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Vegetable protein and vegetable fat intakes in pre-adolescent and adolescent girls, and risk for benign breast disease in young women

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Abstract

Previous investigations, of adolescent diet recalled in adulthood, found lower risk for benign breast disease (BBD) with higher intakes of vegetable fat and nuts during high school. We investigate whether vegetable protein and fat, derived from diets reported during pre-adolescence and adolescence, are associated with subsequent risk for BBD in young women. The Growing Up Today Study includes 9,039 females, 9–15 years in 1996, who completed questionnaires annually through 2001, and then in 2003, 2005, 2007, and 2010. Food frequency questionnaires (1996–2001) obtained intake data on a variety of foods. Beginning in 2005, women (18–30 years) reported whether they had ever been diagnosed with BBD that was confirmed by breast biopsy ($n = 112$ cases). Logistic regression estimated associations between intakes of vegetable protein and fat and biopsy-confirmed BBD. Those individual foods that were the largest contributors of protein and fat in this cohort were also investigated. In analyses of intakes from 1996 through 1998, when our cohort was youngest, vegetable fat (OR = 0.72/(10 gm/day), 95 % CI 0.53–0.98; $p = 0.04$) was inversely associated with BBD risk. The greatest sources of vegetable fat and protein in these girls were peanut butter, peanuts, nuts, beans (beans, lentils, and soybeans), and corn. A daily serving of any one of these was associated with lower risk (OR = 0.32/(serv/day), 95 % CI 0.13–0.79; $p = 0.01$). Peanut butter (and nuts) at age 11 years was inversely associated with risk (p

= 0.01). In analyses of intakes at age 14 years, vegetable protein was associated with lower BBD risk (OR = 0.64/(10 gm/day), 95 % CI 0.43–0.95; $p = 0.03$). A daily serving at 14 years of any one of the foods was associated with lower risk (OR = 0.34, 95 % CI 0.16–0.75; $p = 0.01$), as was peanut butter (and nuts) ($p = 0.02$). Girls with a family history of breast cancer had significantly lower risk if they consumed these foods or vegetable fat. In conclusion, consumption of vegetable protein, fat, peanut butter, or nuts by older girls may help reduce their risk of BBD as young women.

Keywords

Childhood diet; Vegetable protein; Vegetable fat; Peanut butter; BBD; Prospective

Introduction

Substantial evidence implicates the period in life before a woman's initial pregnancy, when mammary gland cells are undergoing rapid proliferation, as a critical time for carcinogenic exposures that may increase her lifetime risk for breast cancer [1]. Certain childhood and adolescent exposures have been shown to be more important than adult exposures in breast cancer development [2-5]. Early-life diet may play a role in breast carcinogenesis by altering the hormonal environment [6, 7]. Research on soy intake in Asian and Asian-American populations provides impressive evidence that soy consumption in children (ages 5–11, more so than in adults after age 20) is responsible, in part, for lower breast cancer rates in Asian women [8]. High versus low soy intake in childhood was associated with a relative risk of breast cancer of 0.40 (95 % confidence interval (CI) 0.18–0.83).

Because benign breast disease (BBD) is a well-established risk factor for breast cancer [9], the investigation of exposures in girls and their subsequent development of BBD may provide insight into the etiology of breast cancer and present possible new strategies for prevention. We previously reported that alcohol consumption recorded during the teen years was associated with increased risk of BBD in young women [10], though dairy intakes (including milk, dairy protein, and dairy fat) were not [11]. Using adolescent dietary data recalled by adult women in the Nurses' Health Study II (NHSII), alcohol was associated with a greater risk for proliferative BBD [12] and vegetable fat and nuts were associated with a substantially lower risk [13, 14].

We investigated whether prospectively reported adolescent intakes of vegetable protein and vegetable fat, and individual foods high in vegetable protein or fat, were associated with subsequent BBD in young women, using data from a cohort study initiated in 1996 of 9–15-year-old females followed to age 30 years.

