Dietary Fat Intake, Serum Estrogen Level and Obesity as Risk Factors of Breast Cancer in Saudi Females: A Case-Control Study

Mostafa A. Abolfotouh1*, Omalkhair Abulkhair2, Suha E. Sbitan3, Fasih Ahmad4 and May N. Al-Muammar4

1King Abdullah International Medical Research Center (KAIMRC), King Saud bin-Abdulaziz University for Health Sciences, Riyadh, KSA.  
2King Abdulaziz Medical City, Riyadh, KSA.  
3King Saud bin Abdulaziz University for Health Sciences, Riyadh, KSA.  
4College of Applied Medical Sciences, King Saud University, Riyadh, KSA.

Authors’ contributions

This work was carried out in collaboration between all authors. Author MAA made the statistical analysis and wrote the final manuscript. Author OA recruited the patients and wrote the first draft of manuscript. Author SES collected the data and managed the literature searches. Authors FA and MNA designed the study and managed the analyses. All authors read and approved the final manuscript.

ABSTRACT

**Aims:** The aim of our study was to examine the associations of dietary fat intake, serum estrogen level and obesity with the risk of breast cancer in a case-control study among Saudi females including newly diagnosed breast cancer patients.  
**Study Design:** Case-control study.  
**Place and Duration of Study:** King Abdulaziz Medical City, Riyadh, Saudi Arabia, during the period between 1st February and 30th May, 2008  
**Methodology:** Dietary histories were collected 40 newly diagnosed female breast cancer cases and 82 randomly selected control subjects matched for age, parity, gravidity, number of children, breast feeding practice and age at marriage. A modified food frequency questionnaire (FFQ) was applied. Anthropometric measurements and blood tests that measured LDL, HDL, triglycerides (TGs) and estrogen levels were performed. Significance was considered at P≤0.05.

*Corresponding author: Email: mabolfotouh@gmail.com;*
**Results:** Breast cancer was significantly associated with overall obesity based on BMI (OR = 3.10, 95%, CI = 1.17–8.25, \(P=0.02\)) and central obesity based on WC (OR = 3.95, 95%, CI = 1.27–12.28, \(P=0.01\)). Cases exhibited significantly higher fat mass (39.6 vs. 36.9 kg, \(p=0.04\)) and significantly lower Fat intake (46.0±27.5 vs 59.0±38.9 g/day, \(P=0.034\)) than did the control group. The mean levels of TGs (2.9±1.1 vs 1.8±1.1 mmol/L, \(P<0.0001\)) and estradiol (131.0 vs 70.6 pmol/L, \(P≤0.008\)) were significantly higher in the study patients compared with the control subjects, whereas the mean level of low density lipoprotein (LDL-C) was significantly higher in the control subjects (3.1±0.8 vs. 1.6±1.0 mmol/L, \(P≤0.0001\)) compared with the study patients.

**Conclusion:** Both overall obesity and central obesity were significantly associated with breast cancer. Higher fat mass and lower fat intake and increased estrogen level were significantly associated with breast cancer cases. Further prospective studies on the Saudi population are recommended to explore the mechanisms of these findings.

**Keywords:** Breast cancer; estrogen; dietary fat; obesity; risk factors.

1. **INTRODUCTION**

Breast cancer is the most common cancer worldwide and is the leading type of cancer among women [1]. The breast cancer burden predictably will continue to increase in the coming years; there could be a nearly 50% increase in global incidence and mortality between the years 2002 and 2020 because of demographic changes alone [1-3].

The etiology of breast cancer is multifactorial, however, the exact cause of breast cancer is still unclear because the disease presumably represents a complex interplay of genetic susceptibility [4] and environmental factors, such as dietary fat intake [5], hazardous effects of hormonal exposures [6-9] and long-term use of hormone replacement therapy [10].

