British Journal of Nutrition (2010), **103**, 1049–1055
doi:10.1017/S0007114509992674

Dietary glycaemic index, dietary glycaemic load and incidence of myocardial infarction in women

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(Received 8 July 2009 – Revised 28 September 2009 – Accepted 5 October 2009 – First published online 14 December 2009)

The association of dietary glycaemic index (GI) and glycaemic load (GL) with CVD has been examined in several populations with varying results. We tested the hypothesis that women with diets high in GI or GL would have higher rates of myocardial infarction (MI), and the associations would be stronger in overweight women. We measured dietary GI and GL in 36 234 Swedish Mammography Cohort participants aged 48–83 years using FFQ. Cox models were used to calculate incidence rate ratios (RR) and 95 % CI for hospitalisation or death due to MI assessed using the Swedish inpatient and cause-of-death registers from 1 January 1998 until 31 December 2006. Over 9 years of follow-up, 1138 women were hospitalised or died due to a first MI. In multivariable-adjusted models, the RR comparing top to bottom quartile of dietary GI were 1·12 (95 % CI 0·92, 1·35, *P*-trend=0·24), and the RR comparing top to bottom quartile of dietary GL were 1·22 (95 % CI 0·90, 1·65, *P*-trend=0·23). Among overweight women, the RR comparing top to bottom quartile of dietary GI were 1·20 (95 % CI 0·91, 1·58, *P*-trend=0·22), and the RR comparing top to bottom quartile of dietary GL were 1·45 (95 % CI 0·93, 2·25, *P*-trend=0·16). There were no statistically significant associations of dietary GI or GL with MI in this population.

Glycaemic index: Glycaemic load: Myocardial infarction

Glycaemic index (GI), the relative increase in blood glucose due to carbohydrate in a test food compared with a reference food, was initially developed as a meal planning tool for people with diabetes mellitus[1]. More recently, there have been experimental and observational studies examining whether the carbohydrate quality influences the risk of CVD.

Randomised diet trials have demonstrated that diets with reduced GI or glycaemic load (GL), the product of GI and carbohydrate content of a food[2,3], influence total cholesterol[4–6], LDL cholesterol[4,6,7], HDL cholesterol[8–10], TAG concentration[11,12], plasminogen activator inhibitor 1[12], insulin resistance[4,11] and inflammation[11,13,14] in ways that would be expected to reduce cardiovascular risk. A meta-analysis of trials showed that low-GI and -GL diets reduced BMI, total body mass and fat mass more than control diets[15], and a low-GL diet was better than a control diet for maintenance of weight loss[16], possibly because low-GI and -GL meals result in higher satiety and lower food intake[17,18]. In addition, low-GI or -GL diets improve glucose control in people with diabetes mellitus in randomised trials[9,19] and are associated with lower risk of developing diabetes mellitus in observational studies[20–22].

There have been several previous studies examining the association of dietary GI, the average GI of carbohydrates consumed, and dietary GL, the product of dietary GI and total carbohydrate, with CHD[3,23–26]. A meta-analysis that pooled data from prospective studies of men and women reported a statistically significant association[27]. However, dietary GI and GL were associated with increased rates of CHD in women[3,23], but studies in men, including our previous report on a population of men from central Sweden have not shown an association[24,26].

We therefore conducted a prospective study of the association of dietary GI and GL with incidence of myocardial infarction (MI) in a population of middle-aged and older women from central Sweden. Dietary GI and GL were assessed using the same FFQ as in our previous study of men[24]. Because the associations of dietary GI and GL with CVD were stronger in overweight than normal weight women in some studies[3,23] possibly because of a greater prevalence of insulin resistance among overweight women, we tested for variation by BMI and by physical activity, another determinant of insulin sensitivity. We also examined the possibility of interaction of dietary GI and GL with fibre consumption, because dietary GL was associated with survival

Abbreviations: GI, glycaemic index; GL, glycaemic load; MI, myocardial infarction; RR, rate ratio.

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among men with established CVD only in the context of low fibre intake[28].

