Computerized tomography assessment of women with weight changes associated with adjuvant treatment for breast cancer

Carrie L Cheney, Janice Mahloch, and Patrick Freeny

ABSTRACT It is common for women undergoing treatment for breast cancer to gain weight, although the characteristics of the weight change have not been described. We investigated the changes in abdominal fat accumulation that accompanied the change in weight associated with treatment for breast cancer in longitudinal and cross-sectional clinical studies in 34 women aged 39-73 y with early-stage primary breast cancer. Computerized tomography scans of abdominal subcutaneous and visceral adipose depots, bioelectrical impedance measurements of body fat mass, and measurements of body weight and girth were obtained early in the course of treatment and 6 mo later (longitudinal study; n = 8) or within 12 mo of treatment (cross-sectional study; n = 26). The longitudinal study found that, irrespective of the direction of weight change, seven of eight women gained body fat and lost lean body mass. In the five women who gained weight (median: 3.2 kg) two lost and three gained subcutaneous adipose fat (median: 19%) whereas all gained visceral fat (median: 23%). In the cross-sectional study 19 women gained weight and 7 lost weight or had stable weight since diagnosis. Change in weight was correlated with abdominal subcutaneous adipose fat (r = 0.39; P = 0.06) and hip circumference (r = 0.43; P = 0.03) but not abdominal visceral fat, the ratio of subcutaneous to visceral fat, or the ratio of waist to hip size. In the longitudinal sample, weight gain resulted in a variable response in subcutaneous adipose volumes but a consistent increase in visceral adipose depot. Although these results are preliminary, it appeared that regardless of weight gain or loss women were likely to lose lean body mass and gain fat mass during treatment for breast cancer. Am J Clin Nutr 1997;66:141-6.

INTRODUCTION Weight gain is common in women receiving adjuvant chemotherapy for early-stage breast cancer. One review noted that weight gain during treatment was observed in from 30% to 90% of samples described in the literature (1). Weight gains of 2.5-6.2 kg were most common although gains > 10 kg were not unusual. Weight gain was also observed in women with breast cancer who did not receive adjuvant chemotherapy although the amount gained was less (< 2 kg) (2, 3).

The gain in weight associated with breast cancer treatment is an important clinical problem that may affect the course of disease. High body weight, obesity, or other measures of adipose accumulation at the time of diagnosis are risk factors for recurrence of disease and are related to poor prognostic factors, such as larger tumor size and greater numbers of involved axillary nodes (4-12). Evidence suggests that weight gain during adjuvant chemotherapy is also associated with a poorer prognosis. Camoriano et al (13) found that women who gained more than the median weight gain (5.9 kg) had a 1.5-fold increase in the risk of recurrence and a 1.6-fold increase in the risk of death (P = 0.04). It is not known whether these findings reflected a causal relation and whether preventing weight gain will result in a favorable effect on prognosis.

The anthropometric characteristics of weight gain in patients being treated for breast cancer are not well studied. A single study reported a 2.2% gain in body fat and a 1.3-kg loss in lean body mass assessed by multiple skinfold measurements in 12 patients with breast cancer after 10 wk of chemotherapy. The patients gained an average of 2 kg in body weight during that period (14).

The metabolic consequences of the weight gain are also not known. As a surrogate for alterations in lipid, carbohydrate, and hormone metabolism, information about fat patterning would be helpful but is also lacking. For example, it is not known whether distribution of body fat is altered, even though distribution of body fat may be important in breast cancer etiology (15-19) and prognosis (20). To our knowledge, circumference measurements in patients with breast cancer undergoing treatment have not been reported. The only study to report changes in subcutaneous fat distribution assessed by skinfold-thickness measurements during 10 wk of treatment showed that, in conjunction with an average weight gain of 2 kg, the greatest increase in subcutaneous fat occurred in the supraclavicular sites whereas smaller increases were observed in triceps and anterior thigh sites (14).

This suggests that preferential accumulation of fat in abdominal sites may occur. Because hormonal concentrations, espe-

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cially serum androgen, estrogen, and cortisol concentrations, appear to influence adipose tissue distribution (21), differential deposition of adipose tissue in abdominal sites may reflect an altered hormonal profile and thus possibly affect prognosis. Alternatively, the increased metabolic activity of adipose tissue in abdominal depots could enhance release of fatty acids or plasma insulin, which in turn may affect bioavailability of plasma sex steroids (18, 22, 23).

