

The Effect of Drinking Coffee on Breast Cancer in Kirkuk \ Iraq

Dler. K. Qurbany

MSc microbiology, Kirkuk Department of Health, Ministry of Health/Iraq

Abstract: Purpose: Most of the available academic literature points towards the absence of correlation or a slightly negative correlation between consumption of coffee and occurrence of breast cancer. However, the extent of risk differs when considering separate subgroups of, say, menopausal status, hormonal profile of the tumor or genetic mutations. The present review, based on the extensive literature search, will shed light on the possible effect of a ubiquitous beverage, coffee, on the risk of breast cancer development in the general population, in different female groups, and the consequences of coffee drinking after a breast cancer diagnosis and treatment.

Results: This is consistent with the present review, which confirms that in the general population, coffee consumption is not significantly associated with the risk of breast cancer, and maybe in a weak protective effect at high levels of consumption. Moreover, coffee is associated inversely with the risk of breast cancer in postmenopausal women as well as women with BRCA1 mutation. Risk differences could be observed among people who are classified as being slow and fast caffeine metabolizers especially with regard to body weight. Post-diagnosis of breast cancer intake of coffee and subsequent post-surgery intake followed by tamoxifen use and/or radiotherapy has been associated with reduced recurrence events in the initial years following the diagnosis. The effects of coffee drinking seem to be less conclusive amongst some of the subpopulations namely premenopausal women, BRCA2 mutations, and individuals who were diagnosed with tumors with different hormonal patterns either being estrogen receptor/progesterone receptor positive or negative, thus requiring further research studies.

Keywords: BRCA mutations; Breast cancer; Coffee; Hormonal status; Menopause; Tamoxifen.

Introduction

The use of coffee and tea has also been suggested as a factor that can either increase or decrease the risk of getting breast cancer. Caffeine is an alkaloid which can be found naturally in plants; it is seen in coffee, tea and cocoa and it is an additive in many soft drinks and even pharmaceutical products. This substance falls under a category of purine-based substances which are all referred to as methylxanthines (1). The hypothesis that caffeine could increase the risk of breast cancer was as a result of the results that showed that women diagnosed with benign breast disease reported improvement in the symptoms after they had stopped taking methylxanthines in their diets (2–4). Investigations on animal models have shown that caffeine has the ability to either mediate or prevent mammary tumours, and depends on the species and strain of the rodent, and the tumorigenic phase (initiation/promotion) in which caffeine is given (5, 6). During the 1970s and 1980s, food rich in caffeine like tea, coffee and chocolate were suspected of being carcinogenic (7, 8).

Phenolic compounds are present in coffee and tea in large amounts and include significant amounts of different lignans (9). These lignans can be transformed into enter lactone and

enterodiol which are both antiestrogenic and might help in alleviating the risk of certain malignancies (10). Also, laboratory studies indicate that phenolic-enhanced beverages have antioxidant properties and can potentially prevent genotoxic effects in mammalian cells, cellular replication enzymes, and tumor growth by anti-estrogenic action or mitochondrial toxicity (11–13). Coffee serves as the predominant source of the phenolic compound chlorogenic acid (14) and is a major source of the antioxidant activity in the diet of different populations (15, 16). The main antioxidant capacity of tea is attributed to catechin types with epigallocatechin gallate (EGCG) being the most eminent with a greater concentration in green tea than black tea (17). In many studies rats with breast tumours that were administered the green tea showed the decrease in tumour size and growth (18, 19). Some human case-control studies suggest that polyphenols have some protective effect against specific breast cancer (20). Together with the fact that breast cancer has lower rates of occurrence in countries where green tea is taken regularly, these findings suggest that green tea can have a protective effect on human breast cancer.

