Ethnicity, Obesity, and Risk of Type 2 Diabetes in Women

A 20-year follow-up study

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OBJECTIVE — To examine ethnic differences in risk of type 2 diabetes, taking dietary and lifestyle risk factors into account.

RESEARCH DESIGN AND METHODS — A prospective (1980–2000) cohort (from The Nurses' Health Study) including 78,419 apparently healthy women (75,584 whites, 801 Asians, 613 Hispanics, and 1,421 blacks) was studied. Detailed dietary and lifestyle information for each participant was repeatedly collected every 4 years.

RESULTS — During 1,294,799 person-years of follow-up, we documented 3,844 incident cases of diabetes. Compared with whites, the age-adjusted relative risks (RRs) were 1.43 (95% CI 1.08–1.90) for Asians, 1.76 (1.32–2.34) for Hispanics, and 2.18 (1.82–2.61) for blacks. After adjustment for BMI, the RRs changed to 2.26 (1.70–2.99) for Asians, 1.86 (1.40–2.47) for Hispanics, and 1.34 (1.12–1.61) for blacks. For each 5-unit increment in BMI, the multivariate RR of diabetes was 2.36 (1.83–3.04) for Asians, 2.21 (1.75–2.79) for Hispanics, 1.96 (1.93–2.00) for whites, and 1.55 (1.36–1.77) for blacks (P for interaction <0.001). For each 5-kg weight gain between age 18 and the year 1980, the risk of diabetes was increased by 84% (95% CI 58–114) for Asians, 44% (26–63) for Hispanics, 38% (28–49) for blacks, and 37% (35–38%) for whites. A healthy diet high in cereal fiber and polyunsaturated fat and low in *trans* fat and glycemic load was more strongly associated with a lower risk of diabetes among minorities (RR 0.54 [95% CI 0.39–0.73]) than among whites (0.77 [0.72–0.84]).

CONCLUSIONS — The risk of diabetes is significantly higher among Asians, Hispanics, and blacks than among whites before and after taking into account differences in BMI. Weight gain is particularly detrimental for Asians. Our data suggest that the inverse association of a healthy diet with diabetes is stronger for minorities than for whites.

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Ethnic differences in prevalence of type 2 diabetes are well documented. In 2004, the prevalence of diagnosed diabetes in the U.S. was higher for blacks and Hispanics than for whites across all age-groups (1). For women aged 45–64 years, the prevalence was 7.8% among whites, 13.5% among Hispanics, and 15.4% among blacks. From 1980 through 2004, the age-adjusted prevalence increased by 65% among white women and 37% among black men (1–5). A random sample of 5% of Medicare fee-for-service beneficiaries ≥65 years of age (6) showed the prevalence of type 2 diabetes to be the highest among Hispanics, the lowest among whites, and intermediate for blacks and Asians. The greatest increase in diabetes prevalence was observed among Asians (68%) during the 7-year period from 1997 to 2004 (6). In a recent population-based national U.S. telephone survey (7), the prevalence of diabetes was significantly higher among Asian Americans than among non-Hispanic whites, after accounting for a lower BMI among Asians.

Diabetes is primarily determined by obesity and lifestyle factors such as diet and exercise (8). However, no previous study has examined whether these factors can explain ethnic differences in prevalence of diabetes. Therefore, we conducted a prospective analysis of ethnic differences in type 2 diabetes risk among 78,419 apparently healthy middle-aged women in the Nurses' Health Study during 20 years of follow-up.

A unique feature of this study is the detailed information on diet and lifestyle factors that has been repeatedly obtained during follow-up.

RESEARCH DESIGN AND METHODS

The Nurses' Health Study population

The Nurses' Health Study cohort was established in 1976 when 121,700 female registered nurses, aged 30–55 years and residing in 11 states, completed a mailed questionnaire about their medical history and lifestyle. Follow-up questionnaires have been sent every 2 years to update information on potential risk factors and to identify newly diagnosed cases of cancer, coronary heart disease, diabetes, and other medical conditions. For the present analysis, we used information from respondents to the 1980 questionnaire, when we first inquired about diet. We excluded women who reported a diagnosis of diabetes, cancer, or cardiovascular disease on the 1980 or a previous questionnaire. The remaining 78,419 women were followed-up for diabetes incidence dur-

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ing the subsequent 20 years (1980–2000).

