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Title: Dietary modification for women after breast cancer treatment: A narrative review

Abstract

Diet is thought to account for about 25% of cancers in developed countries. It is well-documented that the risks associated with both the breast cancer itself and its treatments are important for women previously treated for breast cancer. Women are at risk of recurrence of the primary disease and prone to develop treatment-induced comorbidities, some of which are thought to be modified by diet. With a view to making dietary recommendations for the breast cancer patients we encounter in our clinical nursing research, we mined the literature to scope the most current robust evidence concerning the role of the diet in protecting women against the recurrence of breast cancer and its potential to ameliorate some of the longer-term morbidities associated with the disease. We found that the evidence about the role of the diet in breast cancer recurrence is largely inconclusive. However, drawing on international guidelines enabled us to make three definitive recommendations: women at risk of breast cancer recurrence, or who experience co-morbidities as a result of treatment, should limit their exposure to alcohol; moderate their nutritional intake so it does not contribute to post-menopausal weight gain; and should adhere to a balanced diet. Nursing education planned for breast cancer patients about dietary issues should ideally be individually-tailored, based on a good understanding of the international recommendations and the evidence underpinning them.

Introduction

With approximately 10 million new cases reported internationally each year, cancer is a significant health problem (Parkin et al., 2005). Due to advances in early detection and treatment, 66% of people diagnosed with cancer in Western countries are now expected to be alive five years after diagnosis (Jemal et al., 2009). Within the last decade women treated for breast cancer represent the majority (27%) of this growing group (Parkin and Fernandez, 2006, Jemal et al., 2009). Due to these encouraging developments in mortality rates, while breast cancer is still not considered curable it is now often conceptualised as a 'chronic' disease (Eakin et al., 2006).

An emergent discourse in Western countries is the potential for breast cancer chronicity to become a significant public health issue. This concern arises from the well-documented risks associated with both the breast cancer itself and its treatments. Women previously treated for breast cancer have, for example, an increased risk of recurrence of the primary disease (Tolaney and Winer, 2007); the development of second primary cancers (Mellemkjaer et al., 2006); and a propensity to develop treatment-induced comorbidities in a variety of organ systems (Carver et al., 2007, Eakin et al., 2006, Gupta et al., 2006, Hooning et al., 2006, Maxwell and Viale, 2005, Yeh et al., 2004). These adverse outcomes threaten the quality of life and health of the individual after breast cancer treatment. Many of these outcomes are, however, believed to be modifiable. Hence a second theme in the discourse, related to the first, emphasizes the responsibility of women previously treated for breast cancer to observe exemplary health and lifestyle behaviours to prevent or lessen the severity of treatment-related morbidities (Ganz and Hahn, 2008, Jones and Demark-Wahnefried, 2006). Women previously treated for breast cancer are now encouraged to consistently practise health-promotion and risk-reduction strategies such as regular health screening; resistance and aerobic training; and alcohol and tobacco restriction.

A health behaviour strategy also thought to protect health after cancer treatment is dietary modification. It is, however, also one of the most contested in the literature and in clinical practice (Hankinson et al., 2004, Norman et al., 2007). The dietary discourse in cancer emerged from epidemiological studies, which quantified that women from countries with previously low breast cancer rates developed identical

incidence rates to their host countries within one or two generations (McCracken et al., 2007, Pike et al., 2002). These studies, which implicated environmental changes in the increased incidence of breast cancer in migrant populations, indicated that Westernised diets could play a significant part in the development or recurrence of the disease.

Diet is now thought to account for approximately 25% of cancers in developed countries (Key et al., 2002); hence its role as a modifiable risk factor in the context of breast cancer is of increasing interest to both health professionals and lay people. There is considerable confusion however about the precise role of specific dietary components (such as fats and vitamins) in breast cancer initiation, recurrence or protection; and the part they play in offsetting the long-term toxicities of breast cancer treatments such as ovarian failure (McCullough and Giovannucci, 2004, Duncan, 2004).

