Are the 2005 Dietary Guidelines for Americans Associated With Reduced Risk of Type 2 Diabetes and Cardiometabolic Risk Factors?

Twenty-year findings from the CARDIA study

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OBJECTIVE—To examine the prospective association between accordance with the 2005 Dietary Guidelines for Americans (DGA) and subsequent diabetes incidence and changes in cardiometabolic risk factors.

RESEARCH DESIGN AND METHODS—The sample consisted of 4,381 black and white young adults examined repeatedly from 1985 to 2005. We used the 2005 Diet Quality Index (DQI) to rate participants' diets based on meeting key dietary recommendations conveyed by the 2005 DGA.

RESULTS—Overall, we found no association between DQI score and diabetes risk using Cox models adjusted for potential confounders. Higher DQI scores were associated with favorable changes in HDL cholesterol and blood pressure overall (*P* for trend <0.05), but with increased insulin resistance among blacks (*P* for trend <0.01).

CONCLUSIONS—Our findings highlight the need for evaluation of the DGA's effectiveness, particularly among ethnic minority populations. Clinicians should be aware that following the DGA might not lower diabetes risk.

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The Dietary Guidelines for Americans (DGA) are the basis for federal nutrition programs (1), yet there is little evidence that diets congruent with the guidelines are effective in preventing chronic disease and thus are relevant to clinical care. We examined the prospective association between a diet consistent with the key dietary recommendations of the 2005 DGA and *1*) 20-year incidence of type 2 diabetes; and *2*) 13-year changes in HDL cholesterol, insulin resistance, blood pressure, and triglycerides in a cohort of black and white Americans.

RESEARCH DESIGN AND
METHODS—The Coronary Artery
Risk Development in Young Adults (CARDIA) study consists of 5,115 black and white young adults recruited in 1985–1986 from four U.S. metropolitan areas and reexamined up to 20 years later (2). We excluded subjects who had type 2 diabetes at baseline, were pregnant, had missing data for key variables, or had unusually high or low daily energy intake (<800 or >8,000 kcal for men and <600 or >6,000 kcal for women; as per previous CARDIA research), resulting in 4,381 individuals.

Dietary intake was assessed with the CARDIA Diet History (3), an interviewer-administered instrument that includes a quantitative food frequency questionnaire. The 2005 Diet Quality Index (DQI) was designed to rate participants' diets based on meeting 2005 DGA dietary recommendations. Details on the development of the 2005 DQI are published elsewhere (4). Cardiometabolic outcomes were measured at exam years 0, 7, 10, 15, and 20. Type 2 diabetes was defined as fasting plasma glucose ≥126 mg/dL, nonfasting glucose ≥200 mg/dL, postprandial 2-h glucose ≥200 mg/dL from an oral glucose tolerance test, or current drug treatment for elevated glucose. Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated as (fasting glucose/fasting insulin)/22.5.

Statistical methods
Risk of type 2 diabetes was assessed using Cox proportional hazards regression models according to DQI score quartile (based on the cumulative average of DQI scores at years 0 and 7). Linear regression models were used to estimate 13-year changes in continuous HDL cholesterol, HOMA-IR, blood pressure, and triglycerides. Effect modification was assessed through the inclusion of interaction terms (likelihood ratio test α = 0.10).

RESULTS—Among blacks, higher DQI was associated with higher baseline BMI, but the opposite relation was seen in whites (Supplementary Table 1). We found race (but not sex) to be an effect modifier of the association between DQI score and diabetes risk (Table 1). In Cox models adjusted for lifestyle and sociodemographic characteristics, there was

Table 1—Results of multivariable Cox regressions for 20-year incidence of type 2 diabetes#

	DQI quartiles			
	1st	2nd	3rd	4th
Mean DQI score (SD)	32.1 (5.1)	43.8 (2.8)	54.2 (3.3)	69.3 (6.8)
Overall				
IR†	0.0042	0.0045	0.0042	0.0030
Model 1‡	1.00	1.08 (0.79–1.47)	1.15 (0.83–1.61)	1.05 (0.71–1.56)
Model 2	1.00	1.14 (0.84–1.56)	1.15 (0.83–1.59)	1.16 (0.79–1.71)
Blacks§				
IR†	0.0045	0.0058	0.0070	0.0046
Model 1‡	1.00	1.16 (0.81–1.66)	1.49 (1.02–2.18)	1.10 (0.65–1.86)
Model 2	1.00	1.23 (0.86–1.75)	1.40 (0.97–2.03)	0.96 (0.57–1.62)
Whites				
IR†	0.0035	0.0029	0.0022	0.0025
Model 1‡	1.00	0.83 (0.46–1.51)	0.62 (0.34–1.12)	0.78 (0.44–1.37)
Model 2	1.00	0.90 (0.49–1.65)	0.73 (0.41–1.32)	1.14 (0.65–2.00)