Materials and methods

Study population

The Growing Up Today Study (GUTS; founding PI, Dr. Colditz) includes 9,039 girls from all 50 states who are daughters of NHSII participants [15]. The study, approved by the Institutional Review Boards at Harvard School of Public Health and Brigham and Women's Hospital, is described elsewhere [16]. Mothers provided informed consent and their 9–15-year-old daughters assented by completing baseline questionnaires. The cohort returned questionnaires annually (by mail or Internet) from 1996 through 2001, and then in 2003, 2005, 2007, and 2010. The response rate to one or more follow-ups after baseline was 97 %. Over 80 % returned at least one of the 2005 through 2010 surveys inquiring about BBD.

Benign breast disease

The 2005, 2007, and 2010 surveys inquired “Has a health care provider ever diagnosed you as having Benign Breast Disease?” and, if yes, whether it had been “Confirmed by breast biopsy.” A total of 7,222 females (aged 18–30 year) reported whether a health care provider ever diagnosed them with BBD ($n = 266$ said yes), and if a diagnosis had been confirmed by breast biopsy ($n = 112$). After excluding six girls whose mothers reported childhood cancer in their daughters, those 6,950 females who never reported a BBD diagnosis provided the non-cases for our analyses.

Most BBD cases were likely diagnosed because participants (or their physicians) found a clinically palpable mass which was then biopsied, as participants were too young to be undergoing routine screening mammography. The most common type of BBD occurring in adolescents and young women is fibroadenoma, which accounts for nearly 70 % of benign breast lesions [17]. The remaining types are primarily cysts and fibrocystic changes [17]. A validation study conducted in NHSII women confirmed the accuracy of self-reports of biopsy-confirmed BBD [18].

Dietary intakes

Our self-administered semi-quantitative food frequency questionnaire (FFQ), designed specifically for older children and adolescents, has good validity and reproducibility for children ages 9–18 years [19]. The mean correlation for nutrients from the FFQ compared to three 24-hour recalls was $r = 0.54$, comparable to the performance of a similar FFQ for adults [20].

From 1996 through 2001, our surveys annually inquired about the usual frequency of past-year intake of a wide variety of foods. Vegetable fat, vegetable protein, and total energy were calculated based upon reported food and beverage intakes; further details on our childhood FFQ and the derivation of nutrient values are provided elsewhere [21]. Total daily energy intakes below 500 kcal/day or greater than 5,000 kcal/day were considered implausible and set to missing. Here, we use intakes occurring before age 20 years to represent the adolescent diet.

Other variables

At baseline, participants reported their race/ethnic group by marking all (of six) options that applied to them; most are white/non-Hispanic (95 %), as are BBD cases. We computed ages (to the month) from dates of questionnaire return and birth. Early surveys annually asked “Have you started having menstrual periods?” and “If yes, age when periods began.” Childhood adiposity was derived from baseline body mass index (BMI, kg/m^2), adjusted to age 10 years [22]. Multiple surveys asked females whether they had been pregnant. Cumulative alcohol intake was obtained from alcohol reported on the 2000–2003 surveys when participants were ages 13–22 [10]. Participants’ family history of breast disease was derived from information provided by their mothers [23].

Statistical analyses

To assess the potential for selection bias, using baseline (1996) data, we compared participants who returned the 2005, 2007, and/or 2010 surveys including BBD questions with those who did not.

Risk of biopsy-confirmed BBD was the outcome for all analyses. Logistic regression models, estimated using SAS [24], provided odds ratios (OR) and 95 % confidence intervals (CI). We did not have information regarding month and year when cases were diagnosed. Because age was related to being diagnosed during follow-up, all models adjusted for

baseline age (to the month) along with energy intake. Multivariable-adjusted models additionally included age at menarche, childhood adiposity, adolescent alcohol intake, and ever pregnant (yes, no).