We recently undertook a study that examined women's experiences of cancer, their health practices once treatment is completed, and their educational needs with respect to their post-treatment health behaviours (McCarthy et al., In review-a, McCarthy et al., In review-b, McCarthy et al., In review-c, Tramm, 2010, Tramm et al., In review). This paper scopes the evidence concerning the role of diet after breast cancer treatment in protecting women against recurrence, and the potential of diet to ameliorate some of the longer-term morbidities associated with previous breast cancer treatment. Our aim in this paper is to present the narrative review of the evidence that was undertaken prepatory to the development of education programs for breast cancer patients in our treatment catchment area. The evidence presented in this paper is selected from the latest meta-analyses and systematic reviews, and explained where necessary with reference to other higher-quality evidence such as large epidemiological and case-control studies, and significant theoretical and clinical papers. Drawing on current international guidelines, the paper concludes with a discussion of current dietary recommendations for women who have completed breast cancer treatment and the educational implications of these for oncology nurses.

Individual nutrients and breast cancer

Dietary fat

The role of dietary fat in the recurrence of breast cancer is controversial (Greenwald and Dunn, 2009). Suspicion first arose when cumulative evidence from animal studies indicated that dietary fat might contribute to breast cancer initiation and promotion in some way (Fay et al., 1997). Subsequent large epidemiological studies in humans supported the association between fat intake and breast cancer, with early estimates of a 2.5-fold risk reduction for breast cancer if fat intake was reduced by 50% (Prentice and Sheppard, 1990). A further pooled meta-analysis of case-control and cohort studies appeared to confirm this relationship (Boyd et al., 1993); with a larger meta-analysis of 45 studies (31 case-control and 14 cohort studies) concluding that a high fat intake increased the risk of breast cancer irrespective of whether the fats were saturated or unsaturated, and calculating an overall increased risk of 13% (Boyd et al., 2003). In contrast, a meta-analysis of 8 cohort studies at approximately the same time detected only a modestly-increased risk, which was associated only with saturated fat intake (Smith-Warner et al., 2001a). Later population-based cohort studies, however, tended to support an inverse relationship between fat intake and breast cancer incidence in pre- and postmenopausal women (Prentice et al., 2006, Sieri et al., 2008, Wang et al., 2008). For example, Thiebaut et al. (2007) analysed a large prospective cohort of postmenopausal women and reported direct relationships between all subgroups of fat and breast cancer. However, this trend has been challenged by the latest published meta-analysis, which reports no overall association between animal fat intake and breast cancer development (Alexander et al., 2010).

There are two reasons for these contradictory conclusions. First, methodological critiques of these studies highlight a consistent failure to control for a variety of confounding variables (McCullough and Giovannucci, 2004). They also argue that data are often ‘tainted’ by recall bias; and the accuracy and validity of the instruments used to collect the data is frequently challenged (Michels et al., 2007). Equally important, the second reason questions the precise role of fat in breast cancer recurrence. The problem with fats appears not to be what they contain, but what happens when fat intake exceeds metabolic requirements after menopause. At present, it is considered probable that the components of saturated and unsaturated fats are not

implicated in the development of breast cancer itself, although saturated fat reduction is clearly important in the reduction of the longer-term cardiovascular problems precipitated by cancer treatments containing high-dose anthracyclines (Linos and Willett, 2007). It is more likely that the post-menopausal weight gain arising from a diet rich in energy-dense foods such as fats is the real culprit (Howell and Harvie, 2008, van den Brandt et al., 2000).

