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Table of Content: Volume 10 Number 1 January 2018

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Full Length Research Paper

The influence of life behaviour, diet, physical activities on the incidence of breast cancer among women attending Obafemi Awolowo University Teaching Hospital Complex, Ile-Ife

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The aetiopathogenesis of cancer has been linked to life behaviour in a significant percentage of cases. Overweight, sedentary lifestyle, smoking and the excessive use of alcohol have been shown to play important roles. This study is aimed at determining the effect of life behavioural patterns and diets in the incidence of breast cancer in Nigerians population. A prospective case-controlled study of 125 breast cancer patients and 89 normal were recruited in Obafemi Awolowo University Teaching Hospital Complex, (OAUTHC), Ile-Ife, Nigeria. Questionnaire was administered to determine life behavioural patterns and nutritional habits. The variables on nutrition items were pooled to form 23 aggregate variables renamed "total variables" and exposure to endogenous estrogens was calculated. The means age in the patient and control groups were 45.9±10.4 and 44.5±15.2 years, respectively. A significantly higher number of patients in the breast cancer group had at least one previous abortion compared to the control group. The two groups have similar contraceptive use history and mean body mass index (BMI). 14 derived variables on univariate analysis were found to be significant, but only four (menarche [p = 0.002], "total beans" [p = 0.003], "total oils" [p = 0.021] and alcohol intake [p = 0.034]) reached statistical significance when subjected to multivariate regression analysis. Most were premenopausal but onsets of menarche were significantly higher among breast cancer patients. The onset of menarche, intake of beans, oils and alcohol may play a role in the incidence of breast cancer.

Key words: Life behaviour, diet, physical activities, breast cancer among women, Nigeria.

INTRODUCTION

About 5 to 10% of all cancers can be attributed to genetic factors; the remaining 90 to 95% have their causative

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factors rooted in environmental and behavioural factors. These include factors such as cigarette smoking, diet, alcohol, sun exposure, environmental pollutants, infections, stress, obesity, and physical inactivity (Anand et al., 2008).

A lower risk of breast cancer has been linked to increase in physical activities; an expert panel of the International Agency for Research on Cancer estimated 20 to 40% decrease in the risk of developing breast cancer among the most physically active women, regardless of menopausal status. The types or intensity of activities have significant positive effects on quality of life and prolonged survival after breast cancer diagnosis. Apart from burning of excess fat thus leading weight reduction, there was lowering effect on serum ovarian hormones, which may explain the relationship between physical activity and breast cancer.

Lower oestrogen levels among physically active women with breast cancer is suspected to be responsible for improved survival while lack of physical activity is related to weight gain which may impact negatively on breast cancer survival (Holmes et al., 2005; McTiernan et al., 2003; Lonning et al., 1996; Holmberg et al., 2001; Demark-Wahnefried et al., 2001). Obesity, overweight and sedentary lifestyles are responsible for over 25% of all cancer cases (Anand et al., 2008)). There are numerous evidence which show that, women who are obese have 50 to 250% increased risk of developing breast cancer especially during their post-menopausal period (Holmes et al., 2005).

In a study of women in USA, increased risk of breast cancer mortality was found with higher level of obesity, the relative risk of breast cancer mortality increased significantly with increase in BMI (IARC Working Group on the Evaluation of Cancer-Preventive Agents, 2002; McTiernan, 2000; Calle et al 2003; Rossi et al., 2014). Conversely, engaging in moderate to vigorous exercises, 3 to 4 times a week reduced the incidence and increased survival of breast cancer by 30 to 40% than in sedentary and overweight women. Intake of even moderate quantities of alcohol increases the chance of developing breast cancer in pre- and post-menopausal women. Diet high in fat, low in fruits, vegetables and in fibre, and high simple carbohydrates may increase the risk of breast cancer, although this is not supported by overwhelming evidence (Anand et al., 2008; IARC Working Group on the Evaluation of Cancer-Preventive Agents 2002; McTiernan., 2000; Calle et al., 2003; Anderson et al., 2016; Peeters et al., 2017). These factors are, possibly, mediated through the hormonal pathway (Calle et al 2003; Cerhan et al., 1998; Dirx et al., 2001). The current trend of increasing obesity and very little exercise may be responsible for the increase in the incidence of breast cancer in developing countries. That lifestyle modification can change the risk of developing breast cancer supported by several lines of evidence, therefore, most breast cancer prevention strategies involves some degree of lifestyle modification and positive attitudinal changes toward good dietary pattern and health living, engaging in regular exercise, avoiding tobacco and reduction in alcohol intake (Rossi et al., 2014; Ferrini et al., 2015; Hu et al., 2002).

This study was designed to examine the contribution of life behavioural activities and dietary pattern on the incidence of breast cancer in OAUTHC, Ile-Ife, Osun State, Nigeria.

MATERIALS AND METHODS

This was a prospective, questionnaire-based case-controlled study, involving 126 breast cancer patients and attending the Surgical Out-patient Department or admitted unto surgical wards of the Obafemi Awolowo University Teaching Hospital Complex, (OAUTHC), Ile-Ife, Nigeria and 97 age-matched controls recruited among non-cancer female patients of the hospital or normal volunteers.

