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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

Summary *Background* Excess adiposity has been shown to be associated with increased risk for breast cancer recurrence, and a plant-based eating pattern has been hypothesized to be protective. Whether a plant-based diet without specific energy goals will result in weight loss or changes in body composition in women who have been diagnosed with breast cancer has not been fully explored. *Aim of the study* This study was conducted to identify changes in body weight, anthropometric measures, and body composition over a four year period in a sub-sample of breast cancer survivors participating in a dietary intervention targeting increased intake of vegetables, fruit and fiber and decreased dietary fat intake. *Methods* This randomized, controlled dietary intervention study compared longitudinal changes in intakes, body weight, waist:hip ratio (WHR), body mass index (BMI) and body composition by treatment group among fifty-two women previously treated for Stage I, II, or IIIA breast cancer from the Arizona site of the Women's Healthy Eating and Living Study. The dietary intervention aimed for eight servings of fruit and vegetables, 30 g fiber, ≤20 % total energy from fat per day, as well as daily intake of vegetable juice. The comparison group was advised to follow general dietary guidelines for cancer prevention. *Results* The dietary intervention resulted in a significant and sustained increase in fiber, fruit, vegetable, and vegetable juice consumption ($p < 0.05$) among intervention group subjects as compared to comparison group subjects. The first 6 months resulted in a reduction in body weight and body fat among the intervention group subjects while the comparison group subjects remained stable. Subsequent measurements, at 12, 24 or 36, and 48 months, showed no significant differences in mean body weight, BMI, WHR, or body composition by study group. Also, no significant changes in these measures were demonstrated for either study group between baseline and 48 months. *Conclusions* The dietary intervention efforts resulted in significant changes in diet toward an increase in plant foods and a decrease in dietary fat. Changes in weight, WHR, BMI, and body composition were not different over time or by study group assignment. Interventions that promote a plant-based diet without specific energy restriction do not appear to promote changes in body weight or body composition in women who have been diagnosed with breast cancer. To adequately examine the role of energy restriction in reducing obesity-associated breast cancer recurrence, future interventions should include prescribed energy imbalance either through reduced intake and/or increased expenditure.

Key words breast cancer – body weight – body composition – diet

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Introduction

In recent years, an expanding body of research has focused on the role of diet and nutritional factors, such as body weight, as modifiable risk factors for the development and progression of breast cancer. A positive association between postmenopausal breast cancer and obesity, especially abdominal obesity, has been widely reported in the scientific literature [1–9], particularly among women who have never used hormone replacement therapy [10], African-American women [11] and those of advanced age [12]. However, this association has not been generally observed in women diagnosed with premenopausal breast cancer, in which higher body mass index (BMI) at age 18 years appears to provide protection against breast cancer [10, 13–15]. Studies assessing the relationship between anthropometric measurements and survival indicate that greater BMI is associated with poorer prognosis and higher recurrence rates [16–21], and that a highly significant association exists between body weight (less than versus greater than 60 kg) and survival ($p = 0.0002$) [22]. A recent study found a hazard ratio for survival from breast cancer of 2.6 (95 % CI 1.63–4.17) for women with android adiposity (high suprailiac:thigh circumference ratio) [23]; however, another study assessing the association between weight and cancer recurrence and survival did not identify a significant hazard ratio [24]. A recent report showed that women with BMI below 18.5 kg/m^2 at time of diagnosis had significantly reduced survival as compared to women with BMI over 18.5 kg/m^2, including those with BMI greater than 25 kg/m^2 [25]. Although limited data are available regarding risk for breast cancer recurrence and weight gain post-diagnosis, survivors with an increased BMI appear to be at greater risk for breast cancer recurrence in the majority of studies that have examined this issue [26]. Whether body composition (proportion of lean body mass, body fat percentage) is also of relevance has not been previously examined. Evaluating body weight and body composition among women previously treated for breast cancer is central to advancing our understanding of the interplay between these modifiable factors and long-term survival.

In 1993, the Women's Healthy Eating and Living (WHEL) Study was initiated [27]. This multi-center diet intervention trial is being conducted to determine if diet modification can reduce breast cancer recurrence rates. Presented here are the results of an ancillary study of the larger WHEL study, which investigated the association between reported changes in dietary intake and body weight and body composition measures.

Subjects and methods

Subjects

A convenience sample of seventy-seven women who were recruited into the larger WHEL study at the Arizona site between 1996–1997 was recruited into this ancillary study cohort. All subjects underwent an initial eligibility screening, and medical records were reviewed to verify diagnosis, staging and treatment of breast cancer. According to the WHEL study eligibility criteria, patients were considered to be eligible to participate in the study if they had completed conventional therapy for Stage I, II, or IIIA primary operable invasive breast cancer within the past four years, were between the ages of 18 and 70 years, were accessible by telephone, and had completed a physician evaluation within 3 months of randomization to confirm no evidence of recurrent disease. Exclusion criteria included current smokers, pregnancy, liver or renal dysfunction, other primary or recurrent invasive cancer and an inability to commit to intervention schedule.

