

**FREQUENCY OF SOME ENVIRONMENTAL FACTORS
WITH POTENTIAL RELATIONS TO BREAST DISEASES
AMONG A GROUP OF EGYPTIAN FEMALES**

[3]

**El Sobky, Abeer, F.⁽¹⁾; El Bokhary, M.⁽¹⁾; AwadAllah, Hala, I.⁽¹⁾
and Abdel Hamid, Hanaa, A.⁽²⁾**

1) Medical Sciences Department, Institute of Environmental Studies and Research, Ain Shams University 2) Radio-diagnosis Department, Faculty of Medicine, Ain Shams University

ABSTRACT

This study was conducted to highlight the significant environmental factors that may have impacts on breast benign and malignant diseases. This study was carried out on one hundred females who came to radiology department to perform mammographic examination. A complete personal and family history of patients was taken, clinical examination was done, all cases were asked to fill a questionnaire about how often they deal with each item of the environmental factors including (alcohol, caffeine, smoking, use of some material that are commonly used in daily life and known to have estrogenic effect including (food and soda cans, plastic containers, insecticides, detergents and cleaning agent, deodorants and cosmetics) then digital mammographic examination was done for all cases. Cases with high 17 β Estradiol hormone blood level were excluded from the study. The quantitative data were presented as mean and standard deviations. Also qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by using Chi-square test while the comparison between more than two groups with quantitative data were done by using One Way ANOVA. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant at the level of < 0.05 . Occurrence of benign and malignant lesions, breast calcifications and axillary lymphadenopathy was significantly associated to environmental estrogens containing items namely; use of diet & soda cans, use of insecticides, use of detergents & cleaning agents, use of deodorants, use of

cosmetics and use of plastic containers. Age, BMI of the patient and previous mammographic examinations was significantly associated to occurrence of benign and malignant lesions, breast calcifications and axillary lymphadenopathy. No significant association found between alcohol intake and axillary lymphadenopathy and between caffeine intake and breast calcifications. Caffeine intake, alcohol intake and smoking had no significant association with occurrence of benign and malignant lesions.

Keywords: Environmental estrogens, breast disease, benign breast lesions, digital mammography, breast calcification, breast cancer.

INTRODUCTION

Breast disease in women encompasses a spectrum of benign and malignant disorders (Morrow, 2000). Benign breast disease (BBD) has a high prevalence and a noticeable impact on women's quality of life (Friedenreich *et al.*, 2000). In less-developed countries, breast cancer is the leading cause of cancer death in women; in developed countries, however, it has been surpassed by lung cancer as a cause of cancer death in women. In United States, breast cancer accounts for 29% of all cancers in women and is second only to lung cancer as a cause of cancer deaths. Several risk factors have been found to be clinically useful for assessing a patient's risk of breast cancer. Many of these factors form the basis of breast cancer risk assessment tools currently being used in the practice (Chalasani, 2017).

There are several breast imaging modalities available such as Ultrasound, CT, Digital Mammography, MRI and scintimammography. Mammograms are X-ray images of the breast. The images can be captured on film or stored directly onto a computer (digital). The aim of mammography is to obtain an optimum image along with maximum breast tissue visualization (Popli *et al.*, 2014).

A potential risk factor for breast cancer is exposure to environmental estrogens, a group of synthetic substances found in the environment that, when absorbed into a person's system, function in a similar way to estrogen. Estrogen stimulates breast cell growth, and exposure to estrogen over long periods of time, without any breaks, can increase the risk of breast cancer (Kane, 2013). Environmental estrogens are connected to everything from PMS (Premenstrual Syndrome) to cancer and reproductive problems in animals. In fact, environmental estrogens have been found to change our genes and give our bodies the instructions to produce cancer (Evans, 2009).

The present study was designed as a cross-sectional study in 2015 and 2016 to detect the association of exposure to some environmental factors (with more concern to those related to environmental estrogens) with different breast pathologies especially malignant breast diseases, aiming to reduce risk of breast cancer as much as possible.

SUBJECTS AND METHODS

The present study was designed as a cross-sectional study. It was conducted from March 2015 to July 2016 on one hundred females who came to radiology department to perform mammographic examination; this was done in Central Hospital Egypt (Nasr City next to City stars).

A standardized epidemiological questionnaire including age, smoking status, alcohol use, and family history of cancer was used to collect personal data through in-person interviews.

Inclusion criteria are:

- Patient of known breast disease who came for follow up.
- Patients who came for regular check up.
- Patient who discovered breast abnormality during self-examination or medical assessment.

Exclusion criteria are:

- Taking any hormonal treatment.
- Having any endocrinal disease.
- Having high blood estrogen level.
- Lactating women.
- Lesions due to trauma.
- Females less than 40 years old.

