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## Intake of Energy-Dense Foods, Fast Foods, Sugary Drinks, and Breast Cancer Risk in African American and European American Women

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### Abstract

Limiting energy-dense foods, fast foods, and sugary drinks that promote weight gain is a cancer prevention recommendation, but no studies have evaluated intake in relation to breast cancer risk in African American (AA) women. In a case-control study with 1692 AA women (803 cases and 889 controls) and 1456 European American (EA) women (755 cases and 701 controls), odds ratios (OR) and 95% confidence intervals (CI) for risk were computed, stratifying for menopausal and estrogen receptor (ER) status. Among postmenopausal EA women, breast cancer risk was associated with frequent consumption of energy-dense foods (OR=2.95; 95% CI: 1.66-5.22), fast foods (OR=2.35; 95% CI: 1.38-4.00), and sugary drinks (OR=2.05; 95% CI: 1.13-3.70). Elevated risk of ER+ tumors in EA women was associated with energy-dense (OR=1.75; 95% CI: 1.14-2.69) and fast foods (OR=1.84; 95% CI: 1.22-2.77). Among AA women, frequent fast food consumption was related to premenopausal breast cancer risk (OR=1.97; 95% CI: 1.13-3.43), and with ER+ tumors. Energy adjustment attenuated risk estimates in AA women, while strengthening them among EA women. Frequent consumption of energy-dense and fast foods that have poor nutritive value appeared to increase breast cancer risk in AA and EA women, with differences by menopausal status and ER status.

### Keywords

race; breast cancer; energy-dense; African American

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## Introduction

Weight gain is a critical issue in the US with obesity rates increasing in women regardless of education and income (1). The obesity problem is even graver among African American (AA) women in whom the prevalence of obesity has significantly increased from 1999-2010 (2). Weight gain and obesity have been linked to several chronic diseases including cancer. In particular, body fatness and adult weight gain have been judged as “convincing” and “probable” causes of postmenopausal breast cancer, respectively (3). Consumption of energy-dense foods and sugary drinks could promote weight gain by contributing to increased caloric intake. Energy-dense foods and fast foods mainly include processed food items that contain large amounts of fat or sugar and are commonly consumed, such as baked goods (e.g. cakes, pastries, cookies, and other desserts and confectionery), burgers, and deep fried foods (e.g. French fries, chips, chicken pieces)(3). Sugary drinks mainly include fruit juices with added sugar, sodas and other soft drinks that lead to overconsumption of energy and resulting weight gain.

According to past NHANES data, energy-dense and nutrient-poor foods contribute about 27% of total daily energy intake, with desserts and sweeteners making up almost 20% among all energy-dense and nutrient-poor food groups (4). Higher frequency of fast food consumption has been associated with diets that are loaded with calories and limited in essential nutrients, which could promote weight gain and obesity (5, 6). Increase in dietary energy density has been associated with obesity (7) and overweight across all ethnic groups (8) among US adults. Similarly sugar-sweetened drinks such as soft drinks have also been linked to weight gain and obesity in both observational and experimental studies (9). Increases in energy intake are closely related to circulating levels of insulin-like growth factor (IGF)-1, sex hormone-binding globulin, and estrogen, all important hormonal factors that can play a critical role in cancer cell proliferation or inhibition (10).

Limiting consumption of foods that are high in energy (including fast foods) and sugary drinks is one of the eight evidence-based recommendations proposed by the World Cancer Research Fund (WCRF) and American Institute for Cancer Research (AICR) published in 2007 (3) and included as part of the 2010 Dietary Guidelines for Americans (11). However, to our knowledge, no studies have operationalized this guideline and investigated the association between energy-dense foods or sugary drinks and breast cancer in AA women despite disproportionate rates of overweight and obesity (80% in AA women vs. 60.2% in European American (EA) women) (12). Even in EA women, to our knowledge, no studies have conducted a comprehensive evaluation (such as stratification by menopausal status or hormone receptor status) of foods and drinks promoting weight gain as defined by the WCRF/AICR report (3) and breast cancer risk. Hence, there is a clear gap to be addressed in the nutritional and cancer epidemiology literature. Research on dietary factors that contribute to increased energy intake and weight gain is critical for health education relating to cancer prevention.

We evaluated the relationship between frequency of consuming foods and drinks that promote weight gain, including energy-dense foods, fast foods, and sugary drinks, by

operationalizing the definition of the specified guideline provided by WCRF/AICR (3), as well as assessed racial differences in the associations among participants in the *Women's Circle of Health Study* (WCHS), a case-control study of EA and AA women in New York (NY) and New Jersey (NJ).

## Materials and Methods

### Study population

The WCHS (13, 14) was a multi-site case-control study conducted in NY and NJ. AA and EA women who were newly diagnosed with histologically confirmed breast cancer, 20-75 years of age, with no prior history of cancer except non-melanoma skin cancer, and able to complete study materials in English were eligible to be cases. Controls did not have breast cancer at the time of recruitment, but met all other eligibility criteria as cases. Case recruitment in NY began in January 2002 and involved major hospitals with the largest referral patterns for AA, and controls were identified through random digit dialing (RDD) matched to cases by area code. Study recruitment in NY ended in 2008. In NJ, the NJ State Cancer Registry implemented rapid case ascertainment in seven NJ counties while controls were recruited using a combination of RDD supplemented by community recruitment for AA women (13). Study recruitment in NJ began in March 2006 and ended in 2012.

### Data collection

Home interviews were conducted by trained interviewers to administer questionnaires and collect body measurements. Participants first completed an informed consent following which they completed a main study questionnaire that queried about demographics, and major factors suspected or known to affect breast cancer risk, such as family history, reproductive factors, hormone use, physical activity, and other medical and lifestyle factors. A Food Frequency Questionnaire (FFQ) developed by the Fred Hutchinson Cancer Research Center collected information on frequency and serving size of approximately 125 food items in the 12 month period prior to reference date. Reference date was date of diagnosis for cases and for controls, it was 97 days before date of interview to ensure comparability in recall period by compensating for the time lag between case diagnosis and reporting to registry. This FFQ was based on questionnaires used in two large NIH-funded studies, the Selenium and Vitamin E Cancer Prevention Trial and the VITamins and Lifestyle study. In the past, studies that have specifically assessed the validity and reliability of FFQs in minority populations observed a need to train participants on using self-administered FFQs especially when participants are poorly educated (15). In the WCHS, although the FFQ was generally self-administered, the study interviewers educated all participants on how to respond to the FFQ as part of the in-person appointment. Anthropometric measurements included standing height and weight, were collected as part of the interview, which were then used to compute body mass index (BMI).

