Article

How Does Dietary Intake Relate to Dispositional Optimism and Health-Related Quality of Life in Germline *BRCA1/2* Mutation Carriers?

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Abstract: *Background*: The Mediterranean diet (MD) is an anti-inflammatory diet linked to improved health-related quality of life (HRQoL). Germline (g)*BRCA1/2* mutation carriers have an increased risk of developing breast cancer and are often exposed to severe cancer treatments, thus the improvement of HRQoL is important. Little is known about the associations between dietary intake and HRQoL in this population. *Methods*: We included 312 g*BRCA1/2* mutation carriers from an ongoing prospective randomized controlled lifestyle intervention trial. Baseline data from the EPIC food frequency questionnaire was used to calculate the dietary inflammatory index (DII), and adherence to MD was captured by the 14-item PREDIMED questionnaire. HRQoL was measured by the EORTC QLQ-C30 and LOT-R questionnaires. The presence of metabolic syndrome (MetS) was determined using anthropometric measurements, blood samples and vital parameters. Linear and logistic regression models were performed to assess the possible impact of diet and metabolic syndrome on HRQoL. *Results*: Women with a prior history of cancer (59.6%) reported lower DIIs than women without it ($p = 0.011$). A greater adherence to MD was associated with lower DII scores ($p < 0.001$) and reduced odds for metabolic syndrome (MetS) ($p = 0.024$). Women with a more optimistic outlook on life reported greater adherence to MD ($p < 0.001$), whereas a more pessimistic outlook on life increased the odds for MetS (OR = 1.15; $p = 0.023$). *Conclusions*: This is the first study in g*BRCA1/2* mutation carriers that has linked MD, DII, and MetS to HRQoL. The long-term clinical implications of these findings are yet to be determined.

Keywords: *BRCA1*; *BRCA2*; DII; Mediterranean diet; HRQoL; metabolic syndrome

1. Introduction

With continual improvements in cancer outcomes, both patients and clinicians are shifting their focus from survival alone towards improving health-related quality of life (HRQoL) and patient-centred functional outcomes [1]. HRQoL is defined as the impact a disease and its treatment have on a patient's physical, functional, psychological, social, and financial well-being [2–4]. In cancer care, there is a growing recognition of the significance of HRQoL, as reduced HRQoL may result in lower treatment adherence [5] and an increased risk of mortality [6]. A more comprehensive definition of HRQoL could encompass dispositional optimism, which is a psychological attribute associated with health advantages [7]. Different aspects of HRQoL have been associated with chronic inflammation, i.e., decreased physical [8] and cognitive functioning [9], increased fatigue [10] and higher pain levels [11].

Pre-treatment inflammatory status may predict the development of common cancer treatment side effects [12], e.g., aromatase inhibitor-induced musculoskeletal syndrome in women with pre-existing musculoskeletal pain. Most importantly, elevated inflammatory markers have been associated with adverse cancer outcomes [13,14], potentially by promoting a microenvironment for tumour growth and metastasis [15]. The quantity, quality, and composition of foods have been shown to regulate inflammation [16–18]. This has prompted research into developing a literature-derived index to reflect the inflammatory potential of diets; the Dietary Inflammatory Index (DII) [19] scores an individual's diet on a continuum from anti- to pro-inflammatory. A pro-inflammatory diet has been linked to an increased cardiovascular risk and mortality [20], and it increases the likelihood of both metabolic syndrome (MetS) [21] and various types of cancer [22–25]. Recent studies indicate a negative association between a pro-inflammatory diet and HRQoL [26–28]. A diet associated with low DII scores is the Mediterranean diet (MD) [29]. MD is characterized by high consumption of fruits, vegetables, legumes, grains, and polyunsaturated fats from olive oil and nuts, moderate consumption of fish and dairy products, and low intake of red meat and processed foods [30]. MD has been shown to be associated with reduced cardiovascular risk [31], prevent MetS and lower cancer risk [32]. Furthermore, adherence to MD has been linked to improved HRQoL in healthy individuals [33,34], as well as cancer survivors [35].

Breast cancer (BC) is the most common type of cancer in women [36]. Particularly vulnerable are women with a germline (g)*BRCA1/2* mutation have a risk of 69–72% of developing breast cancer and a risk of 17–44% of developing ovarian cancer by the age of 80 years [37]. These women are exposed to cancer treatments and/or prophylactic surgeries with detrimental short- and long-term effects on their health [38–42] and HRQoL [6,43–45]. Recent studies suggest that beneficial dietary changes after completing primary cancer treatment, as opposed to during treatment, might be most effective in improving HRQoL [46]. Dietary factors to reduce chronic inflammation and improve metabolic profile may be an approach to improving HRQoL, functional capacity, and cancer outcomes in women with a g*BRCA1/2* mutation. A first step in addressing this issue is to determine the relationship of DII, MD, MetS, and different aspects of HRQoL in g*BRCA1/2* mutation carriers with and without a previous history of cancer.

2. Methods

2.1. Study Design and Participants

The present study is a cross-sectional secondary analysis of the baseline data from the randomized controlled LIBRE-2 trial (a lifestyle intervention study in women with hereditary breast and ovarian cancer) and the associated feasibility study LIBRE-1 [47,48]. The trials are registered at ClinicalTrial.gov (NCT numbers: NCT02087592–registered on 14 March 2014, NCT02516540–registered on 6 August 2015). The LIBRE-2 trial is an ongoing, two-armed randomized (1:1) controlled multicentre trial conducted in Germany aimed at determining the impact of a structured one-year lifestyle intervention program on adherence to MD, cardiorespiratory fitness, and body mass index (BMI) among g*BRCA1/2* mutation carriers. The study cohort includes both women with a previous diagnosis of early stage cancer in remission (diseased) and without a prior cancer diagnosis (non-diseased). Details on the study design have been published elsewhere [47,48]. A total of 312 participants were available for the current analysis.

2.2. Instruments

Blood samples, anthropometric measurements, and medical history. At baseline, participants completed a standardized questionnaire to collect information on their medical history, socio-demographic factors, as well as lifestyle factors. Furthermore, all participants underwent a physical examination to determine systolic and diastolic blood pressure, heart rate, and anthropometric measurements such as height (in m), body weight (in kg), and waist and hip circumferences (in cm). These were used to calculate BMI (kg/m^2) and the

waist-to-hip ratio (waist circumference in cm/hip circumference in cm). Blood samples were taken after a 12-h fasting period, and analysed by the affiliated laboratories of the local institutions. MetS was defined according to the International Diabetes Federation criteria by the presence of a waist circumference $\geq$ 80 cm and at least two metabolic abnormalities, i.e., fasting glucose $\geq$ 100 mg/dL, systolic blood pressure $\geq$ 130 mmHg and/or diastolic blood pressure $\geq$ 85 mmHg, triglycerides $\geq$ 150 mg/dL, HDL-cholesterol < 50 mg/dL and/or treatment with lipid-lowering, glucose-lowering or antihypertensive drugs. Cardiopulmonary exercise testing was conducted to assess cardiorespiratory fitness via peak oxygen uptake (VO_{2peak}).

FFQ, MEDAS and Dietary Inflammatory Index. Dietary intake was determined by two validated questionnaires. The participants completed the German version of the PREDIMED questionnaire, the Mediterranean diet adherence screener (MEDAS), a 14-item questionnaire that captures adherence to MD [49–51]. We calculated the MEDAS score as the percentage of positively answered questions [52]. Additionally, the German version of the EPIC food frequency questionnaire (FFQ) was applied to collect information on the quantity and frequency of 148 food items consumed over the previous year [53,54]. Data from the FFQ were then used to calculate DII using the method reported by Shivappa et al. [19]. Briefly, the DII is based on 1943 scientific papers scoring 45 food parameters according to whether they increased (+1), decreased (−1), or had no effect (0) on six inflammatory biomarkers (IL-1β, IL-4, IL-6, IL-10, TNF-α, and CRPs). As reported in previous studies [22,55–57], not all required food items were assessed by the German FFQ. Hence, the DII was calculated using the corresponding 30 food parameters available from the FFQ used in our study. Those were carbohydrates, protein, saturated fat, polyunsaturated fatty acids (PUFA), monounsaturated fatty acids (MUFA), n-3-fatty-acids, n-6-fatty-acids, cholesterol, total fat, energy, fibre, alcohol, iron, magnesium, zinc, vitamin A, thiamin, vitamin B12, riboflavin, niacin, vitamin B6, folic acid, vitamin C, vitamin D, vitamin E, flavonones, anthocyanidins, flavan-3-ol, flavonols, and flavones.

Psychological questionnaires. All LIBRE trial participants completed several psychological questionnaires. To assess optimism and pessimism as a personality trait, the revised 10-item life orientation test (LOT-R) was applied [58]. The "optimism score" (LOTR-O) ranging from 0 (minimally optimistic) to 12 (maximally optimistic) was calculated as the sum of the three positively formulated items. The "pessimism score" (LOTR-P) was calculated accordingly. The EORTC QLQ-C30 (questionnaire for quality of life assessment in patients with cancer, Version 3.0) [3] was used to evaluate HRQoL. This questionnaire consists of 30 items and is designed for patients receiving cancer treatment regardless of cancer type and location. It measures five functional dimensions (physical, role, emotional, cognitive, and social), three symptom items (fatigue, nausea or vomiting, and pain), six single items (dyspnea, insomnia, appetite loss, constipation, diarrhea, and financial impact), and a global health status, which is the mean of two questions regarding overall health and overall quality of life. The BKAE ("Bewertung körperlicher Aktivität und Ernährung", in English "evaluation of physical activity and nutrition") is a questionnaire designed specifically for the LIBRE trials [47,48] to analyse attitudes and views on physical activity and dietary intake. It is based on the concept of "planned behaviour" by Fishbein & Ajzen (1975) [59] promoting that attitude, subjective norms and perceived behaviour control contribute to behavioural intention, which leads to actual behaviour. We only used the dietary information of the questionnaire for our analysis. The scores *BKAE-AT* (attitude towards healthy eating), *BKAE-SN* (subjective norms about healthy eating), *BKAE-PBC* (perceived behaviour control over healthy eating), *BKAE-IT* (intention to eat healthy in the future) and *BKAE-PB* (past behaviour with regard to healthy eating) range from 0 (minimum) to 100 (maximum). The physical activity part of the questionnaire has been evaluated previously; the strongest predictor for cardiopulmonary fitness was attitudes towards physical activity [60].