A complete personal and family history of patients was taken. Clinical examination was done including inspection and palpation. All cases were asked to fill a questionnaire about how often they deal with each item as follows: Never (meaning that the patient never uses this item), occasionally (meaning that the patient uses this item once or twice per week) or daily (meaning that the patient uses this item every day). Items included in the questionnaire were alcohol, caffeine, smoking, use of some material that are commonly used in daily life and known to have estrogenic effect including (food and soda cans, plastic containers, insecticides, detergents and cleaning agent, deodorants and cosmetics).

Digital mammographic examination: Mammography was done for all cases as follows: One breast at a time was rested on a flat surface that contains the X-ray plate. A compressor was pressed firmly against the breast to help

flatten out the breast tissue. The X-ray picture was taken while patient is holding her breath. Routine views were obtained: (Cranio-caudal view -top to bottom-and Medio- lateral oblique) view.

Laboratory examination: Blood samples were collected for testing 17 β Estradiol hormone at the follicular phase of the menstrual cycle. Cases with high 17 β Estradiol hormone blood level were excluded from the study.

Statistical analysis: Data were collected, revised, coded and entered to the Statistical Package of Social Science (IBM SPSS) version 23. The quantitative data were presented as mean and standard deviations. Also qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by using Chi-square test while the comparison between more than two groups with quantitative data were done by using One Way ANOVA. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant at the level of < 0.05 .

RESULTS

Breast abnormalities seen in digital mammography were as follows:

- a) Breast calcifications: The percentage of cases that showed no calcifications in mammography was 34%, typically benign calcifications was 49% and cases showed suspicious calcifications was 17% as shown in figure (1).
- b) Enlarged axillary lymph nodes: Cases that showed no lymph nodal enlargement in mammography was 36%, inflammatory lymph nodal

enlargement was 56% and malignant lymph nodal enlargement was 8% as shown in figure (2).

c) Benign and malignant breast lesions: Cases that categorized as BIRADS II (benign lesions) in mammography was 51%, cases categorized as BIRADS III (probably benign lesions) in mammography was 14%, cases categorized as BIRADS IV (probably malignant lesions) was 17% and cases categorized as BIRADS V (high suspicion of malignancy) was 18% as shown in figure (3).

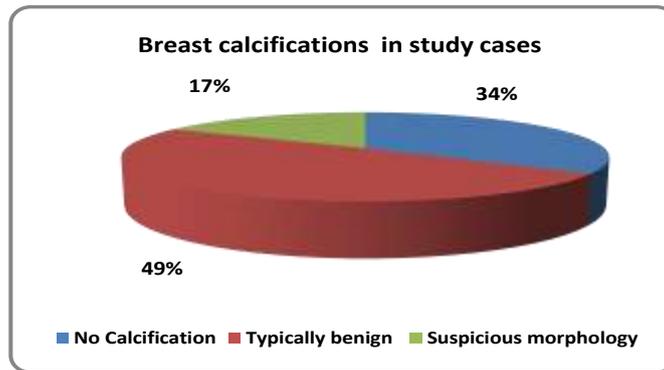


Figure (1): Percentage of breast calcifications in study cases

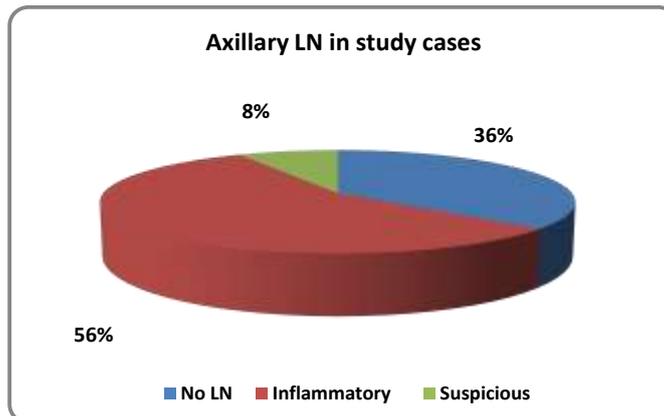


Figure (2): Percentage of axillary lymphadenopathy in study cases

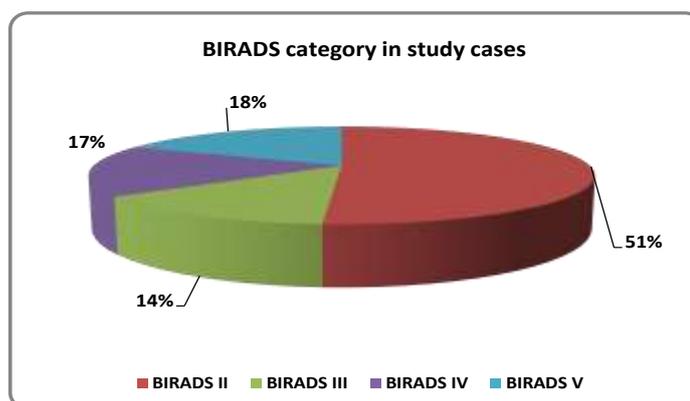


Figure (3): Percentage of different BIRADS categories in study cases

The association between the studied environmental factors and different pathological findings:

Data were collected, revised, coded and entered to the Statistical Package for Social Science (IBM SPSS) version 23. The quantitative data were presented as mean and standard deviations. Also qualitative variables were presented as number and percentages. The comparison between groups regarding qualitative data was done by using Chi-square test while the comparison between more than two groups with quantitative data were done by using One Way ANOVA. The confidence interval was set to 95% and the margin of error accepted was set to 5%. So, the p-value was considered significant at the level of < 0.05 .

Association between axillary lymphadenopathy and studied environmental factors:

Data in table (1) showed that there was high significant association between axillary lymphadenopathy and age, BMI of the patient, smoking, previous mammographic examinations and occupational radiological exposure. All studied factors of exposure to xenoestrogens including use of soda cans, use of insecticides, use of cosmetics, use of

deodorants, use of detergents and cleaning agents and use of plastic containers showed high significant association with axillary lymphadenopathy. Caffeine intake showed significant association with axillary lymphadenopathy. Meanwhile, no significant association was found between axillary lymphadenopathy and alcohol consumption.

Association between breast calcification and studied environmental factors: Data in table (2) showed that there was high significant association between breast calcifications and age, BMI of the patient, smoking, previous mammographic examinations and occupational radiological exposure. All studied factors of exposure to xenoestrogens including use of soda cans, use of insecticides, use of cosmetics, use of deodorants, use of detergents and cleaning agents and use of plastic containers showed high significant association with breast calcifications. Alcohol consumption showed significant association with breast calcifications. Meanwhile, no significant association was found between breast calcifications and caffeine intake.

Association between benign and malignant lesions (according to BIRADS classification) and studied environmental factors: Data in table (3) showed that there was high significant association between occurrence of benign and malignant lesions and age, BMI of the patient and previous mammographic examinations. All studied factors of exposure to xenoestrogens including use of soda cans, use of insecticides, use of cosmetics, use of deodorants and use of detergents and cleaning agents showed high significant association with axillary lymphadenopathy. Occurrence of benign and malignant lesions had significant association with occupational radiological exposure and use of plastic containers. Meanwhile, no significant association was found between

occurrence of benign and malignant lesions and alcohol consumption, Caffeine intake and smoking.

Table (1): Association between studied environmental factors and axillary lymphadenopathy

		Axill LN			Test value	P-value	Sig.
		Negative	Benign	Malignant			
		No. = 18	No. = 28	No. = 4			
Age	Mean±SD	52.00 ± 4.06	52.93 ± 6.45	65.00 ± 1.83	9.628	0	HS
	Range	40 – 56	40 – 61	63 – 67			
BMI	Mean±SD	31.79 ± 3.99	33.76 ± 2.47	39.99 ± 2.31	11.611	0	HS
	Range	24.34 – 35.61	27.69 – 36.57	37.2 – 42.02			
Alcohol	No exposure	15 (83.3%)	28 (100.0%)	4 (100.0%)	5.674	0.059	NS
	Mild exposure	3 (16.7%)	0 (0.0%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)			
Caffeine	No exposure	5 (27.8%)	14 (50.0%)	2 (50.0%)	5.808	0.214	S
	Mild exposure	5 (27.8%)	9 (32.1%)	0 (0.0%)			
	Severe exposure	8 (44.4%)	5 (17.9%)	2 (50.0%)			
Smoking	No exposure	18 (100.0%)	15 (53.6%)	0 (0.0%)	34.091	0	HS
	Mild exposure	0 (0.0%)	9 (32.1%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	4 (14.3%)	4 (100.0%)			
Previous mammogr	No exposure	14 (77.8%)	17 (60.7%)	1 (25.0%)	38.399	0	HS
	Mild exposure	4 (22.2%)	11 (39.3%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	0 (0.0%)	3 (75.0%)			
Occupation rad expo	No exposure	18 (100.0%)	28 (100.0%)	0 (0.0%)	50	0	HS
	Mild exposure	0 (0.0%)	0 (0.0%)	3 (75.0%)			
	Severe exposure	0 (0.0%)	0 (0.0%)	1 (25.0%)			
Soda cans	No exposure	0 (0.0%)	0 (0.0%)	2 (50.0%)	58.691	0	HS
	Mild exposure	0 (0.0%)	24 (85.7%)	2 (50.0%)			
	Severe exposure	18 (100.0%)	4 (14.3%)	0 (0.0%)			
Plastic containers	No exposure	0 (0.0%)	0 (0.0%)	0 (0.0%)	36.702	0	HS
	Mild exposure	0 (0.0%)	0 (0.0%)	3 (75.0%)			
	Severe exposure	18 (100.0%)	28 (100.0%)	1 (25.0%)			
Detr. Clean	No exposure	0 (0.0%)	0 (0.0%)	2 (50.0%)	39.063	0	HS
	Mild exposure	0 (0.0%)	14 (50.0%)	2 (50.0%)			
	Severe exposure	18 (100.0%)	14 (50.0%)	0 (0.0%)			
Insecticides	No exposure	0 (0.0%)	3 (10.7%)	2 (50.0%)	48.357	0	HS
	Mild exposure	0 (0.0%)	23 (82.1%)	2 (50.0%)			
	Severe exposure	18 (100.0%)	2 (7.1%)	0 (0.0%)			
Cosmetics	No exposure	12 (66.7%)	2 (7.1%)	0 (0.0%)	45.971	0	HS
	Mild exposure	6 (33.3%)	23 (82.1%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	3 (10.7%)	4 (100.0%)			
Deodorants	No exposure	18 (100.0%)	2 (7.1%)	0 (0.0%)	53.69	0	HS
	Mild exposure	0 (0.0%)	18 (64.3%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	8 (28.6%)	4 (100.0%)			

