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Dietary glycemic index, dietary glycemic load, and mortality among men with established cardiovascular disease

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Abstract

Background/Objectives—In men with established cardiovascular disease, the effect of diets with high glycemic index (GI) and glycemic load (GL) is unknown. We tested the hypothesis that diets with higher GI and GL are associated with increased mortality in men with established cardiovascular disease.

Subjects/Methods—We measured dietary GI and GL using food-frequency questionnaires in 4 617 men, 45–79 years old, with a history of cardiovascular disease. The men were followed for cardiovascular mortality (6 year follow-up, 608 cases) and all-cause mortality (8 year follow-up, 1 303 cases) using the Swedish cause-of-death and death registers. We used Cox models with age as the time scale and adjusted for body mass index, physical activity, history of hypertension and diabetes, family history of myocardial infarction, aspirin use, cigarette smoking, and dietary factors to estimate incidence rate ratios (RR).

Results—Comparing top to bottom quartiles of dietary GI, the RR for cardiovascular mortality was 0.86 (95% confidence interval (CI) 0.67–1.10, *p* for linear trend = 0.21), and the RR for all-cause mortality was 1.00 (95% CI 0.85–1.19, *p* for linear trend = 0.87). Compared to quartile 1, the RR for men with dietary GL in quartile 4 was 1.02 (95% CI 0.70–1.49, *p* for linear trend = 0.81) for cardiovascular and 1.15 (95% CI 0.89–1.49, *p* for linear trend = 0.20) for all-cause mortality.

Conclusions—In this population of men with prior cardiovascular disease, dietary GI and GL were not associated with cardiovascular or all-cause mortality.

Keywords

glycemic index; glycemic load; mortality

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INTRODUCTION

High dietary glycemic index (GI), a measure of the average propensity of carbohydrate in the diet to increase blood glucose (Jenkins *et al.*, 1981; Salmeron *et al.*, 1997), and high dietary glycemic load (GL), the product of dietary GI and carbohydrate consumed (Salmeron *et al.*, 1997; Brand-Miller *et al.*, 2003), have been associated with increased risk of incident coronary heart disease in women (Liu *et al.*, 2000; Halton *et al.*, 2006; Beulens *et al.*, 2007). To date, prospective studies in men have not demonstrated an association (van Dam *et al.*, 2000; Levitan *et al.*, 2007). Diets high in GL have also been associated with incidence of total stroke in women and hemorrhagic stroke in men (Oh *et al.*, 2005; Levitan *et al.*, 2007). However, the effect of high dietary GI and GL on survival in patients with existing cardiovascular disease is not known. In one study, high carbohydrate diets were linked to accelerated atherosclerosis progression, and high GI carbohydrates were particularly harmful (Mozaffarian *et al.*, 2004). In experimental studies, high GI and GL diets have been documented to have adverse effects on cardiovascular risk factors such as triglyceride concentrations, LDL cholesterol, the ratio of total to HDL cholesterol, and C-reactive protein (Ludwig, 2002; Pereira *et al.*, 2004; Sloth *et al.*, 2004; McMillan-Price *et al.*, 2006). Because these risk factors are associated with worse prognosis among patients with established cardiovascular disease (Smith *et al.*, 2006), we hypothesized that diets high in GI and GL may be associated with increased total and cardiovascular mortality in patients with existing cardiovascular disease. We examined the associations of dietary GI and GL with cardiovascular disease and all-cause mortality among middle-aged and older men with a history of hospitalization for cardiovascular disease at baseline. Because studies suggest that the effects of high GI and GL diets may be more pronounced in the setting of overweight (Liu *et al.*, 2000; Tavani *et al.*, 2003; Oh *et al.*, 2005; Beulens *et al.*, 2007), we tested for variations in the associations by body mass index (BMI) and waist to hip ratio (WHR).