A 450-variable questionnaire to determine life behavioural pattern, dietary habit, the use of alcohol, tobacco, multivitamins, minerals and others was developed. House Surgeons did data collection. They were trained on the administration of the questionnaires to the subjects. The Ethics and Research Committee of OAUTHC, Ile-Ife approved the study, and informed consent was obtained from all respondents.

Data cleaning and processing

All data pertaining to 450 variables interrogated in the questionnaire were entered into MS Excel spreadsheets. Data was examined electronically for accuracy, correctness and consistency, and cleaned appropriately leaving 125 patients and 89 subjects as control.

We used quasi “cluster analysis” Peeters et al. (2017) of dietary pattern, in which consumed items of nutrition were grouped according to their nutritional content, and re-named “Total-variable”. The maximum exposure to each item on the questionnaire was defined as 5, and the items under a group were summed up and converted to percentages of the maximum attainable score. Thus, aggregated exposure to dairy products was called “Total-Dairy”, resulting in 23 aggregated variable groups. New variable “endogenous oestrogen index” was obtained by calculating the total number of month’s respondent was exposed to endogenous oestrogen (based on data about menarche, number of pregnancies, age and menopause).

Data was transferred to SPSS 17 for statistical analysis. Student’s T-test statistics was used to compare the means of the grouped variables for study and the control subjects. A logistic regression analysis was later done to ascertain the strength of association between breast cancer and 14 variables that there were found to be statistically significant as single variables.

RESULTS

Expectedly, the mean age of patients and controls (45.9 ± 10.4 and 44.5 ± 15.2 years, respectively) were not statistically different. All patients and controls were on typical African diets, which consist of starchy food made from tubers or cereals and various forms of vegetables cooked to make soup used in eating starchy food, with a

piece or pieces of beef or chicken or fish. Alcohol consumption was very low but irregular in most of the patients and controls, although the difference was statically significant. Among both groups, there were no significant differences in oral contraceptive use. Similarly, the usage of tobacco (cigarettes), consumption of non-alcoholic beverages, coffee, fruits and seeds and engagement in exercise were very irregular and low (Table 1). Most of the respondents belonged to low and middle socioeconomic groups with low-level education or none at all.

The means body mass index (BMI) for the two groups were also similar. However, the mean age at menarche in subjects (14.8± 2.0) was significantly later than that of the controls (13.5 ± 2.8 years, $p = 0.0004$). The majority in both groups were pre-menopausal, though a significantly higher proportion of the breast cancer patients had reached menopause (36.8% vs. 18.8%; $p = 0.023$) or have had at least one previous abortion (22.4% vs. 6.3%; $p = 0.01$), when compared with the control group. Similarly, there were statistically significant differences in the mean age of first pregnancy, number of pregnancies and number of children, but not in the duration of breast-feeding. A total of ~80% of the patients with breast cancer presented late stage disease (stages 3 and 4; Table 1).

Additionally, the breast cancer patients ate significantly less snacks, cereals, poultry, dairy products, “leafy” vegetables, oils, sweetened products, artificial ingredients, fruits, vitamins and minerals, and alcohol compared to the controls; but consumed more legumes (beans). 14 statistically significant variables were used to test logistic regression model against a constant-only model, which was statistically significant (Chi square = 6.757, $p < 0.001$, df = 8). Prediction success overall was 74% (84.2% for breast cancer and 60.5% for control). The details are shown in Table 3. Only four variables reached statistical significance in this regression analysis: age at menarche ($p = 0.002$), “total beans” ($p = 0.003$), “total oils” ($p = 0.021$) and alcohol ($p = 0.034$) Tables 2 and 3.

DISCUSSION

We found that all patients and controls in this study were on typical African diets, consisting of starchy food made from tubers or cereals and various forms of vegetables with pieces of beef or chicken or fish, cooked to make soup used in eating the starchy food. Therefore, the analysis of contribution of their nutritional content to the incidence of breast cancer in our environment may be difficult. According to Hu (2002), people do not eat isolated nutrients, instead, they eat meals consisting of a variety of foods with complex combinations of nutrients that are likely to be interactive or synergistic in absorption of their effects. Three methods of dietary pattern analysis have been suggested; factor analysis, cluster analysis and healthy eating indices (Hu, 2002). We have used a quasi-cluster analysis in this study, and found significant differences in consumption of snacks, cereals, dairy, poultry, leafy vegetables, beans, oils, artificial ingredients, sweeteners, fruits and vitamins and minerals but on multiple regression analysis and only four of these; the consumption of less vitamins/minerals, less oils, less alcohol and more beans were independently associated with breast cancer.

The means of BMI of patients and controls in this study were within normal limit and were expectedly not significantly different. Various studies had been carried out on the associations between weight or BMI, central fat distribution or adult weight gain and breast cancer incidence (Anand et al., 2008; IARC Working Group on the Evaluation of Cancer-Preventive Agents 2002; McTiernan, 2000; Calle et al., 2003; Friedenreich et al., 2001). These studies found that women who were obese had a 30 to 50% greater risk for post-menopausal breast cancer development than leaner women (IARC Working Group on the Evaluation of Cancer-Preventive Agents, 2002), though another study found that this risk does not extend to pre-menopausal women who are not on hormonal replacement therapy (Morimoto et al., 2002). There is an increase in breast cancer risk with increasing BMI (≥31) as high as 2.5 times risks of developing breast cancer in women. The means of BMI for both subjects and controls were within normal limit, with only 8.5% of subjects having BMI ≥31. The limited numbers consequently do not permit any meaningful comparison in our study population.

