



Published in final edited form as:

Nutr Cancer. 2008 ; 60(1): 31–38. doi:10.1080/01635580701621320.

The Impact of a Long-Term Reduction in Dietary Energy Density on Body Weight Within a Randomized Diet Trial

Nazmus Saquib, Loki Natarajan, Cheryl L. Rock, Shirley W. Flatt, Lisa Madlensky, Sheila Kealey, and John P. Pierce

Cancer Prevention and Control Program, Moores UCSD Cancer Center, University of California, San Diego, California, USA

Abstract

We examined the effect of dietary energy density change on body weight in participants of a randomized trial. Intervention participants markedly increased fruit and vegetable intake while reducing energy intake from fat. Participants were 2,718 breast cancer survivors, aged 26–74 yr, with baseline mean body mass index of 27.3 kg/m² (SD = 6.3). We assessed dietary intake by sets of four 24-h dietary recalls and validated with plasma carotenoid concentrations. Weight and height were measured at baseline, 1 yr, and 4 yr. Dietary energy density was calculated using food but excluding beverages. Intervention participants significantly reduced dietary energy density compared to controls and maintained it over 4 yr—both in cross-sectional ($P < 0.0001$) and longitudinal (Group \times Time interaction, $P < 0.0001$) analyses. Total energy intake or physical activity did not vary between groups. The intervention group had a small but significant weight loss at 1 yr (Group \times Time interaction, $P < 0.0001$), but no between-group weight difference was observed at 4 yr. Our study showed that reducing dietary energy density did not result in a reduction in total energy intake and suggests that this strategy alone is not sufficient to promote long-term weight loss in a free-living population.

INTRODUCTION

Fiber, water, and fat are the 3 most important determinants of dietary energy density (1-3). Consequently, most fruit and vegetables are generally low in energy density due to their high fiber and water content (4-7). It has been observed that the volume of an individual's dietary intake remains more or less constant (8), which has led to the hypothesis that people may regulate their food intake based on volume rather than total energy. Accordingly, replacing energy-dense, high-fat foods with much less energy dense, fiber-rich foods such as vegetables and fruit should result in a reduction of energy intake and weight loss (8,9).

Various cross-sectional studies have found that individuals who eat high-energy-dense foods consume more energy and are relatively heavier than those who consume proportionately greater amounts of low-energy-dense foods (10-13). A number of feeding studies that have manipulated dietary energy density have suggested that a decrease in energy density is associated with weight loss (14,15). Although these feeding studies have addressed important questions about the association between energy density and weight loss, longer term studies of individuals eating in real-life situations are necessary to test the hypothesis.

Ad libitum randomized trials that have encouraged participants to increase their fruit and vegetable intake and/or decrease their fat intake have had mixed results in terms of the amount of validated dietary change as well as weight change (16-23). None of these trials have reported the energy density of the diets in the intervention and control groups, and thus, it is possible that those studies that did not observe a decrease in weight may not have achieved a significant change in dietary energy density.

This article investigates the relationship between change in dietary energy density and body weight as an ancillary report of the Women's Healthy Eating and Living (WHEL) Study—a large-scale randomized trial of the role of a plant-based dietary pattern in reducing breast cancer recurrence and death (24). Participants in the WHEL Study intervention group significantly increased their fruit, vegetable, and fiber intake and decreased their intake of energy from fat (25,26), a pattern characterizing a low-energy density diet, whereas the control participants consumed their usual diet. The WHEL Study has assessed dietary intake at multiple time points, and thus provides the necessary data to assess change in dietary energy density according to method reported in the literature (27). In this article, we compare dietary energy density between the intervention and the control groups at baseline and demonstrate the association between dietary energy density and body weight. Then, we investigate the relationship of change in energy density to change in weight between study groups up to 4 yr postrandomization.

MATERIALS AND METHODS

In this article, we consider participants of the WHEL Study. Population characteristics, eligibility criteria, randomization procedures, and dietary intervention protocol have been described in detail elsewhere (24,26).

Population

All women enrolled in the WHEL Study who did not have a study endpoint (death or recurrence) by 4 yr of follow-up were eligible for this study ($n = 2,718$). WHEL Study participants were aged 18–70 yr at cancer diagnosis; treated for primary, operable, and invasive stage I, II, or IIIA breast carcinoma; and at study entry were not receiving or scheduled for chemotherapy and had no evidence of cancer recurrence after initial treatment. Enrollment in another dietary trial, pregnancy, receiving estrogen replacement therapy, and presence of life-threatening medical conditions or diseases were key exclusion criteria.

In this study, we used WHEL baseline, 1-yr, and 4-yr follow-up data and adopted its randomized design for data analysis (control = 1,363, intervention = 1,355). Dietary data at baseline, 1 yr, and 4 yr were available for 2,713 (control = 1,360, intervention = 1,353), 2,465 (control = 1,270, intervention = 1,195), and 2,324 (control = 1,202, intervention = 1,122) women, respectively. At the same time points, 2,718 (control = 1,363, intervention = 1,355), 2,306 (control = 1,174, intervention = 1,132), and 2,146 (control = 1,116, intervention = 1,030) women had their body weight measured.