2.3. Statistical Analysis

SPSS Version 29.0.0 (IBM Corp., Armonk, NY, USA) was used to analyse data. Descriptive statistics are presented as mean ± standard deviation (SD) for continuous variables or as proportions for categorical variables. The distributions of continuous variables between diseased and non-diseased women were compared using Student's *t*-test. The distributions of categorical variables were compared using the Chi-square test. Linear regression models were created to detect associations between dietary intake and HRQoL. EORTC-QC30 scores were evaluated in diseased women only since the questionnaire was validated for cancer patients. Logistic regression models were performed to estimate odds ratios (ORs) and their associated 95% confidence intervals (95% CI) between MetS, dietary intake and different aspects of HRQoL. Multivariate analyses were carried out to control for potential confounding variables. These analyses were adjusted for body composition (BMI), physical fitness (VO_{2peak}), adherence to MD, and/or dietary inflammatory potential (DII). All *p* values were based on two-sided tests and were considered significant if $p \leq 0.05$.

2.4. Ethics

The study was approved by the ethics committees of both the host institutions Technical University of Munich (Reference No. 5685/13), the University Hospital Cologne (Reference No. 13-053), the University Hospital Schleswig-Holstein in Kiel (Reference No. B-235/13), and the participating study centres. Written consent from all study participants was obtained. All methods were carried out in accordance with relevant guidelines and regulations.

3. Results

A total of 312 women with a g*BRCA1* and/or g*BRCA2* mutation were included in the study. Table 1 summarizes the selected participants' characteristics by health status (diseased vs. non-diseased). The mean age of the entire study cohort was 43.5 years (SD ± 10.3 years). Of all the women, 59.6% had a previous diagnosis of cancer. Among these, breast cancer accounted for 88.7% and ovarian cancer for 7.0% of all cancer cases. Women with a history of breast cancer were older (46.5 years vs. 39.1 years, *p* < 0.001), more likely married (67% vs. 55%, *p* = 0.026), and less educated (high school diploma: 58% vs. 75%, *p* = 0.002). Diseased women had significantly lower hsCRP levels (1.7 vs. 3.3 mg/L, *p* = 0.045) and lower DII scores (−1.1 vs. −0.5, *p* = 0.011) compared to non-diseased women. Non-diseased mutation carriers had better physical fitness (17.3 vs. 16 mL/min/kg, *p* = 0.029), reported significantly higher quality of life (QL2 72.2 vs. 57.7, *p* = 0.041), role (RF 90.3 vs. 79.8, *p* < 0.001), cognitive (CF 82.5 vs. 72.9, *p* < 0.001) and social functioning (SF 85.2 vs. 72.0, *p* < 0.001), and experienced less pain (PA 15.7 vs. 25.6, *p* = 0.001), dyspnea (DY 9.6 vs. 16.1, *p* = 0.015), insomnia (SL 28.0 vs. 39.4, *p* = 0.003), and fewer financial difficulties (FI 4.3 vs. 18.5, *p* < 0.001). On the other hand, diseased mutation carriers reported stronger social norms about healthy eating (BKAE-SN 79.6 vs. 73.7, *p* = 0.008) and greater behavioural control over healthy eating (BKAE-PBC 86.9 vs. 84.2, *p* = 0.010). They also reported a more frequent consumption of healthy foods compared to women without a prior history of cancer (BKAE-PB 58.5 vs. 51.6; *p* = 0.008).

We then analysed associations between DII and various metabolic and lifestyle factors using linear regressions. The results are presented in Table 2. A lower DII score was significantly associated with higher adherence to MD (*p* < 0.001). Among diseased women, higher DII scores were associated with better role functioning (RF) (*p* = 0.032), cognitive functioning (CF) (*p* = 0.003), and social functioning (SF) (*p* = 0.012) as well as decreased fatigue (FA) (*p* = 0.046), dyspnea (DY) (*p* = 0.029) and appetite loss (AP) (*p* = 0.007).

Table 1. Baseline characteristics for participants with and without history of cancer.

Characteristic	Diseased	Non-Diseased	*p*-Value
n (%)	186 (59.6%)	126 (40.4%)	
Socio-demographic Data			
Age, years, mean ± SD	46.5 ± 9.2	39.1 ± 10.4	**<0.001 ***
Married, *n* (%)	(125) 67 %	70 (55%)	**0.026 ***
Number of children, mean ± SD	1.3 ± 0.9	1.1 ± 1.1	0.110
Education, *n* (%)			
● General university entrance qualification	104 (58%)	95 (75%)	**0.002 ***
● University degree	82 (44%)	68 (54%)	0.074
Net income, EUR, mean ± SD, *n*	4043.8 ± 1982.3; *n* = 126	3851.4 ± 2258.8; *n* = 84	0.515
Anthropometric Data			
BMI, kg/m^2, mean ± SD	25.6 ± 4.9	25 ± 5.5	0.301
Waist-to-hip ratio, mean ± SD	0.8 ± 0.1	0.8 ± 0.1	0.256
Metabolic Data			
Metabolic syndrome, *n* (%)	45 (24%)	26 (21%)	0.426
VO$_{2peak}$, mL/min/kg, mean ± SD	16.0 ± 5.0	17.3 ± 4.7	**0.029 ***
Nutritional Data			
DII, mean ± SD	−1.1 ± 1.8	−0.5 ± 1.2	**0.011 ***
MEDAS, mean ± SD	47.9 ± 16.6	46.5 ± 15.3	0.478
Psychological Data			
LOTR-O, mean ± SD	4.2 ± 2.9	4.1 ± 3.0	0.792
LOTR-P, mean ± SD	4.3 ± 2.1	4.0 ± 2.4	0.267
Quality of life (QL2), mean ± SD	67.7 ± 19.1	72.2 ± 8.6	**0.041 ***
Physical Functioning (PF2), mean ± SD	88.8 ± 12.6	91.4 ± 11.0	0.054
Role Functioning (RF2), mean ± SD	79.8 ± 24.0	90.3 ± 18.5	**<0.001 ***
Emotional Functioning (EF), mean ± SD	61.7 ± 27.3	62.6 ± 24.2	0.770
Cognitive Functioning (CF), mean ± SD	72.9 ± 25.9	82.5 ± 19.2	**<0.001 ***
Social Functioning (SF), mean ± SD	72.0 ± 30.0	85.2 ± 23.0	**<0.001 ***
Fatigue (FA), mean ± SD	33.6 ± 26.1	28.9 ± 19.9	0.085
Nausea and vomiting (NV), mean ± SD	3.9 ± 9.6	6.3 ± 14.9	0.096
Pain (PA), mean ± SD	25.6 + 28.3	15.7 ± 21.6	**0.001 ***
Dyspnea (DY), mean ± SD	16.1 ± 24.6	9.6 ± 20.7	**0.015 ***
Insomnia (SL), mean ± SD	39.4 ± 35.7	28.0 ± 29.8	**0.003 ***
Appetite loss (AP), mean ± SD	6.1 ± 16.9	4.0 ± 10.9	0.223
Constipation (CO), mean ± SD	10.0 ± 22.9	8.5 ± 20.3	0.543
Diarrhea (DI), mean ± SD	6.6 ± 15.8	11.5 ± 21.6	**0.033 ***
Financial difficulties (FI), mean ± SD	18.5 ± 29.0	4.3 ± 15.3	**<0.001 ***
BKAE-AT, mean ± SD	79.8 ± 6.8	78.9 ± 7.2	0.308
BKAE-SN, mean ± SD	79.6 ± 15.8	73.7 ± 18.7	**0.008 ***
BKAE-PBC, mean ± SD	86.9 ± 7.8	84.2 ± 10.7	**0.010 ***
BKAE-IT, mean ± SD	78.2 ± 9.8	76.2 ± 11.3	0.108
BKAE-PB, mean ± SD	58.5 ± 20.0	51.6 ± 23.0	**0.008 ***

* Results are statistically significant at a *p*-value of ≤0.05 (in bold).

Table 2. Associations between DII and metabolic, lifestyle and HRQoL factors using linear regression models.

Characteristic	Mean ± SD	Unadjusted Estimate [a] (95% CI)	Unadjusted *p*-Value [a]	Adjusted Estimate [b] (95% CI)	Adjusted *p*-Value [b]
MEDAS	47.3 ± 16.8	−2.340 (−3.579; −1.101)	**<0.001 ***	−2.266 (−3.520; −1.011)	**<0.001 ***
Role functioning (RF2) [1]	79.8 ± 24.0	0.012 (0.001; 0.023)	**0.032 ***	0.014 (0.003; 0.025)	**0.010 ***
Cognitive functioning (CF) [1]	72.9 ± 25.9	0.015 (0.005; 0.025)	**0.003 ***	0.016 (0.006; 0.026)	**0.002 ***
Social functioning (SF) [1]	72.0 ± 30.0	0.011 (0.002; 0.020)	**0.012 ***	0.013 (0.004; 0.021)	**0.005 ***
Fatigue (FA) [1]	33.6 ± 26.1	−0.010 (−0.020; 0.000)	**0.046 ***	−0.012 (−0.022; −0.002)	**0.017 ***
Pain (PA) [1]	25.6 ± 28.3	−0.009 (−0.018; 0.000)	0.057	−0.011 (−0.021; −0.002)	**0.017 ***
Dyspnea (DY) [1]	16.1 ± 24.6	−0.012 (−0.022; −0.001)	**0.029 ***	−0.016 (−0.027; −0.005)	**0.004 ***
Appetite loss (AP) [1]	6.1 ± 16.9	−0.021 (−0.036; −0.006)	**0.007 ***	−0.021 (−0.036; −0.006)	**0.008 ***

* Results are statistically significant at a *p*-value of ≤0.05 (in bold); [1] only for 'Diseased'; [a] univariate linear regression unadjusted for BMI, VO$_{2peak}$ and MEDAS; [b] multivariate linear regression adjusted for BMI, VO$_{2peak}$ and MEDAS.