Our subjects and controls were very low in leisure time physical activities and although many were engaged in occupation-related activities. We found that the level of physical activities was significantly higher in subjects with breast cancer than in controls. Various studies have established the relationship between the leisure and recreational exercises and decrease in the risk of breast cancer (Anand et al., 2008; IARC Working Group on the Evaluation of Cancer-Preventive Agents 2002; McTiernan, 2000; Calle et al., 2003; Ferrini et al., 2015). The relationship between physical activities and decreased risk of breast cancer is dose-related. The risk reduction range from 10 to 70% for the most active women who exercised for 3 to 4 h per week at moderate to vigorous levels (Thune and Furberg, 2001; Thune et al., 1997). Vigorous and moderate physical activities alter the menstrual cycles, suppressing the pulsatile release of GRH, which may lower the cumulative exposure to oestrogen and progesterone, thereby inhibiting breast cancer development. It is believed that energy balance might be important in this; such that caloric restrictions tend to reduce the proliferative activity of the mammary glands (Cohen, 2000) and inhibits carcinogenesis (Thune and Furberg, 2001; Thune et al., 1997).

Several studies have examined dietary pattern

Table 1. Some of the characteristics of the patients and controls.

Variables	PATIENTS		CONTROLS		
	Mean	SD	Mean	SD	P value
Age of subjects	45.9	10.4	44.5	15.2	0.448
BMI	24.1	4.7	24.3	5.5	0.842
Age at menarche	14.7	2.0	13.5	2.8	0.002
Age of first of pregnancy	24.4	4.6	25.2	3.24	0.207
Number of abortions	0.5	1.3	0.1	0.3	0.003
Mean of parity	3.9	2.1	2.9	2.4	0.002
				df	P value
Nulliparity	8		22	3	0.003
History of abortions – Yes	28		4	1	0.007
None	70		75	1	0.653
Menoupausal status- Pre	82		67	1	0.068
Post	43		30	1	0.786
Level of education- None	22		6	1	0.024
Primary	18		16	1	0.432
Junior Secondary Sch	7		3	1	0.235
Senior Secondary Sch	22		18	1	0.335
Tech Sch	6		2	1	0.092
Polytech/College of Education	33		15	1	0.0231
University Education	17		36	1	0.032
Others	1		1		
Annual family income (NGN $x10^3$) <10	15		17	1	0.671
10-49	15		12	1	0.752
50-99	11		16	1	0.478
100-249	27		17	1	0.043
250-449	22		14	1	0.009
≥500	29		18	1	0.032
Freq of leisure-time exercises – Never	13		9	1	0.568
≥1x/year	18		4	1	0.020
≥1x/month	13		13	1	
1x/week	7		14	1	0.045
2x/week	6		2	1	0.068
3-4x/week	10		12	1	0.791
>4x/week	0		0	1	
Fr eq of work-related exercises – sedentary	12		4	1	0.014
Mild strenuous	25		12	1	0.023
Moderately strenuous	44		27	1	0.036
Strenuous	14		2	1	0.042
Very strenuous	14		9	1	0.093

differences throughout the world and suggested that dietary plans which are low in fat, high in fruits, vegetables, fibre, and complex carbohydrates might lower the risk for breast cancer (Marshall et al 1992; Prentice et. al., 1988; Welsch 1992; World Cancer Research Fund Panel (Potter JD Chair), 1997). The diet in our community is high in fibre with complex carbohydrates but low in fat, fruits and vegetables; and may be contributing to the increased incidence of breast cancer, among other things. Many prospective studies women have not been able to unanimously establish this assertion, though, animal studies have demonstrated the value of diet in reducing the incidence of breast cancer (Welsch, 1992; World Cancer Research Fund Panel (Potter JD Chair),

Table 2. Showing analysis of variables.