The mechanisms underlying the relationship between weight gain and the recurrence of breast cancer in post-menopausal women are not fully understood. They are complex and possibly inter-related. For example, a high intake of dietary fat can result in the deposition of adipose tissue. Adipose tissue produces its own oestrogenic compounds called leptins. High levels of adipose tissue are believed to elevate intrinsic oestrogen levels, which can be implicated in the development of some breast cancers (Sugimura, 2000). However, the accumulation of adipose tissue can also lead to metabolic syndrome and subsequent hyperinsulinemia and therefore, promote tumour growth via pathways other than oestrogen exposure (Blackburn and Wang, 2007). Irrespective of the precise aetiology, there are now convincing data that implicate postmenopausal weight gain arising from excess fat consumption as a risk factor for breast cancer. The latest report from the World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR) states unequivocally that "there is abundant and consistent epidemiological evidence that … greater body fatness is a cause of postmenopausal breast cancer" (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010) (p. 3). Given the menopause-inducing nature of many breast cancer treatments, this risk factor should be seriously considered where women have previously been treated for breast cancer.

Carbohydrates

The potential role of carbohydrates has also been investigated in the breast cancer context. Earlier cohort studies seeking associations between carbohydrate intake, glycaemic index or glyacemic load and breast cancer incidence did not reveal strong associations (Cho et al., 2003, Higginbotham et al., 2004, Sieri et al., 2008). Recent meta-analyses of cohort studies supported these initial findings, reporting only weak associations between carbohydrate intake and breast cancer development (George et

al., 2009, Larsson et al., 2009). These findings were challenged by a similarly recent population-based cohort study that reported significant associations between high carbohydrate intake, high glycaemic load and breast cancer in premenopausal women younger than 50 years (Wen et al., 2009). Notably, Larrson et al. (2009) also recently reported associations between carbohydrate intake and the risk of oestrogen receptor-positive breast cancer, a finding the authors argued implicates insulin in the development of the disease. Unfortunately, contradictory evidence collated during one meta-analysis of 39 case-control and cohort studies across all types of cancers only found significant associations between glycaemic load or glycaemic index and cancers of the colorectum and the endometrium (Gnagnarella et al., 2008); and even these findings were recently questioned by the latest meta-analysis that found no associations between glycaemic index, glycaemic load and colorectal cancer risk at all (Mulholland et al., 2009).

Several reasons for these contradictory results are proposed. It could be that like fats, carbohydrate metabolism is implicated in complex carcinogenic processes that are not necessarily related to the precise components of these compounds. It is increasingly accepted that where breast cancer is concerned *too much* carbohydrate resulting in the deposition of fatty tissue, which leads to higher intrinsic oestrogen levels, is one potential culprit. A high carbohydrate load might also increase the bioavailability of sex hormones, which is consistently suggested as a mechanism that encourages breast carcinogenesis and discourages apoptosis (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010). Furthermore, like fats, carbohydrate metabolism alters insulin levels. Carbohydrate in excess of requirements can result in compensatory hyperinsulinaemia and insulin resistance, which may promote tumour growth and also stimulate proliferative factors such as insulin-like growth factor (Sugimura, 2000, Longo and Fontana, 2009).

Alcohol

Alcohol is an important dietary factor to consider in this context for three reasons. First, it disinhibits appetite and this coupled with its high caloric density can contribute to unnecessary weight gain (Michels et al., 2007). This could increase the risk of recurrence in postmenopausal women (Kwan et al., 2010). Second, the

metabolites of alcohol are considered potentially carcinogenic (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010). Third, alcohol mediates the actions of oestrogen and other proliferative factors in numerous ways (Dumitrescu and Shields, 2005). Hence cumulative evidence over the last four decades has firmly established alcohol as a risk factor for breast cancer (Singletary and Gapstur, 2001, Smith-Warner et al., 1998, Williams and Horm, 1977, Knight et al., 2009). Evidence from large pooled meta-analyses indicates there is a 7 % increased risk of breast cancer with every 10g of alcohol consumed per day (Hamajima et al., 2002, Smith-Warner et al., 1998).

