The Breast 69 (2023) 491–498

Contents lists available at ScienceDirect

The Breast

journal homepage: www.journals.elsevier.com/the-breast

Factors associated with weight gain after breast cancer: Results from a community-based survey of Australian women

Carolyn Ee [a,b,c,d,*], Adele Cave [a], Vaishnavi Vaddiparthi [a], Dhevaksha Naidoo [a], John Boyages [e,f]

[a] NICM Health Research Institute, Western Sydney University, Locked Bag 1797, Penrith, Sydney, New South Wales, Australia
[b] Translational Health Research Institute, Western Sydney University, Locked Bag 1797, Penrith, Sydney, New South Wales, Australia
[c] LivingRoom, Chris O' Brien Lifehouse Cancer Centre, PO Box M33, Missenden Rd, Camperdown, Sydney, New South Wales, Australia
[d] Caring Futures Institute, Flinders University, Bedford Park, Adelaide, South Australia, Australia
[e] ICON Cancer Centre, Sydney Adventist Hospital, 185 Fox Valley Road, Wahroonga, Sydney, New South Wales, Australia
[f] Faculty of Health and ANU College of Health & Medicine, Canberra, Australian Capital Territory, Australia

ARTICLE INFO

Keywords:
Breast cancer
Weight gain
Menopause
Tamoxifen
Physical activity
Diet

ABSTRACT

Purpose: Weight gain after breast cancer is common. The aim of this study was to determine factors associated with weight gain after breast cancer in Australian women.
Methods: A cross-sectional online survey was conducted between November 2017 and January 2018. Women living in Australia who self-identified as having breast cancer or ductal carcinoma in-situ were eligible. We created stepwise linear and logistic regression models to evaluate predictors for absolute and clinically significant (≥5%) weight gain respectively.
Results: Data from 276 women were analysed. Most were Caucasian and 92% had been diagnosed with Stage 0-III breast cancer. Absolute weight gain was associated with hot flushes, being in the menopausal transition at diagnosis, being less physically active than at diagnosis, lower eating self-efficacy when watching television or using a computer, and higher self-efficacy when anxious or nervous (F-ratio = 3.26, R^2-adjusted = 0.16, $p <$ .001). Clinically significant weight gain was associated with tamoxifen use (OR 2.7), being less physically active than at diagnosis (OR 3.1), and lower eating self-efficacy when watching television or using a computer (OR 0.82) (Chi-square 64.94, $df = 16$, $p <$ .001). Weight gain was not associated with chemotherapy, radiotherapy, aromatase inhibitor use, number of lymph nodes removed, or body mass index at diagnosis.
Conclusions: Interventions to prevent weight gain after breast cancer, particularly aiming to maintain physical activity, should be targeted at women receiving tamoxifen. The role of eating self-efficacy, especially attentive eating, in managing weight after breast cancer should be explored.

1. Background

Weight gain after breast cancer is common and can have a substantial negative impact on quality of life [1]. We previously demonstrated that two thirds of respondents to our national survey in Australia reported gaining weight after being diagnosed with breast cancer [2,3] at an average of 9.09 kg and in excess of what might be expected in age-matched controls [2]. Weight gain after breast cancer has been associated with an increase in disease recurrence and all-cause mortality [1]. Given the growing population of breast cancer survivors and the link between weight gain and adverse health outcomes, research into the factors associated with weight gain after breast cancer is of critical importance.

A prospective study [3] of 287 Australian women diagnosed with early-stage breast cancer reported that more extensive lymph node removal, being treated on the non-dominant side, receiving radiation therapy and lower physical activity (PA) levels at 6 months was associated with higher weight after breast cancer. Another retrospective clinical audit reported that women with grade II or III breast cancer were more likely to gain weight than women with grade I breast cancer [4]. However, these studies were limited to single cities or regions in cities in Australia. The aim of this study was to determine, from our national sample, the predictors of weight gain after breast cancer in Australian women.

* Corresponding author. NICM Health Research Institute, Western Sydney University, Locked Bag 1797, Penrith, Sydney, New South Wales, Australia.
E-mail address: c.ee@westernsydney.edu.au (C. Ee).

https://doi.org/10.1016/j.breast.2023.01.012
Received 10 December 2022; Received in revised form 23 January 2023; Accepted 24 January 2023
Available online 25 January 2023

2. Methods

2.1. Study design and inclusion criteria

Our methods have been described previously [2]. Briefly, this online cross-sectional survey was completed by women residing in Australia who self-identified as having breast cancer or ductal carcinoma in-situ. The sample included members of the Breast Cancer Network Australia (BCNA) Review and Survey Group, representing 1857 BCNA members who have agreed to receive emails about participating in research studies. BCNA is the largest breast cancer consumer advocacy organization in Australia. The survey was emailed to the 1857 BCNA members on December 5th, 2017, and a reminder email was sent to 1835 BCNA members on January 15th, 2018. A smaller sample was drawn from online communities of women by posting the survey link to Australian women's health organization websites and social media pages, during the months of November and December 2017.

2.2. Survey instrument

Survey questions were developed after reviewing previous literature on weight after breast cancer and revised after feedback from six Consumer Representatives from BCNA. Ethics approval was provided by the Human Research Ethics Committee, Western Sydney University (H12444, Oct 2017).

Women were asked to self-report their weight at the time of diagnosis, and current weight and height. From this we calculated Body Mass Index (BMI) as weight/height [2] and absolute weight gain as current weight - weight at diagnosis. Weight gain percentage was calculated as absolute weight gain/weight at diagnosis × 100 and dichotomised to clinically significant weight gain (weight gain ≥5%) and weight maintenance/weight loss (weight gain <5%). A cut-off of 5% was chosen because this cut-off has been reported in studies aiming to improve health outcomes among people with overweight/obesity [5], and is frequently used in other studies on weight gain after breast cancer therefore facilitating comparisons between studies [6–9].