Randomization and intervention

Eligible participants signed an informed consent form that was approved and determined to be in accord with ethical standards of the Human Subjects Committee at The University of Arizona. Prior to randomization into a study group, participants were stratified by age and stage of tumor. Randomization to the intervention group or comparison group was in a 1:1 ratio using a computer-generated random permuted block design. After randomization, subjects participated in a three-hour dietary counseling session conducted by a registered dietitian. Participants in the intervention group were prescribed a diet high in fruit, vegetables and fiber, and low in fat, aiming for eight servings of fruits and vegetables per day, ≥30 g/d of fiber, and 20 % energy as fat. The intervention group received intensive telephone dietary counseling by trained diet counselors located at the Study Coordinating Center, which initially was provided on a weekly basis (4 weeks), then monthly (6 months), and then quarterly (9 to 48 months). In addition, monthly cooking classes and newsletters were provided to support their efforts to achieve and maintain study dietary goals. Participants in the comparison arm of the study were provided general instructions and materials consistent with current guidelines for cancer prevention, including five fruits and/or vegetables daily, < 30 % of energy from fat, and a diet which provides > 100 % recommended intakes to meet requirements for all nutrients. The comparison group participants received no telephone dietary counseling; however, they were invited to participate in quarterly cooking classes

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and were also provided a quarterly newsletter. Details regarding the WHEL Study design have been published elsewhere [28].

Anthropometric and body composition measurements

Measurements were taken at five time points: baseline, 6 and 12 months, 24 or 36 months, and 48 months. Anthropometric measurements recorded during the study include height, weight, and waist and hip circumferences using standardized procedures [29, 30]. Waist:hip ratio (WHR) and BMI were calculated for each participant using standard formulas.

Body composition data were estimated using bioelectrical impedance. Measurements were made at baseline 6 months and end of study using the Bioresistance Body Composition Analyzer 1990B (Valhala Scientific, Inc, San Diego, CA). Participants were measured after an overnight fast and reportedly had abstained from caffeine, alcohol or heavy exercise for at least 24 hours prior to measurement. Participants were measured in a prone position with shoes, socks and with metallic jewelry or accessories removed.

Dietary intake assessment

Dietary intake was measured at baseline, 6, 12 and 48 months using the Arizona Food Frequency Questionnaire (AFFQ). The questionnaire is a modification of the Block questionnaire [31], with improved assessment of Southwestern U. S. food items, fiber and plant foods. The questionnaire was revised in 1995 to be more sensitive to intake of plant-based foods, a change necessary for its use in this diet intervention study. An earlier, but highly similar, version of the AFFQ has been described elsewhere and validated with plasma carotenoids [32, 33]. Analysis of intake was completed using the U. S. Department of Agriculture Nutrient Database for Standard Reference [34]. Participants were provided the questionnaire by mail with written instructions. The participant then brought the completed questionnaire to the clinic at which time it was reviewed for completeness by the study staff.

Statistical analysis

Descriptive statistics, including mean, median, standard deviation and percentage distributions were used to describe the demographic characteristics of the study population by treatment group. Anthropometric and body composition data were analyzed for change from pre- to post-intervention using conditional change score analysis, controlling for baseline status. The difference in change at 6 months between intervention and comparison groups was analyzed by t-test (change at 6 months was defined as the difference between baseline and 6 months). Repeated measures ANOVA using a mixed model approach was conducted to determine if significant differences existed between the study groups for dietary intake, anthropometric measurements and body composition over time (baseline, 6, 12, 24 or 36, and 48 months). For each variable, the difference between intervention and comparison groups and the change over time was tested. In the analysis, the intervention by time interaction effect was also tested. If there was no significant interaction effect, the change over time was analyzed regardless of treatment effect. The statistical significance was defined as $p < 0.05$ in all analyses. The repeated measures ANOVA was performed using SAS/STAT version 6.12 software (SAS Institute Inc., Cary, NC); all other data analyses were performed using SPSS version 11.5.0 statistical software (SPSS Inc., Chicago, IL).

