

Glycemic Index and Dietary Fiber and the Risk of Type 2 Diabetes

ALLISON M. HODGE, MENVSC¹
DALLAS R. ENGLISH, PHD¹

KERIN O'DEA, PHD²
GRAHAM G. GILES, PHD¹

OBJECTIVE— To examine associations between type 2 diabetes and fiber, glycemic load (GL), dietary glycemic index (GI), and fiber-rich foods.

RESEARCH DESIGN AND METHODS— This was a prospective study of 36,787 men and women aged 40–69 years without diabetes. For all self-reported cases of diabetes at 4-year follow-up, confirmation of diagnosis was sought from medical practitioners. Case subjects were those who reported diabetes at follow-up and for whom there was no evidence that they did not have type 2 diabetes. Data were analyzed with logistic regression, adjusting for country of birth, physical activity, family history of diabetes, alcohol and energy intake, education, 5-year weight change, sex, and age.

RESULTS— Follow-up was completed by 31,641 (86%) participants, and 365 cases were identified. The odds ratio (OR) for the highest quartile of white bread intake compared with the lowest was 1.37 (95% CI 1.04–1.81; *P* for trend = 0.001). Intakes of carbohydrate (OR per 200 g/day 0.58, 0.36–0.95), sugars (OR per 100 g/day 0.61, 0.47–0.79), and magnesium (OR per 500 mg/day 0.62, 0.43–0.90) were inversely associated with incidence of diabetes, whereas intake of starch (OR per 100 g/day 1.47, 1.06–2.05) and dietary GI (OR per 10 units 1.32, 1.05–1.66) were positively associated with diabetes. These relationships were attenuated after adjustment for BMI and waist-to-hip ratio.

CONCLUSIONS— Reducing dietary GI while maintaining a high carbohydrate intake may reduce the risk of type 2 diabetes. One way to achieve this would be to substitute white bread with low-GI breads.

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The prevalence of type 2 diabetes is increasing rapidly worldwide, hence the need for widely applicable strategies to reduce incidence (1). Intensive interventions focusing on diet, physical activity, and weight loss have reduced or delayed the incidence of type 2 diabetes in high-risk individuals (2–5). Such interventions are not feasible at the population level, but the dramatic change from butter to margarine in Australia during the 1970s (6) suggests that relatively

small changes from one version of a food to another may be achievable.

Current Australian dietary recommendations promote the consumption of cereal products (7); however, some studies suggest that refined or high-glycemic index (GI) cereal products may increase the risk of diabetes (8–10), and benefits from cereal products may relate only to whole grains or cereal fiber (8,9,11–13). It is important to clarify the associations between cereals, GI, glycemic load (GL),

and fiber and type 2 diabetes so that, if necessary, recommendations can be made more specific.

Our aims were, therefore, to examine the associations between type 2 diabetes and fiber, GL, GI, and fiber-rich foods in a prospective study in Melbourne, Australia.

RESEARCH DESIGN AND METHODS

The Melbourne Collaborative Cohort Study recruited 41,528 people (17,049 men) between 1990 and 1994. The subjects' age range was 27–75 years at baseline (99.3% were 40–69 years of age). The study included 5,425 migrants from Italy and 4,535 from Greece or Macedonia. The Cancer Council Victoria's Human Research Ethics Committee approved the study protocol. Subjects gave written consent to participate and to obtain access to their medical records.

Before analyses of diabetes incidence, we excluded people with diabetes at baseline (self-reported, *n* = 1,549, or elevated plasma glucose, *n* = 324), those who reported having angina or suffering a heart attack before baseline, those who did not report diabetes at baseline but later reported a date of diabetes diagnosis before baseline, those with energy intakes in the top or bottom 1% of the sex-specific distributions, and those with missing values for relevant risk factors measured at baseline. After these exclusions, 36,787 subjects remained available for analysis.

Baseline glucose measurement

Plasma glucose was measured using a Kodak Ektachem analyzer (Rochester, NY). For the 68% who were fasting, plasma glucose values ≥ 7.8 mmol/l were considered diabetic, and for those who were not fasting, diabetes was defined as ≥ 11.1 mmol/l, according to the World Health Organization criteria current at the time (14).

Dietary assessment

Dietary information was collected using a 121-item, self-administered, food frequency questionnaire (FFQ) that was specifically developed for the Melbourne

From the ¹Cancer Epidemiology Centre, The Cancer Council Victoria, Melbourne, Australia; the ²Menzies School of Health Research, and Institute of Advanced Studies, Charles Darwin University, Darwin, Australia.