Menopausal status at the time of diagnosis was assessed as either "Premenopausal (regular periods with no menopausal symptoms such as hot flushes)", "Perimenopausal/in the menopausal transition (no periods for at least 2 months, plus hot flushes)", "Postmenopausal (no periods for at least 12 months)" or "Previous surgical menopausal (both ovaries or uterus/womb had been removed)." For the purposes of this analysis, we combined the latter two variables into a single "postmenopausal" variable.

Women were asked whether they met the recommended daily intake of five serves of vegetables and two serves of fruit, and whether their physical activity levels were less, similar, or more than at diagnosis. The validated Weight Self Efficacy Scale Short Forms (WEL-SF) [10] was used to evaluate how confident women now felt about being able to successfully resist the desire to overeat in eight different situations on an 11-point Likert scale from 0 (not confident at all) to 10 (very confident). Higher scores indicate higher weight self-efficacy. Physical activity (PA) levels were calculated according to the number of 20-min sessions of less vigorous exercise or more vigorous exercise a week, given a weighting and described in terms of MET (metabolic cost) minutes where MET minutes less than 80 were coded as no PA, 80 to 400 as low, 400 to 560 as moderate and more than 560 as high. A value of 4 METs was given to moderate PA and 7.5 to vigorous PA [11].

2.3. Statistical analysis

We tested correlations of patient and treatment factors for absolute and clinically significant weight gain (weight gain <5%, and weight gain ≥5%). The independent variables tested in our model were determined *a priori* based on clinical importance and are listed as follows.

2.4. Patient factors

- Age
- Presence of hot flushes
- Lifestyle: Fruit and vegetable intake, current physical activity levels in METs, self-reported change in physical activity levels since diagnosis
- Weight self-efficacy
- Menopausal status at diagnosis
- Presence and severity of lymphedema

2.5. Treatment factors

- Hormone therapy (received any hormonal therapy)
- Tamoxifen use
- Aromatase inhibitor use
- Number of lymph nodes removed
- Radiotherapy
- Chemotherapy
- Systemic treatment (received any of chemotherapy, hormonal therapy, and/or targeted therapy)

Statistical analyses were completed using SPSS Statistics for Windows version 26 [12]. Descriptive statistics were used to describe the cohort studied, including whether continuous variables formed normal distributions. Checks for normal distribution were performed using the Shapiro-Wilk test (p values $< .05$ indicated the distribution of the data set was significantly different from that expected for a normal distribution). Appropriate non-parametric tests were used to determine the association between patient and treatment factors and absolute and clinically significant weight gain and were deemed to be significant at $p < .05$. Variables that were statistically significant for an association with absolute or clinically significant weight gain were retained for use in the final linear and logistic regression models.

A linear regression model for absolute weight gain was formed in a stepwise manner with a threshold of retained variables set to $p < .05$. A logistic regression model was formed for clinically significant weight gain as a binary outcome (weight gain <5%, or weight gain ≥5%) with a threshold of retained variables set to $p < .05$.

The degree to which the predictors explained variation in the weight gain outcomes was evaluated by calculating the coefficient of determination (r^2). The regression model formed was evaluated for homoscedasticity by inspecting the plot of residuals versus predicted values, and testing whether residuals conformed to a normal distribution using the Shapiro-Wilk test.

3. Results

3.1. Survey response

A total of 309 women participated in the survey. Thirty-three were excluded due to non-reporting of current weight or weight at time of diagnosis. In total, the data set included 276 participants who self-reported weight at diagnosis and at the time of the survey.

3.2. Participant characteristics

Demographic and medical characteristics are described in Table 1. Most women were Caucasian (92.4%, $n = 255$) with a mean age of 58.8 years ($SD = 9.6$, range 33–78, $n = 267$) and had been diagnosed with localised (Stage I-III) breast cancer (83.4%, 230/276) with 10.1% (28/276) diagnosed with DCIS. The majority (66.3%, 183/276) of women reported they had been prescribed hormonal therapy, of which 61.7% (113/183) were still using hormonal therapy. Table 1 describes the hormonal therapies that women had ever been prescribed. Most women who indicated using tamoxifen in our study were premenopausal at

Table 1
Demographic and medical characteristics of respondents ($n = 276$).