Variables	Sub Types	N	Means	SD	F	df	P value	95% CI	
								lower	upper
Endoge-estr ind	Controls	80	300.8	123.7	30.15	196	0.621	-36.34	21.77
	Pts	118	307.4	83.7					
T-snacks	Controls	89	31.5	12.8	0.220	213	0.022*	0.59	7.54
	Pts	125	27.5	12.7					
T-cereal	Controls	89	45.14	13.7	9.80	213	0.001*	2.173	8.471
	Pts	125	39.8	9.7					
T-poultry	Controls	89	31.8	13.5	0.282	213	0.002*	2.21	9.45
	Pts	125	26.0	13.1					
T-dairy	Controls	89	22.5	14.2	13.53	213	0.004*	1.51	7.68
	Pts	125	17.9	8.8					
T-fish	Controls	89	33.6	16.9	4.14	213	0.200	-1.35	6.56
	Pts	125	30.7	12.6					
T-other meats	Controls	89	20.4	18.7	6.73	212	0.253	-1.88	7.09
	Pts	125	17.8	14.5					
T-fruits/veg soup	Controls	89	38.1	18.0	6.67	213	0.860	-3.87	4.65
	Pts	125	37.7	13.7					
T-Leafy vegetables	Controls	89	30.5	16.9	26.66	213	<0.001*	4.17	11.44
	Pts	125	22.6	10.0					
T-tuber	Controls	89	36.9	16.1	9.06	213	0.058	-0.136	7.30
	Pts	125	33.3	11.5					
T-beans	Controls	89	40.9	22.9	0.012	213	<0.001*	8.56	20.75
	Pts	125	26.2	21.9					
T-oils	Controls	89	34.8	15.0	8.31	213	<0.001*	3.88	10.84
	Pts	125	27.4	10.8					
T-natural ingredients	Controls	89	66.1	22.7	5.51	213	0.087	-0.64	9.39
	Pts	125	63.7	14.6					
T-artficial ingredients	Controls	89	42.5	23.1	8.24	213	0.011*	1.56	12.19
	Pts	125	35.6	16.5					
T-sweet	Controls	89	23.0	16.4	24.8	213	0.005*	1.54	8.53
	Pts	125	18.0	9.7					
T-beverage	Controls	89	30.5	17.2	0.27	213	0.53	-6.06	3.13
	Pts	125	31.9	16.5					
T-soda/juice	Controls	89	28.3	17.7	5.6	213	0.13	-0.95	7.33
	Pts	125	25.1	13.1					
T-dairy/fat	Controls	89	21.38	11.5	1.59	213	1.00	-2.92	2.92
	Pts	125	21.38	10.0					
T-fruits	Controls	89	28.3	12.8	5.9	213	0.004	1.45	7.58
	Pts	125	23.8	9.9					
T-nuts	Controls	89	34.5	20.7	3.1	213	0.571	-3.69	6.68
	Pts	125	33.0	17.8					

Table 2. Contd.

T-vitamins/miner	Controls	89	37.4	28.1	12.2	213	<0.001	8.77	22.07
	Pts	125	22.0	21.4					
T-alcohol	Controls	89	7.2	9.4	94.8	213	<0.001	-4.58	7.30
	Pts	125	3.1	3.8					
T-fr/BK/ros	Controls	89	45.5	22.6	0.39	213	0.652	-4.58	7.300
	Pts	125	44.1	21.1					

Table 3. Multivariate logistic regression modeling (Model Summary).

Model Summary			
Step	**-2 Log likelihood**	**Cox & Snell R Square**	**Nagelkerke R Square**
1	223.384[a]	0.221	0.297

Variables in the Equation

		B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
								Lower	**Upper**
Step 1[a]	Menarche	0.229	0.084	7.512	1	0.006130	1.258	1.068	1.482
	T-Beans	-0.020	0.008	6.840	1	0.008912	0.980	0.966	0.995
	T-Oils	-0.027	0.014	3.872	1	0.049096	0.973	0.947	1.000
	T-VitsMin	-0.011	0.007	2.694	1	0.100724	0.989	0.976	1.002
	T-Alcohol	-0.055	0.026	4.594	1	0.032081	0.946	0.900	0.995
	Constant	-0.871	1.265	0.474	1	0.490974	0.418		

Variables in the Equation

		B	**S.E.**	**Wald**	**df**	**Sig.**	**Exp(B)**	**95% C.I.for EXP(B)**	
								Lower	**Upper**
Step 1[a]	Menarche	0.257	0.082	9.861	1	0.001688	1.293	1.101	1.518
	T-Beans	-0.022	0.007	9.127	1	0.002519	0.978	0.964	0.992
	T-Oils	-0.032	0.014	5.360	1	0.020602	0.969	0.943	0.995
	T-Alcohol	-0.055	0.026	4.510	1	0.033697	0.947	0.900	0.996
	Constant	-1.384	1.216	1.295	1	0.255089	0.251		

1997). Additionally, some studies have found that diets which are low in fat, but high in fibre, vegetable and fruit have some influence on human breast cancer biomarkers and also cause less dense mammographic findings (Chang et al., 2017; Catsburg et al., 2015; Boyd et al., 1997; Mourouti et al., 2015; Lelièvre and Weaver, 2013). Similarly, it has been shown that a decrease in circulating oestrogen levels following modification

in dietary pattern, (Marshall et al.,1992; Wu et al., 1998) whether it was due to the diet or weight loss resulting from the dietary changes have reduced the risk of breast cancer (Cohen, 2000). While intake of dietary fat has not been proven to be associated with risk for breast cancer, increased dietary fat causes increased caloric intake and may lead to overweight and obesity, which have been linked to increase risk for breast cancer.