Associations between adherence to MD (MEDAS) and various factors were carried out using linear regressions (see Table 3). Adherence to MD was associated with higher VO_{2peak} ($p = 0.024$), as well as lower DII scores ($p = <0.001$). Furthermore, adherence to MD was associated with dispositional optimism ($p = 0.001$).

Table 3. Associations between MEDAS and metabolic, lifestyle, and HRQoL factors using linear regression models.

Characteristic	Mean ± SD	Unadjusted Estimate [a] (95% CI)	Unadjusted *p*-Value [a]	Adjusted Estimate [b] (95% CI)	Adjusted *p*-Value [b]
VO_{2peak}, mL/min/kg	16.7 ± 4.9	0.005 (0.001; 0.009)	**0.014 ***	0.004 (0.000; 0.008)	0.053
DII	-0.9 ± 1.9	-0.018 (-0.028; -0.009)	**<0.001 ***	-0.017 (-0.027; -0.008)	**<0.001 ***
LOTR-O	4.2 ± 2.9	0.011 (0.004; 0.017)	**<0.001 ***	0.010 (0.004; 0.016)	**0.002 ***

* Results are statistically significant at a *p*-value of ≤0.05 (in bold); [a] univariate linear regression unadjusted for BMI, VO_{2peak} and DII; [b] multivariate linear regression adjusted for BMI, VO_{2peak} and DII.

We then carried out logistic regression models to estimate odds ratios (OR) and their associated 95% confidence intervals (95% CI) of having metabolic syndrome (MetS) by different dietary variables and different aspects of HRQoL (see Table 4). Higher adherence to MD (MEDAS ≥ 0.50) reduced odds for MetS (OR = 0.538, $p = 0.024$). Women with dispositional pessimism had increased odds for MetS (OR = 1.147, $p = 0.023$). Among diseased women, those who had poorer physical functioning (OR = 0.955, $p < 0.001$) or experienced more dyspnea (OR = 1.017, $p = 0.012$) had increased odds for MetS.

Table 4. Associations between MetS, lifestyle and HRQoL factors using logistic regression models.

Predictor	Unadjusted OR [a] (95% CI)	Unadjusted *p*-Value [a]	Adjusted OR [b] (95% CI)	Adjusted *p*-Value [b]
High adherence to MD (MEDAS ≥ 0.50) vs. low adherence to MD (<0.50)	0.538 (0.314; 0.922)	**0.024 ***	0.602 (0.343; 1.058)	0.078
LOTR-P	1.147 (1.019; 1.292)	**0.023 ***	1.150 (1.012; 1.307)	**0.032 ***
Physical functioning (PF2) [1]	0.955 (0.930; 0.980)	**<0.001 ***	0.963 (0.937; 0.990)	**0.007 ***
Dyspnea (DY) [1]	1.017 (1.004; 1.030)	**0.012 ***	1.013 (0.999; 1.026)	0.061

* Results are statistically significant at a *p*-value of ≤0.05 (in bold); [1] only for 'Diseased'; [a] univariate logistic regression unadjusted for VO_{2peak} and MEDAS; [b] multivariate logistic regression adjusted for VO_{2peak} and MEDAS.

4. Discussion

The aim of this analysis was to evaluate the relationship between anti-inflammatory diet, metabolic syndrome (MetS), and different aspects of health-related quality of life (HRQoL) in g*BRCA1/2* mutation carriers.

g*BRCA1/2* mutation carriers have a very high lifetime risk of developing breast and/or ovarian cancers. The average age of cancer diagnosis is substantially younger than in the general population (37). *BRCA*-associated cancers exhibit pathological features suggestive of an aggressive phenotype [61–63], and therefore, most patients undergo chemotherapy with detrimental side effects. When diagnosed with ER-positive breast cancer, patients might benefit from an extended adjuvant endocrine therapy [64,65], especially premenopausal women [66]. However, adjuvant endocrine therapy impacts HRQoL negatively [67]. Thus, identifying modifiable lifestyle factors to improve HRQoL is of particular relevance to g*BRCA1/2* mutation carriers, possibly resulting in better treatment adherence and (cancer-free) survival.

We observed that diseased women consumed a more anti-inflammatory diet compared to non-diseased women (DII -1.1 vs. -0.5, $p = 0.011$). Moreover, diseased participants perceived greater behavioural control over selecting healthier food options and were more likely to make healthier food choices than women without a history of cancer. This conforms to previous research that a breast cancer diagnosis can lead to beneficial dietary

changes [68]. The German breast cancer guideline issued by the German Association of the Scientific Medical Societies (AWMF) and the German Agency for Quality in Medicine (AeZQ) acknowledges the importance of lifestyle factors, such as diet and physical activity, in the aftercare of breast cancer patients. However, it was not until 2017 that the guideline included this recommendation [69]. The guideline suggests adhering to the dietary guidelines set by the German Society for Nutrition (DGE), which emphasize the consumption of plant-based foods such as cereal, grains, fruits, and vegetables as the foundation of a healthy diet, with small portions of animal products such as dairy, eggs, meat, and fish [70]. Compared to the MD, the consumption of olive oil, fish, seafood, and red wine is less emphasized in the DGE guidelines. As four items of the MEDAS focus on these food groups, it is possible that the lack of emphasis on them in the DGE guidelines may explain why diseased women in our study did not report a higher adherence to the MD compared to non-diseased women.

Of interest is the significant inverse association between adherence to MD and DII, indicating that MD is an anti-inflammatory diet (p = <0.001). In a prospective study, Hodge et al. (2016) identified MD as an anti-inflammatory diet that significantly reduced the risk of lung cancer [29]. The PREDIMED trial was the first randomized controlled trial to support these findings in a group of postmenopausal females; adherence to MD reduced the risk of breast cancer by 68% (95% CI 0.13–0.79) [69].

Porciello et al. [35] reported that adherence to MD in breast cancer survivors was associated with better HRQoL, i.e., improved physical functioning, better sleep quality and lower pain. We were not able to show an association between adherence to MD and HRQoL among diseased g*BRCA1/2* mutation carriers in our univariate and multivariate analyses. In our analysis, the median time from cancer diagnosis to study enrolment was four years (range: 1–48 years). To be eligible for participation in the LIBRE study, women had to be physically fit and functional, and several criteria that could hinder participation in the intervention program had to be excluded at study entry. These criteria included ongoing chemotherapy and/or radiation therapy, metastatic tumor disease, Karnofsky index below 60%, and exercise capacity below 50 watts. Consequently, our study participants were likely much fitter and more functional than those in Porciello et al.'s study, where women had to be diagnosed with breast cancer within the previous twelve months and had a mean age that was ten years older than our study participants.

In our study, adherence to MD was positively associated with dispositional optimism ($p \leq 0.001$). This finding is consistent with the results of a study by Ait-Hadad et al. [70], which investigated the relationship between dietary intake and dispositional optimism in a sample of over 32,000 participants. The authors reported a positive association between optimism and overall diet quality; high intake of fruits, vegetables, legumes, whole grains, seafood, and fats was positively associated with optimism, while high intake of meat and dairy products was negatively associated with optimism. Dispositional optimism is characterized as a general expectation or belief in positive outcomes in the future [71]. It has been associated with improved cardiovascular health and reduced all-cause as well as cause-specific mortality in large epidemiological studies [72,73]. Among breast cancer patients, optimism has been linked to psychological well-being and improved quality of life [74,75]. Boehm et al. found that dispositional optimism was associated with higher serum levels of antioxidants [76]. This association was partially influenced by dietary intake. Scheier and Carver [77] suggest that there are two underlying mechanisms linking optimism to health. Firstly, dispositional optimism facilitates the engagement in health promoting behaviours, i.e., diet and physical activity. Secondly, optimistic individuals better cope with adverse life events better than pessimistic individuals, which results in reduced stress levels and increased physiological wellbeing. Although dispositional optimism is considered to be relatively stable across one's lifespan, some studies found that cognitive therapy can increase optimism levels [78,79]. Since dispositional optimism and MD are linked in gBRCA1/2 mutation carriers, identifying further strategies to increase dispositional optimism might help to implement a healthy diet.

Moreover, adherence to MD was associated with reduced odds for MetS (OR = 0.54, p = 0.024). An Italian randomized controlled trial found that an MD-based dietary intervention in g*BRCA1/2* mutation carriers improved adherence to MD and reduced components of MetS [80]. Recent studies suggest that MetS is associated with impaired HRQoL [81–83]. Cohen et al. [84] reported a positive association between pessimism and the prevalence of MetS in patients with coronary heart disease. In our analysis, we found a positive association between MetS and dispositional pessimism (OR = 1.15, p = 0.023). In univariate analyses, MetS was associated with poorer physical functioning (OR = 0.96, p = <0.001) and higher levels of dyspnea (OR = 1.02; p = 0.012) among diseased women. However, these associations diminished following adjustment for physical fitness (VO_{2max}).