Description	*n* (responses) or Mean	Percentage (%) or SD
Education		
High school- year 10	20	7.2
High school- year 12	32	11.6
TAFE or Vocational College	49	17.8
Bachelor's degree	83	30.1
Postgraduate degree	90	32.6
Missing	2	0.7
Ethnicity		
European/Anglo Saxon/Caucasian	255	92.4
Oceanic (incl. Australian and New Zealand first peoples, Polynesian and Micronesian)	9	3.3
Asian	5	1.8
North/South/Central American	2	0.7
Mixed ethnicity	2	0.7
Indian	2	0.7
Missing	1	0.4
Employment		
Employee	125	45.3
Retired	89	32.2
Self-employed	31	11.2
Home duties/caring for children or family	13	4.7
Doing voluntary work	7	2.5
Unable to work because of illness	6	2.2
In education (going to school, university, etc.)	4	1.5
Missing	1	0.4
Diagnoses		
Localised breast cancer	230	83.4
Ductal Carcinoma in Situ	28	10.1
Metastatic breast cancer	12	4.3
Inflammatory breast cancer	1	0.4
Other including second primary	3	1.1
Unclear/missing	2	0.7
Treatment to the Breast		
Lumpectomy alone	1	0.4
Lumpectomy and radiation	114	41.3
Mastectomy alone	76	27.5
Mastectomy and radiation	79	28.6
Double mastectomy	5	1.8
Missing	1	0.4
Treatment to the Axilla		
Sentinel node biopsy only	24	8.7
Axillary dissection ± Sentinel node biopsy	51	18.5
Axillary dissection ± Sentinel node biopsy + radiation	62	22.5
Radiation only	2	0.7
None/not reported	137	49.6
Intravenous Systemic Therapy		
Chemotherapy without Herceptin	145	52.6
Herceptin only	2	0.7
Chemotherapy + Herceptin	45	16.3
None/not reported	84	30.4
Hormonal Treatments		
Tamoxifen ± goserelin/oophorectomy	57	20.6
Aromatase inhibitors ± goserelin/ oophorectomy	72	26.1
Tamoxifen and aromatase inhibitors ± goserelin/oophorectomy	51	18.5
Goserelin only	3	1.1
None/not reported	93	33.7
Current use of hormone therapy		
Yes	113	40.9
No	69	25.0
No response	94	34.1
Menopausal stage at diagnosis		
Premenopausal	120	43.5
Menopausal transition	29	10.5
Postmenopausal - spontaneous	102	37.0
Postmenopausal - surgical	23	8.3
No response	2	0.7
Change in menopausal stage since diagnosis and treatment ($n = 131$)		
Pre-menopause to post-menopause	93	71.0
Menopausal transition to post-menopause	21	16.0
Still premenopausal	1	0.8
Still in menopausal transition	16	12.2
Physical activity level (METS)		
None (<80)	16	5.8
Low (80-<400)	100	36.2
Moderate (400-<560)	57	20.6
High (≥560)	102	37.0
Missing	1	0.4
Current physical levels compared to pre diagnosis		
I'm more active	66	23.9
I'm less active	108	39.1
I'm as active as I was	102	37.0
Age (mean, SD)		
Age at time of survey (years) ($n = 267$)	58.8	9.6
Range 33–78 years		
Age at time of diagnosis (years) ($n = 268$)	50.9	9.0
Range 29–74 years		
Weight (mean, SD)		
Weight at diagnosis (kg)	71.0	13.6
BMI at diagnosis ($n = 269$)	26.2	5.3
Weight at time of survey	75.6	15.3
BMI at time of survey ($n = 269$)	27.8	5.9
Weight gain		
Weight gain (kilograms)	4.6	8.7
Clinically significant weight gain (>5%) (number of participants)	162/276	58.70

BMI= Body Mass Index; SD = Standard deviation; METS = metabolic equivalents.

diagnosis (66/108, 61.1%) compared to 26.39% of women who had used AIs alone (19/72). Of the 108 women who were prescribed tamoxifen, nine (8.3%) had also been prescribed goserelin while 52 women had taken tamoxifen alone. Most women (162/276, 58.7%) had gained greater than 5% of their weight at baseline/diagnosis.

3.3. Univariate analyses

Increased weight gain (both absolute and clinically significant (≥5%)) was associated with the following patient factors in univariate analyses (see also Tables 2–5).

- Younger age
- Having hot flushes
- Inadequate fruit and vegetable intake
- Lower weight self-efficacy
- Earlier menopausal status at diagnosis
- Increasing lymphedema severity
- Less physical activity than before diagnosis (self-reported)
- Higher levels of current physical activity in METs

The only treatment factor that predicted weight gain in univariate analyses was use of tamoxifen.

Weight gain was not associated with the following factors.

- BMI at diagnosis
- Chemotherapy
- Aromatase inhibitor use
- Systemic treatment
- Hormonal therapy
- Number of lymph nodes removed
- Radiotherapy

3.4. Absolute weight gain linear regression model

In linear regression analysis, the predictors for higher absolute weight gain that were retained included: 1) having hot flushes, 2)

Table 2
Correlation with absolute weight gain – univariate analyses of patient factors.

Patient Factors	N	Test	Test statistic	95% CI [LL, UL]	*p*-value
Age at diagnosis	268	Kendall's tau	−0.18	[-0.26, −.103]	**<.001**
BMI at diagnosis	269	Kendall's tau	0.06	[-0.03, 0.14]	.178
Menopausal status at diagnosis (Premenopausal, Menopausal transition, Postmenopausal, Previous surgical menopause)	274	Kruskal Wallis test	22.90 (*df* = 3)	–	**<.001**
Hot flushes (Yes/No)	276	Mann-Whitney *U* test	−3.44	–	**.001**
Lymphedema severity (nil, mild, moderate, severe)	267	Kendall's tau	0.14	[0.04, 0.24]	**.004**
Intake of 2 fruit/5 veg serves per day (Yes/No)	276	Mann-Whitney *U* test	−1.97	–	**.049**
Change in physical activity (Less active than before diagnosis, similar, more active)	276	Kendall's tau	−0.52	[-0.14, .043]	.277
Current level of physical activity (None, Low, Moderate, High)	275	Kendall's tau	−0.12	[-0.22, −0.02]	**.014**
Current level of physical activity – dichotomised (None or low/Moderate or High)	275	Mann-Whitney *U* test	−2.64	–	**.008**
WELS1	267	Kendall's tau	−0.14	[-0.22, −0.05]	**.002**
WELS2	266	Kendall's tau	−0.17	[-0.26, −0.09]	**<.001**
WELS3	267	Kendall's tau	−0.12	[-0.21, −0.03]	**.007**
WELS4	266	Kendall's tau	−0.20	[-0.28, −0.11]	**<.001**
WELS5	266	Kendall's tau	−0.19	[-0.27, −0.09]	**<.001**
WELS6	266	Kendall's tau	−0.07	[-0.17, 0.02]	.103
WELS7	262	Kendall's tau	−0.17	[-0.26, −0.08]	**<.001**
WELS8	267	Kendall's tau	−0.15	[-0.23, −0.06]	**.001**

Notes: LL = Lower-limit, UL=Upper-limit, WELS=Weight Self-Efficacy Short Form. Bolded values indicate significance at $p < .05$. Veg = vegetables; BMI = XXX.

menopause status at diagnosis, 3) lower WELS4 score (overeating when watching TV or using the computer), 4) higher WELS1 score (overeating when anxious or nervous). The model predicted weight gain (F-ratio = 3.26, R^2-adjusted = 0.16, $p < .001$) and all retained predictors were significant ($p < .05$).