Methods

Population

The present study included 36 234 women who participated in the Swedish Mammography Cohort. The recruitment process, characteristics and study methods of this prospective cohort have been previously described[29]. The Swedish Mammography Cohort includes women born between 1914 and 1948 living in Västmanland and Uppsala counties in central Sweden. The women completed the questionnaires with items on demographic, behavioural and anthropometric factors and consumption of foods and beverages in late 1997. Participants who did not provide or provided incorrect national identification numbers, who reported implausible energy intakes (>3 SD from the natural logarithm-transformed mean) or who had a previous diagnosis of cancer (other than nonmelanoma skin cancer) were excluded (*n* 792). Additionally for these analyses, participants who at baseline had a history of heart failure, MI or diabetes were excluded (*n* 2201). Participants with baseline diabetes were excluded because people who developed diabetes were received dietary counselling and may change both their diet and their reporting of diet. History of heart failure and MI was determined through linkage to the inpatient register; history of diabetes was assessed using self-report and linkage to the inpatient register. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki and all procedures involving human subjects/patients were approved by the Regional Ethical Review Board at Karolinska Institute, Stockholm, Sweden. Completion and return of the self-administered questionnaire was taken to imply consent.

Diet assessment

Self-administered food-frequency items in the questionnaires asked participants to report usual frequency of consumption of ninety-six foods and beverages over the previous year. Participants reported servings/d or servings/week for commonly consumed foods and beverages such as milk, coffee, cheese and bread. For other foods there were eight predefined responses ranging from never to ≥ 3 times/d. Portion sizes for most foods were not specified. In validation studies using weighed diet records, habitual portion sizes were found to vary by age. The total consumption of foods and beverages was calculated by multiplying the frequency of consumption by age-specific portion sizes. Nutrient values were calculated using food composition data from the Swedish National Food Administration[30].

A database of GI and GL values with white bread as the reference food was created based primarily on the international GI and GL tables[31]. Food items and mixed meals with no reported GI value were assigned the value for a comparable food. We calculated average dietary GI using the formula:

$$\text{Dietary GI} = \sum_{\text{foods}} \text{F} \times \text{C} \times \text{GI} / \sum_{\text{foods}} \text{F} \times \text{C},$$

where F represents the frequency of consumption, C represents the available carbohydrate content of an age-specific food portion and GI represents the GI of a specific food[32]. Dietary GL was calculated as the product of dietary GI and total carbohydrate intake divided by 100[32]. Using the residuals method[33], nutrient values and dietary GL were adjusted to 7113 kJ/d, the approximate mean energy intake from the validation study diet records. The correlation between the FFQ used in the present study and two 1-week weighed diet records was 0·62 for dietary GI and 0·77 for dietary GL in a population of men from central Sweden[34].

Outcome assessment

Participants were followed for hospitalisation due to or death from MI through record linkage to the Swedish inpatient and cause-of-death registers. These registers are more than 99 % complete[35]. We included only hospitalisations or deaths with MI listed as the primary diagnosis and only the first MI event recorded in the registers for each individual. Follow-up time accrued from 1 January 1998 until the date of hospitalisation or death from MI, the date of death from other causes or 31 December 2006, whichever was earliest.

Statistical analysis

Because some of the participants were missing data needed to calculate BMI (1·6 %) and a physical activity score (22·1 %), we used Markov chain Monte Carlo multiple imputation to simulate five complete datasets[36]. All statistical analyses described were performed in each of the datasets separately. The results were averaged, and CI and *P*-values were calculated accounting for the uncertainty in the imputed estimates[36].

