

Am Diet Assoc. Author manuscript; available in PMC 2009 August 1.

Published in final edited form as:

J Am Diet Assoc. 2008 August; 108(8): 1323–1329. doi:10.1016/j.jada.2008.05.008.

Comparison of Baseline Dietary Intake of Hispanic and Matched Non-Hispanic White Breast Cancer Survivors Enrolled in the Women's Healthy Eating and Living Study

MARÍA A. HERNÁNDEZ-VALERO, DrPH, CYNTHIA A. THOMSON, PhD, RD, FADA, MIKE HERNÁNDEZ, MS, TAYLOR TRAN, RD, MICHELLE A. DETRY, PhD, RICHARD L. THERIAULT, DO, RICHARD A. HAJEK, PhD, JOHN P. PIERCE, PhD, SHIRLEY W. FLATT, MS, BETTE J. CAAN, DrPH, and LOVELL A. JONES, PhD

M. A. Hernández-Valero is an instructor, T. Tran is program coordinator and research dietitian, R. A. Hajek is a senior research scientist, and L. A. Jones is a professor, Department of Health Disparities Research, M. Hernández and M. A. Detry are statistical analysts, Department of Biostatistics and Applied Mathematics, R. L. Theriault is a professor, Department of Breast Medical Oncology, Center for Research on Minority Health, The University of Texas M. D. Anderson Cancer Center, Houston. C. A. Thomson is an associate professor, Department of Nutritional Sciences, University of Arizona, Tucson. J. P. Pierce is a professor and S. W. Flatt is a senior statistician, University of California-San Diego Cancer Center, La Jolla. B. J. Caan is a senior epidemiologist, Division of Research, Kaiser Foundation Research Institute, Oakland, CA

Abstract

Objective—To assess the reported baseline dietary intake of Hispanic and non-Hispanic white breast cancer survivors in the Women's Healthy Eating and Living study, a randomized plant-based dietary intervention clinical trial.

Design—Dietary data from 4 days repeated 24-hour recalls within 3 weeks included daily total intake of energy, protein, carbohydrates, cholesterol, total fat, monounsaturated fat, saturated fat, polyunsaturated fat, fruit/vegetable servings, carotenoids, alcohol, caffeine, and percentage of energy from protein, carbohydrates, alcohol, and fats.

Subjects—One hundred sixty-five Hispanic breast cancer survivors age-matched to 165 non-Hispanic white breast cancer survivors diagnosed with Stage I, II, or IIIA primary operable breast cancer.

Statistical analyses—Two-sample *t* tests and Wilcoxon rank sum tests to compare dietary intake, and logistic and ordinal logistic regression analyses to examine the association between ethnicity, alcohol, and lycopene consumption, while controlling for place of birth, education, body mass index, and time since diagnosis.

Results—Hispanics were more likely to be foreign-born (P<0.001), less educated (P<0.0001) and to consume higher amounts of lycopene (P=0.029), while non-Hispanic whites were more likely to consume alcohol (P=0.001). However, no differences were observed in the average amounts of alcohol consumed or total percents of energy from alcohol. Both groups consumed more than five servings of fruits and vegetables daily. Being Hispanic remained a significant predictor of lower alcohol use (P=0.004) and higher lycopene consumption (P=0.005) after controlling for place of birth, education, body mass index, and time since diagnosis.

Conclusions—There are more similarities than differences in the dietary intake of Hispanic and non-Hispanic white breast cancer survivors in the Women's Healthy Eating and Living study. Further analysis is needed to determine if higher lycopene consumption shown among the Hispanic participants will translate to greater protection against breast cancer recurrence or increased survival.

In the United States the risk of developing breast cancer differs significantly among women of different ethnicities or racial groups. The risk is highest for non-Hispanic white women, followed in decreasing frequency by African Americans, Asians, Pacific Islanders, Hispanics, American Indians, and Alaskan Natives (1,2). These differences may potentially be explained by culture and environmental factors, such as diet (3-13).

Dietary studies have suggested that diets high in fruits and vegetables and low in alcohol and total and saturated fats may reduce the risk of breast cancer (4-8,11) and breast cancer recurrence (14-16). Among women with a high incidence of familial breast cancer, an inverse association between carotenoid-rich foods and breast cancer has been reported (6), and alcohol consumption has been associated with an increased risk of breast cancer (16,17), particularly among women with folate insufficient diets. One study designed to evaluate variations in nutrient intake among breast cancer patients demonstrated statistically significant ethnic variation in dietary intake between African-American, Chinese, Hispanic, Japanese and non-Hispanic white breast cancer female patients (18).

To develop effective dietary intervention strategies for breast cancer survivors from different ethnicities or racial backgrounds, researchers must be familiar with the dietary patterns of the population they are targeting. Further, identifying significant differences in dietary patterns across ethnic groups may help to explain ethnic differences in breast cancer and breast cancer recurrence rates. To this end, before the study intervention, we evaluated the baseline reported dietary intake of the Hispanic breast cancer survivors and a subset of non-Hispanic white breast cancer survivors enrolled in a randomized controlled dietary intervention study.