Overall, among those who were contacted and eligible, participation rates were 78.7% in AA cases, 48.2% in AA controls, 79% in EA cases, and 49% in EA controls. Hence, a total of 1732 AA women (827 cases and 905 controls) and 1487 EA women (772 cases and 715 controls) participated in the study. Of these, over 97% of women completed the FFQ. Thus,

the study sample for this analysis involved 803 AA cases, 889 AA controls, 755 EA cases, and 701 EA controls who completed both the main study questionnaire and the FFQ. The study was approved by the Institutional Review Boards at the University of Medicine and Dentistry of New Jersey (now Rutgers University), Mount Sinai School of Medicine (now Icahn School of Medicine at Mount Sinai), and Roswell Park Cancer Institute.

### **Operationalization of foods and drinks that promote weight gain**

As per WCRF/AICR guidelines(3), foods and drinks that promote weight gain were classified into energy-dense foods, fast foods, and sugary drinks. Food items from the FFQ that were included for each of these food groups are listed in Appendix 1. Using the USDA National Nutrient Database for Standard Reference (16), food items in the FFQ that delivered more than 225 kcal per 100 grams of food were identified as energy-dense foods. Although fast foods are generally energy dense, they were listed under their own category to enable us to evaluate that food group independently. All other foods that met the criteria (3) for being energy dense were included in the energy-dense foods category.

As the recommendation was to consume these foods (and drinks) sparingly, we calculated total frequency of consumption per week for the medium serving of foods included in each of these three groups. As there are no established recommended thresholds, we computed quartiles for frequency of consumption of energy-dense and fast foods based on distribution among controls. Due to the limited consumption of sugary drinks in general, we categorized frequency of consumption as none, low (below median), and high (above median). Frequency of consuming fast foods and sugary drinks was lower in EA women than in AA women, resulting in skewed distributions and very small cell counts especially in the extreme categories when using similar cut points in both races. Hence, to be consistent across all three food groups, race-specific quartiles were used to categorize frequency of energy dense foods, fast foods and sugary drink intake. Since the distribution relating to frequency of energy-dense food consumption was very similar in both races, the race-specific cut points for frequency of energy-dense foods are the same in both EA and AA women. To facilitate interpretation of cut points relating to frequency of intake per week, the thresholds were rounded to the nearest whole number, due to which distribution of frequencies may not be exactly equal in the four groups. Except for one AA woman missing value for sugary drinks, there were no other missing values for energy-dense foods, fast foods, and sugary drinks.

### **Statistical analyses**

Chi square statistics were used to compare distribution of demographics and other characteristics between cases and controls in each race. Summary statistics (mean, median, standard deviation) were computed to compare distributions relating to total caloric intake as well as frequency of consumption of energy-dense and fast foods, and sugary drinks between cases and controls separately in AA and EA women. The non-parametric Wilcoxon Rank Sum Test provided p values for the difference in distributions. Unconditional logistic regression was used to obtain odds ratios (OR) and 95% confidence intervals (CI). Tests of linear trend were computed by assigning the median value to each category. Statistical significance was defined as a p value 0.05.

Multivariable models were adjusted for age, ethnicity (Hispanic or Non-Hispanic), country of origin (“US born”, “Caribbean born”, “Other”), education (“less than 12<sup>th</sup> grade”, “high school graduate or equivalent”, “some college”, “college graduate”, “post-graduate degree”), age at menarche (continuous), age at menopause (continuous; only for postmenopausal women), menopausal status (if not stratified by this variable), parity (continuous), age at first birth (“nulliparous”, “0-19”, “20-24”, “25-30”, “31”), breastfeeding status (ever/never), history of benign breast disease (yes/no), family history of breast cancer (yes/no), hormone replacement therapy (HRT) use (ever/never), oral contraceptive (OC) use (ever/never), body mass index (BMI - continuous), and study site (NY/NJ). As the purpose of this study was to investigate the independent association between frequency of consumption of foods and drinks that promote weight gain and breast cancer risk (as per AICR/WCRF recommendations (3)), results are presented for models with and without adjustment for total caloric intake. Estimates were also assessed with adjustment for total fat intake in place of total calories. Observations that had missing data for any of the covariates were dropped from regression models (n=29 for AA and n=32 for EA women).

All analyses were stratified by race and further stratified by menopausal status and hormone receptor status. As over 70% of cases had information for ER status of their tumor, polytomous logistic regression was used to compute risk estimates for estrogen receptor positive (ER+) and estrogen receptor negative (ER-) tumors with controls as reference. Statistical interactions were evaluated by including a cross product term involving the potential effect modifier in logistic models.

In sensitivity analyses, models were repeated after excluding women with extreme caloric intake (n=157) i.e. less than 500 kcal (n=81 in AA and 21 in EA) or greater than 4500 kcal in a day (n=50 in AA and 5 in EA). All analyses were completed using SAS version 9.2 (SAS Institute, Cary NC).

## Results

Study population characteristics are presented in Table 1. Higher proportions of EA women had higher education and were non-obese compared to AA women. In both races, cases were more likely to be HRT users, to have a family history of breast cancer and personal history of benign breast disease. More AA cases had hormone receptor negative tumors than EA cases. The differences in frequency of consuming energy-dense or fast foods were not significantly different between cases and controls in either race (Table 2), but AA women (cases and controls) reported higher total caloric intake compared to EA women. The distribution of reporting frequency of consuming sugary drinks in a week was marginally higher in EA cases than controls (p=0.06), although the opposite was true for total caloric intake (p=0.06).

Among AA women (Table 3), after adjustment for total energy intake, consuming fast foods more than five times a week as compared to once or less a week was associated with increased premenopausal breast cancer risk (OR=1.97; 95% CI: 1.13-3.43, p-trend=0.04). Although a significant positive trend between frequency of consuming energy-dense foods and breast cancer was also observed in premenopausal AA women (OR=1.73; 95% CI:

1.13-2.65,  $p$ -trend<0.01), this association was not independent of caloric intake. No clear associations were found for postmenopausal women. None of the statistical interaction terms across menopausal status were significant regardless of calorie adjustment [data not shown].

