ORIGINAL ARTICLE

Association between coffee or tea drinking and Barrett's esophagus or esophagitis: an Italian study

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BACKGROUND/OBJECTIVES: Only a few papers have treated of the relationship between Barrett's esophagus (BE) or erosive esophagitis (E) and coffee or tea intake. We evaluated the role of these beverages in BE and E occurrence.
SUBJECTS/METHODS: Patients with BE (339), E (462) and controls (619) were recruited. Data on coffee and tea and other individual characteristics were collected using a structured questionnaire.
RESULTS: BE risk was higher in former coffee drinkers, irrespective of levels of exposure (cup per day; $\leqslant 1$: OR = 3.76, 95% CI 1.33–10.6; > 1: OR = 3.79, 95% CI 1.31–11.0; test for linear trend (TLT) $P = 0.006$) and was higher with duration (> 30 years: OR = 4.18, 95% CI 1.43–12.3; TLT $P = 0.004$) and for late quitters, respectively ($\leqslant 3$ years from cessation: OR = 5.95, 95% CI 2.19–16.2; TLT $P < 0.001$). The risk of BE was also higher in subjects who started drinking coffee later (age > 18 years: OR = 6.10, 95% CI 2.15–17.3). No association was found in current drinkers, but for an increased risk of E in light drinkers (< 1 cup per day OR = 1.85, 95% CI 1.00–3.43). A discernible risk reduction of E (about 20%, not significant) and BE (about 30%, $P < 0.05$) was observed in tea drinkers.
CONCLUSIONS: Our data were suggestive of a reduced risk of BE and E with tea intake. An adverse effect of coffee was found among BE patients who had stopped drinking coffee. Coffee or tea intakes could be indicative of other lifestyle habits with protective or adverse impact on esophageal mucosa.

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INTRODUCTION

Barrett's esophagus (BE) is a condition in which the normal esophageal squamous mucosa is replaced by a metaplastic columnar mucosa, conferring a predisposition to esophageal adenocarcinoma. In Western areas, the prevalence of BE is estimated to be between 0.5 and 6.8%, to arrive at 15% in symptomatic patients.[1] In Italy it has been estimated to be around 1.5%.[2,3]

Chronic gastroesophageal reflux disease (GERD), a spectrum of hiatal hernia, gastroesophageal reflux, heartburn and regurgitation is a risk factor for both BE and erosive esophagitis (E). It is worth noting that GERD is not always present in all patients with endoscopic diagnosis of BE or E and they may also share other modifiable risk factors such as smoking and overweight.[1,4,5]

So far, epidemiologic data regarding coffee and tea consumption and risk of BE and E in Western areas are scarce and inconclusive. Only a few papers examined the relationship between coffee or tea intake and BE or E, while more data exist on the association with some types of cancer.

Coffee consumption did not seem to be associated with risk of BE, nor with GERD.[6–8]

A recent study did not also support a correlation between tea intake and the risk of BE,[6] while there was a reduced risk of esophageal and other digestive cancers in Asian areas, at higher consumption of green tea.[9] On the other side, no association with cancers of the esophagus was found integrating a series of case–control studies conducted in Italy.[10]

About 90% of adults drink espresso coffee in Italy, while consumption of tea, in particular black tea, is still low.

In this multicenter case–control study we sought to assess the relationship between coffee, tea and herbal tea consumption and risk of BE and E.

MATERIALS AND METHODS

Between March 2009 and October 2012, 339 BE patients, 462 E patients and 619 C with no BE or E undergoing upper gastrointestinal endoscopy for other digestive disorders were consecutively enrolled in 12 Endoscopic Units in the North ($n = 5$), Center ($n = 2$) and South ($n = 5$) of Italy.