A study of alterations in fat distribution could provide insights about the mechanisms by which weight gain influences prognosis in patients with breast cancer. The purpose of this study was to describe the changes in fat distribution that accompany the change in weight associated with breast cancer treatment. We used computerized tomography (CT) to assess changes in abdominal subcutaneous and visceral adipose depots. In a subset of women who provided only one measurement, we sought to relate CT-derived measures of adipose volume to change in weight.

SUBJECTS AND METHODS

This study was approved by the Human Subjects Review Office of the University of Washington. Women with a diagnosis of early-stage (I-IIIA) ductal or lobular breast cancer were recruited when they responded to information provided by their oncologist or the breast cancer support group they attended. The original intent of the study was to obtain measurements at or near the beginning of therapy and again after adjuvant treatment but because of difficulty in recruiting a sufficient number of women, it was necessary to also include women who had recently completed therapy. This created a two-group study.

Women were included if they were within 6 mo of diagnosis of breast cancer and currently undergoing adjuvant treatment of the disease (longitudinal group) or were within 1 y of completing definitive surgical or adjuvant treatment (cross-sectional group). Premenopausal and postmenopausal women of all ages were included. Women with diabetes and pregnant women were excluded as were women with more extensive breast cancer (stage IIIB-IV). Two serial measurements were obtained in women in the longitudinal group, one at entry into the study and one ~6 mo later. A single measurement was obtained in women in the cross-sectional group.

Demographic and clinical characteristics were obtained by interviews and reviews of medical records. If weight at diagnosis was not given in the medical record, the women were asked to recall it. All anthropometric and body-composition measurements were obtained by the study’s registered nurse according to standard methods (24). Body weight was measured on a balance scale calibrated to 0.2 kg, with subjects dressed in light clothing and without shoes. Height was measured to the nearest 0.1 cm, with subjects standing and barefoot. Body mass index (BMI) was computed (in kg/m²). Circumferences were measured with a plastic tape while subjects were standing; values for the waist at the level of the umbilicus and the hips at the greatest circumference were recorded. Body composition was measured by means of bioimpedance analysis done according to the manufacturer’s instructions (Bioelectric Impedance Analyzer 101-A; RJL Systems, Inc, Clinton Township, MI). To minimize alterations in fluid status, women were asked to refrain from exercising or consuming food or caffeine for ≥ 4 h before arrival at the clinic. Measurements from the bioelectrical impedance analysis were used to estimate lean body mass and (assuming a two-compartment model) fat mass.

CT scans were obtained with a General Electric HiSpeed Advantage scanner (General Electric Medical Systems, Milwaukee) in the axial plane by using 140 kV, 170 mA, a 2-s scanning speed, and 10-mm collimation. Field of view was selected according to measured patient size. No oral or intravenous contrast agents were administered. A digitized scout-view was obtained for slice localization and three axial scans were then done at the levels of T-12, the iliac crest, and the midpelvis. CT computer-image analysis software was used to calculate the volume of extraabdominal (subcutaneous) fat and the volume of intraperitoneal fat. Regions of interest were first traced with cursor and trackball; these regions anatomically defined the subcutaneous and intraperitoneal fat compartments. Areas in the regions of interest (mm²) were calculated by the computer. By using a predetermined range of fatty tissue attenuation (pixel range: from −50 to −250 Hounsfield units), we included only the fat-containing tissues in the area measurements, thus obtaining a specific measurement of the area of fat in each compartment (25).

Statistical analysis was conducted by the Statistical Section of the Clinical Nutrition Research Unit at the University of Washington. In the cross-sectional group, weight at diagnosis, selected clinical characteristics, and CT scan measurements in women who gained weight and women who lost weight were compared by using the Mann-Whitney U test and chi-square or Fisher’s exact tests as appropriate; all tests of significance were two sided. The association between weight change from diagnosis and CT scan measurements was evaluated by the Spearman nonparametric correlation coefficient. Only descriptive data are presented for the longitudinal group; no inferential statistical tests were done because of the small sample size. Because the distributions of some variables were skewed, median as well as mean ± SD values are provided for consistency.

