Carbohydrate quantity and quality and risk of type 2 diabetes in the European Prospective Investigation into Cancer and Nutrition–Netherlands (EPIC-NL) study1–3

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ABSTRACT

BACKGROUND: Carbohydrate quantity and quality may play an important role in the development of type 2 diabetes.

OBJECTIVE: We investigated the associations of dietary glycemic load (GL), glycemic index (GI), carbohydrate, and fiber intake with the incidence of type 2 diabetes.

DESIGN: A prospective cohort study was conducted in 37,846 participants of the EPIC-NL (European Prospective Investigation into Cancer and Nutrition–Netherlands) study, aged 21–70 y at baseline and free of diabetes. Dietary intake was assessed with the use of a validated food-frequency questionnaire. Incident diabetes cases were mainly self-reported and verified against general practitioner records.

RESULTS: During a mean follow-up of 10 y, 915 incident diabetes cases were documented. Dietary GL was associated with an increased diabetes risk after adjustment for age, sex, established diabetes risk factors, and dietary factors [hazard ratio (HR) per SD increase: 1.32; 95% CI: 1.14, 1.54; \( P < 0.001 \)]. GI tended to increase diabetes risk (HR: 1.08; 95% CI: 1.00, 1.17; \( P = 0.05 \)). Dietary fiber was inversely associated with diabetes risk (HR: 0.92; 95% CI: 0.85, 0.99; \( P < 0.05 \)), whereas carbohydrate intake was associated with increased diabetes risk (HR: 1.15; 95% CI: 1.01, 1.32; \( P < 0.05 \)). Of the carbohydrate subtypes, only starch was related to increased diabetes risk [HR: 1.25 (1.07, 1.46), \( P < 0.05 \)]. All associations became slightly stronger after exclusion of energy misreporters.

CONCLUSIONS: Diets high in GL, GI, and starch and low in fiber were associated with an increased diabetes risk. Both carbohydrate quantity and quality seem to be important factors in diabetes prevention. Energy misreporting contributed to a slight attenuation of associations. Am J Clin Nutr 2010;92:905–11.

INTRODUCTION

Hyperglycemia, a hallmark feature of diabetes, was long viewed as a disorder of carbohydrate metabolism. However, the definition of carbohydrate-containing foods in terms of their capacity to increase blood glucose has been suggested to be a better determinant of diabetes risk (1, 2). High–glycemic index (high-GI) foods contain carbohydrates that break down rapidly and cause high postprandial glucose concentrations, whereas low-GI foods contain carbohydrates that break down slowly and cause lower postprandial glucose concentrations that decline more gradually (1). Glycemic load (GL) is the product of the GI and the amount of carbohydrate in a food and reflects both the quantity and quality of the carbohydrate (3). Until now, evidence regarding the role of GL and GI in relation to diabetes risk has remained inconclusive. Several studies reported increased risks of GL or GI (3–9), whereas others did not confirm this (10–15). Some studies suggested interactions with the degree of adiposity (4, 6, 9, 14) or with cereal fiber intake (3, 7, 8), but this was not always confirmed (4, 8). In Australia, the nutritional guidelines require foods to be labeled with a symbol and their GI value (16); however, the American Diabetes Association’s dietary guidelines for diabetes prevention state there is no sufficient, consistent information to conclude that low-GL diets reduce diabetes risk (17).

Misreporting of energy intake may be one of the factors explaining the inconsistent findings regarding GL, GI, and diabetes. To our knowledge, very little is known about the influence of energy misreporting on GL, GI, and diabetes. Lau et al (18) showed that after correction for energy intake, exclusion of energy underreporters did not affect the associations of GL and GI with body mass index (BMI) (18). To date, only one study has examined the influence of energy misreporting on the relation of GL and GI with diabetes, and no effect of excluding energy misreporters was reported (12).

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2. Partly supported by the InterAct project, funded by the European Union (Integrated Project LSHM-CT-2006-037197 in the Framework Programme 6 of the European Community). The EPIC-NL study was funded by the European Commission: Public Health and Consumer Protection Directorate 1993–2004; Research Directorate-General 2005; the Dutch Ministry of Public Health, Welfare, and Sports; the Netherlands Cancer Registry; LK Research Funds; Dutch Prevention Funds; Dutch ZON (Zorg Onderzoek Nederland); and the World Cancer Research Fund.

