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Research Article

Association of Total Dietary Fats and Its Subtypes with Risk of Breast Cancer

Jwan I. Jawzali*

Department of Nursing-Basic Science, Nursing College, Hawler Medical University, Erbil, Iraq

ABSTRACT

Specific classes of dietary fatty acids may be important modifiers of breast cancer (B-Ca) risk. Aim of this study was identification risk of subtypes of dietary fat for B-Ca**.** This case–control study carried out in Rizgary Teaching Hospital in Erbil city. Data collected by interview questionnaire and included demographic and reproductive properties; anthropometric measurements; and medical history. Dietary data collected by food frequency questionnaire. They were analyzed by program for Mosby's Nutritrac Nutrition Analysis Software, for calculation intake of dietary; fiber, total fat, and its subtypes, energy intake, acceptable macronutrient distribution range, and antioxidant nutrients. Statistical analysis was performed using SPSS program. Polyunsaturated fats decreased risk of B-Ca while saturated and monounsaturated fats (Cis form) increased risk among all and postmenopause obese women, respectively. Risk of cancer increased significantly in high percentage of energy intake from monounsaturated fats**,** cooking oil, and dietary red meats. The study concluded that total polyunsaturated fatty acids (PUFA) decrease risk of B-Ca among obese menopause woman. Increase risk of B-Ca by cooking oils and animal origin diet may due to increased intake of saturated monounsaturated and specific PUFA. These subtypes of dietary fats may promote hormones imbalance and inflammation.

Keywords: Animal origin diet, breast cancer, monounsaturated fats, saturated fats, unsaturated fats

INTRODUCTION

Breast cancer (B-Ca) is the most frequent cancer among women in Kurdistan/Iraq and increased in the past 10 years. It has been reported that 40% of cancers can be averted by reducing risk factors and by primary prevention.[1] Role of dietary fat could result in initiation and development of breast tumor by stimulation production of estrogen and other endogenous hormone, regulation of immune function, and modulation of gene expression.[2]

Diet may modify risk of B-Ca. Specific classes of dietary fatty acids may be important modifiers of B-Ca risk. Unique chemical and biophysical properties of dietary fatty acids have impact on health and disease.[3] The inconsistent and limited evidence warrants research to assess the impact of consumption of fat subtypes on B-Ca recurrence and mortality.[4] Epidemiologic, animal experiments, and some case–control studies supported the hypothesis that dietary fat can increase B-Ca risk, and still remains one of the most controversial hypotheses in nutritional epidemiology.[5] Inconsistent results due to limited ranges of fat intake among populations, errors of dietary fat measurement, and high correlation between specific types of dietary fat and B-Ca risk. The confounders such as body weight and huge energy intake and food components such as fiber and antioxidants can affect the exact association of total and specific subtypes of fat intake with B-Ca risk.[2]

Fatty acids potentially associated with an increase in cancer risk in humans include saturated fatty acids (SFA), monounsaturated fatty acids (MUFA), and trans-fatty acids, and two major classes of polyunsaturated fatty acids (PUFA); omega-6 PUFA, (PUFA 18:2n6) and arachidonic acid (AA, 20:4n6). In contrast, specific PUFA, n-3 PUFA (α-linolenic acid), may have anticancer effects. Studies have shown that SFAs associated with an increased risk in contrast omega-3 PUFA intake reduced risk of B-Ca.[3]

PUFAs are biologically active food components that elicit pro- and anti-inflammatory responses through several signaling pathways that regulate cell proliferation, apoptosis, and angiogenesis. There is increasing evidence that PUFAs play a role in cancer risk and progression.[6]

Corresponding Author:
Jwan I. Jawzali, Department of Nursing-Basic Science, Nursing College, Hawler Medical University, Erbil, Iraq.
E-mail: jwanjawzali@gmail.com

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Epidemiologic studies provide unpredictable reports about the associations of dietary PUFAs and risks of cancer. The previous reviews evaluate overall dietary fat but do not take in to account the variation in the impact of fat subtypes. Dietary fat risk to the B-Ca may due to the complex composition of dietary fat. Therefore, this study conducted to identify contribution of some subclasses of dietary fat to total energy and their risk to B-Ca. The study also identify socio demographic and lifestyle factors which act as confounder and some antioxidant nutrients that modify the association of dietary fats and its subtype with risk of B-Ca.