Fibre

Fibre derived from fruits, vegetables, cereals and legumes is often discussed as a protective dietary factor in the literature (McCullough and Giovannucci, 2004). Considerable attention has subsequently been directed towards the compounds found in cruciferous vegetables such as broccoli, which are often hypothesised to help reduce the risk of lung, colorectal and to a lesser extent breast cancer (Higdon et al., 2007). In breast carcinogenesis, fibre is also thought to reduce the enterohepatic circulation of steroids, which results in lower overall exposure to oestrogen (Hanf and Gonder, 2005). Unfortunately neither hypothesis is supported by subsequent meta-analyses (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010, Key et al., 1999).

As with fats and carbohydrates, however, covariates must be accounted for when investigating the potential risks or protectors associated with specific dietary components in the breast cancer context. The picture with fibre becomes more complex, for example, when menopausal status and tumour receptivity are taken into account. Although earlier large cohort trials did not describe significant associations between dietary fibre and breast cancer incidence in pre- or postmenopausal women (Willett et al., 1992, Terry et al., 2002, Smith-Warner et al., 2001b), a more recent large prospective cohort study clearly suggested an inverse relationship between fibre intake and breast cancer incidence in pre-menopausal women (Cade et al., 2007). Another large cohort trial documented associations between fibre intake, postmenopausal breast cancer risk and oestrogen/progesterone receptor status (Park et

al., 2009); while Suzuki et al (2009) reported significant associations in their meta-analysis of trials between specific types of fibre intake, positive oestrogen receptor status and postmenopausal breast cancer incidence. They calculated a risk reduction of 34% for women with a high fibre intake derived from fruit sources (Suzuki et al., 2009).

Despite some indications that fibre might be protective against breast cancer, at present no firm conclusions can be drawn about its precise role after breast cancer treatment. Pooled studies to date have either provided too much low quality or inconsistent data, or the number of studies are too few to enable conclusions to be made (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010). Nonetheless, it is likely that dietary fibre will remain a biological mechanism of interest in breast cancer recurrence. This is because it is known to improve insulin sensitivity and also helps counteract the weight gain common in treatment-induced menopause by enhancing the sensation of satiety (Hanf and Gonder, 2005).

Micronutrients

The oxidative stress reduction and other 'anti-cancer' properties of vitamins A, C and E have received considerable attention in the cancer literature (Lee et al., 2004, Nagel et al., 2010, Greenwald et al., 2007). Unfortunately, results concerning these vitamins are inconclusive about protective associations (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010). There is similarly limited evidence that adequate folate and beta carotene intake could partially offset the risk of breast cancer in women who consume moderate to high levels of alcohol (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010). Folate and beta carotene have antioxidant properties that mop up the damage caused by cellular processes and they also enhance the ability of cells to function properly; inadequacies of both of these activities are implicated in the development and recurrence of cancers (Willcox et al., 2004).

Two other commonly-studied micronutrients are also believed to play a protective role in breast cancer, but their mechanism of action is not as well-hypothesised. These

are vitamin D and calcium. Data about the possible benefits of vitamin D first emerged from epidemiological studies conducted in the 1990s, which indicated inverse correlations between greater sun exposure at higher altitudes (implying higher vitamin D levels) and breast cancer incidence (Gorham et al., 1990, John et al., 1999). Recent meta-analyses supported this inverse relationship, reporting a 9% risk reduction when low and high Vitamin D intakes were compared (Chen et al., 2009). Chen et al (2009) also documented a 45% lower risk of breast cancer in cohorts with the highest levels of endogenous Vitamin D compared to cohorts with the lowest levels. A later case-control study, not available to Chen et al (2009), further reported that Vitamin D obtained from supplements was independently associated with breast cancer risk reduction (Anderson et al., 2010).