Table (2): Association between studied environmental factors and breast calcifications

		Calcifications			Test value	P-value	Sig.
		Negative	Benign	Malignant			
		No. = 17	No. = 24	No. = 9			
Age	Mean±SD Range	51.76 ± 4.05 40 – 56	52.29 ± 6.45 40 – 60	60.33 ± 5.74 49 – 67	8.018	0.001	HS
BMI	Mean±SD Range	31.89 ± 4.09 24.34 – 35.61	33.64 ± 2.65 27.69 – 36.33	36.44 ± 3.92 32.13 – 42.02	5.205	0.009	HS
Alcohol	No exposure Mild exposre Severe exposure	14 (82.4%) 3 (17.6%) 0 (0.0%)	24 (100.0%) 0 (0.0%) 0 (0.0%)	9 (100.0%) 0 (0.0%) 0 (0.0%)	6.195	0.045	S
Caffeine	No exposure Mild exposre Severe exposure	5 (29.4%) 5 (29.4%) 7 (41.2%)	14 (58.3%) 7 (29.2%) 3 (12.5%)	2 (22.2%) 2 (22.2%) 5 (55.6%)	8.252	0.083	NS
Smoking	No exposure Mild exposre Severe exposure	17 (100.0%) 0 (0.0%) 0 (0.0%)	14 (58.3%) 7 (29.2%) 3 (12.5%)	2 (22.2%) 2 (22.2%) 5 (55.6%)	22.321	0	HS
Previous mammogr	No exposure Mild exposre Severe exposure	14 (82.4%) 3 (17.6%) 0 (0.0%)	12 (50.0%) 12 (50.0%) 0 (0.0%)	6 (66.7%) 0 (0.0%) 3 (33.3%)	22.071	0	HS
Occupation rad expo	No exposure Mild exposre Severe exposure	17 (100.0%) 0 (0.0%) 0 (0.0%)	24 (100.0%) 0 (0.0%) 0 (0.0%)	5 (55.6%) 3 (33.3%) 1 (11.1%)	19.807	0.001	HS
Soda cans	No exposure Mild exposre Severe exposure	0 (0.0%) 0 (0.0%) 17 (100.0%)	0 (0.0%) 19 (79.2%) 5 (20.8%)	2 (22.2%) 7 (77.8%) 0 (0.0%)	41.511	0	HS
Plastic containers	No exposure Mild exposre Severe exposure	0 (0.0%) 0 (0.0%) 17 (100.0%)	0 (0.0%) 0 (0.0%) 24 (100.0%)	0 (0.0%) 3 (33.3%) 6 (66.7%)	14.539	0.001	HS
Detr. Clean	No exposure Mild exposre Severe exposure	0 (0.0%) 0 (0.0%) 17 (100.0%)	0 (0.0%) 14 (58.3%) 10 (41.7%)	2 (22.2%) 2 (22.2%) 5 (55.6%)	25.434	0	HS
Insecticides	No exposure Mild exposre Severe exposure	0 (0.0%) 0 (0.0%) 17 (100.0%)	0 (0.0%) 21 (87.5%) 3 (12.5%)	5 (55.6%) 4 (44.4%) 0 (0.0%)	61.521	0	HS
Cosmetics	No exposure Mild exposre Severe exposure	12 (70.6%) 5 (29.4%) 0 (0.0%)	2 (8.3%) 22 (91.7%) 0 (0.0%)	0 (0.0%) 2 (22.2%) 7 (77.8%)	57.808	0	HS
Deodorants	No exposure Mild exposre Severe exposure	17 (100.0%) 0 (0.0%) 0 (0.0%)	3 (12.5%) 18 (75.0%) 3 (12.5%)	0 (0.0%) 0 (0.0%) 9 (100.0%)	70	0	HS