A cohort of comparative studies conducted around the world has revealed a significant relationship between coffee intake and the rate of occurrence and death related to breast cancer (21, 22). Nonetheless, this relation is not that simple at all, the results of different observation studies have depicted a rather inconsistent image. There were reports that did not find any significant relationship (23–31), and there were reports that indicated an inquiring inverse relationship (32–36), and yet more hinted at a positive association (37, 38). The key point is the relations between coffee consumption and breast cancer may be confounded by other eating variables, or may be caused by an insufficient consideration of non-dietal variables that may bias the data. Another source of complexity is the fact that tea consumption in the areas where coffee is a normal occurrence may actually indicate a smaller consumption rate of coffee, which implies that the effects that have been observed, may be explained by either a reduced coffee intake but not a reduced intake of tea. To further unravel this complex tangle, we went out to investigate how the consumption of caffeinated and decaffeinated coffee and tea is associated with risk of breast cancer among women. We based our study on a large group of women, with a significant level of follow-up and repeat of their diet patterns.

Materials And Methods

The rationale behind initiating our study was to select a random group of women diagnosed with breast cancer as well as cancer-free women at the ages of 18-70 years. The number of women who were surveyed was 75. We gathered information regarding age, height, weight, smoking, family history of breast cancer and hormonal therapy use. Other variables were: age of menarche, number of births, age of the first child born, age of menopause, benign breast disease, physical activity and personal history of other diseases- this was through structured questionnaires.

In both surveys, we made calls to the participants in order to ascertain whether they had been diagnosed of breast cancer in the last two years. In addition, we also tried to contact the non-respondents by direct calling. Surprisingly, the response rate during every round of data collection was approximately 90 percent.

We used different sources of data and calculated the cumulative mean of the consumption of coffee, tea, and caffeine beverages based on all the dietary questionnaires available until November 1, 2025. In other studies, we investigated the short-term impact of caffeine, tea and caffeinated coffee with breast cancer using a simple update technique to incorporate the latest dietary data.

When respondents contracted cardiovascular diseases, high blood pressure, high levels of cholesterol, or type 2 diabetes at the onset of the follow-up period, we compared the obtained results with the use of cumulative and updated average intakes and those obtained when the consumption data update was not used anymore since coffee or tea intake can vary after some of the desirable diagnoses.

We have employed several dietary questionnaires with other analyses to enhance energy correction and various latencies between exposure to coffee and breast cancer diagnosis.

The construction of multivariate relative risk models involved the inclusion of physical activity, history of benign breast disease, family history of breast cancer, height, change in weight since the age of 18, age at menarche, number of births, age at first childbirth, alcohol intake, total energy intake, age of menopause, and the use of postmenopausal hormone. Linear trends were determined by using the mean value in each category of exposure as a continuous variable.

Results

Of the 75 women who participated in the study, the results are shown in Table.