We calculated means and proportions of baseline covariates. To estimate incidence rate ratios (RR) for the association of quartiles of dietary GI and GL with incidence of MI, we used Cox proportional hazards models that accounted for the effect of age by allowing the baseline rate to vary and adjusted for physical activity (linear term), BMI (linear term), cigarette smoking (current, past and never), living alone (yes, no), postmenopausal hormone use (yes, no), aspirin use (yes, no), education (less than high school, high school and university), family history of MI before 60 years (yes, no), self-reported history of hypertension (yes, no), self-reported history of high cholesterol (yes, no), total energy intake (linear term), alcohol intake (linear term), fibre intake (linear term), saturated fat (linear term), polyunsaturated fat (linear term) and protein (linear term). The dietary GI models were additionally adjusted for carbohydrate (linear term) to estimate the effect of varying the quality of the carbohydrate while holding the quantity constant. As a sensitivity analysis, we also estimated the RR associated with dietary GI without adjustment for carbohydrate. We tested for linear trend by entering the median in each quartile as a continuous variable. We performed additional analyses examining the association of available carbohydrate with MI events using models adjusted for the covariates listed earlier.

We examined the associations of dietary GI and GL with MI events in subpopulations defined by BMI (<25 *v.* ≥25 kg/m^2), by physical activity (active (≥40 min/d walking or bicycling

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Table 1. Baseline characteristics by quartile of dietary glycaemic index and load

(Mean values and standard deviations or percentage)

	Quartile dietary glycaemic index								Quartile dietary glycaemic load							
	1		2		3		4		1		2		3		4	
	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD	Mean	SD
Age (years)	60·2	8·8	60·5	8·8	61·8	9·1	64·0	9·4	59·9	8·9	60·8	8·9	62·0	9·1	63·7	9·3
Physical activity MET (h/d)	42·3	4·7	42·4	4·7	42·6	4·7	42·4	4·9	42·0	4·7	42·3	4·7	42·6	4·8	42·8	4·9
BMI (kg/m^2)	25·0	3·9	24·9	3·8	24·9	3·9	25·0	4·1	24·8	4·0	24·9	3·8	24·9	3·8	25·2	4·0
Cigarette smoking (%)																
Never	47·7		53·7		56·8		56·9		43·4		52·9		57·7		61·3	
Past	27·3		24·5		21·7		18·4		25·8		24·1		22·1		19·8	
Current	24·9		21·8		21·5		24·7		30·8		23·0		20·2		18·9	
Living alone (%)	25·8		21·8		22·0		26·8		25·2		22·4		22·7		26·2	
Post menopausal hormone use (%)	54·5		53·3		51·0		47·4		52·5		52·3		51·9		49·5	
Aspirin use (%)	43·7		43·6		44·0		41·9		44·8		43·8		42·4		42·1	
Education (%)																
Less than high school	64·3		68·9		76·0		85·2		66·4		71·2		75·8		81·1	
High school	9·5		9·1		7·5		5·7		9·5		8·5		7·5		6·4	
University	26·2		22·0		16·46		9·1		24·1		20·37		16·7		12·56	
Family history of myocardial infarction (%)	17·6		17·0		16·9		16·6		17·4		16·4		17·2		17·1	
Hypertension (%)	18·3		19·0		20·7		21·9		16·9		19·4		20·7		22·8	
High cholesterol (%)	7·1		8·2		7·8		8·3		5·8		7·1		8·4		10·2	
Energy intake (kJ/d)	7188	2268	7238	2075	7385	2151	7297	2280	7376	2406	7217	2100	7222	2038	7293	2218
Alcohol (g/d)	5·6	6·4	4·7	5·3	3·8	4·6	2·6	3·9	6·0	6·8	4·6	5·1	3·6	4·2	2·4	3·4
Fibre (g/d)†	22·4	6·0	22·6	5·1	22·1	5·1	21·1	5·6	19·0	4·5	21·6	4·5	23·1	4·9	24·5	6·3
Saturated fat (g/d)†	27·5	6·7	26·9	6·0	27·3	6·1	27·6	6·6	32·8	6·1	28·4	4·7	25·9	4·5	22·1	4·7
Polyunsaturated fat (g/d)†	7·9	2·1	8·1	1·8	8·1	1·8	8·0	1·8	8·4	2·3	8·2	1·8	8·0	1·6	7·4	1·6
Carbohydrate (g/d)†	203	26	210	24	212	25	218	28	180	17	204	11	218	11	241	17
Protein (g/d)†	76·7	12·3	71·7	10·2	68·9	9·6	64·5	9·8	78·5	12·6	72·2	9·2	68·6	8·4	62·7	8·7
Dietary glycaemic index	68·6	3·1	73·3	0·8	76·1	0·9	80·5	2·4	71·1	4·8	73·8	3·7	75·5	3·6	78·2	3·9
Dietary glycaemic load†	140	20	154	18	162	19	176	24	128	12	150	4	164	4	188	15