Women who experienced hot flushes were gained 2.79 kg more than women without hot flushes ($p = .013$). Women who were going through the menopausal transition at the time of diagnosis gained an additional 5.12 kg weight gain compared to women who were postmenopausal at diagnosis. Each 1-point increase in WELS4 score predicted 0.78 kgs less weight gain. This item in the WELS-SF asks about ability to resist overeating when watching television or using a computer, with a higher score indicating a greater ability to resist overeating ($p = .007$). Each 1-point increase in WELS1 score predicted an additional 0.76 kg weight gain ($p = .023$). This item asks about ability to resist overeating when anxious or nervous.

Table 3
Correlation with absolute weight gain – univariate analyses of treatment factors.

Treatment Factors	N	Test	Test statistic	95% CI [LL, UL]	*p*-value
Chemotherapy (Yes/No)	276	Mann-Whitney *U* test	−0.50	–	.627
Hormonal therapy (Yes/No)	276	Mann-Whitney *U* test	−1.23	–	.219
Number of lymph nodes removed	140	Kendall's tau	0.08	[-0.04, 0.19]	.159
Systemic treatment (Received either chemotherapy and/or hormonal therapy and/or targeted therapy)	276	Mann-Whitney *U* test	−1.53	–	.127
Tamoxifen use (Yes/No)	276	Mann-Whitney *U* test	−3.00	–	**.003**
Aromatase inhibitor use (Yes/No)	276	Mann-Whitney *U* test	−.249	–	.803
Radiotherapy (Yes/No)	276	Mann-Whitney *U* test	−1.19	–	.233

Notes: LL = Lower-limit, UL=Upper-limit. Bolded values indicate significance at $p < .05$.

3.5. *Clinically significant weight gain (weight gain ≥5%) logistic regression model*

The logistic regression model formed to identify predictors of ≥5% weight gain was significant (Chi-square 64.94, $df = 16$, $p < .001$). The predictors of the binary weight gain outcome were as follows.

- Tamoxifen use compared to no tamoxifen use (OR 2.7, 95% CI: 1.38 to 5.12)
- Each 1-point increase in WELS4 score reduced the risk of weight gain ≥5% (OR 0.82, 95% CI: 0.70 to 0.97).
- Being less physically active than at diagnosis compared to being as active/more active (OR 3.1, 95% CI: 1.54 to 6.37).<0.001).

4. Discussion

In this national survey of women with breast cancer and/or ductal carcinoma in situ, we found several patient factors to be associated with greater self-reported weight gain. These were menopausal transition stage at diagnosis, experiencing hot flushes, and lifestyle and behavioural factors. Of the treatment factors, only tamoxifen use was correlated with weight gain.

4.1. *Younger age, menopausal status, tamoxifen, and hot flushes*

Younger age was correlated with increasing weight gain in univariate analyses, consistent with earlier observational studies [6–8,13–15]. In the general population, greater weight gain has also been reported people aged <55 years compared to older people [16,17]. However, age was not retained in our model, suggesting that younger age may be a correlate of undergoing the menopausal transition rather than being postmenopausal at diagnosis. Indeed, many of the concerns that face younger (<50 years) breast cancer survivors include premature menopause and managing menopausal symptoms [18].

Premenopausal status at breast cancer diagnosis has been consistently associated with increased risk of weight gain in observational studies [7,13,15,19–21] whereas a cohort study that included only postmenopausal women reported stable weight or weight loss [22]. While there was no difference between being premenopausal at diagnosis vs undergoing the menopausal transition in our study in terms of

Table 4
Correlation with clinically significant weight gain (≥5%) - univariate analyses of patient factors.

Patient Factors	N	Test	Test statistic	95% CI [LL, UL]	*p*-value
Age at diagnosis	268	Mann-Whitney U	−3.68	–	**<.001**
BMI at diagnosis	269	Mann-Whitney U	−1.67	–	.096
Menopausal status at diagnosis (Premenopausal, Menopausal transition, Postmenopausal, Previous surgical menopause)	274	Chi square test	19.60	–	**.003**
Hot flushes (Yes/No)	276	Chi square test	11.36	–	**.003**
Lymphedema severity (nil, mild, moderate, severe)	267	Chi square test	11.87	–	.065
Intake of 2 fruit/5 veg serves per day (Yes/No)	276	Chi square test	4.35	–	.114
Change in physical activity (Less active than before diagnosis, similar, more active)	276	Chi square test	23.20	–	**<.001**
Current level of physical activity (None, Low, Moderate, High)	275	Chi square test	5.12	–	.529
Current level of physical activity – dichotomised (None or low/ Moderate or High)	275	Chi square test	3.93	–	.140
WELS1	267	Logistic regression Odds ratio	0.87	[0.79, 0.95]	**.001**
WELS2	266	Logistic regression Odds ratio	0.84	[0.77, 0.92]	**<.001**
WELS3	267	Logistic regression Odds ratio	0.89	[0.82, 0.97]	**.010**
WELS4	266	Logistic regression Odds ratio	0.82	[0.74, 0.91]	**<.001**
WELS5	266	Logistic regression Odds ratio	0.89	[0.82, 0.96]	**.004**
WELS6	266	Logistic regression Odds ratio	0.91	[0.84, 1.00]	**.046**
WELS7	262	Logistic regression Odds ratio	0.85	[0.78, 0.93]	**.001**
WELS8	267	Logistic regression Odds ratio	0.87	[0.79, 0.95]	**.002**

Notes: LL = Lower-limit, UL=Upper-limit, WELS=Weight Self-Efficacy Short Form. Bolded values indicate significance at $p < .05$.

Table 5
Correlation with clinically significant weight gain (≥5%) - univariate analyses of treatment factors.