Informed written consent from study participants was collected in the WHEL Study. The Human Subjects Committee of the University of California, San Diego, and all participating institutions approved the study procedures.

Dietary Intervention

Participants in the intervention group were encouraged to maintain a dietary pattern that included a daily consumption of at least 5 vegetable servings, 16 ounces of vegetable juice (or equivalent vegetable servings), 3 fruit servings, 30 g of fiber (18 g/1,000 kcal), and 15–20%

energy from fat (24,26). Telephone counseling, monthly cooking classes, and newsletters were the principal methods to promote dietary change in the intervention participants. Control group participants received print materials that included dietary guidelines from the U.S. Department of Agriculture (28) and the National Cancer Institute (29) and a bimonthly cohort maintenance newsletter with general health and nutrition information unrelated to the intervention group's dietary goals.

Dietary Assessment

Dietary intake was assessed through a set of four 24-h dietary recalls at baseline, 1 yr, and 4 yr. Trained dietary assessors conducted these recalls by telephone on randomly selected days, stratified for weekend vs. weekdays, over a 3-wk period. The Nutrition Data System for Research (NDS-R) software was used to collect and estimate dietary intakes (NDS-R version 6.0, 2006, University of Minnesota, Minneapolis, MN). NDS-R included more than 18,000 food codes, including many ethnic foods, and over 8,000 brand-name products.

A number of strategies were used to maximize the accuracy of dietary recall data (30). Dietary assessors completed a training program that included standardized data collection, proper interview technique, and efficient use of dietary analysis software. Participants were trained, before study enrollment, to estimate serving sizes with food models, measuring cups, and spoons, and were provided with 2-dimensional food models for reference during recalls. In addition, assessors used a multipass method that improved recall accuracy by prompting to obtain detailed data about type, amount, and preparation method of foods eaten.

Calculation of Dietary Energy Density

We determined a participant's dietary energy density (kcal/g; 1 kcal = 4.18 kJ) for a dietary recall day by estimating total energy intake (kcal) for that day and dividing it by the total amount (g) of food reported being consumed on that day. Energy density values of the set of 4 days were averaged to derive a mean dietary energy density value for each participant. In our calculations, we excluded all beverages.

Physical Activity Assessment

Physical activity was determined from the Personal Habits questionnaire developed for Women's Health Initiative (WHI) (31), expressed as metabolic equivalents per week (Metmin/wk) (32), and completed at baseline, 1 yr, and 4 yr. For the WHEL Study, this questionnaire was calibrated with the standard 7-Day Physical Activity Recall (PAR) (33) and validated with an accelerometer reading (34). The accelerometer measured an average of 165 total min of physical activity per week, which was not statistically different from the 187 min reported for the PAR or the 171 min reported with the WHI 9-item questionnaire.

Ascertainment of Body Weight

Weight and height were measured—with the participants wearing light clothing and no shoes—during clinic visits (baseline, Yr 1, and Yr 4) scheduled in the WHEL Study. Body mass index (BMI) was calculated as weight (kg)/height (m²).

Other Variables

Information on cancer stage (I, II, IIIA) and demography was ascertained through medical records and questionnaire, respectively. Age at study entry was categorized into 10-yr age groups (<44, 45–54, 55–64, and ≥65 yr), and race was categorized as non-Hispanic White, African American, Hispanic, Asian American, and others. Other variables included were education (college graduate vs. nongraduate), employment status (yes, no), marital status (married vs. not married), and smoking (current, past, and never). We calculated summary

variables such as total fruit and vegetable intake (servings/day) and percent energy intake from fat/day from 24-h dietary recalls.

Validation of Dietary Intake With Biomarkers

Plasma carotenoids are well-known biomarkers of fruit and vegetable intake (35). The WHEL Study measured plasma carotenoid concentrations on a 28% random sample of subjects identified at baseline and has published plasma carotenoid measurement procedures and baseline to 1-yr results (25,36). In this analysis, we report total plasma carotenoid concentrations on the available population ($n = 881$) at baseline, 1 yr, and 4 yr. Total plasma carotenoids are the sum of the individual carotenoids separated and quantified (α -carotene, β -carotene, β -cryptoxanthin, lycopene, and lutein plus zeaxanthin) using high-performance liquid chromatography methodology (25). The mean laboratory day-to-day coefficient of variation for total plasma carotenoids was less than 7%.

Statistical Analyses

We compared baseline characteristics of the control and the intervention groups; demographic, behavioral, and cancer related variables, thought to be potential confounders of the relationship between dietary intake and weight, were examined in this respect.

Energy density was calculated using “food only” values. We used baseline values to assess univariable associations of energy density with categories of age, race, and BMI; one-way analysis of variance compared category means against a referent category. We also grouped participants into tertiles of baseline dietary energy density, calculated mean values of total energy intake, physical activity, and body weight for each tertile and compared tertiles using the lowest tertile as referent. We then compared baseline dietary energy density between the control and the intervention group and graphed energy density in each study group at each time period.

We also computed and compared total energy intake, physical activity, and body weight values in each study group at baseline, 1 yr, and 4 yr, testing for group differences with t -tests.