Results

Seventy-seven women were randomized into this study. Fifty-two were included in the final analysis for the study. Of the 25 participants not included in the final dataset, ten were in the comparison arm and 15 were in the intervention arm. Four participants died, four relocated, one was intolerant of the study diet, four became inactive in the study, and ten were unable to be rescheduled for collection of study measures in a timely manner. Two severely obese subjects (BMI greater than 40 kg/m^2 [35]), one from each study arm, were excluded from the final analyses due to concerns with invalid data because of the known error in body composition analysis using bioelectrical impedance in severely obese subjects [36].

Sociodemographic characteristics of the study population are shown in Table 1. Demographic comparisons of the two study groups (intervention and comparison arms) show no significant differences between groups. Overall, the study participants were mainly well-educated, post-menopausal, Caucasian females previously diagnosed with stage I or II invasive breast cancer. On average, study participants reported a 20.6 kg increase in body weight over adult life (data not shown). Demographic characteristics of this cohort are comparable to the larger WHEL population with the exception of antiestrogen therapy status. At enrollment, approximately 35 % of the participants in this study, compared to 61 % of the WHEL population, reported current antiestrogen therapy use. Previous antiestrogen therapy was reported by approximately 19 % of participants in this study compared to 7.2 % of the WHEL population.

Table 1 Socio-demographic characteristics of breast cancer survivors participating in a dietary intervention trial

	Sub-study participants (n = 52)	
	Intervention	Comparison
Age (years)[1]	55.5 ± 9.2 (21)	52.3 ± 9.0 (31)
Ethnicity[2]		
White, not Hispanic	100.00 (21)	90.3 (28)
Hispanic	0.00 (0)	9.7 (3)
College education[2]	53.2 (16)	52.8 (19)
Stage of cancer at diagnosis[2]		
I	38.1 (8)	48.4 (15)
II	57.1 (12)	48.4 (15)
IIIA	4.8 (1)	3.2 (1)
Menopausal status[2]		
Premenopausal	9.5 (2)	16.1 (5)
Perimenopausal	14.3 (3)	6.5 (2)
Postmenopausal	76.2 (16)	77.4 (24)
Antiestrogen therapy[2]		
Current	38.1 (8)	32.3 (10)
Past	14.3 (3)	22.6 (7)
Never	47.6 (10)	45.2 (14)
Weight age 18 years (kg)[1]	53.5 ± 7.0 (21)	55.8 ± 6.0 (31)
Highest adult weight (kg)[1]	74.4 ± 16.0 (17)	76.2 ± 10.7 (24)

Premenopausal defined as regular menses, *perimenopausal* defined as irregular menses for > one year, and *postmenopausal* defined as no reported menstruation for > one year

[1] Values are mean ± standard deviation (n)

[2] Values are percentage of study population (n)

Dietary intake

Baseline, 6-, 12-, and 48-month, and change in dietary intakes from baseline to 48 months are shown in Table 2. Analysis of dietary intake indicated that at baseline, dietary intake was similar for the intervention and comparison groups with the exception of the intervention group consuming more fruit. Analysis of intakes by treatment group indicated that the intervention group reported significant increases in intakes from baseline of fiber and vegetables compared to the comparison group at 6 and 12 months, but not at 48 months. Compared to the intervention group, the comparison group reported reduced protein intake from baseline that was significant only at 12 months. Significant differences in dietary intake change over time were detected for energy, carbohydrate, fiber, alcohol, vegetables, fruit and percentage energy as fat.

Anthropometric and body composition measurements

There were no significant differences in mean height, weight, BMI, WHR, percentage body fat, and lean body mass prior to a randomization to a study treatment group (Table 3). In addition, we observed no significant differences in these measures by treatment group after 6, 12, 24, 36 or 48 months of dietary intervention. The intervention and comparison groups were marginally significantly different in regard to changes in body weight (loss of 3.1 kg versus gain of 1.6 kg, respectively) at the 6-month time point ($p = 0.07$) but by 12 months, weight regain to baseline level was demonstrated in the intervention group while the comparison group subjects showed a relatively stable weight pattern throughout the study period. Body composition data also indicated an initial (6 month) marginally significant decline in fat stores for intervention group but not for comparison group subjects (loss of 1.6% versus loss of 0.4%) ($p = 0.10$). Repeated measures analysis of longitudinal changes by treatment group showed a significant change over the course of the study (48 months) for percentage body fat ($p < 0.0439$). Concurrently, the intervention group showed a significant decline in lean body mass over time as compared to the comparison group ($p < 0.0484$).

Discussion

Results of this study indicate minimal short-term changes in body weight and composition occur in response to a high-vegetable, low-fat diet in breast cancer survivors, but these changes were not sustained over time.