Address correspondence and reprint requests to Allison M. Hodge, The Cancer Council, Victoria, 1 Rathdowne St., Carlton, Victoria 3053, Australia. E-mail: allison.hodge@cancervic.org.au.

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Abbreviations: FFQ, food frequency questionnaire; GI, glycemic index; GL, glycemic load; WHR, waist-to-hip ratio.

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

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Collaborative Cohort Study (15). To calculate nutrient intakes, sex-specific standard portions were used, together with Australian food-composition data (16). GI is a method of ranking foods on the basis of the blood glucose response to a given amount of carbohydrate from that food. GI values of individual food items were obtained from the 2002 international table of GI and GL values (17). Where there was more than one value, GI values were averaged, with preference given to Australian figures. The GI values used for the 17 foods making up the category "cereal foods, cakes and biscuits" on the FFQ were "wheatgerm" (41), "muesli" (46), "other breakfast cereal" (62), "rice, boiled (including brown rice)" (65), "fried rice" (48), "mixed dishes with rice" (48), "white bread, rolls, or toast" (75), "whole-wheat or rye bread, rolls, or toast" (54), "fruit bread" (48), "crackers or crisp-breads" (67), "sweet biscuits" (63), "cakes or sweet pastries" (64), "puddings" (39), "pasta or noodles" (48), "pizza" (38), "dim sims or spring rolls" (45), and "pies or savory pastries" (45). Dietary GL was computed by summing the product of carbohydrate intake from each food by the GI for that food. GL was divided by total carbohydrate intake to obtain dietary GI, i.e., an average of individual food GI values, weighted according to their contribution to carbohydrate intake (17). Alcoholic beverages were not included in the overall GI.

Information was not sought separately for whole-grain and other cereal foods. It was assumed that pasta was not whole grain and that breakfast cereals were predominantly whole-grain or high-fiber types. Within the study of weighed food records on which the FFQ was based, muesli, bran-based cereals, and porridge made up 85 and 76% of total breakfast cereals by energy in men and women, respectively (18). "White" and "whole-wheat or rye breads" were separately identified on the FFQ. Details of which foods were included in each food group are listed in the online appendix (available from <http://care.diabetesjournals.org>).

Measurement of nondietary risk factors

A structured interview schedule was used to obtain information on potential risk factors including country of birth, smoking, alcohol consumption, physical activity (walking, vigorous, and less-vigorous

exercise), education, weight change over the last 5 years, and history of diabetes in first-degree relatives. Standard methods were used to measure height, weight, and waist and hip circumferences, from which BMI (in kilograms per meters squared) and waist-to-hip ratio (WHR) were calculated.

Follow-up and ascertainment of diabetes status

Incident cases of diabetes were identified from a self-administered questionnaire mailed to participants ~4 years after baseline. Participants were asked: "Has a doctor ever told you that you have had diabetes?" Those who responded in the affirmative were asked to provide the year of diagnosis. For all self-reported incident case subjects, except those who reported a diagnosis date before baseline and who were excluded, confirmation of diagnosis was sought from doctors nominated by participants. Doctors were asked to specify if the participant had diabetes and, if so, to indicate whether it was type 1 or 2.

Reproducibility study

From July 1992 to June 1993, 275 subjects were invited to participate in a further study that involved completing a second FFQ 12 months after baseline. Selection was stratified by sex, country of birth (i.e., Australia, Italy, and Greece), 10-year age-group, and month of attendance.

Statistical analysis

Age, country of birth, sex, physical activity score, weight change, education level, alcohol intake, and family history of diabetes, but not smoking status, were associated with type 2 diabetes and were included as covariates in all analyses to avoid confounding. Dietary variables were tested in logistic regression models including these covariates and energy intake, both with and without BMI and WHR, which were considered possible intermediates in the causal pathway. Because the incidence of diabetes was low, the odds ratios (ORs) from the models are approximately equal to cumulative incidence ratios. Nutrients, GI, and GL were analyzed as continuous variables; the ORs presented correspond approximately to the difference between the 87.5th and the 12.5th percentiles, i.e., the approximate medians within the highest and lowest quartiles. Food groups were analyzed as approximate quarters of consumption,

except fruit and vegetables, which were classified according to recommended daily intakes in Australia. Tests for trend were performed using the medians in each group.