Finally, we used mixed effect models to assess change in energy density, total plasma carotenoids, total energy intake, physical activity, and body weight over the study follow-up period. We chose mixed models, as they are the best option available for correlated data and for data with random missing values. “Unstructured” covariance provided the smallest Akaike's information criterion value and was used in the mixed models.

All calculations were performed using SAS version 9.1 (SAS Institute, Cary, NC). All statistical tests were two-tailed with an alpha level of 0.05.

RESULTS

Baseline characteristics did not differ significantly between the randomly assigned control and intervention groups (Table 1). Women were 26–74 yr of age (mean age = 53.4, SD = 8.8). The mean BMI was 27.3 (SD = 6.3), and 57% were overweight or obese. Although predominantly non-Hispanic White (85%), the cohort also included a small but varied group of minority women (4% African American, 3% Asian American, 5% Hispanic, and 3% other ethnicities). Well-educated [college graduate (54%)] and predominantly employed (72%), 70% of the WHEL women were also married. Only a small percentage (<5%) was diagnosed with either stage IIIA cancer or was currently smoking. The mean energy intake and physical activity were 1,717 kcal/day (SD = 407) [7,184(1,703) kJ/day] and 868 metabolic equivalent task (MET)-min/wk (SD = 879), respectively (data not shown).

At baseline, energy density was inversely associated with age (P for trend < 0.0001) and directly associated with BMI (P for trend < 0.0001). Asian-American participants reported the highest intake of fruit and vegetables and the lowest energy intake from fat (data not shown), making the energy density of their diets significantly lower than any other racial/ethnic group (Table 2). We observed strong linear trends ($P < 0.0001$) across tertiles of energy density, with energy intake and body weight having strong positive associations and physical activity having a strong negative association. Participants in the highest tertile of energy density consumed, on average, approximately 300 kcal/day (1 kcal = 4.18 kJ) more and performed 450 MET-min/wk less physical activity than participants in the lowest tertile; mean body weight differed by 6.8 kg between these 2 tertiles (Table 3).

Mean dietary energy density did not differ between the intervention and the control subjects at baseline, although we observed a significant difference in dietary energy density between groups at 1 yr and 4 yr (P values < 0.0001). At 1 yr, the intervention group reported consuming a diet that was 25% less energy dense than their baseline diet. At 4 yr, this difference was still highly significant but had declined to 15% (Fig. 1). The multivariate analysis (Table 4) shows that these group differences in energy density were statistically significant at both 1 yr and 4 yr (P values for group by time interaction < 0.0001).

Total plasma carotenoid concentrations corroborated the between-group differences in fruit and vegetable intake as assessed by 24-h recall. In the validation sample (36), no significant differences were observed between groups at baseline, and carotenoid values in the control group were relatively unchanged at 1 yr and 4 yr. In contrast, total plasma carotenoid concentrations in the intervention group increased substantially, resulting in a 66% difference between groups at 1 yr and a 41% difference at 4 yr (data not shown).

Data for energy intake, physical activity, and body weight are presented in Fig. 1 and Table 4. At baseline, mean weight in the intervention group was slightly higher than the control group (+0.2%). At 1 yr, weight in the control group increased by 0.71 kg, whereas weight decreased by 0.05 kg in the intervention group, resulting in a mean weight in the intervention group that was 0.7% lower than that of the control group. The multivariate analysis identified this difference as statistically significant (Group \times Time interaction: $P < 0.0001$). At 4 yr, both groups had gained weight, and the mean weight for the intervention group was 0.7% higher than that of the control group. The longitudinal analysis did not identify this as statistically significant (Group \times Time interaction: $P = 0.23$).

Reported energy intake was essentially the same at baseline and 1 yr, and there was a nonsignificant 1.4% difference between groups at 4 yr. At baseline, the intervention group performed 5% less physical activity than the control group. Although both groups reported increasing their physical activity, the intervention group performed 3.6% less physical activity than the control group at 1 yr and 0.3% less at 4 yr. This change in physical activity was borderline significant at 4 yr (Group \times Time interaction: $P = 0.04$).

DISCUSSION

In this group of breast cancer survivors participating in a long-term dietary trial, we observed that an increase in fruit and vegetable intake and decrease in percent energy from fat resulted in a substantial decrease in dietary energy density that was not accompanied by weight loss. Specifically, intervention participants significantly increased their intake of fruit and vegetables (2.7 and 2.3 servings/day, respectively, at 1 and 4 yr; data not shown) and decreased their percent energy intake from fat (5.7% and 4.3%, respectively, at 1 and 4 yr; data not shown). These dietary changes resulted in a large decrease in dietary energy density compared to the control group whose diets and energy density remained relatively unchanged.

At 1 yr, we observed a 25% between-group difference in dietary energy density, which was associated with small (0.7%) difference in weight in the hypothesized direction; although significant, this weight loss was much less than meets general guidelines for successful weight change (37-39). However, the intervention group sustained their reduction of dietary energy density through 4 yr, and this reduction was not associated with a maintained lower weight. Accordingly, the results of this study do not support the hypothesis that a major reduction in dietary energy density will independently result in weight loss.