The study groups showed similar patterns of dietary intake of total fat, saturated fat, fiber and vegetables at baseline, and the dietary counseling program was able to produce significant dietary changes for the intervention group, including increased fiber and vegetable intakes and decreased fat intake within six months, and these changes were sustained through 12 months. Although both study groups reported a net reduction in energy intake over time, the body weight and composition data do not corroborate these reports. These data likely indicate probable under-reporting of energy intake among overweight women as has been reported [37] because no reductions in body weight, BMI or body fat were observed as would be expected with sustained reduced energy consumption. An earlier report on another sub-sample of WHEL study participants showed that even when significant reductions in fat intake were demonstrated among intervention group participants, body weight was not reduced at 12 months [38]; however, no data on body composition were available for that subset. In this study, a significant decrease in body fat (percentage and mass) was observed at 6 months for the intervention group as compared to baseline. In addition, a significant difference ($p = 0.0439$) in percentage body fat change over time was observed between diet groups (1.04% versus 2.27% for intervention and comparison groups, re-

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Table 2 Change in dietary intake in response to a high fruit and vegetable, low fat dietary intervention among women previously treated for breast cancer

Food group/Nutrient	Months on study				Mean change 0 to 48 months	Difference between groups[1] (p)	Difference across time points[1] (p)
	Baseline	6	12	48			
	mean ± standard deviation						
Energy (kcal/day)							
Intervention	2120±626 (20)	1992±642 (18)	1937±821 (13)	1878±923 (21)	−283±794 (20)	0.0698	0.0385
Comparison	1915±824 (31)	1752±819 (27)	1485±613 (24)	1556±707 (31)	−359±668 (31)		
Carbohydrate (g/day)							
Intervention	339.9±143.8 (20)	327.7±114.1 (18)	318.3±169.4 (13)	276.8±168.7 (21)	−71.3±142.8 (20)	0.0828	0.0010
Comparison	289.7±133.4 (31)	265.3±125.0 (27)	223.0±96.7 (24)	221.9±98.7 (31)	−67.8±109.8 (31)		
Protein (g/day)							
Intervention	74.4±25.4 (20)	77.6±30.2 (18)	72.9±30.8 (13)	76.0±36.3 (21)	1.6±29.4 (20)	0.0475	0.3137
Comparison	71.9±31.8 (31)	68.5±32.8 (27)	57.1±24.2 (24)	60.3±29.8 (31)	−11.6±26.8 (31)		
Total fat (g/day)							
Intervention	54.7±17.5 (20)	49.2±18.3 (18)	45.1±13.9 (13)	56.3±30.0 (21)	0.8±22.8 (20)	0.6403	0.7811
Comparison	56.3±25.5 (31)	51.2±30.3 (27)	43.9±21.2 (24)	49.9±28.4 (31)	−6.4±23.5 (31)		
Energy from fat (%)							
Intervention	24.2±7.1 (20)	22.4±4.7 (18)	22.6±5.1 (13)	28.1±9.0 (21)	4.1±7.8 (20)	0.0287	0.0051
Comparison	26.5±4.3 (31)	26.2±6.2 (27)	26.3±4.7 (24)	28.0±5.7 (31)	1.5±5.8 (31)		
Saturated fat (g/day)							
Intervention	17.9±7.0 (20)	14.2±5.8 (18)	14.4±4.1 (13)	19.1±12.8 (21)	1.1±10.4 (20)	0.5239	0.7580
Comparison	18.0±8.2 (31)	15.6±10.1 (27)	13.5±6.6 (24)	15.8±8.8 (31)	−2.1±7.8 (31)		
Cholesterol (mg/day)							
Intervention	174.9±68.9 (20)	159.4±78.5 (18)	156.4±55.3 (13)	195.1±125.5 (21)	20.3±112.6 (20)	0.2202	0.1565
Comparison	159.3±81.4 (31)	182.1±203.7 (27)	128.3±71.5 (24)	176.8±162.1 (31)	17.5±158.8 (31)		
Total fiber (g/day)							
Intervention	27.1±15.0 (20)	38.6±17.5 (18)	31.8±16.5 (13)	28.6±18.1 (21)	1.1±17.6 (20)	0.0653	0.0214
Comparison	25.8±12.2 (31)	26.1±12.4 (27)	20.3±10.2 (24)	21.1±13.9 (31)	−4.7±12.1 (31)		
Meat (g/day)							
Intervention	28.0±17.5 (19)	18.8±9.7 (16)	20.3±7.2 (11)	35.5±33.8 (20)	6.5±25.0 (19)	0.8007	0.3205
Comparison	29.7±22.2 (29)	28.2±25.1 (26)	19.3±15.3 (21)	27.1±28.1 (28)	−2.7±27.4 (28)		
Fruit (servings/day)[2]							
Intervention	4.4±4.2 (20)	5.0±2.6 (18)	5.0±3.3 (13)	4.0±3.2 (21)	−0.6±3.6 (20)	0.0014	0.0372
Comparison	3.0±1.7 (31)	3.5±2.3 (27)	2.6±1.4 (24)	2.5±1.8 (31)	−0.5±1.8 (31)		
Vegetables (servings/day)[3]							
Intervention	4.7±3.5 (20)	8.4±4.3 (18)	7.3±4.3 (13)	5.3±4.1 (21)	0.6±4.6 (20)	0.1509	0.0329
Comparison	3.8±2.7 (31)	4.0±2.8 (27)	3.4±3.3 (24)	3.5±2.8 (31)	−0.3±3.2 (31)		
Alcohol (g/day)							
Intervention	114.9±131.0 (12)	84.7±67.7 (10)	131.6±126.9 (7)	90.1±102.3 (14)	−46.4±107.9 (10)	0.0145	0.0090
Comparison	72.8±56.8 (18)	40.2±33.0 (19)	54.3±53.5 (16)	69.1±78.5 (22)	9.9±78.7 (17)		