RESULTS

Thirty-four women participated in the study, 8 in the longitudinal group and 26 in the cross-sectional group. Overall, 24 women (71%) had gained weight since diagnosis (computed as the difference between weight measured at the first and second visits in the longitudinal group). In the longitudinal group three women lost a median of 1.1 kg (range: 0.5–3.0 kg) and five women gained a median of 3.3 kg (range: 0.4–6.5 kg). In the cross-sectional group 7 women lost a median of 0.2 kg (range: 0.0–4.6 kg) whereas 19 women gained a median of 3.2 kg (range: 0.1–18.7 kg) and 6 (31%) of these women gained ≥ 5 kg.

Characteristics of the subjects stratified by weight change (loss or gain) and study group (longitudinal or cross-sectional) are shown in Table 1. Overall, the women ranged in age from 39 to 73 y and age distribution was similar in the two weight-change groups. Most of the premenopausal women gained weight [13 of 15 (86%)] compared with 11 of 19 (58%) of the postmenopausal women (P = 0.14). Eighteen of 23 women (78%) who received systemic chemotherapy (excluding tamoxifen) gained weight compared with 6 of 11 women (55%) who
## TABLE 1
Characteristics of subjects in the study groups

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Longitudinal group</th>
<th>Cross-sectional group</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Weight-loss group</td>
<td>Weight-gain group</td>
</tr>
<tr>
<td></td>
<td>(n = 3)</td>
<td>(n = 5)</td>
</tr>
<tr>
<td>Age (y)</td>
<td>54 (49, 56)</td>
<td>61 (46, 66)</td>
</tr>
<tr>
<td>Postmenopausal (n)</td>
<td>3</td>
<td>3</td>
</tr>
<tr>
<td>Adjuvant therapy (n)</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Tumor size (cm)</td>
<td>2.0 (1.4, 3.6)</td>
<td>2.5 (0.8, 3.5)</td>
</tr>
<tr>
<td>Receptor status (n)</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Estrogen positive</td>
<td>3</td>
<td>6</td>
</tr>
<tr>
<td>Estrogen negative</td>
<td>0</td>
<td>0</td>
</tr>
<tr>
<td>Progesterone positive</td>
<td>2</td>
<td>4</td>
</tr>
<tr>
<td>Progesterone negative</td>
<td>0</td>
<td>4</td>
</tr>
<tr>
<td>Positive nodes</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>≥4</td>
<td>2</td>
<td>2</td>
</tr>
</tbody>
</table>

| Characteristic                              | Weight-loss group  | Weight-gain group     |
|                                             | (n = 7)            | (n = 19)              |
|                                             |                    |                       |

1 Median; minimum and maximum in parentheses.
2 CMF: cyclophosphamide, methotrexate, and fluorouracil; CAF/AC, cyclophosphamide, doxorubicin, fluorouracil/doxorubicin, and cyclophosphamide.

received either tamoxifen or radiation alone \( P > 0.20 \). Tumor size was slightly although not significantly larger in women who gained weight than in women who lost weight. Weight change was not associated with estrogen- or progesterone-receptor status or number of positive nodes.

The baseline and change in anthropometric and body-composition measurements in the longitudinal group are shown in Table 2. Women who gained weight were 4 kg heavier and had greater fat mass and less lean body mass at baseline than women who lost weight. Irrespective of direction of weight change, seven of eight women gained body fat and lost lean body mass. The CT results in women who lost weight are incomplete because one woman chose not to have a second scan and it was not possible to evaluate another woman’s measure because of poor respiratory effort. Thus, CT results for only one woman in this group are shown.

In the five women who gained weight, two lost and three gained subcutaneous adipose fat (change as a percentage of baseline; median: 19%; range: \(-3\%\)–\(-42\%\)) whereas all five women gained visceral adipose fat (median: 23%; range: \(14\%\)–\(186\%\)). The woman whose visceral adipose increased \(186\%\) (the largest increase in the other four women was \(39\%\)) was the only one in this series who received prednisone as part of her adjuvant chemotherapy. Age, nodal status, and tumor size were similar in the five women. In the woman who lost weight and had both CT scans, a larger proportion of adipose loss occurred in the abdominal visceral depot (\(-15\%)\) than in the subcutaneous depot (\(-2\%)\).