In women with a history of cancer, higher DII scores were associated with better role functioning (RF), cognitive functioning (CF), and social functioning (SF), as well as reduced fatigue (FA), reduced dyspnea (DY), and reduced appetite loss (AP). These findings were robust after adjustment for body composition (BMI), physical fitness (VO_{2peak}), and adherence to MD. To rule out that different types of cancer treatment or time since diagnosis influenced these associations, we calculated further multivariate regression models (see Appendix A). Our results were similar (see Tables A1–A3). This is surprising since it is contradictory to prior findings indicating that a more pro-inflammatory diet is associated with reduced HRQoL [28]. A possible explanation could be that diseased women with better HRQoL were not concerned about healthy eating. According to the theory of planned behaviour by Fishbein and Ajzen, behaviours are influenced by intentions, which are determined by three factors: attitudes, subjective norms, and perceived behavioural control [59]. To test our hypothesis, we adjusted our multivariate regression models for associations between DII and different dimensions of EORTC QLQ-C30 for the three core components of the Fishbein and Ajzen model, i.e., attitudes, subjective norms and perceived behavioural control. None of the three factors were associated with a pro-inflammatory diet nor did they influence the link between greater DII scores and reduced role, cognitive and social functioning (see Appendix A—Table A4). After inserting the variables attitudes, social norms and perceived behavioural control into the linear regression models for DII and fatigue, dyspnea and appetite loss, the models no longer reached significant levels (p = 0.059–0.143). Thus, in our analysis, the positive associations between pro-inflammatory diet patterns and HRQoL were likely not influenced by attitudes and beliefs towards healthy eating.

Strengths and Limitations

The strengths of the current study include the comprehensive evaluation of predictors that could be linked to HRQoL in g*BRCA1/2* mutation carriers. After adjusting for body composition, physical fitness and eating patterns, the adjusted and unadjusted results did not differ significantly. Therefore, any additional confounding was likely to be small. This supports our hypothesis that dietary intake is linked to different aspects of HRQoL. Although our results provide an interesting direction for HRQoL research, this study had several limitations. Firstly, the nature of a cross-sectional secondary analysis cannot establish a cause-and-effect relationship. The prospective nature of the LIBRE trials will allow for evaluating the impact of dietary changes on HRQoL. Secondly, as the number and type of food components to compute DII vary between studies, our results can hardly be compared to other populations of g*BRCA1/2* mutation carriers. Our study cohort was not representative for the average German population regarding education, net income, marital status, and parity [85–88]. Considering that our study cohort consisted of health-conscious females [89], the results obtained in this analysis likely underestimate the true associations between a pro-inflammatory diet and HRQoL outcomes. Finally, our cohort was not sufficiently powered to conduct analyses stratified by the g*BRCA* mutation type.

5. Conclusions

We were able to show that adherence to MD is linked to a more anti-inflammatory diet, dispositional optimism, and lower MetS prevalence among *gBRCA1/2* mutations carriers. Further research is needed to determine the long-term clinical implications of these findings.

Author Contributions: A.E. participated in the conceptualization of the study, was involved in the acquisition of data, carried out data analyses, and drafted the initial manuscript. J.L. designed and conceptualized this study, analysed and interpreted data, and critically revised the manuscript. L.N., S.G., S.C.B., M.H., M.S. and M.Y.-D. were involved in the acquisition and interpretation of patient data and contributed to the critical revision of the manuscript. M.K. conceived and supervised the study and acquired funding, and critically revised the manuscript. All authors have read and agreed to the published version of the manuscript.

Institutional Review Board Statement: The study was approved by the institutional ethics review boards of both the host institutions (Technical University of Munich: Reference No. 5686/13, University Hospital Cologne: Reference No. 13-053 and University Hospital Schleswig-Holstein in Kiel: Reference No. B-235/13) and the participating study centres. Trial registrations: NCT02087592; NCT02516540.

Informed Consent Statement: Written informed consent was obtained from all study subjects.

Data Availability Statement: Data are available upon reasonable request to the corresponding author.

Conflicts of Interest: The authors declare no conflict of interest.

Appendix A

Table A1. Characteristics of participants with a prior history of cancer (59.6%).

Characteristic	Mean $\pm$ SD/*n* (%)
Type of cancer	
• breast cancer (BC)	• 165 (88.7%)
• ovarian cancer (OC)	• 13 (7.0%)
• other	• 8 (4.3%)
Age at diagnosis, years	40.3 $\pm$ 9.0
Time since diagnosis, years	6.1 $\pm$ 6.9
Tumour biology of breast cancer	
• hormone receptor-positive	• 60 (36.3%)
• Her2-positive	• 6 (3.6%)
• triple-negative	• 85 (51.5%)
Breast cancer treatment	
• chemotherapy	• 138 (83.6%)
• chest radiation therapy	• 112 (67.9%)
• antihormonal treatment	• 65 (39.4%)
• HER2-targeted therapy	• 6 (3.6%)

Table A2. Associations between DII and EORTC-QC30 scores adjusted for different types of cancer treatment.

Characteristic		Estimates (95% CI) [a]	*p*-Value
Quality of life (QL2) [1]	unadjusted	0.006 (−0.008; 0.021)	0.395
	adjusted for 'chemotherapy'	0.006 (−0.008; 0.021)	0.392
	adjusted for 'chest radiation therapy'	0.006 (−0.008; 0.021)	0.391
	adjusted for 'antihormonal treatment'	0.006 (−0.008; 0.021)	0.380
	adjusted for 'HER2-targeted therapy'	0.006 (−0.008; 0.020)	0.409
Physical Functioning (PF2) [1]	unadjusted	−0.004 (−0.027; 0.019)	0.752
	adjusted for 'chemotherapy'	−0.004 (−0.027; 0.020)	0.761
	adjusted for 'chest radiation therapy'	−0.004 (−0.027; 0.020)	0.753
	adjusted for 'antihormonal treatment'	−0.004 (−0.027; 0.019)	0.749
	adjusted for 'HER2-targeted therapy'	−0.004 (−0.027; 0.019)	0.869
Role Functioning (RF2) [1]	unadjusted	0.012 (0.001; 0.024)	**0.037 ***
	adjusted for 'chemotherapy'	0.013 (0.001; 0.024)	**0.037 ***
	adjusted for 'chest radiation therapy'	0.013 (0.001; 0.026)	**0.031 ***
	adjusted for 'antihormonal treatment'	0.013 (0.001; 0.024)	**0.037 ***
	adjusted for 'HER2-targeted therapy'	0.013 (0.001; 0.024)	**0.034 ***
Emotional Functioning (EF) [1]	unadjusted	0.005 (−0.006; 0.015)	0.379
	adjusted for 'chemotherapy'	0.005 (−0.006; 0.015)	0.380
	adjusted for 'chest radiation therapy'	0.005 (−0.006; 0.015)	0.379
	adjusted for 'antihormonal treatment'	0.005 (−0.006; 0.015)	0.377
	adjusted for 'HER2-targeted therapy'	0.004 (−0.006; 0.015)	0.425
Cognitive Functioning (CF) [1]	unadjusted	0.015 (0.005; 0.026)	**0.005 ***
	adjusted for 'chemotherapy'	0.016 (0.005; 0.026)	**0.005 ***
	adjusted for 'chest radiation therapy'	0.016 (0.005; 0.026)	**0.005 ***
	adjusted for 'antihormonal treatment'	0.016 (0.005; 0.026)	**0.004 ***
	adjusted for 'HER2-targeted therapy'	0.015 (0.005; 0.026)	**0.004 ***
Social Functioning (SF) [1]	unadjusted	0.011 (0.001; 0.020)	**0.024 ***
	adjusted for 'chemotherapy'	0.011 (0.001; 0.020)	**0.024 ***
	adjusted for 'chest radiation therapy'	0.011 (0.002; 0.021)	**0.021 ***
	adjusted for 'antihormonal treatment'	0.011 (0.002; 0.021)	**0.023 ***
	adjusted for 'HER2-targeted therapy'	0.011 (0.002; 0.021)	**0.020 ***
Fatigue (FA) [1]	unadjusted	−0.010 (−0.021; 0.000)	0.057
	adjusted for 'chemotherapy'	−0.010 (−0.021; 0.000)	0.057
	adjusted for 'chest radiation therapy'	−0.010 (−0.021; 0.000)	0.058
	adjusted for 'antihormonal treatment'	−0.010 (−0.021; 0.000)	0.058
	adjusted for 'HER2-targeted therapy'	−0.010 (−0.020; 0.001)	0.070
Nausea and Vomiting (NV) [1]	unadjusted	−0.009 (−0.037; 0.020)	0.544
	adjusted for 'chemotherapy'	−0.009 (−0.038; 0.020)	0.539
	adjusted for 'chest radiation therapy'	−0.009 (−0.038; 0.020)	0.543
	adjusted for 'antihormonal treatment'	−0.009 (−0.038; 0.020)	0.540
	adjusted for 'HER2-targeted therapy'	−0.011 (−0.039; 0.018)	0.468
Pain (PA) [1]	unadjusted	−0.010 (−0.019; 0.000)	0.053
	adjusted for 'chemotherapy'	−0.010 (−0.019; 0.000)	0.054
	adjusted for 'chest radiation therapy'	−0.010 (−0.019; 0.000)	0.052
	adjusted for 'antihormonal treatment'	−0.009 (−0.019; 0.000)	0.055
	adjusted for 'HER2-targeted therapy'	−0.009 (−0.019; 0.000)	0.060
Dyspnea (DY) [1]	unadjusted	−0.010 (−0.021; −0.002)	0.089
	adjusted for 'chemotherapy'	−0.010 (−0.021; −0.002)	0.086
	adjusted for 'chest radiation therapy'	−0.010 (−0.021; −0.002)	0.089
	adjusted for 'antihormonal treatment'	−0.010 (−0.021; −0.002)	0.091
	adjusted for 'HER2-targeted therapy'	−0.010 (−0.021; −0.002)	0.087
Insomnia (SL) [1]	unadjusted	−0.007 (−0.015; 0.001)	0.088
	adjusted for 'chemotherapy'	−0.007 (−0.015; 0.001)	0.089
	adjusted for 'chest radiation therapy'	−0.007 (−0.015; 0.001)	0.088
	adjusted for 'antihormonal treatment'	−0.007 (−0.015; 0.001)	0.091
	adjusted for 'HER2-targeted therapy'	−0.007 (−0.014; 0.001)	0.094

