Nutrition and Cancer

ISSN: (Print) (Online) Journal homepage: https://www.tandfonline.com/loi/hnuc20

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To cite this article: M. Karen Flores-García, Ángel Mérida-Ortega, Edgar Denova-Gutiérrez & Lizbeth López-Carrillo (2021) Dietary Patterns and Breast Cancer Risk in Women from Northern Mexico, Nutrition and Cancer, 73:11-12, 2763-2773, DOI: 10.1080/01635581.2020.1860241

To link to this article: https://doi.org/10.1080/01635581.2020.1860241

NUTRITION AND CANCER
2021, VOL. 73, NOS. 11–12, 2763–2773
https://doi.org/10.1080/01635581.2020.1860241

Dietary Patterns and Breast Cancer Risk in Women from Northern Mexico

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ABSTRACT

We evaluated the association between dietary patterns and breast cancer (BC) subtypes among women from Northern Mexico. From a study of incident cases and population controls that was carried out from 2007 to 2011, a subsample of 509 cases matched 1:1 by age with 509 controls was selected. Information about expression of estrogen receptor (ER), progesterone receptor (PR), and human epidermal growth factor 2 (HER2) was available from medical records to classify BC on luminal (ER + and/or PR+/HER2–), HER2+ (ER+/– and/or PR+/–/HER2+), or triple negative (ER– and PR–/HER2–). Dietary information was gathered using a semiquantitative food frequency questionnaire and a factor analysis was used to obtain dietary patterns. The association between each dietary pattern and BC molecular subtypes was assessed through conditional logistic regression models. Two dietary patterns were identified. The first (mainly characterized by meat, high fat, and sugary cereals) was positively associated with BC (odds ratio, OR = 12.62; 95% CI: 7.42, 21.45); the second (consisting of corn, legumes, and other vegetables) was inversely associated with BC (OR = 0.50; 95% CI: 0.40, 0.62). Both associations remained significant by BC molecular subtypes. These findings could contribute to the development of public health strategies for BC prevention.

ARTICLE HISTORY
Received 11 September 2020
Accepted 22 November 2020

Introduction

Breast cancer (BC) incidence has increased from 39.0 in 2008 to 46.3 per 100,000 women in 2018 and shows a wide geographical variation worldwide (1,2), which may be explained by differences in lifestyles and environmental factors, including diet (3). BC tumors can express estrogen (ER) and/or progesterone (PR) hormone receptors and/or the human epidermal growth factor receptor 2 (HER2), or none of those markers. Based on them, BC can be divided into three main subtypes: luminal (ER + and/or PR+/HER2–), HER2+ (ER+/– and/or PR+/–/HER2+), or triple negative (TN) (ER– and PR–/HER2–) (4–7). BC subtypes have different incidences and survival and respond differentially to treatment (4,5).

According to the latest report from the World Cancer Research Fund (WCRF), alcohol is the only dietary risk factor for BC with convincing scientific evidence; nevertheless, there are some foods and nutrients that have been linked to BC, but lack sufficient evidence (8). To improve understanding of the relationship between diet and BC, the use of dietary patterns has been proposed as an alternative strategy to evaluate the whole diet (9,10).

Accordingly, in a recent meta-analysis of 32 epidemiological studies (14 cohorts and 18 case–control), a 14% BC excess risk was estimated for women with a "Western" dietary pattern, characterized by consumption of red and processed meat, as well as potatoes and foods high in fats and sugars. In contrast, a 18% reduction in BC risk was identified with a dietary pattern named "Prudent," represented by fruits, vegetables, fish, whole grains, and low-fat dairy products (10). Although this information contributes to the evidence of diet's role in BC development, it does not consider the heterogeneity of this disease.

In some studies, performed in the United States, Europe, and Asia, inverse associations between ER + and PR + or - BC and "Prudent" as well as "Mediterranean" dietary patterns have been reported. The latter pattern was characterized by fruits,

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Supplemental data for this article is available online at https://doi.org/10.1080/01635581.2020.1860241.

vegetables, fish, olive oil, and legumes. In addition, ER + BC has been positively associated with "Western" and "Alcohol" dietary patterns, which included the consumption of processed meats and alcoholic beverages (3,11–14). Moreover, HER2+ BC subtype has been inversely associated with the "Prudent/Vegetarian" pattern (15), as well as positively associated with the "Western" dietary pattern (13). Furthermore, a "Mediterranean" dietary pattern was inversely associated with TN (11).

Epidemiological evidence regarding dietary patterns and BC molecular subtypes is scarce and contradictory (10). By classifying the subtypes solely based on the expression of ER and/or PR, comparability may be difficult, since the presence or absence of HER2 overexpression is not explicit. In addition, there is little information on the relationship between BC and Eastern dietary patterns and the evidence is almost null regarding dietary patterns in Latin America, Africa, and other regions.