Table (3): Association between studied environmental factors and occurrence of benign and malignant breast lesions

		BIRADS		Test value	P-value	Sig.
		Benign	Malignant			
		No. = 32	No. = 18			
Age	Mean±SD	51.72 ± 5.16	56.83 ± 7.13	-2.926	0.005	HS
	Range	40 – 57	43 – 67			
BMI	Mean±SD	32.67 ± 3.53	35.10 ± 3.60	-2.317	0.025	HS
	Range	24.34 – 36.33	29.72 – 42.02			
Alcohol	No exposure	29 (90.6%)	18 (100.0%)	1.795	0.18	NS
	Mild exposre	3 (9.4%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	0 (0.0%)			
Caffeine	No exposure	11 (34.4%)	10 (55.6%)	2.567	0.277	NS
	Mild exposre	11 (34.4%)	3 (16.7%)			
	Severe exposure	10 (31.3%)	5 (27.8%)			
Smoking	No exposure	22 (68.8%)	11 (61.1%)	3.282	0.194	NS
	Mild exposre	7 (21.9%)	2 (11.1%)			
	Severe exposure	3 (9.4%)	5 (27.8%)			
Previous mammogr	No exposure	17 (53.1%)	15 (83.3%)	15.413	0	HS
	Mild exposre	15 (46.9%)	0 (0.0%)			
	Severe exposure	0 (0.0%)	3 (16.7%)			
Occupation rad expo	No exposure	32 (100.0%)	14 (77.8%)	7.729	0.021	S
	Mild exposre	0 (0.0%)	3 (16.7%)			
	Severe exposure	0 (0.0%)	1 (5.6%)			
Soda cans	No exposure	0 (0.0%)	2 (11.1%)	23.291	0	HS
	Mild exposre	10 (31.3%)	16 (88.9%)			
	Severe exposure	22 (68.8%)	0 (0.0%)			
Plastic containers	No exposure	0 (0.0%)	0 (0.0%)	5.674	0.017	S
	Mild exposre	0 (0.0%)	3 (16.7%)			
	Severe exposure	32 (100.0%)	15 (83.3%)			
Detr. Clean	No exposure	0 (0.0%)	2 (11.1%)	12.565	0.002	HS
	Mild exposre	6 (18.8%)	10 (55.6%)			
	Severe exposure	26 (81.3%)	6 (33.3%)			
Insecticides	No exposure	0 (0.0%)	5 (27.8%)	22.917	0	HS
	Mild exposre	12 (37.5%)	13 (72.2%)			
	Severe exposure	20 (62.5%)	0 (0.0%)			
Cosmetics	No exposure	14 (43.8%)	0 (0.0%)	20.366	0	HS
	Mild exposre	18 (56.3%)	11 (61.1%)			
	Severe exposure	0 (0.0%)	7 (38.9%)			
Deodrants	No exposure	20 (62.5%)	0 (0.0%)	32.639	0	HS
	Mild exposre	12 (37.5%)	6 (33.3%)			
	Severe exposure	0 (0.0%)	12 (66.7%)			

DISCUSSION

Results of the present study showed highly significant association between age of the patient, BMI of the patient and previous mammographic examinations with occurrence of benign and malignant lesions, axillary lymphadenopathy and breast calcifications. These findings are close to that mentioned by Chalasani (2017) who stated that increasing age is an established risk factor for breast cancer.

Our results were matching with the results obtained by Goehring and Morabia (1997) and Friedenreich *et al.*, (2000) who stated that obesity has been identified as one of the only consistent risk factors for BBD.

Our results are not matching with the results obtained by other investigators as Hislop *et al.*, (1990), Soini *et al.*, (1981), Brinton *et al.*, (1981), Bianchi *et al.*, (1993) and Cole *et al.*, (1978) who mentioned that Obesity has been consistently shown to decrease risk of BPBD. Our results agreed with the results of Chalasani (2017) who mentioned that increased risk of postmenopausal breast cancer has been consistently associated with adult weight gain of 20-25 kg above body weight at age 18.

Considering relation between alcohol intake and breast diseases, our study showed a significant association between alcohol intake and breast calcifications. There was no significant correlation between alcohol intake and axillary lymphadenopathy and occurrence of benign lesions and occurrence of malignant lesions. These results contradict with the results of many other investigators as Chalasani (2017) who mentioned that increased risk of postmenopausal breast cancer has been consistently associated with regular, moderate consumption of alcohol (3-5 alcoholic beverages per week).