Vegetables and their products were not taken as meals by cohort of subjects and controls, but rather as parts of meals in which carbohydrates is a predominant portion. Although, vegetables have been shown to be associated with lower risk of breast cancer by providing high-fibre diets, low in energy and fat (Chang et al., 2017; Catsburg et al., 2015; Mourouti et al., 2015; Lelièvre and Weaver, 2013), there is no consensus about their ultimate value nevertheless studies have found the consumption of soy products associated with a lower risk for breast cancer (Wu et al., 1998; Greenwald, 2002). Aside from this, there is also no consensus as regard to their value when it comes to breast cancer risk. Indeed, there were studies which suggested that some soy products may promote the growth of some oestrogen-sensitive tumours and reduce the efficacy of tamoxifen (Jones et al., 2002; Ju et al., 2002). Vegetables contain high content of phytoestrogens, a weak oestrogen-like compound, and oestrogen antagonists. Thus, high phytoestrogen intake can compete with endogenous oestrogens in premenopausal women and reduce overall oestrogen exposure to target tissue (World Cancer Research Fund Panel (Potter JD Chair), 1997; Herman et al., 1995; Cassidy et al., 1993). Conversely, phytoestrogens can increase oestrogen activities in women with low endogenous levels of oestrogens, for example, postmenopausal women (World Cancer Research Fund Panel (Potter JD Chair), 1997; Herman et al., 1995; Cassidy et al., 1993). Fruits apart from being rich in fibres, also contain antioxidants which can stem cancerous transformation but in this environment the typical meals are usually poor in fruit contents (Catsburg et al., 2015; Boyd et al., 1997; Mourouti et al., 2015; Lelièvre and Weaver, 2013).

High consumption of meats has been linked to increased risk for breast cancer, but this has not been supported by many studies (World Cancer Research Fund Panel (Potter JD Chair), 1997; Catsburg et al., 2015; Boyd et al., 1997; Lelièvre and Weaver, 2013). Although, this inconsistency may be due to the different levels of carcinogens and mutagens included in meat in different parts of the world, as a result of different breeding, storage and food additive practices. A recent report from a large cohort study suggests a lower risk cancer with greater intake of low-fat dairy products in premenopausal women (Catsburg et al., 2015; Boyd et al., 1997; Mourouti et al., 2015; Lelièvre and Weaver, 2013; Shin et al., 2002; Dorgan et al., 1998). The authors proposed that, the fat content of dairy products may promote a greater risk for breast cancer through a greater energy intake in the diet. It has also been suggested that high calcium and vitamin D contents of many of these products may be protective against breast cancer (Shin et al., 2002). Our patients and controls were not consuming meats in proportions that will provide any observable effect on the risk of breast cancer.

Vitamin and mineral pills are not part of an average African dish; the vitamins (usually vitamins B and C tablets, 100 or 400 mg daily) were taken irregularly by some of our patients and subjects. Among the vitamins and minerals, only carotenoids, folate, calcium, vitamin D, lycopene, and vitamin C have been associated with a lower risk of breast cancer (World Cancer Research Fund Panel (Potter JD Chair), 1997; Dorgan et al., 1998; Hulten et al 2001; Sato et al., 2002; Egnell et al., 2017). Although, the vitamins intake was statistically significant between the groups in our study, our patients and controls were not taking enough doses to be effective as regular use of such micronutrients after food is alien to our community. For intake of vitamins and minerals to have a tangible effect on the risk of cancer, the vitamins have to be taken regularly and doses of vitamins should be very large. Analyses of prospective studies suggest that women with low folate intakes and greater alcohol consumption ran a greater risk for breast cancer than non-drinkers with normal folate intakes (Sellers et al., 2002; Zhang et al., 1999). In a studies involving a large cohort of normal women taking vitamin A, B, C and E supplements, followed-up for 2 decades, it was discovered that taking large doses of thiamine and vitamins C and E did reduce the risk of breast cancer but low intake of vitamin A increased the risk of breast cancer (Egnell et al., 2017; Hunter et al., 1993).

Similarly, the alcohol consumption among women in our community was very low and irregular.The subjects and controls sparingly drink alcohol and smoke cigarette. It is a taboo for ladies to engage in smoking in our environment. Chronic alcohol intake has been linked with cancers of the breast, upper respiratory tract and upper GIT. Studies had found increase in risk of breast cancer due to alcohol consumption (Longnecker et al., 1988). It has been discovered in many studies, that chronic alcohol intake induce carcinogenesis; alcohol metabolism leads to increased free radicals in the system which in turn bind to DNA and protein destroying folate leading to secondary hyper proliferation, induction of cytochrome P4502E1, resulting in increased free radicals, that promote of pro-carcinogens in the alcoholics (Longnecker et al., 1988; Collaborative Group on Hormonal Factors in Breast Cancer, 2002). Among smokers who are also chronic alcohol users, this changes the metabolism and distribution of carcinogens, alters cell-cycle behaviour and shorten the duration resulting in hyperproliferation (Longnecker et al., 1988; Collaborative Group on Hormonal Factors in Breast Cancer, 2002). Alcohol depresses the appetite leading to low intake of vitamins

and essential trace elements thereby altering the immune system (Anand et al., 2008). All these attributes to alcohol regarding the risk breast cancer would play, little or no role in the risk of breast cancer in our environment.

Majority of our patients were premenopausal, which is in tandem with previous findings in Nigeria where by 67 to 80% patients with breast cancer were premenopausal (Adesunkanmi et al., 2006). The onset of menarche in both groups was 12 to 15 years, with the menarche setting-in on average 2 years later in breast cancer patients. This finding is at variance to what is known in breast cancer. The mean ages of first pregnancies were also significantly different, 22 years in breast cancer patients and 18 years in controls; and breast cancer patients had more previous abortions than controls. Both groups had many children, and the differences were not significant. Similarly, the subjects in this study and controls breastfed their children for an average of 18 months. Early menarche (before the age of 12 years), higher frequency of anovulatory cycles, late first birth or no birth, scanty number in lack of lactation, late menopause, prolong interval between menarche and menopause lead to high concentrations and prolonged exposure to endogenous oestrogen and have been found to increase the risk of breast cancer (Key et al., 2002; Kumle et al., 2002; McTiernan et al., 2004).