Dietary assessment

We used validated semiquantitative food-frequency questionnaires to assess the participants' diets. Full descriptions of the food-frequency questionnaire in its abbreviated (61 items: 1980) and expanded (116–136 items: 1984, 1986, 1990, 1994, and 1998) forms, the procedures for calculating nutrient intakes, and data on reproducibility and validity in this cohort have been reported previously (9). The glycemic load was calculated by multiplying the grams of carbohydrate in each serving by its glycemic index value. Each unit of dietary glycemic load represents the equivalent effects of 1 g of carbohydrate from pure glucose (8).

Measurement of nondietary factors

In 1980, participants provided information on their weight and smoking status. We updated this information every 2 years during follow-up. The validity of self-reported weight in this cohort was reported previously ($r = 0.96$ between self-reported and measured weight) (10). Recalled weight at age 18 years was also highly correlated ($r = 0.87$, mean difference with measured weight from the physical examination records for the same age among 118 women [recalled − measured weight] = −1.4 kg) (11).

Information about physical activity was first obtained in 1980 and updated periodically by a validated questionnaire (12). The presence or absence of a family history of diabetes (first-degree) was assessed in 1982 and 1988. Ethnicity was reported in 1992. Participants were classified as white only if they reported being white (i.e., Southern European/Mediterranean origin, Scandinavian, and other Caucasians). Participants were classified as Asians if they reported that they were Asians other than whites. Participants were considered Hispanic if they reported that they were Hispanic other than whites and Asians. Participants were classified as blacks if they reported that they were African American other than the above-mentioned ethnic groups.

Follow-up and ascertainment of incident cases of type 2 diabetes

On the baseline and follow-up questionnaires mailed every 2 years, we inquired about whether diabetes had been newly diagnosed. When a diagnosis of diabetes was reported, participants were asked to complete a supplementary questionnaire to confirm the report and to ascertain the date of diagnosis and the details of the diagnostic tests, presenting symptoms, and medications prescribed. After the exclusion of women who had type 1 or gestational diabetes only, the diagnosis of type 2 diabetes was established if one or more of the following criteria were met: *1*) one or more classic symptoms (excessive thirst, polyuria, weight loss, or pruritus) plus a fasting plasma glucose concentration ≥140 mg/dl (7.78 mmol/l) or a random plasma glucose concentration ≥200 mg/dl (11.1 mmol/l), *2*) two elevated plasma glucose concentrations on different occasions (fasting ≥140 mg/dl [7.78 mmol/l], random ≥200 mg/dl [11.1 mmol/l], or ≥200 mg/dl [11.1 mmol/l] after ≥2 h of oral glucose tolerance testing) in the absence of symptoms, or *3*) treatment with medication for hyperglycemia (insulin or oral hypoglycemic agents). These criteria correspond with those proposed in 1979 by the National Diabetes Data Group (13). In 1997, the fasting plasma glucose concentration indicative of type 2 diabetes was lowered (≥126 mg/dl [7.0 mmol/l]) (14). Here we used the previous criterion because the National Diabetes Data Group definition was the standard during most of the follow-up period. The high validity of self-reported diabetes in this cohort on the supplementary questionnaire was previously documented, and the diagnosis was confirmed by a review of medical records in 98% of cases (15).

Statistical analysis

For each participant, person-time of follow-up was counted from the date of return of the 1980 questionnaire to the date of diabetes diagnosis, the return of the most recent follow-up questionnaire, or 1 June 2000, whichever came first. We used general linear models to compare the age-adjusted means and proportions of the population baseline characteristic across the ethnic groups. We conducted Cox proportional hazard analysis stratified on 5-year age categories and over each 2-year follow-up interval to estimate the relative risks (RRs) of diabetes. To evaluate whether obesity explained ethnic differences in diabetes risk, we first added BMI values as a continuous variable (updated every 2 years) to the age-adjusted model. Subsequently, we tested a multivariable model by further adjusting for family history of diabetes in a first-degree relative, smoking status, physical activity, and alcohol intake. We further adjusted for dietary score and total energy intake. The dietary score was calculated as the sum of the quintile values of cereal fiber and the ratio of polyunsaturated to saturated fats in ascending order and of *trans* fats and glycemic load in descending order (9). We evaluated the contribution of BMI (in 5-unit increments) to diabetes incidence among each ethnic group in stratified analyses. The test for interaction was performed using the likelihood ratio test for nested models with and without interaction terms for ethnicity and BMI (16). All statistical analyses were performed with SAS statistical software version 8.0 (SAS Institute, Cary, NC).