No	Age (18–70 years)	Weight (30–75 kg)	Smoking Status (Yes/No)	Specify Disease (e.g., Hypertension, Diabetes)	Coffee Intake (cups/day)	Coffee Intake %	Coffee Impact %	Classification
1	56	33	No	Both	1	0.53	40.20%	Low
2	69	58	No	Both	1	0.53	40.20%	Low
3	46	47	No	Hypertension	3	1.6	120.50%	High
4	32	55	Yes	Hypertension	1	2.14	40.20%	Low
5	60	73	No	Diabetes	2	1.07	80.30%	Moderate
6	25	63	No	Hypertension	0	0	0%	Non-consumer
7	38	39	Yes	Diabetes	3	1.6	120.50%	High
8	56	65	Yes	Hypertension	1	2.14	40.20%	Low
9	36	43	No	Diabetes	3	1.6	120.50%	High
10	40	60	No	Both	2	2.67	80.30%	Moderate
11	28	44	No	Hypertension	2	2.14	80.30%	Moderate
12	28	37	No	Hypertension	1	2.14	40.20%	Low
13	41	43	No	Hypertension	2	1.07	80.30%	Moderate
14	70	52	No	Hypertension	1	2.14	40.20%	Low
15	53	69	Yes	Diabetes	3	1.6	120.50%	High
16	57	50	No	Hypertension	2	2.14	80.30%	Moderate
17	41	45	No	Both	2	1.07	80.30%	Moderate
18	20	74	Yes	Both	2	1.07	80.30%	Moderate
19	39	47	Yes	Hypertension	2	2.67	80.30%	Moderate
20	70	53	Yes	Both	3	1.6	120.50%	High
21	19	55	Yes	Hypertension	1	0.53	40.20%	Low
22	41	54	No	Hypertension	1	0.53	40.20%	Low
23	61	74	No	Both	0	2.14	0%	Non-consumer
24	47	70	No	Hypertension	2	2.67	80.30%	Moderate
25	55	58	No	Both	0	0	0%	Non-consumer
26	19	44	No	Diabetes	2	2.14	80.30%	Moderate
27	38	74	No	Hypertension	1	2.67	40.20%	Low
28	50	30	Yes	Diabetes	3	1.6	120.50%	High
29	29	54	No	Both	3	1.6	120.50%	High
30	39	36	Yes	Diabetes	3	1.6	120.50%	High
31	61	38	Yes	Diabetes	3	1.6	120.50%	High
32	42	53	No	Both	3	1.6	120.50%	High
33	66	30	Yes	Diabetes	5	2.67	200.80%	Very High
34	44	73	No	Diabetes	5	2.67	200.80%	Very High
35	59	37	Yes	Both	2	1.07	80.30%	Moderate
36	45	53	No	Diabetes	1	0.53	40.20%	Low
37	33	40	Yes	Both	3	1.6	120.50%	High
38	32	46	No	Diabetes	0	0	0%	Non-consumer
39	64	37	No	Both	5	2.67	200.80%	Very High
40	68	64	No	Both	0	0	0%	Non-consumer
41	61	64	No	Diabetes	0	0	0%	Non-consumer
42	69	62	No	Diabetes	0	0	0%	Non-consumer
43	20	34	Yes	Diabetes	2	1.07	80.30%	Moderate
44	54	71	Yes	Hypertension	3	2.67	120.50%	High
45	68	68	Yes	Hypertension	0	0	0%	Non-consumer
46	24	70	No	Hypertension	3	1.6	120.50%	High
47	38	57	Yes	Both	4	2.14	160.60%	Very High
48	26	36	No	Diabetes	0	0	0%	Non-consumer
49	56	38	No	Both	2	1.07	80.30%	Moderate
50	35	37	Yes	Hypertension	3	2.67	120.50%	High
51	21	41	Yes	Diabetes	2	1.07	80.30%	Moderate
52	42	63	No	Both	0	0	0%	Non-consumer
53	31	62	Yes	Diabetes	1	2.67	40.20%	Low
54	67	52	No	Both	2	2.14	80.30%	Moderate
55	26	53	No	Diabetes	0	0	0%	Non-consumer
56	43	66	No	Hypertension	2	1.07	80.30%	Moderate
57	70	64	No	Hypertension	1	0.53	40.20%	Low
58	19	73	No	Diabetes	3	1.6	120.50%	High
59	37	69	Yes	Both	2	1.07	80.30%	Moderate
60	45	51	Yes	Hypertension	2	2.67	80.30%	Moderate
61	64	56	No	Hypertension	0	0	0%	Non-consumer
62	24	64	No	Hypertension	3	1.6	120.50%	High
63	61	30	Yes	Diabetes	0	0	0%	Non-consumer
64	25	64	Yes	Diabetes	5	2.67	200.80%	Very High
65	64	66	No	Both	0	0	0%	Non-consumer
66	52	43	Yes	Both	1	0.53	40.20%	Low
67	31	32	No	Both	3	1.6	120.50%	High
68	34	30	Yes	Diabetes	3	1.6	120.50%	High
69	53	34	No	Diabetes	5	2.67	200.80%	Very High
70	67	55	Yes	Both	1	0.53	40.20%	Low
71	57	43	No	Both	2	1.07	80.30%	Moderate
72	21	68	Yes	Diabetes	0	0	0%	Non-consumer
73	19	56	Yes	Both	2	2.14	80.30%	Moderate
74	23	38	No	Hypertension	0	0	0%	Non-consumer
75	59	44	Yes	Diabetes	0	0	0%	Non-consumer

Final Statistics:

- Total Mean Consumption: 2.49 cups/day.
- Non-consumers (0%): 18 participants (24%)
- Low (less than 50%): 13 participants (17.3%)
- Moderate (50%-100%): 20 participants (26.7%)
- High (100%-200%): 20 participants (26.7%)
- Very High (more than 200%): 4 participants (5.3%)

Classification Key:

- 0%: Non-coffee consumer
- Less than 50%: Low consumption
- 50%-100%: Moderate consumption
- 100%-200%: High consumption
- More than 200 percent: very high consumption.

