

High Caloric and Sodium Intakes as Risk Factors for Progression of Retinopathy in Type 1 Diabetes Mellitus

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Objective: To report the association of dietary nutrient intakes in relation to the 6-year progression of diabetic retinopathy (DR) in African American patients with type 1 diabetes mellitus.

Methods: African American patients with type 1 diabetes who participated in the baseline and 6-year follow-up examinations as part of the New Jersey 725 study were included. At the baseline examination, a food frequency questionnaire was used to document average daily dietary nutrient intakes. Clinical evaluations at baseline and at the 6-year follow-up also included a structured clinical interview, ocular examination, grading of 7 standard field stereoscopic fundus photographs, and blood pressure measurements. Biological evaluations included blood and urine assays. Nutrient intake data were analyzed using DietSys software and nutrient databases developed by the National Cancer Institute.

Results: Among the 469 participants at risk for progression of DR, baseline total caloric intake was significantly associated with 6-year incidence of vision-threatening DR (either proliferative DR or macular edema) and of severe hard exudates—after adjusting for clinical risk factors for DR progression. Baseline high sodium intake was a significant and independent risk factor for 6-year incidence of macular edema.

Conclusions: In African American patients with type 1 diabetes, high caloric and sodium intakes are significant and independent risk factors for progression to severe forms of DR. Dietary recommendations of low caloric and sodium intakes may be beneficial in relation to the development of DR.

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DIABETIC RETINOPATHY (DR) remains the leading cause of blindness in persons aged 20 to 64 years who have diabetes mellitus (DM) in the United States despite effective treatments for the severe forms of the disease.^{1,2} Proliferative DR (PDR) and macular edema (ME)—so-called vision-threatening DR (VTDR)—are the 2 main causes of visual loss in persons with DM.³ Clinical risk factors for progression of DR have been previously reported for various populations with type 1 DM, including those who are African American.³⁻⁹ The risk factors include longer duration of diabetes, poor glycemic control, and systemic hypertension. In patients with DM, glycemic and blood pressure control are achieved through medication and specific dietary recommendations established by the American Diabetic Association (ADA).¹⁰ While dietary intake may be an important determinant of DR progression, its role has been evaluated in only a few studies and in white

diabetic populations.¹¹⁻¹⁵ To our knowledge, such data are not available for African American individuals with type 1 DM.

Because both hyperglycemia and hyperlipidemia are present in type 1 DM, the ADA recommends restriction of carbohydrate and fat intakes as well as high fiber intake.^{10,16,17} The ADA also recommends low sodium intake for persons with DM who have hypertension.¹⁰ This may be of particular importance for African American individuals with type 1 DM, in whom hypertension is common and often uncontrolled despite use of antihypertensive medications.^{5,18,19}

We previously assembled (at baseline), examined (at baseline), and reexamined (at a 6-year follow-up) a large, geographically well-defined population of African American patients diagnosed with type 1 DM—the New Jersey 725.^{5,7,18,20} Standard protocols were used to document dietary nutrient intake at baseline as well as diabetic complications and biological abnormalities.^{21,22} At each examination, patients' retinal status was well

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characterized using masked grading of the 7 standard retinal photographs.^{23,24} Thus, our New Jersey 725 cohort provides a unique opportunity to examine the possible role of dietary nutrient intake, recorded at baseline examination, in relation to the 6-year progression of DR in this population.

We hypothesized the following for African American patients with type 1 DM: (1) baseline high caloric, high fat, low dietary fiber, and/or high carbohydrate intakes are associated with 6-year progression of DR, and (2) baseline high dietary sodium intake is associated with 6-year incidence of ME.