Number of abortions \(≥ 3\), family history of breast cancer, age at first live birth \(≥ 30\) (year), smoking, no live births, no breast feeding, age at menarche \(≤ 12\) (year), and alcohol drinking were among the priorities in the establishment of breast cancer risk assessment model for Asian women [11]. Recently, it has been hypothesized that overall caloric intake and obesity with weight gain are associated with increased breast cancer risk with different effects upon comparing pre- and postmenopausal women [12,13]. Obesity is known to be associated with excess mortality from all causes combined [14], but less is known concerning the magnitude of obesity-induced effects on cancer. In the KSA, studies have provided evidence that the prevalence of moderate obesity (BMI =30-40 kg/m\(^2\)) and severe obesity (BMI >40 kg/m\(^2\)) were 41.9% and 5.1%, respectively [15]. Furthermore, a health survey questioning both Saudi genders in the age range of 30 to 70 years demonstrated that females were significantly more obese compared with males (prevalence of 44% vs. 26.4%, respectively) [16].

Breast cancer is the most common cancer in Arab women [17]. Despite the relatively low incidence of breast cancer in the Kingdom of Saudi Arabia (KSA) compared with other countries, breast cancer in Saudi Arabia is the most common cancer in women [18]. The Saudi National Cancer Registry reported a rising proportion of breast cancer among cancer females of all ages, from 10.2% in 2000 to 24.3% in 2005 [19]. The average age at presentation of breast cancer in Arab countries is 48 years, which is a decade earlier than in western countries [20]. A recent study has estimated that the future burden of breast cancer in the KSA is expected to increase by almost 350% by the year 2025 [21]. Therefore, the aim of our study was to examine the associations of dietary fat intake, serum estrogen level and
obesity with the risk of breast cancer in a case-control study among Saudi females including newly diagnosed breast cancer patients.

2. MATERIALS AND METHODS

This is a case-control study of patients at the breast cancer clinic and the outpatient clinics of King Abdul-Aziz Medical city, National Guard Hospital, Riyadh, Saudi Arabia.

2.1 Subject Selection
An estimated sample size of 121 was derived from two-sided 95% confidence interval (α=0.025), a power of 0.90(β=0.10), a ratio of control subjects to cancer patients of 2 (r=2), and a difference between cases and control of 2 standard deviations (Z score=2), assuming that high fat intake is the potential risk factor of interest in the present study [22]. Therefore, the study involved all breast cancer Saudi female patients (age range: 25-60 years) who were newly diagnosed with breast cancer by histopathology during the periods between 1 February 2008 and 30 May 2008 (n=40). Control group consisted of 82 females who exhibited no clinical symptoms of the disease. One of the investigators was responsible for the recruitment of controls from the patients with minor ailments in different outpatient clinics by allocating those who match cases for age (±3 years), socioeconomic status, parity, gravidity, number of children, breastfeeding practice and age at marriage (±2.5 years). Then, a total of 82 patients were chosen randomly from those who were willing to participate in the study. After each patient was interviewed, corresponding control subjects were recruited within a 3-month period.

2.1.1 Exclusion criteria
Patients were excluded based on the following criteria: 1) a history of any malignancy or a palpable breast lump, 2) history of undergoing a bilateral ovariectomy, 3) chronic or acute liver disease, or 4) current pregnancy or lactation.

2.2 Data Collection

A. Questionnaires were used to collect information from the study patients and the control subjects. The information collected included the following:

i. Demographic data: Personal information, such as age, marital status, and family history of breast cancer, and information concerning risk factors, such as breast cancer status, use of hormones, supplements (e.g.; vitamins, minerals, herbs, etc.) and oral contraceptives (OCP), were collected.