[1] Repeated measures ANOVA; [2] Includes fruit juice; [3] Includes vegetable juice

spectively). Given the small degree of change in percentage body fat, however, this may not be clinically relevant. Of interest is that our body composition findings are different from patterns seen in other studies of survivors in that lean body mass was relatively stable, particularly in the comparison study group, over the 48 months. Other studies following women immediately post-diagnosis have shown an increase in body weight with decreased lean body mass [39, 40], whereas the women in this study were on average 2 years post-diagnosis. These data suggest that perhaps there are different patterns of body composition at different phases post-diagnosis. Although small, the differences in lean body mass over time by treatment group were significant. If sustained, a pattern of increased percentage body fat and decreased lean body mass demonstrated in the intervention group could have long-term health consequences. Thus, body composition should be evaluated in diet intervention trials among breast cancer survivors to further monitor these compartments.

Small but non-significant increases in body weight, WHR and BMI were also observed in this study, regardless of treatment group assignment. For each of these parameters, the net increase was greater in comparison

Table 3 Change in anthropometric and body composition measurements in response to a plant-based dietary intervention or comparison diet among women previously treated for breast cancer

Anthropometric measure	Months on study						Mean change 0 to 48 months	Difference between groups[1] (p)	Difference among time points[1] (p)
	Baseline	6	12	24	36	48			
	mean ± standard deviation (n)								
Weight (kg)									
Intervention	70.4 ± 15.3 (21)	67.0 ± 13.5 (19)	69.9 ± 16.0 (20)	75.6 ± 10.6 (9)	71.8 ± 18.4 (11)	71.1 ± 15.8 (21)	0.72 ± 6.52 (21)	0.1098	0.3287
Comparison	72.2 ± 9.5 (31)	72.6 ± 9.6 (29)	72.4 ± 9.9 (30)	75.4 ± 10.7 (15)	71.1 ± 11.2 (13)	74.2 ± 10.8 (31)	1.93 ± 8.10 (30)		
Body mass index (kg/m²)									
Intervention	26.4 ± 4.6 (21)	25.7 ± 4.2 (19)	26.5 ± 4.8 (20)	28.4 ± 3.0 (9)	27.9 ± 5.2 (11)	27.1 ± 5.0 (21)	0.71 ± 1.96 (21)	0.2763	0.1033
Comparison	26.9 ± 4.5 (31)	27.3 ± 4.7 (29)	27.0 ± 4.6 (30)	27.8 ± 3.6 (15)	27.5 ± 5.3 (13)	28.1 ± 4.7 (31)	1.26 ± 3.02 (30)		
Waist:Hip ratio									
Intervention	0.80 ± 0.08 (21)	0.81 ± 0.08 (19)	0.81 ± 0.07 (20)	0.83 ± 0.06 (9)	0.80 ± 0.07 (10)	0.83 ± 0.09 (21)	0.03 ± 0.05 (21)	0.1972	0.1828
Comparison	0.78 ± 0.06 (31)	0.80 ± 0.08 (28)	0.80 ± 0.07 (30)	0.81 ± 0.07 (15)	0.80 ± 0.08 (13)	0.80 ± 0.07 (31)	0.02 ± 0.05 (30)		
Body fat (%)									
Intervention	31.6 ± 5.0 (21)	30.1 ± 4.8 (19)				32.6 ± 6.3 (21)	1.04 ± 4.99 (21)	0.7591	0.0439
Comparison	31.1 ± 5.8 (30)	31.0 ± 4.4 (29)				33.1 ± 5.6 (30)	2.27 ± 4.19 (29)		
Body fat (kg)									
Intervention	22.4 ± 8.6 (21)	20.6 ± 7.0 (19)				24.0 ± 9.4 (21)	1.54 ± 5.58 (21)	0.2403	0.1990
Comparison	22.7 ± 6.4 (30)	24.8 ± 10.8 (29)				24.9 ± 7.0 (30)	2.60 ± 3.91 (29)		
Lean body mass (kg)									
Intervention	47.7 ± 7.3 (21)	46.4 ± 7.4 (19)				47.1 ± 7.2 (21)	−0.62 ± 2.65 (21)	0.0484	0.9275
Comparison	48.6 ± 5.8 (30)	49.7 ± 4.9 (29)				49.0 ± 4.8 (30)	0.64 ± 4.74 (29)		