RESULTS — Table 1 shows age-adjusted baseline characteristics of 78,419 women across the ethnic groups. Blacks were heavier, more sedentary, and had a higher prevalence of hypertension than whites. Hispanics had risk-factor patterns similar to those of whites, except for a lower prevalence of smoking. Asians were leaner than whites but more sedentary, had a lower prevalence of smoking, and tended to consume less alcohol and to eat a healthier diet, as assessed by our dietary score.

During 20 years of follow-up (1,294,799 person-years), we documented 3,844 incident cases of type 2 diabetes. In age-adjusted models (Table 2), minority groups, especially blacks, had significantly higher risk of diabetes than whites. Further adjustment for updated BMI attenuated the RR among the blacks to 1.34 (95% CI 1.12–1.61) and appreciably increased the RR among Asians to 2.26 (1.70–2.99). Adjustment for BMI did not materially change the RR of type 2 diabetes among Hispanics. Further adjustment for family history of diabetes, alcohol intake, smoking status, and physical activity only slightly attenuated these RRs. Additional adjustments for updated dietary score and energy intake did not appreciably alter these RRs.

During the 20 years of follow-up, BMI increased across all the ethnic groups (Fig. 1). In multivariate analyses, for each 5-unit increment in BMI, the RRs of diabetes were 2.36 (95% CI 1.83–3.04) for Asians, 2.21 (1.75–2.79) for Hispanics, 1.96 (1.93–2.00) for whites, and 1.55 (1.36–1.77) for blacks (P for interaction between BMI and ethnicity <0.001) (Table 3). We further evaluated weight change between age 18 years and the year 1980 in relation to incidence of diabetes

Table 1—*Age-adjusted diabetes-related risk factors of 78,419 women without diabetes at baseline (1980) across ethnic groups*

	Ethnic group			
	White*	Asian	Hispanic	Black
n	75,584	801	613	1,421
Age (years)	45.7	45.9	46.2	46.1
BMI (kg/m^2)	24.3 ± 4.4	22.7 ± 0.43†	24.3 ± 4.0	26.0 ± 5.3†
Parental history of diabetes (%)	17.1	21.1‡	21.4‡	17.7
Moderate/vigorous exercise (h/week)	4.0 ± 2.7	3.4 ± 3.7†	3.8 ±4.2	3.4 ± 3.8†
Current smoker (%)	28.2	14.6†	15.1†	25.9
Hypertension (%)	14.0	13.4	10.4‡	28.1†
Hypercholesterolemia (%)	4.6	6.5‡	5.4	4.9
Alcohol consumption (g/day)	6.5 ± 10.4	2.6 ± 8.1†	4.7 ± 14.6‡	4.3 ± 12.8
Polyunsaturated fat (% energy)	5.3 ± 1.56	5.0 ± 1.4†	4.8 ± 0.2†	5.1 ± 3.8†
Saturated fat (% energy)	15.6 ±3.5	14.5 ± 3.9†	15.2 ± 5.0‡	14.0 ± 3.8†
Polyunsaturated-to-saturated fat ratio	0.4 ± 0.1	0.4 ± 0.2†	0.3 ± 0.02‡	0.4 ± 0.4†
Trans fat (% energy)	2.2 ± 0.7	1.9 ± 0.6†	2.0 ± 0.02†	1.9 ± 0.4†
Total fat intake (% energy)	39.0 ± 8.2	37.6 ± 9.3†	37.9 ± 10.9‡	36.3 ± 9.8†
Cereal fiber (g/day)	12.2 ± 4.5	11.0 ± 4.1	8.7 ± 2.2†	7.4 ± 3.8†
Total crude fiber intake (g/day)	53.5 ± 1.5	55.2 ± 1.7	73.7 ± 2.0†	55.1 ± 1.8‡
Glycemic load	121 ± 35.3	132 ± 36.3†	124 ± 49.5	137 ± 45.2†
Dietary score§	6.2 ± 2.7	6.4 ± 0.3‡	6.2 ± 2.5	6.1 ± 3.7
Magnesium (mg/day)	295 ± 69.9	282 ± 69.0†	293 ± 71.5	271 ± 86.7†
Caffeine (mg/day)	395 ± 302	345 ± 11.5†	335 ± 379†	269 ± 336†
Total energy intake (kcal/day)	1,571 ± 492	1,617 ± 561‡	1,561 ± 698	1,578 ± 622