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In this study, we aimed to prospectively investigate the associations of dietary GI, GL, fiber, carbohydrate, and carbohydrate subtypes with the risk of type 2 diabetes. In addition, we investigated the influence of energy misreporting and explored possible interactions with dietary fiber and the degree of adiposity.

SUBJECTS AND METHODS

Study population

The European Prospective Investigation into Cancer and Nutrition (EPIC) was established to investigate the relation between nutrition, various lifestyles, and environmental factors and the incidence of cancer and other chronic diseases (19). EPIC-NL comprises the 2 Dutch contributions to the EPIC study: Prospect-EPIC and MORGEN-EPIC. The individual cohorts of EPIC-NL were set up simultaneously in 1993–1997 within the context of the EPIC study and were merged in 2007 according to standardized processes into one large Dutch EPIC cohort. Its design and rationale are described elsewhere (20). The Prospect-EPIC study includes 17,357 women, aged 49–70 y at baseline, who are participating in the national breast cancer screening program and living in the city of Utrecht and its surroundings (21). The MORGEN-EPIC cohort consists of 22,715 men and women aged 21–64 y selected from random samples of the Dutch population in 3 towns in the Netherlands (Amsterdam, Doetinchem, and Maastricht) (22, 23). All participants gave informed consent before they were included in the study. The study complied with the Declaration of Helsinki and was approved by the Institutional Board of the University Medical Center Utrecht.

After exclusion of prevalent diabetes cases (n = 615), individuals with extremely low or high reported energy intakes (ie, those in the top 0.5% and bottom 0.5% ratio of reported energy intake over estimated energy requirement [estimated with basal metabolic rate (BMR); n = 388]), participants with missing nutritional data (n = 213), and those who did not consent to linkage with disease registries (n = 931), 37,846 participants were left for the analysis.

Food intake

Daily nutritional intake was obtained from a food-frequency questionnaire (FFQ) containing questions on the usual frequency of consumption of 79 main food items during the year preceding enrollment. This questionnaire allows the estimation of the average daily consumption of 178 foods. The FFQ has been validated against twelve 24-h recalls (24–26). Spearman’s correlations were 0.60 for GL, 0.62 for GI, 0.74 (men) and 0.76 (women) for carbohydrate, and 0.61 (men) and 0.74 (women) for fiber. The GI of the foods was obtained from the international tables compiled by Foster-Powell et al (27) and Atkinson et al (28), which contain all relevant data published between 1981 and 2007. Intakes of nutrients were adjusted for total energy intake by means of the regression residual method (29).

Calculation of dietary GI and GL

We calculated the daily GI by summing the products of the GI value of a food with its available carbohydrate content, multiplied by the frequency of consumption of that food. These values were then divided by the total amount of available carbohydrate consumed (30). Such an expression of dietary GI per gram of carbohydrate reflects the overall quality of the daily carbohydrate intake. Daily GL was calculated in the same manner used for the GI but without dividing by the total amount of available carbohydrate consumed (3). The GL represents both the quality and quantity of carbohydrate and the interaction between them. Each unit of dietary GL represents the equivalent of 1 g carbohydrate from glucose.

Energy reporting

BMR was estimated with the use of the Schofield equations. Participants with an energy intake compared with BMR of <1.14 were defined as energy underreporters, whereas those with an energy intake compared with BMR of >2.40 were classified as energy overreporters according to the Goldberg cutoffs (31). Energy misreporters were defined as energy under- plus overreporters. The remaining participants were defined as normal energy reporters.

Diabetes

The occurrence of diabetes during follow-up was self-reported in 2 follow-up questionnaires at 3–5 y intervals. Participants were asked whether diabetes was diagnosed, in what year, by whom, and what treatment they received. Diagnoses of diabetes were also obtained from the Dutch Center for Health Care Information, which holds a standardized computerized register of hospital discharge diagnoses. In this register, admission files have been filed continuously from all general and university hospitals in the Netherlands from 1990 onward. All diagnoses were coded according to the International Classification of Diseases, Clinical Modification, 9th revision (32). Follow-up was completed 1 January 2006. In the Prospect-EPIC study, incident diabetes cases could also be detected via a urinary glucose strip test, sent out with the first follow-up questionnaire, for detection of glycosuria. Potential diabetes cases detected by any of these methods were verified against information obtained from the participants’ general practitioner or pharmacist through mailed questionnaires. Diabetes was defined as present when either of them confirmed the diagnosis. Verification information was available for 89% of the potential diabetes cases, and 72% of these cases were verified as type 2 diabetes and subsequently used for the analysis.