We first investigated cumulative dietary intakes from baseline (1996) through 1998, representing the earliest diet available for each girl in our cohort, which is appealing because all 112 BBD cases and 6,950 non-cases are included in these analyses. We also examined dietary intakes at age 11, but these analyses had fewer girls as many participants were older than 11 at baseline. We further analyzed dietary intakes at age 14, which falls within the critical period between menses onset and first pregnancy. We also derived intakes separately by menarche status: diet before the onset of menses was the cumulative average of intakes from baseline up to the survey before menses onset, while post-menses-onset diet was the mean of intakes reported afterward.

Our key exposure variables were vegetable (from all plant sources) protein and fat intakes. We further determined the greatest contributors to these in our girls, which were peanut butter (sandwiches), nuts or peanuts (bags), corn, and beans (beans/lentils/soybeans). Servings of peanut butter and nuts (combined) contributed 14 % of the variance in vegetable fat and 20 % of the variance in vegetable protein intake in these girls. Corn intakes contributed 4 % of the variance in vegetable fat and 6 % for protein, while bean intakes contributed 1 % of the variance in vegetable fat, but 15 % of the variance in protein. Peanut butter is very high in both protein (64 g/cup) and fat (129 g/cup) (25). Our survey did not specify type of nuts, but peanuts have 35–40 g/cup of protein and 72–76 g/cup of fat; other types of nuts range from 9 g/cup protein (pecans) to 30–33 g/cup protein (almonds), and fat in other nuts ranges from 62 to 88 g/cup. Beans (12–17 g/cup protein and 1–3 g/cup fat), soybeans (30 g/cup protein and 16 g/cup fat), and lentils (17 g/cup protein and 1 g/cup fat) have high protein contents, but lower fat content, while corn (6 g/cup protein and 2 g/cup fat) is lower in both [25]. We analyzed the associations between BBD and these foods individually (modeled as number of servings every 3 days). We also analyzed the intakes (servings/day) of these foods combined (peanut butter, nuts, corn, beans, lentils, and soybeans). For contrast, we also considered green (string) bean and broccoli intakes as exposures, each very low in protein and in fat.

Because risk factors for BBD may differ by family history of breast disease, we also fit stratified models.

Results

Eighty percent of our cohort returned, between years 2005 and 2012, at least one of the surveys containing questions about BBD. Comparing baseline (1996) data of these females with the 20 % returning none of those surveys, the included girls were slightly younger (by 5.6 weeks) than those not included, were slightly taller (+0.17 inch), consumed more vegetable protein (+0.5gm/day) but less vegetable fat (−0.7 gm/day), and more beans (+0.01 serving/day of beans/lentils/soybeans) (all age-adjusted, dietary variables energy-adjusted, all $p < 0.05$). However, baseline BMI, energy intake, menarche status, intakes of corn, peanut butter, and nuts, and the high protein/fat foods combined were each similar for the included and missing females. These small differences are unlikely to present serious sources of bias.

Table 1 presents mean values at baseline by subsequent BBD status of the dietary intake variables of interest, and mean values or percentages of potential confounders included in multivariable-adjusted models. Though some of these factors (age at menarche and ever pregnant) were not significantly associated with risk for BBD, we still include them in our

multivariable-adjusted models because they are well-established risk factors for breast cancer.

Vegetable protein intake during the first 3 years of follow-up (cumulative intakes in 1996, 1997, and 1998, the earliest dietary data for each girl) was not associated with risk of biopsy-confirmed BBD (Table 2), though cumulative vegetable fat intake (included in the same model) was inversely associated (age–energy-adjusted OR = 0.72/(10 gm/day), $p = 0.04$). We next investigated those foods that were the greatest contributors to vegetable protein and fat intakes in these girls. Early intake of peanut butter and nuts ($p = 0.01$), and the foods combined ($p = 0.01$), was associated with lower BBD risk (Table 2). Peanut butter (and nut) intakes were higher than corn intakes, and bean consumption was the lowest, so the combined food findings are likely driven by peanut butter consumption. Analyses of categorized intakes (1 serving every 3 days vs none) of peanut butter (and nuts), beans, and corn, individually, provided similar results (Table 2 footnote). The mean age, corresponding to the Table 2 analyses of cumulative diet from 1996 through 1998, was 12.8 years.