[1] Repeated measures ANOVA

group subjects compared to intervention group subjects. Perhaps larger sample sizes may have detected significant differences, but trend analyses also did not achieve significance (data not shown). Weight loss or reductions in WHR, BMI and body fat are not among the aims of the WHEL Study, which instead focuses on modified diet composition. Nonetheless, consuming a low fat, high fruit and vegetable, high fiber diet has been previously hypothesized to lead to improvements in these health status parameters [41]. One explanation for the lack of significant change, of course, is the exclusion of energy goals in the diet intervention and counseling. Other explanations include the observation that dietary restraints and avoidance of select foods can result in weight gain among breast cancer survivors [42] or that survivors with abnormal eating attitudes and behaviors demonstrate higher BMIs (as with the study participants described here) [43].

These data support the need for additional studies that address energy balance and increased energy expenditure (physical activity) in an effort to improve body weight and composition and thus positively impact risk for breast cancer recurrence. The association between body weight and breast cancer risk has been examined in several epidemiological studies [29, 44, 45], but seldom in the context of a clinical trial.

Other limitations of this study need to be considered. First, body composition changes (percentage body fat) were unchanged when comparing baseline values to end of study values. Fat mass is relatively stable in adults; however, standard error in measurement has been estimated as 2–3 % [46, 47]; thus, even the 1.5 % reduction seen at 6 months is likely not clinically significant. The use of dual energy x-ray absorptiometry (DXA) for body composition assessment, which is now more readily available and cost effective than it was at the time when this study was initiated, may have allowed for more precise measurements. Second, although repeated measures over time provide significant power to detect differences between groups, the small sub-group sample size made it statistically inappropriate to run sub-analyses in regard to factors such as menopausal status or previous hormone replacement therapy, factors which may modify body weight.

In summary, changes in dietary intake in this cohort of breast cancer survivors were not associated with significant changes in adiposity or body weight. What remains to be determined is whether reductions in weight or body fat in women previously treated for breast cancer will reduce their risk for recurrence. Additional research which includes larger cohorts and longer-term serial measures of body composition, in response to modulation of dietary intake and physical activity to promote healthy body weight, is necessary before clinical recommendations can be formulated.