Interactions between carbohydrate intake and GI and between cereal fiber intake and GI and GL were evaluated. Previous studies (19) of GI and other outcomes have suggested that the associations may be stronger in obese or inactive people, who are likely to be insulin resistant. Hence interactions between GI and BMI (underweight or normal, overweight, or obese), physical activity (inactive or active), and glucose tolerance (normal or impaired) were examined. Interactions were evaluated by including interaction terms in the models with both main-effects terms and calculating *P* values using the likelihood ratio test. The analyses were repeated in Australian- and New Zealand-born participants only to ensure that residual confounding by country of birth would not have influenced our results. We also repeated the analyses using only the 303 confirmed cases of type 2 diabetes.

RESULTS— A total of 31,641 (86% of eligible participants) completed the follow-up question on diabetes. People who completed follow-up had similar levels of risk factors for type 2 diabetes compared with those who did not complete the questionnaire (mean age 54.3 vs. 54.5 years and mean fasting plasma glucose 5.5 vs. 5.5 mmol/l, respectively), although BMI was slightly lower (mean 26.6 vs. 27.2 kg/m², respectively). Eighty-seven percent of women and 85% of men completed follow-up. Greek-born participants (80%) were slightly less likely to complete follow-up than those born elsewhere (86–87%).

Of those who completed follow-up, 459 reported a diagnosis of diabetes after baseline and attempts were made to verify this diagnosis with the participant's doctor. Of 399 people for whom a response was obtained, 303 (76%) were confirmed as having type 2 diabetes. Because the proportion confirmed was high, the two people for whom the doctor did not know diabetes type or status were considered to be case subjects, as were the 60 people for whom no response was available. Participants whose doctors reported that they had type 1 diabetes (*n* = 11), had impaired glucose tolerance (*n* = 1), or did

Table 1—Risk factor levels by quartiles of GI

	GI quartile groups			
	20.8–46.0	46.1–48.6	48.7–51.5	51.6–67.7
Medians within GI quartiles				
Age (years)	54	54	55	55
BMI (kg/m ²)	26.0	25.8	26.1	26.6
WHR	0.82	0.82	0.83	0.84
Carbohydrate (g/day)	236.7	226.4	229.2	238.4
Sugars (g/day)	127.9	113.8	109.3	103.0
Starch (g/day)	104.9	109.5	115.4	130.9
Fiber (g/day)	30.8	31.0	29.2	26.3
Magnesium (g/day)	473.3	390.0	362.0	323.0
GL	104.9	108.0	115.3	131.1
Cereal fiber (g/day)	8.9	10.4	10.6	10.4
Fruit fiber (g/day)	7.5	7.5	6.5	5.1
Vegetable fiber (g/day)	5.4	5.0	4.4	3.6
Legume fiber (g/day)	1.5	1.5	1.4	1.3
Potato fiber (g/day)	0.6	0.7	0.8	0.8
Energy (kcal/day)	2,204	2,079	2,060	2,107
Cereal (times/week)	22.5	28.0	31.5	37.0
Breakfast cereal (times/week)	3.5	5.5	5.5	3.0
Rice (times/week)	1.5	1.5	1.5	1.0
Bread (times/week)	7.0	7.5	8.5	17.5
White bread (times/week)	0.5	1.0	3.0	17.5
Whole-meal bread (times/week)	5.5	7	5.5	0.5
Savory cereal (times/week)	0.5	0.5	0.5	0.5
Pasta (times/week)	1.0	1.0	1.0	1.0
Other cereal (times/week)	4.0	6.5	8.0	9.0
Vegetables (times/day)*	6.0	5.0	5.0	4.0
Potato (times/week)	3.0	3.5	4.0	4.0
Legumes (times/week)	3.0	3.0	3.0	3.0
Fruit (times/day)*	4.0	4.0	3.0	3.0
Alcohol (g/day)	7.9	5.0	4.3	2.0
Percentage distribution†				
Born in Australia	25.2	25.5	25.7	23.6
Born in U.K./Malta	27.1	26.8	24.8	21.2
Born in Greece/Macedonia	28.8	25.5	21.7	24.0
Born in Italy	19.4	20.5	23.9	36.2
Active‡	29.0	27.7	24.3	19.0
Family history of diabetes	24.2	23.0	25.8	27.0
Increased weight in the last 5 years	24.2	23.5	25.7	26.6
Did not go past primary school	23.5	22.0	23.6	30.9

*Vegetables and fruit are in times per day rather than per week in order to correspond to Australian dietary recommendations; †percentage of the group falling within each GI quartile; ‡active is defined as the most active of four physical activity groups, based on frequency of walking, less vigorous activity, and twice the frequency of vigorous activity.

not have diabetes ($n = 82$) were classified as noncase subjects, along with those who did not report diabetes at follow-up. After all exclusions, 365 case and 31,276 non-case subjects were identified as eligible for analysis.