BE cases were enrolled among those with an endoscopic 15 mm upward displacement of the squamocolumnar junction (Z-line) from the gastro-esophageal junction at endoscopy, and with specialized intestinal metaplasia with 'goblet' cells on histology.[11] BE length at endoscopy was defined according to the Prague C & M criteria.[12]

The E group was identified among patients with Los Angeles grade A or B reflux esophagitis with mucosal breaks proven by endoscopy.[13] E and C patients were identified in the same centers and in the same period as BE patients.

Multiple biopsies were taken for BE, according to the Seattle protocol, while four biopsies were taken in E patients (two at the Z-line and two at 2 cm above it).[14] Biopsies were interpreted in every center by experienced gastrointestinal pathologists.

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Controls were recruited from subjects undergoing endoscopy for any reason both in the presence or absence of GERD. We defined as GERD positive those patients reporting at least weekly heartburn or pyrosis (i.e. burning retrosternal sensation, rising from the epigastric region and radiating to the neck, throat or angle of the jaw) and/or acid regurgitation (a sour or bitter taste in the mouth) 1 year before diagnosis.[15]

Overall, we elected subjects aged 18 years or older, with no serious chronic diseases and prepared to undergo a questionnaire. Only patients with a new diagnosis of BE or E were recruited. The study was approved by the Ethics Committee in each Center and all participants signed an informed consent.

A questionnaire on symptoms or lifestyle habits preceding the diagnosis of BE and E, or endoscopy for controls, was administered by centrally trained interviewers. The questionnaire covered individual characteristics (weight, height, education, occupation), smoking, alcohol consumption, diet, medical history and presence and duration of GERD symptoms. Each subject was asked to report about lifetime consumption of both alcoholic and non-alcoholic beverages, in particular coffee, decaffeinated coffee, tea and herbal tea.

For coffee and tea, subjects' entire drinking histories were recalled according to his/her drinking status, namely non-drinker, current drinker and former drinker (who had stopped drinking at least 1 year before enrollment). Subjects were considered 'drinkers' if they consumed beverages at least monthly for 6 months or longer. Questions were asked about the frequency of consumption and the number of units consumed on each drinking occasion, age at initiation, duration and, for former drinkers, years since cessation. One unit was equivalent to 1 cup of coffee or tea (about 30 ml and 170 ml, respectively).

Statistical methods

Data were explored through the analysis of contingency tables and χ^2 test was used to assess the independence between each individual characteristic and the three-category health outcome (i.e., BE, E and C). For this reason, continuous variables (e.g., age at interview, BMI) were categorized according to percentiles.

The association between coffee/tea drinking characteristics and health outcome was evaluated using the multinomial logistic regression (MLR) modeling.[16] MLR is a particular logistic modeling in that it allows to carry out simultaneously two binary comparisons, namely E vs C and BE vs C. Within each comparison, odds ratio (OR), along with the corresponding and 95% confidence interval (95% CI), was computed and considered as an index of association between each binary health outcome and each putative risk factor.

Coffee/tea drinking habit can be represented through some quantitative characteristics (frequency of consumption, number of units consumed,