† Values are adjusted for energy using the residuals method.

and ≥1 h/week exercising) *v.* inactive)[37] and by fibre intake (<18·4 g/d (quartile 1) *v.* ≥18·4 g/d (quartiles 2–4)). We tested whether the associations varied across subpopulations using a likelihood ratio test with four df. We tested for violation of the assumption of proportional hazards by entering the product of the exposures and the natural logarithm of time in the models; we did not find evidence for violation of the assumption of proportional hazards. Statistical analyses were performed using Statistical Analysis Systems version 9.1 (SAS Institute, Cary, NC, USA). A two-sided *P*-value <0·05 was considered statistically significant.

Results

Over 9 years of follow-up, 1138 women were hospitalised or died due to a first MI. Compared with women with dietary GI or GL in the lowest quartile, women with higher dietary GI or GL were on average older and were more likely to have a history of hypertension, to have a less than high school education and to be a nonsmoker (Table 1). They consumed less alcohol and less protein than women with low dietary GI or GL. In addition, women with high dietary GL were more likely to have a history of high cholesterol and consumed more fibre and less saturated and polyunsaturated fat than women with lower dietary GL.

There was a modest association between dietary GI and incidence of MI in age-adjusted analyses (RR comparing top to bottom quartile were 1·16, 95 % CI 0·98, 1·37, *P*-trend=0·06), which was attenuated in multivariable-adjusted models (RR comparing top to bottom quartile were 1·12, 95 % CI 0·92, 1·35, *P*-trend=0·24; Table 2). Results were similar when we did not adjust for carbohydrate intake. RR across quartiles were 1 (reference), 1·05 (95 % CI 0·88, 1·27), 1·10 (95 % CI 0·92, 1·32) and 1·11 (95 % CI 0·92, 1·34, *P*-trend=0·27). There was a suggestion of a direct association between dietary GL and incidence of MI in multivariable-adjusted models, which was not statistically significant (RR comparing top to bottom quartile were 1·22, 95 % CI 0·90, 1·65, *P*-trend=0·23). RR across quartiles of carbohydrate intake were 1 (reference), 0·90 (95 % CI 0·73, 1·11), 0·90 (95 % CI 0·70, 1·17) and 0·95 (95 % CI 0·67, 1·35, *P*-trend=0·82).

Although tests of interaction were not statistically significant, there was a trend towards an association of dietary GI and GL with rates of MI in overweight women (BMI ≥ 25 kg/m^2) but not in normal weight (BMI < 25 kg/m^2) women (*P*-interaction=0·20 for dietary GI, *P*-interaction=0·14 for dietary GL; Table 3). However, the associations of dietary GI and GL with MI appeared stronger in active women, though the differences were not statistically significant. The RR comparing top to bottom quartile of dietary GI in the active women were 1·29 (95 % CI 0·86–1·95, *P*-trend=0·22) and 1·06 (95 % CI 0·85–1·31, *P*-trend=0·61) among inactive women (*P*-interaction=0·18). The RR comparing top to bottom quartile of dietary GL were 1·42 (95 % CI 0·72, 2·79, *P*-trend=0·38) in the active women and 1·14 (95 % CI 0·82, 1·60, *P*-trend=0·46) in inactive women (*P*-interaction=0·40). The association between dietary GI and MI was similar across levels of fibre intake (RR comparing top to bottom quartile among those with fibre <18·4 g/d were 1·05, 95 % CI 0·74, 1·49, *P*-trend=0·73; RR comparing top to bottom quartile among those with fibre ≥ 18·4 g/d were 1·14, 95 % CI 0·91, 1·42, *P*-trend=0·28; *P*-interaction=0·53). The association between dietary GL and MI also did not vary significantly by fibre intake (RR comparing top to bottom quartile among those with fibre <18·4 g/d were 0·85, 95 % CI 0·49, 1·48, *P*-trend=0·63; RR comparing top to bottom quartile among those with fibre ≥ 18·4 g/d were 1·41, 95 % CI 0·98, 2·05, *P*-trend=0·09; *P*-interaction=0·39).