ii. Dietary assessment and history: Food Frequency Questionnaire [23]. A previously validated food frequency questionnaire, in the National Nutrition Survey for Saudi Arabia [14], was applied. It includes food items most commonly found in the Saudi diet. The 40 food items listed in the questionnaire were: beef, lamb, poultry, fish, camel meat, goat meat, eggs, corn oil, olive oil, sunflower oil, ghee/samnah, butter, shaham, ghusdah, tahinah, other fats and oils, salt, pepper, sauces, vegetables, fruits, cow’s milk, laban milk, yoghurt, cheese, powdered milk, camel’s milk, goat’s milk, concentrated milk, sweetened condensed milk, milk with other flavour, tea with milk, coffee with milk, coffee without milk, bran flakes, corn, bread, rice, cakes, pie, pudding, ice cream, crème caramel. Food frequency questionnaire was
administered by interview using well-trained nutritionists. Intake frequencies were expressed as number of times each item was consumed per week. Food frequency data were crosschecked with a 3-day food record. Patients were asked about intake frequency and standard serving size for each food item. Interviews were conducted using food models to help respondents estimate the serving size.

To verify the accuracy of questions, the questionnaire was translated into Arabic with a back translation into English by two bilingual professionals. Diet analysis software (NSL Diet Analyser and WinDiet version 5, Robert Gordon University Garthdee AB10 7QE), and the Saudi food tables [23] were used to indirectly calculate nutrient consumption from the reported food intakes of the individuals.

B. Anthropometric measurements:

These measurements included the following details: height, weight, waist circumference, hip circumference and arm skinfold thickness. Body weight was measured to the nearest 0.1 kilograms (kg) using a beam. Height was measured, in duplicate, to the nearest 0.5 centimeters (cm) using a stadiometer. Patients were without head cover; they were bare footed with their feet placed together against the measuring board. Head was kept upright with eyes parallel to the ground. BMI was calculated using the following equation: BMI = weight (kg) ÷ height (m$^2$). Because most patients rejected the standard caliper, the body fat mass percentage was calculated by means of body fat pilot (Soehnle). Waist and hip circumferences were measured to the nearest 0.5 cm using standard measuring tape. Central obesity was considered with elevated WC ≥80 cm (female). [this is the European cut-off recommended by the IDF for use in East Mediterranean and Middle East (Arab) populations until more specific data are available] [24].

C. Blood tests:

The subjects were asked to fast for 12 hours before their blood samples were collected. None of the patients had treatment before blood samples were collected. Blood parameters were determined as follows: total cholesterol (TC), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL), triglycerides (TGs) and estrogen level. These parameters were estimated using enzymatic methods (Abbott Diagnostics USA).

TGs are enzymatically hydrolyzed by lipase into free fatty acids and glycerol, which is phosphorylated. Glycerol-3-phosphate is oxidized to create H$_2$O$_2$. In a peroxidase-catalyzed colorimetric reaction, H$_2$O$_2$ reacts with 4-aminoantipyrine (4-AAP) and 4-chlorophenol (4-CP) to produce a pink dye. The absorbance of this dye is directly proportional to the original concentration of the triglycerides present in the sample.

Estrogen levels were estimated in the blood samples using an estradiol assay; a one-step chemiluminescent microparticle immunoassay (CMIA) was used to determine the presence of estradiol in the human serum. For patients, blood parameter data were collected from the laboratory records, whereas blood samples were collected from each control subject and analyzed at the hospital laboratory to produce the blood parameter data.
2.3 Ethical Issues

This study protocol was approved by the institutional review board of King Saud University (KSU), Riyadh, Saudi Arabia, in partial fulfillment of a master's degree in clinical nutrition, in the College of Applied Medical Sciences, KSU. Written informed consent was obtained from participants before interview and any testing procedure. All participants had the right not to participate in the study or to withdraw from interview and/or measurements prior to completion. The study protocol received ethical approval from the Institutional Review Board of The Saudi National Guard Affairs, Riyadh, Saudi Arabia.

2.4 Data analysis

The data were analyzed using the Statistical Package for the Social Sciences (SPSS version 11.5). Categorical variables were presented as a percentage (%), whereas normally distributed continuous variables were presented as the mean ± standard deviation. Estradiol data were log transformed and presented as the median (interquartile range). Mann-Whitney U-test was utilized to compare continuous variables, and chi-squared test and Fisher’s exact test were used to compare frequencies. Significance was at a $P$-value $\leq 0.05$.

