# **RESEARCH ARTICLE**

# Dietary Fat and Physical Activity in Relation to Breast Cancer among Polish Women

Joanna Kruk<sup>1\*</sup>, Mariola Marchlewicz<sup>2</sup>

### Abstract

<u>Background</u>: Dietary fat has been inconsistently associated with the risk of breast cancer. The purpose of this study was to examine the relationship between meat and animal and plant fat intake and breast cancer risk in subgroups by total lifetime physical activity, using data from a case-control study conducted in the Region of Western Pomerania, Poland. <u>Materials and Methods</u>: The study included 858 women with histological confirmed breast cancer and 1,085 controls, free of any cancer diagnosis. The study was based on a self-administered questionnaire including questions about socio-demographic characteristics, current weight and height, reproductive factors, family history of breast cancer and lifestyle habits. Unconditional logistic regression was performed to calculate odds ratios (ORs) and 95% confidence intervals (CIs). <u>Results:</u> High animal fat intake significantly increased OR from 1.7 times (OR=1.66, 95% CI=1.07-3.59) to 2.9 times (OR=2.9, 95% CI=1.37-6.14) independent of physical activity level, comparing the third versus the lowest quartile. Women with a high intake of red meat or processed meat and low physical activity showed increased risk of breast cancer: OR=2.70, 95% CI=1.21-6.03 and 1.78, 95% CI=1.04-3.59, respectively. The plant fat dietary pattern was negatively associated with breast cancer in sedentary women (OR=0.57, 95% CI=0.32-0.99). <u>Conclusions</u>: These results indicated that a diet characterized by a high consumption of animal fat is associated with a higher breast cancer risk in sedentary women, while consumption of plant fat products may reduce risk in the same group.

Keywords: Breast cancer risk - animal fat - plant fat - physical activity - case-control study - Poland

Asian Pacific J Cancer Prev, 14 (4), 2495-2502

# Introduction

Breast cancer is the most frequent cancer in women in industrialized countries (Jemal et al., 2008). The breast cancer incidence rate vary widely around the world. For example, the age-standardized incidence rate (per 100000) in North America is 99.4, 18.7 in China (Parkin et al., 2002), 44.5 in Poland (Brużewicz, 2008). The incidence rate is the lowest in most parts of Africa (16.5 per 100000) (Parkin et al., 2002). Moreover, the rising incidence of breast cancer has been observed in countries in parallel with their economic development (Zhang et al., 2011).

Extensive research has shown that body fatness can decrease the risk of breast cancer in premenopausal women, while it increases the risk of postmenopausal breast cancer (WCRF/AICR, 2007; Renehan et al., 2008). Abdominal fatness and adult weight gain are positively associated with breast cancer, while physical activity is negatively associated with the risk (WCRF/AICR, 2007). Several studies examined the role of diet in relation to breast cancer risk (Kushi and Giowannucci, 2002; Linos et al., 2007; Michels et al., 2007; Kellen et al., 2009; Brennan et al., 2010) as well as physical activity (Friedenreich,

2010a; 2010b). Data regarding diet suggested that breast cancer risk is positively associated with high intake of red and processed meat, saturated fat, and polyunsaturated fatty acids, especially among women with estrogen receptor positive breast cancer. In turn, increased fiber, vegetable and fruit intakes was found to be negatively associated with breast cancer risk. The findings from these studies have been inconsistent, as diet vary among populations.

However, independently of the geographical region, the strongest protective effect against breast cancer development was exerted by exercise, pregnancy, lactation, consumption of soy products, followed by intake of fish and vegetables. These modifying factors for breast cancer were also reported for Asian women in recently published overview by Moore and Sobue (2009). Recent studies have focused on the role of the increased risk of dietary fat (Velie et al., 2000) and protective effect of vegetables and fruits (Michels et al., 2007), and phytoestrogens (Horn-Ross et al., 2001) in the etiology of breast cancer. The evidence for a protective effect of diet, physical activity and risk of 17 types of cancer based on systematic world literature reviews was judged by

<sup>1</sup>Faculty of Physical Culture and Health Promotion, University of Szczecin, <sup>2</sup>Department of Aesthetic Dermatology, Pomeranian Medical University, Szczecin, Poland \*For correspondence: joanna.kruk@univ.szczecin.pl

#### Joanna Kruk and Mariola Marchlewicz

the Expert Panel in their second report: Food, Nutrition, Physical Activity and the Prevention of Cancer: a Global Perspective (WCRF/AICR, 2007). The judgments made by the Panel the strength for evidence for food, nutrition and physical activity with the risk of cancer and personal recommendations were given.

The recommendations include: moderate physical activity for at least 30 minutes daily, sparingly consumption of energy-dense food, avoiding of sugary drinks, eating of at least five portion/servings ( $\geq$ 400g) of non-starchy vegetables and fruits daily. In addition, people who eat red meat should limit its consumption to <500g a week. For people who intake alcohol its consumption should be limited to no more than two drinks (10-15 grams of ethanol) daily for men and one drink daily for women. Although dietary energy restrictions resulted in reduction of the incidence of mammary tumors as observed in animals, a number of studies in humans showed that high levels of energy intake have not been consistently linked with breast cancer, especially among physically active women (Chang et al., 2006).

The present study was designed to evaluate the influence of the joint effect of dietary fat and overall physical activity on breast cancer risk. Our a priori hypothesis was that women declaring the low physical activity and high fat intake of animal fat would have an increased risk of breast cancer, compared to women that were moderate or vigorous active or their dietary fat intake was less.