Table A2. *Cont.*

Characteristic		Estimates (95% CI) [a]	*p*-Value
Appetite Loss (AP) [1]	unadjusted	−0.021 (−0.037; −0.006)	**0.010 ***
	adjusted for 'chemotherapy'	−0.021 (−0.037; −0.005)	**0.010 ***
	adjusted for 'chest radiation therapy'	−0.021 (−0.037; −0.005)	**0.010 ***
	adjusted for 'antihormonal treatment'	−0.021 (−0.037; −0.005)	**0.010 ***
	adjusted for 'HER2-targeted therapy'	−0.022 (−0.038; −0.006)	**0.08 ***
Constipation (CO) [1]	unadjusted	0.001 (−0.012; 0.013)	0.884
	adjusted for 'chemotherapy'	0.001 (−0.012; 0.013)	0.884
	adjusted for 'chest radiation therapy'	0.001 (−0.012; 0.013)	0.886
	adjusted for 'antihormonal treatment'	0.001 (−0.012; 0.013)	0.890
	adjusted for 'HER2-targeted therapy'	0.002 (−0.012; 0.013)	0.795
Diarrhea (DI) [1]	unadjusted	−0.007 (−0.025; 0.010)	0.451
	adjusted for 'chemotherapy'	−0.007 (−0.025; 0.010)	0.413
	adjusted for 'chest radiation therapy'	−0.007 (−0.025; 0.010)	0.416
	adjusted for 'antihormonal treatment'	−0.007 (−0.026; 0.010)	0.429
	adjusted for 'HER2-targeted therapy'	−0.007 (−0.025; 0.011)	0.423
Financial Difficulties (FI) [1]	unadjusted	−0.002 (−0.012; 0.007)	0.637
	adjusted for 'chemotherapy'	−0.002 (−0.012; 0.007)	0.637
	adjusted for 'chest radiation therapy'	−0.002 (−0.012; 0.007)	0.636
	adjusted for 'antihormonal treatment'	−0.002 (−0.012; 0.007)	0.619
	adjusted for 'HER2-targeted therapy'	−0.002 (−0.012; 0.008)	0.682

* Results are statistically significant at a *p*-value of ≤0.05 (in bold); [1] only for women with a prior history of breast cancer, [a] linear regressions models.

Table A3. Associations between DII and EORTC-QC30 scores adjusted for 'time since cancer diagnosis'.

Characteristic	Unadjusted Estimate [a] [95% CI]	Unadjusted *p*-Value [a]	Adjusted Estimate [b] (96% CI)	Adjusted *p*-Value [b]
Quality of Life (QL2) [1]	0.008 [−0.005; 0.022]	0.234	0.008 (−0.048; 0.034)	0.730
Physical Functioning (PF2) [1]	0.000 [−0.021; 0.020]	0.966	−0.002 (−0.025; 0.022)	0.896
Role Functioning (RF2) [1]	0.012 [0.001; 0.023]	**0.032 ***	0.014 (0.002; 0.026)	**0.026 ***
Emotional Functioning (EF) [1]	0.006 [−0.003; 0.016]	0.203	0.006 (−0.004; 0.016)	0.224
Cognitive Functioning (CF) [1]	0.015 [0.005; 0.025]	**0.003 ***	0.017 (0.006; 0.028)	**0.002 ***
Social Functioning (SF) [1]	0.011 [0.002; 0.020]	**0.012 ***	0.013 (0.004; 0.022)	**0.005 ***
Fatigue (FA) [1]	−0.010 [−0.020; 0.000]	**0.046 ***	−0.012 (−0.022; −0.001)	**0.031 ***
Nausea and Vomiting (NV) [1]	−0.003 [−0.031; 0.024]	0.802	−0.009 (−0.038; 0.020)	0.542
Pain (PA) [1]	−0.009 [−0.018; 0.000]	0.057	−0.010 (−0.020; −0.001)	**0.034 ***
Dyspnea (DY) [1]	−0.012 [−0.022; −0.001]	**0.029 ***	−0.014 (−0.025; −0.002)	**0.018 ***
Insomnia (SL) [1]	−0.006 [−0.014; 0.001]	0.095	−0.007 (−0.015; 0.001)	0.079
Appetite Loss (AP) [1]	−0.021 [−0.036; −0.006]	**0.007 ***	−0.026 (−0.042; −0.010)	**0.002 ***
Constipation (CO) [1]	0.000 [−0.011; 0.012]	0.999	−0.001 (−0.014; 0.012)	0.876
Diarrhea (DI) [1]	−0.008 [−0.025; 0.008]	0.331	−0.009 (−0.027; 0.009)	0.323
Financial Difficulties (FI) [1]	−0.006 [−0.015; 0.003]	0.217	−0.006 (−0.016; 0.004)	0.205

* Results are statistically significant at a *p*-value of ≤0.05 (in bold); [1] only for 'Diseased'; [a] univariate linear regression unadjusted for 'time since cancer diagnosis'; [b] multivariate linear regression adjusted for 'time since cancer diagnosis'.

Table A4. Associations between DII and EORTC-QC30 scores adjusted for attitudes towards, social norms about, and perceived behavioural control over healthy eating.

Characteristic	Mean ± SD	Unadjusted Estimate [a] (95% CI)	Unadjusted *p*-Value [a]	Adjusted Estimate [b] (96% CI)	Adjusted *p*-Value [b]
BKAE-AT	79.4 ± 7.0	−0.027 (−0.060; 0.005)	0.102	-	-
BKAE-SN	77.2 ± 17.3	−0.008 (−0.021; 0.006)	0.265	-	-
BKAE-PBC	85.8 ± 9.2	−0.010 (−0.034; 0.014)	0.394	-	-
Quality of life (QL2) [1]	67.7 ± 19.1	0.008 (−0.005; 0.022)	0.234	0.009 (−0.007; 0.025)	0.265
Physical Functioning (PF2) [1]	88.8 ± 12.6	0.000 (−0.021; 0.020)	0.966	−0.005 (−0.029; 0.019)	0.671
Role Functioning (RF2) [1]	79.8 ± 24.0	0.012 (0.001; 0.023)	**0.032 ***	0.014 (0.001; 0.027)	**0.040 ***
Emotional Functioning (EF) [1]	61.7 ± 27.3	0.006 (−0.003; 0.016)	0.203	0.008 (−0.003; 0.019)	0.148
Cognitive Functioning (CF) [1]	72.9 ± 25.9	0.015 (0.005; 0.025)	**0.003 ***	0.011 (0.000; 0.023)	**0.047 ***
Social Functioning (SF) [1]	72.0 ± 30.0	0.011 (0.002; 0.020)	**0.012 ***	0.014 (0.004; 0.024)	**0.005 ***
Fatigue (FA) [1]	33.6 ± 26.1	−0.010 (−0.020; 0.000)	**0.046 ***	−0.009 (−0.020; 0.003)	0.143
Nausea and Vomiting (NV) [1]	3.9 ± 9.6	−0.003 (−0.031; 0.024)	0.802	0.001 (−0.030; 0.033)	0.928
Pain (PA) [1]	25.6 ± 28.3	−0.009 (−0.018; 0.000)	0.057	−0.010 (−0.020; 0.001)	0.086
Dyspnea (DY) [1]	16.1 ± 24.6	−0.012 (−0.022; −0.001)	**0.029 ***	−0.010 (−0.022; 0.003)	0.131
Insomnia (SL) [1]	39.4 ± 35.7	−0.006 (−0.014; 0.001)	0.095	−0.004 (−0.012; 0.005)	0.411
Appetite Loss (AP) [1]	6.1 ± 16.9	−0.021 (−0.036; −0.006)	**0.007 ***	−0.019 (−0.039; 0.001)	0.059
Constipation (CO) [1]	10.0 ± 22.9	0.000 (−0.011; 0.012)	0.999	0.000 (−0.014; 0.014)	0.971
Diarrhea (DI) [1]	6.6 ± 15.8	−0.008 (−0.025; 0.008)	0.331	−0.010 (−0.028; 0.008)	0.284
Financial Difficulties (FI) [1]	18.5 ± 29.0	−0.006 (−0.015; 0.003)	0.217	−0.008 (−0.018; 0.002)	0.131

* Results are statistically significant at a *p*-value of ≤0.05 (in bold); [1] only for 'Diseased'; [a] univariate linear regression unadjusted for BKAE-AT, BKAE-SN and BKAE-PBC; [b] multivariate linear regression adjusted for BKAE-AT, BKAE-SN and BKAE-PBC.