A key component of the energy density–weight loss hypothesis is the assumption that people who adopt a low energy density dietary pattern will regulate their food intake by volume rather than by total energy. We did not observe this phenomenon in our study population. Despite a substantial increase in fruit and vegetable intake in the intervention group, their total energy intake did not change at either follow-up point. Likewise, we observed no meaningful difference in change of physical activity, a surrogate marker of energy expenditure, between the study groups. Thus, physical activity does not explain the finding of no difference in weight change between groups.

This study is one of the few to examine a longitudinal association between a change in dietary energy density and body weight. Our findings differ from the results of the 2 other trials in the literature that have examined this association (40,41). In both trials, weight loss was significantly correlated with decrease in dietary energy density. However, differences in the study population, intervention, and duration of follow-up between those 2 trials (40,41) and this one are substantial. The intervention in PREMIER trial (41) involves many more components than the dietary intervention in our study. In addition to promoting a high-fiber and low-fat diet, it also promoted weight loss and physical activity and restricted alcohol and sodium in-take. Unlike our study, both trials (40,41) focused on overweight or obese participants, setting up the possibility of a regression to the mean effect on weight. Further, subjects in our study maintained their dietary pattern across 4 yr, allowing us to investigate the long-term influence of such a dietary pattern.

All dietary studies need to address measurement error, and low-energy reporting is a concern, as several studies have observed higher frequency of low-energy reporting in their intervention groups (42-45). A related issue is whether intervention subjects were more prone to bias and reported eating more “socially desirable” foods such as vegetables and fruits or less fat than actually consumed, which would directly influence dietary energy density. Although differential underreporting and social desirability bias among intervention subjects is possible, that could not explain the dietary difference observed between our study groups. Total plasma carotenoids—a biomarker of fruit and vegetable consumption—increased significantly among intervention subjects throughout the follow-up period but remained unchanged in the control group (Table 4).

This study has a number of strengths; primarily, its randomized trial design whereby randomization theoretically distributes all attributes of the study subjects, both measured and unmeasured, evenly between the groups. Neither reported caloric intake nor physical activity expenditure were different between study groups at any time point. The huge difference achieved in dietary energy density was confirmed with the accepted biomarker of vegetables and fruit. Further, in this study, we measured body weight and height, unlike many other studies that have used self-reported weight and height (46-48). Hence, the accuracy of outcome measures was higher. Finally, the cross-sectional associations of dietary energy density we described in this article are consistent with findings from previous studies (10,11,27,49).

However, this study was not a random sample of the population. WHEL participants were breast cancer survivors, generally White, highly educated, and predominantly employed;

therefore, these results may not be generalizable to the population at large. Follow-up measured weight data were not available for 10% of subjects who did not attend clinic visits; however, this missing data did not differ between study groups (control = 9.9%, intervention = 11.2%). Finally, this study could not address the hypothesis of whether low energy density in conjunction with caloric restriction leads to long-term weight loss.

In summary, the intervention in this randomized trial significantly reduced dietary energy density and maintained this change over 4 yr. This change in dietary pattern was not associated with a change in energy balance (total energy intake vs. expenditure), and it did not result in a meaningful change in weight in free-living individuals. As a strategy to specifically reduce total energy intake, reducing dietary energy density may be a useful component of weight management. However, changing this characteristic of the diet without a targeted reduction in energy intake does not appear to result in either reduced energy intake or weight loss.

ACKNOWLEDGMENTS

The authors thank Christine Hayes for her editorial support. This study was initiated with the support of the Walton Family Foundation and continued with funding from National Cancer Institute (NIH) Grant CA 69375. Some of the data were collected from General Clinical Research Centers, NIH grants M01-RR00070, M01-RR00079, and M01-RR00827.

REFERENCES

1. Drewnowski A. The role of energy density. *Lipids* 2003;38:109–115. [PubMed: 12733741]
2. Drewnowski A. Energy density, palatability, and satiety: implications for weight control. *Nutr Rev* 1998;56:347–353. [PubMed: 9884582]
3. Howarth NC, Saltzman E, Roberts SB. Dietary fiber and weight regulation. *Nutr Rev* 2001;59:129–139. [PubMed: 11396693]
4. Poppitt SD, Prentice AM. Energy density and its role in the control of food intake: evidence from metabolic and community studies. *Appetite* 1996;26:153–174. [PubMed: 8737167]
5. Rolls BJ, Ello-Martin JA, Tohill BC. What can intervention studies tell us about the relationship between fruit and vegetable consumption and weight management? *Nutr Rev* 2004;62:1–17. [PubMed: 14995052]
6. Tohill BC, Seymour J, Serdula M, Kettel-Khan L, Roll BJ. What epidemiologic studies tell us about the relationship between fruit and vegetable consumption and body weight. *Nutr Rev* 2004;62:365–374. [PubMed: 15508906]
7. Yao M, Roberts SB. Dietary energy density and weight regulation. *Nutr Rev* 2001;59:247–258. [PubMed: 11518179]
8. Ledikwe JH, Blanck HM, Kettel Khan L, Serdula MK, Seymour JD, et al. Dietary energy density is associated with energy intake and weight status in US adults. *Am J Clin Nutr* 2006;83:1362–1368. [PubMed: 16762948]
9. Bell EA, Rolls BJ. Energy density of foods affects energy intake across multiple levels of fat content in lean and obese women. *Am J Clin Nutr* 2001;73:1010–1018. [PubMed: 11382653]
10. de Castro JM. Dietary energy density is associated with increased intake in free-living humans. *J Nutr* 2004;134:335–341. [PubMed: 14747669]
11. Kant AK, Graubard BI. Energy density of diets reported by American adults: association with food group intake, nutrient intake, and body weight. *Int J Obes (London)* 2005;29:950–956. [PubMed: 15917854]
12. Stookey JD. Energy density, energy intake and weight status in a large free-living sample of Chinese adults: exploring the underlying roles of fat, protein, carbohydrate, fiber and water intakes. *Eur J Clin Nutr* 2001;55:349–359. [PubMed: 11378808]
13. Prentice AM, Poppitt SD. Importance of energy density and macronutrients in the regulation of energy intake. *Int J Obes Relat Metab Disord* 1996;20(2 Suppl):18–23.

