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Consumption of sweet foods and breast cancer risk: a case-control study of women on Long Island, New York

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Abstract

Several epidemiologic studies have reported a positive association between breast cancer risk and high intake of sweets, which may be due to an insulin-related mechanism. We investigated this association in a population-based case-control study of 1434 cases and 1440 controls from Long Island, NY. Shortly after diagnosis, subjects were interviewed in-person to assess potential breast cancer risk factors, and self-completed a modified Block food frequency questionnaire (FFQ), which included 11 items pertaining to consumption of sweets (sweet beverages, added sugars and various desserts) in the previous year. Using unconditional logistic regression models, we estimated the association between consumption of sweets and breast cancer. Consumption of a food grouping that included dessert foods, sweet beverages and added sugars was positively associated with breast cancer risk [adjusted odds ratio (OR) comparing highest to lowest quartile: 1.27, 95% confidence interval (CI): 1.00-1.61]. The OR was slightly higher when only dessert foods were considered (OR: 1.55, 95% CI: 1.23-1.96). The association with desserts was stronger among premenopausal women (OR: 2.00, 95% CI: 1.32-3.04) than postmenopausal women (OR: 1.40, 95% CI: 1.07-1.83), although the interaction with menopause was not statistically significant. Our study indicates that frequent consumption of sweets, particularly desserts, may be associated with an increased risk of breast cancer. These results are consistent with other studies that implicate insulin-related factors in breast carcinogenesis.

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Keywords

sweets consumption; insulin; breast cancer; estrogen receptor; progesterone receptor

Introduction

Research suggests that diets high in refined sugars, sweets and desserts may increase a woman's risk for breast cancer (1-6) although several null findings (7-9) have resulted in a lack of consensus on the issue. Dietary behaviors that result in elevated blood glucose levels, such as frequent consumption of refined carbohydrates, consequently increase plasma insulin. Hyperinsulinemia may be a risk factor for breast cancer as insulin is mitogenic, thereby encouraging cellular proliferation and promoting tumor growth; excess insulin is also indirectly related to increased levels of free estrogen through inhibition of the production of sex-hormone binding globulin (10). Chronically elevated insulin may be one of the pathways involved in the observed associations between energy balance and breast carcinogenesis as evidenced by the results from a recent meta-analysis on C-peptide and insulin levels and breast cancer risk (11), although the positive association was limited to case-control studies. Thus recent research has focused on evaluating lifestyle factors, such as dietary behaviors, that may influence insulin exposure.

We evaluated the association between increasing consumption of sweet foods and breast cancer risk in a large, population-based case-control study of breast cancer among women residing in Long Island, NY. We also examined the potential for effect modification by exposures known to be associated with hyperinsulinemia or related hormonal pathways.

Methods

The Long Island Breast Cancer Study Project (LIBCSP) is a population-based case-control study of breast cancer conducted in Nassau and Suffolk counties in New York (12). The study was approved by the Institutional Review Board of participating institutions.

Study Population

Cases were English speaking adult women with a first primary *in situ* or invasive breast cancer diagnosed between 1996-1997. Potentially eligible women were identified through pathology departments of participating hospitals and their physicians were contacted to confirm the diagnosis and obtain permission to contact the patients for participation in the study. Controls were frequency matched to the expected distribution of cases in 5-year age groups in 1996-1997; potentially eligible controls under the age of 65 were identified by random digit dialing, while those over age 65 were identified from the Health Care Finance Administration rosters. A total of 1,508 eligible cases (82.1%) and 1,556 eligible controls (62.7%) agreed to participate in the study. Participants ranged in age from 20 to 98 years, and 94% were white and 4% were African American.

Exposure Assessment

Signed informed consent was obtained for all subjects prior to data collection. Risk factor information was assessed by structured questionnaire, which was administered by trained interviewers during in-home visits. Information collected included menstrual and reproductive history, exogenous hormone use, cigarette smoking, alcohol use, non-steroidal anti-inflammatory (NSAID) use, anthropometric measures, and physical activity (http://epi.grants.cancer.gov/LIBCSP/projects/Questionnaire.html). Factors found to increase breast cancer risk in this study population have been previously published (12). For case women who signed a medical record release form (97.7%), tumor stage and hormone receptor status were ascertained from the medical records.