References

1. Ewertz, M.; Jensen, A.B. Late effects of breast cancer treatment and potentials for rehabilitation. *Acta Oncol.* **2011**, *50*, 187–193. [CrossRef] [PubMed]
2. Sitlinger, A.; Zafar, S.Y. Health-Related Quality of Life: The Impact on Morbidity and Mortality. *Surg. Oncol. Clin. N. Am.* **2018**, *27*, 675–684. [CrossRef] [PubMed]
3. Aaronson, N.K.; Ahmedzai, S.; Bergman, B.; Bullinger, M.; Cull, A.; Duez, N.J.; Filiberti, A.; Flechtner, H.; Fleishman, S.B.; de Haes, J.C.; et al. The European Organization for Research and Treatment of Cancer QLQ-C30: A quality-of-life instrument for use in international clinical trials in oncology. *J. Natl. Cancer Inst.* **1993**, *85*, 365–376. [CrossRef] [PubMed]
4. Ware, J.E., Jr.; Sherbourne, C.D. The MOS 36-item short-form health survey (SF-36). I. Conceptual framework and item selection. *Med. Care* **1992**, *30*, 473–483. [CrossRef] [PubMed]
5. Chantzaras, A.; Yfantopoulos, J. Association between medication adherence and health-related quality of life of patients with diabetes. *Hormones* **2022**, *21*, 691–705. [CrossRef]
6. Park, J.; Rodriguez, J.L.; O'Brien, K.M.; Nichols, H.B.; Hodgson, M.E.; Weinberg, C.R.; Sandler, D.P. Health-related quality of life outcomes among breast cancer survivors. *Cancer* **2021**, *127*, 1114–1125. [CrossRef]
7. Carver, C.S.; Scheier, M.F. Dispositional optimism. *Trends Cogn. Sci.* **2014**, *18*, 293–299. [CrossRef]
8. Imayama, I.; Alfano, C.M.; Neuhouser, M.L.; George, S.M.; Wilder Smith, A.; Baumgartner, R.N.; Baumgartner, K.B.; Bernstein, L.; Wang, C.Y.; Duggan, C.; et al. Weight, inflammation, cancer-related symptoms and health related quality of life among breast cancer survivors. *Breast Cancer Res. Treat.* **2013**, *140*, 159–176. [CrossRef]
9. Sartori, A.C.; Vance, D.E.; Slater, L.Z.; Crowe, M. The impact of inflammation on cognitive function in older adults: Implications for healthcare practice and research. *J. Neurosci. Nurs.* **2012**, *44*, 206–217. [CrossRef]
10. Alfano, C.M.; Imayama, I.; Neuhouser, M.L.; Kiecolt-Glaser, J.K.; Smith, A.W.; Meeske, K.; McTiernan, A.; Bernstein, L.; Baumgartner, K.B.; Ulrich, C.M.; et al. Fatigue, inflammation, and ω-3 and ω-6 fatty acid intake among breast cancer survivors. *J. Clin. Oncol.* **2012**, *30*, 1280–1287. [CrossRef] [PubMed]
11. Panis, C.; Pavanelli, W.R. Cytokines as Mediators of Pain-Related Process in Breast Cancer. *Mediat. Inflamm.* **2015**, *2015*, 129034. [CrossRef] [PubMed]
12. Henry, N.L.; Pchejetski, D.; A'Hern, R.; Nguyen, A.T.; Charles, P.; Waxman, J.; Li, L.; Storniolo, A.M.; Hayes, D.F.; Flockhart, D.A.; et al. Inflammatory cytokines and aromatase inhibitor-associated musculoskeletal syndrome: A case-control study. *Br. J. Cancer* **2010**, *103*, 291–296. [CrossRef]
13. Pierce, B.L.; Ballard-Barbash, R.; Bernstein, L.; Baumgartner, R.N.; Neuhouser, M.L.; Wener, M.H.; Baumgartner, K.B.; Gilliland, F.D.; Sorensen, B.E.; McTiernan, A.; et al. Elevated biomarkers of inflammation are associated with reduced survival among breast cancer patients. *J. Clin. Oncol.* **2009**, *27*, 3437–3444. [CrossRef] [PubMed]
14. Villaseñor, A.; Flatt, S.W.; Marinac, C.; Natarajan, L.; Pierce, J.P.; Patterson, R.E. Postdiagnosis C-reactive protein and breast cancer survivorship: Findings from the WHEL study. *Cancer Epidemiol. Biomark. Prev.* **2014**, *23*, 189–199. [CrossRef]
15. Hanahan, D.; Weinberg, R.A. Hallmarks of cancer: The next generation. *Cell* **2011**, *144*, 646–674. [CrossRef] [PubMed]

16. Giugliano, D.; Ceriello, A.; Esposito, K. The effects of diet on inflammation: Emphasis on the metabolic syndrome. *J. Am. Coll. Cardiol.* **2006**, *48*, 677–685. [CrossRef]

17. Cui, X.; Jin, Y.; Singh, U.P.; Chumanevich, A.A.; Harmon, B.; Cavicchia, P.; Hofseth, A.B.; Kotakadi, V.; Poudyal, D.; Stroud, B.; et al. Suppression of DNA damage in human peripheral blood lymphocytes by a juice concentrate: A randomized, double-blind, placebo-controlled trial. *Mol. Nutr. Food Res.* **2012**, *56*, 666–670. [CrossRef]

18. Roager, H.M.; Vogt, J.K.; Kristensen, M.; Hansen, L.B.S.; Ibrügger, S.; Mærkedahl, R.B.; Bahl, M.I.; Lind, M.V.; Nielsen, R.L.; Frøkiær, H.; et al. Whole grain-rich diet reduces body weight and systemic low-grade inflammation without inducing major changes of the gut microbiome: A randomised cross-over trial. *Gut* **2019**, *68*, 83–93. [CrossRef]

19. Shivappa, N.; Steck, S.E.; Hurley, T.G.; Hussey, J.R.; Hébert, J.R. Designing and developing a literature-derived, population-based dietary inflammatory index. *Public Health Nutr.* **2014**, *17*, 1689–1696. [CrossRef]

20. Shivappa, N.; Godos, J.; Hébert, J.R.; Wirth, M.D.; Piuri, G.; Speciani, A.F.; Grosso, G. Dietary Inflammatory Index and Cardiovascular Risk and Mortality—A Meta-Analysis. *Nutrients* **2018**, *10*, 200. [CrossRef]

21. Zhang, X.; Guo, Y.; Yao, N.; Wang, L.; Sun, M.; Xu, X.; Yang, H.; Sun, Y.; Li, B. Association between dietary inflammatory index and metabolic syndrome: Analysis of the NHANES 2005–2016. *Front. Nutr.* **2022**, *9*, 991907. [CrossRef]

22. Vahid, F.; Shivappa, N.; Hatami, M.; Sadeghi, M.; Ameri, F.; Jamshidi Naeini, Y.; Hebert, J.R.; Davoodi, S.H. Association between Dietary Inflammatory Index (DII) and Risk of Breast Cancer: A Case-Control Study. *Asian Pac. J. Cancer Prev.* **2018**, *19*, 1215–1221. [CrossRef] [PubMed]

23. Shivappa, N.; Hébert, J.R.; Paddock, L.E.; Rodriguez-Rodriguez, L.; Olson, S.H.; Bandera, E.V. Dietary inflammatory index and ovarian cancer risk in a New Jersey case-control study. *Nutrition* **2018**, *46*, 78–82. [CrossRef]

24. Vahid, F.; Shivappa, N.; Faghfoori, Z.; Khodabakhshi, A.; Zayeri, F.; Hebert, J.R.; Davoodi, S.H. Validation of a Dietary Inflammatory Index (DII) and Association with Risk of Gastric Cancer: A Case-Control Study. *Asian Pac. J. Cancer Prev.* **2018**, *19*, 1471–1477. [CrossRef] [PubMed]

25. Shivappa, N.; Godos, J.; Hébert, J.R.; Wirth, M.D.; Piuri, G.; Speciani, A.F.; Grosso, G. Dietary Inflammatory Index and Colorectal Cancer Risk—A Meta-Analysis. *Nutrients* **2017**, *9*, 1043. [CrossRef] [PubMed]

26. Golmohammadi, M.; Kheirouri, S.; Ebrahimzadeh Attari, V.; Moludi, J.; Sulistyowati, R.; Nachvak, S.M.; Mostafaei, R.; Mansordehghan, M. Is there any association between dietary inflammatory index and quality of life? A systematic review. *Front Nutr.* **2022**, *9*, 1067468. [CrossRef]

27. Toopchizadeh, V.; Dolatkhah, N.; Aghamohammadi, D.; Rasouli, M.; Hashemian, M. Dietary inflammatory index is associated with pain intensity and some components of quality of life in patients with knee osteoarthritis. *BMC Res. Notes* **2020**, *13*, 448. [CrossRef]

28. Yaseri, M.; Alipoor, E.; Hafizi, N.; Maghsoudi-Nasab, S.; Shivappa, N.; Hebert, J.R.; Hosseinzadeh-Attar, M.J. Dietary Inflammatory Index Is a Better Determinant of Quality of Life Compared to Obesity Status in Patients with Hemodialysis. *J. Ren. Nutr.* **2021**, *31*, 313–319. [CrossRef] [PubMed]

29. Hodge, A.M.; Bassett, J.K.; Shivappa, N.; Hébert, J.R.; English, D.R.; Giles, G.G.; Severi, G. Dietary inflammatory index, Mediterranean diet score, and lung cancer: A prospective study. *Cancer Causes Control* **2016**, *27*, 907–917. [CrossRef] [PubMed]

30. Willett, W.C.; Sacks, F.; Trichopoulou, A.; Drescher, G.; Ferro-Luzzi, A.; Helsing, E.; Trichopoulos, D. Mediterranean diet pyramid: A cultural model for healthy eating. *Am. J. Clin. Nutr.* **1995**, *61*, 1402s–1406s. [CrossRef]

31. Martínez-González, M.A.; Gea, A.; Ruiz-Canela, M. The Mediterranean Diet and Cardiovascular Health. *Circ. Res.* **2019**, *124*, 779–798. [CrossRef] [PubMed]

32. Di Daniele, N.; Noce, A.; Vidiri, M.F.; Moriconi, E.; Marrone, G.; Annicchiarico-Petruzzelli, M.; D'Urso, G.; Tesauro, M.; Rovella, V.; De Lorenzo, A. Impact of Mediterranean diet on metabolic syndrome, cancer and longevity. *Oncotarget* **2017**, *8*, 8947–8979. [CrossRef] [PubMed]

33. Bonaccio, M.; Di Castelnuovo, A.; Bonanni, A.; Costanzo, S.; De Lucia, F.; Pounis, G.; Zito, F.; Donati, M.B.; de Gaetano, G.; Iacoviello, L. Adherence to a Mediterranean diet is associated with a better health-related quality of life: A possible role of high dietary antioxidant content. *BMJ Open* **2013**, *3*, e003003. [CrossRef] [PubMed]

34. Galilea-Zabalza, I.; Buil-Cosiales, P.; Salas-Salvadó, J.; Toledo, E.; Ortega-Azorín, C.; Díez-Espino, J.; Vázquez-Ruiz, Z.; Zomeño, M.D.; Vioque, J.; Martínez, J.A.; et al. Mediterranean diet and quality of life: Baseline cross-sectional analysis of the PREDIMED-PLUS trial. *PLoS ONE* **2018**, *13*, e0198974. [CrossRef]

35. Porciello, G.; Montagnese, C.; Crispo, A.; Grimaldi, M.; Libra, M.; Vitale, S.; Palumbo, E.; Pica, R.; Calabrese, I.; Cubisino, S.; et al. Mediterranean diet and quality of life in women treated for breast cancer: A baseline analysis of DEDiCa multicentre trial. *PLoS ONE* **2020**, *15*, e0239803. [CrossRef] [PubMed]

36. WHO. *WHO Report on Cancer: Setting Priorities, Investing Wisely and Providing Care for All*; World Health Organization: Geneva, Switzerland, 2020.