METHODS

Study Population

This study represents a subgroup analysis of data collected from breast cancer survivors participating in the Women's Healthy Eating and Living (WHEL) study, a multicenter randomized controlled clinical trial of 3,088 women previously treated for breast cancer designed to determine if a diet high in fiber, fruit, and vegetables, and low in fat increases breast cancer survival (19). The data presented in this article include all Hispanic participants enrolled in the WHEL study at baseline (N=165). Hispanic ethnicity was self-reported and included participants born in the United States, Mexico, the Caribbean, Central America, Europe, South America, and Asia. Hispanic participants interested in participating in the WHEL study were required to be able to speak, write, and read the English language. For this analysis, the Hispanic participants were randomly matched (1:1 ratio) to a subset of WHEL study participants on age at the time of study enrollment using a range of ±5 years. Our rationale for not including all the non-Hispanic white participants was twofold: the WHEL study was not balanced by ethnicity, and the total number of non-Hispanic white participants far exceeded the total number of Hispanic participants.

The WHEL study was approved by the institutional review boards of the seven participating centers (University of California at San Diego and Davis; Kaiser Permanente Northern California, Oakland, CA; The University of Texas M. D. Anderson Cancer Center, Houston, TX; Stanford Prevention Research Center, Stanford, CA; University of Arizona, Tucson, AZ; and Center for Health Research, Portland, OR). Further description of the study design and methodology of the WHEL study has been published elsewhere (20-25).

Dietary Intake Data

Dietary intake data were collected using four 24-hour recalls collected within a 3-week period on randomly selected days that were stratified for weekend vs weekdays (24). Recalls were conducted by telephone by trained dietary assessors located at the University of California, San Diego, using the US Department of Agriculture Multi-pass recall method (26). Quality control for the 24-hour recalls was conducted (24,25). Participants were educated on how to complete recalls during the baseline clinic visit and were provided written guidelines and pictures of portion estimates to assist during the recall process (20,25). All telephone interviews were conducted in English. Dietary assessors were not involved in the administration of dietary interventions and this analysis included only baseline dietary recalls which were conducted before randomization. Dietary data collected during the recall were entered into the Nutrition Data System (version 4.0, 2000, University of Minnesota Nutrition Coordinating Center, Minneapolis, MN) computer-based software for nutrient analysis. Nutrition Data System data were used to assess intake of the following: daily total energy (kilocalories/day); fiber intake (grams/day); cholesterol (grams/day); protein (grams/day); total fat (grams/day); monounsaturated fat (grams/day); saturated fat (grams/day); polyun-saturated fat (grams/day); alcohol (grams/day); caffeine (grams/day); percent energy from all types of fats, protein, carbohydrates, and alcohol; and reported carotenoid intake. The average daily number of servings of fruits and vegetables (1 serving=1/2 c), which also included mixed dishes such as tacos, burritos, soups, sandwiches, and stews, were calculated using software developed by the University of California at San Diego Cancer Prevention and Control Program, La Jolla, CA (23). Estimates of dietary carotenoid intake were obtained from the Nutrition Data System nutrient analysis, which includes food analytical estimates from the US Department of Agriculture carotenoid database (27).

Sociodemographic data for the WHEL study were collected by telephone during the screening interview (20), and included date of birth, age, self-reported ethnicity (eg, African American, Asian, Hispanic, or white), date of breast cancer diagnosis and stage at diagnosis confirmed through medical records review, education level, marital status, and occupation. Place of birth was also self-reported and collected via a written questionnaire, and height and weight were measured during the first clinic visit by standard procedures to calculate body mass index (BMI).

Statistical Analyses

All analyses were performed with the Statistical Analysis System (version 8.2, 2001, SAS Institute, Cary, NC). Two-sample t tests and χ^2 tests were used to compare the sociodemographic and clinical characteristics of the study population by ethnicity. Wilcoxon rank sum test was used to compare the dietary intake of study participants because the nutrition variables tended to be non-normally distributed. Univariate logistic regression models were fit to examine the independent association between ethnicity, place of birth, education, BMI, time since diagnosis, and the nutrition variables. In addition, multivariate logistic models were conducted to examine the association between ethnicity and those nutrition variables that were found to be statistically significantly different between the ethnic groups, while controlling for place of birth, education, BMI, and time since diagnosis. Finally, ordinal logistic regression models were used to examine the association between ethnicity and the nutrition variables that had a large number of participants who reported "no intake." Each nutrient was categorized into quartiles, and tested for the proportional odds assumption.

RESULTS

A comparison of the sociodemographic and clinical characteristics of study participants by ethnic group shows the groups had similar marital status, BMI, menopausal status, and breast

cancer stage at diagnosis (Table 1). Due to matching, the mean age at enrollment for both Hispanic and non-Hispanic white participants was identical (50.6 years). The mean time since breast cancer diagnosis and enrollment into the WHEL study was 2 years for both ethnic groups. One third of Hispanic participants were born outside the United States vs 10% of non-Hispanic white participants (P<0.001). A higher percentage of the non-Hispanic white women were college or university graduates (53%) compared to only one third of the Hispanics (P<0.0001).

Table 2 compares, by ethnic group, the baseline reported daily mean dietary intake of selected nutrients among study participants. Overall, the diets of the two groups were similar, with the exception of the proportion of women who reported any alcohol use during the 4 days of intake recalled and the average daily consumption of lycopene. A higher percentage of non-Hispanic white participants reported using alcohol than Hispanic participants (75.8% vs 59.4%; P=0.001); however, among those reporting any alcohol intake, there was no statistically significant difference in the amount of alcohol consumed or in the percent energy from alcohol consumed (Table 2). Hispanic participants had higher mean intakes of lycopene than non-Hispanic white participants (P=0.029). In regard to fruit and vegetable consumption, both ethnic groups reported consuming, on average, more than five servings of fruits and vegetables daily (5.4 and 5.9 servings for Hispanics and non-Hispanic whites, respectively).