In analyses that stratified by hormone receptor status (Table 4), there was a suggestion of an increased risk of ER+ tumors among AA women who reported frequency of consuming energy-dense foods (OR=1.65; 95% CI: 1.15-2.37,  $p$ -trend=0.02) or fast foods (OR=1.73; 95% CI: 1.16-2.59;  $p$ -trend=0.02) in the highest quartile compared to lowest. However, the estimates were attenuated when total energy intake was added to the model, and confidence interval included the null value. Findings related to risk of ER- tumors were mostly null for all food groups.

Results from analyses in EA women stratified by menopausal status are presented in Table 5. A statistically significant increased breast cancer risk associated with more frequent consumption of energy-dense foods (OR=1.57; 95% CI: 1.10-2.24,  $p$ -trend=0.02) and fast foods (OR=1.75; 95% CI: 1.24-2.46,  $p$ -trend<0.01) was observed after adjusting for caloric intake. These associations appeared to be stronger in postmenopausal EA women. In addition, a positive trend between consuming more than one sugary drink a week compared to no consumption and postmenopausal breast cancer risk was also observed in EA women (OR=2.05; 95% CI: 1.13-3.70,  $p$ -trend=0.02). There was no significant statistical interaction by menopausal status.

When evaluating associations stratified by hormone receptor status in EA women (Table 6), increased reported frequency of consuming energy-dense foods (OR=1.75; 95% CI: 1.14-2.69,  $p$ -trend=0.01) and fast foods (OR=1.84; 95% CI: 1.22-2.77,  $p$ -trend<0.01) was strongly associated with elevated risk of ER+ tumors. There was a suggestion of elevated risk of ER- tumors with higher frequency of sugary drink intake, but the association was not statistically significant (OR=1.82; 95% CI: 0.93-3.57,  $p$ -trend=0.08), possibly due to limited power as there were only 17 EA ER- cases consuming more than one sugary drink per week.

In sensitivity analyses, overall, there was no change in direction of odds ratios or in main study conclusions when women reporting extremely low or high energy intake were excluded [data not shown]. Adjusting for fat instead of energy intake produced very similar estimates.

## Discussion

In the first study to evaluate associations between frequency of consuming foods and drinks that promote weight gain (operationalized as per the AICR/WCRF guideline) and breast cancer risk in a large sample of AA and EA women, higher consumption of energy-dense foods and fast foods was associated with increased breast cancer risk in both AA and EA women, with some differences by menopausal and ER status of the tumor. The positive associations with frequency of fast food intake were stronger among premenopausal AA women and postmenopausal EA women, in whom a positive trend was also observed with frequent intake of energy-dense foods and sugary drinks. Higher frequency of consuming fast foods was associated with increased risk of ER+ tumors in AA women while among EA

women, significant risk increase with frequent consumption of energy-dense and fast foods were also mostly observed for ER+ tumors. Adjustment for total energy intake attenuated odds ratios in AA women, but strengthened risk estimates in EA women.

In the EPIC study (17), greater adherence to following the guideline on restricting foods and drinks that promote weight gain was not associated with total cancer incidence in women, and the population was mostly Caucasian. An investigation from the VITamins and Lifestyle Study cohort (18) that focused on adherence to the cancer prevention recommendations and breast cancer risk in postmenopausal women also found no association with meeting the guideline on limiting foods and drinks that promote weight gain and disease risk, but operationalization of this guideline was different from our study. Although there is a plethora of evidence on other AICR/WCRF dietary guidelines in relation to red meat, fruits, vegetables, and alcohol intakes and breast cancer risk, the majority of the literature have focused on EAs (19), with very scarce research on evaluating specific foods and drinks that promote weight gain. Evaluating frequency of commonly consumed energy-dense foods and drinks instead of assessing total energy or dietary fat intake not only serves to directly operationalize the cancer prevention guideline (3), but also presents both a measure and actual food groups that can be more easily comprehended by health promoters and the public. No studies have operationalized and examined racial differences in meeting the guideline on energy-dense foods, fast foods, and sugary drinks in relation to breast cancer risk. Hence, there are no direct comparisons available for most of our study findings.

In the only study that presented breast cancer risk estimates associated with dietary fat in AA and EA women, no significant relationships were observed in AA women while percentage of energy from total fat increased risk in EA women (20). In our study, findings were very similar when adjusting for fat or adjusting for calories. We report results before and after energy adjustment to account for diet composition as well as the total volume of food. Although energy-dense foods increased breast cancer risk in both races, adjustment for energy attenuated associations in AA women and strengthened odds ratios in EA women. To understand potential reasons for these differences, we obtained estimates pertaining to proportion of variation in energy intake accounted for by frequency of consuming energy-dense foods, fast foods, and sugary drinks. Each of these food groups seemed to explain a much higher proportion of total energy intake in AA than in EA women, which suggests that these foods are a bigger part of the diet composition in AA women. In contrast, EA women reported lower intakes of these food groups, and the total energy intake in these women is probably explained by a wider variety of foods. Past NHANES data that investigated dietary diversity in US adults also observed lower diet diversity scores in AA than in EAs (21). Hence, it is possible that among EA women, relatively higher proportions of energy coming from foods that are dense in calories results in stronger associations with risk, while in AA women, the diet composition is not varied enough to detect differences in risk. However, we were unable to evaluate the contrasting findings on energy adjustment after accounting for potential racial differences in metabolism rate, as data on energy expenditure was not collected in our study.

In addition to differences following energy adjustment, EA women appeared to experience increased risks associated with frequent fast food and sugary drink intake at lower thresholds

than AA women. These racial differences once again support the notion of more distinct diet composition in EA compared to AA women, due to which small differences in intake levels could result in significant associations. In addition, the differences could also potentially indicate varied levels of susceptibility to physiological changes impacted by dietary intake in addition to BMI. In fact, the relationship between BMI and breast cancer risk has not been consistent in EA and AA women in the literature. Although BMI is an established risk factor for breast cancer in postmenopausal EA women, the associations in AA women are inconclusive(19), including a null association that was observed between BMI and breast cancer risk using anthropometric data collected in the WCHS(22). Furthermore, the body composition publication from WCHS also observed increased premenopausal breast cancer risk with higher waist and hip circumferences in AA women(22), while among premenopausal EA women, the literature has shown reduced breast cancer risk with increased body fatness(3). Put together, this evidence suggests plausible racial differences in nutritional factors and breast cancer risk and possibly suggests that the increased risk may not just be mediated by increases in body weight.