Upon examining dietary intakes at age 11 (with less power because many girls were too old at baseline to be included), vegetable protein intake was not associated with BBD ($p = 0.98$; not shown), though vegetable fat (OR = 0.78/10 gm, $p = 0.16$) appeared more important. At age 11, intake of peanut butter and nuts (combined) was inversely associated with BBD (OR = 0.56/(serving every 3 days), $p = 0.02$), suggesting a 44 % reduction in risk.

Investigating diet at older ages, analysis of vegetable protein intake at age 14 found a significant association with biopsy-confirmed BBD (age–energy-adjusted OR = 0.64/(10 gm/day), $p = 0.03$), though vegetable fat intake at age 14 (in the same model) was not (Table 3) associated with BBD. Age 14 intakes of peanut butter and nuts (combined) were associated with lower risk for BBD (OR = 0.61/(serving every 3 days), $p = 0.02$). Corn was marginally significant (OR = 0.56/(serving every 3 days), $p = 0.06$), while beans/lentils/soybeans were not significant (their consumption in these girls was considerably lower than the other foods). Analyses of intakes (of peanut butter, beans, or corn, individually) categorized (1 serving every 3 days versus never) provided similar results (Table 3 footnote). Eating any one of these foods (peanut butter, nuts, corn, beans, lentils, and soybeans) daily at age 14 was inversely associated with BBD (OR = 0.34/(daily serving), $p = 0.01$). Again, the combined food findings are likely driven by peanut butter consumption.

Analyses of cumulative intakes before menses onset provided results similar to those at age 11 and to cumulative early (1996–1998) diet, while analyses of intakes after menses onset provided results similar to those at age 14, though some of the statistically significant ($p < 0.05$) associations outlined in the above paragraphs became marginally significant ($p < 0.10$) (due to smaller N's). These similarities were expected because dietary intakes at age 11 were highly correlated with cumulative intakes before menses onset (Spearman's $\rho = 0.82$ – 0.87) and intakes at age 14 were also highly correlated with intakes after menses onset ($\rho = 0.75$ – 0.80). On the other hand, correlations between the earlier (age 11 or pre-menses) and later (age 14 or post-menses onset) intakes were considerably lower, ranging from $\rho = 0.37$ to 0.47 , reflecting dietary changes as children go from childhood to adolescence.

We further considered salad dressings, as our surveys asked about low calorie and regular calorie (high in vegetable oil) separately, but neither was associated with BBD (all $p \geq 0.44$). Only our earliest (1996 and 1997) surveys collected data on intakes of seeds (pumpkin, sunflower) because their intakes were so low (0.03 serving/day) and they were subsequently dropped; seed intake appeared protective, but not statistically significant (OR = 0.67/(serving every 3 days); $p = 0.52$). Tofu and soy milk intakes were even lower in these girls (at baseline, less than 0.01 serving/day), preventing an analysis of amounts consumed,

though girls who ever reported drinking soy milk had non-significantly lower risk (OR = 0.68, $p = 0.60$).

To contrast with the high protein and high fat foods investigated above, we also considered green (string) beans and broccoli, both very low in protein and fat. In spite of reasonable consumption levels (mean baseline intakes: string beans 0.24 serving every 3 days and broccoli 0.31 serving every 3 days), we observed no evidence that either green beans or broccoli might lower BBD risk.

Because our earlier work indicated that some BBD risk factors vary by family history [23], we further investigated diet separately by family history of breast disease. Among the 1,199 women with a family history of breast cancer (in mother, maternal aunt, or maternal grandmother), 26 were themselves biopsy-confirmed BBD cases and 1,173 were non-cases; apparent benefit (against BBD) was suggested for peanut butter and nut consumption at age 14, after the onset of menses, and at younger ages (cumulative early intake), all $p < 0.05$. Cumulative early intakes (1996–1998) of vegetable fat and of the foods combined were significantly associated with lower BBD risk, and higher intakes of the foods combined at age 14 were also associated with lower risk for BBD among those with a family history of breast cancer. Among the 4,840 women with no family history of breast cancer or maternal BBD, 63 were themselves BBD cases and 4,777 were non-cases, all ORs were below 1.00, but a significant inverse association was found only for consumption of the foods combined after the onset of menses. With our smaller number of cases within family history subgroups, power is more limited, yet we still observed the significant associations described above.