37. Kuchenbaecker, K.B.; Hopper, J.L.; Barnes, D.R.; Phillips, K.A.; Mooij, T.M.; Roos-Blom, M.J.; Jervis, S.; van Leeuwen, F.E.; Milne, R.L.; Andrieu, N.; et al. Risks of Breast, Ovarian, and Contralateral Breast Cancer for BRCA1 and BRCA2 Mutation Carriers. *JAMA* **2017**, *317*, 2402–2416. [CrossRef]

38. Shapiro, C.L.; Recht, A. Side Effects of Adjuvant Treatment of Breast Cancer. *N. Engl. J. Med.* **2001**, *344*, 1997–2008. [CrossRef]

39. Bouillon, K.; Haddy, N.; Delaloge, S.; Garbay, J.R.; Garsi, J.P.; Brindel, P.; Mousannif, A.; Lê, M.G.; Labbe, M.; Arriagada, R.; et al. Long-term cardiovascular mortality after radiotherapy for breast cancer. *J. Am. Coll. Cardiol.* **2011**, *57*, 445–452. [CrossRef]

40. Tao, J.J.; Visvanathan, K.; Wolff, A.C. Long term side effects of adjuvant chemotherapy in patients with early breast cancer. *Breast* **2015**, *24* (Suppl. S2), S149–S153. [CrossRef]

41. Ramchand, S.K.; Cheung, Y.M.; Yeo, B.; Grossmann, M. The effects of adjuvant endocrine therapy on bone health in women with breast cancer. *J. Endocrinol.* **2019**, *241*, R111–R124. [CrossRef]

42. Barish, R.; Gates, E.; Barac, A. Trastuzumab-Induced Cardiomyopathy. *Cardiol. Clin.* **2019**, *37*, 407–418. [CrossRef] [PubMed]

43. Schmidt, M.E.; Scherer, S.; Wiskemann, J.; Steindorf, K. Return to work after breast cancer: The role of treatment-related side effects and potential impact on quality of life. *Eur. J. Cancer Care* **2019**, *28*, e13051. [CrossRef]

44. Hickey, I.; Jha, S.; Wyld, L. The psychosexual effects of risk-reducing bilateral salpingo-oophorectomy in female BRCA1/2 mutation carriers: A systematic review of qualitative studies. *Gynecol. Oncol.* **2021**, *160*, 763–770. [CrossRef]

45. D'Alonzo, M.; Piva, E.; Pecchio, S.; Liberale, V.; Modaffari, P.; Ponzone, R.; Biglia, N. Satisfaction and Impact on Quality of Life of Clinical and Instrumental Surveillance and Prophylactic Surgery in BRCA-mutation Carriers. *Clin. Breast Cancer* **2018**, *18*, e1361–e1366. [CrossRef] [PubMed]

46. Kassianos, A.P.; Raats, M.M.; Gage, H.; Peacock, M. Quality of life and dietary changes among cancer patients: A systematic review. *Qual. Life Res.* **2015**, *24*, 705–719. [CrossRef]

47. Kiechle, M.; Engel, C.; Berling, A.; Hebestreit, K.; Bischoff, S.; Dukatz, R.; Gerber, W.D.; Siniatchkin, M.; Pfeifer, K.; Grill, S.; et al. Lifestyle intervention in BRCA1/2 mutation carriers: Study protocol for a prospective, randomized, controlled clinical feasibility trial (LIBRE-1 study). *Pilot Feasibil. Stud.* **2016**, *2*, 74. [CrossRef]

48. Kiechle, M.; Engel, C.; Berling, A.; Hebestreit, K.; Bischoff, S.C.; Dukatz, R.; Siniatchkin, M.; Pfeifer, K.; Grill, S.; Yahiaoui-Doktor, M.; et al. Effects of lifestyle intervention in BRCA1/2 mutation carriers on nutrition, BMI, and physical fitness (LIBRE study): Study protocol for a randomized controlled trial. *Trials* **2016**, *17*, 368. [CrossRef]

49. Schröder, H.; Fitó, M.; Estruch, R.; Martínez-González, M.A.; Corella, D.; Salas-Salvadó, J.; Lamuela-Raventós, R.; Ros, E.; Salaverría, I.; Fiol, M.; et al. A short screener is valid for assessing Mediterranean diet adherence among older Spanish men and women. *J. Nutr.* **2011**, *141*, 1140–1145. [CrossRef] [PubMed]

50. Martínez-González, M.; Corella, D.; Salas-Salvadó, J.; Ros, E.; Covas, M.I.; Fiol, M.; Wärnberg, J.; Arós, F.; Ruíz-Gutiérrez, V.; Lamuela-Raventós, R.M.; et al. Cohort profile: Design and methods of the PREDIMED study. *Int. J. Epidemiol.* **2012**, *41*, 377–385. [CrossRef]

51. Hebestreit, K.; Yahiaoui-Doktor, M.; Engel, C.; Vetter, W.; Siniatchkin, M.; Erickson, N.; Halle, M.; Kiechle, M.; Bischoff, S.C. Validation of the German version of the Mediterranean Diet Adherence Screener (MEDAS) questionnaire. *BMC Cancer* **2017**, *17*, 341. [CrossRef]

52. Seethaler, B.; Basrai, M.; Vetter, W.; Lehnert, K.; Engel, C.; Siniatchkin, M.; Halle, M.; Kiechle, M.; Bischoff, S.C. Fatty acid profiles in erythrocyte membranes following the Mediterranean diet—Data from a multicenter lifestyle intervention study in women with hereditary breast cancer (LIBRE). *Clin. Nutr.* **2020**, *39*, 2389–2398. [CrossRef]

53. Boeing, H.; Bohlscheid-Thomas, S.; Voss, S.; Schneeweiss, S.; Wahrendorf, J. The relative validity of vitamin intakes derived from a food frequency questionnaire compared to 24-hour recalls and biological measurements: Results from the EPIC pilot study in Germany. European Prospective Investigation into Cancer and Nutrition. *Int. J. Epidemiol.* **1997**, *26* (Suppl. S1), S82–S90. [CrossRef]

54. Bohlscheid-Thomas, S.; Hoting, I.; Boeing, H.; Wahrendorf, J. Reproducibility and relative validity of energy and macronutrient intake of a food frequency questionnaire developed for the German part of the EPIC project. European Prospective Investigation into Cancer and Nutrition. *Int. J. Epidemiol.* **1997**, *26* (Suppl. S1), S71–S81. [CrossRef]

55. Gholamalizadeh, M.; Afsharfar, M.; Fathi, S.; Tajadod, S.; Mohseni, G.K.; Shekari, S.; Vahid, F.; Doaei, S.; Shafaei Kachaei, H.; Majidi, N.; et al. Relationship between breast cancer and dietary inflammatory index; a case-control study. *Clin. Nutr. ESPEN* **2022**, *51*, 353–358. [CrossRef]

56. Jalali, S.; Shivappa, N.; Hébert, J.R.; Heidari, Z.; Hekmatdoost, A.; Rashidkhani, B. Dietary Inflammatory Index and Odds of Breast Cancer in a Case-Control Study from Iran. *Nutr. Cancer* **2018**, *70*, 1034–1042. [CrossRef]

57. Huang, W.Q.; Mo, X.F.; Ye, Y.B.; Shivappa, N.; Lin, F.Y.; Huang, J.; Hébert, J.R.; Yan, B.; Zhang, C.X. A higher Dietary Inflammatory Index score is associated with a higher risk of breast cancer among Chinese women: A case-control study. *Br. J. Nutr.* **2017**, *117*, 1358–1367. [CrossRef] [PubMed]

58. Glaesmer, H.; Hoyer, J.; Klotsche, J.; Herzberg, P.Y. Die deutsche Version des Life-Orientation-Tests (LOT-R) zum dispositionellen Optimismus und Pessimismus. *Z. Gesundh.* **2008**, *16*, 26–31. [CrossRef]

59. Flanders, N.A.; Fishbein, M.; Ajzen, I. *Belief, Attitude, Intention, and Behavior: An Introduction to Theory and Research*; Addison-Wesley Publishing Company: Boston, MA, USA, 1975.

