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Carbohydrates Raise Blood Sugar Levels and Increase the Risk of Developing Pancreatic Cancer

Johrj Jefresz*

Department of Endocrinology, University of Brazil, Brazil

Abstract

Differences across quartiles of Glycemic load, pancreatic cancer, Angeles Multi-ethnic Cohort, dietary carbohydrates, were tested with the Cochran-Aromatase test for trend for categorical variables and with the t test for slope in linear regression models of mean values on GL for continuous variables. Person-times were determined by beginning with the date of cohort entry, defined as the date of questionnaire completion, and ending at the earliest of the following dates: date of pancreatic cancer diagnosis, date of death. Tests based on residuals showed no evidence that proportional hazard assumptions were violated for any analysis. We present models including both sexes, because there was no evidence of interaction by sex, after adjustment for sex and follow-up time on study from baseline as strata variables, to allow for different baseline hazard rates. A separate analysis suggested some differences between RRs across 3 follow-up time strata, although the differences were not statistically significant. Quartiles of nutrient and food intakes were based on the distribution of the

Keywords: Glycemic; Cochran-Aromatase; Pancreatic cancer; Ethnic Cohort; Carbohydrates

Introduction

Pancreatic cancer is the most fatal cancer in adults, with a 5-y survival rate. More than 33 000 new pancreatic cancer cases were expected in the United States in 2006. Because of the poor prognosis and the minimal effect of conventional treatment methods, it is important to focus on prevention of this disease. Cigarette smoking is the most important etiologic factor yet identified current smokers have a pancreatic cancer risk approximately double that of non-smokers. The risk attributable to smoking has been estimated. Obesity and a family history of pancreatic cancer have also been associated with the disease [1]. Other risk factors include increasing age, male sex, and Native Hawaiian or African American race-ethnicity. Dietary factors may also be important. The hypothesized mechanism involves insulin and this hypothesis has recently been supported by findings linking higher insulin concentrations and insulin resistance to pancreatic cancer risk. Postprandial blood glucose concentrations are influenced by food consumption. Different foods cause different absolute peaks in blood glucose and different rates of change in blood glucose concentrations during the period after consumption . An empirical measure of blood glucose response after consumption of a specific food, the Glycemic index, has been developed to classify foods according to their postprandial Glycemic effects and, hence, according to their effects on blood insulin concentrations [2]. Because the amount of carbohydrate in a diet is a major determinant of blood glucose concentrations, the GI of a food item is multiplied by its carbohydrate content to derive the Glycemic load (GL) per 100-g intake of the food. The GL of a diet can then be calculated from the amounts and types of foods consumed. On the basis of the assumption that glucose metabolism plays a role in the development of pancreatic cancer, we hypothesized that a high dietary GL is positively associated with the risk of pancreatic cancer. Both GI and GL have been investigated with respect to pancreatic cancer in prospective studies and the results have been inconsistent. We analyzed 8-y prospective data from the Multi-ethnic Cohort Study to investigate associations between dietary GL and pancreatic cancer risk. Furthermore, we examined various carbohydrates and sugars, especially added sugars, to fully investigate the associations [3].

Method

The percentage of men increased across quartiles of dietary GL. Mean BMI was slightly elevated in the last quartile of GL only. All dietary variables of interest were positively associated with GL. Because all of these dietary variables are likely to be highly associated with each other, we calculated Spearman correlation coefficients between GL and the intakes of total carbohydrates, sucrose, fructose, total sugars, and added sugars. During follow-up, 434 incident pancreatic cancer cases occurred in the cohort. GL was not associated with pancreatic cancer risk in the overall cohort. The RR for fructose was significantly elevated in the highest quartile [4]. Although the risks for the intakes of total sugars as well as sucrose were highest in the fourth quartile and were suggestive of an association, neither the point estimates nor the test for trend were significant. Because obesity can be a determinant of insulin resistance, we stratified our analysis to examine whether the effects of GL or carbohydrate intakes varied by BMI. The risks of pancreatic cancer were seen in the overweight and obese group than in the normal-weight group in the top quartiles of intakes of all dietary variables, and there were stronger trends across quartiles. A significant interaction was evident for sucrose only [5]. The use of calibration-adjusted nutrient intakes gave similar results. Additional adjustment for total physical activity did not alter the findings, although a further stratification by level of physical activity among the overweight or obese participants suggested a higher RR for those with a higher level of physical activity than for those with a low level of physical activity. Fruit and juices combined were the largest contributor to fructose intake in the cohort, followed by non-diet sodas. To confirm our findings for fructose, we analyzed the intakes of several food groups—soda, fruit

***Corresponding author:** Johrj Jefresz, Department of Endocrinology, University of Brazil, Brazil, E-mail: johrjjrfresz921@gmail.com

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juices, total fruit, citrus fruit, and yellow-orange fruit—in disease risk models.