Discussion

In the present study of middle-aged and older women, we did not find statistically significant associations of dietary GI and

Table 2. Dietary glycaemic index, dietary glycaemic load and incidence of myocardial infarction (Ratio rate and 95 % confidence intervals)

				Age-adjusted		Multivariable-adjusted	
	Median	Cases	Person (years)	Incidence rate ratio	95 % CI*	Incidence rate ratio	95 % CI†
Dietary glycaemic index							
Quartile 1	69·5	229	78 498	1	Reference	1	Reference
Quartile 2	73·3	246	78 575	1·04	0·87, 1·25	1·06	0·88, 1·27
Quartile 3	76·1	295	77 914	1·10	0·93, 1·31	1·11	0·92, 1·33
Quartile 4	79·9	368	76 515	1·16	0·98, 1·37	1·12	0·92, 1·35
P-trend				0·06		0·24	
Dietary glycaemic load							
Quartile 1	131	229	78 423	1	Reference	1	Reference
Quartile 2	150	264	78 187	1·06	0·89, 1·27	1·14	0·93, 1·39
Quartile 3	164	296	77 858	1·07	0·90, 1·27	1·18	0·94, 1·50
Quartile 4	184	349	77 035	1·09	0·92, 1·29	1·22	0·90, 1·65
P-trend				0·34		0·23	

* Cox proportional hazards model accounting for age.

† Additionally adjusted for education (less than high school, high school and university), BMI (linear term), physical activity (linear term), cigarette smoking (current, past and never), living alone (yes, no), postmenopausal hormone use (yes, no), aspirin use (yes, no), total energy intake (linear term), alcohol intake (linear term), fibre intake (linear term), saturated fat (linear term), polyunsaturated fat (linear term), protein (linear term), carbohydrate (linear term, dietary glycaemic index only), family history of myocardial infarction before 60 years (yes, no), self-reported history of hypertension (yes, no) and self-reported history of high cholesterol (yes, no).

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Table 3. Dietary glycaemic index, dietary glycaemic load and incidence of myocardial infarction by BMI
(Ratio rate and 95 % confidence intervals)

	Normal weight		Overweight		
	Incidence rate ratio	95 % CI*	Incidence rate ratio	95 % CI*	*P*-interaction
Dietary glycaemic index					
Quartile 1	1	Reference	1	Reference	
Quartile 2	0·94	0·73, 1·23	1·19	0·91, 1·54	
Quartile 3	0·99	0·76, 1·28	1·23	0·94, 1·60	0·20
Quartile 4	1·03	0·79, 1·34	1·20	0·91, 1·58	
P-trend	0·68		0·22		
Dietary glycaemic load					
Quartile 1	1	Reference	1	Reference	
Quartile 2	0·99	0·74, 1·32	1·34	1·00, 1·80	
Quartile 3	1·11	0·80, 1·53	1·30	0·92, 1·85	0·14
Quartile 4	1·07	0·70, 1·62	1·45	0·93, 2·25	
P-trend	0·67		0·16		

* Normal weight defined as BMI $<25\,kg/m^2$; Overweight defined as $\geq 25\,kg/m^2$. Cox proportional hazards model accounting for age and adjusted for education (less than high school, high school and university), BMI (linear term), physical activity (linear term), cigarette smoking (current, past and never), living alone (yes, no), postmenopausal hormone use (yes, no), aspirin use (yes, no), total energy intake (linear term), alcohol intake (linear term), fibre intake (linear term), saturated fat (linear term), polyunsaturated fat (linear term), protein (linear term), carbohydrate (linear term, dietary glycaemic index only), family history of myocardial infarction before 60 years (yes, no), self-reported history of hypertension (yes, no) and self-reported history of high cholesterol (yes, no).