In Mexico, some authors have identified dietary patterns as the "Western," which also include local foods like corn tortillas (16–18). Likewise, the "Prudent" pattern has been identified with some additional foods such as fresh legumes (16,17,19). A third pattern has been characterized by Mexican foods, refined cereals, as well as animal proteins and fats (16,18,20,21).

Thus, the purpose of this study was to assess the relationship between dietary patterns and BC molecular subtypes in women from Northern states of Mexico.

Methods

Previously, our research group carried out a population-based case–control study from 2007 to 2011 in five states of Northern Mexico (Chihuahua, Coahuila, Durango, Nuevo León, and Sonora). The aim of the original study was to evaluate environmental and genetic factors associated with BC; detailed information regarding its methodology have been reported elsewhere (22). Briefly, in the original study, 1,045 histopathologically confirmed BC cases were identified in 17 hospitals, both public and academic. The inclusion criteria included a minimum age of 18 years, no personal history of any other type of cancer, and at least one year of residency in the study area. A total of 1,030 controls with no personal history of cancer and at least one year of residence in the study area, were matched by age to cases (±5 years). Controls were identified through the Master Sample Framework used in the Mexican National Health and Nutrition Survey (ENSANUT, by its Spanish acronym), which provides a probabilistic list of households in urban and rural areas. In households where there was more than one eligible woman, only one participant was chosen at random, while if there was no eligible woman or she declined participation, another house was systematically located according to standardized procedures. The participation rates were above 90% for cases and controls. Both cases and controls were interviewed face to face about sociodemographic, reproductive, lifestyle, and dietary characteristics. Height and weight were obtained to calculate body mass index (BMI). Patients were interviewed before any type of treatment and after receiving the diagnosis (the average time from diagnosis to interview was two months). This study was approved by the Ethics, Biosafety, and Research Committees of the National Institute of Public Health. The present study was developed and performed according to the Declaration of Helsinki guidelines; a written informed consent was obtained from all participants.

For the purpose of this report, 509 cases that had information on BC molecular subtype were included, which were age-matched 1:1, with 509 controls. In addition, information about tumor stage according to the Tumor, Node, Metastasis (TNM) system was available: stage *in situ* ($n = 3$), I ($n = 57$), II ($n = 277$), III ($n = 130$), and IV ($n = 14$), no information ($n = 28$) (23).

Diagnosis in cases was established through the immunohistochemical expression of ER, PR, and HER2 in breast tumors of the patients. This information was available in clinical records of each participating hospital. The tumor was ER positive (ER+) and/or PR positive (PR+) if at least 1% of cells were reactive (24). And those who showed a pattern of intense and complete staining in at least 30% of cells were considered HER2+ (25,26). Molecular subtypes were: luminal (ER + and/or PR +/HER2–), HER2+ (ER+/– and/or PR+/–/HER2+), or TN (ER– and PR–/HER2–).

Food Consumption

To evaluate food consumption, an already validated semiquantitative food frequency questionnaire (FFQ) was used (27). Participants were asked to report their usual food consumption during the last year prior to diagnosis between cases and prior to interview between controls. This questionnaire consists of previously defined portion sizes of 119 foods and 14 dishes, with 10 response options from "never" up to "six or more times a day." The portions were as follows: a glass (milk and wine), a cup (yogurt, some fruits and

vegetables, tea, juices, alcoholic and nonalcoholic beverages), a spoon (oils, sour cream, sauces, and nuts), a slice (cheeses, some fruits, and meats), a dish (local vegetables and dishes), and a piece (some fruits and breads).

Previously, our research group reviewed the consistency of foods included in our questionnaire with those in the reference tables of the United States Department of Agriculture (28), from which energy consumption was obtained. Two food items were not found in those tables (quince and tejocote) and their energy values were gathered from the reference tables of the National Institute of Medical Sciences and Nutrition in Mexico "Salvador Zubiran" (29). For each participant, daily intake of total energy was estimated based on food portion size and its frequency of consumption. According to their availability throughout the year, the frequency of consumption of fruits and vegetables was adjusted. For example, only half the consumption of plums was accounted for because they are available during 6 mo of the year only.

Dietary Patterns

Individual foods and beverages contained in our FFQ were categorized into 27 food groups based on: Similarity of nutrient content (e.g., fat, carbohydrates, protein, vitamins, alcohol), their added sugar content (e.g., added or not), and the type of fat (e.g., saturated or vegetable fat). Some individual foods were considered as groups by themselves because their nutritional content did not meet the criteria to belong to any group (egg, chicken, and root beer), because they were consumed very frequently within the population (corn tortilla), or due to a special culinary use (corn). Likewise, two more food groups were considered: Fast food (pizza and hamburger) and 12 Mexican dishes. Energy consumption from each food group was converted to a percentage of the total daily energy intake that was later standardized using Z score. Result values were used to obtain the dietary patters and factor loadings through factor analysis among the entire study population. Factors with an eigenvalue greater than 1.5 were maintained to facilitate interpretability. Each factor was defined by a subset of at least four food groups with an absolute load factor equal to or greater than –0.20 or 0.20. If a food group had a load factor $\geq$0.20 in both patterns, only the one with the highest load factor was considered in the pattern, because individuals tend to follow the pattern with the higher score (30). The Kaiser–Meyer–Olkin index was computed to assess the adequacy of the data in relation to the factor analysis, observing a value of 0.5. In addition, the Bartlett sphericity test was performed to evaluate the correlations between the variables, and we observed a P value of 1.0.