Treatment Factors	N	Test	Test statistic	95% CI [LL, UL]	*p*-value
Chemotherapy (Yes/No)	276	Chi square test	0.65	–	.723
Hormonal therapy (Yes/No)	276	Chi square test	4.59	–	.101
Number of lymph nodes removed	140	Logistic regression Odds ratio	1.02	[0.98, 1.06]	.338
Systemic treatment (Received either chemotherapy and/or hormonal therapy and/or targeted therapy)	276	Chi square test	4.43	–	.109
Tamoxifen use (Yes/No)	276	Chi square test	15.69	–	**<.001**
Aromatase inhibitor use (Yes/No)	276	Chi square test	.825	–	.662
Radiotherapy (Yes/No)	276	Chi square test	3.83	–	.148

Notes: LL = Lower-limit, UL=Upper-limit. Bolded values indicate significance at $p < .05$.

increase in weight gain, women who were undergoing the menopausal transition gained 5.12 kg more weight than postmenopausal women. Changes in body composition, with increased fat mass, waist circumference, and decreased lean mass, have been demonstrated during the menopausal transition in the general (non-cancer) population [23]. Changes in sleep and circadian rhythms may also influence changes in appetite and energy expenditure after menopause [24]. The acceleration of these changes with breast cancer treatment may be a factor in weight gain.

Tamoxifen was associated with a 2.7 times increased risk of clinically significant weight gain in our study, whereas neither hormonal therapy overall or use of aromatase inhibitors was associated with weight gain. Although a number of cohort studies [3,6,8,15] and randomized controlled trials have not reported differences in weight with tamoxifen use [25,26], predictors of weight gain >5 kg within these trials included age younger than 60 years at entry Additionally, women in adjuvant settings gained more weight than women in preventive settings [27] and women in these trials were postmenopausal. Tamoxifen has been shown to have a negative impact on body composition [28] which may further exacerbate the body composition changes with the onset of the menopause.

Women who experienced hot flushes were more likely to have reported greater absolute, but not clinically significant, weight gain. This has been reported in some studies [29,30] but not others [9]. It is likely that there is a bidirectional relationship between weight gain and vasomotor symptoms (hot flushes and night sweats). Weight gain, in particular body fat gain, is a risk factor for experiencing hot flushes in both women with breast cancer and women without [29,31]. On the other hand, vasomotor symptoms are associated with sleep complaints which in turn have been linked to appetite dysregulation and an increase in food intake in the non-cancer population [32].

4.2. *Lifestyle factors*

Change in physical activity (PA) levels compared to pre-diagnosis were retained in the absolute and clinically significant weight gain models, but there was no correlation between weight gain and current PA levels. It has been reported that energy balance becomes positive after cancer treatment partly due to fluctuations in dietary intake and PA but also due to reduced resting energy expenditure. Physical activity decreases during and after breast cancer treatment, particularly if women are premenopausal at diagnosis [33,34]. Lower PA levels have predicted weight gain after breast cancer in some [3,14,35] but not all [36] studies. This difference may be due to methodology as studies that used self-reported PA reported an association with weight gain while objectively measured PA was not associated with weight gain. Indeed, a limitation of our study is the subjective nature of PA reporting.

Despite this, it is important to note that in the general population, low PA levels are also a risk factor for transitioning from normal weight to overweight status particularly in younger women [37]. PA slows the rate of weight gain and attenuates changes in body composition experienced by menopausal women, in the general population [23]. PA may

also play a role in reducing the stress response which can affect thermoregulation (and therefore VMS) at the level of the hypothalamus [23], and has multiple benefits for people with cancer beyond preventing weight gain, including improving cancer-specific survival, fatigue, and quality of life [38]. Women with breast cancer should be prescribed exercise as a means to improve overall health and wellbeing, as well as for its ability to prevent or slow weight gain, and interventions should focus on maintenance of pre-diagnosis PA. Incorporation of behaviour change techniques such as those associated with goals and planning, shaping of knowledge, feedback and monitoring, comparisons of outcomes, repetition and substitution, social support and association have been demonstrated to have a moderate effect on increasing both self-reported and objectively measured PA levels in women with breast cancer [39]. The use of wearable technology such as pedometers has also been shown to increase PA and daily step counts in breast cancer survivors [40]. A combination of home- and facility-based settings, and individual and group delivery of aerobic exercise interventions increases PA levels in women with breast cancer who have undergone adjuvant therapy [41]. These interventions should be prescribed to women with breast cancer to support maintenance of pre-diagnosis PA.

Weight self-efficacy when watching television or using a computer was associated with higher risk of both absolute and clinically significant weight gain in our multivariate models, while weight self-efficacy in response to anxiety was associated with decreased likelihood of absolute (but not clinically significant) weight gain. Also known as eating self-efficacy, weight self-efficacy is a measure of confidence for controlling eating behaviour in a variety of challenging situations [42] and improves as weight loss is achieved [43]. In general, higher baseline levels of eating self-efficacy predict greater weight loss during treatment as well as weight maintenance [44–46], and is an important predictor of the initiation and performance of weight control behaviours, such as adherence to an eating plan and calorie monitoring [47]. However, this relationship is not consistent, with some studies demonstrating an increase in weight gain with increased weight self-efficacy. The reasons for this are unclear, however it has been suggested that increased self-efficacy may lead to overconfidence with dietary restrictions [48].

Higher energy intake has predicted weight gain in women with breast cancer [49,50]. It is possible that lower self-efficacy, particularly while watching TV or using the computer, led to increased energy intake among the women in our study. This pattern of eating reflects eating while distracted or without paying attention to visual and satiety cues, and has been shown in experimental studies to produce a moderate increase in immediate intake, and a greater increase in later intake [51], possibly due to a decrease in neural taste processing [52]. This increase in food intake can occur regardless of conscious restriction attempt and is consistent with more recent evidence that eating while using smartphones increases caloric intake in young people [51] and that eating while performing a high perceptual load task on a computer decreased processing of satiety cues [53]. It would be reasonable for health practitioners caring for women with breast cancer to prescribe behavioural modification particularly around attentive eating. However, studies on eating while distracted were conducted in laboratory settings with people with healthy weights, and the role of attentive eating is unclear in people with overweight/obesity. Additionally, no studies have examined the role of eating self-efficacy in women with breast cancer.