Reasons for the observed racial differences by menopausal status are unclear. In our study, the positive associations observed with increased frequency of consuming energy-dense foods were more dominant in premenopausal AA and postmenopausal EA women. Energy imbalance was related to increased breast cancer risk particularly in premenopausal women in the National Breast Screening Study (23), and total energy intake increased breast cancer risk in premenopausal women in a study involving sisters in a recent study (24), but race-specific estimates were not provided. Aside from consideration of chance findings, potential racial differences in the way menopausal status modifies the relationship between consuming energy-dense foods and breast cancer risk should be investigated further.

The elevated risks associated with energy-dense and fast food intakes were more pronounced for ER+ tumors in both AA and EA women, however the risk estimates were attenuated with energy adjustment in AA women. Our findings are consistent with a recent study based on data from the Breast Cancer Family Registry that reported elevated risk of both hormone receptor positive and negative tumors corresponding to energy intake, but significant increased risk was found only for hormone receptor positive tumors (24). In addition to being more common than ER- tumors, ER+ tumors are more closely related to hormonal factors and therefore may be more responsive to hormonal levels impacted by diet. However, findings for ER- tumors warrant further investigation on potential non-estrogenic pathways that could mediate the role of consuming energy-dense foods and drinks. The small sample sizes in some cells could also have caused significant differences in risk, and hence the strong odds ratios should be viewed with caution.

Certain limitations of the study should be noted. Bias in recalling intake of 'seemingly' unhealthy foods cannot be ignored. Data on change in behaviors since diagnosis showed that more AA and EA cases (49% and 35%) had decreased total energy intake since diagnosis compared to AA and EA controls (35% and 30%), respectively. A similar pattern was observed for fast food consumption. However, if cases had under-reported intake of energy-dense foods by incorrectly reporting dietary behavior since diagnosis, then such an under-reporting would probably underestimate the observed associations. Racial differences in

recall should also be considered, especially in studies that use self-reported data. For instance, more EA respondents (both cases and controls) reported ‘no change’ in their intake of calories and fast foods than AA participants, while the majority of AA cases and controls reported ‘decreasing’ total caloric and fast food intake (data not shown), despite having higher caloric intake (Table 2) and more frequent intake compared to EA women, as demonstrated by the higher cut points for fast foods and sugary drinks. Nutrition studies that have evaluated racial differences have also used self-reported dietary data in the past (25, 26), and results should be viewed with caution until investigations are repeated in other EA and AA populations.

The main strength of this study was the ability to conduct a comprehensive evaluation of the AICR/WCRF guideline on restricting foods and drinks that promote weight gain and using a large sample of AA and EA women. As WCHS was specifically designed to evaluate breast cancer risk factors in AA women, the study sample enabled analyses further stratified by menopausal and ER status while evaluating racial differences in the associations. Moreover, as this study involved population-based recruitment and is an association study rather than a prevalence study, findings can be generalized to AA and EA women in the US.

In summary, frequent consumption of energy-dense and fast foods that are poor in nutritive value appeared to increase breast cancer risk in AA and EA women, with some differences by menopausal status and hormone receptor status. Hence, public health education programs should continue to promote the cancer prevention guidelines, with specific attention to diet interventions that could have multifaceted benefits by reducing weight gain while improving dietary quality.

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## Appendix

### Appendix 1

List of food items from the FFQ that were included in the energy-dense foods, fast foods, and sugary drinks groups as per Second Expert Report(3).

Category of foods/drinks that promote weight gain	Food Items from the FFQ
Energy-dense foods	“Ice cream and milkshakes”, “doughnuts, pies and pastries”, “cookies and cakes”, “chocolate, candy bars and toffee”, “other candy”, “buttered or regular microwave popcorn”, “regular crackers”, “regular potato chips, tortilla chips, corn chips, and puffs”, “pancakes, French toast, waffles”, “muffins, scones, croissants and biscuits”, “cornbread and corn muffins”
Fast foods	“hot dogs and sausage”, “ground meat including hamburgers and meatloaf”, “fried chicken, including nuggets and tenders”, “fried fish, fish sandwich, fried shellfish”, “pizza”, “burritos, tacos, quesadillas”, “French fries, fried potatoes and hash browns”, “enchiladas”
Sugary drinks	“Fruit drinks fortified with Vitamin C such as Hi-C, Fruitopia, and Kool-Aid”, “regular soft drinks”

## References

- Ogden CL, Lamb MM, Carroll MD, Flegal KM. Obesity and socioeconomic status in adults: United States, 2005-2008. 2010:1-8.
- Flegal KM, Carroll MD, Kit BK, Ogden CL. Prevalence of obesity and trends in the distribution of body mass index among US adults, 1999-2010. JAMA. 2012; 307:491-7. jama.2012.39 [pii]. 10.1001/jama.2012.39 [PubMed: 22253363]
- World Cancer Research Fund/American Institute for Cancer Research. Food, Nutrition, Physical Activity, and the Prevention of Cancer: A Global Perspective. Washington DC: American Institute for Cancer Research; 2007.
- Kant AK. Consumption of energy-dense, nutrient-poor foods by adult Americans: nutritional and health implications. The third National Health and Nutrition Examination Survey, 1988-1994. Am J Clin Nutr. 2000; 72:929-36. [PubMed: 11010933]
- Anderson B, Rafferty AP, Lyon-Callo S, Fussman C, Imes G. Fast-food consumption and obesity among Michigan adults. Prev Chronic Dis. 2011; 8:A71. A71 [pii]. [PubMed: 21672395]
- Bowman SA, Vinyard BT. Fast food consumption of U.S. adults: impact on energy and nutrient intakes and overweight status. J Am Coll Nutr. 2004; 23:163-8. [PubMed: 15047683]
- Ledikwe JH, Blanck HM, Kettel Khan L, Serdula MK, Seymour JD, et al. Dietary energy density is associated with energy intake and weight status in US adults. Am J Clin Nutr. 2006; 83:1362-8. 83/6/1362 [pii]. [PubMed: 16762948]
- Howarth NC, Murphy SP, Wilkens LR, Hankin JH, Kolonel LN. Dietary energy density is associated with overweight status among 5 ethnic groups in the multiethnic cohort study. J Nutr. 2006; 136:2243-8. 136/8/2243 [pii]. [PubMed: 16857848]
- Malik VS, Schulze MB, Hu FB. Intake of sugar-sweetened beverages and weight gain: a systematic review. Am J Clin Nutr. 2006; 84:274-88. 84/2/274 [pii]. [PubMed: 16895873]
- Harvie M, Howell A. Energy balance adiposity and breast cancer - energy restriction strategies for breast cancer prevention. Obes Rev. 2006; 7:33-47. OBR207 [pii]. 10.1111/j.1467-789X.2006.00207.x [PubMed: 16436101]
- U.S Department of Agriculture and U.S. Department of Health and Human Services. Dietary guidelines for Americans, 2010. U.S. Government Printing Office; Washington, DC: 2010.
- National Center for Health Statistics. Health, United States, 2011: With special feature on socioeconomic status and health. Hyattsville, MD: 2012.
- Bandera EV, Chandran U, Zirpoli G, McCann SE, Ciupak G, et al. Rethinking sources of representative controls for the conduct of case-control studies in minority populations. BMC Med