14. Shintani TT, Beckham S, Brown AC, O'Connor HK. The Hawaii Diet: ad libitum high carbohydrate, low fat multi-cultural diet for the reduction of chronic disease risk factors: obesity, hypertension, hypercholesterolemia, and hyperglycemia. *Hawaii Med J* 2001;60:69–73. [PubMed: 11320614]
15. Shintani TT, Hughes CK, Beckham S, O'Connor HK. Obesity and cardiovascular risk intervention through the ad libitum feeding of traditional Hawaiian diet. *Am J Clin Nutr* 1991;53:1647S–1651S. [PubMed: 2031501]
16. Howard BV, Manson JE, Stefanick ML, Beresford SA, Frank G, et al. Low-fat dietary pattern and weight change over 7 years: the Women's Health Initiative Dietary Modification Trial. *JAMA* 2006;295:39–49. [PubMed: 16391215]
17. Lanza E, Schatzkin A, Daston C, Corle D, Freedman L, et al. Implementation of a 4-y, high-fiber, high-fruit-and-vegetable, low-fat dietary intervention: results of dietary changes in the Polyp Prevention Trial. *Am J Clin Nutr* 2001;74:387–401. [PubMed: 11522565]
18. Rock CL, Thomson C, Caan BJ, Flatt SW, Newman V, et al. Reduction in fat intake is not associated with weight loss in most women after breast cancer diagnosis: evidence from a randomized controlled trial. *Cancer* 2001;91:25–34. [PubMed: 11148556]
19. Smith-Warner SA, Elmer PJ, Tharp TM, Fosdick L, Randall B, et al. Increasing vegetable and fruit intake: randomized intervention and monitoring in an at-risk population. *Cancer Epidemiol Biomarkers Prev* 2000;9:307–317. [PubMed: 10750670]
20. Zino S, Skeaff M, Williams S, Mann J. Randomised controlled trial of effect of fruit and vegetable consumption on plasma concentrations of lipids and antioxidants. *BMJ* 1997;314:1787–1791. [PubMed: 9224079]
21. Maskarinec G, Chan CL, Meng L, Franke AA, Cooney RV. Exploring the feasibility and effects of a high-fruit and -vegetable diet in healthy women. *Cancer Epidemiol Biomarkers Prev* 1999;8:919–924. [PubMed: 10548322]
22. Djuric Z, Poore KM, Depper JB, Uhley VE, Lababidi S, et al. Methods to increase fruit and vegetable intake with and without a decrease in fat intake: compliance and effects on body weight in the nutrition and breast health study. *Nutr Cancer* 2002;43:141–151. [PubMed: 12588694]
23. Singh RB, Dubnov G, Niaz MA, Ghosh S, Singh R, et al. Effect of an Indo-Mediterranean diet on progression of coronary artery disease in high risk patients (Indo-Mediterranean Diet Heart Study): a randomised single-blind trial. *Lancet* 2002;360:1455–1461. [PubMed: 12433513]
24. Pierce JP, Faerber S, Wright FA, Rock CL, Newman V, et al. 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* 2002;23:728–756. [PubMed: 12505249]
25. Pierce JP, Natarajan L, Sun S, Al-Delaimy W, Flatt S, et al. Increases in plasma carotenoid concentrations in response to a major dietary change in the Women's Healthy Eating and Living Study. *Cancer Epidemiol Biomarkers Prev* 2006;15:1886–1892. [PubMed: 17035395]
26. Pierce JP, Newman VA, Flatt SW, Faerber S, Rock CL, et al. Telephone counseling intervention increases intakes of micronutrient- and phytochemical-rich vegetables, fruit and fiber in breast cancer survivors. *J Nutr* 2004;134:452–458. [PubMed: 14747688]
27. Ledikwe JH, Blanck HM, Khan LK, Serdula MK, Seymour JD, et al. Dietary energy density determined by eight calculation methods in a nationally representative United States population. *J Nutr* 2005;135:273–278. [PubMed: 15671225]
28. U.S. Department of Health and Human Services. Home Health And Garden Bulletin No. 232, Dietary Guidelines for Americans, 1995. Department of Health and Human Services; Washington, DC: 1995.
29. National Cancer Institute. Action Guide for Healthy Eating. National Cancer Institute, NIH Publication No. 95–3877; Bethesda, MD: 1995.
30. Newman VA, Thomson CA, Rock CL, Flatt SW, Kealey S, et al. Achieving substantial changes in eating behavior among women previously treated for breast cancer—an overview of the intervention. *J Am Diet Assoc* 2005;105:382–391. 488. [PubMed: 15746825]
31. The Women's Health Initiative Study Group. Design of the Women's Health Initiative clinical trial and observational study. *Control Clin Trials* 1998;19:61–109. [PubMed: 9492970]
32. Ainsworth BE, Haskell WL, Whitt MC, Irwin ML, Swartz AM, et al. Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc* 2000;32:S498–S504. [PubMed: 10993420]