Result

The combined intake of fruit and juices and the intake of fruit alone were associated with increase in the risk of pancreatic cancer in the overall cohort. Among overweight or obese participants, total fruit and juice intake was associated with a 51% higher risk, but weaker associations were seen when fruit and fruit juices were separated. When total fruit intake was divided into citrus fruit or yellow-orange fruit, the intake of yellow-orange fruit was positively associated with pancreatic cancer risk among normal-weight participants. On statistical evidence of an interaction of any fruit or juice variable with BMI was present. A high intake of regular sodas was not associated with pancreatic cancer risk in the overall cohort or in the subgroup of obese or overweight participants [6]. To determine whether the association was limited to fruit, we also investigated jointly the trends for fructose from fruit, soda, and other sources. All trends were positive and non significant. Therefore, the association with fruit may be due to the fructose content of fruit or another factor. Because smoking is the most well-established risk factor for pancreatic cancer, we stratified our analysis according to smoking status. Overall, there was little evidence of effect modification by smoking status (never, former, or current), although the greater risk associated with fruit intake was most apparent among never smokers

Discussion

The finding of a greater risk with higher fruit and juices intake was surprising, and it merits some discussion. Fruit, most often in combination with vegetables, generally is thought to have beneficial effects in terms of cancer prevention at various sites, including the pancreas. Prospective studies have reported on fruit intake and pancreatic cancer risk, and none detected a significant association, either positive or inverse. A recent study from Sweden included pancreatic cancer cases in a cohort of men and women. When the highest and lowest quartiles were compared [7], overall fruit intake was not significantly associated with pancreatic cancer risk, nor was citrus fruit intake. Participants in the highest quartile consumed ≥2.5 servings. A study in Finnish smokers found a non-significant the highest consumption of all fruit and berries in an analysis of cases and non-cases.. Four studies with mortality as the outcome did not find any associations. One of these studies included the largest number of pancreatic cancer deaths thus far, although the dietary assessment was very limited and included only one question about consumption of citrus fruit or juices. Our study, in contrast to these previously published studies, examined the largest number of incident pancreatic cancer cases by using a comprehensive and detailed dietary assessment, which enabled us to detect statistically significant associations of smaller magnitude. Because the intake of fruit and juices combined is the largest contributor to fructose intake in our cohort, the increase in risk with high fruit intake may be explained by fructose and total sugars, both of which are highly correlated with fruit intake. Models with fructose separated by sources showed similar associations for all sources [8, 9]. An effect of fruit other than through sugars, therefore, cannot be ruled out at this point. Furthermore, although the null association with soda does not seem to support this conjecture, underreporting of the intake of low-nutrient-density beverages such as soda may attenuate the true association.

The hypothesized mechanism linking an impaired glucose metabolism or diabetes mellitus to pancreatic cancer involves insulin. Insulin can promote tumors development by inhibiting apoptosis and stimulating cell proliferation, and it has been argued that insulin acts as a promoter for pancreatic carcinogenesis. Our findings support this hypothesis to some extent, because slightly elevated risks, especially those with sucrose, were seen in overweight or obese participants who may already have an underlying degree of insulin resistance. We reported a higher pancreatic cancer risk in obese men but not in obese women in the Multi-ethnic Cohort Study, and evidence in the literature for a positive association between BMI and pancreatic cancer risk is fairly consistent .

As discussed in the literature, the GI is a controversial concept. It has been pointed out that the GIs of foods vary by types, by processing or preparation, and by combinations of foods consumed together, which renders the actual determination of a GI value for a specific food difficult [10]. In addition, the Glycemic response after ingestion of a food does not necessarily predict the insulin response. Most important for our study, it has been debated whether the GI or GL is accurate when using dietary intakes collected with an, because some questionnaire items may group foods with differing GL values. However, the quantitative FFQ used for the Multi-ethnic Cohort Study is lengthy and thus less likely than most to group dissimilar foods.

In our study, we used GL rather than GI because the blood glucose response after ingestion of a food is determined by both its carbohydrate content and its GI [11], and GL combines the 2. We further explored the relations between variables by calculating correlation coefficients. Indeed, the correlation between GL and carbohydrate intake was very high, and we found almost identical RRs for pancreatic cancer. We suggest that dietary GL in general may not add important new information about the quality of carbohydrates in the diet of our cohort participants. However, models with fructose separated by sources showed similar associations for all sources. An effect of fruit other than through sugars, therefore, cannot be ruled out at this point. Furthermore, although the null association with soda does not seem to support this conjecture, underreporting of the intake of low-nutrient-density beverages such as soda may attenuate the true association [12]. Because the intake of fruit and juices combined is the largest contributor to fructose intake in our cohort, the increase in risk with high fruit intake may be explained by fructose and total sugars, both of which are highly correlated with fruit intake.

Conclusion

High fructose and sucrose intakes may play a role in pancreatic cancer etiology. Conditions such as overweight or obesity in which a degree of insulin resistance may be present may also be important. The finding of a greater risk with higher fruit and juices intake was surprising, and it merits some discussion. Fruit, most often in combination with vegetables, generally is thought to have beneficial effects in terms of cancer prevention at various sites, including the pancreas. Prospective studies have reported on fruit intake and pancreatic cancer risk, and none detected a significant association, either positive or inverse. When the highest and lowest quartiles were compared, Participants in the highest quartile consumed ≥2.5 servings of fruit/d. A study in Finnish smokers found a no significant RR for the highest consumption of all fruit and berries in an analysis. Median consumption for cases and non-cases was ≈100 g/d. Four studies with mortality as the outcome did not find any associations. One of these studies included the largest number of pancreatic cancer deaths thus far, although the dietary assessment was very limited and included only one question about consumption of citrus fruit or juices. Our study, in contrast to these previously published studies, examined the largest number of incident pancreatic cancer cases by using a comprehensive and detailed dietary assessment, which enabled us to detect statistically significant associations of smaller magnitude.

Acknowledgement

None

Conflict of Interest

None

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