Since the percentage of non-Hispanic whites who reported any alcohol consumption during the 4 days recalled was significantly higher than the percentage of Hispanic women reporting any alcohol consumption, we fit logistic regression models (Table 3) to determine whether ethnicity was associated with any alcohol use. Both the univariate and multivariate analyses showed a significant association between ethnicity and alcohol use. In the univariate analysis, Hispanic participants were 53% less likely to use alcohol compared to non-Hispanic white participants (odds ratio [OR] 0.47, 95% confidence interval [CI] [0.29, 0.75]), even after controlling for place of birth, education, BMI, and time since diagnosis (OR=0.44, 95% CI [0.26, 0.74]). The educational status of study participants was also associated with alcohol use. Participants with a posthigh school/some college education were almost three times more likely to use alcohol than participants with only a high school education, in both the univariate (OR 2.98, 95% CI [1.60, 5.55]) and multivariate analyses (OR 2.67, 95% CI [1.40, 5.05]). To a lesser extent, the same association was observed among participants who were college/university graduates.

Because lycopene consumption was found to be significantly different between the two ethnicities (Table 2), an ordinal logistic regression model (Table 4) was used to measure the association between ethnicity and lycopene consumption. Because the lycopene data were highly skewed, quartiles of mean daily intake were used to convert it into proportionally equal categories: $<1,203\,\mu g, 1,203\,\mu g$ to $2,564.4\,\mu g, 2,564.5\,\mu g$ to $4393.7\,\mu g,$ and $>4393.7\,\mu g.$ Results of the univariate ordinal logistic regression analysis showed that ethnicity was significantly associated with lycopene consumption, with Hispanic women being 52% more likely to consume foods with higher amounts of lycopene than their non-Hispanic white counterparts (OR 1.52, 95% CI [1.03, 2.42]). Even after controlling for place of birth, education, BMI, and time since diagnosis (Table 3), Hispanic participants remained more likely (56%) to consume significantly greater amounts of lycopene daily than non-Hispanic white participants (Table 4). In addition to ethnicity, the BMI status of study participants was also associated with lycopene consumption. Overweight participants were 70% more likely to consume lycopene than participants of normal weight, in both the univariate (OR 1.70, 95% CI [1.06, 2.72]) and multivariate analyses (OR 1.70, 95% CI [1.05, 2.73]).

DISCUSSION

We compared baseline reported dietary intakes of Hispanic and non-Hispanic white women enrolled in a plant-based dietary intervention trial, on average, 2 years after their breast cancer diagnosis. Overall, the two ethnic groups were sociodemographically and clinically similar, with the exception of their place of birth and educational status, with more Hispanic participants being born outside of the United States, and non-Hispanic white participants having a higher education level. The latter finding supports the findings of the US Census Bureau, which shows the education level of Hispanics in the United States are lower than those of other ethnic groups (28). Nevertheless, the educational status of the Hispanic participants in this study was found to be higher than the educational level of Hispanics reported in other cancer studies (29-31). This may reflect the overall greater socioeconomic status of WHEL participants, who tend to be middle-class or upper-class as defined by their educational status. Further, as we excluded women who reported Spanish-only literacy and language, we may have selected for a more acculturated subgroup of Hispanic breast cancer survivors resulting in greater similarity in sociodemographic variables than would be expected.

The dietary intakes of both ethnic groups were similar, with the exception of the percentage who consumed alcohol during the 4 days recalled. The mean alcohol intakes for both groups were below the national average (32). The study findings suggesting very low alcohol consumption are consistent with a report from Hernández-Valero and colleagues (33) of 3,384 Hispanic women of Mexican origin, which found that <20% of the population reported ever consuming alcoholic beverages. The alcohol–breast cancer association also previously has been investigated by Baumgartner and colleagues (34) in a biethnic study between non-Hispanic white and Hispanic breast cancer survivors. In that study alcohol intake did not appear to have a consistent or significant association with breast cancer risk in Hispanic women (33). Whether the lower quantity of alcohol intake will be associated with a significant reduction in breast cancer recurrence risk for the Hispanic participants in this study remains to be evaluated.

In our analysis, both ethnic groups reported similar mean daily consumption of fruits and vegetables, which was also supported by similar carotenoid intake, with the exception of lycopene for which Hispanics reported greater intake than non-Hispanic whites. Because the study population had been diagnosed with breast cancer 2 years before their enrollment into the WHEL study, there is the possibility that fruit and vegetable intake increased in response to the breast cancer diagnosis, as has been previously reported for the total WHEL study population (21). Alternatively, these levels of fruit and vegetable intake may be more representative of breast cancer survivors volunteering for a dietary intervention trial, and not representative of breast cancer survivors as a whole, as previously shown in nonintervention dietary studies (35,36). Further, the significant difference in lycopene intake may reflect the daily use of tomato products (eg, sauce and paste) in many Hispanic dishes such as salsas (tomato-based sauces), and *sofritos* (a combination of sautéed onions, garlic, bell peppers, and tomato sauce and oil), which are an integral part of the daily cuisine among certain Hispanic groups (37-39).