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References

1. de Waard F (1975) Breast cancer incidence and nutritional status with particular reference to body weight and height. Cancer Res 35:3351–3356
2. Ballard-Barbash R, Birt DF, Kestin M, King IB (1997) Perspectives on integrating experimental and epidemiologic research on diet, anthropometry and breast cancer. J Nutr 127:936S–939S
3. Huang Z, Willett WC, Colditz GA, Hunter DJ, Manson JE, Rosner B, Speizer FE, Hankinson SE (1999) Waist circumference, waist:hip ratio, and risk of breast cancer in the Nurses' Health Study. Am J Epidemiol 150:1316–1324
4. Hunter DJ, Willett WC (1993) Diet, body size, and breast cancer. Epidemiol Rev 15:110–132
5. Sonnenschein E, Toniolo P, Terry MB, Bruning PF, Kato I, Koenig KL, Shore RE (1999) Body fat distribution and obesity in pre- and postmenopausal breast cancer. Int J Epidemiol 28:1026–1031
6. Stoll BA (2000) Adiposity as a risk determinant for postmenopausal breast cancer. Int J Obes 24:527–533
7. Ziegler RG (1997) Anthropometry and breast cancer. J Nutr 127:924S–928S
8. Wenten M, Gilliland FD, Baumgartner K, Samet JM (2002) Associations of weight, weight change, and body mass with breast cancer risk in Hispanic and non-Hispanic white women. Ann Epidemiol 12:435–444
9. Sellers TA, Davis J, Cerhan JR, Vierkant RA, Olson JE, Pankratz VS, Potter JD, Folsom AR (2002) Interaction of waist/hip ratio and family history on the risk of hormone receptor-defined breast cancer in a prospective study of postmenopausal women. Am J Epidemiol 155:225–233
10. Huang Z, Hankinson SE, Colditz GA, Stampfer MJ, Hunter DJ, Manson JE, Hennekens CH, Rosner B, Speizer FE, Willett WC (1997) Dual effects of weight and weight gain on breast cancer risk. JAMA 278:1407–1411
11. Moorman PG, Jones BA, Millikan RC, Hall IJ, Newman B (2001) Race, anthropometric factors, and stage at diagnosis of breast cancer. Am J Epidemiol 153: 284–291
12. La Vecchia C, Negri E, Franceschi S, Talamini R, Bruzzi P, Palli D, Decarli A (1997) Body mass index and postmenopausal breast cancer: an age-specific analysis. Br J Cancer 75:441–444
13. Brinton LA, Swanson CA (1992) Height and weight at various ages and risk of breast cancer. Ann Epidemiol 2:597–609
14. Ursin G, Longnecker MP, Haile RW, Greenland S (1995) A meta-analysis of body mass index and risk of premenopausal breast cancer. Epidemiology 6:137–141
15. Magnusson C, Baron J, Persson I, Wolk A, Bergstrom R, Trichopoulos D, Adami HO (1998) Body size in different periods of life and breast cancer risk in post-menopausal women. Int J Cancer 76:29–34
16. Tartter PI, Papatestas AE, Ioannovich J, Mulvihill MN, Lesnick G, Aufses AH, Jr (1981) Cholesterol and obesity as prognostic factors in breast cancer. Cancer 47:2222–2227
17. Senie RT, Rosen PP, Rhodes P, Lesser ML, Kinne DW (1992) Obesity at diagnosis of breast carcinoma influences duration of disease-free survival. Ann Intern Med 116:26–32
18. Newman SC, Lees AW, Jenkins HJ (1997) The effect of body mass index and oestrogen receptor level on survival of breast cancer patients. Int J Epidemiol 26:484–490
19. Hebert JR, Hurley TG, Ma Y (1998) The effect of dietary exposures on recurrence and mortality in early stage breast cancer. Breast Cancer Res Treat 51:17–28
20. Overvad K (2002) Body weight, physical activity and cancer. IARC Sci. Publ 156: 236–236
21. Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ (2003) Overweight, obesity, and mortality from cancer in a prospectively studied cohort of US adults. N Engl J Med 348:1625–1638
22. Haybittle J, Houghton J, Baum M (1997) Social class and weight as prognostic factors in early breast cancer. Br J Cancer 75:729–733
23. Kumar NB, Cantor A, Allen K, Cox CE (2000) Android obesity at diagnosis and breast carcinoma survival – Evaluation of the effects of anthropometric variables at diagnosis, including body composition and body fat distribution and weight gain during life span, and survival from breast carcinoma. Cancer 88:2751–2757
24. Saxe GA, Rock CL, Wicha MS, Schottenfeld D (1999) Diet and risk for breast cancer recurrence and survival. Breast Cancer Res Treat 53:241–253
25. Marret H, Perrotin F, Bougnoux P, Descamps P, Hubert B, Lefranc T, Le Floch O, Lansac J, Body G (2001) Low body mass index is an independent predictive factor of local recurrence after conservative treatment for breast cancer. Breast Cancer Res Treat 66:17–23
26. Rock CL, Flatt SW, Newman V, Caan BJ, Haan MN, Stefanick ML, Faerber S, Pierce JP (1999) Factors associated with weight gain in women after diagnosis of breast cancer. Women's Healthy Eating and Living Study Group. J Am Diet Assoc 99:1212–1221
27. Pierce JP, Faerber S, Wright FA, Newman V, Flatt SW, Kealey S, Rock CL, Hryniuk W, Greenberg ER (1997) Feasibility of a randomized trial of a high-vegetable diet to prevent breast cancer recurrence. Nutr Cancer 28:282–288
28. 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, Thomas RG, Gilpin EA, Women's Healthy Eating and Living (WHEL) Study Group (2002) A randomized trial of the effect of a plant-based dietary pattern on additional breast cancer events and survival: the Women's Healthy Eating and Living (WHEL) Study. Control Clin Trials 23: 728–756
29. London SJ, Colditz GA, Stampfer MJ, Willett WC, Rosner B, Speizer FE (1989) Prospective study of relative weight, height, and risk of breast cancer. JAMA 262:2853–2858