Besides, several of these reproductive hormones have been found predispose to obesity in the absence of physical activity thereby increase the risk of breast cancer. The use of oral contraceptive intake was occasional between the two groups studied and there was no case of hormone replacement therapy. Some studies found associations of oral contraceptive, most especially oestrogen-based pills and increased breast cancer risk among premenopausal women (McTiernan et al., 2004; Hulka, 1996). Combined oral contraceptives, the combination of hormones, induce more cell divisions than oestrogen alone. The use of combined oral contraceptive pills increases the levels of oestrogen as well as progestogen, whereas progestin-only pills only increase levels of progestogen without influencing oestrogen level, thereby removing the suspected carcinogenic effect of oestrogen (McTiernan et al., 2004; Hulka, 1996).

Conclusion

The role of diets, physical activities and other life behavioural patterns in the risk of breast cancer in community needs further studies. This is because the diets consist mainly of carbohydrates with over cooked vegetables, very poor in protein content, fruits and low fat diet. Also, food supplements are rarely consumed; chronic alcohol users and cigarette smoking are uncommon among our patients and controls. While the poor nutritional value, poor fruit and vegetable content, poor vitamins and minerals supplements and almost lack of physical activities may contribute to increase in the risk of breast cancer; low fat, poor intake oral contraceptives, rarity of alcohol intake and cigarette smoking may negatively influence the risk of breast cancer.

Further prospective studies in this direction will be necessary in Nigeria to be able to make clear pronouncements on the effect of the various dietary components, on the risk of breast cancer in our environment.

CONFLICT OF INTERESTS

The authors have not declared any conflict of interests.

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REFERENCES

Adesunkanmi ARK, Lawal OO, Adelusola KA, Durosimi MA (2006). The severity, outcome and challenges of breast cancer in Nigeria. The Breast 15(3):399-409.

Anand P, Kunnumakara AB, Sundaram C, Harikumar KB, Tharakan ST, Lai OS, Sung B, Aggarwal BB (2008). Cancer is a Preventable Disease that Requires Major Lifestyle Changes. Pharm. Res. 25(9):2097-2116.

Anderson C, Harrigan C, George SM, Ferrucci LM, Sanft T, Irwin ML, Cartmel B (2016). Changes in diet quality in a randomized weight loss trial in breast cancer survivors: The lifestyle, exercise, and nutrition (LEAN) study. Nature Partner J. (npj) Breast Cancer 2:16026

Boyd NF, Greenberg C, Lockwood G, Little L, Martin L, Tritchler D, Byng J, Yaffe M (1997). Effects at two years of a low-fat, high-carbohydrate diet on radiologic features of the breast: results from a randomized trial. Canadian Diet and Breast Cancer Prevention Study Group. J. Natl. Cancer Inst. 89:488-496.

Calle EE, Rodriguez C, Walker-Thurmond K, Thun MJ (2003). Overweight, obesity, and mortality from cancer in a prospectively studied cohort of U.S. adults. N. Engl. J. Med. 348:1625-1638.

Cassidy A, Bingham S, Carlson J, Setchell KDR (1993). Biological effects of plant oestrogens in premenopausal women. FASEB J. 7:A866.

Catsburg C, Kim RS, Kirsh VA, Soskolne CL, Kreiger N, Rohan TE (2015). Dietary patterns and breast cancer risk: A study in 2 cohorts. Am. J. Clin. Nutr. 101(4):817-823.

Cerhan JR, Chiu BC, Wallace RB, Lemke JH, Lynch CF, Tomer JC, Rubenstein LM (1998). Physical activity, physical function, and the risk of breast cancer in a prospective study among elderly women. J. Gerontol. A Biol. Sci. Med. Sci. 53(4):M251-M256.

Chang YJ, Hou YC, Chen LJ, Wu JH, Wu CC, Chang YJ, Chung KP (2017). Is vegetarian diet associated with a lower risk of breast cancer in Taiwanese women?. BMC public health, 17(1):800.

Cohen LA (2000). Re: Meta-analysis: dietary fat intake, serum estrogen levels, and the risk of breast cancer. J. Natl. Cancer Inst. 92(1):78-78.

Collaborative Gropu on Hormonal Factors in Breast Cancer (2002). Alcohol, tobacco and breast cancer- collaborative reanalysis of individual data from 53 epidemio- logical studies, including 58,515 women with breast cancer and 95,067 women without the disease. Br. J. Cancer. 87:1234-45.

Demark-Wahnefried W, Peterson BL, Winer EP, Marks L, Aziz N, Marcom PK, Blackwell K, Rimer BK (2001). Changes in weight, body composition, and factors influencing energy balance among premenopausal breast cancer patients receiving adjuvant chemotherapy. J. Clin. Oncol. 19(9):2381-2389.