Only a few studies have examined the relationship between breast cancer and tomato products (6,40,41) or serum or plasma lycopene levels (42-46). Dietary-based studies (6,44,45) generally have not found an association between breast cancer risk and tomato intake. However, other studies that have investigated the risk of breast cancer in relation to plasma or serum lycopene levels (42-49) have observed a significant gradient of decreasing risk with increasing lycopene concentration. Further, mechanistic evidence also exists to support a protective role for lycopene in breast cancer. For example, Levy and colleagues (47) found lycopene to have antiproliferative effects against breast cancer cells in culture and Sharoni and

colleagues (48) have shown that rats treated with tomato oleoresin developed fewer 7,12-dimethylbenz(a)anthracene-induced mammary tumors. In addition to lycopene, tomatoes have other potential health-enhancing compounds like ascorbic acid (vitamin C), and hypothetically the complex interactions of these compounds may also contribute to the anticancer properties of tomato products (49). A study conducted by Porrini and colleagues (50) among healthy individuals found that the daily intake of a formulated tomato drink significantly reduced (by about 42%) DNA damage in lymphocytes, suggesting that oxidative stress may be favorably modulated with tomato foods thus indirectly reducing cancer risk.

The relationship between lycopene, tomatoes, and breast cancer risk remains unclear and more evidence is needed. Furthermore, recent findings suggest that currently there is little evidence from epidemiologic studies to support a protective relationship between fruits and vegetable intake and breast cancer, although ethnic-specific associations have not been fully explored (6-9,11,49,50). One reason for the inconsistent findings maybe dose; another maybe the nutrient and/or phytochemical density of the specific plant foods consumed by the study populations. Thus, it is not inconceivable that the higher tomato intake among Hispanics may play a role in breast cancer risk reduction, including reduction in risk for breast cancer recurrence, and contribute to lower rates of breast cancer in this population. However, much more research is needed before any clinical recommendations can be made (14,15).

Caution must be taken in generalizing the findings of this study to all Hispanic and non-Hispanic white women in the United States because of possible bias due to differences in the sociodemographic and educational status between the ethnic groups, and the small number of Hispanic women enrolled in the WHEL study, which did not allow for stratification by place of birth.

CONCLUSIONS

Our research shows that overall there are more similarities than differences in the reported dietary intake of Hispanic and non-Hispanic white breast cancer survivors after diagnosis. However, a few interesting differences (ie, alcohol use and lycopene consumption) were observed that may influence the main study measured outcomes. Further analysis is needed to determine if higher lycopene consumption provides protection against breast cancer recurrence or increased survival in this group of women. In addition, similar studies need to be conducted with greater numbers of breast cancer survivors from this ethnic group, including non-English speaking Hispanics and/or those reporting lower acculturation levels.

Acknowledgements

Funding for this research came in part from the National Institutes of Health grants nos. NCI CA-69375, NCI CA-69375A Minority Supplement, NCMHHD P60 MD000503, M01-RR00827, M01-RR00079, and M01-RR00070.

The authors thank Stephanie Deming, Department of Scientific Publications, and Patricia C. Pillow, MS, RD, Department of Epidemiology, The University of Texas M. D. Anderson Cancer Center, for their valuable editorial comments.

References

- 1. Doll R, Peto R. The causes of cancer: Quantitative estimates of avoidable risks of cancer in the United States today. J Natl Cancer Inst 1981;66:1191–1308. [PubMed: 7017215]
- 2. Muir, C.; Waterhouse, J.; Mack, T.; Powell, J.; Whelan, S.; Smans, M., editors. Cancer Incidence in Five Continents. Agency for Research on Cancer; Lyon, France: 1987.
- 3. Zaridze DG, Muir CS, McMichael AJ. Diet and cancer: Value of different types of epidemiological studies. Nutr Cancer 1985;7:155–166. [PubMed: 3001657]

4. Goldin BR, Gorbach SL. Hormone studies and the diet and breast cancer connection. Adv Exp Med Biol 1994;364:35–46. [PubMed: 7725958]