Estrogenic Index

We estimated years of exposure to endogenous estrogens through an estrogenic index that has been used elsewhere (5). For postmenopausal women, the difference in years between the age at menopause minus age at menarche was obtained; likewise, for premenopausal women the difference of age at the time of the study minus age at menarche was estimated; from those respective results, the pregnancies and breastfeeding duration in years were subtracted.

Statistical Analysis

Sociodemographic, reproductive, and lifestyle characteristics were compared between included and not included cases and controls, using the Mann–Whitney U, Student t and χ^2 test, as appropriate. Similarly, according to the distribution of tertiles, those characteristics were compared between each BC subtype and their controls.

Conditional logistic regression models were used to evaluate the association between the dietary patterns in continuous scale (with and without fast food and Mexican dishes) and each BC molecular subtype. Potential confounders (alcohol, smoking, BMI, estrogenic index, family history of BC, years of education, and total energy) were evaluated by comparing the adjusted versus crude odds ratios, those that were different by more than 10% were maintained as covariates in multivariable models: total energy, estrogenic index, years of education, and family history of BC. In addition, the full model was adjusted by each dietary pattern. Results with a P value $<$0.05 were considered statistically significant.

All statistical analyses were done using the statistical package Stata version 14.0 (StataCorp, College Station, TX).

Results

Most selected characteristics did not show significant differences between included and not included cases and controls. However, compared to the included, the nonincluded cases had a significantly higher percentage of BC family history. Likewise, age and age at

menopause of the included controls was lower than that of the nonincluded (Supplementary Table S1).

Compared to their respective controls, BC family history and age at first pregnancy were greater in the three molecular subtypes. Despite the fact that the median age at menarche and education in luminal cases and controls were the same, they were statistically different because there was higher proportion of women with a younger age at menarche and with more years of education among cases. In addition, HER2+ cases had statistically significant more years of education. On the other hand, luminal and HER2+ cases had fewer pregnancies and breastfeeding, while the estrogenic index was statistically significantly higher among luminal and HER2+ BC subtypes. Among postmenopausal women, BMI was significantly lower in HER2+ cases. Only luminal BC cases reported significantly higher alcohol consumption. Due to the small sample size, BC family history and alcohol consumption are not shown in Table 1.

Table 2 shows two dietary patterns obtained without considering fast food and Mexican dishes. The first, with 13.70% of explained variance, was characterized mainly by having positive loads in the consumption of fruits, fish and other seafoods, red meat, and fat and sugary dairy products, as well as a negative load in the consumption of corn tortillas. The second, with 7.49% of variance, resulted in higher positive loads of consumption of cruciferous, corn, starchy, and allium vegetables, and negative loads of high fat and sugary cereals, red meat, and saturated fat. In both patterns, at least 43% of the total energy was comprised of four food groups: corn tortillas, refined cereals, vegetable oils, and legumes. These results were maintained when fast food and Mexican dishes were included in the patterns estimation (data not shown).

BC was 12.62 (95% CI: 7.42, 21.45) fold times more frequent by unit change of pattern 1, after adjusting for energy, estrogenic index, education, and family history of BC. This association remained significantly positive in all molecular subtypes: luminal (odds ratio, OR = 10.16; 95% CI: 5.33, 19.37), HER2+ (OR = 20.94; 95% CI: 4.42, 99.21), and TN (OR = 17.62; 95% CI: 4.41, 70.39). In contrast, for each unit change in the pattern 2, inverse associations with BC (OR = 0.50; 95% CI: 0.40, 0.62) and molecular subtypes were observed: luminal (OR = 0.50; 95% CI: 0.37, 0.67), HER2 + (OR = 0.34; 95% CI: 0.17, 0.65), and TN (OR = 0.55; 95% CI 0.36, 0.83) (Table 3). The above results did not change when adjusting by each other dietary pattern (data not shown), nor with patterns derived from the inclusion of fast foods and Mexican dishes (Supplementary Table S2).

Discussion

Our results showed the existence of two dietary patterns in the study sample. The first was positively associated with BC, whereas the second was inversely associated. These results remained by molecular subtype. The first pattern was like the called "Western" (3), and the second to the "Healthy/Prudent" (11).