SUBJECTS AND METHODS

We studied 4 617 men who had at least one hospitalization for cardiovascular disease recorded in the Swedish inpatient register and who were participants in the Cohort of Swedish Men. Of these men, 1 060 had ever been hospitalized for stroke (including 173 who had a history of both myocardial infarction (MI) and stroke), 2 027 had been hospitalized for MI and had never been hospitalized for stroke, and 1 530 had been hospitalized for a cardiovascular disease other than stroke or MI. On average the first cardiovascular event occurred 8.8 years prior to baseline, January 1, 1998, (range 1 day-33 years). The Cohort of Swedish Men population and exposure assessment have been described previously (Messerer *et al.*, 2004; Larsson *et al.*, 2006). The ethics committees for the Karolinska Institute, Uppsala University Hospital, and the Örebro region approved the Cohort of Swedish Men.

At baseline the men completed a 96-item food-frequency questionnaire (FFQ) and items regarding sociodemographic factors, anthropometric data, and physical activity. The self-administered FFQ asked participants to report their usual frequency of consumption of foods and beverages over the previous year. There were eight possible responses ranging from

never to three or more times per day. Total consumption of each of the foods and beverages was calculated by multiplying the frequency of consumption by age-specific portion sizes determined from two weeks of diet records completed by 152 men. Nutrient values were calculated using food composition data from the Swedish National Food Administration (Bergström *et al.*, 1991).

A database of GI and GL values with white bread as the reference food was created primarily based on the 2002 international GI and GL tables (Foster-Powell *et al.*, 2002). We calculated average dietary GI using the formula dietary GI = (Σ frequency of consumption X carbohydrate per age-specific serving X GI) / total carbohydrate. Dietary GL was calculated as the product of dietary GI and total carbohydrate intake divided by 100. The correlation between the FFQ and two one-week diet records was 0.77 for dietary GL and 0.62 for dietary GI in a validation study of men (Levitan *et al.*, 2007).

Nutrient values and dietary GL were adjusted for energy using the residuals method (Willett, 1998). All nutrient values were adjusted to 9 209 kJ (2 200 kcal)/d, the mean energy intake determined from validation study diet records. We estimated the minutes per day of activity from self-reported time spent walking, cycling, and exercising. We calculate BMI and WHR from self-report anthropometric data.

Participants were followed from January 1, 1998 until December 31, 2003 for cardiovascular disease mortality by linkage to the Swedish cause-of-death register. All-cause mortality was determined by linkage to the Swedish death register with follow-up through December 31, 2005.

We calculated means or percentages of dietary and lifestyle covariates by quartile of dietary GI and GL. To estimate the incidence rate ratios (RR) of cardiovascular and all-cause mortality by quartile of dietary GI and GL, we used Cox proportional hazard models with age and calendar time as the joint time scales. The models were adjusted for BMI (< 20 kg/m², 20–24.9 kg/m², 25–29.9 kg/m², ≥ 30 kg/m²), physical activity (approximate tertiles), history of hypertension (yes, no), history of diabetes (yes, no), family history of myocardial infarction before the age of 60 (yes, no), aspirin use (yes, no), cigarette smoking (current, past, never), and quartiles of total energy, saturated fat, polyunsaturated fat, protein, alcohol, and cereal fiber. Tests of linear trend were performed by entering the median of each quartile as a predictor into the models. Further adjustment for quartiles of magnesium, folate, vitamin E, and coffee consumption, use of multivitamins, marital status, and education level did not substantially affect the results. We tested for violation of the proportional hazards assumption by entering interaction terms between the exposures and the natural logarithm of time into the model. When we observed a deviation from proportionality of the hazards over time, we conducted separate analyses during the first and second halves of the follow-up period.

We conducted stratified analysis and tests for interaction by BMI (< 25 kg/m² or ≥ 25 kg/m² and < 30 kg/m² or ≥ 30 kg/m²) and WHR (< 0.9 or ≥ 0.9). The analysis stratified by WHR was limited to 3 640 men who reported both waist and hip circumference. In sensitivity analyses, we examined whether the associations of dietary GI and GL with cardiovascular

and all-cause mortality varied by cereal fiber and saturated fat consumption. We additionally stratified by baseline history of cardiovascular disease (any hospitalization for stroke, hospitalization for MI but no hospitalization for stroke, or hospitalization for cardiovascular diseases other than stroke or MI). Analyses were conducted using SAS version 9.1 (SAS Institute, Cary, NC, USA). A two-sided p -value of 0.05 was considered statistically significant.