3. RESULTS

In Table 1, cancer case group showed a significantly higher proportion of subjects with a positive family history of breast cancer (20.0% vs. 6.1%, OR=3.8, $P=0.03$) and oral contraceptive use (57.5% vs. 15.9%, OR=7.04, $P<0.0001$) as well overweight and obesity (85.0% vs 64.6%, OR=3, 8.25, $P=0.02$) compared with the control group. However, there was no difference between the study patients and control subjects in supplement use (OR=2.11, $P=0.054$), eating a main meal (OR=0.53, $P=0.09$), consumption of fast food (OR=1.49, $P=0.33$), or eating between meals (OR=2.34, $P=0.14$).

Table 1. Demographics, physical activity and diet history comparisons

| Characteristics                      | Cases (N=40) | Controls (N=82) | OR (95% CI) | $P$-value |
|--------------------------------------|--------------|-----------------|-------------|-----------|
| Age in years (mean±SD)              | 46±8.5       | 44.8±5.9        | 0.30        |
| Family History of BC                |              |                 | 0.03        |           |
| Yes                                 | 8, (20.0)    | 5, (6.1)        | 3.80        |
| No                                  | 32, (80.0)   | 77, (93.9)      | (1.14-13.67)|           |
| Marital Status                      |              |                 | 0.99$^a$    |           |
| Single                              | 2, (5)       | 3, (3.6)        | 1.38        |
| Married/widow/divorced             | 38, (85)     | 79, (91.5)      | (0.16-9.65) |           |
| Breast feeding history              |              |                 | 0.97        |           |
| Yes                                 | 30, (83.3)   | 63, (85.1)      | 1.144       |
| No                                  | 6, (16.7)    | 11, (14.9)      | (0.36-3.39) |
| Oral Contraceptive use              |              |                 | < 0.0001    |           |
| Yes                                 | 23, (57.5)   | 13, (15.9)      | 7.04        |
| No                                  | 17, (42.5)   | 69, (84.1)      | (2.30-17.18)|           |
| Supplement use                      |              |                 | 0.054       |           |
| Yes                                 | 23, (57.5)   | 32, (37.8)      | 2.11        |
| No                                  | 17, (42.5)   | 50, (62.2)      | (0.98-4.56) |

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Table 1 continued …

|                          | Cases     | Controls   | P-Value |
|--------------------------|-----------|------------|---------|
| Eats main meal           | 16, (40.0)| 46, (52.0) | 0.53    |
| Yes                      | 24, (60.0)| 36, (48.0) | (0.24-1.13) |
| Consumes fast food       |           |            | 0.33    |
| Yes                      | 26, (66.7)| 47, (57.3) | 1.49    |
| No                       | 13, (33.3)| 35, (42.7) | (0.67, 3.37) |
| Eats between meals       |           |            | 0.14    |
| Yes                      | 36, (90.0)| 65, (79.3) | 2.34    |
| No                       | 4, (10.0) | 17, (20.7) | (0.76-8.65) |

Note: Data are presented as N, %; ( ) denotes ratio; significance at p <0.05.

@---Odds ratio represents the odds of association between breast cancer and overweight and/or obesity;
#Fisher’s exact test was applied.

3.1 Response to Food Frequency Questionnaire

In Table 2, intakes of energy were significantly lower in the study patients compared with the control subjects from fat (18.7±9.1% vs. 22.5±10.4%, p=0.044), fat intake (46.0±27.5 vs. 59.0±38.9 g/day, p=0.034), and monounsaturated fat intake (17.6±10.8 vs. 22.6±15.3 g/day, p=0.045).