As is common in these studies, when covariates are considered, the picture becomes more subtle. For example, while one meta-analysis (Gissel et al., 2008) and one randomised control trial of 36,282 postmenopausal women (Chlebowski et al., 2008) reported no associations between vitamin D intake and breast cancer development, when the meta-analysis was restricted to doses ≤ 400 IU daily, higher doses were associated with lower breast cancer incidence (Gissel et al., 2008). Significant risk reduction was also observed in a large randomised controlled trial of 1179 women when daily vitamin D doses ≥ 1100 IU (Lappe et al., 2007). Current recommendations for vitamin D do not exceed 400 IU per day but such cumulative evidence of preventive properties at higher dosages may change this threshold in the near future (Hines et al., 2010). There are already calls to increase the recommended vitamin D intake for all women to 2000 IU per day in order to reduce the estimated incidence of breast cancer by as much as 25% (Garland et al., 2009). Nonetheless, a comprehensive systematic review undertaken by the United States Agency for Healthcare Research and Quality classified available studies with respect to vitamin D as inconclusive (Chung et al., 2009); and this stance is supported by other international guidelines (National Health and Medical Research Council (NHMRC), 2003, Food Standards Agency, 2010, World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010, Cancer Research UK, 2009). Concerns exist mainly because significant associations between vitamin D intake and breast cancer risk reduction were not evident during a follow-up period of 7 to 12 years in either pre- or postmenopausal cohorts (Chung et al., 2009).

The situation is similarly confusing for calcium. In their meta-analysis of the available studies, Chen et al. (2009) reported a decreased overall breast cancer risk reduction of 19% when comparing high and low quintiles of calcium intake. However, stratified analyses reached significance only for premenopausal women. There are also reports of consistent associations of breast cancer risk reduction with a daily intake of 780mg to 1750mg of calcium for premenopausal but not for postmenopausal women (Chung et al., 2009). When the role of calcium was considered in a randomised controlled study of 36,282 women, the authors concluded that calcium supplementation does not reduce invasive breast cancer incidence in postmenopausal women (Chlebowski et al., 2008). The evidence concerning the role of calcium in breast carcinogenesis is therefore considered so limited and contradictory that no conclusions can reliably be drawn (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010).

Vitamin D and calcium nonetheless have significant roles to play after treatment for breast cancer. Recent high quality evidence indicates that women who have had breast cancer often have intrinsically insufficient levels of both nutrients prior to diagnosis, and that this deficiency carries forward after treatment (Coleman et al., 2008, Crew et al., 2009, Neuhouser et al., 2008). The reason for this is not currently clear, as is the possible role of this deficiency in the initiation or recurrence of breast cancers. However the importance of both of these micronutrients is unquestioned with respect to bone health. The cytotoxic and hormonally-active treatments used during breast cancer are often associated with the induction of menopause; meaning the women receiving them are subsequently prone to impaired bone density and osteoporotic fractures (Hines et al., 2010, Hillner et al., 2003). Adequate levels of vitamin D and calcium are recommended to lessen the risk of these adverse outcomes (National Health and Medical Research Council (NHMRC), 2003).

Phyto-oestrogens

Phyto-oestrogens have been extensively studied in the context of breast cancer prevention and recurrence. Plant-derived oestrogens are classed as lignans (which are more common in Western diets and derived from flaxseed, cereals and berries) and

isoflavones (derived from soya bean and its products). Interest in isoflavones arose when it was first noticed that the high rate of soy products consumed by Asian women seemed to correlate with a low breast cancer incidence. When these women migrated to the West, where soy consumption is not so entrenched, the breast cancer incidence equalled that of Western women within a few generations. Soy products have been the main focus of research activity because they are so structurally similar to human oestrogen. Depending on the researcher's point of view, they are therefore considered either helpful in preventing the recurrence of cancer because they are capable of oestrogen receptor binding and blocking; or they are viewed as harmful because they can mimic the action of the naturally-produced oestrogens that are considered tumour-promoting in a significant proportion of breast cancers (Duncan, 2004, Gold et al., 2006, Hankinson and Eliassen, 2007, World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010).