RESULTS

Among 4 617 men with established cardiovascular disease at baseline, there were 608 cardiovascular deaths over 6 years of follow-up and 1 303 deaths due to all causes combined over 8 years of follow-up. Men with dietary GI in the top quartile on average spent less time physically active and consumed more carbohydrate and cereal fiber and less protein and alcohol than those with lower dietary GI (Table 1). Compared to men with dietary GL in the lowest quartile, men with dietary GL in the highest quartile were slightly older and less likely to have hypertension and consumed less fat, protein, and alcohol and more carbohydrate and cereal fiber.

We did not find significant associations of dietary GI or GL with cardiovascular or all-cause mortality (Table 2). The interaction terms between quartiles of dietary GI and the natural logarithm of time were statistically significant in models for all-cause mortality ($p = 0.02$), indicating a violation of the proportional hazards assumption. To explore this deviation, we examined association between dietary GI and all-cause mortality in two time periods—1998–2001 and 2002–2005. During the first 4 years of follow-up, we found that compared to men in the lowest quartile, men with dietary GI in quartiles 2–4 had non-significantly decreased rates of mortality (RR = 0.91, 0.84, and 0.84 respectively, p for linear trend = 0.15). During the second 4 years of follow-up, men with dietary GI in quartiles 2–4 had non-significantly increased rates of mortality (RR = 1.04, 1.19, and 1.17 respectively, p for linear trend = 0.13).

Among men with cereal fiber intake below the median (< 16.8 g/day), the RR of all-cause mortality comparing extreme quartiles of dietary GL was 1.64 (95% CI 1.17–2.30), and among men with cereal fiber intake above the median (≥ 16.8 g/day) the RR of all-cause mortality comparing extreme quartiles of dietary GL was 0.64 (95% CI 0.42–0.98). The test for interaction was statistically significant ($p = 0.006$). Results for cardiovascular mortality were similar. We did not find evidence for variation of the association by saturated fat consumption. When we stratified by type of previous cardiovascular disease (stroke, MI, or other cardiovascular disease), we found that high dietary GI and GL appeared to be associated with increased risk of death among men with previous MI and decreased risk of death among men with previous stroke. However, when we further stratified by cereal fiber intake, high dietary GL appeared to be associated with increased mortality among men who consumed less cereal fiber and decreased mortality among men who consumed more cereal fiber in all disease groups.

The associations did not vary by overweight (BMI < 25 kg/m² or ≥ 25 kg/m²), obesity (BMI < 30 kg/m² or ≥ 30 kg/m²), WHR (< 0.9 or ≥ 0.9), or saturated fat consumption.

DISCUSSION

Overall, we did not find associations of dietary GI and GL assessed using self-administered questionnaires with cardiovascular and all-cause mortality. However, dietary GL was associated with increased mortality in the subgroup who consumed relatively little cereal fiber and with decreased mortality in the subgroup who consumed more cereal fiber.

Although we are not aware of other studies of the association between dietary GI and GL and survival in cardiovascular disease patients, dietary GI and GL have been associated with incident coronary heart disease in women, particularly overweight women (Liu *et al.*, 2000; Halton *et al.*, 2006; Beulens *et al.*, 2007). This association has not been observed in men (van Dam *et al.*, 2000; Tavani *et al.*, 2003; Levitan *et al.*, 2007). Dietary GL was associated with total stroke in women and hemorrhagic stroke in men (Oh *et al.*, 2005; Levitan *et al.*, 2007). In controlled feeding studies, diets high in GI and GL have been shown to have adverse effects on total cholesterol, HDL cholesterol, triglycerides, C-reactive protein, and, among people with diabetes, glycosylated hemoglobin (Bouche *et al.*, 2002; Ludwig, 2002; Brand-Miller *et al.*, 2003; Pereira *et al.*, 2004; Sloth *et al.*, 2004; Ebbeling *et al.*, 2005; McMillan-Price *et al.*, 2006). Similar associations have been observed in cross-sectional studies of self-reported diet (Frost *et al.*, 1999; Ford & Liu, 2001; Liu *et al.*, 2001; Liu *et al.*, 2002; Amano *et al.*, 2004; McKeown *et al.*, 2004; Slyper *et al.*, 2005). In addition, high carbohydrate diets that were also high in GI were associated with accelerated atherosclerosis progression (Mozaffarian *et al.*, 2004). This evidence lead us to hypothesis that high GI and GL diets would be associated with decreased survival in men with cardiovascular disease.