PATIENTS AND METHODS

The study was case control, carried out in Rizgary Teaching Hospital in Erbil governorate, Kurdistan region, Iraq. The patients age ranged between 27 and 74 years and included 55 cases of women with diagnosed B-Ca (after mastectomy), at different stage, attending outpatient unit of chemotherapy and 55 cancer-free women attending outpatient clinic of Rizgary Teaching Hospital. Sample size determined by sample size mode[7] using odds ratio (OR) and percentage of controls exposed to total fats from previous case–control study conducted in the same hospital. Women whose weighs changed after diagnoses and women who did not complete the interview questionnaire were excluded from the study.

They were interviewed after their consent had been taken by questionnaire, which included sociodemographic properties; reproductive history, family history of B-Ca, anthropometric measurements; body mass index and waist-to-hip ratio, and medical history. Diet history collected by semi-quantitative food frequency questionnaire to estimate; type, frequency, and quantity of food using household measurements and standard portion size photographs of common foods.

Foods intake data were analyzed by program for Mosby's Nutritrac Nutrition Analysis Software, version IV (CD-ROM), for calculation; total energy, dietary fat intake and its subtypes, cholesterol, dietary fibers, and some antioxidants per individual by, taking into account intake of margarine, butter, oils, and fats used in cooking and baking. Calculating amount of omega 6 in cooking oil by RightTrak Nutrient Calculator. Acceptable macronutrient distribution range (AMDR) represents the percentage of energy contributed by total fat calculated. Food groups contributing to intake of fat, red meat, chicken, fish, and eggs, were used in association with risk of B-Ca.

Statistical Analysis

All data were analyzed by SPSS version 22.0. Descriptive statistics were used to study the mean and standard deviation and standard error for continuous variables. Chi-square test was used for association measurements between categories, independents *t*-test used for measurement differences between the means. Odds ratio (OR), and binary logistic regression were used in measuring the risk of B-Ca. *P*-value was considered significant when $P \leq 0.05$ and was considered highly significant when $P \leq 0.01$.

RESULTS AND DISCUSSION

Table 1 shows demographic reproductive properties of the studied samples. There was no significant difference between B-Ca and control groups in age, residency, marital status, education levels, age of menarche, age at first pregnancy, and menopause age.

Table 2 shows no significant difference in anthropometric measurement between B-Ca and controls. Most were overweight and had normal value (0.8–0.85) of waist-to-hip ratio. In contrast to familial absence of cancer, this showed significant association and decrease in the risk (OR=0.14**) and confident interval (CI) ranged to decrease (0.04–0.44) risk of B-Ca and indicates to the importance of genetic factors, and increasing rate of B-Ca incidence in Iraq. This view the same results of the previous studies conducted in Iraq by Lafta *et al.* and Safil *et al.*[8,9] Nearly 60% of the studied women had family history of B-Ca it is much higher than that reported in the study[10] where only 16% of the patients recorded a positive family history.

Table 3 shows intake of daily energy as kilocalorie (Kcal./d), daily total fats and its subtypes as gram/day (g/d), their AMDR, dietary fiber (g/d), and some antioxidant nutrients. There was a significant difference between means of saturated fats, unsaturated, and selenium as antioxidants. The results reveal the roles of subtypes of fats on the risk of B-Ca.

The most important one was PUFA which represent mainly total form of two major classes of (PUFA); omega-6 and omega 3. Interaction with other subtype fats and antioxidants, and their AMDR in all samples and largely among overweight and obese patients, decreased risk of B-Ca. Increased risk of B-Ca was found among group with high intake of saturated fats more than 10% of total calories and ≥20 g/day of normal value of saturated fat intake, and among obese post-menopause women who had high percentage of energy intake from MUFA. The finding supported by Sieri *et al*.[11] who found positive association between B-Ca and saturated fat and was more pronounced among postmenopausal women who never used hormone therapy. Higher total fat intake (39% of total energy) than recommendations ensures limitation total fat intake between 20% and 35% of total caloric and restriction saturated fat consumption as stated by Makarem *et al*.[4] Furthermore, Canadian cohort study showed that higher intakes of saturated fat as a percentage of total energy significantly increased the risk of B-Ca death in postmenopausal women and not significantly in premenopausal women. Every 5% increase in saturated fat intake, as percentage of total energy, was associated with approximately 65% increased risk of B-Ca mortality.[4] Otherwise, Zhang *et al*.[12] suggested that intakes of total fat, saturated and monounsaturated fat, and *n*-3 and *n*-6 polyunsaturated fat were not associated with risk of B-Ca.