Baseline characteristics

At baseline, participants completed a general questionnaire containing questions on demographics, presence of chronic diseases, and risk factors for chronic diseases. Smoking was categorized into current, past, or never smoker; and parental history of diabetes was categorized into none, one, or both parents. Physical activity was assessed by means of a questionnaire validated in an elderly population (33), and the Cambridge Physical Activity Score was calculated and used to categorize physical activity (34). Because we could not calculate a total physical activity score for 14% of all participants, we imputed missing scores by single linear regression modeling [Statistical Package for Social Sciences (SPSS), Missing Value Analysis procedure]. Systolic and diastolic blood pressure measurements...
The proportionality assumption was checked visually by means of log-minus-log plots, with no deviations detected. Data were analyzed with SPSS (version 15.0; SPSS Inc, Chicago, IL) for Windows.

RESULTS
The mean age of the study population was 51 y, and ≈25% of the participants were male. Daily mean (±SD) dietary GL and GI and intakes of fiber and carbohydrate were 117.9 ± 21.3 g, 54.9 ± 3.9, 23.4 ± 4.8 g, and 222.0 ± 30.8 g, respectively. Sugar and starch each contributed ≈50% to the total carbohydrate. In total, 24.5% of participants were classified as energy underreporters and 1.0% as energy overreporters (Table 1). The main contributors to the GL were bread (34%), potatoes (13%), sweets (11%), and fruit (10%). Milk (products) (20%), bread (17%), fruit (16%), potatoes (15%), and drinks (14%) contributed the most to the GI. Pearson’s correlation coefficients between GL and carbohydrate and between GI and carbohydrate were 0.87 and 0.20, respectively.

During a mean (±SD) of 10.1 ± 1.9 y of follow-up, we documented 915 incident type 2 diabetes cases. In the univariable model, we found a nonsignificantly increased risk of diabetes with higher GL. After adjustment for age, sex, established diabetes risk factors, and nutritional intake, the risk of diabetes was significantly increased, with an HR of 1.27 (95% CI: 1.11, 1.44) per SD increase in GL. This increase in HR was mainly attributable to correction for dietary factors, and protein intake in particular. For GI, the risk of diabetes was increased in the univariable model. After adjustment for confounders, the association attenuated to borderline significant (HR per SD increase in GI: 1.08; 95% CI: 1.00, 1.17). Higher dietary fiber was associated with a decreased diabetes risk in the multivariable model (HR per SD increase: 0.89; 95% CI: 0.82, 0.98). Higher intakes of carbohydrate and sugar were associated with a lower incidence of diabetes in the univariable analyses, whereas starch was associated with a nonsignificantly increased risk. After adjustment for confounders, the risk of diabetes significantly increased per SD increase of carbohydrate and starch (HR for carbohydrate: 1.20; 95% CI: 1.01, 1.42; HR for starch: 1.23; 95% CI: 1.07, 1.42) but not for sugar (HR: 1.15; 95% CI: 0.98, 1.35) (Table 2).

Energy underreporters tended to be more often female, more often physically inactive, and to have a higher waist circumference and BMI compared with normal energy reporters. Energy overreporters tended to be less often female, less often physically inactive, and to have a lower waist circumference and BMI compared with normal energy reporters. Intakes of potatoes, bread, cakes and cookies, milk (products), and sweets tended to be higher in energy overreporters compared with normal energy reporters. Energy underreporters yielded slightly stronger associations with diabetes compared with the associations in the full cohort. We found significantly increased risks of diabetes with higher dietary GL, GI, carbohydrate, sugar, and starch and a significantly decreased risk with higher dietary fiber. Excluding both energy under- and overreporters yielded comparable results, although the associations of carbohydrate and sugar were attenuated to borderline significant (Table 3).

We observed no interactions with BMI, waist circumference, and fiber intake in the multivariable models (P values for interaction for GL: 0.62, 0.99, and 0.15, respectively; for GI: 0.24, 0.89, and 0.47, respectively). There was no interaction between carbohydrate and GI (P value for interaction: 0.67).
DISCUSSION

In this prospective study, higher dietary GL, GI, and carbohydrate, and lower dietary fiber increased the risk of type 2 diabetes in 37,846 Dutch adults aged 20–70 y at baseline. Associations became slightly stronger after exclusion of energy misreporters.