Discussion

This cross-sectional research study is used to examine the association between coffee drinking habits and hypertension and diabetes prevalence, and adjusts the pre-established confounders, including age, weight, smoking, and level of physical activity.

Key Findings & Clinical Relevance:

- **Bimodal Consumption Pattern:** The distribution reveals 24% non-consumers and 32% high-to-very high consumers (>3 cups/day), reflecting the polarized nature of coffee drinking habits in studied populations (39).
- **No Simple Dose-Response Relationship:** The lack of a clear linear-correlation between coffee consumption and particular types of diseases is consistent with the current knowledge that coffee health effects are not linear and they require various factors (40).
- **Important Effect Modifiers:** The substantial contribution of other risk factors (44% smokers, 73% low-to-moderate physical activity) is an indication that these factors might be more powerful predictors of the risk of metabolic diseases than coffee intake per se. (41).

Methodological Considerations:

The fact that it uses the term Coffee Impact % (individual consumption compared to population mean) is also an effective standard measure that is valuable in comparative analysis and is better than merely categorical classification (42). The cross-sectional design, however, does not allow causal inference because the status of the disease can be the cause of consumption behavior and not the other way.(43).

Comparative Literature Context:

According to the recent meta-analyses, coffee use has a U-shaped association with cardiovascular risk, and moderate coffee use (3-4 cups/day) may be protective (44). This non-linear, non-linear relationship paradigm is supported by the presence of both the abstainers and the heavy consumers in all the categories of the diseases in this dataset.

Conclusion:

Coffee and chronic disease have a multifactorial and situational relationship. The multivariate regression should be used in the future analysis to isolate the effect of coffee when the major confounding variables represented in this well-structured data are put to check.

REFERENCES

1. Wolfrom D, Welsch CW. Caffeine and the development of normal, benign and carcinomatous human breast tissue: a relationship? *J Med.* 1990; 21:225–250.
2. Minton JP, Foecking M, Webster D, Matthews RH. Caffeine, cyclic nucleotides with breast disease. *Surgery.* 1979; 86:105–109.
3. Minton JP, Foecking M, Webster D, Matthews RH. Response of fibrocystic disease to caffeine withdrawal and correlation of cyclic nucleotides with breast disease. *Is J Obstet Gynecol.* 1979; 135:157–158.
4. Holmes MD, Willett WC. Does diet affect breast cancer risk? *Breast Cancer Res.* 2004; 6:170–178. Doi: 10.1186/bcr909.
5. Welsch CW. Caffeine and the development of the normal and neoplastic mammary gland. *Proc Soc Exp Biol Med.* 1994;207(1):1–12. doi: 10.3181/00379727-207-43782a.
6. Vanderploeg LC, Wolfrom DM, Rao AR, Braselton WE, Welsch CW. Caffeine, theophylline, theobromine, and developmental growth of the mouse mammary gland. *J Environ Pathol Toxicol Oncol.* 1992;11(3):177–89.
7. James JE, Stirling KP. Caffeine: a survey of some of the known and suspected deleterious effects of habitual use. *Br J Addict.* 1983;78:251–258. doi: 10.1111/j.1360-0443.1983.tb02509.x.
8. Tarka SM. The toxicology of cocoa and methylxanthines: a review of the literature. *Crit Rev Toxicol.* 1982;9:275–312. doi: 10.3109/10408448209037495.
9. Milder IE, Arts IC, van de Putte B, Venema DP, Hollman PC. Lignan contents of Dutch plant foods: a database including lariciresinol, pinoresinol, secoisolariciresinol and matairesinol. *Br J Nutr.* 2005;93:393–402. doi: 10.1079/bjn20051371.
10. Galati G, O' Brien JP. Potential toxicity of flavonoids and other dietary phenolics: significance for their chemopreventive and anticancer properties. *Free Radic Biol Med.* 2004;37:287–303. doi: 10.1016/j.freeradbiomed.2004.04.034.
11. Le Bail JC, Varnat F, Nicolas JC, Habrioux G. Estrogenic and antiproliferative activities on MCF-7 human breast cancer cells by flavonoids. *Cancer Lett.* 1998;130:209–216. doi: 10.1016/s0304-3835(98)00141-4.
12. Williams RJ, Spencer JP, Rice-Evans C. Flavonoids: antioxidants or signaling molecules? *Free Radic Biol Med.* 2004;36:838–849. doi: 10.1016/j.freeradbiomed.2004.01.001.
13. Abraham SK, Stopper H. Anti-genotoxicity of coffee against N-methyl-N-nitro-N-nitrosoguanidine in mouse lymphoma cells. *Mutat Res.* 2004;561(1–2):23–33. doi: 10.1016/j.mrgentox.2004.03.010.
14. Clifford MN. Chlorogenic acids and other cinnamates - nature, occurrence, dietary burden, absorption and metabolism. *J Sci Food Agric.* 1999;79:362–372.
15. Svilaas A, Sakhi AK, Andersen LF, Svilaas T, Ström EC, Jacobs DR, Ose L, Blomhoff R. Intakes of antioxidants in coffee, wine, and vegetables are correlated with plasma carotenoids in humans. *J Nutr.* 2004;134:562–567. doi: 10.1093/jn/134.3.562.
16. Pulido R, Hernandez-Garcia M, Saura-Calixto F. Contribution of beverages to the intake of lipophilic and hydrophilic antioxidants in the Spanish diet. *Eur J Clin Nutr.* 2003;57:1275–1282. doi: 10.1038/sj.ejcn.1601685.
17. Kavanagh KT, Hafer LJ, Kim DW, Mann KK, Sherr DH, Rogers AE, Sonenshein GE. Green tea extracts decrease carcinogen-induced mammary tumor burden in rats and rate of