The results of studies of phyto-oestrogens mirror this controversy. Soy intake was modestly associated with a reduced risk of breast cancer in one pooled meta-analysis of cohort and case control studies comprising pre- and postmenopausal women (Trock et al., 2006). However, the authors of this meta-analysis expressed concerns that few studies included in the pool were originally designed to assess soy as a risk factor and they also noted inconsistent use of validated instruments. Additional evidence from a meta-analysis of later studies reported a dose-response relationship between soy intake and breast cancer prevention and concluded that the amount of soy intake typical of Asian populations could reduce breast cancer risk (Wu et al., 2008). In general, however, meta-analyses of all studies are limited because the studies they have pooled are so heterogeneous. For example, the measure of soy intake is rarely standardised and the differences in the type and quantity of soy consumed vary so widely among studies that it is difficult to reliably determine estimates of risk (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010).

The situation with lignan is less confusing, mainly because relatively few studies have been conducted in this area. *In vivo* studies revealed that lignan might have preventive potential, but to date only one meta-analysis has found significant associations between lignan intake and breast cancer risk reduction in humans, particularly in

postmenopausal women (Velentzis et al., 2009, Bergman et al., 2007). As a result of the confusion surrounding phyto-oestrogens, the overall evidence is considered so limited that no conclusions about their anti-tumour effects can be made at present (World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010).

Implications for women treated for breast cancer

At present there is much conjecture and limited hard fact that plausibly explains how and why diet might be implicated in breast cancer recurrence. There are too many questions about the methodological reliability and validity of most of the research undertaken to date, with the issue further clouded by the complex, synergistic nature of food metabolism. Moreover, it is hard to locate good evidence that explicitly addresses the role of diet in cancer recurrence, as it is usually studied prospectively in women who have never been diagnosed with the disease. The only evidence of a causal relationship convincing enough for any international body to make dietary recommendations related to breast cancer pertains to first, alcohol consumption at any age; and second, to post-menopausal obesity arising from energy-dense food consumption that is excess to metabolic requirements. This evidence applies to women in general to prevent cancer, not specifically to women who have already had breast cancer.

Nonetheless, our nursing research in this area indicates that many women treated for breast cancer are curious about the protective potential of diet and anxious for dietary advice that will help them maximise their health outcomes. Given the menopause-inducing nature of many cancer treatments, and the oestrogen-sensitive nature of many breast tumours, all that can be said in relation to limiting the risk of recurrence is that women should probably minimise their consumption of alcohol to the level recommended by their relevant health authority, which in most Western countries is considered one standard measure of alcohol per day. Post-menopausal weight gain resulting from unnecessary consumption of fats and carbohydrates should also be avoided.

Multimodal cancer therapy is associated with numerous side effects from surgery, radiotherapy and systemic therapies such as ovarian failure, fatigue, cardiotoxicity, anaemia and bone loss (Montazeri, 2008). The scientific community recognises the important role of diet in preventing or managing some of these longer-term morbidities. Unlike recommendations to prevent cancer recurrence, the dietary evidence and the guidelines in this respect are unequivocal and firmly grounded in good evidence. Essentially, the dietary guidelines for the management of treatment toxicities are the same as those recommended for all women. A balanced diet is also considered generally protective of women with chronic conditions like breast cancer because the nature and proportions of nutrients it contains helps to maintain good cellular function; to mop up any damage caused by metabolic processes; and to maintain overall health. In terms of individual dietary components, the following strategies are therefore recommended (Food Standards Agency, 2010, National Health & Medical Research Council, 2003, World Health Organization, 2003, Dietitians Association of Australia, 2010, World Health Organization, 2004, World Cancer Research Fund and American Institute of Cancer Research (WCRF/AICR), 2010, Cancer Research UK, 2009):

1. Limit fatty foods and simple sugar intake. This helps prevent excessive weight gain, moderates endogenous hormone levels and facilitates cardiovascular fitness. All fats and sugars are high in kilojoules, but some fats are essential and these should be regularly incorporated into the diet. These include polyunsaturated fats derived from plant and piscine sources that contain cardioprotective fatty acids such as omega-3 and omega-6.
2. Eat plenty of fruit, vegetables, legumes and wholegrain cereals, which are important for several reasons. For example they contain fibre, which enhances feelings of satiety and therefore could help moderate fat intake. These foods are also abundant in the micronutrients vital for optimum cellular function and fatigue minimization.
3. Include daily serves of lean red and white meats, fish or vegetarian sources of protein such as nuts and legumes. These nutrients are essential for the protein that helps maintain muscle strength and endurance; and the vitamin B12 that helps protect DNA. Equally important, lean meats are excellent sources of the iron that assists with fatigue reduction, cognitive function and resistance to infection in the context of breast cancer chronicity.