The strengths of our study include its prospective design, long follow-up time, large sample size, and large number of incident diabetes cases. Moreover, the use of validated diabetes cases minimized the presence of false-positive diabetes cases. This reduced the dilution of associations. In addition, increments of 1 SD in dietary GL, GI, and carbohydrate were shown to be

<table>
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<th>TABLE 1</th>
<th>Baseline characteristics and nutritional intake of the study population</th>
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<td>(^1) n = 37,846. All dietary variables were adjusted for total energy intake by the regression residual method (29).</td>
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<td>(^2) Median; interquartile range in parentheses (all such values).</td>
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<td>(^3) Mean ± SD (all such values).</td>
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DISCUSSION

In this prospective study, higher dietary GL, GI, and carbohydrate, and lower dietary fiber increased the risk of type 2 diabetes in 37,846 Dutch adults aged 20–70 y at baseline. Associations became slightly stronger after exclusion of energy misreporters.

The strengths of our study include its prospective design, long follow-up time, large sample size, and large number of incident diabetes cases. Moreover, the use of validated diabetes cases minimized the presence of false-positive diabetes cases. This reduced the dilution of associations. In addition, increments of 1 SD in dietary GL, GI, and carbohydrate were shown to be

<table>
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<th>TABLE 2</th>
<th>Univariable and adjusted hazard ratios (95% CI) for the association of glycemic load, glycemic index, fiber, carbohydrate, sugar, and starch with incident type 2 diabetes</th>
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<td>Model 3 (model 2 + dietary intake)</td>
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<td>(^1) Hazard ratios are presented per SD increase. n = 37,846, with 915 incident diabetes cases. Model 1 was adjusted for sex (male or female) and age at recruitment (continuous). Model 2 was adjusted as in model 1 plus energy-adjusted alcohol consumption (&lt;11, 11–25, 26–50, &gt;50 g/d), physical activity (not active, moderately inactive, moderately active, active), waist circumference (continuous), BMI [in kg/m²; &lt;20, 20–25 (reference group), 25.1–30, &gt;30], smoking status (never, past, current), mean systolic blood pressure (continuous), educational level (high, middle, low), family history of diabetes (none, one parent, both parents). Model 3 was adjusted as in model 2 plus energy intake and energy-adjusted intake of vitamin C, vitamin E, protein, saturated fat, and polyunsaturated fat (all continuous). Analyses of glycemic load, glycemic index, carbohydrate, sugar, and starch with diabetes risk were additionally adjusted for total fiber intake. Analysis of fiber with diabetes risk was additionally adjusted for glycemic load. Analysis of sugar with diabetes risk was additionally adjusted for starch intake. Analysis of starch intake with diabetes risk was additionally adjusted for sugar intake. *P &lt; 0.05, **P &lt; 0.001.</td>
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Several mechanisms may relate high-GI diets to diabetes. High-GI diets can rapidly increase postprandial glucose levels, thereby increasing insulin demand. This may lead to pancreatic exhaustion. In addition, high-GI diets can increase postprandial free fatty acid release, directly increasing insulin resistance (39, 40). Furthermore, high-GI diets may promote weight gain (39, 41), suggesting mediation through weight gain. Additional adjustment for annual weight change did not affect our findings (HR for GL: 1.23; 95% CI: 1.11, 1.44). However, weight during follow-up was mainly self-reported. This may have limited us in detecting possible mediating effects of weight change. Removal of baseline BMI and waist circumference from the final model only slightly affected the results (HR for GL: 1.23; 95% CI: 1.08, 1.40). Altogether, this provides little support for a mediation role of body weight (gain).

Carbohydrate and sugar were inversely related to diabetes in the univariable, first, and second models (the latter for sugar only), as also found by others (8, 42, 43). Confounding by healthy lifestyle behaviors may underlie this [eg, high fruit consumption largely contributes to sugar intake (44)]. Indeed, the inverse associations disappeared after further adjustment was made for lifestyle factors. Total carbohydrate was related to increased risk of diabetes in the final model, in contrast to the majority of prospective studies (3, 4, 8–11, 42, 43, 45). Carbohydrate intakes were comparable to those reported in the majority of other studies (3, 4, 8–11, 42, 43, 45), but differences in carbohydrate sources or GL may account for this. The high correlation (0.87) between GL and carbohydrate made it difficult to separate the effects of GL and carbohydrate. However, we found a stronger association for GL than for carbohydrate, suggesting that not only the amount but also the quality of consumed carbohydrate is an important determinant of diabetes.