Approximately 98% of the respondents completed a self-administered modified Block food frequency questionnaire (FFQ) that took an average of approximately 30 minutes to complete (12). The FFQ assessed frequency and relative portion size for 101 food items in the year prior to the interview, with line items for sweet foods including desserts (ice cream, low-fat ice cream/frozen yogurt/frozen tofu/sherbet, pies, doughnuts and pastry, chocolate cake/brownies/cookies, chocolate candy), sweetened beverages (fruit drinks, regular sweetened soft drinks) and added sweeteners (sugar added to cereal, sugar added to coffee or tea).

Food groupings were developed to be consistent with previous studies (2, 3) and to help distinguish the effect of desserts, which are more calorie and sugar rich, from total intake of sweets. For each line item, we created a variable representing the number of average sized servings per week by multiplying the relative portion size (less then average=0.5, average=1.0, larger than average=2.0) by the number of servings per week reported. We summed these variables to define number of servings per week of food groups corresponding to total sweets (all items) as well as dessert-foods only.

To eliminate the influence of outliers we excluded those subjects where the log-transformed total energy intake was more than 3 standard deviations of the log-transformed mean (45 cases and 56 controls). We also eliminated those subjects with missing data on menopausal status (30 cases and 63 controls) yielding a final sample of 1,434 cases and 1,440 controls.

Statistical Analysis

Unconditional logistic regression (13), adjusting for age at reference date (date of diagnosis for cases, date of interview for controls) in 5-year categories, was used to estimate odds ratios (OR) and 95% confidence intervals (CI) for the association between sweets and breast cancer. Intake of total sweets and desserts were categorized according to quartiles of intake among all controls. Potential confounders included total energy (kcal, continuous), family history of breast cancer (yes/no), parity (nulliparous/parous), oral contraceptive use (ever/never), Body Mass Index one year prior to reference date (BMI, kg/m^2: <25, 25-29, >30), physical activity from menarche to reference date (0, 0.01-7.55, >7.55 METS), weight change since age 20 (categorized as <3kg increase, 3-14.4 kg increase, >14.4 kg increase), fruit and vegetable intake (0-14, >14 servings per week), meat intake (grams per day, continuous), alcohol use (ever/never), and smoking (ever/never). Only total energy intake

notably altered the effect estimates (>10% change), therefore our final models are adjusted for age and energy intake. Tests for linear trend were conducted using the continuous variable in the model and examining the p-value for the corresponding beta coefficient from the logistic regression model (14). We used multinomal logistic regression to examine if the effects differed by *in situ* (n =235) or invasive (n=1270) cancers by estimating multinomial models allowing the effect to vary by *in situ* or invasive type and separate models restricting the effect to be the same for both types. These models were then compared using a likelihood ratio test. All statistical analyses were conducted using Stata version 9.2 (College Station, TX).

Multiplicative interaction (14) between sweets consumption and select variables was assessed using the likelihood ratio test to compare models with and without interaction terms; these interactions were deemed significant for p-values < 0.10. Potential effect modifiers included factors thought to directly or indirectly affect endogenous insulin exposure: menopausal status, body mass index, weight change since age 20, physical activity and alcohol intake.

Results

As shown in table 1, we observed a modest increase in the risk of breast cancer with increasing frequency of consumption of total sweets among all women (OR comparing highest to lowest quartile: 1.27, 95% CI: 1.00-1.61). We noted a slightly higher effect among premenopausal women (OR: 1.43, 95% CI: 0.95-2.14) compared to postmenopausal women (OR: 1.24, 95% CI: 0.94-1.64), although the test for interaction was not statistically signficant. The observed association was stronger when only dessert foods were considered. Among all women, those in the highest quartile of dessert consumption showed a 55% increase in risk of breast cancer compared to those with the lowest quartile of intake (OR: 1.55, 95% CI: 1.23-1.96). We again found a stronger association among premenopausal women (OR: 2.00, 95% CI: 1.32-3.04) compared to postmenopausal women (OR: 1.40, 95% CI: 1.07-1.83), however effect modification by menopausal status was not significant. The observed patterns generally suggest that risk increases monotonically with increasing consumption although tests for linear trend were not statistically significant. In analyses that considered individual food items, no single sweet item appeared to be responsible for the associations noted with total sweets or desserts (data not shown). The multinomial logistic regression models comparing the effects between *in situ* and invasive cancers showed no significant difference in effect of total sweets (likelihood ratio p-value = 0.66) or desserts only (likelihood ratio p-value=0.71).