Res Methodol. 2013; 13:71. 1471-2288-13-71 [pii]. 10.1186/1471-2288-13-71 [PubMed: 23721229]

14. Ambrosone CB, Ciupak GL, Bandera EV, Jandorf L, Bovbjerg DH, et al. Conducting Molecular Epidemiological Research in the Age of HIPAA: A Multi-Institutional Case-Control Study of Breast Cancer in African-American and European-American Women. *J Oncol*. 2009; 2009:871250. 10.1155/2009/871250
15. Kristal AR, Feng Z, Coates RJ, Oberman A, George V. Associations of race/ethnicity, education, and dietary intervention with the validity and reliability of a food frequency questionnaire: the Women's Health Trial Feasibility Study in Minority Populations. *Am J Epidemiol*. 1997; 146:856–69. [PubMed: 9384206]
16. United States Department of Agriculture ARS, Nutrient Data Laboratory. National nutrient database for standard reference. Release 24. 2012
17. Romaguera D, Vergnaud AC, Peeters PH, van Gils CH, Chan DS, et al. Is concordance with World Cancer Research Fund/American Institute for Cancer Research guidelines for cancer prevention related to subsequent risk of cancer? Results from the EPIC study. *Am J Clin Nutr*. 2012; 96:150–63. *ajcn*.111.031674 [pii]. 10.3945/ajcn.111.031674 [PubMed: 22592101]
18. Hastert TA, Beresford SA, Patterson RE, Kristal AR, White E. Adherence to WCRF/AICR Cancer Prevention Recommendations and Risk of Postmenopausal Breast Cancer. *Cancer Epidemiol Biomarkers Prev*. 2013; 22:1498–508. 1055-9965.EPI-13-0210 [pii]. 10.1158/1055-9965.EPI-13-0210 [PubMed: 23780838]
19. Chandran U, Hirshfield KM, Bandera EV. The role of anthropometric and nutritional factors on breast cancer risk in African-American women. *Public Health Nutr*. 2012; 15:738–48. S136898001100303X [pii]. 10.1017/S136898001100303X [PubMed: 22122844]
20. Wang J, John EM, Horn-Ross PL, Ingles SA. Dietary fat, cooking fat, and breast cancer risk in a multiethnic population. *Nutr Cancer*. 2008; 60:492–504. 794508066 [pii]. 10.1080/01635580801956485 [PubMed: 18584483]
21. Kant AK, Block G, Schatzkin A, Ziegler RG, Nestle M. Dietary diversity in the US population, NHANES II, 1976–1980. *J Am Diet Assoc*. 1991; 91:1526–31. [PubMed: 1960344]
22. Bandera EV, Chandran U, Zirpoli G, Gong Z, McCann SE, et al. Body fatness and breast cancer risk in women of African ancestry. *BMC cancer*. 2013; 13:475.10.1186/1471-2407-13-475 [PubMed: 24118876]
23. Silvera SA, Jain M, Howe GR, Miller AB, Rohan TE. Energy balance and breast cancer risk: a prospective cohort study. *Breast Cancer Res Treat*. 2006; 97:97–106.10.1007/s10549-005-9098-3 [PubMed: 16319973]
24. Zhang FF, John EM, Knight JA, Kaur M, Daly M, et al. Total energy intake and breast cancer risk in sisters: the Breast Cancer Family Registry. *Breast Cancer Res Treat*. 2013; 137:541–51.10.1007/s10549-012-2342-8 [PubMed: 23225141]
25. Wang, Ya; C, X. How much of racial/ethnic disparities in dietary intakes, exercise, and weight status can be explained y nutrition- and health related psychological factors and socioeconomic status among US adults? *Journal of American Deitetic Association*. 2011:1904–911.
26. Newby PK, Noel SE, Grant R, Judd S, Shikany JM, et al. Race and region have independent and synergistic effects on dietary intakes in black and white women. *Nutr J*. 2012; 11:25. 1475-2891-11-25 [pii]. 10.1186/1475-2891-11-25 [PubMed: 22500645]

## Abbreviations

<b>AA</b>	African American
<b>EA</b>	European American
<b>OR</b>	odds ratio
<b>CI</b>	confidence interval

<b>WCRF</b>	World Cancer Research Fund
<b>AICR</b>	American Institute for Cancer Research
<b>WCHS</b>	Women's Circle of Health Study
<b>NY</b>	New York
<b>NJ</b>	New Jersey
<b>RDD</b>	random digit dialing
<b>FFQ</b>	food frequency questionnaire
<b>HRT</b>	hormone replacement therapy
<b>OC</b>	oral contraceptive
<b>BMI</b>	body mass index
<b>ER</b>	estrogen receptor