Our results are consistent with those in previous reports that have evaluated the association between dietary patterns and different molecular subtypes of BC. In a meta-analysis that included seven cohort and five case–control studies conducted in Europe, Asia, United States, Australia, and Uruguay, it was reported that the "Western" dietary pattern significantly increased the risk of ER+ and/or PR + BC (relative risk, RR 1.18; 95% CI 1.04, 1.33). The "Prudent" dietary pattern evaluated in 11 of these studies was inversely and significantly associated with ER+ and/or PR + BC (RR 0.80; 95% CI 0.66, 0.98) and ER– and/or PR– (RR 0.68; 95% CI 0.55, 0.83) (10). Additionally, in a study of 1,017 cases and 1,017 controls carried out in Spain that considered HER2 expression, it was found that the "Mediterranean" pattern was inversely associated with ER+/PR+/HER2– (OR 0.57; CI 95% 0.40, 0.82) and ER–/PR–/HER2– (OR 0.32; CI 95% 0.15, 0.66) BC molecular subtypes (11).

Some biological mechanisms have been described that could explain the positive association between pattern 1 (like "Western") and luminal BC. Exogenous hormones found in chicken, red and processed meat activate hormonal receptors in breast tissue and stimulate tumor growth through its proliferative and metastatic activity (31–34). In addition, the consumption of foods with a high glycemic index such as sugary drinks and some fruits can increase the endogenous production and stimulation of estrogens (35–38). Also, foods with a high glycemic index increase insulin-like growth factor-1 (IGF-1), which promotes the growth, proliferation, and survival of tumor cells in the three BC molecular subtypes (35). Furthermore, there are mechanisms that may not be specific for any of these molecular subtypes. As an example, red and processed meats have been reported to contain heme iron and heterocyclic amines that enhance the formation of *N*-nitroso compounds (NOC) and may contribute to the development of BC through DNA alkylation (34,39). The saturated fats in

Table 1. Selected characteristics in the study population according to breast cancer molecular subtype sets.