#### **Materials and Methods**

The details of the study design and data collection for the Region of Western Pomerania study have been previously described (Kruk, 2007; 2009). Briefly, case subjects were diagnosed with histologically confirmed invasive breast cancer, operated between 1999 through 2006, and had not secondary breast cancer. Controls were frequency matched on 5-year age group and place of residence. They were required to have no personal history cancer and earlier physical limitation. Eligibility criteria for both cases and controls were age between 25 and 79. The final study sample for the analysis was 858 cases and 1,085 controls (49.3% and 67.2%, respectively). The study was approved by the Ethics Committee of the Pomeranian Medical Academy in accordance with the Polish Department of Health and Human Services.

An 8 page self-administered structured questionnaire was used to collect detailed information on demographic factors, menstrual and reproductive history, family history of cancer, lifestyle habits (physical activity, dietary habits, sleeping, active and passive cigarette smoking, alcohol drinking, use of hormones, multivitamins supplement and breast screening history). All of the data were obtained up to the year before diagnosis (for cases) or recruitment (for controls).

Information on dietary intake, and alcohol drinking was gathered from each women using a separate section of questionnaire modeling on the Block et al. (1990) and Franceschi et al. (1993) food frequency questionnaires. This section included 18 main Polish-specific food groups: red meats (pork, beef, lamb; boiled, fried, canned) and alternatives, animal fat (bacon, grease and butter), milk and its products, grain products, plant fat (oils, margarine), vegetables, fruits, and juices. Subjects were asked to report types of food and alcoholic drinks as well as consumption frequency per week and portion size.

Lifetime physical activity patterns were assessed using a questionnaire modeled of those of Kriska et al. (1990) and Friedenreich et al. (1998) to allow for self-administration. Regular exercise and sports (40 items) participation beginning at age 14 and ending in the reference year, household and outdoor chores, the jobs held outside the home (20 items the most popular occupations) over lifetime were reported. Number years, months per year, weeks per month, day per week, and hours per day for each activity reported was recorded. For every specific activity a metabolic equivalent (MET) score was assigned using the Compendium of Physical Activities (Ainsworth et al., 1993; 2000). Total lifetime activity was calculated as the sum of the averages household, occupational and leisure-time activity, and evaluated by MET-hours per week per year (MET-h/week/year). The reproducibity of the reports of diet intake and physical activity was tested in 14 cases and 16 controls 2-3 months later

Unconditional logistic regression modeling was applied to assess the relationship between the diet factor score and breast cancer risk. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated and tests for a linear trend in the risk calculation were conducted using the statistical package STATISTICA 98 (Stat Soft Polsca, Poland).

The lifetime physical activity was categorized into quartile according to the distribution of the variable among control subjects. A two-sided P-test was used; P-values below 0.05 were considered significant. For each analyses, ORs were estimated separately in a model adjusted only for age (univariate model) and in a multivariate logistic model. Since the age adjusted ORs were similar compared to those for multivariate analyses, only the results from the latter are reported.

Trends were tested by entering the categorical variables as continuous variables in the models (the Wald test). All multivariate models included adjustment for age at enrolment, place of residence, education, marital status, family income, Body Mass Index (BMI) at reference year, age at menarche, menstrual cycles, parity, age at menopause, age at first birth, duration of breast feeding, use of oral contraceptives, Hormone Replacement Therapy (HRT), family history of breast cancer, psychological stress experience, active smoking, passive smoking, alcohol drinking, screening mammography or ultrasonic examination, physical activity and diet.

Discriminate analysis was performed in order finding an optimal model. The effect of adding and removing confounders on a model was evaluated by F-Fisher's test. The final models included only those confounders that were associated significantly with breast cancer and a risk factor and were found to influence the goodness of the model fit, as noted in the footnotes to the Tables reported in the paper.

## Table 1. Selected Demographic Characteristics and Known and Probably Risk Factors of the Study Subjects

Variables			Cases N=858	Controls N=1085	P-value <sup>a</sup>
Age (years) in reference year, mean (SD*)			55.30 (9.70)	54.80 (9.50)	0.2400
Education level					
Elementary school			262(30.50)	253 (23.30)	
Middle school			339(39.50)	379 (34.90)	
High school (university, academy)			257(30.00)	453 (41.80)	< 0.0001
Marital status (number, %)					
Never married			51 (5.90)	56 (5.20)	
Married	100.0		580(67.60)	<u>7</u> 36 (67.80)	
Widowed/divorced		67	227(26.50)	293 (27.00)	0.7400
Body mass index (kg/m <sup>2</sup> ), mean (SD)		0.5	26. <u>20<sup>0</sup>(4.7</u> 0)	20 <u>3</u> 3.30 (4.10)	0.0001
Age at menarche (years), mean (SD)			13.82 (1.61)	13.80 (1.57)	0.7600
Parity	75.0		1.90 (1.10)	1.91 ( <b>2.5:9</b> )	0.8000
Age at first birth (years), mean (SD)			23.35 (3.94)	23.97 (3.95)	0.0900
Age at menopause (years), mean (SD)#		56.3	48.7 <b>46.8</b> 5.3)	49.22 (4.65)	0.0700
Breast-feeding (months), mean (SD)			6.62 (7.52)	10.82 (10.37)	< 0.0001
Red meat consumption (servings/week), mean (SD)	50.0		2.24 (1.66)	<b>54.2</b> .10 (34.3)	0.0860
Saturated fat consumption (servings/week), mean (SD)			5.12 (2.70)	4.15 (2.72)	0.0001
Alcohol drinking (drinks <sup>‡</sup> /week), mean (SD)			1.20 (0.87)	1.14 (0.95)	0.2700
Vegetable consumption (servings/week), mean (SD)	25.0		3.35 (2.25)	4.31 (2.38)	< 0.0001
Fruits consumption (servings/week), mean (SD)	25.0		5.24 (2.63)	5.62 (2.41)	0.0009
Ever users of OCP (number, %)		31.3	153(17.80)	158 (1 <b>3.163</b> )	<0.0110
Ever users of HRT (number, %)			301(35.10)	<b>23.7</b> 371 (34.20)	0.6800
Stress experience (yes, number, %)	0		492(57.30)	497 (45.80)	< 0.0001
Active smokers (number, %)	0		413(48.20)	a) 355 (32.80)	< 0.0001
Passive smokers (number, %)		ent	458 (5.70)	ຍູ້ 347 (32.ອີ້ອີ)	< 0.0001
Family history of breast cancer in first degree female relativ	atm	138(∰6.10)	Ĕ 82 (7.∰))	< 0.0001	
Total lifetime physical activity, mean (SD)		crea	reg	ect Rer	
MET-hours/week/year		nt t	137.60 466.7)	J 58.5 (73.34)	< 0.0001