Table 1 shows associations between dietary GI and dietary and other characteristics. The most consistent patterns among nutrients were observed for sug-

ars, fiber, and magnesium, which decreased with increasing GI, whereas starch and GL were positively associated with GI, although GI was not strongly correlated with any of these (absolute correlations between 0.14 and 0.35). Australian-, U.K.-, and Greek-born participants were slightly less likely to be in the top category of GI, whereas Italian-born participants were almost twice as likely to be

in the top category of GI as in the lowest category. High intakes of total cereal foods, bread, white bread, and “other cereal,” which includes mostly sweet cereal products, were associated with higher GI. High intakes of fruit and vegetables were associated with lower GI. Physical activity and alcohol intake were inversely associated with GI. The cumulative incidence of type 2 diabetes across GI quartiles was 0.9, 1.0, 1.2, and 1.6%, respectively.

Bread intake was weakly positively associated with diabetes risk (Table 2). White bread showed a J-shaped relationship with risk of diabetes, which was highest in the top quartile of intake. Intake of savory cereal products was positively associated with diabetes risk, whereas the “other cereal” group was inversely associated.

Intakes of total carbohydrate, sugars, and magnesium were inversely associated with diabetes incidence, and starch was positively associated (Table 3). Dietary GI was positively associated with diabetes. When BMI and WHR were included in the model, associations with bread, savory cereal products, “other” cereal products, total carbohydrate, sugars, magnesium, and dietary GI were attenuated. GL showed little association with diabetes. Neither total fiber nor fiber from different sources was associated with diabetes. Results were not materially altered if analyses were limited to the 303 confirmed case subjects with type 2 diabetes.

There was little evidence that the association between risk of diabetes and GI was modified by carbohydrate intake (interaction, $P = 0.6$), cereal fiber (interaction, $P = 0.5$), or glucose tolerance at baseline (interaction, $P = 0.4$). The association between diabetes and GL was not modified by cereal fiber (interaction, $P = 0.6$). In people with BMI <25 kg/m² (case subjects, $n = 20$), GI was inversely associated with diabetes (OR 0.29, 95% CI 0.10–0.91). In people with $25 \leq$ BMI <30 kg/m² (case subjects, $n = 134$), GI was not associated with diabetes (1.00, 0.68–1.46), whereas in those with BMI ≥ 30 kg/m² (case subjects, $n = 211$), a positive association (1.64, 1.22–2.21) was observed (interaction, $P = 0.01$). The association of GI with type 2 diabetes was somewhat stronger in people who reported doing physical activity (case subjects, $n = 241$) than those who reported none (case subjects, $n = 124$), although

Table 2—ORs and CIs for quartiles of intake of food groups and tests for trend across quartiles