**Table 1**  
**Distribution of selected characteristics for breast cancer among women participating in WCHS, n=3148**

	AA women		EA women	
	Cases (n=803) N (%)	Controls (n=889) N (%)	Cases (n=755) N (%)	Controls (n=701) N (%)
<b>Age at reference date (yrs)</b>				
20-34	44 (5.5)	74 (8.3)	27 (3.6)	35 (5.0)
35-44	173 (21.5)	190 (21.4)	153 (20.3)	162 (23.1)
45-54	261 (32.5)	325 (36.6)	252 (33.4)	258 (36.8)
55-64	254 (31.6)	259 (29.1)	245 (32.5)	245 (35.0)
65-76	71 (8.8)	41 (4.6)	78 (10.3)	1 (0.1)
<i>Chi square p value</i>	<i>0.001</i>		<i>&lt;0.001</i>	
<b>Education</b>				
<High school	118 (14.7)	112 (12.6)	21 (2.8)	10 (1.4)
High school graduate	241 (30)	227 (25.5)	127 (16.8)	69 (9.8)
Some college	213 (26.5)	259 (29.1)	165 (21.9)	132 (18.8)
College graduate	141 (17.6)	180 (20.2)	230 (30.5)	226 (32.2)
Post-graduate degree	90 (11.2)	111 (12.5)	212 (28.1)	264 (37.7)
<i>Chi square p value</i>	<i>0.11</i>		<i>&lt;0.001</i>	
<b>Country of origin</b>				
United States	552 (68.7)	711 (80)	639 (84.6)	617 (88)
Caribbean countries	189 (23.5)	129 (14.5)	25 (3.3)	2 (0.3)
Other	62 (7.7)	49 (5.5)	91 (12.1)	82 (11.7)
<i>Chi square p value</i>	<i>&lt;0.001</i>		<i>&lt;0.001</i>	
<b>Ethnicity</b>				
Hispanic	45 (5.6)	26 (2.9)	62 (8.2)	15 (2.1)
Non-Hispanic	758 (94.4)	863 (97.1)	693 (91.8)	686 (97.9)
<i>Chi square p value</i>	<i>0.01</i>		<i>&lt;0.001</i>	
<b>Marital Status</b>				
Married	287 (35.7)	306 (34.5)	468 (62.1)	477 (68)
Living as married	13 (1.6)	19 (2.1)	22 (2.9)	22 (3.1)
Widowed	74 (9.2)	58 (6.5)	40 (5.3)	19 (2.7)
Separated	62 (7.7)	57 (6.4)	14 (1.9)	16 (2.3)
Divorced	138 (17.2)	136 (15.3)	91 (12.1)	73 (10.4)
Single, never married or never lived as married	229 (28.5)	312 (35.1)	119 (15.8)	94 (13.4)
<i>Chi square p value</i>	<i>0.03</i>		<i>0.06</i>	
<b>Age at menarche (yrs)</b>				
<12	228 (28.4)	250 (28.2)	175 (23.4)	157 (22.6)
12-13	365 (45.4)	399 (44.9)	416 (55.6)	368 (53)

	AA women		EA women	
	Cases (n=803) N (%)	Controls (n=889) N (%)	Cases (n=755) N (%)	Controls (n=701) N (%)
>13	210 (26.2)	239 (26.9)	157 (21)	170 (24.5)
<i>Chi square p value</i>	0.94		0.28	
<b>Menopausal status</b>				
Premenopausal	408 (50.8)	463 (52.1)	389 (51.5)	385 (54.9)
Postmenopausal	395 (49.2)	426 (47.9)	366 (48.5)	316 (45.1)
<i>Chi square p value</i>	0.60		0.20	
<b>Age at menopause (yrs)</b>				
45	36 (9.4)	52 (12.3)	29 (8.1)	27 (8.7)
46-49	60 (15.6)	108 (25.6)	73 (20.4)	71 (22.9)
50-54	247 (64.2)	220 (52.1)	204 (57)	175 (56.4)
>55	42 (10.9)	42 (10)	52 (14.5)	37 (11.9)
<i>Chi square p value</i>	0.001		0.70	
<b>Parity (livebirths)</b>				
0	124 (15.4)	148 (16.7)	237 (31.4)	206 (29.4)
1-2	414 (51.6)	438 (49.3)	355 (47)	355 (50.6)
3-4	200 (24.9)	237 (26.7)	146 (19.3)	117 (16.7)
>5	65 (8.1)	66 (7.4)	17 (2.3)	23 (3.3)
<i>Chi square p value</i>	0.67		0.24	
<b>Age at first birth (yrs)</b>				
Nulliparous (0 birthcount)	124 (15.5)	148 (16.7)	237 (31.4)	206 (29.4)
19	253 (31.6)	294 (33.1)	36 (4.8)	32 (4.6)
20-24	195 (24.3)	220 (24.8)	134 (17.8)	110 (15.7)
25-30	149 (18.6)	120 (13.5)	190 (25.2)	170 (24.3)
>31	81 (10.1)	106 (11.9)	158 (20.9)	183 (26.1)
<i>Chi square p value</i>	0.07		0.24	
<b>Breastfeeding</b>				
Never	470 (58.5)	529 (59.5)	430 (57)	355 (50.6)
Ever	333 (41.5)	360 (40.5)	325 (43)	346 (49.4)
<i>Chi square p value</i>	0.68		0.02	
<b>Family history of breast cancer</b>				
No	687 (85.6)	786 (88.4)	578 (76.5)	584 (83.3)
Yes	116 (14.4)	103 (11.6)	177 (23.4)	117 (16.7)
<i>Chi square p value</i>	0.08		0.001	
<b>Past benign breast disease</b>				
No	547 (68.3)	685 (77.1)	431 (57.6)	466 (66.7)
Yes	254 (31.7)	203 (22.9)	317 (42.4)	232 (33.3)
<i>Chi square p value</i>	<0.001		<0.001	

	AA women		EA women	
	Cases (n=803) N (%)	Controls (n=889) N (%)	Cases (n=755) N (%)	Controls (n=701) N (%)
<b>HRT use</b>				
Never	682 (85.4)	785 (88.5)	559 (74)	540 (77.1)
Ever	117 (14.6)	102 (11.5)	196 (26)	160 (22.9)
<i>Chi square p value</i>	0.06		0.17	
<b>Oral contraceptive use</b>				
Never	333 (41.5)	387 (43.6)	261 (34.7)	203 (29)
Ever	470 (58.5)	501 (56.4)	492 (65.3)	498 (71)
<i>Chi square p value</i>	0.38		0.02	
<b>BMI</b>				
Underweight/Normal	151 (18.8)	157 (17.7)	342 (45.3)	317 (45.3)
Overweight	235 (29.3)	255 (28.7)	206 (27.3)	191 (27.3)
Obese	416 (51.9)	477 (53.7)	207 (27.4)	192 (27.4)
<i>Chi square p value</i>	0.73		0.99	
<b>Estrogen receptor status</b>				
ER positive	409 (69)	-	413 (82.1)	-
ER negative	184 (31)	-	90 (17.9)	-