\*SD, standard deviation; MET, metabolic equivalent; HRT, hormonal replacement therapy; OCP, oral contraceptives; \* and a construction of wine or 30g of high-grade alcohols; \* due to missing values, some categories do not sum to 100%; \* and postmer pausal wore postme

# Results

The distribution of demographic characteristics and breast cancer risk factors among 858 breast cancer cases and 1085 controls have been previously described (Kruk, 2009) and are presented briefly in Table 1. Compared to the controls, cases subjects were somewhat less educated, had higher BMI, higher frequency of histories of breast cancer risk in the first degree female relatives, were more frequent active and passive smokers, saturated fat consumers, an oral contraceptive users, and more frequent experienced psychological stress. In addition, cases declared less frequent consumption of vegetables and fruits and were less likely to be physically active. No significant differences were found between the case and control subjects in age in the reference year, marital status, age at menarche, parity, age at first birth, age at menopause, average frequency of red meat consumption, alcohol intake and use of hormonal replacement therapy. Table 2 gives the results of multiple regression analyses conducted for the relationship between animal fat consumption and the breast cancer ratio in women of different physical activity levels. High animal fat consumption (≥5 servings/ week) significantly increased breast cancer risk from about 1.7 times (OR=1.66,95%CI=1.07-3.59) to 2.9 times (OR=2.90,95%CI=1.37-6.14), independently on physical activity level. The risk increased with increasing frequency of fat consumption, the results were statistically significant and supported by a trend-test.

The adjusted OR<sup>®</sup> and 95% CI for each tertile level of red meat consumption and quartile of total lifetime physical activity ar presented in Table 1. Only women in the lowest quartile of physical activity and the highest tertile of red meat consumption were at statistically significant 2.7 times increased risk (OR=2.7,95% CI=1.21-6.03, P trend<0.02), relative to their counterparts consuming red meat rarely.

30.0

30.0

30.0

None

Table 2 presents adjusted ORs for processed meat consumption among women of which physical activity was classified into quartiles. In multiple regression analysis, the highest quartile of the processed meat consumption was positively and statistically significantly associated with breast cancer risk (OR=1.78,95%CI=1.04-3.59) only among women of the lowest level of physical activity.

The relationship between poultry consumption and breast cancer risk did not differ according to physical activity level (Table 2). For example, the OR between the highest and the lowest tertile of poultry consumption was 0.86 (95%CI=0.37-2.01) for the lowest quartile of physical activity, and 0.57 (95%CI=0.22-1.51) for the highest quartile of physical activity and were not statistically significantly associated with breast cancer risk. However, the risks were suggestively decreased among women in the highest quartile of physical activity (P trend<0.01).

The plant fat consumption was negatively associated with breast cancer risk among women in the lowest and the highest quartiles of physical activity with the ORs (95%CI) of 0.57 (0.32-0.99) and 0.46 (0.26-0.81) (P trend<0.02 and <0.05, respectively), comparing the