Table 2. A case-control comparison of macronutrient and vitamin intake based on food frequency questionnaire

| Nutrients (units)                          | Cases (N=40) | Control (N=82) | P-Value* |
|-------------------------------------------|--------------|----------------|---------|
| Energy intake (kcal/day)                  | 2147± 791    | 2193± 868      | NS      |
| Energy distribution:                      |              |                |         |
| % Carbohydrate                            | 58.9± 12.6   | 57.1± 13.4     | NS      |
| % Fat                                     | 18.7± 9.1    | 22.5± 10.4     | 0.04    |
| % Protein                                 | 22.4± 7.6    | 20.2± 6.4      | NS      |
| Carbohydrate intake (g/day)               | 311± 124     | 304± 124       | NS      |
| Protein intake (g/day)                    | 121± 64.3    | 109.9± 50.4    | NS      |
| Fat intake (g/day)                        | 46.0± 27.5   | 59.0± 38.9     | 0.03    |
| Saturated fat (g/day)                     | 16.4± 11.7   | 22.6± 16.0     | NS      |
| Monounsaturated (g/day)                   | 17.6± 10.8   | 22.6± 15.3     | 0.045   |
| Polyunsaturated (g/day)                   | 12.0± 10.5   | 14.7± 12.5     | NS      |
| Cholesterol (mg/day)                      | 224± 128     | 242± 190       | NS      |
| Phytosterol (mg/day)                      | 27.0± 26.1   | 36.3± 33.9     | NS      |
| Fiber (g/day)                             | 11.8± 5.3    | 16.5± 6.3      | NS      |
| Vitamin A (µg)                            | 1730 ± 1658  | 1768 ± 1584    | NS      |
| Retinol (µg)                              | 189 ± 128    | 197 ± 127      | NS      |
| Vitamin C (mg)                            | 117 ± 90     | 82.3 ± 84.0    | NS      |
| Vitamin B1 (mg)                           | 4.2 ± 4.0    | 4.7 ± 4.4      | NS      |
| Vitamin B2 (mg)                           | 2.4 ± 1.7    | 2.3 ± 1.3      | NS      |
| Niacin (mg)                               | 65.6 ± 49.5  | 65.4 ± 55.7    | NS      |
| Vitamin B6 (mg)                           | 5.9 ± 5.4    | 6.5 ± 6.4      | NS      |
| Vitamin B12 (µg)                          | 5.3 ± 5.2    | 4.7 ± 4.6      | NS      |
| Folate (µg)                               | 377 ± 210    | 360 ± 208      | NS      |
| Pantothenic (mg)                          | 13 ± 10.3    | 14.0 ± 11.7    | NS      |
| Vitamin D (µg)                            | 6.6 ± 6.5    | 8.1 ± 7.8      | NS      |

Note: Data are presented as the mean ± standard deviation, *Student’s t-test was applied
3.2 Body and Laboratory Measurements

In Table 3, the study patients exhibited a significantly higher mean BMI (30.9±5.3 vs. 27.9±5.6 kg/m², p=0.005) and fat mass (39.6 vs. 36.9 kg, p=0.04), whereas mean height was significantly higher in the control subjects. (161.7±8.1 vs. 157.0±6.0 cm, p=0.002). The mean TG (2.9±1.1 vs. 1.8±1.1 mmol/L, p<0.0001) and estradiol levels (131.0 vs. 70.6 pmol/L, p=0.008) were significantly higher in the study patients, whereas the mean LDL-C level was significantly higher in the control subjects (3.1±0.8 vs. 1.6±1.0 mmol/L, p=0.0001). The mean HDL and TC levels were not significantly different between the study patients and the control subjects.