**Table 2**  
**Distribution of the frequency of consuming energy-dense foods, fast foods, and sugary drinks per week among women participating in WCHS, n=3148**

	AA		EA	
	Cases	Controls	Cases	Controls
<b>Total caloric intake (kcal)</b>				
Mean ± SD	1807.79±1227.46	1765.84±1132.48	1683.32±721.79	1742.19±721.18
Median	1510.08	1553.52	1570.47	1657.20
<i>P value</i> *	0.97		0.06	
<b>Frequency of ED foods intake/week</b>				
Mean ± SD	8.24±8.57	8.46±8.70	8.16±6.90	7.64±6.64
Median	6.08	5.69	6.46	6.08
<i>P value</i> *	0.85		0.10	
<b>Frequency of fast foods intake/week</b>				
Mean ± SD	4.09±4.51	4.07±4.10	4.07±4.10	2.53±2.41
Median	2.77	2.85	2.15	2
<i>P value</i> *	0.53		0.07	
<b>Frequency of sugary drink intake/week</b>				
Mean ± SD	5.00±10.14	5.18±9.82	1.31±3.98	1.19±4.33
Median	1	1	0	0
<i>P value</i> *	0.67		0.06	

\* P values are from non-parametric tests

**Table 3**  
**Consumption of foods and drinks that promote weight gain and breast cancer risk among AA women by menopausal status**

(frequency of intake/week)	All women						Premenopausal (n=871)						Postmenopausal (n=821)					
	Ca (n)	Co (n)	ORI	95% CI	OR2	95% CI	Ca (n)	Co (n)	ORI	95% CI	OR2	95% CI	Ca (n)	Co (n)	ORI	95% CI	OR2	95% CI
<b>Energy dense foods</b>																		
Q1 ( 3)	226	256	Ref		Ref		99	120	Ref		Ref		127	136	Ref		Ref	
Q2 (3.1-6)	173	214	1.07	0.80-1.42	1.04	0.78-1.39	91	121	1.15	0.76-1.75	1.13	0.74-1.72	82	93	1.01	0.66-1.53	0.99	0.65-1.51
Q3 (6.1-11)	210	196	1.52	1.14-2.02	1.43	1.06-1.92	114	108	1.54	1.01-2.33	1.43	0.93-2.19	96	88	1.47	0.97-2.23	1.42	0.92-2.18
Q4 (>11)	194	223	1.31	0.98-1.75	1.15	0.82-1.61	104	114	1.73	1.13-2.65	1.45	0.89-2.37	90	109	1.04	0.69-1.58	0.97	0.59-1.57
<i>P for linear trend</i>				0.05		0.41				0.008		0.14				0.77		0.93
<b>Fast foods</b>																		
Q1 ( 1)	155	166	Ref		Ref		59	71	Ref		Ref		96	95	Ref		Ref	
Q2 (1.1-3)	271	298	1.24	0.92-1.66	1.22	0.91-1.64	125	144	1.52	0.95-2.44	1.49	0.93-2.39	146	154	1.09	0.74-1.63	1.08	0.73-1.61
Q3 (3.1-5)	158	177	1.25	0.90-1.75	1.20	0.86-1.68	85	95	1.71	1.02-2.86	1.61	0.95-2.71	73	82	1.01	0.64-1.60	0.98	0.61-1.57
Q4 (>5)	219	248	1.53	1.10-2.13	1.36	0.94-1.96	139	153	2.30	1.38-3.82	1.97	1.13-3.43	80	95	1.11	0.70-1.77	1.02	0.60-1.74
<i>P for linear trend</i>				0.02		0.18				0.002		0.04				0.77		0.93
<b>Sugary drinks</b>																		
None	285	315	Ref		Ref		119	146	Ref		Ref		166	169	Ref		Ref	
Low ( 3)	281	282	1.16	0.91-1.48	1.15	0.90-1.48	147	147	1.41	0.98-2.03	1.42	0.99-2.05	134	135	0.92	0.65-1.31	0.91	0.64-1.29
High (>3)	237	291	1.07	0.83-1.39	0.97	0.74-1.27	142	169	1.35	0.92-1.96	1.17	0.79-1.74	95	122	0.81	0.56-1.18	0.76	0.51-1.12
<i>P for linear trend</i>				0.94		0.45				0.41		0.98				0.30		0.18

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Ca – case; Co- control

ORI : Adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status, parity, age at first birth, breastfeeding status, family history of breast cancer, HRT use, OC use, history of benign breast disease, study site, BMI, total MET hours per week

OR2: Further adjusted for total energy intake

**Table 4**  
**Consumption of foods and drinks that promote weight gain and breast cancer risk among AA women by hormone receptor status**

(frequency of intake/week)	ER+Ca (n)		ER-Ca (n)		Co (n)		ER+ vs. Co		ER- vs. Co		
	OR1	95% CI	OR2	95% CI	OR1	95% CI	OR2	95% CI	OR1	95% CI	
<b>Energy dense foods</b>											
Q1 ( 3)	99	63	256	Ref	Ref	Ref	Ref	Ref	Ref	Ref	
Q2 (3.1-6)	100	30	214	1.47	1.03-2.09	1.43	1.00-2.04	0.63	0.39-1.03	0.61	0.37-1.01
Q3 (6.1-11)	108	50	196	1.80	1.26-2.58	1.68	1.17-2.43	1.15	0.73-1.78	1.07	0.67-1.69
Q4 (>11)	102	41	223	1.65	1.15-2.37	1.40	0.92-2.13	0.81	0.51-1.30	0.69	0.40-1.19
<i>P for linear trend</i>				0.02			0.25		0.72		0.34
<b>Fast foods</b>											
Q1 ( 1)	77	41	166	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Q2 (1.1-3)	144	55	298	1.33	0.93-1.91	1.31	0.91-1.88	0.89	0.55-1.44	0.87	0.54-1.41
Q3 (3.1-5)	68	44	177	1.08	0.71-1.65	1.03	0.67-1.57	1.25	0.75-2.09	1.19	0.70-2.01
Q4 (>5)	120	44	248	1.73	1.16-2.59	1.49	0.95-2.33	0.91	0.53-1.56	0.79	0.43-1.44
<i>P for linear trend</i>				0.02			0.19		0.96		0.59
<b>Sugary drinks</b>											
None	143	63	315	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Low ( 3)	145	64	282	1.23	0.91-1.66	1.23	0.91-1.65	1.11	0.74-1.66	1.11	0.74-1.66
High (>3)	121	57	291	1.14	0.83-1.57	1.01	0.73-1.41	0.97	0.63-1.49	0.91	0.58-1.41
<i>P for linear trend</i>				0.74			0.65		0.69		0.48