Food group*	Multivariate adjusted†					Further adjusted for BMI and WHR				
	Quartiles of intake					Quartiles of intake				
	2	3	4	Increase of one time/week	P trend	2	3	4	Increase of one time/week	P trend
Cereal	0.92 (0.68–1.24)	0.86 (0.63–1.19)	0.95 (0.66–1.36)	1.00 (0.99–1.01)	0.8	0.93 (0.69–1.26)	0.97 (0.70–1.35)	1.05 (0.73–1.52)	1.00 (0.99–1.01)	0.7
Breakfast cereal	1.11 (0.81–1.50)	0.77 (0.56–1.07)	0.81 (0.61–1.08)	0.96 (0.93–1.00)	0.05	1.14 (0.84–1.56)	0.80 (0.58–1.12)	1.01 (0.75–1.35)	0.99 (0.95–1.03)	0.6
Rice	0.74 (0.53–1.02)	0.86 (0.64–1.15)	0.89 (0.66–1.21)	1.00 (0.92–1.08)	0.99	0.77 (0.56–1.07)	0.91 (0.67–1.22)	0.93 (0.68–1.27)	1.01 (0.93–1.09)	0.9
Bread	0.83 (0.59–1.16)	1.13 (0.83–1.54)	1.17 (0.83–1.65)	1.02 (1.00–1.03)	0.05	0.81 (0.57–1.14)	1.06 (0.77–1.44)	1.12 (0.79–1.58)	1.02 (1.00–1.03)	0.1
White bread	0.69 (0.47–1.04)	1.08 (0.77–1.52)	1.37 (1.04–1.81)	1.18 (1.07–1.29)	0.001	0.66 (0.44–0.99)	0.95 (0.67–1.35)	1.13 (0.86–1.50)	1.11 (1.02–1.22)	0.04
Whole-meal bread	0.71 (0.53–0.94)	0.76 (0.57–1.01)	0.75 (0.55–1.02)	0.94 (0.83–1.05)	0.3	0.72 (0.54–0.97)	0.82 (0.61–1.09)	0.86 (0.63–1.18)	0.99 (0.88–1.11)	0.8
Savory cereal products	1.00 (0.76–1.33)	0.98 (0.72–1.35)	1.43 (1.04–1.96)	1.23 (1.00–1.51)	0.05	0.95 (0.71–1.26)	0.95 (0.69–1.30)	1.22 (0.89–1.69)	1.12 (0.91–1.38)	0.3
Pasta	0.84 (0.60–1.19)	0.77 (0.55–1.07)	0.84 (0.59–1.19)	0.88 (0.42–1.84)	0.7	0.89 (0.63–1.27)	0.84 (0.60–1.18)	0.86 (0.60–1.23)	0.83 (0.39–1.77)	0.6
Other cereal	0.86 (0.64–1.15)	0.73 (0.54–1.00)	0.64 (0.46–0.90)	0.98 (0.96–1.00)	0.01	0.93 (0.70–1.25)	0.82 (0.60–1.12)	0.79 (0.56–1.10)	0.99 (0.97–1.01)	0.2
Vegetables	1.00 (0.71–1.40)	0.92 (0.65–1.31)	0.86 (0.59–1.24)	0.97 (0.92–1.03)	0.3	1.09 (0.78–1.54)	0.97 (0.68–1.39)	0.88 (0.60–1.28)	0.97 (0.91–1.03)	0.3
Potatoes	0.80 (0.60–1.06)	0.79 (0.58–1.07)	0.90 (0.64–1.25)	0.98 (0.93–1.03)	0.4	0.84 (0.63–1.12)	0.82 (0.60–1.12)	0.98 (0.70–1.37)	0.99 (0.94–1.04)	0.7
Legumes	1.05 (0.80–1.39)	0.78 (0.55–1.12)	1.10 (0.82–1.47)	1.01 (0.96–1.06)	0.6	1.08 (0.81–1.43)	0.81 (0.56–1.16)	1.09 (0.81–1.47)	1.01 (0.96–1.06)	0.7
Fruit	0.72 (0.52–0.98)	0.70 (0.50–0.98)	0.75 (0.53–1.07)	0.97 (0.92–1.03)	0.3	0.81 (0.59–1.12)	0.82 (0.58–1.16)	0.85 (0.59–1.22)	0.99 (0.93–1.04)	0.6

Data are OR (95% CI). *Cut points for food group quartiles 1–4, respectively, were the following: total cereal: <20, 20–28, 29–40, and ≥41 times/week; breakfast cereal: <0.01, 0.01–2.9, 3.0–6.9, and ≥7.0 times/week; rice: <1.0, 1.0–1.4, 1.5–2.4, and ≥2.5 times/week; bread: <6.0, 6.0–7.9, 8.0–18.0, and ≥18.0 times/week; white bread: <0.5, 0.5–2.9, 3.0–6.9, and ≥7.0 times/week; whole-meal bread: <0.5, 0.5–1.4, 1.5–17.4, and ≥17.5 times/week; other cereal: <2.0, 2.0–5.9, 6.0–11.0, and ≥11.0 times/week; savory cereal products: <0.5, 0.5–0.9, 1.0–1.4, and ≥1.5 times/week; pasta: <0.5, 0.5–0.9, 1.0–2.9, and ≥3.0 times/week; vegetables: <3.0, 3.0–4.9, 5.0–6.9, and ≥7.0 times/day; potatoes: <2.0, 2.0–3.9, 4.0–6.4, and ≥6.5 times/week; legumes: <2.0, 2.0–3.4, 3.5–4.9, and ≥5.0 times/week; and fruit: <2.0, 2.0–3.9, 4.0–5.9, and ≥6.0 times/day. †Adjusted for age, sex, country of birth, physical activity, family history of diabetes, alcohol intake, education level, weight change in the last 5 years, and energy intake.

the interaction had a large *P* value (interaction, *P* = 0.2).

In contrast to the observations of Salmeron et al. (8,9), adjustment for cereal fiber did not strengthen the association of GL with diabetes (OR 0.84, 95% CI 0.54–1.33).