GL with MI. However, women in the top quartile of dietary GI had a nonsignificant 12 % higher rate of MI than those in the bottom quartile and women in the top quartile of dietary GL had a nonsignificant 22 % higher rate of MI than those in the bottom quartile. The associations may have been stronger in overweight women. The results are consistent with those observed in other populations of women.

In the Nurses' Health Study, women with dietary GI in the highest quintile had a 31 % higher rate of CHD, and women with dietary GL in the highest quintile had a 98 % higher rate of CHD[3]. The highest compared to the lowest quartile of dietary GI and GL was associated with 44 % higher rates of CHD in a study of Dutch women[23]. The associations tended to be stronger in heavier women in both the studies. In contrast, the two previous prospective studies of men did not find associations of dietary GI or GL with CHD[24,26]. The associations between dietary GI and GL and MI appeared to be stronger in women than in men in a case-control study conducted in Italy[25], but to our knowledge the interaction between dietary GI and GL and sex has not been evaluated in a prospective setting.

In the present study, the median dietary GL ranged from 131 in the lowest quartile to 184 in the highest. By comparison, the median dietary GL ranged from 117 in the lowest quintile to 206 in the highest in the Nurses' Health Study[3] and from 78·5 in the lowest quartile to 121·8 in the highest using a glucose standard in a study of Dutch women (approximately 112 and 174 converted to a white bread standard)[23]. It is possible that the limited range of dietary GL in the present study prevented us from detecting a significant association. Populations also vary in which foods contribute most to dietary GL. Boiled or mashed potatoes and cold breakfast cereals were the most important contributors to dietary GL in the Nurses' Health Study[3]; among Swedish men, white bread contributed the most to dietary GL, followed by boiled potatoes, crispbread and whole grain bread[34].

Experimental studies comparing diets with high and low GI or GL support a link with CVD. Feeding studies have shown numerous metabolic effects of high-GI or -GL diets that could increase the risk of CVD including alternations in lipid profiles[4–12], inflammation[11,13,14] and insulin resistance[4,11]. The data on the effects of GI and GL on HDL cholesterol and TAG concentration suggest a possible explanation for the differences observed in men and women[24]. Diet trials have suggested that the decrease in HDL cholesterol and increase in TAG concentration caused by increased carbohydrate consumption may be greater in women than in men[38]. Additionally, elevated TAG concentration may be a stronger cardiovascular risk factor for women than for men[39].

There are several important limitations of the present study. Our assessment of medical history and other covariates was based on self-report, which is inherently less reliable than clinical measurement. Correlations between the FFQ and weighed diet records were 0·62 for dietary GI and 0·77 for dietary GL in a population of Swedish men from the same region[34]. However, dietary GI and GL were measured using FFQ with a simple procedure to match FFQ items to values in the published tables. A more sophisticated algorithm has been described, which is based on the frequency of consumption of the foods underlying the queried items[40]. Using questionnaires to assess diet resulted in some exposure misclassification. If the misclassification of diet was unrelated to MI incidence, the results would likely be biased towards the null. However, this assumption was not verifiable with available data. As with all observational studies, we cannot rule out residual or unmeasured confounding that could account for the association of dietary GI and GL with MI.

In summary, there was no statistically significant association of dietary GI or GL with MI in women in this middle-aged and older population. However, there was a suggestion of a higher rate of MI among women with high dietary GI or GL, which was consistent with previous reports.

Acknowledgements

Supported in part by grants from the Swedish Research Council/Committee for Infrastructure and the Committee for Medicine, a grant from the Swedish Foundation for International

Cooperation in Research and Higher Education and National Institutes of Health grant F32 HL091683 (supporting E. B. L.). The authors have no conflicts of interest to disclose. E. B. L. and A. W. will serve as guarantors for the contents of the present paper. E. B. L., M. A. M. and A. W. were responsible for the study concept and design, the statistical analysis and interpretation of data and drafting of the manuscript. A. W. was responsible for the data collection.

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