33. Blair, SN. How to assess exercise habits and physical fitness.. In: Matarazzo, JD.; Herd, JA.; Miller, NE.; Weiss, SSM., editors. Behavioral Health: A Handbook of Health Enhancement and Disease Prevention. Wiley; New York: 1984. p. 424-447.
34. Johnson-Kozlow M, Rock CL, Gilpin EA, Rock CL, Pierce JP. Validation of the WHI brief physical activity questionnaire among women diagnosed with breast cancer. *Am J Health Behav* 2007;31:193–202. [PubMed: 17269909]
35. McEligot AJ, Rock CL, Flatt SW, Newman V, Farber S, et al. Plasma carotenoids are biomarkers of long-term high vegetable intake in women with breast cancer. *J Nutr* 1999;129:2258–2263. [PubMed: 10573560]
36. Natarajan L, Flatt SW, Sun X, Gamst AC, Major JM, et al. Validity and systematic error in measuring carotenoid consumption with dietary self-report instruments. *Am J Epidemiol* 2006;163:770–778. [PubMed: 16524958]
37. Ball K, Brown W, Crawford D. Who does not gain weight? Prevalence and predictors of weight maintenance in young women. *Int J Obes Relat Metab Disord* 2002;26:1570–1578. [PubMed: 12461673]
38. Harris TB, Savage PJ, Tell GS, Haan M, Kumahika S, et al. Carrying the burden of cardiovascular risk in old age: associations of weight and weight change with prevalent cardiovascular disease, risk factors, and health status in the Cardiovascular Health Study. *Am J Clin Nutr* 1997;66:837–844. [PubMed: 9322558]
39. Meltzer AA, Everhart JE. Self-reported substantial 1-year weight change among men and women in the United States. *Obes Res* 1995;3(2 Suppl):123s–134s. [PubMed: 8581768]
40. Rolls BJ, Roe LS, Beach AM, Kris-Etherton PM. Provision of foods differing in energy density affects long-term weight loss. *Obes Res* 2005;13:1052–1060. [PubMed: 15976148]
41. Ledikwe JH, Rolls BJ, Smiciklas-Wright H, Mitchell DC, Ard JD, et al. Reductions in dietary energy density are associated with weight loss in overweight and obese participants in the PREMIER trial. *Am J Clin Nutr* 2007;85:1212–1221. [PubMed: 17490955]
42. Caan B, Ballard-Barbash R, Slattery ML, Pinsky JL, Iber FL, et al. Low energy reporting may increase in intervention participants enrolled in dietary intervention trials. *J Am Diet Assoc* 2004;104:357–366. 491. [PubMed: 14993857]
43. Harnack L, Himes JH, Anliker J, Clay T, Gittelsohn J, et al. Intervention-related bias in reporting of food intake by fifth-grade children participating in an obesity prevention study. *Am J Epidemiol* 2004;160:1117–1121. [PubMed: 15561991]
44. Buzzard IM, Faucett CL, Jeffery RW, McBane L, McGovern P, et al. Monitoring dietary change in a low-fat diet intervention study: advantages of using 24-hour dietary recalls vs. food records. *J Am Diet Assoc* 1996;96:574–579. [PubMed: 8655904]
45. Kristal AR, Andrilla CH, Koepsell TD, Diehr PH, Cheadle A. Dietary assessment instruments are susceptible to intervention-associated response set bias. *J Am Diet Assoc* 1998;98:40–43. [PubMed: 9434649]
46. Togo P, Osler M, Sorensen TI, Heitman BL. A longitudinal study of food intake patterns and obesity in adult Danish men and women. *Int J Obes Relat Metab Disord* 2004;28:583–593. [PubMed: 14770197]
47. Newby PK, Muller D, Hallfrisch J, Qiao N, Andres R, et al. Dietary patterns and changes in body mass index and waist circumference in adults. *Am J Clin Nutr* 2003;77:1417–1425. [PubMed: 12791618]
48. He K, Hu FB, Colditz GA, Manson JE, Willett WC, et al. Changes in intake of fruits and vegetables in relation to risk of obesity and weight gain among middle-aged women. *Int J Obes Relat Metab Disord* 2004;28:1569–1574. [PubMed: 15467774]
49. Marti-Henneberg C, Capdevila F, Arijia V, Perez S, Cuco G. Energy density of the diet, food volume and energy intake by age and sex in a healthy population. *Eur J Clin Nutr* 1999;53:421–428. [PubMed: 10403576]