Table 2. Odds Ratios Associated with Dietary Components

Servings of animal fat	Cases	Controls	OR (95% CI)		Cases	;	Controls	OR (95% CI)	_	
Animals fat consumption	n by quartiles	and total lifeti	me physical activity l	evels						
i initialis fat consumption	in by quartites <	:105 MET-h/we	æk	evens		105	<138 MET-h/v	veek		
≤2/wk	126	125	1.00 <sup>a</sup>		71		119	1.00 <sup>b</sup>		
3-4/wk	35	31	1.14(0.61-2.14)		29		43	0.91(0.46-1.80)		
5-6/wk	43	26	1.66(1.07-3.59)		43		32	2.82(1.44-5.53)		
≥7/wk	94	53	1.86(1.15-3.02)		70		45	2.80(1.59-4.53)		
P trend			<0.01					<0.0001		
	138	8-<170 MET-h/	week			≥1′	70 MET-h/wee	ek		
≤2/wk	54	118	1.00° <b>100.0</b>		89		206	$1.00^{d}$	100.0	
3-4/wk	19	36	1.24(0.59-2.62)	6.3	28	10.1	68	0.85(0.49-1.49)		6.3
5-6/WK	26	21	2.90(1.37-6.14)		50		240/2.3	1.76(1.00-3.10)		
≥//WK D trond	39	43	1.8/(1.00-3.53)		59		//	2.02(1.25-3.27)	75 200	
P trellu Ped meat consumption k	ny tartilas and	l total lifetime	<0.01 73.0	0				<b>40,0</b> 01	/ 5.00.0	
Red meat consumption t	by tertiles and	105 MFT_h/we	vek	.5		4605	-138 MET.h/v	veek		
<2/wk	210	181	1 00ª	56.3	158	10:0/-	179	1 00 <sup>b</sup>		56.3
3-4/wk	55	44	1.12(0. <b>50.0</b> 82)		44		5482	<b>a1.0</b> 1(0.62-1.65)	50.0	
≥5/wk	33	10	2.70(1.21-6.03)		10		11	1.14(0.44-2.96)	30.0	
P trend			<0.02					<0.59		
	138	8-<170 MET-h/	week		1	≥1′	70 MET-h/wee	ek 👘		
≤2/wk	101	160	<sub>1.00°</sub> 25.0		152	20.0	292	$1.00^{d}$	25.0	
3-4/wk	29	49	1.02(0.57-1.81)	31.3	36	38.0	75_	<b>31.3</b> 0(0.52-1.92)	30.0	31.3
≥5/wk	7	9	1.16(0.39-3.44)		23		2377	1.45(0.77-2.73)		
P trend			<0.79					<0.30		
Processed meat consump	ption by quar	tiles and total li	ifetime physical activ	ity level	S		۵	C	U U	>
	<	:105 MET-h/we	ek	lent		<b>B</b> 05	<138 M ET-h/v	veek	lon	rap
≤2/wk	100	102	1.00ª	atm	87	atm	102	₽ E OOp	2	the
3-4/wk	121	79	1.39(0.88-2.18)	tre	77	tre	80	1226(0.77-2.04)		mo
5-6/wk	54	39	1.62(0.90-2.91)	out	33	ith	32	1.84(0.97-3.48)		Che
≥/ D turn d	23	15	1.78(1.04-3.59)	ithe	16	≥ q	192	1.62(0.68-3.88)		itho
P trend	120	2 .170 MET L	<0.05	₹ q		) J S C 1'		<0.07		≥ q
~2/mlr	50	02		See	02	gssī	12m	1 00d		See
$\leq 2/WK$	38 56	93	$1.00^{\circ}$ 1.21(0.72.2.02)	gnc	83 77	dia	10 <u>4</u> 154	$1.00^{\circ}$ 1.01(0.67, 1.52)		gno
5-4/WK	15	80 26	1.21(0.72-2.03) 0.82(0.40, 1.72)	dia	22	۲۲	134	1.01(0.07 - 1.03) 1.11(0.65, 1.80)		dia
5-0/WK	0	9	1.72(0.62.4.8)	Ý	16	Nev	23	1.11(0.05-1.09) 1.25(0.59, 2.62)		Ź
≥7 Ptrend	)	)	~0.95	Nev	10		23	<pre></pre>		Nev
Poultry consumption by	tertiles and to	otal lifetime ph	vsical activity levels					<0.50		
roundy consumption of	<	:105 MET-h/we	ek			105	<138 MET-h/v	veek		
≤2/wk	194	160	1.00ª		160		149	1.00 <sup>b</sup>		
3-4/wk	87	60	1.11(0.71-1.73)		46		80	1.49(0.29-0.82)		
≥5/wk	17	15	0.86(0.37-2.01)		7		10	0.73(0.24-2.26)		
P trend			<0.93					<0.02		
	138	8-<170 MET-h/	week			≥1′	70 MET-h/wee	ek		
≤2/wk	98	141	1.00°		144		228	$1.00^{d}$		
3-4/wk	30	66	0.80(0.33-1.03)		57		143	0.60(0.38-0.85)		
≥5/wk	10	11	1.15(0.47-3.58)		8		22	0.57(0.22-1.51)		
P trend			< 0.42					<0.01		
Plant fat consumption	by quartiles	s and total life	etime physical activ	ity leve	ls					
2/ 1	<	:105 MET-h/we	ek		0.4	105	<138 MET-h/v	veek		
≤2/wk	120	71	$1.00^{a}$		84		80	1.00 <sup>b</sup>		
3-4/WK	13	/4	0.64(0.38-1.06)		55 27		65	0.70(0.39-1.27)		
⊃-0/WK	49 56	49 51	0.55(0.31-0.99)		31		49	0.04(0.34-1.21)		
≥//WK D trand	20	51	0.37(0.32-0.99)		51		45	0.79(0.42-1.49)		
r uena	120	R_~170 MET L	<0.02 week			<u>\</u> 1'	70 MET h/w~	<0.20		
<7/wk	130 44	60	1 00°		58	≥1	104	1 00 <sup>d</sup>		
$\leq 2/WK$ $3_2/Wk$	44	67	1.00		50 66		80	1 09/0 71 2 00		
>5/wk	19	36	0.48(0.22-1.05)		36		109	0.46(0.26-0.81)		
>7/wk	27	45	0.78(0.38-1.62)		49		100	0.73(0.44-1.24)		
P trend	2,	12	<0.17				100	0.055		

\*OR – odds ratio; CI – 95% confidence interval. \*Adjusted for age, BMI, education, breast-feeding, psychological stress, multivitamins supplement, family history of breast cancer, passive smoking; <sup>b</sup>Adjusted for age, BMI, education, breast-feeding, psychological stress, family history of breast cancer, passive smoking; <sup>c</sup>Adjusted for age, BMI, education, breast-feeding, psychological stress, family history of breast cancer, passive smoking; <sup>c</sup>Adjusted for age, BMI, education, breast-feeding, psychological stress, family history of breast cancer, passive smoking; <sup>c</sup>Adjusted for age, BMI, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, education, breast-feeding, psychological stress, passive smoking; <sup>d</sup>Adjusted for age, psychological stress, psychological stress, psychological stress, psychological stress, psychological stress, psychological stress, psychologic

highest versus the lowest quartile of intake (Table 2).