Antioxidant nutrients (Vitamins C, E, and selenium) and their modulation effect appeared only with selenium that showed significant association with B-Ca. This result is in line with Pouchieu *et al*.[13] who suggests that specific SFAs, MUFAs, and PUFAs were prospectively differentially associated with cancer risk. Antioxidants may modulate these associations with interaction with other subtype fats and counteracting the potential effects of these fatty acids on carcinogenesis.

Significant high risk of MUFA which occupies high percentage from total energy among obese patient and decreased risk by polyunsaturated and its AMDR mainly among obese postmenopause women is related to body fatness

Table 1: Comparison of sociodemographic characteristics for cases and controls and their association with B-Ca

Demographic data	**B-Ca cases**	**Controls**	***P*-value of *t*-test**
	Mean±SD	**Mean±SD**	
Age (years)	47.6±10.0	47.1±11.5	0.74
Residency	**F %**	**F %**	***P*-value Chi-square**
Urban	30 (54.5)	38 (69.1)	0.169
Rural	25 (45.5)	17 (30.9)	
Educational levels	**F (%)**	**F (%)**	***P*-value Chi-square**
Illiterate	38 (69.1)	30 (54.)5	0.068
Primary school	8 (10.9)	13 (23.6)	
Secondary school	6 (14.5)	4 (7.3)	
Institute and above	3 (5.5)	8 (14.5)	
Marital status	**F (%)**	**F (%)**	***P*-value Chi-square**
Married	48 (87.3)	49 (89.1)	0.77
Single	7 (12.7)	6 (10.9)	
	Mean±SD	**Mean±SD**	***P*-value *t*-test**
Menarche age	13.4±1.5	13.5±0.68	1.4
Age at first pregnancy	16.8±10.8	18.2±9.5	0.48
Menopause age	43.6±7.6	43.7±9	0.97

F.: Frequency, ***P*-value ≤0.01, **P*-value ≤0.01

Table 2: Comparisons of anthropometric factors and familial history between cases and controls

Anthropometric	**B-Ca cases**	**Controls**	***P*-value–*t*-test**
	Mean±SD	**Mean±SD**	
BMI (Kg/m^2)	31.7±5.5	30.2±4.6	0.13
Waist/hip ratio	0.84±0.06	0.83±0.07	0.89
BMI category	**F. (%)**	**F. (%)**	
< 25	5 (9.1)	5 (9.1)	
25-29	18 (32.7)	20 (36.4)	
> 30	32 (58.2)	30 (54.5)	
Family history	**F. (%)**	**F (%)**	***P*-value of Chi-square**
Negative	35 (63.6)	51 (92.7)	0.00**
Positive	20 (36.4)	4 (7.3)	

F: Frequency, **P≤0.01, *P≤0.01

and estrogen hormone production. This result reveals that the idea of other case–control studies has shown a positive role of MUFA in the pathogenesis of B-Ca and that body adiposity enhances the production of estrogen, hormonal imbalances in insulin-like growth factor-1 and insulin. Increased bioavailable estrogens that are theorized to increase cell proliferation through activation of target genes, thereby promoting carcinogenesis. Makarem *et al.*, Singh and Nimbkar[4,14] concluded that reduction of dietary fat would result in a 2.5-fold reduced risk of B-Ca among postmenopausal women.

Table 4 shows significant differences in the mean levels of cooking oils, dietary red meats, poultry meats, and dietary egg between B-Ca and controls. This may due to content of cooking oils (largely sunflower and corn oils intake that was abundant in studied samples) of omega-6. Effectively sunflower oils showed approximately significant difference between B-Ca and controls. Highest percentage 77% of sunflower oil and 50% of corn oil composed of omega 6 (*n*3:6) linoleic unsaturated fatty acids.[15] This result explores pro-inflammatory effect of omega 6 that increases risk of B-Ca as stated by Prentice and Sheppard[16] who concluded increased B-Ca risk with increasing dietary ratio of pro-inflammatory (*n*-6 PUFA) to anti-inflammatory (*n*-3 PUFAs) and increases in pro-inflammatory eicosanoids such as prostaglandin E2, promotes angiogenesis, and hinders apoptosis.

In addition, red meats may contain other carcinogenic compounds formed during meat process and preparing that increase risk of B-Ca as stated by Khodarahmi and Azadbakht[2] that carcinogens such as heterocyclic amines, N-nitroso compound, and polycyclic aromatic hydrocarbons, which are found largely in cooked red meat and soluble hormones or growth factor, could increase risk of B-Ca and they may confound the exact relationship of the subtypes of fat with B-Ca.