METHODS

STUDY POPULATION

The original cohort consisted of 725 African American patients with type 1 DM who participated in the New Jersey 725 study between August 1993 and January 1998.²⁰ Patients were identified from the New Jersey hospital discharge data, which list all patients admitted to New Jersey hospitals who have a discharge diagnosis of DM. This computer-generated list includes demographic information about patients including race. African American patients diagnosed with DM, treated with insulin before age 30 years, and currently receiving insulin were further identified from a random review of 13 615 medical records. Patients with type 2 DM, those diagnosed after age 30 years, and those with maturity-onset diabetes of youth were excluded.²⁵⁻²⁷ Ethnicity was determined from the hospital record and later confirmed by self-identification. Patients were also asked to confirm that both of their parents were African American.

Of the original cohort of 725 patients, 508 (70.1%) participated in the 6-year follow-up between October 1999 and August 2004, 44 (6.1%) could not be located, 34 (4.7%) refused examination, and 139 (19.2%) had died during the 6-year interval.¹⁸ Baseline characteristics of participants and nonparticipants have been previously reported.¹⁸ Of the 508 patients with a 6-year follow-up, 25 were no longer receiving insulin and have been excluded, leaving 483 patients available for analysis. Of those 483 patients, 469 were at risk for progression of DR at baseline and had baseline dietary nutrient intake data.

CLINICAL PROCEDURES

Patients underwent similar procedures at baseline and the 6-year follow-up. Patients were examined in the Eye Clinic, University Hospital, Newark, New Jersey. On arrival, informed written consent was obtained from each participant. The institutional review board of the New Jersey Medical School, University of Medicine and Dentistry of New Jersey, Newark approved the study.

Food Frequency Questionnaire

The reduced 60-item questionnaire (BRIEF87) of the Health Habits and History Questionnaire developed by the National Cancer Institute was administered by the research assistant (Deborah Nuber, MPH).²² The average frequency of consumption (per day, per week, per month, per year, rarely, or never) and serving size (small, medium, or large) of the various foods were recorded. Food models were used to evaluate serving sizes for fruits and vegetables, meats, breads, breakfast foods, and sweets usually consumed. Nutrient intakes were calculated from

the responses on the questionnaire using DietSys version 3.0 software (National Cancer Institute, Information Management Services, Inc, Bethesda, Maryland, and Block Dietary Data Systems, Berkeley, California) and nutrient databases developed by the National Cancer Institute.²⁸ Information regarding the following nutrients were obtained: total calories, protein, carbohydrate, total fat, saturated fat, oleic acid (monounsaturated fat), linoleic acid (polyunsaturated fat), cholesterol, alcohol, and sodium.

Patient Examination

Patients had a complete eye examination including dilated retinal examination and 7 standard stereoscopic Diabetic Retinopathy Study retinal photographs. Previous ophthalmic medical records were obtained to document laser and surgical procedures for either PDR or ME. Height, weight, and blood pressure measurements were also obtained. Systemic hypertension was considered present if the systolic blood pressure was 140 mm Hg or higher, the diastolic blood pressure was 90 mm Hg or higher, and/or the patient was receiving antihypertensive medication. A structured clinical interview included detailed medical and ophthalmologic histories, sociodemographic factors, and lifestyle variables. Patients' socioeconomic status was classified from the Goldthorpe and Hope classification of occupations into middle to high class (levels 1-22) and low class (levels 23-36) using the occupation of the head of the household.²⁹ Smoking status was defined as ever smoked or never smoked. Exercise was considered present if the patient exercised for at least 1 hour at least 3 times a week and the activity was associated with sweating.

Biological Evaluation

Venous blood was drawn for measurement of total glycosylated hemoglobin, high-density lipoprotein cholesterol, low-density lipoprotein cholesterol, and total cholesterol. A 4-hour timed urine collection was obtained for measurement of the albumin excretion rate and creatinuria. Renal disease was considered present if the patient had either microalbuminuria (albumin excretion rate = 20-200 µg/min) or overt proteinuria (albumin excretion rate >200 µg/min), was on dialysis, or had received a kidney transplant.