Factors and levels	Control		Esophagitis		Barrett's esophagus		P-value
	n	%	n	%	n	%	
Gender			< 0.001				
Male	252	40.7	285	61.7	229	67.6	
Female	367	59.3	177	38.3	110	32.4	
Age at interview (years)							< 0.001
< 41	126	20.4	113	24.5	58	17.1	
41–50	129	20.8	92	19.9	66	19.5	
51–60	143	23.1	90	19.5	66	19.5	
61–68	129	20.8	101	21.9	58	17.1	
> 68	93	15.0	66	14.3	91	26.8	
Smoking status							0.001
Never smoker	330	53.3	218	47.2	135	39.8	
Former smoker	156	25.2	138	29.9	126	37.2	
Cig/day ⩽ 13	71	11.5	51	11.0	57	16.8	
Cig/day > 13	85	13.7	87	18.8	69	20.4	
Current smoker	133	21.5	106	22.9	78	23.0	
Cig/day ⩽ 13	72	11.6	45	9.7	38	11.2	
Cig/day > 13	61	9.9	61	13.2	40	11.8	
Alcohol habit							0.001
Never drinker	169	27.3	114	24.7	77	22.7	
Former drinker	29	4.7	16	3.5	29	8.6	
Current drinker	304	49.1	260	56.3	194	57.2	
Ever drinker	117	18.9	72	15.6	39	11.5	
Body mass index							< 0.001
< 22.28	157	25.4	64	13.9	60	17.7	
22.28–24.22	148	23.9	86	18.6	65	19.2	
24.23–25.95	115	18.6	98	21.2	60	17.7	
25.96–28.09	104	16.8	103	22.3	76	22.4	
> 28.09	95	15.3	111	24.0	78	23.0	
Duration of GERD							< 0.001
Never	280	45.2	91	19.7	73	21.5	
< 3 years	201	32.5	184	39.8	51	15.0	
3–10 years	92	14.9	142	30.7	127	37.5	
> 10 years	46	7.4	45	9.7	88	26.0	
Years of schooling							< 0.001
< 6	193	31.2	104	22.5	96	28.3	
6–8	216	34.9	145	31.4	85	25.1	
9–13	183	29.6	171	37.0	116	34.2	
> 13	27	4.4	42	9.1	42	12.4	
Whole sample	619	100.0	462	100.0	339	100.0	1420

Table 1. Baseline characteristics of Barrett's esophagus, esophagitis and controls

Table 2. Relative risk of Barrett's esophagus and esophagitis according to coffee drinking habit estimated through multinomial logistic regression modeling among former drinkers using never drinkers as a reference

Model	Coffee drinking characteristics		Former drinkers (n = 131) vs non-drinkers (n = 158)					
			E vs C			BE vs C		
	Covariates	Main predictor	OR	95% CI		OR	95% CI	
1	Years of duration	Cups per day						
	Age at initiation	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
	Years since cessation	≤ 1	1.22	0.39	3.75	3.76	1.33	10.6
		> 1	1.09	0.36	3.33	3.79	1.31	11.0
		Test for linear trend	0.823			0.006		
2	Cups per day	Years of duration						
	Age at initiation	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
	Years since cessation	≤ 30	1.59	0.50	5.08	3.37	1.10	10.3
		> 30	0.88	0.29	2.72	4.18	1.43	12.3
		Test for linear trend	0.975			0.004		
3	Cups per day	Years since cessation						
	Age at initiation	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
	Years of duration	> 3	0.94	0.30	2.96	2.19	0.71	6.76
		≤ 3	1.39	0.46	4.19	5.95	2.19	16.2
		Test for linear trend	0.619			< 0.001		
4	Cups per day	Age at initiation						
	Years since cessation	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
	Years of duration	> 18 years	1.47	0.47	4.58	6.10	2.15	17.3
		≤ 18 years	1.09	0.37	3.19	2.15	0.73	6.33
		Test for linear trend	0.743			0.049		

Abbreviations: BE vs C, Barrett's esophagus patients versus control group; E vs C, esophagitis patients versus control group; OR, odds ratio (relative risk) point estimate, adjusted for age, gender, body mass index, alcohol consumption, years of schooling, duration of GERD and collaborative center; test for linear trend, P-value of the likelihood-based chi-square test for linear trend; ref., reference category; 95% CI, 95% confidence interval for OR.

years of duration, age at initiation and years since cessation), each of which should be properly addressed and analyzed in order to evaluate their distinct effect on individual health status. Such characteristics are generally well correlated and this occurrence (collinearity) can seriously impede to assess their joint role on health outcomes estimated through a regression analysis, and therefore prevent from controlling for the reciprocal confounding effect.[17] In an attempt to reduce this statistical drawback, data were stratified according to drinking status (former and current drinkers) and in each stratum an MLR analysis was performed using non-drinkers as a reference category. Only one quantitative drinking variable (main predictor) at a time entered the regression equation after categorization based on specific thresholds (percentiles) a priori defined on the distribution of the C group. The remaining quantitative characteristics, appropriately transformed (centered), entered the equation as continuous variables (covariates).[17] In addition to quantitative drinking variables, all MLRs also included, as confounding variables, gender, age at interview, years of schooling, body mass index, smoking habit, alcohol drinking, duration of GERD and categorical terms for collaborative centers.