Data are means ± SD unless otherwise indicated. *Includes those with Southern European/Mediterranean ancestry, Scandinavian ancestry, and other Caucasian ancestry. ‡$P < 0.05$, †$P < 0.0001$ as compared with whites. §The dietary score is the sum of quintile values (0–4) for cereal fiber, ratio of polyunsaturated fat to saturated fat (ascending order), glycemic load, and *trans* fat (descending order). A higher score indicates a healthier diet.

between 1980 and 2000 (Table 3). Weight gain since age 18 years was the lowest among Asians (mean 5.5 kg) and the highest among blacks (mean 13.4 kg). In a multivariate model including BMI at age 18 years, weight gain during adulthood appeared to be most strongly associated with diabetes in Asians; for each 5-kg increment in weight gain since age 18 years, the RR of diabetes was 1.84 (1.58–2.14) for Asians, 1.44 (1.26–1.63) for Hispanics, 1.38 (1.28–1.49) for blacks, and 1.37 (1.35–1.38) for whites (all P values <0.05 comparing Asians with each of the other ethnic groups).

We attempted to examine the associations between other risk factors (dietary score, alcohol, and family history of diabetes) and risk of diabetes for each ethnic group. The directions of the associations between these variables and diabetes were generally similar for the minority groups, but the 95% CIs were wide because of the relatively small number of diabetes cases. Thus, we combined the three minority groups. In the combined analyses adjusting for age, BMI, ethnicity, and other covariates, a higher diet score (above median) was more strongly associated with a lower risk of diabetes among minorities (RR 0.54 [95% CI 0.39–0.73]) than among whites (0.77 [0.72–0.84]). Moderate alcohol intake (5 g or more per day) was equally beneficial among minorities (0.64 [0.40–1.02]) and whites (0.64 [0.58–0.70]). Family history of diabetes was associated with a higher risk of diabetes among both minorities (2.17 [1.66–2.84]) and whites (2.44 [2.29–2.61]).

Table 2—*RR (95% CI) of type 2 diabetes associated with ethnicity during 20 years of follow-up among 78,419 women*

	Ethnic group			
	White	Asian	Hispanic	Black
n	75,584	801	613	1,421
Person-years of follow-up	1,254,454	11,671	9,248	19,427
Incident cases of diabetes	3,624	49	48	123
Age adjusted	1.0 (ref.)	1.43 (1.08–1.90)	1.76 (1.32–2.34)	2.18 (1.82–2.61)
Age and BMI adjusted	1.0 (ref.)	2.26 (1.70–2.99)	1.86 (1.40–2.47)	1.34 (1.12–1.61)
Multivariate*	1.0 (ref.)	1.94 (1.46–2.58)	1.70 (1.28–2.26)	1.36 (1.14–1.63)
Additional adjustment for dietary score† and energy intake	1.0 (ref.)	1.99 (1.50–2.64)	1.73 (1.30–2.31)	1.38 (1.15–1.66)

*Adjusted for age (5-year categories), BMI (continuous), family history of diabetes, alcohol intake (none, 0.1–4.9, 5–14.9, and 15+ g/day), smoking status (never, past smoker, current 1–14, current 15–24, and current 25+ cigarettes/day), and moderate/vigorous exercise (0–0.9, 1–1.9, 2–3.9, 4–6.9, and 7+ h/week) (updated variables). †The dietary score is the sum of quintile values for cereal fiber, ratio of polyunsaturated fat to saturated fat (ascending order), glycemic load, and *trans* fat (descending order). A higher score indicates a healthier diet (updated variables).