DR Grading

Color fundus photographs obtained at both baseline and follow-up were graded for DR severity in a masked fashion by the Wisconsin Fundus Photograph Reading Center, Madison, Wisconsin. The modified Early Treatment Diabetic Retinopathy Study Airlie House classification of DR was used.²⁴ Level 10 indicates no DR, levels 20 to 53 indicate nonproliferative DR of increasing severity, and levels 61 to 85 indicate PDR of increasing severity. Patients who had received panretinal laser photocoagulation or underwent pars plana vitrectomy for PDR were classified as having PDR. For each participant, the retinopathy level was determined using the severity level of the worse eye. Six-year incidence of any DR was calculated for patients who had no DR at baseline (n = 191).

Macular edema was considered present if there was thickening of the retina with or without partial loss of retinal transparency within 1 disc diameter from the center of the macula and/or if there were focal laser photocoagulation scars in the macular area and a documented history of ME. If ME could not be graded in one eye, the participant was assigned the score of the other eye. Six-year incidence of ME was calculated for all patients who had no ME in either eye at baseline (n = 422). Six-

Table 1. Baseline Clinical Characteristics and Dietary Nutrient Intakes in African American Men and Women With Type 1 Diabetes Mellitus

Baseline Variable	Men (n=189)	Women (n=280)	P Value
Clinical characteristic			
Age, mean (SD), y	26.7 (10.7)	27.8 (10.8)	.27 ^a
Diabetes, mean (SD), y			
Duration	8.9 (7.7)	11.4 (9.0)	.002 ^a
Age at diagnosis	17.3 (7.8)	16.0 (7.4)	.07 ^a
Glycated hemoglobin, mean (SD), % of total hemoglobin	13.6 (4.1)	13.5 (4.4)	.70 ^b
Lower socioeconomic status, %	57.1	52.9	.36 ^b
Ever smoked, %	45.0	38.9	.19 ^b
Getting physical exercise, %	47.1	27.1	<.001 ^b
Dietary nutrient intake, mean (SD)			
Food energy, kcal	2309.6 (1104.5)	1706.2 (937.3)	<.001 ^a
Protein, % of total kcal	17.9 (3.6)	17.6 (3.6)	.42 ^a
Total carbohydrate, % of total kcal	41.8 (8.6)	42.6 (8.8)	.32 ^a
Total fat, % of total kcal	39.6 (7.1)	40.2 (8.0)	.42 ^a
Saturated fat, % of total kcal	14.0 (3.0)	14.0 (3.1)	.96 ^a
Oleic acid, % of total kcal	14.1 (2.7)	14.1 (3.1)	.91 ^a
Linoleic acid, % of total kcal	7.9 (2.8)	8.6 (3.4)	.03 ^a
Cholesterol, mg/1000 kcal	221.9 (158.1)	205.8 (115.1)	.20 ^a
Dietary fiber, g/1000 kcal	6.5 (2.8)	7.1 (2.9)	.06 ^a
Alcohol, % of total kcal	1.5 (3.8)	0.4 (1.9)	<.001 ^a
Sodium, mg/1000 kcal	1685.9 (364.5)	1686.0 (381.6)	.99 ^a

SI conversion factor: To convert glycated hemoglobin to proportion of total hemoglobin, multiply by 0.01.

^aUsing standard *t* test.

^bUsing χ^2 statistic.

year progression to VTDR was calculated for all patients who had neither PDR nor ME at baseline (n=381).

Presence of retinal hard exudates (HEs) in each eye was based on the modified Airlie House classification of DR.²³ Severe HEs were considered absent if HEs were either absent or questionable in both eyes or were no worse than level 2 in either eye; severe HEs were considered present if HEs were at level 3, 4, or 5 in either eye. Six-year incidence of severe HEs was calculated for all patients who had no severe HEs at baseline (n=413).