The statistical significance (two-tailed P-value < 0.05) was assessed using the likelihood-based chi-square test for linear trend (TLT).[16] All statistical analyses were performed using STATA software.[18]

RESULTS

Characteristics of BE patients (n = 339), E patients (n = 462) and C subjects (n = 619) are reported in Table 1. Mean age was 56.2 ± 15.2 for BE, 52.6 ± 14.7 for E and 53.7 ± 14.1 for C. Controls had a higher percentage of females (59.3% vs 32.4% BE and 38.3% E) and a lower BMI (49.3% had BMI < 24.23 vs 36.9% of BE and 32.5 of E). C had also a lower percentage of smokers (46.7% vs 60.2% of BE and 52.8% of E). Controls underwent endoscopy because of epigastric pain (38%), regurgitation (25%), dyspepsia (24%), pyrosis or dysphagia (9%), gastric or duodenal ulcer (3%) and anemia (1%). GERD symptoms were reported by 78.5% of BE, 80.3% of E and in 54.8% of C. Among GERD-positive subjects,

80.8% of BE patients had suffered from symptoms for more than 3 years vs 50.4% of E and 40.7% of C.

No differences were observed with regard to percentage of subjects consuming coffee (BE 90.9%, E 87.4% and C 88.9%), but BE were more likely to be former drinkers (BE 16.5% vs C and E 6.9%, P-value < 0.001). When compared with C, BE patients reported a slightly higher frequency (≥3 cups/day: 37.2% vs 31.3% C, P-value = 0.069) and drank for more time (≥30 years: 58.7% vs 50.1%, P-value = 0.011). In addition, BE started drinking at earlier age (age at initiation ≤ 15 years: 19.2% vs 14.4%, P-value = 0.056) and quitted drinking coffee later than C (time since cessation ≤ 3 years: 10.0% vs 3.4% C, P-value < 0.001). In comparison with C subjects, E patients were more likely to be younger when starting drinking (age at initiation ≤ 15 years: 19.0%, P-value = 0.041), while no differences were observed as for frequency (≥3 cups/day: 32.1%), duration (≥30 years: 48.1%) and time since cessation (≤3 years: 3%).

Tables 2 and 3 show the results of MLR modeling reporting the risk of BE and E in former and current coffee drinkers, respectively, using non-drinkers as a reference category. Adjusting for confounding, MLR analysis pointed out a noteworthy risk of BE in former drinkers for all coffee-related predictors (Table 2). In particular, BE risk was high (about fourfold), irrespective of levels of exposure (Table 2, model 1; TLT P-value = 0.006) and was four to six times more with duration (Table 2, model 2, > 30 years: OR = 4.18, 95% CI = 1.43–12.3; TLT P-value = 0.004) and for late quitters (Table 2, model 3; ≤ 3 years: OR = 5.95, 95% CI = 2.19–16.2; TLT P-value < 0.001), respectively. The risk of BE was very high also in subjects who started drinking coffee later (Table 2, model 4; > 18 years: OR = 6.10, 95% CI = 2.15–17.3).

No correlation was found in current drinkers, except an increased risk of E in light drinkers (Table 3, model 1; < 1 cup/day: OR = 1.85, 95% CI = 1.00–3.43).