- Freudenheim JL, Marshall JR, Graham S, Laughlin R, Vena JE, Swanson M, Ambrosone C, Nemoto T. Lifetime alcohol consumption and risk of breast cancer. Nutr Cancer 1995;23:1–11. [PubMed: 7739910]
- Freudenheim JL, Marshall JR, Vena JE, Laughlin R, Brasure JR, Swanson MK, Nemoto T, Graham S. Premenopausal breast cancer risk and intake of vegetables, fruits, and related nutrients. J Natl Cancer Inst 1996;88:340–348. [PubMed: 8609642]
- Steinmetz KA, Potter JD. Vegetables, fruit, and cancer prevention: A review. J Am Diet Assoc 1996;96:1027–1039. [PubMed: 8841165]
- 8. Greenwald P, Sherwood K, McDonald SS. Fat caloric intake and obesity: Lifestyle risk factors for breast cancer. J Am Diet Assoc 1997;97(suppl 7):S24–S30. [PubMed: 9216564]
- Jones LA, González R, Pillow PC, Gómez-Garza SA, Foreman CJ, Chilton JA, Linares A, Yick J, Badrei M, Hajek RA. Dietary fiber, Hispanics, and breast cancer risk? Ann NY Acad Sci 1997;837:524–536. [PubMed: 9472361]
- 10. Sasco AJ. Epidemiology of breast cancer: An environmental disease? APMIS 2001;109:321–332. [PubMed: 11478680]
- 11. Lee MM, Lin SS. Dietary fat and breast cancer. Annu Rev Nutr 2002;20:221–248. [PubMed: 10940333]
- 12. Ziegler RG, Hoover RN, Pike MC, Hildesheim A, Nomura AM, West DW, Wu-Williams AH, Kolonel LN, Horn-Ross PL, Rosenthal JF. Migration patterns and breast cancer risk in Asian-American women. J Natl Cancer Inst 1993;85:1819–1827. [PubMed: 8230262]
- 13. Kouris-Blazos A, Wahlqvist ML, Trichoupolou A, Polychronopoulos E, Trichopolous D. Health and nutritional status of elderly Greek migrants to Melbourne, Australia. Age Ageing 1996;25:177–189. [PubMed: 8670549]
- 14. Rohan TE, Hiller JE, McMichael AJ. Dietary factors and survival from breast cancer. Nutr Cancer 1993;20:167–177. [PubMed: 8233982]
- 15. Ingram D. Diet and subsequent survival in women with breast cancer. Br J Cancer 1994;69:592–595. [PubMed: 8123493]
- Jain MG, Ferrence RG, Rehm JT, Bondy SJ, Rohan TE, Ashley MJ, Cohen JE, Miller AB. Alcohol and breast cancer mortality in a cohort study. Breast Cancer Res Treat 1994;64:201–209. [PubMed: 11194456]
- 17. Egan K, Stampfer MJ, Rosner BA, Trichopoulos D, Newcomb PA, Trent-Dietz A, Longnecker MP, Mittendorf R, Greenberg ER, Willett WC. Risk factors for breast cancer in women with a breast cancer family history. Cancer Epidemiol Biomarkers Prev 1998;7:359–364. [PubMed: 9610783]
- 18. Huang MH, Schocken M, Block G, Sowers M, Gold E, Sternfeld B, Seeman T, Greendale GA. Variation in nutrient intakes by ethnicity: results from the Study of Women's Health Across the Nation (SWAN). Menopause 2002;9:309–319. [PubMed: 12218719]
- 19. Pierce JP, Faerber S, Wright FA, Rock CL, Newman V, Flatt SW, Kealey S, Jones VE, Caan BJ, Gold EB, Haan M, Hollenbach KA, Jones L, Marshall JR, Ritenbaugh C, Stefanick ML, Thomson C, Wasserman L, Natarajan L, Gilpin EA, Thomas RG, the Women's Healthy Eating and Living (WHEL) Study Group. A randomized trial of the effect of a plant-based dietary pattern on additional breast cancer events and survival: Women's Healthy Eating and Living (WHEL) Study. Control Clin Trials 2002;23:728–756. [PubMed: 12505249]
- 20. Pierce JP, Faerber S, Wright FA, Newman V, Flatt SW, Kealey S, Rock CL, Hryniuk W, Greenberg ER. Feasibility of a randomized trial of high-vegetable diet to prevent breast cancer recurrence. Nutr Cancer 1997;28:282–288. [PubMed: 9343838]
- Thomson CA, Flatt SW, Rock CL, Ritenbaugh C, Newman V, Pierce JP. Increased fruit, vegetable, and fiber intake and lower fat intake reported among women previously treated for invasive breast cancer. J Am Diet Assoc 2002;102:801–808. [PubMed: 12067045]
- 22. Thomson CA, Rock CL, Giulano AR, Cui H, Reid PM, Green TL, Alberts DS. Longitudinal changes in body weight and body composition among women previously treated for breast cancer consuming a high-vegetable, fruit and fiber, low-fat diet. Eur J Nutr 2005;44:18–25. [PubMed: 15309460]

23. Newman V, Faerber S, Zoumas C, Rock CL. Amount of raw vegetables and fruits needed to yield 1 c juice. J Am Diet Assoc 2002;102:975–977. [PubMed: 12146562]