4.3. Strengths and limitations

Our is the first study to use a nationally representative sample of women in Australia to identify predictors of weight gain after breast cancer. We collected a comprehensive dataset of potential predictors of weight gain, including eating self-efficacy. However, there is a risk of bias within our small convenience sample, which was heterogeneous with regards to stage, age, and time since diagnosis. We were not able to collect objectively measured data and relied on self-report of weight, change in PA levels, and other variables. We also did not collect comprehensive data on dietary intake. Our study was cross-sectional rather than longitudinal and prospective, which limits the validity of our findings. Last, body weight in kilograms may be a poor indicator of body composition, and we were unable to determine change in body composition or central adiposity, which may be more relevant to long-term health outcomes than body weight. Sarcopenia is defined as low muscle strength confirmed by the presence of low muscle quantity or quality [54], and sarcopenic obesity is a relatively new concept that describes sarcopenia together with increased adiposity [55]. Sarcopenia is common in women with breast cancer [56] and is associated with higher mortality rates [57], and change in body fat percentage after surgery has been shown to increase the risk of distant metastasis [58], The distribution of body fat also has implications for health outcomes in people with cancer with central adiposity being associated with an increase in all-cause mortality [59]. Sarcopenia increases insulin resistance due to reduction in GLUT4 expression in skeletal muscle and reduced demand for insulin-dependent glucose uptake [55] while adipose tissue plays an important role in tumour growth and metastasis and creates a tumour-promoting microenvironment by increasing circulating levels of sex hormones, adipokines and pro-inflammatory cytokines, and activating the IGF-1 – insulin pathway [60]. Body fat and muscle mass may therefore be a better indicator prognostic indicator in breast cancer than body weight alone and can be routinely measured using bioimpedance measures.

4.4. Implications for research

There is a need for longitudinal studies to confirm or refute our preliminary findings among a national cohort of Australian women. Studies should be designed as prospective cohort studies and should collect data on patient factors such as menopausal stage at diagnosis and change in menopausal stage after treatment, objectively measured physical activity levels, caloric intake and weight self-efficacy, and treatment factors such as use of tamoxifen. Outcomes should include changes in body composition (e.g. fat mass, fat percentage and fat free mass) and central adiposity (such as waist circumference and waist-hip ratio). To diagnose sarcopenic obesity, validated measures of muscle strength, such as grip strength, could also be utilized [54]. Intervention studies addressing factors associated with increased weight gain after breast cancer are also warranted, such as multimodal interventions to reduce the burden of hot flushes and to increase eating self-efficacy and mindful eating as an adjunct to diet and exercise interventions. These interventions can be compared with standard diet and exercise interventions in randomized controlled trials, and randomization should be stratified according to menopausal stage at diagnosis.

4.5. Conclusion

Exercise should be prescribed to all women with breast cancer, focusing on maintaining pre-diagnosis physical activity levels, with particular emphasis on women prescribed tamoxifen. The role of eating self-efficacy, particularly attentive eating, in managing food intake and weight after breast cancer should be explored. Multimodal supportive care interventions addressing maintenance of physical activity, dietary modification, behavioural strategies to increase eating self-efficacy, and strategies to mitigate treatment-induced hot flushes should be prescribed, and should be targeted at women undergoing the menopausal transition at diagnosis.

Statements and declarations

Ethics approval and consent to participate

Ethics approval was provided by the Human Research Ethics Committee, Western Sydney University (H12444, Oct 2017).

Availability of data and materials

The datasets used and/or analysed during the current study are available from the corresponding author **(CE)** on reasonable request.

Funding

This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Author's contributions

CE conceived of the study. CE and JB designed the study and collected data. AEC led the data analysis. VV and DN contributed to data analysis. VV, AEC, DN and JB contributed to the writing and revision of the paper.

Declaration of competing interest

None.

Acknowledgements

Participants in this research were recruited from Breast Cancer Network Australia's (BCNA) Review and Survey Group, a national, online group of Australian women living with breast cancer who are interested in receiving invitations to participate in research. We acknowledge the contribution of the women involved in the Review and Survey Group who participated in this project. We would also like to acknowledge the statistics assistance provided by Dr. Russell Thomson at Western Sydney University. CE's salary as a Principal Fellow is supported by an endowment from the Jacka Foundation of Natural Therapies.

