Is the Association Between Dietary Glycemic Index and Type 2 Diabetes Modified by Waist Circumference?

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Data on dietary intake characterized by high glycemic index and glycemic load and development of type 2 diabetes have been inconsistent. A total of four studies have shown positive associations (1–4). One study showed consistent associations for both glycemic index and glycemic load (1), while in the other three, only glycemic index was predictive of diabetes (2–4). In contrast, the Atherosclerosis Risk in Communities (5) and Iowa Women's Health Study (6) showed no association between glycemic index and glycemic load with incidence of type 2 diabetes. Furthermore, studies focusing on precursors for diabetes are equally inconsistent, the majority not supporting a role of glycemic index in insulin resistance (7–9).

The aim of our study was to evaluate the impact of dietary glycemic index and glycemic load on risk of type 2 diabetes in the multiethnic Insulin Resistance Atherosclerosis Study (IRAS). Given our previous findings on abdominal adiposity predicting insulin sensitivity (10), which are key risk factors for the development of diabetes (11,12), we specifically focused on the role of glycemic index and glycemic load relative to abdominal obesity and waist change.

RESEARCH DESIGN AND METHODS

— Details of the IRAS study design have been published (13). More than 1,600 participants were recruited at four clinical centers between 1992 and 1994, aiming for equal representation across glucose tolerance status (normal, impaired glucose tolerance, and non–insulin-taking type 2 diabetes), ethnicity (African American, Hispanic, and non-Hispanic white), sex, and age (40–49, 50–59, and 60–69 years). The cohort was followed-up 5 years later.

At baseline, habitual dietary intake was assessed by using a 1-year, semiquantitative, 114-item food frequency interview (14). Details of the glycemic index and glycemic load estimation procedures in our study have been published (15). Glycemic index was assigned from published data (16) and other available resources (T. Wolever, personal communication) using the glucose = 100 scale (17,18) to food frequency questionnaire line items. Anthropometric measures were taken in a standardized manner according to the IRAS protocol.

At 5-year follow-up, individuals who met World Health Organization criteria for diabetes on their follow-up oral glucose tolerance test or who were taking hypoglycemic medication not previously reported at baseline were considered incident type 2 diabetic patients.

We included 892 participants who were free from type 2 diabetes at baseline, who returned for the follow-up examination, and had no missing data relevant to this analysis.

Multiple logistic regression analysis was used to assess the relationship between glycemic index and glycemic load and risk of type 2 diabetes. Parameter estimates and corresponding *P* values were computed for continuous variables and odds ratios (ORs) and 95% CIs for glycemic index/glycemic load tertiles. The models were stratified by abdominal obesity (19) (waist >102 cm [men] or >88 cm [women]) at baseline and change in waist (±2 cm, no change; −2 cm, decrease; and +2 cm = increase) during follow-up.

RESULTS

— At follow-up, 146 incident cases of type 2 diabetes were identified. Case subjects with diabetes were slightly older and had a higher BMI compared with nondiabetic case subjects. The average glycemic index and glycemic load of diabetic case subjects were 59.5 and 127.9, respectively, being similar to the values of nondiabetic case subjects (58.6 and 121.8, respectively). In multivariate regression models, glycemic index and glycemic load were not associated with risk of type 2 diabetes (glycemic index: $\beta = 0.0234$, $P = 0.2$; glycemic load: $\beta = -0.0018$, $P = 0.6$)

Results of the evaluation of the association between baseline glycemic index and glycemic load and risk of type 2 diabetes by abdominal obesity and waist change are shown in the table. Stratification by abdominal obesity status at baseline revealed a positive association between dietary glycemic index and risk of type 2 diabetes among nonabdominally obese subjects, whereas no association was detected among those with abdominal obesity. Furthermore, stratification by 5-year waist change demonstrated a positive association between glycemic index and diabetes risk among those who experienced an increase in waist size. This association was even stronger among nonabdominally obese subjects: diabetes risk was elevated by 12% (OR 1.12 [95% CI 1.03–1.21]) for a 1-unit increase in glycemic index among persons with waist increase and no abdominal obesity.

With regard to dietary glycemic load,

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Received for publication 9 January 2006 and accepted in revised form 23 January 2006.

Abbreviations: IRAS, Insulin Resistance Atherosclerosis Study.

A table elsewhere in this issue shows conventional and Système International (SI) units and conversion factors for many substances.