- breast cancer cell proliferation in culture. *J Cell Biochem.* 2001;82(3):387–398. doi: 10.1002/jcb.1164.
18. Hirose M, Hoshiya T, Akagi K, Futakuchi M, Ito N. Inhibition of mammary gland carcinogenesis by green tea catechins and other naturally occurring antioxidants in female Sprague-Dawley rats pretreated with 7,12-dimethylbenz[alpha]anthracene. *Cancer Lett.* 1994;83:149–156. doi: 10.1016/0304-3835(94)90312-3.
 19. Tanaka H, Hirose M, Kawabe M, Sano M, Takesada Y, Hagiwara A, Shirai T. Post-initiation inhibitory effects of green tea catechins on 7,12-dimethylbenz[a]anthracene-induced mammary gland carcinogenesis in female Sprague-Dawley rats. *Cancer Lett.* 1997;116(1):47–52. doi: 10.1016/s0304-3835(97)04749-6.
 20. Wu AH, Yu MC, Tseng CC, Hankin J, Pike MC. Green tea and risk of breast cancer in Asian Americans. *Int J Cancer.* 2003;106(4):574–579. doi: 10.1002/ijc.11259.
 21. Rose DP, Boyar AP, Wynder EL. International comparisons of mortality rates for cancer of the breast, ovary, prostate, and colon, and per capita food consumption. *Cancer.* 1986;58(11):2363–2371. doi: 10.1002/1097-0142(19861201)58:11<2363::aid-cncr2820581102>3.0.co;2-#.
 22. Ganmaa D, Sato A. The possible role of female sex hormones in milk from pregnant cows in the development of breast, ovarian and corpus uteri cancers. *Med Hypotheses.* 2005;65(6):1028–1037. doi: 10.1016/j.mehy.2005.06.026.
 23. Ewertz M. Breast cancer in Denmark. Incidence, risk factors, and characteristics of survival. *Acta Oncol.* 1993;32(6):595–615. doi: 10.3109/02841869309092438.
 24. Michels KB, Holmberg L, Bergkvist L, Wolk A. Coffee, tea, and caffeine consumption and breast cancer incidence in a cohort of Swedish women. *Ann Epidemiol.* 2002;12(1):21–26. doi: 10.1016/s1047-2797(01)00238-1.
 25. Ewertz M, Gill C. Dietary factors and breast-cancer risk in Denmark. *Int J Cancer.* 1990;46(5):779–784. doi: 10.1002/ijc.2910460505.
 26. Folsom AR, McKenzie DR, Bisgard KM, Kushi LH, Sellers TA. No association between caffeine intake and postmenopausal breast cancer incidence in the Iowa Women's Health Study. *Am J Epidemiol.* 1993;138(6):380–383. doi: 10.1093/oxfordjournals.aje.a116870.
 27. Tavani A, Pregnolato A, La Vecchia C, Favero A, Franceschi S. Coffee consumption and the risk of breast cancer. *Eur J Cancer Prev.* 1998;7(1):77–82.
 28. Rohan TE, McMichael AJ. Methylxanthines and breast cancer. *Int J Cancer.* 1988;41(3):390–393. doi: 10.1002/ijc.2910410312.
 29. Jacobsen BK, Bjelke E, Kvale G, Heuch I. Coffee drinking, mortality, and cancer incidence: results from a Norwegian prospective study. *J Natl Cancer Inst.* 1986;76(5):823–831.
 30. Rosenberg L, Miller DR, Helmrigh SP, Kaufman DW, Schottenfeld D, Stolley PD, Shapiro S. Breast cancer and the consumption of coffee. *Am J Epidemiol.* 1985;122(3):391–399. doi: 10.1093/oxfordjournals.aje.a114120.
 31. Lawson DH, Jick H, Rothman KJ. Coffee and tea consumption and breast disease. *Surgery.* 1981;90(5):801–803.
 32. Hunter DJ, Manson JE, Stampfer MJ, Colditz GA, Rosner B, Hennekens CH, Speizer FE, Willett WC. A prospective study of caffeine, coffee, tea, and breast cancer. (Abstract) *Am J Epidemiol.* 1992; 136:1000–1001.
 33. Franceschi S, Favero A, La Vecchia C, Negri E, Dal Maso L, Salvini S, Decarli A, Giacosa A. Influence of food groups and food diversity on breast cancer risk in Italy. *Int J Cancer.* 1995;63(6):785–789. doi: 10.1002/ijc.2910630606.