30. Trentham-Dietz A, Newcomb PA, Egan KM, Titus-Ernstoff L, Baron JA, Storer BE, Stampfer M, Willett WC (2000) Weight change and risk of postmenopausal breast cancer (United States). Cancer Causes Control 11: 533–542
31. Block G (1989) Health, Habits, and History Questionnaire – Diet History and Other Risk Factors, Personal Computer System Packet. National Cancer Institute, Bethesda, MD
32. Ritenbaugh C, Peng YM, Aickin M, Graver E, Branch M, Alberts DS (1996) New carotenoid values for foods improve relationship of food frequency questionnaire intake estimates to plasma values. Cancer Epidemiol Biomarkers Prev 5:907–912
33. Ritenbaugh C, Aickin M, Taren D, Teufel N, Graver E, Woolf K, Alberts DS (1997) Use of a food frequency questionnaire to screen for dietary eligibility in a randomized cancer prevention phase III trial. Cancer Epidemiol Biomarkers Prev 6:347–354
34. US Department of Agriculture, Agricultural Research Service (2001) USDA Nutrient Database for Standard Reference, releases 11–13. Nutrient Data Laboratory, Beltsville Human Nutrition Research Center, Beltsville, MD (http://www.nal.usda.gov/fnic/foodcomp)
35. North American Association for the Study of Obesity; National Heart, Lung, and Blood Institute; National Institutes of Health (U. S.); NHLBI Obesity Education Initiative (2000) The Practical Guide: Identification, Evaluation, and Treatment of Overweight and Obesity in Adults. NIH Publication no. 00–4084. National Institutes of Health; National Heart, Lung, and Blood Institute; NHLBI Obesity Education Initiative; North American Association for the Study of Obesity, Bethesda, MD, p1 (http://www.nhlbi.nih.gov/guidelines/obesity/prctgd%5Fb.pdf)
36. National Institute of Diabetes and Digestive and Kidney Disease and the NIH Office of Medical Applications of Research (1994) Bioelectrical impedance analysis in body composition measurement. Technology assessment conference statement, 1994 Dec 12–14. National Institutes of Health, Bethesda, MD (http://consensus.nih.gov/ta/015/015_intro.htm)
37. Caan BJ, Flatt SW, Rock CL, Ritenbaugh C, Newman V, Pierce JP (2000) Low-energy reporting in women at risk for breast cancer recurrence. Women's Healthy Eating and Living Group. Cancer Epidemiol. Biomarkers Prev 9: 1091–1097
38. Rock CL, Thomson C, Caan BJ, Flatt SW, Newman V, Ritenbaugh C, Marshall JR, Hollenbach KA, Stefanick ML, Pierce JP (2001) Reduction in fat intake is not associated with weight loss in most women after breast cancer diagnosis. Cancer 91:25–34
39. Aslani A, Smith RC, Allen BJ, Pavlakis N, Levi JA (1999) Changes in body composition during breast cancer chemotherapy with the CMF-regimen. Breast Cancer Res Treat 57:285–290
40. Demark-Wahnefried W, Peterson BL, Winer EP, Marks L, Aziz N, Marcom PK, Blackwell K, Rimer BK (2001) Changes in weight, body composition, and factors influencing energy balance among premenopausal breast cancer patients receiving adjuvant chemotherapy. J Clin Oncol 19:2381–2389
41. Bray GA, Popkin BM (1998) Dietary fat intake does affect obesity! Am J Clin Nutr 68:1157–1173
42. DeGeorge D, Gray JJ, Fetting JH, Rolls BJ (1990) Weight gain in patients with breast cancer receiving adjuvant treatment as a function of restraint, disinhibition, and hunger. Oncol Nurs Forum 17:23–28
43. Rock CL, McEligot AJ, Flatt SW, Sobo EJ, Wilfley DE, Jones VE, Hollenbach KA, Marx RD (2000) Eating pathology and obesity in women at risk for breast cancer recurrence. Int J Eat Disord 27: 172–179
44. Hunter DJ, Manson JE, Colditz GA, Stampfer MJ, Rosner B, Hennekens CH, Speizer FE, Willett WC (1993) A prospective study of the intake of vitamins C, E, and A and the risk of breast cancer. N Engl J Med 329:234–240
45. Sellers TA, Kushi LH, Potter JD, Kaye SA, Nelson, CL, McGovern PG, Folsom AR (1992) Effect of family history, body-fat distribution, and reproductive factors on the risk of postmenopausal breast cancer [erratum appears in N Engl J Med 1992, 327:1612.]. N Engl J Med 326: 1323–1329
46. Cohn SH (1985) How valid are bioelectric impedance measurements in body composition studies? Am J Clin Nutr 42: 889–890
47. Hansen S, Cold S, Petersen PH, Rose C (1997) Estimates of the sources of variation (variance components) of bioelectric impedance and anthropometric measurements in an epidemiological case-control study of breast cancer. Eur J Clin Nutr 51:764–770