In addition to a true absence of biological effect of dietary GI or GL on survival in men with cardiovascular disease, there are a number of potential explanations for the lack of association observed in our primary analysis. First, cereal fiber consumption was quite high in this population (median 16.8 g/day), and dietary GL appeared to be harmful only in those with relatively low cereal fiber. High fiber intake may alleviate any harmful effect of a high GL diet though improvements in insulin response, glucose control, and blood lipids (Flight & Clifton, 2006).

Second, dietary GI and GL may be causes of morbidity but not mortality. However, among men from the same source population without baseline cardiovascular disease, dietary GI and GL were not associated with ischemic cardiovascular disease or cardiovascular mortality; we did observe a marginally significant association between dietary GL and hemorrhagic stroke (Levitan *et al.*, 2007). The dietary GI and GL calculated from the FFQ may not reflect the glucose-raising potential of the diet. However, we used methods similar to those used in other populations, and we have validated the measures against diet records and the correlations were in the same range as other dietary factors measured using FFQ (Levitan *et al.*, 2007). Random errors on the FFQ that are not associated with mortality will usually tend to bias the estimates towards 1. If, on the other hand, the errors in measurement are associated with mortality, for example through the participant's health status when the questionnaire was completed, the estimated effects could be biased in unpredictable directions. If men susceptible to ill effects of high GI and GL diets did not survive long enough to enroll, leaving only those relatively non-responsive, a harmful effect could have

been masked. Men with high dietary GI and GL had characteristics that would be expected to have both harmful and protective effects on survival. As in all observational studies we can not rule out confounding due to factors not measured or measured poorly.

The unexpected differences between the men with prior stroke and men with prior MI appeared to be due to differential intake of cereal fiber and dietary GL, though the results could also have been due to chance. Alternatively, stroke patients with more severe disease may consume a diet lower in GI and GL causing high dietary GI and GL to look protective; we were not able to measure disease severity or to assess this hypothesis. This is in contrast to the observation that incidence of hemorrhagic stroke was elevated in men with high dietary GL who did not have a history of cardiovascular disease at baseline (Levitan *et al.*, 2007).

In summary, we did not observe an association of dietary GI and dietary GL with cardiovascular or all-cause mortality overall in this population of middle-aged and older men with established cardiovascular disease. The results suggested dietary GL may be harmful in the setting of lower cereal fiber intake, but further research will be necessary to confirm or refute these findings.

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Table 1

Age-standardized characteristics of 4,617 middle-aged mean with established cardiovascular disease by quartile of dietary glycemic load (means or percentages)