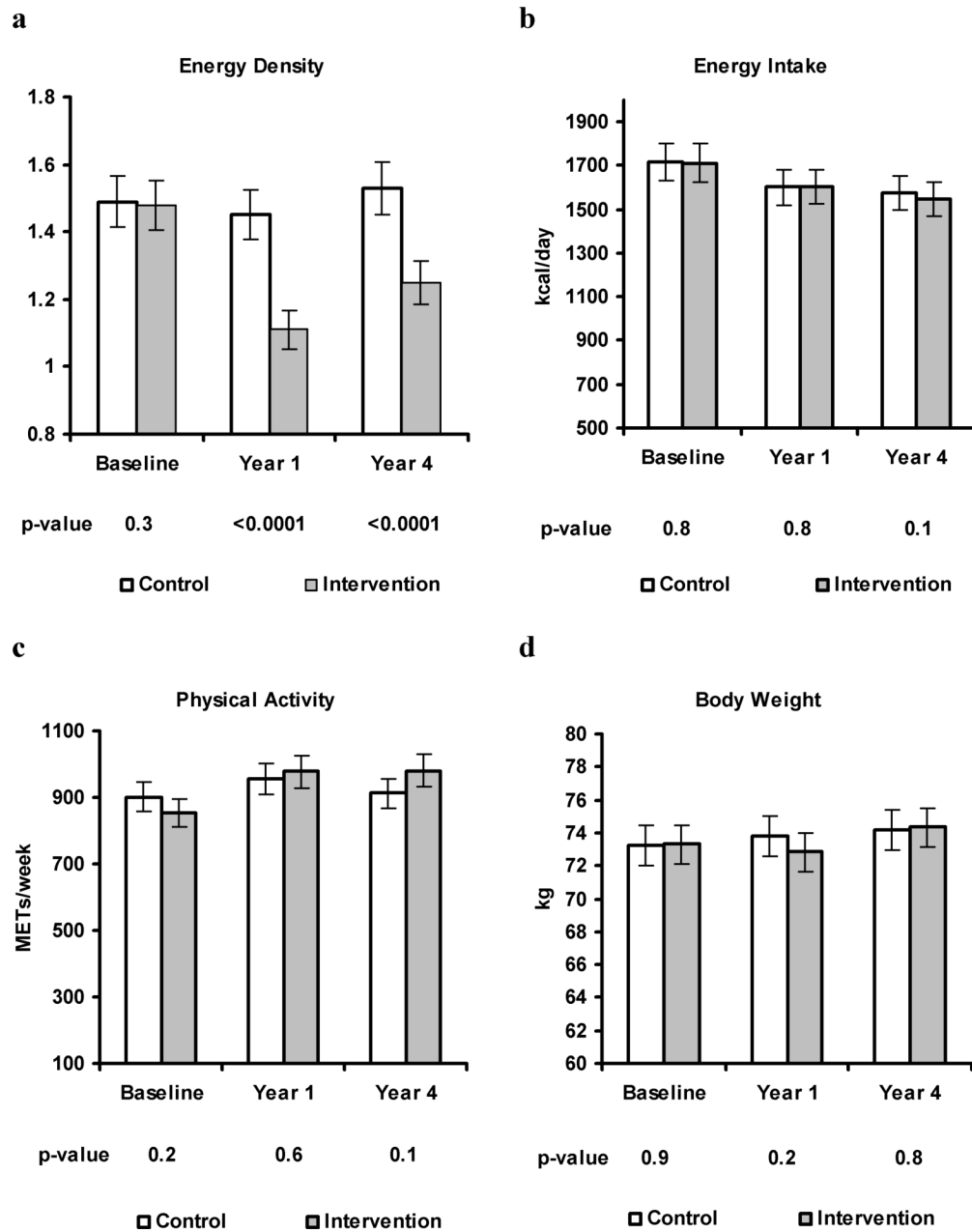


FIG. 1. Mean energy density (food only), energy intake, physical activity, and body weight in the control and in the intervention group over the study period: The Women's Healthy Eating and Living (WHEL) Study. 1 kcal = 4.18 kJ. MET, metabolic equivalent task.

TABLE 1Baseline characteristics of the control and intervention group^a

Characteristic	Control (%)(n = 1,363)	Intervention (%)(n = 1,355)	P Value
Age at study entry (yr)			
20–14	16.9	15.3	0.56
45–54	41.5	43.0	
55–64	30.2	29.5	
≥ 65	11.4	12.2	
BMI (kg/m ²)			
<25	42.7	42.3	0.74
25–29.9	32.1	31.3	
≥ 30	25.2	26.4	
Race/ethnicity			
Non-Hispanic White	85.4	85.5	0.93
African American	3.7	3.7	
Asian American	3.5	3.0	
Hispanic	5.1	5.6	
Others	2.3	2.1	
College graduate	53.9	55.3	0.47
Married	70.6	70.3	0.83
Employed	72.8	71.9	0.61
Current smoker	4.8	4.1	0.16
Stage III cancer	4.2	3.9	0.83
Energy intake (kcal/day) ^b	1,718 ± 11.2	1,714 ± 10.9	0.76
Physical activity (MET-min/wk) ^b	901 ± 24.3	854 ± 24.3	0.18

^a Abbreviations are as follows: BMI, body mass index; MET, metabolic equivalent tasks; min, minute; SE, standard error of the mean. Mean and standard error for continuous variables and frequency for categorical variables are presented.