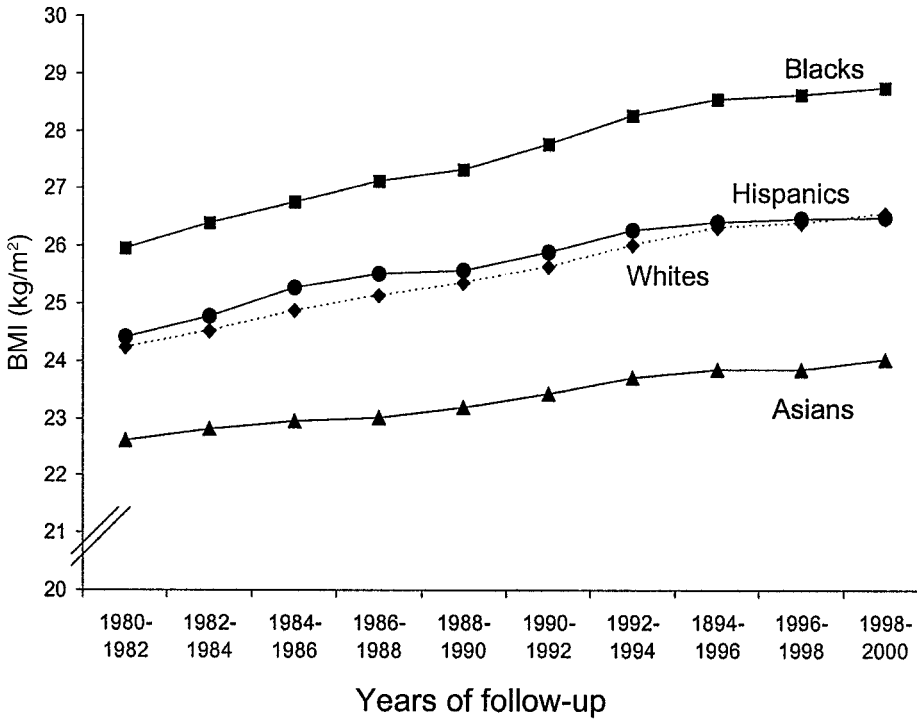

Figure 1—*Age-adjusted means of BMI during 20 years of follow-up among 78,419 women by ethnic group.*

CONCLUSIONS — In this 20-year prospective study, Asians, Hispanics, and blacks all had a significantly higher risk of type 2 diabetes than whites after controlling for BMI. Our data suggest that a diet high in cereal fiber and polyunsaturated fat and low in *trans* fat and glycemic load appears to have a stronger inverse association with diabetes risk among the minorities than among whites.

The strengths of our study include the large sample size, long follow-up, and detailed and repeated ascertainment of dietary and lifestyle measurements. However, some limitations of our study warrant consideration. We had no information about specific countries of origin, a variable that represents genetic and cultural factors within each ethnic group. We also had no information about length of residence in the U.S. A recent study showed that residency of immigrants in the U.S. for longer than 10 years was associated with increased obesity (17). However, our analyses took into account both baseline and current BMI levels in different ethnic groups. Even though we controlled for several major diabetes risk factors in the analyses, we cannot exclude the possibility of unmeasured confounders. The participants are predominantly white women, reflecting the ethnic background of women who trained as registered nurses through the 1960s. However, the cohort provides important data on diet and lifestyle across different ethnic groups. Finally, although this cohort is not a random sample of U.S. women, the relative socioeconomic homogeneity of the cohort can reduce the impact of unknown confounders.

In the Third National Health and Nutrition Examination Survey, the prevalence rates of diabetes for non-Hispanic blacks were 1.6 times the rate for non-Hispanic whites (3). In our cohort, the age-adjusted RR of type 2 diabetes among blacks was more than twice that in whites. However, the increased risk was attenuated after adjustment for BMI. In general, BMI was much higher among blacks than among whites, but its contribution to diabetes risk was lower among blacks. Stud-

Table 3—*RR (95% CI) of type 2 diabetes associated with a 5-unit increment in BMI during follow-up (1980–2000) and with a 5-kg increment in weight between age 18 years and the year 1980 among 78,419 women, stratified by ethnic group*

	Ethnic groups			
	White	Asian	Hispanic	Black
n	75,584	801	613	1,421
Person-years of follow-up	1,254,454	11,671	9,248	19,427
Incident cases of diabetes	3,624	49	48	123
Change of BMI between 1980–2000				
RR of BMI change (increments of 5 units, multivariate model)*	1.96 (1.93–2.00)[a]	2.36 (1.83–3.04)[a]	2.21 (1.75–2.79)[a]	1.55 (1.36–1.77)[b]
Change of weight between age 18 years and the year 1980				
Weight at age 18 years (kg)	57.3 ± 8.5	50.6 ± 7.4	53.4 ± 7.8	55.8 ± 8.2
BMI at age 18 years (kg/m²)	21.3 ± 2.9	20.3 ± 2.6	20.7 ± 2.7	20.8 ± 3.1
Weight change since age 18 years until 1980 (kg)	7.6 ± 9.9	5.5 ± 7.6	8.8 ± 9.0	13.4 ± 10.9
RR of weight change (increments of 5 units), multivariate model†	1.37 (1.35–1.38)[a]	1.84 (1.58–2.14)[b]	1.44 (1.26–1.63)[a]	1.38 (1.28–1.49)[a]