Characteristics	(*n*)	Tertile 1	Tertile 2	Tertile 3	Total
Age, years [Mean (SD)]					
Luminal	(289)	41.67 (5.63)	55.25 (2.95)	68.67 (6.19)	54.78 (12.28)
Controls	(289)	41.64 (5.69)	55.25 (2.95)	68.62 (6.14)	54.75 (12.28)
HER2 +	(117)	38.38 (4.06)	49.39 (2.14)	60.69 (6.07)	49.39 (10.19)
Controls	(117)	38.38 (3.99)	49.39 (2.14)	60.69 (6.07)	49.39 (10.18)
TN	(103)	36.70 (5.65)	51.31 (3.48)	69.68 (6.62)	51.59 (14.42)
Controls	(103)	36.70 (5.60)	51.31 (3.48)	69.71 (6.71)	51.60 (14.44)
Age at menarche, years [p50 (p25, p75)]					
Luminal	(289)	12 (11, 12)	13 (13, 13)	15 (14, 15)	13 (12, 14)
Controls	(289)	12 (11, 12)	13 (13, 14)	15 (15, 16)	13 (12, 14)*
HER2+	(116)	12 (11, 12)	13 (13, 14)	15 (15,16)	13 (12, 14)
Controls	(117)	12 (11, 12)	14 (13, 14)	16 (15, 17)	13 (12, 14)
TN	(103)	12 (11, 12)	13 (13, 13)	14 (14, 15)	13 (12, 14)
Controls	(103)	12 (11, 12)	13 (13, 14)	15 (15, 16)	13 (12, 14)
Education, years [p50 (p25, p75)]					
Luminal	(288)	4.00 (2.00, 6.00)	9.00 (9.00, 9.00)	12.00 (12.00, 16.00)	6.00 (4.00, 11.50)
Controls	(289)	2.00 (1.00, 3.00)	6.00 (5.00, 6.00)	9.00 (9.00, 12.00)	6.00 (3.00, 6.00)*
HER2+	(117)	6.00 (4.00, 6.00)	9.00 (9.00, 11.00)	12.00 (12.00, 15.00)	9.00 (6.00, 12.00)
Controls	(117)	4.50 (2.00, 6.00)	9.00 (9.00, 9.00)	12.00 (12.00, 12.00)	6.00 (4.00, 9.00)*
TN	(103)	5.00 (2.00, 6.00)	9.00 (9.00, 9.00)	12.00 (12.00, 16.00)	6.00 (4.00, 9.00)
Controls	(103)	3.00 (1.00, 4.00)	6.00 (6.00, 6.00)	9.00 (9.00,12.00)	6.00 (4.00,9.00)
Number of pregnancies [p50 (p25, p75)]					
Luminal	(262)	2.00 (2.00, 3.00)	4.00 (4.00, 5.00)	8.00 (6.00, 9.00)	4.00 (3.00, 6.00)
Controls	(281)	2.50 (2.00, 3.00)	5.00 (4.00, 6.00)	9.00 (8.00, 12.00)	5.00 (3.00, 7.00)*
HER2+	(114)	2.00 (1.00, 2.00)	3.00 (3.00, 4.00)	5.50 (5.00, 8.00)	3.00 (2.00, 5.00)
Controls	(114)	3.00 (2.00,3.00)	5.00 (4.00, 5.00)	7.50 (6.00, 9.00)	4.00 (3.00, 6.00)*
TN	(93)	2.00 (2.00, 3.00)	4.00 (4.00, 5.00)	7.00 (6.00, 8.00)	4.00 (3.00, 6.00)
Controls	(98)	3.00 (2.00, 3.00)	4.00 (4.00, 5.00)	8.00 (6.00, 12.00)	4.00 (3.00, 6.00)
Age at first pregnancy, years [p50 (p25, p75)][a]					
Luminal	(259)	18.00 (17.00, 19.00)	22.00 (21.00, 23.00)	29.00 (26.00, 32.00)	22.00 (19.00, 25.00)
Controls	(280)	17.00 (16.00, 18.00)	20.00 (19.00, 20.00)	24.50 (23.00, 27.00)	19.00 (17.00, 22.00)*
HER2+	(110)	18.00 (17.00, 19.00)	23.00 (22.00, 24.00)	29.00 (27.00, 32.00)	22.00 (19.00, 26.00)
Controls	(114)	17.00 (16.00, 18.00)	19.00 (19.00, 20.00)	24.00 (23.50, 26.50)	19.00 (17.00, 23.00)*
TN	(92)	18.00 (16.00, 18.00)	21.00 (20.00, 22.00)	27.00 (25.00, 28.00)	21.00 (18.00, 24.00)
Controls	(98)	16.00 (15.00, 18.00)	20.00 (20.00, 21.00)	24.00 (22.00, 28.00)	19.00 (17.00, 22.00)*
Total breastfeeding, months [p50 (p25, p75)][a]					
Luminal	(262)	2.00 (0.00,6.00)	20.00 (16.00, 27.00)	79.00 (60.00, 108.00)	19.50 (5.00, 60.00)
Controls	(281)	9.00 (3.00, 16.00)	42.00 (30.00, 51.00)	120.00 (90.00, 183.50)	41.00 (15.00, 84.00)*
HER2+	(114)	0.00 (0.00, 3.00)	12.00 (10.00, 17.00)	38.00 (29.00, 56.00)	12.00 (2.00, 29.00)
Controls	(114)	6.0 (0.00, 11.00)	36.00 (32.00, 50.00)	96.00 (78.00, 120.00)	36.00 (11.00, 78.00)*
TN	(93)	4.00 (1.00, 7.00)	30.00 (22.00, 36.00)	75.00 (50.00, 118.00)	30.00 (7.00, 50.00)
Controls	(98)	9.00 (0.00,16.00)	34.50 (26.50, 42.00)	129.00 (72.00, 161.00)	32.50 (15.00, 72.00)
Age at menopause, years [p50 (p25, p75)]					
Luminal	(191)	41.00 (37.50, 44.50)	48.00 (47.00, 50.00)	52.00 (52.00, 54.00)	47.00 (42.00, 50.00)
Controls	(199)	40.00 (36.00, 40.50)	45.00 (44.00, 47.00)	50.00 (50.00, 51.00)	45.00 (40.00, 50.00)*
HER2+	(62)	41.00 (36.00, 44.00)	48.00 (48.00, 50.00)	52.50 (52.00, 54.00)	48.00 (44.00, 50.00)
Controls	(72)	39.00 (35.00, 40.00)	45.00 (43.00, 46.00)	50.00 (49.00, 50.00)	44.50 (40.00, 48.00)*
TN	(65)	37.50 (32.00, 43.00)	49.00 (47.00, 50.00)	53.00 (52.00, 55.00)	48.00 (43.00, 50.00)
Controls	(61)	40.00 (35.00, 41.00)	45.00 (45.00, 46.00)	50.50 (50.00,53.00)	45.00 (40.00, 50.00)
Body mass index, kg/m^2 [p50 (p25, p75)]					
Premenopause					
Luminal	(104)	23.32 (22.59, 24.64)	28.30 (27.14, 29.38)	33.30 (31.33, 36.98)	28.29 (24.51, 31.28)
Controls	(91)	23.95 (22.52, 26.30)	29.71 (28.19, 31.00)	36.02 (33.76, 40.46)	29.55 (26.14, 33.76)
HER2+	(57)	23.31 (20,24, 24.80)	28.14 (26.17, 29.75)	34.70 (32.42, 36.17)	28.14 (24.80,32.42)
Controls	(47)	22.63 (21.47, 25.02)	28.18 (27.26, 29.68)	36.58 (33.20, 38.95)	28.16 (24.12, 33.20)
TN	(43)	22.91 (21.33, 25.33)	28.09 (27.34, 29.09)	33.55 (32.45, 36.12)	27.91 (24.03, 32.45)
Controls	(45)	23.53 (21.93, 26.30)	30.10 (28.67, 31.24)	36.74 (35.09, 43.43)	30.10 (26.30, 35.09)
Postmenopause					
Luminal	(183)	25. 40 (23.70, 26.48)	29.84 (28.88, 31.20)	35.42 (34.11, 39.08)	29.84 (26.48, 34.11)
Controls	(198)	25.07 (23.83, 26.67)	29.64 (28.80, 30.63)	35.21 (33.33, 38.87)	29.64 (26.67, 33.33)
HER2+	(57)	22.99 (21.80, 24.65)	28.73 (27.99, 29.55)	33.33 (32.03, 37.16)	28.73 (24.65, 32.03)
Controls	(70)	25.20 (23.70, 26.26)	31.25 (30.43, 32.89)	35.88 (34.96, 41.41)	31.15 (26.16, 34.96)*
TN	(59)	25.66 (23.65, 25.98)	29.33 (28.33, 30.27)	34.28 (31.62, 39.54)	29.22 (25.88, 31.62)
Controls	(58)	25.76 (24.60, 27.06)	30.49 (29.15, 31.62)	36.40 (34.17, 39.12)	30.36 (26.84, 34.17)
Smoking, cigarettes/day [p50 (p25, p75)][b]					
Luminal	(77)	2.00 (1.00, 3.00)	6.00 (4.00,10.00)	20.00 (20.00, 30.00)	4.00 (2.00, 10.00)
Controls	(88)	2.00 (1.00, 3.00)	5.00 (4.50, 5.50)	10.00 (9.00, 20.00)	5.00 (2.00, 9.00)
HER2+	(31)	2.00 (1.00, 2.50)	5.00 (4.00, 5.00)	10.00, (10.00, 20.00)	4.00 (2.00, 10.00)
Controls	(30)	2.50 (1.00, 3.00)	5.00 (4.50, 5.00)	9.00 (7.00, 15.00)	3.00 (2.00, 6.00)