#Statistical analyses were set up so that diet at baseline predicted incidence from baseline to year 7, and the average of baseline and year 7 diet predicted incidence from year 7 to years 10, 15, and 20. Based on 328 incident cases of diabetes ($n = 4,381$). †Data are incidence rates = number of cases divided by person-years. ‡Data are hazard ratios (95% CI). Model 1: adjusted for age, sex, race, education, income, smoking, physical activity, energy intake, family history of type 2 diabetes, clinic, and baseline HOMA-IR. Model 2: further adjusted model 1 for baseline BMI. §Models include interaction terms for race*DQI score. IR, insulin resistance.

no significant association between DQI score and diabetes risk in whites. However, blacks in the third (vs. lowest) DQI quartile had 49% higher risk of developing diabetes. This association was no longer statistically significant after further adjusting for baseline BMI.

Participants in the highest (vs. lowest) DQI quartile had significantly less increase in blood pressure (systolic and diastolic) and greater increase in HDL cholesterol (Supplementary Table 2). Among blacks, higher DQI scores were associated with greater increase in insulin resistance, even after adjusting models for initial BMI (P for trend <0.01).

CONCLUSIONS—In this longitudinal study, we found no evidence that higher accordance with the 2005 DGA was associated with lower type 2 diabetes risk. This finding is consistent with results from a large 8-year dietary modification trial among postmenopausal women in which a diet similar to that recommended by the DGA (i.e., a diet lower in fat and higher in fruits, vegetables, and grains compared with the control diet) was not associated with lower diabetes incidence (5). Indeed, most of the individual DGA recommendations have not been proven to reduce diabetes risk (6). We also found that accordance with the 2005 DGA was inversely associated with blood pressure and HDL cholesterol, but not triglycerides. In addition, our results for type 2

diabetes and insulin resistance suggest a differential effect of diet by race, consistent with beneficial weight associations for whites but not blacks (4) and null findings for type 2 diabetes incidence but evidence of effect modification by race/ethnicity (7). It is possible that physiological/metabolic differences between blacks and whites underlie divergent results for type 2 diabetes and insulin resistance (8–11). For example, studies have found that regardless of age or adiposity, blacks have higher insulin secretion than whites (12,13), which could make them more susceptible to the glycemic effects of a high-carbohydrate diet (14).

Based on the baseline associations between DQI score and BMI, adjusting for initial BMI was expected to attenuate the relation between DQI score and diabetes risk, as well as attenuate effect modification by race. However, this was not the case. Further, even after adjusting for initial BMI, we observed a greater increase in insulin resistance among blacks with higher DQI scores. This suggests that the racial differences in initial BMI do not underlie these findings.

Although our study offers many strengths, potential weaknesses include factors related to the self-reported dietary data and the interval of measurement. However, CARDIA research suggests the dietary data are reasonably reliable and relatively stable over time (15). Further, scoring of the DQI involves quantitative

interpretation, albeit a priori and based on a validated index (4).

In terms of clinical care, it is important to note that our results do not characterize the effects of strictly following the 2005 DGA (no one in our sample received a DQI score of 100). However, the 2005 DGA executive summary states that "even following some of the recommendations can have health benefits" (1). Our results for insulin resistance in black participants do not support this statement. Indeed, a possible interpretation of our results is that, compared with blacks with low adherence to the DGA, those following some (but not all) of the dietary recommendations may have higher risk of diabetes. Our findings highlight the need for evaluation of the effectiveness of the DGA, particularly among ethnic minority populations, as has been noted by the 2010 Dietary Guidelines Advisory Committee (6). Until then, clinicians should be aware that advising African Americans to eat a diet congruent with the DGA in an effort to reduce type 2 diabetes might be premature.

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