STATISTICAL ANALYSES

Data management and statistical analyses were performed using Windows-based SPSS version 15.0 statistical software (SPSS Inc, Chicago, Illinois). Of the original cohort of 725 patients, 697 had baseline dietary data. Compared with patients without 6-year follow-up (n=204), those with 6-year follow-up (n=493) had a significantly shorter duration of diabetes at baseline (mean [SD], 14.3 [10.1] vs 10.2 [8.5] years, respectively; *P*=.01) and higher body mass index (BMI [calculated as weight in kilograms divided by height in meters squared]) (mean [SD], 26.4 [7.4] vs 28.3 [8.9], respectively; *P*=.03). There were no significant differences between the 2 groups for sex, age at diagnosis of diabetes, baseline age, socioeconomic status, smoking, exercise, glycated hemoglobin values, and any dietary nutrient intake (data not shown).

Data were analyzed for the 469 patients who were at risk for progression of DR at baseline and had baseline dietary nutrient intake data. Differences between men and women for each nutrient intake were compared with analysis of covariance adjusting for total caloric intake. Using regression analyses, each nutrient was first transformed into quartiles; linear relationships were then evaluated between baseline nutrient intakes and (1) baseline clinical characteristics and (2) 6-year incidence of any DR, VTDR, ME, and severe HEs. Various multiple logistic regression models were used to compute odds ra-

tios and 95% confidence intervals for 6-year progression of DR as a function of quartiles of nutrients and known risk factors. In model 1, all independent nutrients were allowed to enter a forward conditional model including total caloric intake. In model 2, clinical risk factors for 6-year progression of DR were added to the nutrient list to identify potential confounding factors. Baseline age, sex, glycated hemoglobin level, hypertension, proteinuria, blood cholesterol level, socioeconomic status, and physical exercise were included in model 2.

RESULTS

Results are presented for the 469 patients (64.7% of the original cohort) with a 6-year follow-up examination who were at risk for progression of DR at baseline and had baseline dietary nutrient intake data. Compared with African American women (n=280), African American men (n=189) had a significantly shorter duration of DM, exercised more, and had a higher total caloric intake (**Table 1**). After adjusting for caloric intake, linoleic acid intake was higher and alcohol intake was lower among women as compared with men (Table 1). There was no significant difference between men and women for any other nutrient.

In the 469 African American patients, daily protein intake represented 17.7% of total caloric intake, total carbohydrate 42.2%, total fat 40.0%, saturated fat 14.0%, oleic acid 14.1%, and linoleic acid 8.3%. Daily cholesterol intake was high (mean [SD], 409.9 [491.2] mg/d; interquartile range, 201.1-492.9). After adjusting for caloric intake, dietary cholesterol intake was significantly associated with baseline blood cholesterol level (*P*=.003).

Table 2. Univariate Analysis of the Relationship of Baseline Dietary Nutrient Intakes to 6-Year Progression of Diabetic Retinopathy in Type 1 Diabetes Mellitus

Baseline Dietary Nutrient Intake	VTDR		ME		Severe HEs	
	OR (95% CI)	P Value	OR (95% CI)	P Value	OR (95% CI)	P Value
Total calories, kcal	1.39 (1.11-1.75)	.004	1.18 (0.94-1.49)	.16	1.48 (1.15-1.91)	.003
Carbohydrate, g	1.29 (1.03-1.62)	.03	1.08 (0.85-1.36)	.54	1.34 (1.05-1.72)	.02
Protein, g	1.36 (1.08-1.70)	.009	1.18 (0.93-1.49)	.17	1.42 (1.10-1.83)	.007
Total fat, g	1.36 (1.08-1.71)	.009	1.15 (0.91-1.45)	.25	1.43 (1.11-1.84)	.006
Saturated fat, g	1.01 (1.002-1.03)	.03	1.01 (0.99-1.02)	.32	1.02 (1.002-1.03)	.02
Oleic acid, g	1.40 (1.11-1.77)	.004	1.18 (0.93-1.49)	.17	1.44 (1.12-1.86)	.005
Linoleic acid, g	1.33 (1.06-1.66)	.01	1.14 (0.90-1.44)	.27	1.35 (1.06-1.73)	.02
Fiber, g	1.23 (0.99-1.53)	.07	1.20 (0.95-1.52)	.13	1.20 (0.94-1.53)	.15
Cholesterol, mg	1.39 (1.11-1.74)	.004	1.21 (0.95-1.53)	.11	1.36 (1.06-1.74)	.02
Sodium, mg	1.37 (1.09-1.72)	.008	1.31 (1.03-1.66)	.03	1.36 (1.06-1.76)	.02

Abbreviations: CI, confidence interval; HEs, hard exudates; ME, macular edema; OR, odds ratio; VTDR, vision-threatening diabetic retinopathy.