(*Continued*)

Table 1. Continued.

Characteristics	(*n*)	Tertile 1	Tertile 2	Tertile 3	Total
TN	(29)	2.00 (1.00, 2.50)	5.50 (4.50, 10.00)	20.00 (20.00, 40.00)	5.00 (2.00, 20.00)
Controls	(27)	2.00 (1.00, 3.00)	5.00 (4.00, 6.00)	20.00 (10.00, 20.00)	4.00 (2.00, 10.00)
Estrogenic index, years [p50 (p25, p75)]					
Luminal	(288)	19.25 (14.75, 22.00)	27.67 (26.00, 29.00)	33.92 (32.04, 36.33)	27.25 (21.54, 32.04)
Controls	(288)	12.54 (7.79, 16.25)	22.50 (21.00, 24.42)	30.00 (27.67, 32.50)	22.50 (16.25, 27.63)*
HER2+	(115)	21.08 (18.38, 23.42)	27.17 (25.75, 29.00)	34.08 (32.67, 35.75)	26.83 (23.17, 32.67)
Controls	(116)	14.50 (8.17, 18.50)	22.75 (21.17, 24.08)	29.71 (27.75, 32.00)	22.67 (18.50, 27.75)*
TN	(103)	17 (12.33, 19.08)	24.25 (23.17, 26.00)	31.83 (29.83, 34.58)	24.25 (19.00, 29.75)
Controls	(102)	11.75 (5.50, 18.00)	22.75 (21.25, 24.25)	29.75 (27.25, 32.50)	22.50 (16.75, 27.25)

HER2+ = human epidermal growth factor receptor 2-postive, TN = triple negative breast cancer.
[a]Numbers that add to less than the total number of controls or cases are due to missing values among women with pregnancies.
[b]Numbers that add to less than the total number of controls or cases are due to missing values among women who have smoked more than 100 cigarettes in their lives.
*Mann–Whitney's *U*-test *P* value <0.05.

red meat, cheese, and milk generate free radicals and mutagenic compounds, as well as modulate the expression of genes that regulate breast carcinogenesis (40). In addition, non-starchy vegetables, such as spinach and purslane, may contain pesticides and/or heavy metals (41–43), which could increase BC risk.

Likewise, the inverse relationship between pattern 2 ("Prudent" like) and luminal BC may be explained by the content of β-carotenes in some starchy vegetables (beets and carrots), that may inhibit cell proliferation, induced by17-β-estradiol, in ER + tumors (44,45). Similarly, some phytoestrogens from legumes, such as genistein, activate ERβ, which inhibits cell proliferation in breast tissue (46–48). Furthermore, fiber from vegetables, legumes, and corn inhibits the reabsorption of estrogens in the colon and increases their excretion in the feces (49–51). Moreover, lignans, contained in legumes, have been associated with a reduction in HER2 overexpression, as well as the inhibition of tumor cell proliferation through regulation of IGF-1 (52). In addition, inhibition of DNA oxidation and/or adduct formation, cell proliferation, and tumor growth are related to β-carotenes as well as organosulfur compounds, isothiocyanates, and indoles in cruciferous and allium vegetables (45,53–57).