Results for Australian- and New Zealand-born participants did not differ substantially from results in the whole cohort. For example, without adjusting for BMI and WHR, the OR in the top quartile of white bread relative to the bottom quartile was 1.34 (95% CI 0.91–1.96; *P* trend = 0.006), and the OR for a 10-unit increase in GI was 1.39 (0.98–1.97). Vegetable fiber appeared to be associated with increased risk of diabetes (1.24, 1.03–1.48, per 5 g/day) in this subgroup.

Two hundred forty-two people completed the reproducibility study. The weighted κ -statistics for the reproducibility of food group quartiles ranged from OR 0.26 (95% CI 0.14–0.38) for bread to 0.77 (0.65–0.90) for pasta. The intraclass correlations (the proportion of the total variance due to the between-subject component) for nutrients including GL and GI ranged from 0.27 (0.15–0.38) for fiber from potatoes to 0.49 (0.39–0.58) for GI.

CONCLUSIONS— High dietary GI and intake of white bread and starch were associated with increased risk of type 2 diabetes, while risk was lower with higher intake of sugars, magnesium, and total carbohydrate. These associations were weakened after adjustment for measures of obesity. Intake of fiber was not associated with diabetes.

The high response rate in our study and the small differences between respondents and nonrespondents should minimize response bias. Compared with other studies of diabetes incidence (8–11,19), our study had the advantage of measuring blood glucose at baseline, so the 324 people with diabetic plasma glucose levels could be excluded.

At follow-up, type 2 diabetes was confirmed in 76% of self-reported cases. This compares favorably with the results from the Iowa Women's Health Study (11), where in a substudy, diabetes was confirmed in only 64% of self-reported cases. Excluding the unconfirmed cases did not change our conclusions. Some incident cases would have been missed because we did not screen participants at follow-up, but this would not affect the

Table 3—Association of nutrients and type 2 diabetes*

Nutrient	Multivariate adjusted†		Further adjusted for BMI and WHR	
	OR (95% CI)	P	OR (95% CI)	P
Carbohydrate (200 g/day)	0.58 (0.36–0.95)	0.03	0.84 (0.51–1.39)	0.50
Sugars (100 g/day)	0.61 (0.47–0.79)	<0.001	0.72 (0.56–0.93)	0.01
Starch (100 g/day)	1.47 (1.06–2.05)	0.02	1.52 (1.09–2.11)	0.01
Fiber (20 g/day)	0.93 (0.73–1.18)	0.53	1.02 (0.81–1.30)	0.85
Magnesium (500 mg/day)	0.62 (0.43–0.90)	0.01	0.73 (0.51–1.04)	0.07
Cereal fiber (10 g/day)	0.97 (0.79–1.20)	0.79	1.08 (0.88–1.32)	0.46
Fruit fiber (10 g/day)	0.93 (0.77–1.11)	0.40	0.97 (0.81–1.16)	0.71
Vegetable fiber (5 g/day)	1.01 (0.87–1.18)	0.89	1.00 (0.86–1.17)	0.96
Potato fiber (g/day)	1.04 (0.92–1.17)	0.57	1.03 (0.91–1.16)	0.65
Legume fiber (g/day)	1.01 (0.97–1.06)	0.62	1.01 (0.96–1.06)	0.67
GI (10 units)	1.32 (1.05–1.67)	0.02	1.23 (0.98–1.54)	0.08
GL (100 units/day)	0.85 (0.56–1.29)	0.45	1.04 (0.68–1.58)	0.85

*Variables are scaled so that the ORs correspond approximately to the difference between the 87.5th and 12.5th percentiles, i.e., the approximate medians within the highest and lowest quarters; †adjusted for age, sex, country of birth, physical activity, family history of diabetes, alcohol intake, education level, weight change in the last 5 years, and energy intake.

results if underascertainment was not associated with the exposure (20).

Random error in measuring intake and dietary change during follow-up is likely to have attenuated the associations. Our measurement of diet was based on a single FFQ administered at baseline that may not have been representative of consumption over the longer term. In a subset of 242 participants, the FFQ showed only fair-to-moderate agreement when administered on two occasions 12 months apart. An important limitation of the study was the lack of detail regarding some of the foods, particularly breakfast cereals, which necessitated making assumptions in the calculation of fiber and GI. The lack of information on type of pasta and rice may be less important because whole-grain versions are rarely used compared with refined versions according to our analysis of 24-h recall data from the 1995 National Nutrition Survey (21).

Our finding of a positive association between GI and diabetes is consistent with the Nurses' Health Study and the Health Professionals' Follow-up Study, although in both of those studies, the association was not attenuated after including BMI (8,9). Two other large cohort studies (11,12) did not find any association between GI and diabetes incidence. In the studies in which GI was associated with diabetes, the association was strengthened by adjustment for cereal fiber. We found no evidence of this.