60. Berling-Ernst, A.; Yahiaoui-Doktor, M.; Kiechle, M.; Engel, C.; Lammert, J.; Grill, S.; Dukatz, R.; Rhiem, K.; Baumann, F.T.; Bischoff, S.C.; et al. Predictors of cardiopulmonary fitness in cancer-affected and -unaffected women with a pathogenic germline variant in the genes BRCA1/2 (LIBRE-1). *Sci. Rep.* **2022**, *12*, 2907. [CrossRef]

61. Lee, E.; McKean-Cowdin, R.; Ma, H.; Spicer, D.V.; Van Den Berg, D.; Bernstein, L.; Ursin, G. Characteristics of triple-negative breast cancer in patients with a BRCA1 mutation: Results from a population-based study of young women. *J. Clin. Oncol.* **2011**, *29*, 4373–4380. [CrossRef] [PubMed]

62. Mavaddat, N.; Barrowdale, D.; Andrulis, I.L.; Domchek, S.M.; Eccles, D.; Nevanlinna, H.; Ramus, S.J.; Spurdle, A.; Robson, M.; Sherman, M.; et al. Pathology of breast and ovarian cancers among BRCA1 and BRCA2 mutation carriers: Results from the Consortium of Investigators of Modifiers of BRCA1/2 (CIMBA). *Cancer Epidemiol. Biomark. Prev.* **2012**, *21*, 134–147. [CrossRef] [PubMed]

63. Guzmán-Arocho, Y.D.; Rosenberg, S.M.; Garber, J.E.; Vardeh, H.; Poorvu, P.D.; Ruddy, K.J.; Kirkner, G.; Snow, C.; Tamimi, R.M.; Peppercorn, J.; et al. Clinicopathological features and BRCA1 and BRCA2 mutation status in a prospective cohort of young women with breast cancer. *Br. J. Cancer* **2022**, *126*, 302–309. [CrossRef] [PubMed]

64. Davies, C.; Pan, H.; Godwin, J.; Gray, R.; Arriagada, R.; Raina, V.; Abraham, M.; Medeiros Alencar, V.H.; Badran, A.; Bonfill, X.; et al. Long-term effects of continuing adjuvant tamoxifen to 10 years versus stopping at 5 years after diagnosis of oestrogen receptor-positive breast cancer: ATLAS, a randomised trial. *Lancet* **2013**, *381*, 805–816. [CrossRef] [PubMed]

65. Gray, R.G.; Rea, D.; Handley, K.; Bowden, S.J.; Perry, P.; Earl, H.M.; Poole, C.J.; Bates, T.; Chetiyawardana, S.; Dewar, J.A. *aTTom: Long-Term Effects of Continuing Adjuvant Tamoxifen to 10 Years versus Stopping at 5 Years in 6953 Women with Early Breast Cancer*; American Society of Clinical Oncology: Alexandria, VA, USA, 2013.

66. Burstein, H.; Griggs, J.; Prestrud, A.; Temin, S. American society of clinical oncology clinical practice guideline update on adjuvant endocrine therapy for women with hormone receptor-positive breast cancer. *J. Oncol. Pract.* **2010**, *5*, 243–246. [CrossRef] [PubMed]

67. Condorelli, R.; Vaz-Luis, I. Managing side effects in adjuvant endocrine therapy for breast cancer. *Expert Rev. Anticancer Ther.* **2018**, *18*, 1101–1112. [CrossRef] [PubMed]

68. Maunsell, E.; Drolet, M.; Brisson, J.; Robert, J.; Deschênes, L. Dietary change after breast cancer: Extent, predictors, and relation with psychological distress. *J. Clin. Oncol.* **2002**, *20*, 1017–1025. [CrossRef]

69. Leitlinienprogramm Onkologie (Deutsche Krebsgesellschaft, Deutsche Krebshilfe, AWMF): S3-Leitlinie Früherkennung, Diagnose, Therapie und Nachsorge des Mammakarzinoms, Version 4.0, 2017 AWMF Registernummer: 032-045OL. Available online: http://www.leitlinienprogramm-onkologie.de/leitlinien/mammakarzinom/ (accessed on 5 March 2023).

70. Deutsche Gesellschaft für Ernährung e. V., DGE-Ernährungskreis. 2019. Available online: https://www.dge-ernaehrungskreis.de/orientierungswerte/ (accessed on 5 March 2023).

71. Scheier, M.F.; Carver, C.S. Optimism, coping, and health: Assessment and implications of generalized outcome expectancies. *Health Psychol.* **1985**, *4*, 219–247. [CrossRef]

72. Tindle, H.A.; Chang, Y.F.; Kuller, L.H.; Manson, J.E.; Robinson, J.G.; Rosal, M.C.; Siegle, G.J.; Matthews, K.A. Optimism, cynical hostility, and incident coronary heart disease and mortality in the Women's Health Initiative. *Circulation* **2009**, *120*, 656–662. [CrossRef]

73. Kim, E.S.; Hagan, K.A.; Grodstein, F.; DeMeo, D.L.; De Vivo, I.; Kubzansky, L.D. Optimism and Cause-Specific Mortality: A Prospective Cohort Study. *Am. J. Epidemiol.* **2017**, *185*, 21–29. [CrossRef]

74. Friedman, L.C.; Kalidas, M.; Elledge, R.; Chang, J.; Romero, C.; Husain, I.; Dulay, M.F.; Liscum, K.R. Optimism, social support and psychosocial functioning among women with breast cancer. *Psychooncology* **2006**, *15*, 595–603. [CrossRef]

75. Thieme, M.; Einenkel, J.; Zenger, M.; Hinz, A. Optimism, pessimism and self-efficacy in female cancer patients. *Jpn. J. Clin. Oncol.* **2017**, *47*, 849–855. [CrossRef]

76. Boehm, J.K.; Williams, D.R.; Rimm, E.B.; Ryff, C.; Kubzansky, L.D. Association between optimism and serum antioxidants in the midlife in the United States study. *Psychosom. Med.* **2013**, *75*, 2–10. [CrossRef]

77. Scheier, M.F.; Carver, C.S. Dispositional optimism and physical health: A long look back, a quick look forward. *Am. Psychol.* **2018**, *73*, 1082–1094. [CrossRef] [PubMed]

78. Peters, M.L.; Flink, I.K.; Boersma, K.; Linton, S.J. Manipulating optimism: Can imagining a best possible self be used to increase positive future expectancies? *J. Posit. Psychol.* **2010**, *5*, 204–211. [CrossRef]

79. Meevissen, Y.M.C.; Peters, M.L.; Alberts, H.J.E.M. Become more optimistic by imagining a best possible self: Effects of a two week intervention. *J. Behav. Ther. Exp. Psychiatry* **2011**, *42*, 371–378. [CrossRef] [PubMed]

80. Bruno, E.; Manoukian, S.; Venturelli, E.; Oliverio, A.; Rovera, F.; Iula, G.; Morelli, D.; Peissel, B.; Azzolini, J.; Roveda, E.; et al. Adherence to Mediterranean Diet and Metabolic Syndrome in BRCA Mutation Carriers. *Integr. Cancer Ther.* **2018**, *17*, 153–160. [CrossRef] [PubMed]

81. Ford, E.S.; Li, C. Metabolic syndrome and health-related quality of life among U.S. adults. *Ann. Epidemiol.* **2008**, *18*, 165–171. [CrossRef]

82. Tziallas, D.; Kastanioti, C.; Kostapanos, M.S.; Skapinakis, P.; Elisaf, M.S.; Mavreas, V. The impact of the metabolic syndrome on health-related quality of life: A cross-sectional study in Greece. *Eur. J. Cardiovasc. Nurs.* **2012**, *11*, 297–303. [CrossRef]

83. Lee, H.; Kim, J.S. Factors Affecting the Health-Related Quality of Life of Cancer Survivors According to Metabolic Syndrome. *Cancer Nurs.* **2022**; *adhead of print*. [CrossRef]

84. Cohen, B.E.; Panguluri, P.; Na, B.; Whooley, M.A. Psychological risk factors and the metabolic syndrome in patients with coronary heart disease: Findings from the Heart and Soul Study. *Psychiatry Res.* **2010**, *175*, 133–137. [CrossRef]

85. Bundesamt, S. Bevölkerung Nach FamilienStand 2011 bis 2021 Deutschland. 2022. Available online: https://www.destatis.de/DE/Themen/Gesellschaft-Umwelt/Bevoelkerung/Bevoelkerungsstand/Tabellen/familienstand-jahre-5.html (accessed on 15 February 2023).

86. Bundesamt, S. Zusammengefasste Geburtenziffer nach Kalenderjahren. Available online: https://www.destatis.de/DE/Themen/Gesellschaft-Umwelt/Bevoelkerung/Geburten/Tabellen/geburtenziffer.html (accessed on 15 February 2023).

87. Bundesamt, S. Bevölkerung im Alter von 15 Jahren und Mehr Nach Allgemeinen und Beruflichen Bildungsabschlüssen nach Jahren. Available online: https://www.destatis.de/DE/Themen/Gesellschaft-Umwelt/Bildung-Forschung-Kultur/Bildungsstand/Tabellen/bildungsabschluss.html (accessed on 15 February 2023).
88. Bundesamt, S. Statistik zu Einkommen und Lebensbedingungen (Mikrozensus-Unterstichprobe zu Einkommen und Lebensbedingungen), Fachserie 15, Reihe 3, EU-SILC 2021. Statistisches Bundesamt: Wiesbaden, Germany, 2022; p. 23. Available online: https://www.destatis.de/DE/Themen/Gesellschaft-Umwelt/Einkommen-Konsum-Lebensbedingungen/Lebensbedingungen-Armutsgefaehrdung/Publikationen/Downloads-Lebensbedingungen/einkommen-lebensbedingungen-2150300217004.html (accessed on 15 February 2023).
89. Grill, S.; Yahiaoui-Doktor, M.; Dukatz, R.; Lammert, J.; Ullrich, M.; Engel, C.; Pfeifer, K.; Basrai, M.; Siniatchkin, M.; Schmidt, T.; et al. Smoking and physical inactivity increase cancer prevalence in BRCA-1 and BRCA-2 mutation carriers: Results from a retrospective observational analysis. *Arch. Gynecol. Obs.* **2017**, *296*, 1135–1144. [CrossRef]