Coutelle *et al.*, (2004) and Fan *et al.*, (2000) and Fucic *et al.*, (2012) mentioned that Alcohol is related with increased risk of breast cancer development as even low alcohol consumption increases serum estradiol. In a study made on animal model, alcohol increases estradiol levels in dams, which leads to higher levels of ER alpha receptors in their offspring mammary gland and may launch tumori genesis (Hilakivi *et al.*, 2004).

Our results were similar to those mentioned by Rohan & Cook (1989) and Friedenreich *et al.*, (2000) who found no association between alcohol consumption and BPBD, even after stratification by degree of atypia.

Results of our study showed significant association between caffeine intake and enlarged axillary lymph nodes. No association found between caffeine intake and breast calcifications and occurrence of benign and malignant lesions. Our findings are close to those obtained by Rohan *et al.* (1989) and Boyle *et al.* (1984) who found no strong or consistent association between caffeine consumption and the potential deleterious effect on BPBD. Moreover, Webb *et al.* (2004) stated that caffeine restriction may improve symptoms of BBD. Morrow (2000) mentioned that caffeine avoidance has been a popular treatment measure in women with breast pain. Unfortunately, two randomized clinical trials by Ernster *et al.* (1982) and Allen & Froberg (1987) failed to demonstrate a therapeutic benefit for caffeine restriction.

Friedenreich *et al.* (2000) mentioned that no clear associations with BPBD were observed for some factors as caffeine intake.

Our results showed a high significant association between smoking and enlarged axillary lymph nodes and breast calcifications. Meanwhile, no

significant association was found between smoking and occurrence of benign and malignant lesions. This was similar to previous studies by *Yu et al.* (1992) and *Berkowitz et al.* (1985) who have shown no substantial effect of ever, former or current smoking; however one study by *Pastides et al.* (1987) found decreased risks and another study by *Nomura et al.* (1977) showed increased risks for BPBD associated with smoking. In this study, cigarette smoking as assessed as current, past, or passive smoking was not associated with an increased risk of BPBD. *Friedenreich et al.* (2000) mentioned that no clear associations with BPBD were observed for some factors as smoking.

Our study showed significant association between use of diet and soda cans, use of detergents and cleaning agents, use of insecticides, use of deodorants and use of cosmetics and occurrence of benign and malignant lesions, axillary lymphadenopathy and breast calcifications. Significant association was found between use of plastic containers and occurrence of benign and malignant lesions.

This was matching with *Andersen et al.* (2006) and *Calafat et al.* (2008) who stated that BPA is commonly found in polycarbonate plastic products including baby bottles, water bottles, food containers, in the linings of metal food cans and in dental sealants and composites. BPA has been found in over 90 % of the U.S. population age six and over, with highest concentrations in children ages 6–11.

Our results was similar to results published by *Lankester et al.* (2013), who mentioned that Triclosan is commonly found in antibacterial hand soaps, toothpastes and household cleaning supplies. Phthalate chemicals “soften” plastics to make them pliable. They are also found in personal care products,

food, plastic toys and household dust. Parabens are found in common personal care products including cosmetics and antiperspirants.

These results agreed with those obtained by Brody & Rudel (2003) and Brody *et al.* (2007) who mentioned that currently there are some 160 xenoestrogens that may be involved in breast cancer development. Women are the largest consumers of cosmetic products which may be a significant source of xenoestrogens. Some, such as metalloestrogens (e.g., aluminium salts), parabens, cyclosiloxanes, triclosan, UV screeners, phthalates, Aloe Vera extracts, and musk are present in numerous cosmetics products.

Also Rosenthal *et al.* (2004) mentioned that humans are exposed to these chemicals transcutaneously and measurable levels have been detected in human breast tissue.

Exposure to environmental estrogens was described by Andersen *et al.* (2006) who mentioned that Chemicals like polycyclic aromatic hydrocarbons (PAH), pesticides, polychlorinated biphenyl (PCB), dichlorodiphenyl-trichlorethane (DDT), some drugs (e.g. antiepileptic drugs), fungicides, cotinine, phytoestrogens, mycotoxins, bisphenol A (a plastics additive), phthalates, alkylphenols, and metalloestrogens mimic estrogen action, affect estrogen levels, or bind to estrogen receptors.

Fucic *et al.* (2012) stated that Xenoestrogens are present in a number of substrates such as cigarette smoke, automobile exhaust, chemical industry pollutants, grilled meat, volcano dust, forest fire smoke, milk, water, and cosmetic products. This means that all human population may be exposed to them.