The association of GI with diabetes was weaker after adjustment for BMI and WHR. Because high-GI diets may promote weight gain (22), it is possible that this is one of the ways in which a high dietary GI increases the risk of diabetes, although no association between BMI and GI was observed within the cohort. Two other mechanisms have been suggested, whereby intake of high-GI carbohydrates may increase the risk of type 2 diabetes. By overstimulating insulin secretion, a high-GI diet or high GL could contribute to pancreatic β -cell dysfunction. A high GL or high-GI diet may also lead to insulin resistance (23). GL might be expected to have a greater effect than GI on both of these mechanisms. However, GI and not GL was the stronger predictor of type 2 diabetes.

Because GL is the product of carbohydrate intake and GI, a relatively high GL could result from a relatively low-carbohydrate, high-GI diet, which would increase diabetes risk according to our results, or a relatively high-carbohydrate, low-GI diet, which would reduce risk. Thus dietary patterns with the same GL may not be equivalent (24).

There is some evidence that GI is important only in combination with a degree of insulin resistance as shown by obesity or inactivity (19). We observed the adverse association of GI with type 2 diabetes only in participants with BMI ≥ 30 kg/m². In contrast, GI was more important

for active people, and we did not observe an interaction of GI with glucose tolerance at baseline, i.e., normal versus impaired, although people with impaired glucose tolerance would be likely to be more insulin resistant. Thus, of three relatively insulin-resistant states, we only observed a stronger association between GI and diabetes in one.

The inverse association with diabetes for sugars and some, mostly sweet, high-fat, cereal-based foods is not consistent with animal studies (25) in which high intakes of sucrose or fructose lead to insulin insensitivity. However, the evidence in humans is inconsistent (25). Among participants in the Iowa Women's Study, sucrose was inversely associated with diabetes incidence, whereas fructose was positively associated (11), and intake of sugars was not associated with type 2 diabetes incidence in the Women's Health Study (26). Sugars in the current analysis include sugars from fruit, and these may have a different effect from sugars consumed as added sugar.

Two recent prospective studies (27,28) provide evidence for the benefits of magnesium in reducing diabetes risk, as we observed. Breads were the single most important source of magnesium in the National Nutrition Survey (21), and whole-meal bread has roughly twice the magnesium of white bread (16). Including whole-grain products and other minimally refined plant foods in the diet will increase magnesium intake (27) as well as reduce GI.

Our data suggest that a diet with high carbohydrate content and a low GI may reduce the risk of type 2 diabetes. White bread was the food most strongly related to diabetes incidence and was also the most strongly associated with GI. Thus, the simple change from white bread to lower-GI bread within a high-carbohydrate diet could reduce the risk of diabetes. Changing bread type may be a more acceptable dietary change than one requiring a whole new eating pattern. Reducing dietary GI may also help with weight reduction.