To interpret our results, some methodological considerations must be considered. Our odds ratios between pattern 1 and BC were high compared to those reported in other studies where a "Western" dietary pattern similar to ours was used (10). In this regard, we cannot rule out the possibility that our cases over-reported the consumption of foods included in pattern 1, that they would have considered associated with BC development (i.e., red and processed meats, foods with added sugars, among others). In several previous case–control studies it has been suggested that this differential measurement error (recall bias) could partially explain the associations between the "Western" pattern and BC since in most cohort studies, where the measurement of diet was obtained before BC diagnosis, this association has not been found (9,10). To decrease the probability of this error, in our study the FFQ was applied as close as possible to the BC diagnosis date (average time between diagnosis and interview: two months). In addition, we previously evaluated the known reproductive risk factors of BC which were associated in the expected direction and magnitude (i.e., breastfeeding and number of pregnancies = protective; late age at first birth = risk, among others) (5). If there had been recall bias in the diet report, it is possible that there would have also been bias in the reporting of reproductive factors related to BC, and therefore the odds ratios of reproductive factors would have been distorted, which did not happen in this study (58). Therefore, we think that there is a low probability that our results regarding the relationship between the "Western" dietary pattern and BC are entirely due to recall bias.

The high magnitude of odds ratio between pattern 1 and BC could also be a real increase in risk due to the way foods are cooked. In this report, such information was not considered, but it is known that in the study area, it is common to roast meat (59). In this process, heterocyclic amines, polycyclic aromatic hydrocarbons, and NOC, that are carcinogenic compounds, are formed (60). Therefore, we cannot rule out that the way certain foods included in pattern 1 are cooked affects this relationship.

Most of our cases reported consumption of foods in pattern 1 (data not shown). This could be due to the small sample size that limits representativeness. However, cases were identified from several tertiary hospital units, which covered 90% of the study area population (61). The observed prevalence of BC luminal, HER2+, and TN was 56.77%, 22.99%, and 20.23%, respectively, which were like those reported in other studies in Mexican women (62–64). Likewise, when comparing the median total energy intake

Table 2. Consumption of food groups and factor-loading matrix for the two dietary patterns identified by factor analysis.

	Pattern 1			Pattern 2		
	% Energy/day [Mean (SD)]		Factor loading	% Energy/day [Mean (SD)]		Factor loading
Food groups	<p50	≥p50		<p50	≥p50	
Soda	4.18 (5.15)	3.78 (4.50)	–	5.11 (5.41)	2.84 (3.87)	−0.31
Diet soda	0.01 (0.06)	0.04 (0.12)	–	0.02 (0.10)	0.02 (0.10)	–
Saturated fats	0.63 (0.78)	1.31 (1.56)	0.33	1.23 (1.59)	0.70 (0.78)	−0.33
Fat dairy	3.45 (3.31)	6.56 (5.29)	0.38	4.81 (4.56)	5.19 (4.78)	–
Fat and sugary dairy	0.62 (1.08)	2.57 (3.10)	0.49	1.63 (2.56)	1.56 (2.47)	–
Red meat	2.01 (1.95)	6.26 (4.08)	0.61	5.19 (4.31)	3.08 (2.95)	−0.37
Processed meat	1.11 (1.20)	2.59 (2.26)	0.43	2.27 (2.23)	1.43 (1.53)	−0.30
Refined cereals	14.28 (11.61)	12.20 (7.09)	–	13.17 (9.98)	13.31 (9.36)	–
Starchy vegetables	1.73 (1.22)	2.71 (1.71)	–	1.65 (1.11)	2.79 (1.73)	0.47
Alcoholic drinks	0.03 (0.10)	0.10 (0.35)	–	0.10 (0.35)	0.02 (0.12)	–
Fish and other seafood	0.41 (0.52)	1.58 (1.46)	0.62	0.88 (1.10)	1.11 (1.36)	–
Cruciferous vegetables	0.30 (0.35)	0.53 (0.58)	–	0.24 (0.24)	0.60 (0.60)	0.54
Allium vegetables	0.26 (0.23)	0.25 (0.20)	–	0.19 (0.14)	0.32 (0.25)	0.40
Fruits	2.56 (2.13)	7.43 (4.53)	0.70	4.66 (3.95)	5.34 (4.59)	–
Non-starchy vegetables	2.63 (2.12)	5.11 (3.32)	0.48	3.14 (2.22)	4.61 (3.55)	–
Legumes	9.34 (6.85)	8.86 (6.06)	–	7.80 (5.70)	10.40 (6.92)	0.27
Corn	1.21 (1.21)	1.06 (1.22)	–	0.65 (0.64)	1.62 (1.44)	0.52
Vitamin E	0.76 (0.93)	1.88 (2.16)	0.42	1.12 (1.51)	1.52 (1.95)	–
Eggs	5.52 (5.42)	4.05 (3.87)	–	4.87 (5.03)	4.71 (4.48)	–
Poultry	0.90 (0.71)	1.38 (1.25)	0.32	0.94 (0.72)	1.34 (1.26)	–
Tea and coffee	2.57 (2.58)	2.30 (2.48)	–	2.45 (2.61)	2.42 (2.45)	–
Corn tortilla	34.80 (17.14)	14.07 (8.35)	−0.77	25.58 (19.14)	23.30 (14.49)	–
High fat and sugar cereals	0.91 (1.60)	3.64 (4.06)	0.44	3.32 (4.17)	1.23 (1.81)	−0.46
Sweets	0.04 (0.15)	0.23 (0.75)	0.20	0.21 (0.74)	0.07 (0.22)	−0.23
Vegetable oil	9.34 (5.26)	8.42 (4.73)	–	7.82 (4.65)	9.94 (5.16)	–
Corn-based drinks	0.39 (1.17)	1.10 (1.78)	0.25	0.97 (1.87)	0.53 (1.10)	–
Root beer	0.00 (0.00)	0.00 (0.08)	–	0.00 (0.08)	0.00 (0.01)	–
Eigenvalue			3.70			2.02
Variance explained			13.70			7.49