Table 3. Anthropometric measures and biochemical and estradiol levels among breast cancer case-control subjects

| Parameter                  | Cases (n=40) Mean ± SD | Controls (n=82) Mean ± SD | P-Value |
|----------------------------|------------------------|----------------------------|---------|
| Height (cm)                | 157.0 ± 6.0            | 161.7 ± 8.1                | 0.002   |
| Weight (kg)                | 76.1 ± 12.4            | 73.0 ± 15.0                | NS      |
| BMI (kg/m²)                | 30.9 ± 5.3             | 27.9 ± 5.6                 | 0.005   |
| Waist circumference (cm)   | 91.1 ± 17.5            | 88.6 ± 11.3                | NS      |
| Hip circumference (cm)     | 108.3 ± 19.7           | 109.3 ± 11.8               | NS      |
| Waist-Hip ratio            | 0.84 ± 0.12            | 0.81± 0.08                 | NS      |
| Fat mass (kg)              | 39.6 ± 6.5             | 36.9 ± 7.0                 | 0.04    |
| HDL-Cholesterol (mmol/L)   | 1.4 ± 0.71             | 1.4 ± 0.37                 | NS      |
| LDL-Cholesterol (mmol/L)   | 1.6 ± 1.0              | 3.1 ± 0.8                  | < 0.0001|
| Total Cholesterol (mmol/L) | 5.1 ± 1.3              | 5.0 ± 0.9                  | NS      |
| Triglycerides (mmol/L)     | 2.9 ± 1.1              | 1.8 ± 1.1                  | < 0.0001|
| Estradiol (pmol/L)#        | 131.0 (40.5-426.0)     | 70.6 (31.2-179.8)          | 0.008   |

Note: Data are presented as the mean ± standard deviation; # denotes a non-Gaussian distribution, and data are presented as the median (interquartile range).

Table 4 shows that cases were significantly more obese (85.0% vs 64.6%) and centrally obese (90.0% vs 69.5%) than control subjects, based on BMI (OR=3.10, 95%CI: 1.17-8.25), and WC (OR= 3.95, 95%CI: 1.27-12.28).

Table 4. Body composition of breast cancer cases and their control according to BMI and WC

|                  | Cases(n=40) | Control(n=82) | Statistical difference |
|------------------|-------------|---------------|-----------------------|
| **BMI levels**   |             |               |                       |
| <25kg/m² (normal)| 6 (15.0%)   | 29 (35.4%)    | χ²=5.45,df=1, P=0.02* |
| 25-<30kg/m²(overweight)| 10 (25.0%) | 26 (31.7%) |                       |
| >30kg/m² (obese) | 24 (60.0%)  | 27 (32.9%)    |                       |
| **WC levels**    |             |               |                       |
| <80 cm (normal)  | 4 (10.0%)   | 25 (30.5%)    | χ²=6.23, df,1, P=0.013 |
| ≥80 cm (centrally obese) | 36 (90.0%) | 57 (69.5%) |                       |
| Total            | 40 (100.0%) | 82 (100.0%)   |                       |

*overweight and obese subjects were added before applying the Chi-squared test.
BMI---body mass index, WC----waist circumference.
4. DISCUSSION

Previous data have demonstrated that there is an increase in breast cancer risk with increasing BMI among postmenopausal women, particularly those who consume high-fat food. It has been reported that there is a 3% increase in risk per 1 kg/m² increase in BMI [25]. The BMI of the study subjects (28.9 kg/m²) approximates that of a cross-sectional study of Saudi females, which reported a mean BMI of 29.2 kg/m² [15]. The mean BMI and body weight of the study patients were significantly higher compared with those of the control subjects. Moreover, study patients were significantly obese, based on BMI levels and centrally obese, based on waist circumference cut-off. Premenopausal weight gain during adult life increases the risk of breast cancer among postmenopausal women, whereas weight loss after menopause is associated with a decreased risk [26,27]. Meanwhile, there is an association between obesity (BMI) in premenopausal women and breast cancer risk; this association might explain the increased incidence of breast cancer at a younger age in Saudi Arabia, particularly because the incidence of obesity among Saudi females (age >15) is high.