Discussion

To our knowledge, this is the first investigation of vegetable protein and vegetable fat derived from dietary intakes prospectively reported by pre-adolescent and adolescent girls, rather than recalled later in adult life, and their risk of BBD as young women. Our work suggested that vegetable fat consumed by pre-adolescent girls and vegetable protein consumed by adolescents may be protective, while peanut butter and nuts, high in both protein and fat, consumed at any age may be associated with lower risk for BBD. Our stronger findings for peanut butter and nuts, than for corn and beans (beans, soybeans, lentils), may be due to their higher levels of consumption in these girls, but peanut butter and nuts also contain considerably more protein and fat. It is also possible that certain foods require a longer latency period between exposure and the diagnosis of disease.

Our findings were the strongest among those females whose mother, maternal aunt, or maternal grandmother had been diagnosed with breast cancer, though protective effects were still suggested for girls with no family history, whose ORs were consistently below 1.00 though seldom statistically significant. Girls with a family history of breast cancer may have more valid BBD information than those with no family history, among whom there may be more undiagnosed cases that appear in analyses as non-cases. All analyses within family history subgroups have less power.

Though we fit a large number of models, they address different age periods and thus the participants included sometimes vary among analyses. However, we might expect the estimated effects of diet at age 14 to be consistent with effects of intakes after menses onset, and they generally were, with vegetable protein more important than vegetable fat and peanut butter/nuts and the four foods combined important. Similarly, we may expect intakes before menses onset to provide conclusions similar to intakes at age 11 and to cumulative

early intakes: vegetable fat more important than vegetable protein, while peanut butter and nuts and the foods combined are still important.

Our findings are reasonably consistent with the limited literature on childhood diet and BBD. Analyses of nurses, who at 33–53 years of age recalled their high school diets (comparable to our age 14 or post-menarche measures), found an inverse association between intakes of vegetable fat and nuts and proliferative BBD risk (based on centralized pathology specimen review) [14]. Extended follow-up of that same cohort found that nuts and peanut butter consumed in high school were significantly associated with lower risk of proliferative BBD [13]. Intakes of beans and lentils were inversely associated with risk, though not statistically significant [13]. Looking instead at breast cancer, studies have implicated high total adolescent fat (including animal fat) consumption as a risk factor [26, 27]. More compatible with our BBD analyses, adolescent intakes of vegetable fat (derived from adult dietary recalls) were associated with decreased risk of breast cancer [28]. Our cohort's intake of soy products was too low for us to investigate, though earlier studies showed a strong inverse relationship between childhood soy consumption and risk of breast cancer [8]. Studies of dietary intakes in adulthood have not consistently supported associations between diet and BBD or breast cancer [29], but this is not unexpected if childhood or adolescent diets provide the critical exposures. However, vegetable fat consumed in adulthood was associated with lower risk of proliferative BBD without atypia [30]. Only olive oil (not total vegetable fat) consumption was associated with lower breast cancer risk in Spanish women [31]. Trials in mice and monkeys support a relationship between breast cancer risk and early-life events including diet [32, 33].

Our food frequency question on nut intakes did not specify the types of nuts consumed; however, there is evidence that walnuts contain bioactive molecules (alpha-linolenic acid (ALA) and phytosterols) that affect mammary epithelial cells [34]. Tree nuts and peanuts are rich in unsaturated fatty acids and other bioactive compounds that produce a broad range of metabolic benefits [35, 36].