Results for effect modification by BMI, weight change since age 20, physical activity and alcohol consumption are presented in table 2. For total sweets intake, only physical activity was observed to modify the association with breast cancer risk (p for interaction: 0.01), with more active women apparently more susceptible to the negative effect of frequent sweets consumption (lowest strata of physical activity, OR: 0.71, 95% CI: 0.46-1.09; highest strata of physical activity, OR: 1.85, 95% CI: 1.25-2.74).

For dessert consumption, heterogeneity across strata of other factors that could potentially impact endogenous insulin levels was evident not only for physical activity, but also body size, adult weight gain and alcohol. For example, the association with breast cancer risk was essentially null across all levels of intake among inactive women, whereas among the most active women we observed a nearly doubling of risk comparing the highest to lowest quartile of dessert intake (OR: 1.91, 95% CI: 1.30-2.82). For body size, women with lower BMI exhibited a stronger association with dessert consumption compared to those with higher BMI (low BMI stratum, OR: 1.85, 95% CI: 1.32-2.57; high BMI stratum, OR: 1.39, 95% CI: 1.03-1.87; p for interaction: 0.06). Greater adult weight gain appears to somewhat attenuate the effect of increasing dessert consumption (among those with less than 3 kg gain, OR: 1.70, 95% CI: 1.02-2.81; among those with >14.4 kg gain, OR: 1.30, 95% CI: 0.92-1.83; p for interaction: 0.099) which is not observed when all sweet foods are considered. For alcohol, ever drinkers showed a significantly greater risk of breast cancer across all levels of dessert intake compared to those women who reported never drinking alcohol (p for interaction: 0.01).

Discussion

In a large, population-based case-control study of breast cancer, we found a positive association between consumption of sweet foods, especially desserts, and breast cancer risk among both pre- and post-menopausal women. The adverse effects may be stronger among women who are more physically active; for desserts, high risk subgroups may also include women who are leaner and those who reported ever having consumed alcohol.

Our results are consistent with other recent epidemiologic research (1-5). In a population-based case-control study of breast cancer among American women under age 45, Potischman (2) observed a 32% increase in risk of breast cancer among those who consumed the most sweets, as compared with the least. A 2005 report of an Italian hospital-based case-control study (3) also showed a 19% increase among those in the highest tertile of intake of dessert foods. In a Canadian case-control study, Lubin (5) reported a positive association with sweet desserts of a magnitude similar to what we observe in our data (OR highest to lowest tertile: 1.50). Some authors have postulated that these results could be due to other characteristics of these foods, such as fat content, however the associations for lower-fat foods, such as sweetened beverages and sugar intake (1, 4) would be more consistent with an insulin-related pathway. There may also be a synergistic effect of high-sugar and high-calorie foods on breast cancer development, which could explain the stronger effects for desserts.

Inconsistent findings between consumption of sweets and breast cancer in the epidemiologic literature may be due to differences in exposure definitions, which have included intake of sugars (1, 6), sweets and desserts (2-5), individual foods (3) or an overall dietary pattern that included high intake of sweets (7-9). The studies of individual foods (1, 3) or food groups (1-5) tended to show positive associations between sweets consumption and breast cancer incidence, while a similar association was not seen in those studies that evaluated overall dietary patterns that reflected frequent sweets consumption (7-9). Variations in exposure definitions used across studies may correspond to differences in underlying biologic effects.

Case-control studies that examined overall measures of dietary glycemic burden, such as total carbohydrate intake, glycemic index (GI) and glycemic load (GL), have generally shown a positive association with breast cancer risk (1, 8, 15). In contrast, many cohort studies (16-22), with a few exceptions (23-26), report null associations between total carbohydrate intake, GI, GL and breast cancer risk (27). It is possible that differential recall in case-control studies or the fact that prospective observation may alter usual behavior in cohort studies could explain some of these discrepancies. Alternatively, the inconsistent associations with cancer incidence in the epidemiologic literature may be explained by the use of the GI and/or the GL themselves, which is a controversial practice. Because these measures pertain only to individual foods and not combinations of foods (as they are typically eaten in a mixed meal), some researchers believe that they may not accurately reflect the glycemic burden of an individual diet (28). Further, the postprandial glycemic response to foods explains only about 23% of the variation in insulin levels (29), as noted by other authors (30). Analyses that account for food groupings and dietary patterns may identify exposures that affect a multitude of factors that affect disease risk more than when nutrients or characteristics of the foods are considered individually (31). Notably, a recent analysis that combined dietary pattern analysis with GI and GL showed that the dietary patterns identified (where sweet foods contributed the most to the factor loadings) were associated with increased breast cancer risk among premenopausal women more so than when the simple GI and GL were considered (8).