Table 3. Multivariable adjusted odds ratio for breast cancer according to molecular subtype.

	Cases/Controls (*n*)	Crude[a] OR (CI 95%)	Model 2[b] OR (CI 95%)	Model 3[c] OR (CI 95%)	Model 4[d] OR (CI 95%)
All participants					
Pattern 1	(509/509)	13.38 (8.15, 21.94)	12.69 (7.68, 20.97)	12.56 (7.57, 20.83)	12.62 (7.42, 21.45)
Pattern 2	(509/509)	0.60 (0.50, 0.70)	0.56 (0.46, 0.67)	0.55 (0.45, 0.67)	0.50 (0.40, 0.62)
Luminal A					
Pattern 1	(289/289)	11.69 (6.23, 21.92)	11.15 (5.92, 21.01)	10.83 (5.73, 20.49)	10.16 (5.33, 19.37)
Pattern 2	(289/289)	0.56 (0.45, 0.70)	0.54 (0.42, 0.69)	0.54 (0.41, 0.70)	0.50 (0.37, 0.67)
HER2+					
Pattern 1	(117/117)	25.02 (6.23, 100.43)	24.32 (5.32, 111.22)	23.94 (5.23, 109.49)	20.94 (4.42, 99.21)
Pattern 2	(117/117)	0.66 (0.45, 0.96)	0.47 (0.28, 0.78)	0.45 (0.26, 0.78)	0.34 (0.17, 0.65)
TN					
Pattern 1	(103/103)	11.32 (4.25, 30.19)	10.76 (4.03, 28.76)	12.60 (4.21, 37.68)	17.62 (4.41, 70.39)
Pattern 2	(103/103)	0.61 (0.42, 0.88)	0.60 (0.41, 0.89)	0.58 (0.39, 0.87)	0.55 (0.36, 0.83)

OR = odds ratio; CI = confidence interval; HER2+ = human epidermal growth factor receptor 2-positive; TN = triple negative.
[a]Adjusted for energy (kcals/day).
[b]Ajusted for energy (kcals/day), estrogenic index (yr).
[c]Adjusted for energy (kcals/day), estrogenic index (yr), education (yr).
[d]Adjusted for energy (kcals/day), estrogenic index (yr), education (yr), breast cancer family history.

among controls (1,938 kcal/day), it was similar to that reported in the ENSANUT from 2006 for the Northern region of Mexico (1,743 kcal/day) (65). This suggests that the participating women did not comprise a biased sample of the target population.

To the best of our knowledge, this is the first study of dietary patterns and BC by molecular subtype in Mexican women. Evidence on dietary patterns and BC comes mainly from European, American, and Asian populations (10), whose dietary habits are different from Latin American populations. Our results provide evidence for the prevention of BC; however, they need to be replicated and expanded to other Latin American populations where other foods are consumed in a diverse way.

Acknowledgment

The authors thank the technical assistance of B.Sc Gisela Collado and Ms. Sheyla M. Armas.

Disclosure statement

The authors have no conflict of interest to declare.

Funding

This study was supported by CONACyT. Fondo Sectorial de Investigación en Salud y Seguridad Social (2005-02-14373, 2009-01-11384, 2010-1-140962 and 2016-272632), SEP-CONACYT (2008-79912) and Fondo Institucional del CONACyT (PDCPN2013-01-215464).

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