Table 3. Relative risk of Barrett's esophagus and esophagitis according to coffee drinking habit estimated through multinomial logistic regression modeling among current drinkers using never drinkers as a reference

Model	Coffee drinking characteristics		Current drinkers (n = 1131) vs Non-drinkers (n = 158)					
			E vs C			BE vs C		
	Covariates	Main predictor	OR	95% CI		OR	95% CI	
1	Years of duration	Cups per day						
	Age at initiation	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
		< 1	1.85	1.00	3.43	1.39	0.67	2.87
		1	0.64	0.36	1.15	0.87	0.45	1.68
		2–3	1.26	0.73	2.18	1.23	0.66	2.29
		> 3	0.73	0.34	1.54	1.09	0.48	2.49
		Test for linear trend	0.441			0.797		
2	Cups per day	Years of duration						
	Age at initiation	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
		< 23	1.31	0.60	2.86	0.79	0.33	1.89
		23–34	1.21	0.65	2.24	0.99	0.49	2.02
		35–45	0.98	0.54	1.77	1.17	0.58	2.34
		> 45	0.90	0.45	1.79	1.81	0.82	3.98
		Test for linear trend	0.904			0.228		
3	Cups per day	Age at initiation						
	Years of duration	Non-drinker	1.00	(Ref.)		1.00	(Ref.)	
		> 22 years	1.09	0.59	2.01	1.04	0.52	2.09
		19–22 years	1.49	0.82	2.72	1.01	0.49	2.06
		16–18 years	0.97	0.55	1.70	1.15	0.61	2.17
		< 16 years	0.80	0.40	1.58	1.23	0.57	2.67
		Test for linear trend	0.553			0.539		

Abbreviations: BE vs C, Barrett's esophagus patients versus control group; E vs C, esophagitis patients versus control group; OR, odds ratio (relative risk) point estimate, adjusted for age, gender, body mass index, alcohol consumption, years of schooling, duration of GERD and collaborative center; ref., reference category; test for linear trend, P-value of the likelihood-based chi-square test for linear trend; 95% CI, 95% confidence interval for OR.

Unfortunately, we were unable to apply to decaffeinated coffee, tea and herbal tea data the same regression strategy used for coffee intake given the poorness and sparseness of information on several quantitative characteristics. In this context, Table 4 shows the results of contingency tables and MLR analyses. Only few subjects consumed decaffeinated coffee (BE: 23.0%, E: 18.2% and C: 24.1%), and no remarkable differences in frequency of consumption between the study groups were observed.

Controls were more likely to drink tea, and with a higher frequency, compared with both BE (67.8% vs 55.5%, P-value < 0.001; cups ⩾ 1 week: 39.2% vs 32.3%, P-value = 0.039) and E (48.9%, P-value < 0.001; cups ⩾ 1 week: 31.6%, P-value = 0.017). Tea consumption seemed to decrease the risk of both E and BE, but the observed descending trend is neither monotonic nor statistically significant. However, it is worth noting a discernible risk reduction of E (OR = 0.80, 95% CI = 0.59–1.07, P-value = 0.128) and BE (OR = 0.71, 95% CI = 0.51–0.97, P-value = 0.033) in ever-drinkers when compared with non-drinkers.

No evidence of association between BE or E and herbal tea consumption was found.

DISCUSSION

In this study we investigated coffee and tea consumption in relation to the risk of BE or E. Results were suggestive of a protective effect of tea on BE occurrence, while a remarkable risk of BE for all coffee-related predictors was evidenced.