Dirx MJ, Voorrips LE, Goldbohm RA et al (2001). Baseline recreational physical activity, history of sports participation, and postmenopausal breast carcinoma risk in the Netherlands Cohort Study. Cancer 92(6):1638-1649.

Dorgan JF, Sowell A, Swanson CA, Potischman N, Miller R, Schussler N, Stephenson H (1998). Relationships of serum carotenoids, retinol, alpha-tocopherol, and selenium with breast cancer risk: results from a prospective study in Columbia, Missouri (United States). Cancer Causes Control 9:89-97.

Egnell M, Fassier P, Lécuyer L, Zelek L, Vasson MP, Hercberg S, Latino-Martel P, Galan P, Deschasaux M, Touvier M (2017). B-Vitamin Intake from Diet and Supplements and Breast Cancer Risk in Middle-Aged Women: Results from the Prospective NutriNet-Santé Cohort. Nutrients 9(5):488.

Ferrini K, Ghelfi F, Mannucci R, Titta L (2015). Lifestyle, nutrition and breast cancer: facts and presumptions for consideration. Ecancermedicalscience. 9:557.

Friedenreich CM (2001). Review of anthropometric factors and breast cancer risk. Eur. J. Cancer Prev.10:15-32.

Greenwald P (2002). Cancer prevention clinical trials. J. Clin. Oncol. 20(suppl 18):14S-22S.

Herman C, Adlercreutz T, Goldin BR, Gorbach SL, Höckerstedt KA, Watanabe S, Hämäläinen EK, Markkanen MH, Mäkelä TH, Wähälä KT, Hase TA (1995). Soybean phytoestrogen intake and cancer risk. J. Nutr. 125(suppl 3):757S-770S.

Holmberg L, Norden T, Lindgren A, Wide L, Degerman M, Adami HO (2001). Pre-operative oestradiol levels-relation to survival in breast cancer. Eur. J. Surg. Oncol. 27:152.

Holmes MD, Chen WY, Feskanich D, Kroenke CH, Colditz, GA (2005). Physical Activity and Survival After Breast Cancer Diagnosis. JAMA. 293(20):2479-2486.

Hu FB (2002). Dietary pattern Analysis: A new direction in nutritional epidemiology. Curr. Opin. lipidol. 13(1):3-9.

Hulka BS (1996). Epidemiology of susceptibility to breast cancer. Prog. Clin. Biol. Res. 395:159-174.

Hultén K, Van Kappel AL, Winkvist A, Kaaks R, Hallmans G, Lenner P, Riboli E (2001). Carotenoids, alpha-tocopherols, and retinol in plasma and breast cancer risk in northern Sweden. Cancer Causes Control 12(6):529-537.

Hunter DJ, Manson JE, Coldzt GA, Stamper ME, Rosner B, Hennekens CH, Speiser FE, Willet WC (1993). A prospective study of intake of vitamin C, E and A and the risk of breast cancer. New Engl. J. Med. 329:234-40.

IARC Working Group on the Evaluation of Cancer-Preventive Agents (2002). Weight Control and Physical Activity, IARC Handbooks of Cancer Prevention, Volume 6. Lyon, France: IARC.

Jones JL, Daley BJ, Enderson BL, Jin-Rong Z (2002). Genistein inhibits tamoxifen effects on cell proliferation and cell cycle arrect in T47D breast cancer cells/Discussion. Am. Surg. 68(6):575-577.

Ju YH, Doerge DR, Allred KF, Allred CD, Helferich WG (2002). Dietary genistein negates the inhibitory effect of tamoxifen on growth of estrogen-dependent human breast cancer (MCF-7) cells implanted in athymic mice. Cancer Res. 62(9):2474-2477.

Key T, Appleby P, Barnes I, Reeves G (2002). Endogenous sex hormones and breast cancer in postmenopausal women: reanalysis of nine prospective studies. J. Natl. Cancer Inst. 94:606-616.

Kumle M, Weiderpass E, Braaten T, Persson I, Adami HO, Lund E (2002). Use of oral contraceptives and breast cancer risk. Cancer Epidemiol. Prev. Biomarkers 11(11): 1375-1381.

Lelièvre SA, Weaver CM (2013). Global Nutrition Research: Nutrition and Breast Cancer Prevention as a Model. Nutr. Rev. 71(11): 742-752.

Longnecker MP, Newcomb PA, Mittendorf R, Greenberg ER (1988). Risk of breast cancer in relation to lifetime alcohol consumption. Int. J. Epidemiol. 17:298-306

Lonning PE, Helle SI, Johannessen DC, Ekse D, Adlercreutz H (1996). Influence of plasma estrogen levels on the length of the disease-free interval in postmenopausal women with breast cancer. Breast Cancer Res. Treat. 39(3):335-341.

Marshall JR, Yinsheng Q, Junshi C, Parpia B, Campbell TC (1992). Additional ecological evidence: lipids and breast cancer mortality among women aged 55 and over in China. Eur. J. Cancer 28(10):1720-1727.

McTiernan A (2000). Associations between energy balance and body mass index and risk of breast carcinoma in women from diverse racial and ethnic backgrounds in the U.S. Cancer 88(S5):1248-1255.