The longitudinal design of this investigation comprises its major strength, as dietary data were collected in real time, years prior to the reporting of BBD diagnoses in this large cohort of girls from all over the US. Though all models controlled for age and energy, and other potential confounders were further included in multivariable-adjusted models, with separate analyses stratifying by family history, some residual and unmeasured confounding may remain. We cannot exclude the possibility of incomplete adjustment or confounding through variables not considered, but little is known about childhood risk factors for BBD. Although our cohort is not representative of US females, the comparison of risks within our cohort should still be valid and generalizable [37]. Because our participants are daughters of nurses, this reduces confounding by socioeconomic and other unmeasured factors, while enhancing the accuracy of the data.

The most serious limitation is our small number of cases, though we did obtain significant ORs (for several risk factors) that were reasonably consistent across our analyses. Furthermore, earlier research on this cohort reported significant associations between BBD and adolescent alcohol consumption [10], family history of breast disease [23], and childhood growth variables [22]; though our study of adolescent dairy intakes and BBD was null [11], this is consistent with the literature. Another limitation was the necessity to collect data by self-report, but alternatives were not feasible. Dietary intake reporting errors were likely non-differential with respect to subsequent BBD, possibly biasing our estimates toward the null. Some of the dietary variables in our analyses were cumulative averages of intakes reported over multiple years, which should reduce measurement error bias in risk

estimates. The racial/ethnic makeup of our cohort (95 % white/non-Hispanic) hinders generalization to other races/ethnicities.

In conclusion, we assessed BBD risk in young women (in their 20s) in relation to their vegetable protein and vegetable fat intakes recorded during pre-adolescence and adolescence, periods previously demonstrated to be critical for the development of breast cancer [2-5] and other adult diseases. We found evidence that vegetable protein and fat intakes, and peanut butter and nuts in particular, may help reduce risk for BBD. Older girls may benefit by consuming a variety of foods that are high in vegetable protein and fat, including peanut butter, nuts, corn, beans, lentils, and soybeans. Because our number of cases was relatively small, and because certain dietary factors may require longer latency from exposure to development of disease, continued follow-up of this cohort will be critical to reassess these results as new cases of BBD are diagnosed. In addition, animal models may help refine understanding of pathways and mechanisms, and potential prevention strategies.

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Table 1

Characteristics of girls in this study and their baseline diet by their subsequent benign breast disease status

	Never reported BBD (n = 6950)	Biopsy-confirmed BBD cases (n = 112)	Age-adjusted p value
Characteristics: Mean (SD)			
Baseline age (year)	12.0 (1.6)	12.5 (1.6)	0.01
Childhood BMI ^a (kg/m ²)	18.2 (3.3)	17.3 (2.5)	0.01
Menarche age (year)	12.9 (1.2)	13.0 (1.1)	0.24
Adolesc alcohol (serv/day)	0.21 (0.41)	0.33 (0.54)	0.03
<i>Baseline (1996) intakes:</i>			
Energy intake (kcal/day)	2050 (640)	1936 (612)	0.06
Veg protein (g/day)	24.9 (8.9)	23.0 (8.5)	0.02
Veg fat (g/day)	35.2 (13.5)	32.9 (12.3)	0.09
<i>Servings/day:</i>			
Peanut butter, Nuts	0.19 (0.23)	0.12 (0.17)	0.01
Beans, lentils, soybeans	0.08 (0.15)	0.07 (0.13)	0.31
Corn	0.13 (0.13)	0.12 (0.10)	0.15
Above foods combined	0.40 (0.32)	0.31 (0.26)	0.01
peanut butter, nuts, beans, lentils, soybeans, corn (servings/day)			
Characteristics: percentages			
Pregnancy ever	8.4 %	10.7 %	0.60
Family history BC	16.9 %	23.2 %	0.09
Maternal BBD	18.4 %	29.5 %	0.01

p values from logistic regression models of BBD outcome and risk factor, adjusted for baseline age

^aBaseline BMI, adjusted to age 10 years [22]