## Discussion

In this hospital-based case-control study, the main finding was positive association between high intake of animal fat and breast cancer risk, independently on physical activity level. In addition, an important positive association between high intake of red meat and breast cancer risk was observed among women in the lowest quartile of lifetime total physical activity. This study also showed that increased consumption of processed meat was positively associated with breast cancer risk among physically inactive women.

Yet, the relationship between animal fat and breast cancer risk is still a highly controversial topic. One casecontrol study conducted in Taiwan found that breast cancer risk was significantly increased with a higher consumption of fat intake, with an OR and 95%CI of 2.6 (1.4-5.0) in all women for the highest versus lowest quartile of fat intake (Lee et al., 2005). Some epidemiological studies found the linkage between red meat intake and diagnosis of breast cancer (Toniolo et at., 1994; Zheng et., 1998), others failed to observe any linkage (Ambrosone et al., 1998; Willett et al., 1998). The study by Lee et al. (2005) showed that the highest quartile of beef and pork intake significantly increased the risk among women aged ≤40y and among all women (aged 25-79 years) with an OR and 95%CI of 2.5 (1.0-6.0) and 2.5 (1.1-3.3) versus the lowest quartile of intake, respectively. A case-control study conducted in Shanghai (China) showed that a high consumption of total meat and red meat was positively associated with breast cancer risk (OR=1.58, 95%CI=1.22-2.04 and OR=1.53, 95%CI=1.19-1.96, respectively), for the highest quartile (Dai et al., 2002). In turn, the case-control studies among Chinese women observed a borderline significant increased risk of breast cancer in women who intake processed meat (OR=1.44, 95%CI=0.97-2.15, P trend=0.066 for the highest versus the lowest quartile intake) and a lack of association between total and red meat consumption and the risk (Zhang et al., 2009). These authors observed that the refined grain-meat-pickle was positively linked with breast cancer risk (OR=2.58, 95%CI=1.53-4.34) (Zhang et al., 2011).

A study carried out in Uruguay also reported that a high consumption of fried and processed meat was positively linked with breast cancer risk (OR=2.16, 95%CI=1.46-3.29 for the highest quartile) (Ronco et al., 2006). Positive association between a higher intake of processed and red meats and breast cancer risk was also observed by Knekt et al. (1994) among the Finish women and by Fung et al. (2005) among the US women. Consistent with these results, our study found that animal fat consumption for 5 or more servings per week versus two or fewer servings per week exhibited a 2-fold increase in the risk for breast cancer even among highly physically active women (≥170 MET-h/week) and a 2.8-fold increase in the risk among women who indicated 105-<138 MET-h/week total physical activity. In addition, a modest no significant positive associations were observed between high intake of animal products and breast cancer risk among women

declaring higher levels of activity. However, others studies did not observed a significant relationship between total and red meat intake and breast cancer risk. For example, a case-control study of German women reported no association between high meat and deep-frying fat intake and breast cancer risk (OR=0.96, 95%CI=0.81-1.13) independently on estrogen and progesterone receptor (ER/ PR) status (Buck et al., 2010). In contrast other authors found that consumption of red meat for more than 5 servings per day versus one serving per day was positively associated with ER+/PR+ breast cancer risk (OR=1.42, 95%CI=1.06-1.90) among premenopausal women (Cho et al., 2006).

Our analysis on the relationship between poultry consumption and breast cancer risk in subgroup of physical activity has displayed about 20-40% significant decrease in the risk of breast cancer when the second tertile of poultry serving was compared with the lowest, among subgroups of women with dose of activity  $\leq$ 138 MET-h/ week. This finding is, for example, in contrast to a case-control study of Zhang et al. (2009), who did not observed a significant relationship between poultry consumption and breast cancer risk (OR=0.82, 95%CI=0.53-1.27). Previous studies reported inconsistent findings on the relationships between poultry intake and breast cancer (Franceschi et al., 1995; Dai et al., 2002; Cho et al., 2003; Ronco et al., 2003; Shannon et al., 2005; Taylor et al., 2007).

We found that plant fat consumption was significantly associated with a 43% decreased breast cancer risk in sedentary women, comparing the highest to the lowest tertile of intake. The inverse association between plant fat and the risk of breast cancer in our study agrees with the previously published data on plant fat. Some epidemiological studies reported that olive oil and others oils, the compounds having high monounsaturated fatty acids, decreased breast cancer risk (Willett, 1997; Kushi and Giovannucci, 2002; Kushi et al., 2006; Kushi et al., 2012). Contrary to these observations, some studies found that polyunsaturated fats have not been linked with breast cancer (Micozzi, 1985; Hunter et al., 1996; Holmes et al., 1999).