The mechanism for the association between obesity and breast cancer risk may be partially attributed to an increase in the serum concentration of bioavailable estradiol, which causes both an increase in the production of estrogens by adipose tissue and a decrease in the serum concentration of sex hormone binding globulin (SHBG) [28]. Therefore, the increased risk of breast cancer of heavier postmenopausal women may be related to their having higher estrogen and lower SHBG levels compared with leaner women [29,30]. In this study, the mean estradiol level of the cancer patients was higher compared with that of the control subjects. This finding might explain the significant association between oral contraceptive use and breast cancer in the present study; breast cancer patients were sevenfold more likely to be contraceptive pill users compared with the control subjects.

As it relates to the effect of fat on breast cancer, it has been demonstrated that high fat intake raises endogenous estrogen levels. Body fat can produce estrogen that may fuel the growth of breast cancer cells that need hormones to grow [31]. In the present study, the mean fat mass of the study patients was significantly higher compared with that of the control subjects. This finding is in agreement with previous studies in which high saturated fat intake was reported to be associated with breast cancer [31]. This might explain the finding of significantly higher estradiol levels in cancer patients compared with control subjects.

Among premenopausal women, high intake of low fat dairy foods, especially skim/low-fat milk, was associated with reduced breast cancer risk, whereas high intake of animal fat, mainly from red meat and high-fat dairy foods, was associated with an increased risk [32]. Based on these data, early dietary guidelines have emphasized fat reduction for cancer prevention [33]. In our study, and based on FFQ, the case patients had significantly lower intake of energy from fat and lower fat intake compared with the control subjects. However, history of comorbidities that might have been possibly the cause behind the low fat intake among cancer females was not investigated in our study. This finding was in agreement with findings of previous cohort studies of fat intake and the risk of breast cancer [34]. It appears unlikely that a reduction in total fat consumption by middle-aged and older women will substantially reduce their risk of breast cancer. In the present study, the mean monounsaturated fat and phytosterol intake levels of the control subjects were higher compared with that of the study patients. This difference could be partially explained by the
intake of phytosterol rich foods, such as plant food items, that could prevent breast cancer [35].

The relationship between blood lipids and breast cancer risk is unclear. Until now, the reported results on the association between blood lipids and risk of breast cancer in women have conflicted. Thiebaut et al. [36], demonstrated no association between breast cancer risk and blood lipids. Epidemiological studies have suggested an association between lipids and cancer; various studies have demonstrated an association between lipid abnormalities and oncogenesis [37]. In the present study, TG levels were significantly higher in the patient cases compared with the control subjects; this finding was concordant with the findings of studies by Farid et al. [38] and Michalaki et al. [39] In contrast, HDL and TC levels were not different between both groups. These results were similar to the findings of previous studies [37]. High TC and TG levels have been reported in the tissue of malignant breast tumors compared with benign tumors [40].

Although scientific evidence supports the benefits of lowering low-density lipoprotein cholesterol (LDL-C) to help prevent heart disease, previous studies of cholesterol-lowering drugs have suggested a strong association between low LDL-C levels and cancer risk [41]. In the present study, mean LDL levels were significantly lower in the study patients compared with the control subjects. However, this finding suggests there may be some underlying mechanism that affects both LDL cholesterol levels and cancer risk.

The present study may be limited by its case-control study design, which is subject to recall and selection biases. Dietary assessment based on individual interview may introduce measurement errors and attenuate the association between variables. Although this study included both pre- and postmenopausal women, the total number of subjects was insufficient to allow us to conduct further sub-analyses to investigate the association between the breast cancer risk factors in pre- and postmenopausal women. Finally, the association between breast cancer and other variables such as; generalized obesity, central obesity, estrogen level and fat mass may be subjected to some possible confounders for which the smaller number of cancer cases wouldn’t allow for adjustment.

5. CONCLUSION

In conclusion, this study demonstrated that obesity, a high-fat mass and increased estrogen levels were significantly associated with breast cancer. Further prospective large-scale studies on Saudi females are recommended to draw a strong conclusion.

CONSENT

All authors declare that written informed consent was obtained from the patient (or other approved parties) for publication of this study.

ETHICAL APPROVAL

All authors hereby declare that all experiments have been examined and approved by the appropriate ethics committee and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.
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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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