Our findings for effect modification by body size and physical activity are somewhat unexpected, given that a recent case-control study did not find similar patterns (3), although the discrepancy may be due to differences in study design. The previous study (3) was hospital-based whereas our study was population-based. Given that body size and physical activity are known to contribute to multiple chronic diseases, the distribution of these factors among a control sample that is population-based, rather than hospital-based, is likely to better represent the underlying distribution of the source population from which the cases are drawn (14). However, although the potential interactions between sweets intake and body size and/or physical activity appear plausible, our results on more pronounced effects among special subgoups of women need confirmation by other investigations.

In addition to functioning as a tumor promoter by itself, insulin increases the activity of insulin-like growth factor I (IGF-I), which also functions as a tumor promoter (10) and has been associated with breast cancer in several epidemiologic studies (32, 33). Our results for insulin-related dietary exposures are also consistent with other findings from the LIBCSP, which found an increased risk of breast cancer among pre-menopausal women with polymorphisms for IGF-I (34), which is in agreement with evidence for IGF-I in most other studies (33, 35).

Our analysis benefits from a large, population-based sample that includes both pre- and post-menopausal women and comprehensive assessment of anthropometric, lifestyle and dietary factors. One limitation is the potential for measurement error through use of a FFQ to ascertain usual diet. Although this is a concern, FFQs adequately rank dietary intakes (36), which is the approach we used for our analysis. In case-control studies the potential for bias due to differential recall is always present, where subjects with disease may recall

unhealthy behaviors with greater frequency compared to controls. Additionally, although the completion of the self-administered FFQ was over 98% among both case and control respondents, the response rate to the main questionnaire among controls was lower than expected, primarily among older women (12). Thus, there may be selection bias due to non-response in our data, but its potential influence would primarily affect our results among women 65 years of age of older. Selection bias among controls has the potential to induce a spurious association (37), yet our observed effects were more pronounced among premenopausal women, the group for whom response rates were about 80% and thus control selection was not an issue. Therefore, we believe it unlikely that our findings, particularly among younger women, are the result of selection bias.

In summary, our results suggest that high consumption of sweet foods, particularly desserts, may be positively associated with breast cancer risk, and the effect may be most pronounced among leaner and more physically active women. The implications of these findings may be that the greatest reduction in breast cancer risk involves regular physical activity (38) and maintenance of a healthy weight (39) combined with a diet low in sweets and desserts. These results lend additional evidence to the role of insulin in breast carcinogenesis and may highlight an area for potential intervention.

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Table 1

Age and energy adjusted OR for association between sweets consumption and breast cancer, for all women and separately by menopausal status, Long Island Breast Cancer Study Project, Long Island, NY, 1996-1997.

	All Women		Premenopausal		Postmenopausal	
Quartiles of Servings/week*	**Cases/Controls (n)**	**OR† (95% CI)**	**Cases/Controls (n)**	**OR† (95% CI)**	**Cases/Controls (n)**	**OR† (95% CI)**
Total Sweets						
<2.4	330/359	1.	67/89	1.	263/270	1.
2.4-7.3	369/359	1.17 (0.95-1.45)	105/121	1.21 (0.80-1.84)	264/238	1.18 (0.92-1.51)
7.4-14.1	375/362	1.24 (1.00-1.54)	139/121	1.68 (1.12-2.53)	236/241	1.08 (0.84-1.40)
>14.1	360/360	1.27 (1.00-1.61)	146/156	1.43 (0.95-2.14)	214/204	1.24 (0.94-1.64)
p trend		0.18		0.06		0.82
p interaction				0.21		
Desserts						
<1.1	299/357	1.	58/93	1.	241/264	1.
1.1-2.8	356/363	1.21 (0.98-1.50)	111/114	1.60 (1.05-2.44)	245/249	1.10 (0.85-1.41)
2.9-6.3	377/360	1.36 (1.09-1.69)	134/141	1.61 (1.07-2.43)	243/219	1.29 (0.99-1.67)
>6.3	402/360	1.55 (1.23-1.96)	154/139	2.00 (1.32-3.04)	248/221	1.40 (1.07-1.83)
p trend		0.15		0.10		0.66
p interaction				0.41		

*Among all controls.