## TABLE 2
Baseline and change in anthropometric measurements in the longitudinal group1

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Baseline Weight-loss group (n = 3)</th>
<th>Baseline Weight-gain group (n = 5)</th>
<th>Change Weight-loss group (n = 3)</th>
<th>Change Weight-gain group (n = 5)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight (kg)</td>
<td>69.5 (60.0, 72.7)</td>
<td>73.5 (61.8, 83.5)</td>
<td>(-1.1 (-0.5, -3.0))</td>
<td>3.3 (0.4, 6.5)</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>165 (163, 171)</td>
<td>167 (161, 177)</td>
<td>(-0.4 (-0.2, -1.0))</td>
<td>(-1.7 (0.1, 2.3))</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>23.8 (22.0, 27.4)</td>
<td>26.1 (22.2, 32.3)</td>
<td>(-0.4 (-0.2, -1.0))</td>
<td>(-1.0 (1.0, 8.0))</td>
</tr>
<tr>
<td>Body fat mass (kg)</td>
<td>23.6 (17.9, 28.1)</td>
<td>38.1 (33.1, 46.6)</td>
<td>(-0.1 (-0.8, 0.2))</td>
<td>(-0.4 (0.7, 7.9))</td>
</tr>
<tr>
<td>Body fat mass (%)</td>
<td>34 (30, 39)</td>
<td>47 (34, 55)</td>
<td>(-0.0 (-1.0, 1.0))</td>
<td>(-0.4 (1.0, 4.0))</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>44.6 (42.0, 46.1)</td>
<td>37.6 (29.2, 49.4)</td>
<td>(-0.7 (0.8, -2.9))</td>
<td>(-1.3 (-0.2, -3.5))</td>
</tr>
<tr>
<td>Subcutaneous abdominal fat (cm²)¹</td>
<td>112.5</td>
<td>110.9 (76.2, 156.9)</td>
<td>1.1</td>
<td>19.5 (7.6, 32.0)</td>
</tr>
<tr>
<td>Visceral abdominal fat (cm²) ²</td>
<td>147.9</td>
<td>76.3 (15.0, 125.4)</td>
<td>(-7.4)</td>
<td>14.9 (10.7, 28.8)</td>
</tr>
</tbody>
</table>

1 Median; minimum and maximum in parentheses.
2 n = 1.
The anthropometric measurements in the cross-sectional group are shown in Table 3. Body weight at diagnosis was similar in women who lost weight and women who gained weight although BMI was slightly greater in women who gained weight. Body fat and lean masses were ~5 kg and 4 kg heavier, respectively, in women who gained weight than in women who lost weight although these differences were not significant. Fat and lean masses as a percentage of body weight were similar in the groups. The median abdominal subcutaneous adipose fat was nearly 16 cm$^2$ larger and the median visceral adipose fat was nearly 24 cm$^2$ larger in women who gained weight than in women who lost weight. Accordingly, the ratio of subcutaneous to visceral adipose fat was lower in women who gained weight than in women who lost weight (2.09 compared with 2.28). None of these differences were significant.

The change in weight since diagnosis was positively related to abdominal subcutaneous adipose fat ($r = 0.39$, $P = 0.06$) and hip circumference ($r = 0.43$, $P = 0.03$) but not to abdominal visceral fat ($r = 0.13$, $P = 0.54$), ratio of subcutaneous to visceral fat ($r = 0.04$, $P = 0.85$), or ratio of waist to hip size ($r = -0.23$, $P = 0.26$). Interestingly, body fat mass was strongly positively correlated with abdominal visceral fat ($r = 0.82$, $P < 0.001$) and abdominal subcutaneous fat ($r = 0.60$, $P = 0.002$) and negatively correlated with the ratio of subcutaneous to visceral abdominal fat ($r = -0.55$, $P = 0.004$).

**DISCUSSION**

Our intention was to characterize the changes in abdominal adipose depots resulting from weight changes associated with medical treatment for breast cancer in the hope of gaining clues about metabolic factors that may help explain the reported association between weight gain and poorer survival. To our knowledge, no other reports of assessments of visceral adipose deposition during active weight gain in nonobese women exist although there are many such reports in subjects on weight-loss regimens. Unfortunately, interpretation of the longitudinal study results is limited by the small sample size and substantial individual variability. However, there are consistent findings worthy of note.