4. Low fat dairy products are essential for their contribution of calcium. The bone loss associated with treatment-induced menopause can be mediated with a high calcium intake from dairy foods when combined with adequate vitamin D obtained from sensible sun exposure or from other foods such as margarine, eggs, tuna and salmon. Due to the limited evidence base, recommendations relating to the amount of daily vitamin D intake cannot currently be made.
5. Moderate alcohol intake. This limits exposure to oestrogen through excessive weight gain and the oestrogen-mimicking and carcinogenic metabolites of alcohol. It is recognised that one serve of alcohol daily could protect women at risk of cardiac disease.

Implications for oncology nurses

Education is a key factor with respect to enabling positive health behaviours such as dietary modification (Green and Kreuter, 2005), and oncology nurses are ideally placed to deliver it. However, preparing women for life after breast cancer treatment and advising them on the diet that will maximise their treatment outcomes is a complex business. Many issues related to the cancer itself require careful consideration – such as the hormone receptivity of the primary tumour; the nature of the treatment received; and the short- and long-term health outcomes that can be expected – because all of these factors can influence the specific dietary advice required. Coupled with this is attention to the demographic characteristics of the individual woman and her social circumstances. A woman's dietary and educational needs are dynamic, highly individual and vary according to her phase in the cancer trajectory (Rees and Bath, 2001, Rutten et al., 2005). For example, research indicates that younger breast cancer patients have different educational needs to their older peers (Vivar and McQueen, 2005, Thewes et al., 2004, Beckjord et al., 2008). They tend to seek more information and base their search for information on a broader range of sources (Rutten et al., 2005). Similarly, it is not helpful to advise a woman who can barely afford her cancer treatment to eat salmon three days a week. The poor educational outcomes of neglecting such factors are often reported in the literature (Schmid-Buchi et al., 2008, Cappiello et al., 2007).

Women also describe problems with the content, timing and pedagogical aspects of the education they receive from health care staff in preparation for life after treatment. In the study we conducted in this field (Tramm, 2010, McCarthy et al., In review-a), women's preferred educational preparation for dietary modification occurred in the period one or two months either side of treatment completion, when some sort of normality was re-established in their lives. It is plausible that dietary education is of little benefit while women are physically and emotionally distressed with the symptoms of active treatment during their first few cycles. Their memory can also be significantly affected by cytotoxic treatments and treatment-induced menopause during and after treatment (Cella and Fallowfield, 2008). Cognitive function should therefore be assessed before embarking on patient education and accounted for in both educational content and its method of delivery. Pedagogy is also important. Research indicates that people treated for cancer generally prefer oral group presentations by experienced peers and health professionals (Campbell et al., 2004, Rees and Bath, 2000, Hoey et al., 2008), followed by written summaries and visual resources that account for differences in individual cognitive performance after treatment (McPherson et al., 2001).

Conclusion

This paper has reviewed the evidence about the role of diet after breast cancer treatment. While the evidence is largely inconclusive, three definite recommendations can be made and incorporated into educational programs: women at risk of breast cancer recurrence, or who experience co-morbidities as a result of treatment, should limit their exposure to alcohol; moderate their nutritional intake so that it does not contribute to post-menopausal weight gain; and partake of a balanced diet in accordance with international guidelines. Dietary education about these issues should ideally be individually-tailored, based on a good understanding of the international recommendations and the evidence underpinning them; in addition to a thorough assessment of the woman's educational needs, their treatment outcomes and their specific social conditions to ensure their health outcomes are optimised.

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