- 24. Pierce JP, Newman V, Flatt SW, Natarajan L, Flatt SW, Al-Delaimy WK, Caan BJ, Emond JA, Faerber S, Gold EB, Hajek RA, Hollenbach KA, Jones LA, Karanja N, Kealey S, Madlensky L, Marshall J, Ritenbaugh C, Rock CL, Stefanick ML, Thomson CA, Wasserman L, Parker BA. Telephone counseling helps maintain long-term adherence to a high-vegetable dietary pattern. J Nutr 2007;137:2291–2296. [PubMed: 17885013]
- 25. Thomson CA, Giuliano A, Rock CL, Ritenbaugh CK, Flatt SW, Faerber S, Newman V, Caan B, Graver E, Hartz V, Whitacre FP, Parker F, Pierce JP, Marshall JR. Measuring dietary changes in a diet intervention trial: Comparing food frequency questionnaire and dietary recalls. Am J Epidemiol 2003;157:754–762. [PubMed: 12697580]
- 26. Conway JM, Ingwersen LA, Vinyard BT, Moshfegh AJ. Effectiveness of the US department of Agriculture 5-step multiple-pass method in assessing food intake in obese and non-obese women. Am J Clin Nutr 2003;77:1171–1178. [PubMed: 12716668]
- 27. USDA-NCC Carotenoid database for US food—1998. US Department of Agriculture, Nutrient Data Laboratory Web site. http://www.nal.usda.gov/fnic/foodcomp/Data/car98/car98.html.May 13, 2008
- 28. Educational Attainment in the United States: March 2002. Detailed Tables (PPL-169). US Census Bureau Web site. http://www.census.gov/population/www/socdemo/education/ppl-169.html.May 13, 2006
- 29. O'Malley AS, Kerner J, Johnson AE, Mandelblatt J. Acculturation and breast cancer screening among Hispanic women in New York City. Am J Public Health 1999;89:219–227. [PubMed: 9949753]
- 30. Bowen D, Raczynski J, George V, Feng Z, Fouad M. The role of participation in the women's health trial: Feasibility study in minority populations. Prev Med 2000;31:474–480. [PubMed: 11071827]
- 31. Valdez A, Banerjee K, Ackerson L, Fernández M. A multimedia breast cancer education intervention for low-income Latinas. J Community Health 2002;27:33–51. [PubMed: 11845940]
- 32. Breslow RA, Guenther PM, Smothers BA. Alcohol drinking patterns and diet quality: The 1999-2000 National Health and Nutrition Examination Survey. Am J Epidemiol 2006;163:359–366. [PubMed: 16394204]
- 33. Hernández-Valero, MA.; Bárcenas, C.; Cao, Y.; Wilkinson, A.; Strom, SS.; Spitz, MR.; Bondy, ML. Proceedings of the National Institute for Farm Safety 2004 Summer Conference. National Institute for Farm Safety; Madison, WI: 2004. Long-term health in migrant and non-migrant farmworkers: Results from a population-based Mexican-American cohort study..
- 34. Baumgartner KB, Annegers JF, McPherson RS, Frankowski RF, Gilliland FD, Samet JM. Is alcohol intake associated with breast cancer in Hispanic women? The New Mexico Women's Health Study. Ethn Dis 2002;12:460–469. [PubMed: 12477131]
- Caan B, Sternfeld B, Gunderson E, Coates A, Quesenberry C, Slattery ML. Life After Cancer Epidemiology (LACE) Study: A cohort of early breast cancer survivors (United States). Cancer Causes Control 2005;16:545–556. [PubMed: 15986109]
- 36. Wayne SJ, Lopez ST, Butler LM, Baumgartner KB, Baumgartner RN, Ballard-Barbash R. Changes in dietary intake after diagnosis of breast cancer. J Am Diet Assoc 2004;104:1561–1568. [PubMed: 15389414]
- 37. Alvarez, J. All about Cuban cooking. [May 13, 2006]. http://www.cuban-cooking-cookbook.com.
- 38. Rivera, M. Cocina criolla. [May 13, 2006]. http://welcome.topuertorico.org/cocina/.
- 39. Mexico. Sally's Place Web site. http://www.sallys-place.com/food/cuisines/mexico.htm.May 13, 2006
- 40. Ewertz M, Gill C. Dietary factors and breast-cancer risk in Denmark. Int J Cancer 1990;46:779–784. [PubMed: 2228305]
- 41. Levi F, Vecchia C, Gulie C, Negri E. Dietary factors and breast cancer risk in Vaud, Switzerland. Nutr Cancer 1993;19:327–335. [PubMed: 8346081]
- 42. Potischman N, McCulloch CE, Byers T, Nemoto T, Stubbe N, Milch R, Parker R, Rasmussen KM, Root M, Graham S. Breast cancer and dietary and plasma concentrations of carotenoids and vitamin A. Am J Clin Nutr 1990;52:909–915. [PubMed: 2239767]

43. London SL, Stein EA, Henderson IC, Stampfer MJ, Wood WC, Remine S, Dmochowaki JR, Robert NJ, Willett WC. Carotenoids, retinol, and vitamin E and risk of proliferative benign breast disease and breast cancer. Cancer Causes Control 1992;3:503–512. [PubMed: 1420852]

- 44. Jarvinen R, Knekt P, Seppanen R, Teppo L. Diet and breast cancer risk in a cohort of Finnish women. Cancer Letter 1997;114:251–253.
- 45. Zhang S, Tang G, Russell RM, Mayzel KA, Stampfer MJ, Willett WC, Hunter DJ. Measurement of retinoids and carotenoids in breast adi-pose tissue and a comparison of concentrations in breast cancer cases and control subjects. Am J Clin Nutr 1997;66:626–632. [PubMed: 9280184]
- 46. Dorgan JF, Sowell A, Swanson CA, Potischman N, Miller R, Schussler N, Stephenson HE Jr. Relationships of serum carotenoid, retinol, α-tocopherol, and selenium with breast cancer risk: results from a prospective study in Columbia, Missouri (United States). Cancer Causes Control 1998;9:89–97. [PubMed: 9486468]
- 47. Levy J, Bosin E, Feldman B, Giat Y, Miinster A, Danilenko M, Sharoni Y. Lycopene is a more potent inhibitor of human cancer cell proliferation than either α-carotene or β-carotene. Nutr Cancer 1995;24:257–266. [PubMed: 8610045]
- 48. Sharoni Y, Giron E, Rise M, Levy J. Effects of lycopene-enriched tomato oleoresin on 7,12-dimethylbenzo[a]anthracene-induced rat mammary tumors. Cancer Detect Prev 1997;21:118–123. [PubMed: 9101071]
- 49. Giovannucci E. Tomatoes, tomato-based products, lycopene, and cancer: Review of the epidemiologic literature. J Natl Cancer Inst 1999;9:317–331. [PubMed: 10050865]
- 50. Porrini M, Riso P, Brusamolino A, Berti C, Guarnirei S, Visioli F. Daily intake of a formulated tomato drink affects carotenoid plasma and lymphocyte concentrations and improves cellular antioxidant protection. Br J Nutr 2005;93:93–99. [PubMed: 15705230]