Both animal and human epidemiological studies recognized that moderate regular physical activity and diet effect on susceptibility to breast cancer (Shephard and Shek, 1998). Several biological mechanisms may explain our findings that animal fat, red meat intake were associated with possible increased breast cancer risk. Among them are: modification of body fat content, hormone concentration and immune response. Animal based fat intake has been reported to raise the synthesis of estrogens from androstendione and insulin levels, compounds which promote tumor growth (Cho et al., 2003; 2006).

In turn, heat-processed and cooked red meat is a source of carcinogens, such as heterocyclic amines, N-nitroso containing compounds, and polycyclic aromatic hydrocarbons. The compounds were found to be extremely mutagenic agents in numerous in vitro and in vivo systems; they are able to increase breast cancer risk (Zheng et al., 1998; Felton et al., 2002). Knize and co-workers (1985;

#### Joanna Kruk and Mariola Marchlewicz

1998) observed that concentration of these carcinogens depends on cooking method, time, temperature and protein source. Additionally, red meat is rich in heme iron. Metal ions, specifically iron are necessary for the generation of a highly biologically toxic oxygen species, like hydroxyl radical. When the formation of the oxygen species is high and when the antioxidants concentrations are low, the species may induce cellular changes similar to carcinogens (the state is called oxidative stress) (Wisman and Halliwell, 1996). Several mechanisms have been suggested for the role of iron ions in estrogen induced carcinogenesis, like release of iron from ferritin, iron mediated production of hydroxyl radicals and formation of lipid hydroperoxides as a products of the hydroxyl radical reaction with lipids (Liehr and Jones, 2001). Taken together, iron ions may indirectly affect cytoplasmic and nuclear signal transduction pathways under oxidative stress (Brown and Bicknell, 2001; Liehr and Jones, 2001). The breast cancer preventive abilities of monounsaturated fatty acids present in vegetable oils may be attributed to their antioxidant action, thereby their effect on cell signaling (Lander, 1997; Allen and Tresini, 2000). Monounsaturated acids are able scavenger reactive oxygen species and may play an important function in the regulation of cell growth, differentiation, and apoptosis.

This study has typical weaknesses and strengths of retrospective case-control examination. A lower response rate among cases (54.7%) than among controls subjects (84.3%) may have introduced a potential selection bias. This error should be in part limited on account of the comparable place of residence of cases and controls. In addition, both groups of subjects were similar in terms of age enrollment and several gynaecological factors related to breast cancer risk, alcohol drinking, ever use HRT, and marital status. There is also still limited awareness of the influence of diet and physical activity on the risk of breast cancer among Polish women. Since our study interviewed women retrospectively, there is the likelihood of recall bias. It was possible that subjects could have changed their dietary components over time. In addition this study used a questionnaire that was validated with respect to physical activity assessment but not with respect to diet, thus, misclassification of the dietary pattern could also occur, although the section of questionnaire included 18 main Polish - specific food groups. Recall bias was minimized by estimation cancer risk more comprehensibly. Because intake of different food, nutrients or others lifestyle components may affect cancer risk all logistic models were controlled for other food groups, known breast cancer risk factors and potential confounders. Similarly as most studies we did not analyze the diet in relation to estrogen receptor/progesterone receptor (ER/PR) status of tumor. However, the findings in this respect are inconsistent. Also, this study measured only intake frequencies of dietary variables and a portion size and was unable to adjust the calculated ORs for energy intake, and to control for body weight changes over lifetime. The next methodological limitations, i.e. misclassification bias in the evaluation of physical activity, was addressed by measure of household, occupational and recreational lifetime physical activity. Nevertheless, our positive results for the animal products

intake in relation to breast cancer risk are in accordance with some findings from other studies. This positive association is not surprising as animal fats and processed meat have high loading in the Polish dietary pattern.

Strengths of this study are: a large sample of case and control subjects, histological confirmation of invasive breast cancer, a comprehensive assessment of confounders relating to reproductive, lifestyle, anthropometric risk factors and family history of breast cancer. Major strength was the ability to carry out an adequate adjustment for exposure to potential confounders. Another major strength of this study is an assessment of the full range of lifetime physical activity that contribute importantly to energy expenditure, and that a dose-response relation over different levels of variables was tested in all analyses. To our knowledge, this is the first study that examined the relationship between breast cancer and animal fat intake in subgroups analyses by physical activity level among Polish women.

In conclusion, the findings of this paper indicate that consumption of animal fat, red meat and processed meat was associated with increased breast cancer in Polish sedentary women. Conversely, high intake of plant fat was negatively linked with breast cancer among sedentary women. These results agree with American Cancer Society Guideliness on Nutrition and Physical Activity for Cancer Prevention that emphasize the importance of limitation of processed and red meats consumption and adoption of a physically active lifestyle (Kushi et al., 2006; 2012). The best way to adopt a physically active lifestyle and a healthy body weight by balancing caloric intake with energy expenditure is engaging in different recreational forms of exercise, e.g. Wordic Walking (Piech and Raczyńska, 2010) or tourism (Hunter-Jones 2004; Lee 2010). Although the research on the relationship between health and tourism is limited, the growing popularity of Nordic Walking and international tourism contribute to growth in interest in research in these fields. To obtain public health implication, these findings should be confirmed in studies with a validated food frequency questionnaire, all relevant items in food groups, and analyzed in subgroups by physical activity level and ER/PR status of tumor.

