Quartiles of dietary pattern adherence scores and type 2 diabetes risk among women with a history of GDM

	Q1	Q2		Q3		Q4		
	[Reference]	**HR**	**[95% CI]**	**HR**	**[95% CI]**	**HR**	**[95% CI]**	**p-trend**
aMED								
cases/PY	137/12,198	142/16,161	(8.8)	106/10,961	(9.7)	106/13,423	(7.9)	
(rate per 1,000 PY)	(11.2)							
Model 1	1.0	0.74	[0.56, 0.96]	0.77	[0.57, 1.03]	0.60	[0.44, 0.80]	*0.0011*
Model 2	1.0	0.74	[0.57, 0.97]	0.77	[0.57, 1.04]	0.60	[0.44, 0.82]	*0.0019*
Model 3	1.0	0.83	[0.62, 1.10]	0.90	[0.66, 1.22]	0.76	[0.55, 1.05]	*0.13*
DASH								
cases/PY	152/12,532	110/13,716	(8.0)	130/13,249	(9.8)	99/13,246	(7.5)	
(rate per 1,000 PY)	(12.1)							
Model 1	1.0	0.64	[0.49, 0.84]	0.73	[0.56, 0.95]	0.51	[0.38, 0.69]	*<0.0001*
Model 2	1.0	0.67	[0.51, 0.89]	0.77	[0.58, 1.00]	0.54	[0.39, 0.73]	*0.0002*
Model 3	1.0	0.69	[0.51, 0.93]	0.85	[0.64, 1.14]	0.68	[0.49, 0.94]	*0.042*
aHEI								
cases/PY	152/13,082	130/13,693	(9.5)	120/13,033	(9.2)	89/12,935	(6.9)	
(rate per 1,000 PY)	(11.6)							
Model 1	1.0	0.70	[0.53, 0.91]	0.60	[0.46, 0.80]	0.43	[0.31, 0.58]	*<0.0001*
Model 2	1.0	0.72	[0.55, 0.94]	0.60	[0.45, 0.81]	0.43	[0.31, 0.59]	*<0.0001*
Model 3	1.0	0.80	[0.60, 1.07]	0.74	[0.54, 0.99]	0.65	[0.46, 0.92]	*0.013*

Model 1: age (months), total energy intake (kcal/day)

Model 2: + parity (1, 2, 3, 4+), age at first birth (12–24, 25–29, 30+), race/ethnicity (Caucasian, African-American, Hispanic, Asian, other), parental history of type 2 diabetes (yes, no), oral contraceptive use (current, former, never), menopausal status (premenopausal, peri-menopausal, post-menopausal), smoking status (never, former, current), total physical activity (MET-hours/week: quartiles); DASH analysis only: alcohol (grams/day: 0, 1–14, 15+)

Model 3: + BMI (kg/m^2: categorical <23, 24–25, 26–27, 28–30, 31–35, 36+)

GDM=gestational diabetes mellitus; aMED=alternate Mediterranean Diet; DASH=Dietary Approaches to Stop Hypertension; aHEI=alternate Healthy Eating Index; HR=hazard ratio; CI=confidence interval; PY=person-years