Table 2

Associations between cumulative dietary intakes over initial 3 years of follow-up (1996, 1997, 1998; youngest dietary data for each girl) and risk of BBD in young women: dietary intake means by BBD status and odds ratios (OR)

	Never reported BBD (<i>n</i> = 6950)	Biopsy-confirmed BBD cases (<i>n</i> = 112)	Age-energy-adjusted			Multivariable-adjusted		
	Mean	Mean	OR	(95 % CI)	<i>p</i>	OR	(95 % CI)	<i>p</i>
Vegetable protein (10 gm/day)	2.41	2.38	0.85	(0.55–1.33)	0.48	0.86	(0.55–1.34)	0.50
Vegetable fat (10 gm/day)	3.40	3.26	0.72	(0.53–0.98)	0.04	0.72	(0.52–0.98)	0.03
Peanut butter and nuts (servings/3 days)	0.52	0.38	0.56	(0.36–0.88)	0.01	0.56	(0.35–0.87)	0.01
Beans, lentils, soybeans (servings/3 days)	0.24	0.24	0.94	(0.55–1.61)	0.83	0.95	(0.55–1.62)	0.84
Corn (servings/3 days)	0.40	0.37	0.69	(0.35–1.35)	0.28	0.73	(0.37–1.43)	0.36
Total servings per day of peanut butter, nuts, beans, lentils, soybeans, corn	0.38	0.33	0.32	(0.13–0.79)	0.01	0.33	(0.13–0.82)	0.02

Multivariable-adjusted models further include childhood adiposity, age at menarche, adolescent alcohol intake, and pregnancy (ever). The mean age, for the cumulative 1996, 1997, and 1998 surveys, was 12.8 years. Categorical food analyses provided similar results: Peanut butter and nuts, 1 + serving/(3 days) vs none, OR = 0.44, 95 % CI 0.20–0.99; Beans, lentils, soybeans 1 + servings/(3 days) versus none, OR = 0.61, CI 0.24–1.57; Corn 1 + servings/(3 days) versus 3/month, OR = 0.79, CI 0.34–1.82

Table 3

Associations between adolescent (age 14 years) intakes and risk of BBD in young women: dietary intake means by BBD status and odds ratios (OR)

Intakes at 14 years	Never reported BBD (N = 6503)	Biopsy-confirmed BBD cases (N = 108)	Age-energy-adjusted			Multivariable-adjusted		
			OR	(95 % CI)	p	OR	(95 % CI)	p
Vegetable protein (10 gm/day)	2.48	2.33	0.64	(0.43–0.95)	0.03	0.65	(0.44–0.96)	0.03
Vegetable fat (10 gm/day)	3.45	3.29	0.88	(0.68–1.13)	0.30	0.87	(0.68–1.11)	0.26
Peanut butter, nuts (servings/3 days)	0.49	0.34	0.61	(0.41–0.91)	0.02	0.61	(0.41–0.91)	0.02
Beans, lentils, soybeans (servings/3 days)	0.23	0.22	0.90	(0.57–1.41)	0.64	0.90	(0.57–1.42)	0.65
Corn (servings/3 days)	0.40	0.33	0.56	(0.30–1.03)	0.06	0.57	(0.31–1.06)	0.08
Total servings/day of peanut butter, nuts, beans, lentils, soybeans, corn	0.37	0.29	0.34	(0.16–0.75)	0.01	0.34	(0.16–0.76)	0.01

When dietary variables were not available at age 14, values between ages 13.5 and 15.5 were used, and if still missing, values obtained from 13.0 to 15.99 year intakes. Multivariable adjustment further included age at menarche, childhood adiposity, adolescent alcohol intake, and pregnancy ever. Categorical food analyses provided similar results: Peanut butter and nuts, 1 + serving/(3 days) versus never, OR = 0.51, 95 % CI 0.28–0.93; Beans, lentils, soybeans 1 + servings/(3 days) versus never, OR = 0.78, CI 0.33–1.82; Corn 1 + servings/(3 days) versus never, OR = 0.43, CI 0.19–0.98