### References

- Ainsworth BE, Haskell WL, Leon AS, et al (1993). Compendium of physical activities: classification of energy costs of human physical activities. *Med Sci Sports Exerc*, 25, 71-80.
- Ainsworth BE, Haskell WL, Whitt WC, et al (2000). Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc*, **32**, 498-516.
- Allen RG, Tresini M (2000). Oxidative stress and gene regulation. *Free Radic Biol Med*, **28**, 463-99.
- Ambrosone CB, Freudenheim JL, Sinha R, et al (1998). Breast cancer risk, meat consumption and N-acetyltransferase (NAT2) genetic polymorphisms. *Int J Cancer*, **75**, 825-30.
- Block G, Hatman AM, Naughton D (1990). A reduced dietary questionnaire: development and validation. *Epidemiology*, 1, 58-64.
- Brennan SF, Cantwell MM, Cardwell CR, Velentzis LS, Woodsite JV (2010). Dietary patterns and breast cancer risk: a systematic review and meta-analysis. *Am J Clin Nutr*, **91**,

### DOI:http://dx.doi.org/10.7314/APJCP.2013.14.4.2495 Dietary Fat and Physical Activity Associations with Breast Cancer in Poland

1294-302.

- Brown NS, Bicknell R (2001). Hypoxia and oxidative stress in breast cancer. Oxidative stress: its effect on the growth, metastatic potential and response to therapy of breast cancer. *Breast Cancer Res*, **3**, 323-7.
- Bružewicz S (2008). Epidemiologia raka piersi, Portal Forum Zdrowia, http://forumzdrowia.pl, 14.08.2008.
- Buck K, Vrieling A, Flesch-Janys D, Chang-Claude J (2011). Dietary patterns and the risk of postmenopausal breast cancer in a German case-control study. *Cancer Causes Control*, 22, 273-82.
- Chang SC, Ziegler RG, Dunn B, et al (2006). Association of energy intake and energy balance with postmenopausal breast cancer in the prostate, lung, colorectal, and ovarian cancer screening trial. *Cancer Epidemiol Biomarkers Prev*, 15, 334-41.
- Cho E, Chen WY, Hunter DJ, et al (2006). Red meat intake and risk of breast cancer among premenopausal women, *Arch Int Med*, **166**, 2253-9.
- Cho E, Spiegelman D, Hunter DJ, et al (2003). Premenopausal fat intake and risk of breast cancer. *J Natl Cancer Inst*, **95**, 1079-85.
- Dai Q, Shu XO, Jin F, et al (2002). Consumption of animal foods, cooking methods, and risk of breast cancer. *Cancer Epidemiol Biomarker Prev*, **11**, 801-8.
- Felton JS, Knize MG, Salmon CP, Malfatti MA, Kulp KS (2002). Human exposure to heterocyclic amine food mutagens/ carcinogens: relevance to breast cancer. *Environ Mol Mutagen*, **39**, 112-8.
- Fraceschi S, Favero A, La Vecchia C, et al (1995). Influence of food groups and food diversity on breast cancer in Italy. *Int* J Cancer, 63, 785-9.
- Franceschi S, Negri E, Salvini S, et al (1993). Reproducibility of an Indian food frequency questionnaire for cancer studies: results for specific food items. *Eur J Cancer*, **29**, 2298-305.
- Friedenreich CM (2010). Physical activity and breast cancer: Review of the epidemiologic evidence and biological mechanisms. In "Clinical Cancer Prevention" (Senn H-J and Otto F (eds) Springer-Verlag Berlin Heidelberg, 2010, Chapt.11).
- Friedenreich CM, Courneya KS, Bryant HE (1998). The lifetime total physical questionnaire: development and reliability. *Med Sci Sports Exerc*, **30**, 266-74.
- Friedenreich CM, Neilson HK, Lynch BM (2010). State of the epidemiological evidence on physical activity and cancer prevention. *Eur J Cancer*, 46, 2593-604.
- Fung TT, Hu FB, Holmes MD, et al (2005). Dietary patterns and the risk of postmenopausal breast cancer. *Int J Cancer*, **116**, 116-21.
- Holmes MD, Hunter DJ, Colditz GA, et al (1999). Association of dietary intake of fat and fatty acids with risk of breast cancer. *JAMA*, **281**, 914-20.
- Horn-Ross PL, John EM, Lee M, et al (2001). Phytoestrogen consumption and breast cancer risk in multiethnic population: the Bay Area Breast Cancer Study. Am J Epidemiol, 154, 434-41.
- Hunter DJ, Spiegelman D, Adami HO, et al (1996). Cohort studies of fat intake and the risk of breast cancer: a pooled analysis. *N Engl J Med*, **334**, 356-61.
- Hunter-Jones P (2005). Cancer and tourism. *Ann Tourism Res*, **32**, 70-92.
- Jemal A, Siegel R, Ward E, et al (2008). Cancer Statistics 2008. CA Cancer J Clin, **58**, 71-96.
- Kellen E, Vansant G, Christiaens M-R, Neven P, Limbergen EV (2009). Lifestyle changes and breast cancer prognosis: a review. *Breast Cancer Res Treat*, **114**, 13-22.

Knekt P, Steineck G, Jarvinen R, Hakulinen T, Aroma A (1994).

Intake of fried meat and risk of cancer : a follow-up study in Finland. *Int J Cancer*, **59**, 756-60.