References

[1] Vance V, Mourtzakis M, McCargar L, Hanning R. Weight gain in breast cancer survivors: prevalence, pattern and health consequences. Obes Rev 2011;12(4):282–94.
[2] Ee C, Cave AE, Naidoo D, Bilinski K, Boyages J. Weight before and after a diagnosis of breast cancer or ductal carcinoma in situ: a national Australian survey. BMC Cancer 2020;20(1):113.
[3] Vagenas D, DiSipio T, Battistutta D, et al. Weight and weight change following breast cancer: evidence from a prospective, population-based, breast cancer cohort study. BMC Cancer 2015;15:28.
[4] Pumpa KL, Dalton J, Mara JK, Zhang Y, Yip D. Identifying women at risk of weight gain after a breast cancer diagnosis: results from a cohort of Australian women. Health Promot J Aust 2022;33(1):138–47.
[5] Ryan DH, Yockey SR. Weight loss and improvement in comorbidity: differences at 5%, 10%, 15%, and over. Current obesity reports 2017;6(2):187–94.
[6] Saquib N, Flatt SW, Natarajan L, et al. Weight gain and recovery of pre-cancer weight after breast cancer treatments: evidence from the women's healthy eating and living (WHEL) study. Breast Cancer Res Treat 2007;105(2):177–86.
[7] Raghavendra A, Sinha AK, Valle-Goffin J, Shen Y, Tripathy D, Barcenas CH. Determinants of weight gain during adjuvant endocrine therapy and association of such weight gain with recurrence in long-term breast cancer survivors. Clin Breast Cancer 2018;18(1):e7–13.
[8] Koo H-Y, Seo Y-G, Cho M-H, Kim M-J, Choi H-C. Weight change and associated factors in long-term breast cancer survivors. PLoS One 2016;11(7). e0159098-e0159098.
[9] Imayama I, Alfano C, Neuhouser M, et al. Weight, inflammation, cancer-related symptoms and health-related quality of life among breast cancer survivors. Breast Cancer Res Treat 2013;140(1):159–76.
[10] Ames GE, Heckman MG, Diehl NN, Grothe KB, Clark MM. Further statistical and clinical validity for the weight efficacy lifestyle questionnaire-short form. Eat Behav 2015;18:115–9.
[11] Russell A. Data Technical Report of the ALSWH: derived variables not included in datasets. 2006.
[12] IBM SPSS Statistics for Windows [computer program]. Version 26.0. Armonk, NY: IBM Corp; 2019. Released.
[13] Sedjo RL, Byers T, Ganz PA, et al. Weight gain prior to entry into a weight-loss intervention study among overweight and obese breast cancer survivors. J Cancer Surviv 2014;8(3):410–8.
[14] Lei Y, Ho SC, Kwok C, et al. Dietary pattern at 18-month post-diagnosis and outcomes of breast cancer among Chinese women with early-stage breast cancer. Cancer Manag Res 2021;13:4553–65.
[15] Gu K, Chen X, Zheng Y, et al. Weight change patterns among breast cancer survivors: results from the Shanghai breast cancer survival study. An International Journal of Studies of Cancer in Human Populations 2010;21(4):621–9.
[16] Williamson DF. Descriptive epidemiology of body weight and weight change in U. S. adults. Ann Intern Med 1993;119(7 Pt 2):646–9.
[17] Sammel MD, Grisso JA, Freeman EW, et al. Weight gain among women in the late reproductive years. Fam Pract 2003;20(4):401–9.
[18] Howard-Anderson J, Ganz PA, Bower JE, Stanton AL. Quality of life, fertility concerns, and behavioral health outcomes in younger breast cancer survivors: a systematic review. J Natl Cancer Inst 2012;104(5):386–405.
[19] Reddy SM, Sadim M, Helenowski I, Li J, Yi N, Kaklamani V. Clinical and genetic predictors of weight gain in patients diagnosed with breast cancer. J Clin Oncol 2012;30(15).
[20] Gandhi A, Copson E, Eccles D, et al. Predictors of weight gain in a cohort of premenopausal early breast cancer patients receiving chemotherapy. Breast 2019;45:1–6.
[21] Ekenga CC, Wang X, Pérez M, Schootman M, Jeffe DB. A prospective study of weight gain in women diagnosed with early-stage invasive breast cancer, ductal carcinoma in situ, and women without breast cancer. J Womens Health (Larchmt). 2020;29(4):524–33.
[22] Nyrop KA, Williams GR, Muss HB, Shachar SS. Weight gain during adjuvant endocrine treatment for early-stage breast cancer: what is the evidence? Breast Cancer Res Treat 2016;158(2):203–17.
[23] Sternfeld B, Dugan S. Physical activity and health during the menopausal transition. Obstet Gynecol Clin N Am 2011;38(3):537–66.
[24] Kravitz HM, Kazlauskaite R, Joffe H. Sleep, health, and metabolism in midlife women and menopause: food for thought. Obstet Gynecol Clin N Am 2018;45(4):679–94.
[25] Sestak I, Harvie M, Howell A, Forbes JF, Dowsett M, Cuzick J. Weight change associated with anastrozole and tamoxifen treatment in postmenopausal women with or at high risk of developing breast cancer. Breast Cancer Res Treat 2012;134(2):727–34.
[26] Baum M, Budzar AU, Cuzick J, et al. Anastrozole alone or in combination with tamoxifen versus tamoxifen alone for adjuvant treatment of postmenopausal women with early breast cancer: first results of the ATAC randomised trial. Lancet 2002;359(9324):2131–9.
[27] Cuzick J, Forbes J, Edwards R, et al. First results from the international breast cancer intervention study (IBIS-I): a randomised prevention trial. Lancet 2002;360(9336):817–24.
[28] Scalzo RL, Foright RM, Hull SE, et al. Breast cancer endocrine therapy promotes weight gain with distinct adipose tissue effects in lean and obese female mice. Endocrinology 2021;162(11).
[29] Caan BJ, Kwan ML, Shu XO, et al. Weight change and survival after breast cancer in the after breast cancer pooling project. Cancer Epidemiol Biomark Prev 2012;21(8):1260–71.
[30] Su HI, Sammel MD, Springer E, Freeman EW, DeMichele A, Mao JJ. Weight gain is associated with increased risk of hot flashes in breast cancer survivors on aromatase inhibitors. Breast Cancer Res Treat 2010;124(1):205–11.
[31] Thurston RC, Sowers MR, Sternfeld B, et al. Gains in body fat and vasomotor symptom reporting over the menopausal transition: the study of women's health across the nation. Am J Epidemiol 2009;170(6):766–74.
[32] Kravitz HM, Matthews KA, Joffe H, et al. Trajectory analysis of sleep maintenance problems in midlife women before and after surgical menopause: the Study of Women's Health across the Nation (SWAN). Menopause 2020;27(3):278–88.
[33] Tonorezos ES, Jones LW. Energy balance and metabolism after cancer treatment. Semin Oncol 2013;40(6):745–56.
[34] Orban E, Jung AY, Möhl A, et al. Changes in alcohol consumption, body weight and physical activity among breast cancer survivors and population-based unaffected women in a prospective study. Cancer Epidemiol 2021;70:101852.
[35] Rock CL, Flatt SW, Byers TE, Colditz GA, Demark-Wahnefried W, Ganz PA. Results of the Exercise and Nutrition to Enhance Recovery and Good Health for You (ENERGY) Trial: a behavioral weight loss intervention in overweight or obese breast cancer survivors. J Clin Oncol 2015;33.
[36] Okumatsu K, Yamauchi H, Kotake R, Gosho M, Nakata Y. Association between endocrine therapy and weight gain after breast cancer diagnosis among Japanese patients: a retrospective cohort study. Med Sci 2021;9(3).
[37] Hillemeier MM, Weisman CS, Chuang C, Downs DS, McCall-Hosenfeld J, Camacho F. Transition to overweight or obesity among women of reproductive age. J Womens Health (Larchmt) 2011;20(5):703–10.
[38] Campbell KL, Winters-Stone KM, Wiskemann J, et al. Exercise guidelines for cancer survivors: consensus statement from international multidisciplinary roundtable. Med Sci Sports Exerc 2019;51(11):2375–90.
[39] Liu MG, Davis GM, Kilbreath SL, Yee J. Physical activity interventions using behaviour change theories for women with breast cancer: a systematic review and meta-analysis. J Cancer Surviv 2022;16(5):1127–48.
[40] Singh B, Zopf EM, Howden EJ. Effect and feasibility of wearable physical activity trackers and pedometers for increasing physical activity and improving health outcomes in cancer survivors: a systematic review and meta-analysis. J Sport Health Sci 2022;11(2):184–93.
[41] Lahart IM, Metsios GS, Nevill AM, Carmichael AR. Physical activity for women with breast cancer after adjuvant therapy. Cochrane Database Syst Rev 2018;1(1):Cd011292.