Coffee intake has been related to a lower risk of a number of cancers at different sites such as liver, prostate, breast and colorectum,[19–23] while it seemed to increase the risk of laryngeal cancer.[24]

The studies on the role of coffee on the occurrence of esophageal diseases did not exclude a weak inverse relationship between coffee intake and esophageal cancers,[25] while a meta-analysis of some Italian observational studies provided evidence of an inverse association with cancers of the oral cavity or pharynx, but not with laryngeal and esophageal cancers.[26] Furthermore, other data from a pool of Italian case–control studies revealed no association with tea consumption.[10]

Data regarding the relationship between tea and cancer are inconsistent. Tea consumption has been inversely associated with all cancers and all-cause mortality;[27] nevertheless, meta-analyses suggested an inverse association of high tea consumption (mainly green tea) only with oral, bladder cancer, leukemia and myeloid malignancies, while less clear data were found for other cancers.[24,28–30] Correlation with esophageal cancer remains unclear, too, with data on a protective effect of green tea, especially in studies conducted among Chinese population,[25] or a risk reduction only for subgroups of patients such as females.[31,32]

Among the few studies on non-neoplastic esophageal diseases, a meta-analysis showed that overall coffee did not seem to be a causal factor for GERD, while a significantly higher odds ratio was found for E.[8] No association was found between coffee intake and the presence of BE in an our previous study;[33] furthermore, results from a US survey did not support an association between consumption of coffee or tea and the risk of BE.[6] No other data exist on the relationship between BE and tea consumption, but for a suggestion of green tea as an a potential chemopreventive agent for esophageal adenocarcinoma and BE because of the presence of polyphenols able to inhibit the growth of human Barrett's and aerodigestive adenocarcinoma cells.[34] Some authors, nevertheless, reported of an amount of heartburn due to tea intake when compared with water.[35]

Despite the uncertainty on the role of coffee on esophageal tissue, there are some mechanisms supporting the hypothesis of a beneficial or harmful effect. Overall, coffee, including the

Table 4. Joint distribution of disease status and decaffeinated coffee, tea and herbal tea consumption, and results of multinomial logistic regression modeling

Beverage (cups)	Control		Esophagitis		Barrett's esophagus		E vs C			BE vs C		
	n	%	n	%	n	%	OR	95% CI		OR	95% CI	
Decaffeinated coffee												
Non-drinker	455	73.5	275	59.5	241	71.1	1.00	(Ref.)		1.00	(Ref.)	
< 1 months	50	8.1	30	6.5	21	6.2	1.04	0.63	1.71	0.76	0.42	1.38
1–3 months	25	4.0	12	2.6	15	4.4	0.94	0.45	1.97	1.46	0.69	3.06
1–4 weeks	42	6.8	25	5.4	28	8.3	1.01	0.58	1.74	1.37	0.78	2.41
> 5 weeks	32	5.2	17	3.7	14	4.1	0.88	0.46	1.67	0.86	0.42	1.77
Unknown	15	2.4	103	22.3	20	5.9	—	—	—	—	—	—
Test for linear trend							0.781			0.581		
Tea												
Non-drinker	185	29.9	132	28.6	131	38.6	1.00	(Ref.)		1.00	(Ref.)	
< 1 months	97	15.7	61	13.2	49	14.5	0.87	0.57	1.31	0.70	0.44	1.10
1–3 months	86	13.9	52	11.3	36	10.6	0.73	0.48	1.13	0.49	0.29	0.81
1–4 weeks	109	17.6	53	11.5	51	15.0	0.79	0.52	1.20	0.87	0.56	1.37
> 5 weeks	128	20.7	60	13.0	52	15.3	0.78	0.52	1.16	0.78	0.50	1.21
Unknown	14	2.3	104	22.5	20	5.9	—	—	—	—	—	—
Test for linear trend							0.162			0.266		
Herbal tea												
Non-drinker	292	47.2	186	40.3	171	50.4	1.00	(Ref.)		1.00	(Ref.)	
< 1 months	85	13.7	50	10.8	53	15.6	0.99	0.65	1.50	1.14	0.73	1.77
1–3 months	70	11.3	42	9.1	28	8.3	1.09	0.70	1.71	0.73	0.43	1.25
1–4 weeks	89	14.4	37	8.0	31	9.1	0.84	0.54	1.32	0.79	0.48	1.30
>5 weeks	69	11.1	44	9.5	35	10.3	1.37	0.88	2.15	1.29	0.78	2.13
Unknown	14	2.3	103	22.3	21	6.2	—	—	—	—	—	—
Test for linear trend							0.469			0.983		
Whole sample	619	100.0	462	100.0	339.0	100.0	—	—	—	—	—	—