McTiernan A, Rajan KB, Tworoger SS, Irwin M, Berstein L, Baumgarthe R, Gilliland F, Stanczyk FZ, Yasui Y, Ballar-Barbash R (2003). Adiposity and sex hormones in postmenopausal breast cancer survivors. J. Clin. Oncol. 21:1961-1966.

McTiernan A, Tworoger SS, Ulrich CM, Yasui Y, Irwin ML, Rajan KB, Sorensen B, Rudolph RE, Bowen D, Stanczyk FZ, Potter JD (2004). Effect of exercise on serum estrogens in postmenopausal women: A 12-month randomized clinical trial. Cancer Res. 64(8):2923-2928.

Morimoto LM, White E, Chen Z, Chlebowski RT, Hays J, Kuller L, Lopez AM, Manson J, Margolis KL, Muti PC, Stefanick ML (2002). Obesity, body size, and risk of postmenopausal breast cancer: the Women's Health Initiative (United States). Cancer Causes Control 13:741-751.

Mourouti N, Kontogianni MD, Papavagelis C, Panagiotakos DB (2015). Diet and breast cancer: a systematic review. Int. J. Food Sci. Nutr. 66(1):1-42.

Peeters K, Van Leemputte F, Fischer B, Bonini BM, Quezada H, Tsytlonok M, Haesen D, Vanthienen W, Bernardes N, Gonzalez-Blas CB, Janssens V, Tompa P, Versées W, Thevelei JM (2017). Fructose-1,6-bisphosphate couples glycolyticflux to activation of Ras. Nature Communications 8(1):922.

Prentice RL, Kakar F, Hursting S, Sheppard L, Klein R, Kushi LH (1988). Aspects of the rationale for the Women's Health Trial. J. Natl. Cancer Inst. 80(11):802-814.

Rossi RE, Pericleous M, Mandair D, Whyand T, Caplin ME (2014). The Role of Dietary Factors in Prevention and Progression of Breast Cancer. Anticancer Research 34(12):6861-6875

Sato R, Helzlsouer KJ, Alberg AJ, Hoffman SC, Norkus E P, Comstock GW (2002). Prospective study of carotenoids, tocopherols, and retinoid concentrations and the risk of breast cancer. Cancer Epidemiol. Biomarkers Prev. 11:451-457.

Sellers TA, Vierkant RA, Cerhan JR, Gapstur SM, Vachon CM, Olson JE, Pankratz VS, Kushi LH, Folsom AR (2002). Interaction of dietary folate intake, alcohol, and risk of hormone receptor-defined breast cancer in a prospective study of postmenopausal women. Cancer Epidemiol. Biomarkers Prev.11:1104-1107.

Shin MH, Holmes MD, Hankinson SE, Wu K, Colditz GA, Willett WC (2002). Intake of dairy products, calcium, and vitamin d and risk of breast cancer. J. Natl. Cancer Inst. 94(17):1301-1311.

Thune I, Brenn T, Lund E, Gaard M (1997). Physical activity and the risk of breast cancer. New Engl. Med. J. 336(18):1269-1275.

Thune I, Furberg AS (2001). Physical activity and cancer risk: Dose-response and cancer, all sites and site-specific. Med. Sci. Sports Exerc. 33(6):S530-S550.

Welsch CW (1992). Relationship between dietary fat and experimental mammary tumorigenesis: a review and critique. Cancer Res. 52 (suppl 7):2040s-2048s.

World Cancer Research Fund Panel (Potter JD Chair) (1997). Food, Nutrition and the Prevention of Cancer: a Global Perspective. Washington, DC: American Institute for Cancer Research.

Wu AH, Ziegler RG, Nomura AM, West DW, Kolonel LN, Horn-Ross PL, Hoover RN, Pike MC (1998). Soy intake and risk of breast cancer in Asians and Asian Americans. Am. J. Clin. Nutr. 68(suppl 6):1437S-1443S.

Zhang S, Hunter DJ, Hankinson SE, Giovannucci EL, Rosner BA, Colditz GA, Speizer FE, Willett WC (1999). A prospective study of folate intake and the risk of breast cancer. JAMA 281(17):1632-1637.

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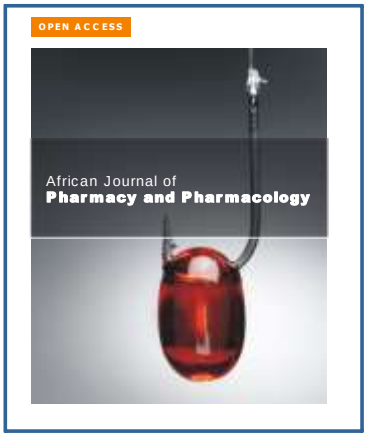

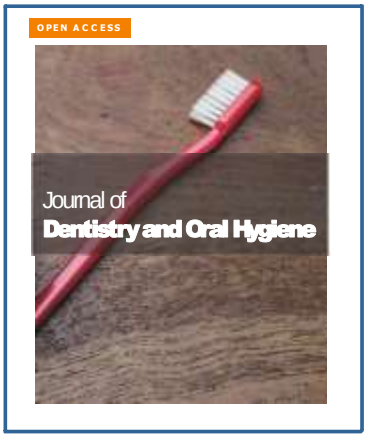

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