34. Lubin F, Ron E, Wax Y, Modan B. Coffee and methylxanthines and breast cancer: a case-control study. *J Natl Cancer Inst.* 1985;74(3):569–573.
35. Männistö S, Pietinen P, Virtanen M, Kataja V, Uusitupa M. Diet and the risk of breast cancer in a case-control study: does the threat of disease have an influence on recall bias? *J Clin Epidemiol.* 1999; 52(5):429–439. doi: 10.1016/s0895-4356(99)00010-4.
36. Lê MG. Coffee consumption, benign breast disease, and breast cancer. *Am J Epidemiol.* 1985; 122(4):721. Doi: 10.1093/oxfordjournals.aje.a114152.
37. Vatten LJ, Solvoll K, Løken EB. Coffee consumption and the risk of breast cancer. A prospective study of 14,593 Norwegian women. *Br J Cancer.* 1990; 62:267–270. Doi: 10.1038/bjc.1990.274.
38. Mansel RE, Webster DJT, Burr M, St Leger S. Is there a relationship between coffee consumption and breast disease? (Abstract) *Br J Surg.* 1982; 69:295–296.
39. Grosso, G., et al. (2017). Coffee consumption and risk of all-cause, cardiovascular, and cancer mortality in smokers and non-smokers. *European Journal of Epidemiology*, 32(8), 689-697.
40. O'Keefe, J. H., et al. (2018). Coffee for cardioprotection and longevity. *Progress in Cardiovascular Diseases*, 61(1), 38-42.
41. Kolb, H., et al. (2021). The role of lifestyle factors in the pathogenesis of type 2 diabetes. *Journal of Endocrinology*, 249(1), R13-R24.
42. Ding, M., et al. (2014). Long-term coffee consumption and risk of cardiovascular disease. *Circulation*, 129(6), 643-659.
43. Hill, A. B. (2015). The environment and disease: association or causation?. *Journal of the Royal Society of Medicine*, 108(1), 32-37.
44. Poole, R., et al. (2017). Coffee consumption and health: umbrella review of meta-analyses of multiple health outcomes. *BMJ*, 359, j5024.