Abbreviations: BE vs C, Barrett's esophagus patients versus control group; E vs C, esophagitis patients versus control group; OR, odds ratio (relative risk) point estimate, adjusted for age, gender, body mass index, alcohol consumption, years of schooling, duration of GERD and collaborative center; ref., reference category; test for linear trend, *P*-value of the likelihood-based chi-square test for linear trend; 95% CI, 95% confidence interval for OR.

decaffeinated type, may cause lower-esophageal-sphincter dysfunction and GERD in susceptible subjects.[36,37] In addition, it contains potentially carcinogenic compounds, including acrylamide.[23]

On the other side, coffee contains polyphenols that inhibit harmful oxidation processes in the body.[38] Unfortunately, we were not able to evaluate the different types of coffee, as well as coffee processing and the method used for roasting. The issue is controversial, but these characteristics might explain some of the variability in esophageal tissue response to coffee consumption.[39–41]

The poorness of information did not allow us to better analyze some quantitative characteristics of tea consumption or to distinguish between green and black tea type. Anyway, it might be difficult to compare our results with other studies since the majority of Italian people consume black tea, and at low levels, while most reports on this topic are from Asian areas where green tea consumption is largely prevalent. As already said, green tea contains high concentrations of polyphenols that have shown inhibitory effects against the development and growth of carcinogen-induced tumors in animal models at different organs, including the esophagus. In humans, green tea polyphenols may suppress cell proliferation and induce apoptosis.[42] This favorable biological activity is nevertheless reduced with the process of black tea production.[43]

The consumption of beverages at high temperature may be a risk factor for esophageal diseases,[44,45] but coffee and tea temperature did not seem to influence the risk of BE among a western population.[6]

Comparisons among the studies on the role of beverages on esophageal diseases may be hampered by different characteristics of the studies. A determinant of esophageal disorders is often the presence of GERD, so the lack of information on the presence of GERD may have led authors to underestimate the role of studied beverages.[46] Moreover, population controls might have an undiagnosed BE or GERD, even though BE is rarely diagnosed among healthy volunteers and is found in less than 10% of subjects with severe reflux.[47,48] Contrasting results may be also obtained with adjustment for different confounders or when considering different temporalities of the associations. With this regard, it is possible that the cases or GERD controls have changed their drinking habits in the years because of symptoms or diagnosis of esophageal abnormality.

In addition, an underestimation of the observed effects may be present, due to the fact that controls underwent endoscopy because of dyspeptic symptoms. There is also the possibility of a recall bias and some confounders, such as diet, were not taken into account. Few data exist on the association between diet and BE: an inverse relationship between fruits, vegetables and antioxidants intake and the risk of developing BE has been reported, while a diet rich in meat and fast food moderately increased the risk in subjects without GERD.[49]

To the best of our knowledge, this is the first study analyzing the association between coffee and tea and BE or E in Italian regions. In Italy consumption of these beverages is presumably different from that in other studies and controls did not seem to differ from the Italian population with regard to coffee consumption. Furthermore, all study subjects had an endoscopic diagnosis, BE and E were incident cases and we could control for intensity of GERD symptoms.

In conclusion, our data are suggestive of a reduced risk of BE and E with tea intake, while an adverse effect of coffee was found

among BE patients who had stopped drinking coffee, probably due to the development of symptoms or diagnosis. These results are interesting; nevertheless, there is the possibility that coffee or tea intakes be indicative of other lifestyle or food habits with protective or adverse impact on esophageal mucosa.

CONFLICT OF INTEREST

The authors declare no conflict of interest.

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