DOI: 10.2337/dc06-0056

Table 1—*Multivariable-adjusted risk estimates for the association between baseline dietary glycemic index and glycemic load and the risk of developing type 2 diabetes, stratified by abdominal obesity at baseline and change in waist during follow-up, IRAS I and II,* n = *892*

	Cases	β (*P* value)	OR (95% CI) 1st tertile	2nd tertile	3rd tertile	*P* value
Glycemic index						
Abdominal obesity*†						
Yes	74	−0.0035 (0.90)	1.00	0.82 (0.39–1.72)	0.84 (0.40–1.79)	0.65
No	72	0.0517 (0.06)	1.00	1.42 (0.68–2.97)	1.90 (0.89–4.00)	0.10
Change in waist‡§						
Decrease	23	0.0404 (0.40)	1.00	—‖	—‖	
Stable	36	−0.0678 (0.14)	1.00	0.85 (0.30–2.42)	0.49 (0.14–1.66)	0.25
Increase	87	0.0571 (0.04)	1.00	1.32 (0.65–2.65)	1.70 (0.84–3.47)	0.14
Glycemic load						
Abdominal obesity*†						
Yes	74	−0.0017 (0.74)	1.00	1.20 (0.58–2.48)	0.82 (0.39–1.75)	0.89
No	72	−0.0019 (0.70)	1.00	1.31 (0.67–2.57)	1.14 (0.57–2.26)	0.66
Change in waist‡§						
Decrease	23	−0.0007 (0.94)	1.00	—‖	—‖	
Stable	36	−0.0131 (0.10)	1.00	—‖	—‖	
Increase	87	−0.0006 (0.90)	1.00	0.81 (0.43–1.53)	0.76 (0.39–1.49)	0.42

*Waist circumference >102 cm for men and >88 cm for women. †Adjusted for age, ethnicity/clinic, baseline BMI, family history of diabetes, smoking status, glucose tolerance status, education, and energy intake (by residual method). ‡Decrease: change in waist ≤−2 cm; stable: change in waist ±2 cm; increase: change in waist ≥2 cm. §Adjusted for age, ethnicity/clinic, baseline BMI, baseline waist circumference, family history of diabetes, smoking status, glucose tolerance status, education, and energy intake (by residual method). ‖Tertile-specific ORs were not estimated if the reference category comprised <10 subjects.

stratification by abdominal obesity or waist change did not reveal any association with type 2 diabetes as did the association with intake of digestible carbohydrates (total carbohydrates minus fiber).

CONCLUSIONS — Previous work in IRAS (10) demonstrating a significant association between waist circumference and change in insulin sensitivity (S_I) confined to nonobese individuals (BMI < 30 kg/m^2) prompted us to evaluate whether waist circumference may modify the association of behavioral factors, such as dietary glycemic index and glycemic load, on the risk of type 2 diabetes. To the best of our knowledge, our study is the first to reveal an association between glycemic index (but not glycemic load) and type 2 diabetes, which was modified by waist circumference, i.e., dietary glycemic index increased the risk of type 2 diabetes among nonabdominally obese subjects and among subjects experiencing increases in waist circumference. The previous positive studies (1–4) controlled their analyses for BMI and waist-to-hip ratio, which impacted the risk estimates in only one study; there, a significant association between glycemic index and diabetes risk was confined to obese subjects (4).

It needs to be mentioned that the IRAS study population differs from other study populations in terms of prevalence of overall and abdominal obesity. Given the sampling design of IRAS (13) (1/3 of the population having impaired glucose tolerance), our cohort is much more overweight (mean BMI 28.4 kg/m^2) than the U.S.-American cohorts (1,2) and has a higher mean waist-to-hip ratio (0.86) than the Melbourne Collaborative cohort (0.83) (4). This may have impacted the chance of detecting an association modified by abdominal obesity and change in waist.

The major limitation of the present study is its small sample size and number of incident cases. This may, at least in part, explain our nonsignificant risk estimates and the fact that formal tests of interaction failed to reject homogeneity of risk across strata of the modifying variables. However, the trend analysis indicated a monotonically increasing relationship between glycemic index and diabetes risk, albeit nonsignificant.

In conclusion, the data of the present study suggest effect modification of the glycemic index–diabetes association by waist circumference, in that a high–glycemic index diet predicts type 2 diabetes risks among nonabdominally obese individuals and individuals with increases in waist but not among abdominally obese individuals. This needs to be confirmed in large-scale prospective studies. No association was apparent for dietary glycemic load or carbohydrate intake.

Acknowledgments— This study was supported by an American Diabetes Association Clinical Research Award to Dr. Liese. The IRAS study was supported by National Institutes of Health/National Heart, Lung, and Blood Institute Grants UO1 HL/17887, UO1 HL/17889, UO1 HL/17890, UO1 HL/17892, UO1 HL/17902, and DK29867.

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