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References

1. Shaw JE, Chisholm DJ: Epidemiology and prevention of type 2 diabetes and the metabolic syndrome. *Med J Aust* 179:379–383, 2003
2. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT, Hamalainen H, Ilanne-Parikka P, Keinanen-Kiukaanniemi S, Laakso M, Louheranta A, Rastas M, Salminen V, Uusitupa M: Prevention of type 2 diabetes mellitus by change in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med* 344:1343–1350, 2001
3. Eriksson K-F, Lindgärde F: Prevention of type 2 (non-insulin-dependent) diabetes mellitus by diet and physical exercise: the 6-year Malmö Feasibility Study. *Diabetologia* 34:891–898, 1991
4. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, Hu ZX, Lin J, Xiao JZ, Cao HB, Liu PA, Jiang XG, Jiang YY, Wang JP, Zheng H, Zhang H, Bennett PH, Howard BV: Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance: the Da Qing IGT and Diabetes Study. *Diabetes Care* 20: 537–544, 1997
5. Diabetes Prevention Program Research Group: Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med* 346:393–403, 2002
6. Australian Bureau of Statistics: *Year Book Australia 2002: Agriculture: Apparent Consumption of Foodstuffs*. Canberra, Australia, Australian Bureau of Statistics, 2002
7. National Health and Medical Research Council: *Food for Health: Dietary Guidelines for Australian Adults: A Guide to Healthy Eating*. Canberra, Australia, National Health and Medical Research Council, Commonwealth of Australia, 2003
8. Salmerón J, Manson JAE, Stampfer MJ, Colditz GA, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of non-insulin-dependent diabetes mellitus in women. *JAMA* 277:472–477, 1997
9. Salmerón J, Ascherio A, Rimm EB, Colditz GA, Spiegelman D, Jenkins DJ, Stampfer MJ, Wing AL, Willett WC: Dietary fiber, glycemic load, and risk of NIDDM in men. *Diabetes Care* 20:545–550, 1997
10. Liu S, Manson JAE, Stampfer M, Hu FB, Giovannucci E, Colditz GA, Hennekens CH, Willett WC: A prospective study of whole-grain intake and risk of type 2 diabetes mellitus in US women. *Am J Public Health* 90:1409–1415, 2000
11. Meyer KA, Kushi LH, Jacobs DR Jr, Slavin J, Sellers TA, Folsom AR: Carbohydrates, dietary fiber, and incident type 2 diabetes in older women. *Am J Clin Nutr* 71:921–930, 2000
12. Stevens J, Ahn K, Juhaeri, Houston DK, Steffan L, Couper D: Dietary fiber intake and glycemic index and incidence of diabetes in African-American and white adults. *Diabetes Care* 25:1715–1721, 2002
13. Fung TT, Hu FB, Pereira MA, Liu S, Stampfer MJ, Colditz GA, Willett WC: Whole-grain intake and the risk of type 2 diabetes: a prospective study in men. *Am J Clin Nutr* 76:535–540, 2002
14. World Health Organization: *Definition, Diagnosis and Classification of Diabetes Mellitus and Its Complications: Report of a WHO Consultation: Part 1: Diagnosis and Classification of Diabetes Mellitus*. Geneva, World Health Org., Department of Noncommunicable Disease Surveillance, 1999
15. Ireland P, Jolley D, Giles G, O’Dea K, Powles J, Rutishauser I, Wahlqvist M, Williams J: Development of the Melbourne FFQ: a food frequency questionnaire for use in an Australian prospective study involving and ethnically diverse cohort. *Asia Pac J Clin Nutr* 3:19–31, 1994
16. Lewis J, Milligan G, Hunt A: *NUTTAB95 Nutrient Data Table for Use in Australia*. Canberra, Australia, Australian Government Publishing Service, 1995
17. Foster-Powell K, Holt HA, Brand-Miller JC: International tables of glycemic index and glycemic load values: 2002. *Am J Clin Nutr* 76:5–56, 2002
18. Ireland PD: Measuring dietary intake in a Melbourne cohort of men and women born in Australia, Italy and Greece. Melbourne, Australia, Monash Medical Centre, 1996, p. 250
19. Jenkins DJ, Kendall CW, Augustin LS, Franceschi S, Hamidi M, Marchie A, Jenkins AL, Axelsen M: Glycemic index: overview of implications in health and disease. *Am J Clin Nutr* 76:266S–273S, 2002
20. Rothman KJ, Greenland S: *Modern Epidemiology*. 2nd ed. Philadelphia, Lippincott-Raven, 1998
21. McLennan W, Podger A: *National Nutrition Survey Nutrient Intakes and Physical Measurements: Australia*. Canberra, Australia, Australian Bureau of Statistics, 1998
22. Brand-Miller JC, Holt SH, Pawlak DB, McMillan J: Glycemic index and obesity. *Am J Clin Nutr* 76:281S–285S, 2002
23. Willett W, Manson J, Liu S: Glycemic index, glycemic load, and risk of type 2 diabetes. *Am J Clin Nutr* 76:274S–280S, 2002
24. Wolever TM, Mehling C: Long-term effect of varying the source or amount of dietary carbohydrate on postprandial glucose, insulin, triacylglycerol, and free fatty acid concentrations in subjects with impaired glucose tolerance. *Am J Clin Nutr* 77:612–621, 2003
25. Daly M: Sugars, insulin sensitivity, and the postprandial state. *Am J Clin Nutr* 78: 865S–872S, 2003
26. Janket SJ, Manson JE, Sesso H, Buring JE, Liu S: A prospective study of sugar intake and risk of type 2 diabetes in women. *Diabetes Care* 26:1008–1015, 2003
27. Lopez-Ridaura R, Willett WC, Rimm EB, Liu S, Stampfer MJ, Manson JE, Hu FB: Magnesium intake and risk of type 2 diabetes in men and women. *Diabetes Care* 27:134–140, 2004
28. Song Y, Manson JE, Buring JE, Liu S: Dietary magnesium intake in relation to plasma insulin levels and risk of type 2 diabetes in women. *Diabetes Care* 27:59–65, 2004

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