The amount of weight gain in the subjects in our study was modest but comparable to that reported by other investigators (2, 26–28), and the prevalence of weight gain (71%) was in line with that reported in the literature (1). In our study, weight gain resulted in increases in both abdominal subcutaneous and visceral adipose volumes although a slightly larger increase oc-

<table>
<thead>
<tr>
<th>Measurement</th>
<th>Weight-loss group $(n = 7)^2$</th>
<th>Weight-gain group $(n = 19)^3$</th>
</tr>
</thead>
<tbody>
<tr>
<td>Body weight at diagnosis (kg)</td>
<td>65.8 (58.1, 81.7)</td>
<td>65.9 (54.0, 95.3)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>67.5 ± 8.9</td>
<td>66.8 ± 11.9</td>
</tr>
<tr>
<td>Weight change from diagnosis (kg)</td>
<td>$-0.2 \pm (-4.6, 0.0)$</td>
<td>$-1.2 \pm 1.7$</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>167 (146, 169)</td>
<td>165 (152, 172)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>163.4 ± 8.0</td>
<td>163.4 ± 5.6</td>
</tr>
<tr>
<td>BMI (kg/m$^2$)</td>
<td>24.2 (21.3, 30.2)</td>
<td>25.3 (19.8, 37.4)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>24.9 ± 3.3</td>
<td>27.0 ± 5.0</td>
</tr>
<tr>
<td>Body fat mass (kg)</td>
<td>23.8 (17.3, 40.8)</td>
<td>28.5 (10.1, 52.1)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>28.1 ± 9.5</td>
<td>31.1 ± 12.2</td>
</tr>
<tr>
<td>(%)</td>
<td>38 (28, 56)</td>
<td>41 (18, 70)</td>
</tr>
<tr>
<td>Lean body mass (kg)</td>
<td>41 ± 10</td>
<td>42 ± 12</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>38.9 (31.9, 43.7)</td>
<td>43.1 (22.0, 53.8)</td>
</tr>
<tr>
<td>(%)</td>
<td>38.3 ± 4.4</td>
<td>41.1 ± 8.8</td>
</tr>
<tr>
<td>Waist circumference (cm)</td>
<td>81 (67, 104)</td>
<td>83 (69, 108)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>82.6 ± 12.1</td>
<td>84.6 ± 11.3</td>
</tr>
<tr>
<td>Hip circumference (cm)</td>
<td>104 (98, 114)</td>
<td>107 (92, 131)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>104.6 ± 5.5</td>
<td>109.5 ± 10.7</td>
</tr>
<tr>
<td>Waist-hip ratio</td>
<td>0.78 (0.67, 0.91)</td>
<td>0.76 (0.68, 0.87)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>0.79 ± 0.08</td>
<td>0.77 ± 0.06</td>
</tr>
<tr>
<td>Subcutaneous abdominal fat (cm$^2$)</td>
<td>111.1 (71.7, 186.4)</td>
<td>126.9 (74.6, 276.9)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>115.4 ± 42.2</td>
<td>145.9 ± 68.4</td>
</tr>
<tr>
<td>Visceral abdominal fat (cm$^2$)</td>
<td>47.7 (15.5, 294.9)</td>
<td>71.6 (20.2, 281.6)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>98.9 ± 109.1</td>
<td>81.0 ± 64.9</td>
</tr>
<tr>
<td>Subcutaneous-visceral fat ratio</td>
<td>2.28 (0.63, 5.00)</td>
<td>2.09 (0.58, 13.68)</td>
</tr>
<tr>
<td>$\bar{x} \pm SD$</td>
<td>2.34 ± 1.63</td>
<td>2.71 ± 2.86</td>
</tr>
</tbody>
</table>

$^1$ Median; minimum and maximum in parentheses.

$^2$ Except for subcutaneous and visceral abdominal fat and subcutaneous-visceral fat ratio, where $n = 6$ in the weight-loss group and $n = 18$ in the weight-gain group.
curred in the visceral adipose depot. The increase in visceral adipose volume was a consistent finding. Individually, all women who gained weight accumulated visceral fat but not all women gained subcutaneous fat. Moreover, regardless of whether there was weight gain or loss, women were likely to lose lean body mass and gain fat mass during treatment. This finding as well as the magnitude of the loss in lean body mass (1.3 kg) is similar to that reported by Winningham et al (14) in their study of women with breast cancer.

Other prospective studies of weight gain have likewise found that fat mass increased with increases in body weight but, contrary to our findings, fat-free mass also increased (29, 30). The difference in our findings was likely due to the fact that the subjects in the other studies were younger healthy obese individuals whereas our sample was composed largely of middle-aged and older nonobese women receiving cytotoxic treatment for malignant disease, which could account for the loss of lean body mass. The greater loss of lean body mass in women who gained weight compared with women who lost weight may have been due to the older age of women who gained weight (61 compared with 54 y) and differences in treatment because one of the women who gained weight received prednisone.