>Table 1

Sociodemographic and clinical characteristics of Hispanic and matched non-Hispanic white breast cancer survivors enrolled in the Women's Healthy Eating and Living Study (N=330)^a

| Characteristic | Hispanic (n=165) | Non-Hispanic white (n= 165) | P value |
|--|--|-----------------------------|----------|
| - | ← mean±standard deviation → | | |
| Age at enrollment (v) | 50.6±9.3 | 50.6± 9.3 | 1.000 |
| Age at diagnosis (y) | 48.8±9.2 | 48.8 ± 9.3 | 0.999 |
| Body mass index | 28.1 ± 6.3 | 27.9 ± 7.0 | 0.843 |
| | ← | (%)──── | |
| Place of birth | | | |
| United States | 111 (67.3) | 148 (89.7) | |
| Outside the United States ^b | 54 (32.7) | 17(10.3) | < 0.001 |
| Education | | | |
| High School | 52 (31.5) | 25 (15.2) | |
| Posthigh school/some college | 62 (37.6) | 52 (31.5) | |
| Postcollege/university graduate | 51 (31.0) | 88 (53.3) | < 0.0001 |
| Marital status | (2, 7, 7, 7, 7, 7, 7, 7, 7, 7, 7, 7, 7, 7, | (, , , , | |
| Single | 25 (15.2) | 24 (14.6) | |
| Married | 109(66.5) | 117(71.3) | |
| Separated/divorced | 22 (13.4) | 15 (9.2) | |
| Widowed | 8 (4.9) | 8 (4.9) | 0.653 |
| Categorical body mass index C | ` ' | , , | |
| Normal | 59 (35.8) | 69 (41.8) | |
| Overweight | 53 (32.1) | 52 (31.5) | |
| Obese | 53 (32.1) | 44 (26.7) | 0.444 |
| Menopausal status | | (, | |
| Premenopausal | 25 (15.2) | 27 (16.4) | |
| Perimenopausal | 16(9.8) | 15 (9.1) | |
| Postmenopausal | 123 (75.0) | 123 (74.5) | 0.948 |
| Cancer stage at diagnosis | ` ' | ` ' | |
| Stage I | 51 (30.9) | 58 (35.2) | |
| Stage II | 101 (61.2) | 100 (60.6) | |
| Stage IIIA | 13(7.9) | 7 (4.2) | 0.324 |

 $^{^{}a}\mathrm{Numbers}$ may vary due to missing values.

b Self-reported place of birth of foreign-born participants: for Hispanics: Caribbean (n=7), Central America (n=2), Mexico (n=29), Asia (n = 1), South America (n = 13), and Europe (n=2); for non-Hispanic whites: Africa (n=1), Asia (n=2), Canada (n=5), and Europe (n=9).

^cCategories of body mass index: normal (18–24.9), overweight (25–29.9), and obese (>30).

Table 2Baseline reported daily nutrient intake of Hispanic and matched non-Hispanic white breast cancer survivors enrolled in the Women's Healthy Eating and Living Study (N=330)

| Nutrient intake | Hispanic (n=165) | Non-Hispanic white (n= 165) | P value ^a |
|--|-----------------------------|-----------------------------|----------------------|
| | ← mean±standard deviation → | | |
| Total energy (kcal) | 1,690.8±414.4 | $1,749.8 \pm 462.5$ | 0.351 |
| Protein (g) | 67.9±17.7 | 70.4 ± 18.4 | 0.305 |
| Carbohydrates (g) | 227.3±60.5 | 237.65±73.1 | 0.564 |
| Energy from carbohydrates (%) | 54.2±7.9 | 54.8±8.0 | 0.549 |
| Cholesterol (mg) | 223.7±98.3 | 207.5 ± 106.9 | 0.070 |
| Total fat (g) | 58.5±22.5 | 58.4 ± 22.0 | 0.811 |
| Energy from total fat (%) | 30.3 ± 6.9 | 29.3 ± 6.5 | 0.155 |
| Monounsaturated fatty acid (g) | 22.1 ±8.7 | 22.1 ± 9.2 | 0.954 |
| Energy from monounsaturated fatty acid (%) | 11.6±3.0 | 11.2±3.0 | 0.211 |
| Saturated fatty acid (g) | 19.1 ±8.6 | 19.2 ± 8.1 | 0.702 |
| Energy from saturated fatty acid (%) | 10.0±2.9 | 9.7±2.8 | 0.519 |
| Polyunsaturated fatty acid (g) | 12.5±5.7 | 12.4 ± 5.2 | 0.898 |
| Energy from polyunsaturated fatty acid (%) | 6.6±2.1 | 6.3±1.9 | 0.378 |
| Dietary fiber (g) | 19.7 ± 7.1 | 20.5±8.1 | 0.408 |
| Caffeine consumed b (g) | 139.6± 122.8 | 164.3 ± 180.7 | 0.561 |
| Fruit servings+fruit juices ^c | 2.7±1.8 | 3.1±2.1 | 0.116 |
| Vegetable servings+vegetable juices ^c | 2.7±1.7 | 2.8±1.7 | 0.398 |
| α -Carotene (μ g) | $953.9 \pm 2.559.3$ | $1,016.0 \pm 1,474.6$ | 0.114 |
| β -Carotene (μ g) | $4,800.9 \pm 6,883.4$ | $5,109.4 \pm 4,985.9$ | 0.283 |
| β-Cryptoxanthin (μg) | 91.4 ± 174.8 | 80.6±120.2 | 0.601 |
| Lutein+zeaxanthin (µg) | $2,377.6 \pm 1,912.9$ | $2,820.3\pm2,981.1$ | 0.191 |
| Lycopene (µg) | $3.682.0 \pm 3.161.7$ | 3.004.2± 2.719.9 | 0.029 |
| | | n (%) — → | |
| Alcohol intake during reported days Yes | 98 (59.4) | 125 (75.8) | 0.001^{d} |
| No. | 67 (40.6) | 40 (24.2) | 0.001 |
| | 4.8±7.2 | 6.7±10.4 | 0.153 |
| Alcohol consumed ^e (g) | | | |
| Energy from alcohol ^e (%) | 2.0±3.1 | 2.5±3.8 | 0.335 |
| Caffeine intake during reported days | 1.52 (00.0) | 1.50 (07.0) | J |
| Yes | 163 (98.8) | 160 (97.0) | 0.252^{d} |
| No | 2(1.2) | 5 (3.0) | |