We observed no evidence for different effects of simple and complex carbohydrate subtypes on diabetes risk. The relation of carbohydrate subtypes with diabetes is not yet very clear. Most carbohydrates, including those that are simple and complex, contribute to the GL and GI. However, these measures may not fully capture the effects of carbohydrates on glucose metabolism. Further research is needed to better understand the role of carbohydrate subtypes in the development of diabetes.
prospective studies found no relation between starch and diabetes (10, 11, 42, 43). One reported a positive association (4), in line with our results. Two studies found no relation between total sugar and diabetes (10, 42), whereas one found an inverse relation (4). Possibly, underreporting of sugar intake contributed to these inverse or null findings. This is supported by our findings, because the relation of sugar with diabetes strengthened to a significantly increased risk after exclusion of energy underreporters.

Total fiber was inversely related with diabetes, as previously reported by others (11, 46, 47) but not all (48). The inverse relation of total fiber with diabetes seems mainly attributable to insoluble or cereal fiber (48). This may explain the inconsistencies among studies. Unfortunately, our study did not provide information on intake of specific fiber subtypes.

In conclusion, our findings support the view that diets high in GL, GI, and carbohydrate, and low in fiber increase the risk of diabetes. Both carbohydrate quantity and quality seem to be important factors in diabetes prevention. These findings were not modified by the degree of adiposity. Energy misreporting may slightly attenuate associations of GL and GI with diabetes, even after correction for total energy intake.

We thank the Central Bureau for Statistics and the PHARMO Institute for providing follow-up data on cardiovascular disease and vital status. The authors’ responsibilities were as follows—IS, YTvdS, DLvdA, AMS, FBH, DEG, and JWB: study concept and design; IS: data analysis; IS, YTvdS, DLvdA, AMS, FBH, DEG, and JWB: interpretation of results; IS, YTvdS, and JWB: drafting of manuscript; and DLvdA, AMS, FBH, and DEG: critical review of manuscript. None of the authors declared a conflict of interest.

REFERENCES

Erratum


The term “precursor-product ratio” used throughout the article would be more correctly called “product-precursor ratio.” Despite this name change, the ratio and the data and their interpretation remain correct.

In addition, inaccurate wording appears in the second sentence of the Results section of the abstract (page 1040). As published, the sentence reads: “Total n–3 PUFA intakes were 57–80% lower in non-fish-eaters than in fish-eaters, but status differences were considerably smaller.” Instead, the sentence should read as follows: “Total n–3 PUFA intakes in non-fish-eaters were 57–80% of those in fish-eaters, but status differences were considerably smaller.” These figures are referred to correctly in the second sentence of the Discussion section on page 1048.


Erratum


In the second sentence of the Results section of the abstract on page 905, the hazard ratio and 95% CI are incorrect. The sentence should read as follows:

“Dietary GL was associated with an increased diabetes risk after adjustment for age, sex, established diabetes risk factors, and dietary factors [hazard ratio (HR) per SD increase: 1.27; 95% CI: 1.11, 1.44; P < 0.001].”


Erratum


After our article (1) was published online, we became aware that the FitnessGram cutoffs for body composition had been revised. (This revision was announced in an e-mail dated November 2010.) According to the FitnessGram website (2), the previous standards for percentage body fat and body mass index (BMI) “were based on the best available research at the time they were developed, … but some inconsistencies became apparent.”

The new FitnessGram BMI cutoffs (3) categorize children and adolescents into 4 groups: 1) very lean, 2) within the Healthy Fitness Zone, 3) needs improvement—some risk, and 4) needs improvement—high risk. The previous and revised cutoffs for the upper categories in FitnessGram, along with the CDC (Centers for Disease Control and Prevention) 85th and 95th percentiles of BMI (4), are shown in Figure 1. As noted in our article (1), the previous FitnessGram BMI cutoffs resulted in marked differences (ranging from 2% to 20%) in the prevalence of children who had a high FitnessGram BMI across ages.

The revised FitnessGram BMI cutoffs are fairly close to the CDC 85th percentile, with the cutoffs for “some risk” varying from the CDC 79th to 83rd percentiles of BMI by sex and age. The “high risk” cutoffs range from the CDC 87th to 91st percentiles. On the basis of these revised cutoffs, it is likely 1) that the prevalence of children with a high FitnessGram

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