REFERENCES

- Allen, S.S. and Froberg, D.G. (1987): The effect of decreased caffeine consumption on benign proliferative breast disease: a randomized clinical trial. *Surgery*. 101:720–30.
- Andersen, H.R.; Bonefeld-Jorgensen, E.C.; Nielsen, F.; Jarfeldt, K.; Jayatissa, M.N. and Vinggaard, A.M. (2006): Estrogen effects in vitro and in vivo of the fungicide fenarimol. *ToxicolLett*. 163(2):142–152.
- Berkowitz, G.S.; Canny, P.F.; Livolsi, V.A.; Merino, M.J.; O'Connor, T.Z. and Kelsey, J.L. (1985): Cigarette smoking and benign breast disease. *J Epidemiol Community Health* 39:308–13.
- Bianchi, S.; Palli, D.; Galli, M. and Zampi, G. (1993): Benign breast disease and cancer risk. *Crit Rev Oncol/Hematol* 15:221–42.
- Boyle, C.A.; Berkowitz, G.S. and LiVolsi, V.A. (1984): Caffeine consumption and fibrocystic breast disease: a case-control epidemiologic study. *J Natl Cancer Inst* 72:1015–19.
- Brinton, L.A.; Vessey, M.P.; Flavel, R. and Yeates, D. (1981): Risk factors for benign breast disease. *Am J Epidemiol* 113:203–14.
- Brody, J.G.; Rudel, R.A.; Michels, K.B.; Moysich, K.B.; Bernstein, L.; Attfield, K.R. and Gray, S. (2007): Environmental pollutants, diet, physical activity, body size and breast cancer. *Cancer (Suppl)* 109(12):2627–2634.
- Brody, J.G. and Rudel, R.A. (2003): Environmental pollutants and breast cancer. *EHP*. 111(8):1007–19.
- Calafat, A.M.; Ye, X.; Wong, L.Y.; Reidy, J.A. and Needham, L.L. (2008): Exposure of the U.S. population to bisphenol A and 4-tertiary-octylphenol: 2003–2004. *Environ Health Perspect*. 116(1):39–44. doi: 10.1289/ehp.10753.
- Cole, P.; Elwood, J.M. and Kaplan, S.D. (1978): Incidence rates and risk factors of benign breast neoplasms. *Am J Epidemiol* 108:112–20.

- Coutelle, C.; Hohn, B.; Benesova, M.; Oneta, C.M.; Quattrochi, P.; Roth, H.J.; Schmidt-Gayk, H.; Schneeweiss, A.; Bastert, G. and Seitz, H.K. (2004): Risk factor in alcohol associated breast cancer: Alcohol dehydrogenase polymorphism and estrogens. *Int J Oncology*. 25(4):1127–1132.
- Ernster, V.L.; Mason, L.; Goodson, W.H. 3rd; Sickles, E.A.; Sacks, S.T. and Selvin, S. (1982): Effects of caffeine-free diet on benign breast disease: a randomized trial. *Surgery* 91:263–7.
- Evans, K. (2009): Environmental Estrogen: Understand the Problems and Solutions. https://www.naturalnews.com/027729_estrogen_plastics.html Monday, December, 14th. Cited June 2017.
- Fan, S.; Meng, Q.; Gao, B.; Grossman, J.; Yadegari, M.; Goldberg, I.D. and Rosen, E.M. (2000): Alcohol stimulates estrogen receptor signaling in human breast cancer cell lines. *Cancer Res*. 60:5635–5639.
- Friedenreich, C.M.; Bryant, H.E.; Alexander, F. and Hugh, J. (2000): Risk factors for benign proliferative breast disease, *Int. J. Epidemiol* 29 (4): 637-644
- Fucic, A.; MarijaGamulin; ZeljkoFerencic; JelenaKatic; Martin, Krayner von Krauss; Alena, Bartonova and Domenico, F. Merlo (2012): Environmental exposure to xenoestrogens and oestrogen related cancers: reproductive system, breast, lung, kidney, pancreas, and brain. *Environ Health*. 11(Suppl 1): S8.
- Goehring, C. and Morabia, A. (1997): Epidemiology of benign breast disease, with special attention to histologic types. *Epidemiol Rev* 19:310–27.
- Hilakivi, C.L.; Cabanes, A.; de Assis, S.; Wang, M.; Khan, G.; Shoemaker, W.J. and Stevens, R.G. (2004): In utero alcohol exposure increases mammary tumorigenesis in rats. *Brit J Cancer*. 90:2225–2231.
- Hislop, T.G.; Band, P.R. and Deschamps, M. (1990): Diet and histologic types of benign breast disease defined by subsequent risk of breast cancer. *Am J Epidemiol* 131:263-70.