- Knize MG, Anderson BD, Healy SK, et al (1985). Effect of temperature, patty thickness and fat content on the production of mutagens in fried ground beef. *Food Chem Toxicol*, 23, 1035-40.
- Knize MG, Sinha R, Rothman N, et al (1998). Heterocyclic amine content in restaurant-cooked hamburgers, steaks, ribs, and chicken. J Agric Food Chem, 46, 4648-51.
- Kriska AM, Knowler WC, La Porte RE, et al (1990). Development of questionnaire to examine relationship of physical activity and diabetes in Pima Indians. *Diabetes Care*, **13**, 401-11.
- Kruk (2007). Association of lifestyle and other risk factors with breast cancer according to menopausal status: A case-control study in the Region of Western Pomerania. Asian Pacific J Cancer Prev, 8, 24-35.
- Kruk J (2009). Intensity of lifetime physical activity and breast cancer risk among Polish women. J Sports Sciences, 27, 437-45.
- Kushi L, Giovannucci E (2002). Dietary fat and cancer. *Am J Med*, **113**, 63-70.
- Kushi LH, Byers T, Doyle C, et al (2006). American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer J Clin*, **56**, 254-81.
- Kushi LH, Doyle C, McCullough M, et al (2012). American Cancer Society Guidelines on nutrition and physical activity for cancer prevention: reducing the risk of cancer with healthy food choices and physical activity. *CA Cancer J Clin*, **62**, 30-67.
- Lander HM (1997). An essential role for free radicals and derived species in signal transduction. *FASEB J*, **11**, 118-24.
- Lee CG (2010). Health care and tourism: Evidence from Singapore. *Tourism Management*, **31**, 486-8.
- Lee MM, Chang IYH, Horng CF, et al (2005). Breast cancer and dietary factors in Taiwanese women. *Cancer Causes and Controls*, **16**, 929-37.
- Liehr JG, Jones S (2001). Role of iron in estrogen-induced cancer. *Curr Med Chem*, **8**, 839-49.
- Linos E, Holmes MD, Willett WC (2007). Diet and breast cancer. *Curr Oncol Report*, **9**, 31-41.
- Michels KB, Mohllajee AP, Roset-Bahmanyar E, Beehler GP, Moysich KB (2007). Diet and breast cancer, a review of the prospective observational studies. *Cancer, Suppl*, **109**, 2712-49.
- Micozzi MS (1985). Nutrition, body size, and breast cancer. *Yearbk Phys Anthropol*, **28**, 175-206.
- Moore MA, Sobue T (2009). Strategies for cancer control on an organ-site basis. *Asian Pacific J Cancer Prev*, **10**, 149-64.
- Parkin DM, Bray F, Ferlay J, Pisani P (2005). Global cancer statistics 2002. CA Cancer J Clin, 55, 74-108.
- Piech K, Raczyńska B (2010). Nordic Walking a versatile physical activity. *Pol J Sport Tourism*, **17**, 69-78.
- Renehan AG, Tyson M, Egger M, et al. 2008). Body-mass index and incidence of cancer: a systematic review and meta-analysis of prospective observational studies. *Lancet*, **371**, 569-78.
- Ronco AL, De Stefani E, Boffetta P, et al (2006). Food patterns and risk of breast cancer; a factor analysis study in Uruguay, *Int J Cancer*, **119**, 1672-8.
- Ronco AL, De Stefani E, Febra (2003). White meat intake and the risk of breast cancer: a case-control study in Montevideo, Uruguay. *Nutr Res*, **23**, 151-62.
- Shannon J, Ray R, Wu C, et al (2005). Food and botanical grouping and risk of breast cancer: case-control study in

Joanna Kruk and Mariola Marchlewicz

Shanghai, China. Cancer Epidemiol Biomarkers Prev, 14, 81-90.

- Shephard RJ, Shek PN (1998). Association between physical activity and susceptibility to cancer. *Sports Med*, **26**, 293-315.
- Taylor EF, Burley VJ, Greenwood DC, Cade JE (2007). Meat consumption and risk of breast cancer in the UK women's cohort study. *Br J Cancer*, **96**, 1139-46.
- Toniolo P, Riboli E, Shore RE, Pasternack BS (1994). Consumption of meat, animal products, protein, and fat and risk of breast cancer: a prospective cohort study in New York. *Epidemiology*, **5**, 391-7.
- Velie E, Kulldorff M, Schairer C, et al (2000). Dietary fat, fat subtypes, and breast cancer in postmenopausal women: a prospective cohort study. J Natl Cancer Inst, 92, 833-9.
- Willett WC (1997). Specific fatty acids and prostate cancer: dietary intake. Am J Clin Nutr, 66, 1557-63.
- Willett WC, Hunter DJ, Stampfer MJ, et al (1998). Dietary fat and fiber in relation to risk of breast cancer: an 8-year follow-up. *JAMA*, **268**, 2037-44.
- Wisman H, Halliwell B (1996). Damage to DNA by reactive oxygen and nitrogen species: role in inflammatory disease and progression to cancer. *Biochem J*, **313**, 17-29.
- World Cancer Research Fund/American Institute for Cancer Research, Food, Nutrition, Physical Activity, and the Prevention of Cancer: a Global Perspective, The Panel's judgements Washington, DC: AICR 2007, 8–14, 198–200, 289–295.
- Zhang C-X, Ho SC, Chen Y-M, et al (2009). Meet and egg consumption and risk of breast cancer among Chinese women. *Cancer Causes Control*, **20**, 1845-53.
- Zhang C-X, Ho SC, Fu J-A, et al (2011). Dietary patterns and breast cancer risk among Chinese women. *Cancer Causes Control*, **22**, 115-24.
- Zheng W, Gustafson DR, Sinha R, et al (1998). Well-done meat intake and the risk of breast cancer. *J Natl Cancer Inst*, **90**, 1724-29.