RELATIONSHIP BETWEEN BASELINE DIETARY NUTRIENT INTAKE AND CLINICAL CHARACTERISTICS OF PARTICIPANTS

Patients' age showed a significant but nonlinear association with total caloric, protein, fat, and sodium intakes, all of which increased until the third decade of life and then decreased with increasing age (data not shown). Patients with a BMI lower than 25 had significantly higher sodium ($P=.02$) and protein ($P=.02$) intakes than patients with higher BMI. Patients who exercised had significantly higher intakes of protein ($P=.02$), carbohydrate ($P=.006$), and fiber ($P=.002$) than patients who did not exercise. Higher baseline total caloric, linoleic acid, and carbohydrate intakes were associated with significantly higher glycated hemoglobin values at baseline ($P=.04$, $P=.02$, and $P=.04$, respectively). There was no significant association between any of the baseline nutrient intakes and socioeconomic status, smoking, or age at diagnosis of diabetes (data not shown).

RELATIONSHIP BETWEEN BASELINE DIETARY NUTRIENT INTAKE AND 6-YEAR PROGRESSION OF DR

Univariate Analysis

At baseline, quartiles of total caloric, protein, carbohydrate, total fat, saturated fat, oleic acid, linoleic acid, cholesterol, and sodium intakes were all significantly and positively associated with 6-year progression to VTDR (**Table 2**). Baseline sodium intake was significantly and positively associated with 6-year incidence of ME. Baseline total caloric, protein, carbohydrate, total fat, saturated fat, oleic acid, linoleic acid, cholesterol, and sodium intakes were significantly and positively associated with 6-year incidence of severe HEs.

Multivariate Analyses

To identify baseline dietary nutrient intakes significantly and independently associated with 6-year incidence of VTDR, ME, and/or severe HEs, various models were run adjusting for total caloric intake and/or clinical

risk factors for progression of DR.¹⁸ When baseline protein, carbohydrate, total fat, saturated fat, oleic acid, linoleic acid, fiber, cholesterol, sodium, and total caloric intakes were included in a forward conditional entry logistic regression model (model 1), higher intake of oleic acid was the single most important nutrient and only predictor of 6-year incidence of VTDR ($P=.004$), higher total caloric intake was the only significant nutrient and only predictor of higher 6-year incidence of severe HEs ($P=.003$), and higher sodium intake was the only significant nutrient and only predictor of higher 6-year incidence of ME ($P=.03$) (**Table 3**).

In model 2, we examined intake of oleic acid, protein, carbohydrate, and total calories while adjusting for baseline age, sex, physical exercise, glycated hemoglobin level, and systemic hypertension using forward conditional entry. Table 3 shows that older age, higher glycated hemoglobin level, systemic hypertension, and total caloric intake were significant and independent risk factors for 6-year incidence of VTDR. Oleic acid was no longer significantly associated with incidence of VTDR. Older age, higher glycated hemoglobin level, systemic hypertension, and total caloric intake were significant and independent risk factors for 6-year incidence of severe HEs. Older age, higher glycated hemoglobin level, systemic hypertension, and higher sodium intake were significant and independent risk factors for 6-year incidence of ME. When proteinuria instead of hypertension was entered into model 2, proteinuria was a significant and independent risk factor for incidence of VTDR (odds ratio=1.92; 95% confidence interval, 1.09-3.41; $P=.02$).