Characteristic	Dietary glycemic index ¹				Dietary glycemic load ¹				p-value trend
	Quartile 1	Quartile 2	Quartile 3	Quartile 4	Quartile 1	Quartile 2	Quartile 3	Quartile 4	
Age (y)	67.5	67.1	67.4	67.6	67.1	67.2	67.6	67.8	0.02
Body mass index (kg/m ²)	26.5	26.1	26.2	29.1	26.4	26.3	26.2	26.1	0.04
Physical activity (min/d)	60.6	60.7	57.8	54.2	56.4	58.8	59.7	58.4	0.31
History of hypertension (%)	49.9	48.5	45.2	45.8	50.6	46.7	47.4	44.8	0.04
History of diabetes (%)	17.2	14.3	14.9	12.9	17.1	14.7	14.1	13.3	0.07
Family history of myocardial infarction at < 60 years (%)	21.5	21.8	20.0	18.9	20.5	20.4	20.9	20.5	0.99
Aspirin use (%)	65.6	67.5	64.9	63.6	62.7	67.3	65.0	66.6	0.09
Cigarette smoking (%)									0.003
Never	24.7	30.8	29.8	27.9	22.8	28.1	30.6	31.8	
Past	52.1	48.6	46.5	44.0	52.6	46.2	46.3	46.2	
Current	23.2	20.7	23.7	28.1	24.7	25.7	23.1	22.1	
Total energy (kJ/d)	10 174	10 213	10 241	9 952	10 356	10 132	10 134	9 957	0.004
Saturated fat (g/d) ²	33.4	31.3	31.8	31.8	39.0	33.5	30.4	25.4	<0.001
Monounsaturated fat (g/d) ²	24.4	24.2	24.5	24.7	27.7	25.3	23.8	20.9	<0.001
Polysaturated fat (g/d) ²	9.6	9.8	9.8	9.7	10.2	9.9	9.7	9.1	<0.001
Carbohydrate intake (g/d) ²	267	282	287	294	242	273	293	323	<0.001
Protein intake (g/d) ²	95.3	89.0	85.9	82.0	97.4	90.3	85.8	78.6	<0.001
Alcohol intake (g/d)	12.8	10.1	7.9	6.1	13.1	10.3	7.9	5.5	<0.001
Cereal fiber intake (g/d) ²	15.2	17.6	18.4	18.9	14.3	17.2	18.5	20.1	<0.001
Dietary glycemic index ¹	71.9	76.6	79.4	83.4	74.0	77.1	78.9	81.3	<0.001
Dietary glycemic load ^{1,2}	192	216	228	245	179	210	231	263	-

¹ White bread used as the reference food

² Energy adjusted using the residuals method

Table 2

Incidence rate ratios of cardiovascular and all-cause mortality by quartile of dietary glycemic load and dietary glycemic index among 4,617 men with cardiovascular disease at baseline

	Quartile 1	Quartile 2	Quartile 3	Quartile 4	P for linear trend
<i>Dietary glycemic index</i>					
Median dietary glycemic index ¹	72.8	76.9	79.4	82.9	
Cardiovascular mortality (n = 608)					
RR (95% CI) ²	1	0.83 (0.66–1.04)	0.85 (0.68–1.07)	0.90 (0.73–1.12)	0.38
RR (95% CI) ³	1	0.91 (0.72–1.15)	0.86 (0.67–1.09)	0.86 (0.67–1.10)	0.21
All-cause mortality (n = 1303)					
RR (95% CI) ²	1	0.90 (0.77–1.06)	1.00 (0.85–1.16)	1.04 (0.89–1.21)	0.46
RR (95% CI) ³	1	0.98 (0.83–1.15)	1.02 (0.87–1.21)	1.00 (0.85–1.19)	0.87
<i>Dietary glycemic load</i>					
Median dietary glycemic load ¹	184	210	231	285	
Cardiovascular mortality					
RR (95% CI) ²	1	0.77 (0.61–0.96)	0.84 (0.67–1.05)	0.82 (0.65–1.02)	0.13
RR (95% CI) ³	1	0.88 (0.68–1.13)	1.02 (0.76–1.38)	1.02 (0.70–1.49)	0.81
All-cause mortality					
RR (95% CI) ²	1	0.84 (0.72–0.98)	0.92 (0.79–1.07)	0.89 (0.76–1.04)	0.27
RR (95% CI) ³	1	0.96 (0.81–1.15)	1.13 (0.92–1.38)	1.15 (0.89–1.49)	0.20

¹ White bread used as reference food

² Incidence rate ratios, 95% confidence intervals, age used as the time scale

³ Incidence rate ratios, 95% confidence intervals, age used as the time scale, adjusted for BMI (< 20 kg/m², 20–24.9 kg/m², 25–29.9 kg/m², ≥ 30 kg/m²), physical activity (approximate tertiles), history of hypertension (yes, no), history of diabetes (yes, no), family history of myocardial infarction before the age of 60 (yes, no), aspirin use (yes, no), cigarette smoking (current, past, never), and quartiles of total energy, saturated fat, polyunsaturated fat, protein, alcohol, and cereal fiber