†Adjusted for age at reference date and total energy intake.

Table 2

Age and energy adjusted OR for association between sweets consumption and breast cancer, by BMI in the year prior to reference date, physical activity from menarche to reference date and alcohol consumption, Long Island Breast Cancer Study Project, Long Island, NY, 1996-1997.

	Adjusted OR[†] (95% CI)				
	Total Sweets[*]				
	<2.4	**2.4-7.3**	**7.4-14.1**	**>14.1**	**p-value[§]**
BMI					
< 25.0	1.	1.42 (1.04-1.94)	1.26 (0.92-1.72)	1.29 (0.93-1.80)	0.28
>= 25.0	1.	1.00 (0.75-1.33)	1.24 (0.92-1.66)	1.27 (0.93-1.73)	
Weight change since age 20 yrs					
<3 kg gain	1.	1.03 (0.62-1.72)	1.29 (0.79-2.11)	1.01 (0.61-1.66)	
3-14.5 kg gain	1.	1.16 (0.83-1.61)	1.12 (0.79-1.58)	1.28 (0.90-1.82)	0.88
>=14.5 kg gain	1.	1.23 (0.88-1.72)	1.33 (0.96-1.86)	1.42 (1.00-2.02)	
Physical Activity (MET-hrs per week)					
0	1.	0.83 (0.54-1.28)	0.91 (0.60-1.38)	0.71 (0.46-1.09)	
0.01-7.54	1.	1.15 (0.80-1.65)	1.47 (1.02-2.12)	1.68 (1.15-2.44)	0.01
>7.54	1.	1.50 (1.04-2.16)	1.23 (0.85-1.79)	1.85 (1.25-2.74)	
Alcohol use					
Never	1.	1.05 (0.74-1.48)	1.13 (0.80-1.60)	1.08 (0.75-1.54)	0.68
Ever	1.	1.25 (0.96-1.63)	1.30 (0.99-1.71)	1.41 (1.06-1.88)	
	Desserts only[*]				
< 1.1		**1.1-2.8**	**2.9-6.3**	**>6.3**	**p-value[§]**
BMI					
< 25.0	1.	1.64 (1.19-2.25)	1.80 (1.30-2.48)	1.85 (1.32-2.57)	0.06
>= 25.0	1.	0.95 (0.71-1.28)	1.10 (0.82-1.47)	1.39 (1.03-1.87)	
Weight change since age 20 yrs					
<3 kg gain	1.	2.06 (1.25-3.40)	1.97 (1.17-3.33)	1.70 (1.02-2.81)	
3-14.5 kg gain	1.	1.30 (0.92-1.85)	1.32 (0.93-1.87)	1.75 (1.22-2.51)	0.10
>=14.5 kg gain	1.	0.86 (0.62-1.20)	1.15 (0.83-1.61)	1.30 (0.92-1.83)	
Physical Activity (MET-hrs per week)					

	Adjusted OR† (95% CI)					
		*Total Sweets**				
		<2.4	**2.4-7.3**	**7.4-14.1**	**>14.1**	**p-value§**
	0	1.	1.06 (0.70-1.60)	0.85 (0.55-1.30)	0.98 (0.64-1.52)	
	0.01-7.54	1.	1.29 (0.90-1.86)	1.37 (0.94-1.98)	1.82 (1.25-2.66)	0.02
	>7.54	1.	1.23 (0.84-1.81)	1.78 (1.23-2.59)	1.91 (1.30-2.82)	
Alcohol use						
	Never	1.	0.82(0.58-1.15)	0.93 (0.66-1.31)	1.19 (0.84-1.69)	0.01
	Ever	1.	1.57 (1.19-2.08)	1.75 (1.32-2.31)	1.88 (1.40-2.51)	

*Categories represent quartiles of number of servings per week among all controls.

†Adjusted for age at reference date and total energy intake.

§p-value for test of interaction using likelihood ratio test.