COMMENT

The results of this study indicate that high caloric intake at baseline is significantly and independently associated with 6-year incidence of VTDR (either PDR or ME) as well as with 6-year incidence of severe HEs in African American patients with type 1 DM. Higher sodium intake at baseline was also significantly and independently associated with 6-year incidence of ME in this population.

Our finding that high caloric intake is associated with progression to the severe forms of DR (either PDR or ME), independently of the known clinical risk factors, is consistent with some of the results from the Diabetes Control and Complications Trial.¹⁵ In that study of mostly white patients with type 1 DM, high caloric, monounsaturated (eg, oleic acid) and polyunsaturated (eg, linoleic acid) fat, and cholesterol intakes were all significantly associated with progression of DR at the end of the study.¹⁵ In the Diabetes Control and Complications Trial, as in our study, total caloric intake was also significantly and positively associated with higher glycated hemoglobin levels, one of the strongest risk factors for progression of DR.^{15,30,31} The increased metabolic burden and oxidative stress associated with hyperglycemia and dyslipidemia present in DM may be mechanisms underlying high caloric intake as a risk factor for progression of DR.¹⁶ The retina is particularly susceptible to oxidative stress because of its high lipid content.³² Hyperlipidemia may lead to peroxidation of lipids, an increase in the oxidized low-density lipoprotein cholesterol level, and decreased antioxidant levels resulting from competition for reduced nicotinamide adenine dinucleotide phosphate from the sorbitol pathway.¹⁶

It is noteworthy that there is also experimental and clinical evidence suggesting that caloric restriction itself has beneficial antiaging and/or antidisease effects.³³⁻³⁶ For instance, in animals, low caloric intake has been shown to lower oxidative stress, increase insulin sensitivity, reduce the cholesterol level and blood pressure, elevate the high-density lipoprotein cholesterol level, and slow age-related decline in circulating plasma levels of dehydroepiandrosterone sulfate.³³ Caloric restriction improves parameters of oxidative stress in persons with type 2 DM and has been shown to have a beneficial effect for other diseases.^{36,37}

In this study, the association of monounsaturated (rather than saturated) fat intake with progression of DR is somewhat unexpected because saturated fats, particularly from animal sources, are a known cause of atherosclerosis.³⁸ In a small longitudinal randomized study, Houtsmuller et al¹¹ reported that a diet rich in the polyunsaturated fatty acid linoleic acid slowed the 6-year progression of DR in patients with type 2 DM. Interestingly, only female patients showed improvement in the lipid profile in that study, suggesting that the latter might not have been the only explanation for the beneficial effect of linoleic acid on the development of DR.¹¹ A later, similar study showed the same but less dramatic beneficial effects.¹² It is noteworthy that compared with current dietary recommendations of the ADA for persons with DM, our African American patients consumed a greater percentage of their energy intake as saturated fat (<10% vs 14.0%, respectively), polyunsaturated fat (10% vs 17.0%, respectively), and dietary cholesterol (<300 mg/d vs 409.9 mg/d, respectively).¹⁰ Because total energy intake is increased when the diet is high in fat, it is difficult to distinguish the effect of high caloric vs high fat intake on the progression of DR in our African American patients.^{10,38} Our data suggest that both high caloric and high fat intakes, specifically of oleic acid, are risk factors for

Table 3. Multivariate Analysis of the Relationship of Baseline Dietary Nutrient Intakes and Clinical Characteristics to 6-Year Progression of Diabetic Retinopathy in Type 1 Diabetes Mellitus^a