NOTE: Information from this table is available online at www.adajournal.org as part of a PowerPoint presentation.

 $^{{}^{}a}\mathrm{Based}$ on Wilcoxon rank sum test.

 $[^]b\mathrm{Among}$ participants who reported coffee intake.

 $c_{1/2}$ c=1 serving.

 $d_{\text{Based on } x^2 \text{ test.}}$

 $^{^{\}it e}{\rm Among}$ the participants who reported alcohol intake.

Table 3

Univariate and multivariate logistic regression models for alcohol intake during the reported days among Hispanic and matched non-Hispanic white breast cancer survivors enrolled in the Women's Healthy Eating and Living Study (N=330)

| Characteristic | Univariate analysis | Multivariate analysis | |
|------------------------------|--|-----------------------|--|
| | ← Odds ratio (95% confidence interval) → | | |
| Ethnicity | | , | |
| Non-Hispanic white | 1.00 | 1.00 | |
| Hispanic | 0.47 (0.29, 0.75) | 0.44 (0.26, 0.74) | |
| Place of birth | (2.0, 0.0) | (3, 7) | |
| United States | 1.00 | 1.00 | |
| Outside the United States | 1.09 (0.62,1.92) | 1.42 (0.77, 2.61) | |
| Education | (2.2. , 2. , | (3.1.1) | |
| High school | 1.00 | 1.00 | |
| Posthigh school/some college | 2.98 (1.60, 5.55) | 2.66 (1.40, 5.05) | |
| College/university graduate | 2.06 (1.16, 3.66) | 1.67 (0.91, 3.07) | |
| Body mass index status | ` ' ' | , , , | |
| Normal | 1.00 | 1.00 | |
| Overweight | 1.70 (0.96, 3.02) | 1.69 (0.93, 3.08) | |
| Obese | 0.99 (0.57, 1.72) | 1.18 (0.66, 2.10) | |
| Time since cancer diagnosis | , , , , , , , , , , , , , , , , , , , | , , , , , , | |
| <2 y | 1.00 | 1.00 | |
| ≥2 y | 1.03 (0.64, 1.67) | 0.99 (0.60, 1.63) | |

Table 4

Ordinal logistic regression models for lycopene consumption among the Hispanic and matched non-Hispanic white breast cancer survivors enrolled in the Women's Health Eating and Living Study (N=330)

| Characteristic | Univariate analysis | Multivariate analysis |
|------------------------------|---|-----------------------|
| | ← Odds ratio a (95% confidence interval) → | |
| Ethnicity | () | , |
| Non-Hispanic white | 1.00 | 1.00 |
| Hispanic | 1.52 (1.03, 2.42) | 1.56 (1.04, 2.36) |
| Place of birth | . , , | . , , |
| United States | 1.00 | 1.00 |
| Outside the United States | 1.21 (0.75, 1.95) | 1.04 (0.64, 1.72) |
| Education | , , , | ` ' ' |
| High school | 1.00 | 1.00 |
| Posthigh school/some college | 0.95 (0.57, 1.60) | 0.96 (0.57, 1.62) |
| College/university graduate | 1.06 (0.65, 1.74) | 1.16 (0.69, 1.95) |
| Body mass index status | , , , | , , , |
| Normal | 1.00 | 1.00 |
| Overweight | 1.70 (1.06, 2.72) | 1.70 (1.05, 2.73) |
| Obese | 0.97 (0.61, 1.55) | 0.94 (0.59, 1.53) |
| Time since cancer diagnosis | , , , , , , | , , , , , , , |
| <2 y | 1.00 | 1.00 |
| ≥2 y | 1.06 (0.71, 1.59) | 1.02 (0.68, 1.54) |

^aThe odds ratio reflects the odds of being in a category of higher lycopene consumption than in a category of less lycopene consumption. The multivariate analysis displays results of the odds ratio for ethnicity adjusted for place of birth, education, body mass index, and time since cancer diagnosis.