Ca – case; Co- control

OR1: Adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status, parity, age at first birth, breastfeeding status, family history of breast cancer, HRT use, OC use, history of benign breast disease, study site, BMI, total MET hours per week

OR2: Further adjusted for total energy intake

**Table 5**  
**Consumption of foods and drinks that promote weight gain and breast cancer risk among EA women by menopausal status**

(frequency of intake/week)	All women											
	Premenopausal (n=774)					Postmenopausal (n=682)						
	Ca (n)	Co (n)	ORI	95% CI	OR2	95% CI	Ca (n)	Co (n)	ORI	95% CI	OR2	95% CI
<b>Energy dense foods</b>												
Q1 ( 3)	181	198	Ref		Ref		93	90	Ref		Ref	
Q2 (3.1-6)	175	151	1.28	0.93-1.75	1.32	0.96-1.81	76	86	0.81	0.51-1.29	0.80	0.50-1.27
Q3 (6.1-11)	202	184	1.27	0.94-1.73	1.36	0.99-1.86	112	105	0.97	0.63-1.50	0.93	0.59-1.45
Q4 (>11)	197	168	1.37	1.01-1.87	1.57	1.10-2.24	108	104	1.14	0.74-1.75	1.04	0.64-1.70
<i>P for linear trend</i>				0.08		0.02				0.31		0.59
<b>Fast foods</b>												
Q1 ( 1)	164	187	Ref		Ref		69	73	Ref		Ref	
Q2 (1.1-2)	196	180	1.49	1.09-2.03	1.51	1.11-2.07	87	98	1.16	0.72-1.88	1.15	0.71-1.86
Q3 (2.1-3)	156	132	1.67	1.19-2.35	1.74	1.23-2.45	86	76	1.53	0.92-2.52	1.49	0.89-2.48
Q4 (>3)	239	202	1.60	1.16-2.19	1.75	1.24-2.46	147	138	1.46	0.92-2.31	1.39	0.85-2.27
<i>P for linear trend</i>				0.02		0.005				0.11		0.21
<b>Sugary drinks</b>												
None	524	512	Ref		Ref		248	263	Ref		Ref	
Low ( 1)	125	120	1.08	0.80-1.45	1.09	0.80-1.46	86	76	1.13	0.77-1.65	1.10	0.75-1.62
High (>1)	106	69	1.28	0.90-1.84	1.31	0.91-1.89	55	46	1.01	0.62-1.64	0.95	0.58-1.56
<i>P for linear trend</i>				0.18		0.15				0.97		0.80

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Ca – case; Co- control

ORI: Adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status, parity, age at first birth, breastfeeding status, family history of breast cancer, HRT use, OC use, history of benign breast disease, study site, BMI, total MET hours per week; OR2: Further adjusted for total energy intake

**Table 6**  
**Consumption of foods and drinks that promote weight gain and breast cancer risk among EA women by hormone receptor status**

(frequency of intake/week)	ER+Ca (n)		ER-Ca (n)		Co (n)		ER+ vs. Co		ER- vs. Co		
	OR1	95% CI	OR2	95% CI	OR1	95% CI	OR2	95% CI	OR1	95% CI	
<b>Energy dense foods</b>											
Q1 ( 3)	103	18	198	Ref	Ref	Ref	Ref	Ref	Ref	Ref	
Q2 (3.1-6)	89	22	151	1.10	0.75-1.61	1.17	0.79-1.72	1.48	0.75-2.93	1.47	0.74-2.92
Q3 (6.1-11)	109	28	184	1.22	0.85-1.76	1.36	0.93-1.99	1.54	0.80-2.99	1.52	0.76-3.01
Q4 (>11)	112	22	168	1.38	0.95-2.00	1.75	1.14-2.69	1.47	0.74-2.94	1.43	0.65-3.13
<i>P for linear trend</i>					0.08		0.01			0.37	0.50
<b>Fast foods</b>											
Q1 ( 1)	89	21	187	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Q2 (1.1-2)	96	21	180	1.37	0.94-2.02	1.42	0.96-2.08	1.07	0.55-2.10	1.06	0.54-2.09
Q3 (2.1-3)	95	17	132	1.86	1.24-2.79	1.98	1.31-3.00	1.25	0.60-2.59	1.24	0.59-2.59
Q4 (>3)	133	31	202	1.60	1.09-2.34	1.84	1.22-2.77	1.46	0.76-2.79	1.42	0.70-2.87
<i>P for linear trend</i>					0.03		0.006		0.22		0.29
<b>Sugary drinks</b>											
None	292	58	512	Ref	Ref	Ref	Ref	Ref	Ref	Ref	Ref
Low ( 1)	67	15	120	1.00	0.70-1.43	1.01	0.71-1.45	1.08	0.57-2.05	1.08	0.57-2.05
High (>1)	54	17	69	1.35	0.88-2.07	1.40	0.91-2.16	1.86	0.96-3.61	1.82	0.93-3.57
<i>P for linear trend</i>					0.17		0.13		0.07		0.08

Ca – case; Co- control

OR1: Adjusted for age, ethnicity, country of origin, education, age at menarche, menopausal status, parity, age at first birth, breastfeeding status, family history of breast cancer, HRT use, OC use, history of benign breast disease, study site, BMI, total MET hours per week

OR2: Further adjusted for total energy intake