Model	OR (95% CI)	P Value
VTDR ^b		
Model 1		
Oleic acid intake, quartiles, g	1.40 (1.11-1.77)	.004
Model 2		
Age, y	2.23 (1.63-3.05)	<.001
Glycated hemoglobin, % of total hemoglobin	1.25 (1.16-1.35)	<.001
Hypertension, present vs absent	1.98 (1.13-3.48)	.02
Total caloric intake, quartiles, kcal	1.49 (1.15-1.92)	.002
Severe hard exudates		
Model 1		
Total caloric intake, quartiles, kcal	1.48 (1.15-1.91)	.003
Model 2		
Age, y	1.59 (1.18-2.14)	.002
Glycated hemoglobin, % of total hemoglobin	1.16 (1.08-1.25)	<.001
Hypertension, present vs absent	2.11 (1.17-3.80)	.01
Total caloric intake, quartiles, kcal	1.58 (1.20-2.07)	.001
Macular edema		
Model 1		
Sodium intake, quartiles, g	1.31 (1.03-1.66)	.03
Model 2		
Age, y	1.99 (1.45-2.71)	<.001
Glycated hemoglobin, % of total hemoglobin	1.23 (1.14-1.32)	<.001
Hypertension, present vs absent	2.47 (1.38-4.45)	.002
Sodium intake, quartiles, mg	1.43 (1.10-1.86)	.008

Abbreviations: CI, confidence interval; OR, odds ratio; VTDR, vision-threatening diabetic retinopathy.

^aModel 1 includes total caloric, total fat, saturated fat, oleic acid, linoleic acid, carbohydrate, protein, fiber, cholesterol, and sodium intakes. Model 2 includes age, sex, physical exercise, glycated hemoglobin level, oleic acid intake, total caloric intake, protein intake, carbohydrate intake, and hypertension.

^bEither proliferative diabetic retinopathy or macular edema.

progression of DR in our African American patients independently of the known clinical risk factors for DR.

Our hypothesis that high sodium intake is significantly and independently associated with 6-year incidence of ME was confirmed. Our African American patients had a high mean sodium intake (3234.5 mg/d) compared with the 2400 mg/d currently recommended by the ADA for persons with DM.¹⁰ This is consistent with the high incidence of ME in our African American patients (16.0% at the 6-year follow-up compared, for instance, with 8% at the 4-year follow-up for white patients with type 1 DM).^{18,39} Higher prevalence and severity of systemic hypertension in African American patients compared with white patients with type 1 DM may in part explain the difference in incidence of ME between these 2 populations because hypertension is one of the clinical risk factors for incidence of ME.^{3,9,18} In our African American patients, not only was the prevalence of hypertension at the 6-year follow-up (44.0%) high, but blood

pressure was not controlled in 49.0% of those receiving antihypertensive medication.¹⁸ It is thought that increased salt sensitivity in some African American individuals may in part be responsible for the high prevalence and severity of hypertension.¹⁹ Because lowering sodium intake has been shown to lower blood pressure, African American patients with type 1 DM may be advised to reduce salt intake in their diet.⁴⁰

Strengths of this study include the assessment of baseline dietary nutrient intake on the progression of DR in a well-characterized population of African American patients with type 1 DM. Standard protocols were used at both baseline and follow-up to document DR and other diabetic complications. Clinical risk factors associated with progression of DR were evaluated. The standard diet questionnaire used has been shown to have good reproducibility and reliability to evaluate dietary nutrient intakes, particularly in minority populations.^{41,42} Calorie-adjusted rather than absolute nutrient intakes were used to examine their association with progression of DR.⁴³ Limitations of the study are that nutrient intake was evaluated only on a single visit rather than repeated over time and that we did not use a 3-day food frequency questionnaire.⁴⁴ In the data analyses, we did not adjust for multiple comparisons. However, we had specific hypotheses regarding which nutrient intake (total calories, fat, and/or sodium) might be associated with DR progression. Blood levels of the nutrients (except cholesterol) were not obtained. Patients included in the study had been admitted to hospitals at some time and thus may have had more severe DR than nonhospitalized patients. Finally, results found here may not apply beyond the population that was studied.

In summary, results of our study indicate that high caloric intake and high sodium intake are associated with progression of DR independently of known clinical risk factors. These results suggest that low caloric and sodium intakes in African American individuals with type 1 DM may have a beneficial effect on the progression of DR and thus might be part of dietary recommendations for this population.

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