*Adjusted for age (5-year categories), family history of diabetes, alcohol intake (</≥5 g/day), smoking status (current/noncurrent), moderate/vigorous exercise (0, 1–2, and 3+ h/week), and dietary score (below/above median). †Adjusted for age (5-year categories), BMI at age 18 (continuous), family history of diabetes, alcohol intake (</≥5 g/day), smoking status (current/non-current), moderate/vigorous exercise (0, 1–2, and 3+ h/week), and dietary score (below/above median) (updated variables). [a, b]Different letters indicate significant differences between groups.

ies have demonstrated greater visceral adipose tissue in whites than in blacks despite the greater amount of total body fat in black women than in white women (18–19). However, black obese women demonstrate a higher degree of insulin resistance than white obese women despite less visceral fat (20).

Several studies have documented increased prevalence of diabetes among Asian-American immigrants (21,22). In contrast to white diabetic patients, whose BMIs are much higher than those of individuals without diabetes, the BMIs of both Japanese (23) and Thai (24) diabetic cohorts are often not in the obesity range as defined for Western populations. Our results suggest that the association between obesity and diabetes was more evident among Asian women than women in other ethnic groups. Previous studies have demonstrated a strong relationship between BMI and cardiovascular risk factors in Asian populations (25), although the range of BMI is at the lower end of the distribution for typical western populations. Recent studies suggest that lower cutoff BMI values for classifying overweight and obesity status are needed to identify Asians at a higher risk of cardiovascular disease and diabetes because Asians tend to have a higher percentage of body fat (26), particularly abdominal visceral fat (27), than whites with the same BMI.

The age-adjusted diabetes prevalence among U.S. Hispanics during 1998–2002 was approximately twice that among non-Hispanic whites (9.8 vs. 5.0%), as reported by the Centers for Disease Control and Prevention (28). Also, at each BMI level, the prevalence of type 2 diabetes among Hispanics was higher than among non-Hispanic whites (28). In the San Antonio Heart Study, the incidence of type 2 diabetes among Mexican Americans tripled during the 7–8 years of follow-up. The trend remained significant after adjusting for obesity and other risk factors (29). The RR of diabetes among Hispanics was not attenuated after controlling for BMI and several risk factors, suggesting that other factors may account for the increased risk of diabetes among Hispanics.

Our study suggests that ethnic differences in diabetes risk are only partially explained by differences in known diabetes risk factors. So far, the biological basis for the ethnic differences has not been fully elucidated. In metabolic studies, postprandial glucose was markedly higher in Asians than in whites (30). Specifically, among lean, healthy subjects matched for age, BMI, waist circumference, birth weight, and current diet, Asians (especially those of Southeast Asian descent) had significantly higher postprandial glycemia and lower insulin sensitivity than whites in response to a 75-g carbohydrate load. In several epidemiologic studies, Asians (31), blacks (31,32), and Mexican Americans (32) were found to be less insulin-sensitive than non-Hispanic whites after adjusting for obesity. These findings suggest that reduced insulin sensitivity may underlie the increased risk of type 2 diabetes in minorities in the U.S., especially among Asians. Because insulin sensitivity is determined partly by genetic factors, one could infer that the racial/ethnic differences in diabetes risk are attributed to genetic predisposition. However, adjustment for family history of diabetes did not appear to alter the risk estimates for different ethnic groups, and genetic factors alone would not explain the marked increase in prevalence of type 2 diabetes during the past decade. We speculate that the observed ethnic difference in diabetes risk is likely due to an interaction between diet and lifestyle and increased genetic susceptibility among minorities.

In our study, the diet score (representing a higher intake of fiber and polyunsaturated-to-saturated ratio and a lower glycemic load and intake of *trans* fat) was more strongly associated with diabetes risk among minorities than whites. These results suggest that dietary intervention may be particularly effective for diabetes prevention among minorities, although this hypothesis needs to be tested in future studies.

In conclusion, after accounting for BMI, our study indicates that Asians, Hispanics, and blacks were all at higher risk for type 2 diabetes than whites. The association between increasing BMI and greater weight gain and risk of diabetes was most pronounced among Asians, suggesting that lower cutoff BMI values are needed to identify Asians at a higher risk of diabetes.

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