Significant difference between cases and controls in dietary egg intake may indicate to its association with risk of B-Ca and it is in line with[17] that high egg intake may be associated with elevated risk of B-Ca. Also agree with Cho *et al.*[18] who attributed association of high egg intake with risk of B-Ca to estrogen hormone levels and concluded that animal fat intake increase B-Ca risk by modulating estrogen levels among women with estrogen receptor (ER)-positive cancers compare to women with ER-negative cancers.

Significant difference in poultry meats intake among B-Ca and controls conforms result of Chandran *et al.*[19] who found positive association between poultry consumption and B-Ca risk in Caucasian women, with over 2-fold greater risk

Table 3: Comparisons of energy, fats, and antioxidants intakes between cases and controls and their risk to B-Ca

Nutrients		B-Ca cases	Controls	*P*-value *t*-test	OR	95% CI
		Mean±SE	Mean±SE			
Total energy (Kcal/day)		4653.3±1212.9	3019.1±641.2	0.23		
Total fat		137.8±49.4	85.2±13.9	0.31		
% Kcal from total fat		39.4±7.6	32.5±1.8	0.32		
Saturated fat intake		**F (%)**	**F (%)**	***P*-value of Chi-square**		
Categories 7–10% of calorie is normal	<7% low	18 (32.7)	29 (52.7)	0.01**	Low intake of SF is reference	
	Normal	11 (20.0)	15 (27.3)		3.8**[a]	1.5–9.5
	>10% high	26 (47.3)	11 (20.0)		3.2*	1.13–9.2
		Mean±SE	Mean±SE	*P*-value *t*-test	OR	95% CI
Saturated fat (SF) (g/d)		58±32.2	18.3±3.0			
% Kcal from saturated fat		10.8±1.6	7.9±0.6			
Monounsaturated (g/d)		53.7±13.6	40.2±4.6	0.35		
% Kcal from MAFA		19.4± 4.7	18.5 ±1.5		1.7*[O]	1.04–2.9
Polyunsaturated (g/d)		12.7± 1.9	7.3 ± 1.2	0.017*	0.93*[a]	0.87–0.98
Polyunsaturated PAFA					0.86**[b]	0.77–0.96
Polyunsaturated PAFA					0.62*[O]	0.4–0.97
% Kcal from PAFA		4.8±0.8	2.7±0.1	0.013*	−0.7**[a]	03–0.8
Cholesterol (mg/d)		400.7±194.4	126.9±18.1	0.66		
Dietary fiber (g/d)		56.2±105	56.8±119	0.97		
Vitamin C (mg/d)		521±139.6	797±400	0.52		
Vitamin E (mg/d)		10.7±2.1	10.6±3.4	0.98		
Selenium (Mcg/d)		97.7±5.2	83.2±4.2	0.03*		

SE is standard error, it is used for comparing between means, **$P \leq 0.01$, *$P \leq 0.05$, O: Risk B-ca among obese, a: Risk of B-ca among all sample, b: Risk B-ca among all sample with antioxidant nutrients (Se and Vitamins C and E). mg/d: Milligram/day, Mcg/d: Microgram/day

Table 4: Comparisons of some food items between cases and controls and their significant differences

Dietary data intake (g/d)	Controls	B-Ca cases	*P*-value of *t*-test
	Mean±SE	Mean±SE	
Cooking oil	31.1±4.0	42.7±4.7	0.05*
Omega-6 (in cooking oil-sunflower oil)	19.9±2.6	27.3±2.9	0.05*
Red meats	11.4±1.9	22.8±3.5	0.005**
Poultry meats	21.6±2.4	33.5±4.8	0.029*
Fish intake	4.2±0.9	5.8±1.5	0.36
Egg intake	10.8±1.5	16.2±2.1	0.035*

g/d: Gram per day

among premenopausal Caucasian women and among women to ER-negative tumors. This result may due to the method of preparation as reported by Bao *et al*.[20] who concluded that B-Ca risk increased when chicken was consumed with the skin, contains fats or some component produced during cooking.

CONCLUSIONS

The study concluded that increased intake of saturated and monounsaturated fats that are derived from animal origin; red meats, poultry and eggs, and specific polyunsaturated fats mainly from cooking oil, may cause hormones imbalance, inflammation, and increase risk of B-Ca, particularly among obese menopause woman. In addition, gaining high calories from monounsaturated fats and method of meat preparation and cooking may increase the risk of B-Ca.

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