[42] Ames GE, Heckman MG, Grothe KB, Clark MM. Eating self-efficacy: development of a short-form WEL. Eat Behav 2012;13(4):375–8.
[43] Gilcharan Singh HK, Chee WSS, Hamdy O, et al. Eating self-efficacy changes in individuals with type 2 diabetes following a structured lifestyle intervention based on the transcultural Diabetes Nutrition Algorithm (tDNA): a secondary analysis of a randomized controlled trial. PLoS One 2020;15(11):e0242487.
[44] Nezami BT, Lang W, Jakicic JM, et al. The effect of self-efficacy on behavior and weight in a behavioral weight-loss intervention. Health Psychol 2016;35(7):714–22.
[45] Martin PD, Dutton GR, Brantley PJ. Self-efficacy as a predictor of weight change in African-American women. Obes Res 2004;12(4):646–51.
[46] Hays LM, Finch EA, Saha C, Marrero DG, Ackermann RT. Effect of self-efficacy on weight loss: a psychosocial analysis of a community-based adaptation of the diabetes prevention program lifestyle intervention. Diabetes Spectr 2014;27(4):270–5.
[47] Linde JA, Rothman AJ, Baldwin AS, Jeffery RW. The impact of self-efficacy on behavior change and weight change among overweight participants in a weight loss trial. Health Psychol 2006;25.
[48] Martin PD, Dutton GR, Brantley PJ. Self-efficacy as a predictor of weight change in african-American women. Obes Res 2004;12(4):646–51.
[49] Chen X, Lu W, Zheng W, et al. Obesity and weight change in relation to breast cancer survival. Breast Cancer Res Treat 2010;122(3):823–33.
[50] Rock CL, Flatt SW, Newman V, et al. Factors associated with weight gain in women after diagnosis of breast cancer. Women's Healthy Eating and Living Study Group. J Am Diet Assoc 1999;99(10):1212–21.
[51] Robinson E, Aveyard P, Daley A, et al. Eating attentively: a systematic review and meta-analysis of the effect of food intake memory and awareness on eating. AJCN (Am J Clin Nutr) 2013;97(4):728–42.
[52] Duif I, Wegman J, Mars MM, de Graaf C, Smeets PAM, Aarts E. Effects of distraction on taste-related neural processing: a cross-sectional fMRI study. AJCN (Am J Clin Nutr) 2020;111(5):950–61.
[53] Morris J, Vi CT, Obrist M, Forster S, Yeomans MR. Ingested but not perceived: response to satiety cues disrupted by perceptual load. Appetite 2020;155:104813.
[54] Cruz-Jentoft AJ, Bahat G, Bauer J, et al. Sarcopenia: revised European consensus on definition and diagnosis. Age Ageing 2019;48(4):601.
[55] Silveira EA, da Silva Filho RR, Spexoto MCB, Haghighatdoost F, Sarrafzadegan N, de Oliveira C. The role of sarcopenic obesity in cancer and cardiovascular disease: a synthesis of the evidence on pathophysiological aspects and clinical implications. Int J Mol Sci 2021;22(9).
[56] Morlino D, Marra M, Cioffi I, et al. Prevalence of sarcopenia in women with breast cancer. Nutrients 2022;14(9).
[57] Zhang XM, Dou QL, Zeng Y, Yang Y, Cheng ASK, Zhang WW. Sarcopenia as a predictor of mortality in women with breast cancer: a meta-analysis and systematic review. BMC Cancer 2020;20(1):172.
[58] Liu L-N, Lin Y-C, Miaskowski C, Chen S-C, Chen M-L. Association between changes in body fat and disease progression after breast cancer surgery is moderated by menopausal status. BMC Cancer 2017;17(1).
[59] Pang Y, Wei Y, Kartsonaki C. Associations of adiposity and weight change with recurrence and survival in breast cancer patients: a systematic review and meta-analysis. Breast Cancer; 2022.
[60] Park J, Morley TS, Kim M, Clegg DJ, Scherer PE. Obesity and cancer–mechanisms underlying tumour progression and recurrence. Nat Rev Endocrinol 2014;10(8):455–65.