The characteristics of weight gain in women and its consequences in fat distribution have received only limited study. Increased fat mass and decreased lean mass and a tendency to accumulate fat centrally were related to menopause (31) and were similar to those we observed. The effect of hormones on these changes in patients undergoing chemotherapy is unclear. Recently, Reubinoff et al (32) studied body-composition changes in women soon after menopause for 1 y. In association with an average weight gain of 2 kg, there was a 2% increase in body fat and a significant increase in waist-hip ratio (+ 0.05) in women not receiving hormone-replacement therapy. With a larger median weight gain, the women in our study also had an increased waist-hip ratio but the increase was less extensive than that reported by Reubinoff et al, possibly because of the age differences in the women in our study.

Our observations that weight changes correlated with abdominal subcutaneous fat and not with visceral fat accord with reports of related findings. Enzi et al (33) reported that as overweight increased, women < 60 y of age tend to store fat preferentially in abdominal subcutaneous sites. Kvist et al (34) found that the relative amount of adipose tissue in visceral depots was not related to degree of obesity in women. Ferland et al (35) assessed total and visceral adipose tissue in obese women and found no association between these variables. These investigators speculated that the subcutaneous and visceral fat compartments may expand independently from each other. More recently, Sjostrom (36) also observed no correlation between relative amount of visceral adipose tissue and amount of total body fat in women. However, in our cross-sectional sample we found that although weight changes were not associated with visceral adipose volume, total body fat was strongly correlated with both visceral and subcutaneous abdominal adipose tissue.

The question of whether weight gain results in preferential deposition of adipose tissue in visceral depots is important. Our observations in the longitudinal sample were unfortunately limited by small sample size but were consistent with a tendency to accumulate a slightly larger amount of fat in visceral sites. This idea is supported by the fact that all women gained visceral fat but not all gained subcutaneous fat. Few other studies have reported on abdominal adipose changes in the context of weight gain; most investigations observed changes during weight-loss regimens or weight cycling. In obese women studied after they regained weight after weight-loss intervention, Wadden et al (37) found that waist-hip ratios returned to preintervention values rather than showing alterations in fat distribution. Similarly, in their study of weight cycling, including weight regain after initial loss, van der Kooy et al (38) found no indication of a preferential deposition of visceral fat with weight regain but reported a slight tendency to accumulate excess subcutaneous fat rather than visceral fat.

Differences in our investigations may explain the discrepant findings. The women in the other studies were all obese (mean BMI: > 31) and premenopausal whereas women in our longitudinal sample who gained weight were not all obese (mean BMI: 27) and were aged 42–66 y. In addition, the women in our sample were undergoing adjuvant treatment for breast cancer and it is possible that such treatment (or the underlying disease) resulted in metabolic changes that affected adipose deposition. Additional study is needed to confirm and expand on our observations.

A secondary aim of our study was to identify factors associated with a tendency to gain weight during treatment for breast cancer. Although we cannot rule out the possibility that chance accounted for our findings, the group of women who gained weight tended to have a larger body fat mass and a larger visceral fat mass (relative to the subcutaneous mass) than their counterparts who lost weight or had stable body weight. It is unlikely that age accounted for these differences because the distribution of ages in weight losers and weight gainers was similar. Whether these differences represented factors associated with a tendency to gain weight or factors resulting from weight gain is unknown.

In the longitudinal group, however, women who gained weight also had a larger fat mass and a higher BMI at baseline than women who lost weight. This may have reflected a relation between larger body size and more advanced breast cancer and thus more aggressive treatment that resulted in larger weight gains. However, tumor size, nodal status, and medical treatment in women who lost weight and women who gained weight did not differ substantially in our sample. It may be that the presence of a larger fat mass and possibly a larger visceral fat volume may predispose a woman to excess weight gain during treatment for breast cancer. Further study is needed to clarify whether there are characteristics, such as a tendency to accumulate visceral fat, that help to identify women at greater risk for weight gain so that prevention strategies may be more successful.

We thank the women who generously gave of themselves to participate in this study, Joanne Maurice for assistance with data collection, and Carolyn Walden and Brian Fish of the Clinical Nutrition Research Unit Statistical Section for assistance with data management and analysis.

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