^b Mean ± SE.

TABLE 2

Dietary energy density (food only) by baseline demographic characteristics: The Women's Healthy Eating and Living (WHEL) Study^a

Variable	<i>n</i>	Baseline dietary energy density (Food only) Mean ± SEM	<i>P</i> Trend
Age (yr)			
≤44 (ref) ^b	437	1.57 ± 0.02	<0.0001
45–54	1,148	1.51 ± 0.02*	
55–64	810	1.44 ± 0.02*	
≥65	318	1.41 ± 0.02*	
BMI (kg/m ²)			
<25 (ref) ^b	1,151	1.41 ± 0.01	<0.0001
25–29.9	862	1.52 ± 0.01*	
≥30	700	1.57 ± 0.01*	
Race/ethnicity			
Non-Hispanic White (ref) ^b	2,318	1.48 ± 0.01	Not applicable
African American	102	1.65 ± 0.04*	
Asian American	88	1.32 ± 0.04*	
Hispanic	145	1.55 ± 0.03	
Others	60	1.45 ± 0.05	

^a *n* = 2,713 (intervention and control group combined). 24-h dietary recalls were used to obtain dietary information via telephone interview. Abbreviations are as follows: SEM, standard error of the mean; BMI, body mass index; ref, reference.

* indicates significant differences.

^b One-way analysis of variance examined group differences.

TABLE 3Energy intake, physical activity, and body weight by tertile of baseline dietary energy density (food only)^a

Variable	Baseline dietary energy density (food only)		
	Bottom tertile (<1.29 kcal/g)	Middle tertile (1.29–1.60 kcal/g)	Top tertile (≥1.61 kcal/g)
Total energy intake (kcal/day) ^b	1,571 ± 12.9	1,698 ± 12.9*	1,874 ± 12.6†
Physical activity (METs/week) ^{b,c}	1,101 ± 29.6	903 ± 29.4*	637 ± 22.9†
Body weight (kg) ^b	70.1 ± 0.56	72.8 ± 0.55*	76.9 ± 0.55†

^a *n* = 2,713 (intervention and control group combined). Reference: bottom tertile; values with different symbols (*, †) are significantly different (*P* < 0.05). 1 kcal = 4.18 kJ. Abbreviation is as follows: METs, metabolic equivalent tasks.

^b Mean ± standard error of the mean.

^c Sum of METs assigned as 2 METs/min of casual strolling, 3 METs/min of mild activity or average walking, 4 METs/min of fast walking, 5 METs/min of moderate activity, 6 METs/min of very fast walking, 8 METs/min of strenuous activity.

TABLE 4

Changes in energy density, total energy intake, physical activity, and body weight over the study follow-up period: The Women's Healthy Eating and Living (WHEL) Study^a

Factor	Group	Baseline (Mean ± SEM)	Change	
			Yr 1 - Baseline (Mean ± SEM)	Yr 4 - Baseline (Mean ± SEM)
Energy density (food only)	Control	1.49 ± 0.01	-0.03 ± 0.01	0.05 ± 0.01
	Intervention	1.48 ± 0.01	-0.35 ± 0.01**	-0.22 ± 0.01**
Total plasma carotenoids (µmol/l)	Control	2.47 ± 0.04	-0.07 ± 0.03	-0.10 ± 0.04
	Intervention	2.40 ± 0.03	1.59 ± 0.05**	0.94 ± 0.06**
Energy intake (kcal ^b /day)	Control	1,718 ± 11.2	-121 ± 10.7	-152 ± 12.2
	Intervention	1,713 ± 10.9	-115 ± 11.5	-172 ± 16.6
Physical activity (METs/week) ^c	Control	901 ± 24.6	51.2 ± 21.5	24.6 ± 24.5
	Intervention	854 ± 24.3	78.2 ± 21.2	72.2 ± 26.8*
Body weight (kg)	Control	73.3 ± 0.5	0.71 ± 0.11	1.43 ± 0.20
	Intervention	73.3 ± 0.5	-0.05 ± 0.12**	1.77 ± 0.23

^aMixed effect models were used to examine difference of change between groups from baseline.

* $P < 0.05$

** $P < 0.0001$: computed for testing Group × Time interaction for each variable. Abbreviations are as follows: SEM, standard error of the mean; METs, metabolic equivalent tasks.

^b 1 kcal = 4.18 kJ.

^c Sum of METs assigned as 2 METs/min of casual strolling, 3 METs/min of mild activity or average walking, 4 METs/min of fast walking, 5 METs/min of moderate activity, 6 METs/min of very fast walking, 8 METs/min of strenuous activity.