Null Results in Brief

No Association Between Dietary Glycemic Index or Load and Pancreatic Cancer Incidence in Postmenopausal Women

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Introduction

Recently, diets with high glycemic index (GI) and glycemic load (GL) values have been implicated in the etiology of chronic diseases owing to their potential to increase postprandial glucose and insulin (1). The GI reflects the relative increase in blood glucose per gram of carbohydrate for carbohydrate-containing foods compared with a control food, usually white bread or glucose. GL further standardizes the GI for the amount of carbohydrate in a particular food by multiplying GI by the grams of carbohydrate in the food (2). The diet of an individual can be characterized by calculating an overall value for dietary GI or GL.

We hypothesized that high dietary GI and GL are associated with increased risk of pancreatic cancer, which could occur through mitogenic effects of insulin and insulin growth factors on the exocrine pancreas (3, 4). One study has examined the relation between pancreatic cancer and high GI and GL diets, finding a positive association between a high dietary GI and pancreatic cancer incidence that was stronger in women who were sedentary, overweight, or both (5). We examined these associations in a group of postmenopausal women in the Iowa Women’s Health Study cohort.

Materials and Methods

Subjects. Iowa Women’s Health Study is a prospective study of dietary and lifestyle risk factors for chronic disease in 41,836 women who were 55 to 69 years old at baseline in 1986. Subjects were sampled from the 1985 Iowa driver’s license list and responded to a mailed questionnaire. Baseline information was collected on education, physical activity, individual and family medical history, anthropometric variables, and smoking history. In addition, the women also completed a 126-item food frequency questionnaire (6).

GI and GL values were calculated as previously described (7) according to the following formulas:

Average daily glycemic index:

\[
\frac{\sum [\text{servings of food per day}]}{\text{(carbohydrate content of food)}} \times (\text{glycemic index})
\]

Average daily glycemic load:

\[
\sum [(\text{servings of food per day}) \times (\text{carbohydrate content of food})] \times (\text{glycemic index})
\]

These variables were adjusted for energy intake using the residual method (8).

Nondietary covariate information obtained at baseline included diabetes status, physical activity level, body mass index, smoking status, pack-years, and multivitamin use. Body mass index and diabetes status were determined as previously described (7). A woman’s physical activity level was categorized from three questions regarding frequency of moderate and vigorous activities and dichotomized as low or moderate/high. Pack-years was calculated as the product of reported number of cigarettes smoked per day and the number of years the subject smoked. Regular multivitamin use was categorized as yes or no.

Subject Follow-up. Subjects were followed through the administration of four follow-up questionnaires (1987, 1989, 1992, and 1997) and through annual determination of vital status through linkage to Iowa death records and the National Death Index. Incident cases of cancer of the exocrine pancreas were ascertained from 1986 to 2002 through linkage with the Iowa Cancer Registry.

Data Analysis. We excluded participants who at baseline had implausible energy intake (>5,000 or <600 calories/d), had ≥30 missing responses on the food frequency questionnaire, reported previous cancer (except nonmelanotic skin cancer), or were premenopausal. We also excluded four pancreatic cancer cases with nontypical tumors. After exclusions, 34,699 participants were considered at risk and 190 developed incident pancreatic cancer.

Data analysis was done using SAS version 8 (SAS Institute, Inc., Cary, NC). Poisson regression was used to calculate crude incidence rates for potential independent risk factors. We used Cox proportional hazards regression to calculate unadjusted and adjusted hazard ratios (HR) and 95% confidence intervals (CI) according to the average daily GI or GL. A total of 33,551 subjects, including 181 cases, had a complete set of covariates for adjusted models. Independent risk factors, as determined by crude rate ratios, were included in the adjusted models if they improved the model fit as judged by the likelihood ratio \( \chi^2 \) test (\( P < 0.05 \)). Using this criterion, we adjusted for age, smoking status,
pack-years, diabetes, and multivitamin use. A P value for trend was calculated using an ordinal variable coded 1 to 4 for successive quartiles. Tests for interaction on the multiplicative scale were done using the likelihood ratio $\chi^2$ test, comparing models with and without the inclusion of the cross-product of the GI/GL quartile and the covariate being tested. Our power was 80% to detect a significant HR of 1.8 in each quartile compared with the reference GI/GL quartile.

### Results

Incidence of pancreatic cancer was higher in those who were 65 to 69 versus 55 to 64 (47 versus 32 of 100,000 person-years), diabetic versus nondiabetic (68 versus 35 of 100,000 person-years), current smokers versus nonsmokers (60 versus 28 of 100,000 person-years), and multivitamin nonusers versus users (40 versus 28 of 100,000 person-years). There was no association between pancreatic cancer incidence and body mass index, physical activity, or any dietary variables examined including fruits and vegetables, meat, fat, carbohydrates, fiber, coffee, energy-intake, and alcohol (data not shown).

There was no increased hazard of pancreatic cancer associated with high dietary GI or GL for the fourth compared with the first quartile, with or without adjustment for covariates, with adjusted HRs of 1.08 and 0.87 for GI and GL, respectively (Table 1). We also tested whether the HR for dietary GI and GL differed by physical activity level (low versus medium/high) or by BMI category (cut point 25 kg/m²). Tests for interaction on the multiplicative scale were not significant (data not shown).

### Strengths, Limitations, and Conclusions

This study has several strengths, including a prospective design and long follow-up time. Limitations include the use of a single questionnaire to collect dietary information and a moderate number of case subjects.

We did not find evidence to support the hypothesis that high dietary GI or GL increases the risk of pancreatic cancer. Our findings are in contrast to those previously reported by the Nurses' Health Study (6). Although an increased risk of pancreatic cancer associated with high GI or GL diets seems biologically plausible, the data from our study do not provide support for such an etiologic link.

### References

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