- Kane, J. (2013): 5 Ways to Reduce Your Exposure to Environmental Estrogens. <https://community.breastcancer.org/blog/for-our-mothers-and-others-environmental-estrogens/> May 8th. Cited July 2017.
- Lankester, J.; Patel, C.; Cullen, M.R.; Ley, C. and Parsonnet, J. (2013): Urinary triclosan is associated with elevated body mass index in NHANES. *PLoS One*. 8(11):e80057.
- Morrow, M. (2000): The Evaluation of Common Breast Problems. *Am Fam Physician*. 61(8):2371-2378.
- Nomura, A.; Comstock, G.W. and Tonascia, J.A. (1977): Epidemiologic characteristics of benign breast disease. *Am J Epidemiol* 105:505–12.
- Pastides, H.; Najjar, M.A. and Kelsey, J.L. (1987): Estrogen replacement therapy and fibrocystic breast disease. *Am J Prev Med* 3:282–86.
- Popli, M.B.; Rahul, Teotia; Meenakshi, Narang and Hare, Krishna (2014): Breast Positioning during Mammography: Mistakes to be Avoided *Breast Cancer (Auckl)*. 8: 119–124.
- Rohan, T.E. and Cook, M.G. (1989): Alcohol consumption and risk of benign proliferative epithelial disorders of the breast in women. *Int J Cancer* 43:631–36.
- Rosenthal, M.D.; Albrecht, E.D. and Pepe, G.J. (2004): Estrogen modulates developmentally regulated gene expression in the fetal baboon liver. *Endocrine*. 23(2-3):219–228.
- Update: February 22nd, Cited June 2017.
- Soini, I.; Aine, R.; Lauslahti, K. and Hakama, M. (1981): Independent risk factors of benign and malignant breast lesions. *Am J Epidemiol* 114: 507–14.
- Yu, H.; Rohan, T.E.; Cook, M.G.; Howe, G.R. and Miller, A.B. (1992): Risk factors for fibroadenoma: case-control study in Australia. *Am J Epidemiol* 135:247–58.

Webb, P.M.; Celia, Byrne; Stuart, J. Schnitt; James, L. Connolly; Timothy, W. Jacobs; Heather, J. Baer; Walter, C. Willett and Graham, A. Colditz (2004): A Prospective Study of Diet and Benign Breast Disease, Cancer Epidemiol Biomarkers Prev. 13(7):1106-13.

أثر بعض العوامل البيئية على أمراض الثدي المختلفة المشخصة بواسطة أشعة الثدي الرقمية

[3]

عبير السبكي⁽¹⁾ - محمود البخاري⁽¹⁾ - هالة عوض الله⁽¹⁾ - هناء عبد الحميد⁽²⁾
(1) قسم العلوم الطبية، معهد الدراسات والبحوث البيئية، جامعة عين شمس (2) قسم الأشعة التشخيصية، كلية الطب، جامعة عين شمس

المستخلص

أجريت هذه الدراسة لتسليط الضوء على العوامل البيئية الهامة التي قد يكون لها تأثير على أمراض الثدي الحميدة والخبيثة.

أجريت هذه الدراسة عشوائيا على مائة سيدة ترددن على قسم الأشعة التشخيصية لإجراء الفحص الإشعاعي للثدي. تم أخذ التاريخ الكامل للمرضى، وتم إجراء الفحص الطبي، وطلب من جميع الحالات الإجابة على استبيان حول مدى تعاملهم مع كل عامل من العوامل البيئية المختلفة بما في ذلك الكحول، والكافيين، والتدخين، استخدام بعض المواد المعروف أن لها تأثير الاستروجين البيئي بما في ذلك علب المواد الغذائية والصودا والحاويات البلاستيكية، والمبيدات الحشرية والمنظفات ومزيل رائحة العرق ومستحضرات التجميل. تم إجراء فحص الثدي الإشعاعي الرقمي لجميع الحالات، وتم استبعاد الحالات ذات مستوى هرمون استراديول عالي بالدم من الدراسة. وتم تحليل البيانات إحصائيا باختبار مربع كاي لتحديد التصاحب المتمثل بين العوامل المدروسة وأمراض الثدي المختلفة.

وكان وجود الأورام الحميدة والخبيثة مصاحبا بشكل كبير بمصادر الإستروجين البيئي وهي: استخدام المعلبات الغذائية والصودا، استخدام المبيدات الحشرية، استخدام الحاويات البلاستيكية، استخدام المنظفات، استخدام مزيل رائحة العرق واستخدام مستحضرات التجميل. عمر المريض، مؤشر كتلة الجسم للمريض، التعرض الإشعاعي المهني وفحوصات التصوير الإشعاعي السابقة للثدي كان لها علاقة كبيرة مع حدوث الأورام الحميدة والخبيثة. لم يتأثر حدوث الأورام الحميدة والخبيثة بتناول الكحول، تناول الكافيين والتدخين.

الكلمات المفتاحية: هرمون الاستروجين البيئي، آفات الثدي الحميدة، التصوير الرقمي الإشعاعي للثدي، نكلس الثدي، سرطان الثدي.